Frequently Asked Questions

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CE for Ohio Dental Professionals

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Chapter 1: Facing the Future: NIDCR Researchers Offer Their Vision of the 21st Century

3 CE Hour

By: Elite Staff

Learning objectives

- Review scientific research on craniofacial development.
- Identify National Institute of Dental and Craniofacial Research (NIDCR) ideas on the systematic model of craniofacial development.
- Explain how neural crest cells assist in the development of the formation of the craniofacial plate.
- Identify the development of replacement tissues for damaged teeth.
- Discover the future possibilities to regenerate damaged gingival, ligament and bone.

Introduction

This course presents researchers and their views on related studies of dental and oral research that promise giant steps in the future. The material consists of questions and answers from grantees of the National Institute of Dental and Craniofacial Research. These scientists use the latest molecular and genetic tools to conduct research on the full spectrum of topics related to craniofacial, oral and dental health and disease. In this course, you will meet each of these researchers and review some of the challenges they are facing.

The six researchers and grantees are:

- Dr. Marianne Bronner-Fraser, a biologist at the California Institute of Technology in Pasadena, California.
- Dr. Paul Trainor, a scientist at the Stowers Institute for Medical Research in Kansas City, Missouri.
- Dr. Richard Maas, a scientist at Brigham and Women's Hospital and Harvard Medical School in Boston, Massachusetts.
- Dr. Malcolm Snead, a scientist at the University of Southern California, Los Angeles, California.
- Dr. William Giannobile, a researcher at the University of Michigan in Ann Arbor, Michigan.
- Dr. Pamela Robey, a National Institute of Dental and Craniofacial Research scientist in Bethesda, Maryland.

On its 60th anniversary, the NIDCR looks to the future and the likelihood of a more systematic model of craniofacial development. The pages that follow offer the perspectives of several NIDCR researchers and grantees on the scientific road ahead to meet this challenge. They also portray some of the likely benefits of this research to the nation's public health. These include a detailed picture of where the molecular glitches might arise in the system, for example, to cleft a lip, omit a tooth bud or malform a bone. By knowing the most frequent problem spots and, more generally, how healthy craniofacial structures are made, scientists will be in a much better position in the years ahead to dispense molecular medicine and repair more naturally a congenital problem or heal a diseased tissue. They also will be more attuned to the early molecular warning signs of developing disease. This will allow earlier and more accurate diagnoses to correct problems before they become advanced, chronic and destructive.

As part of this glimpse forward, the NIDCR highlights related areas of dental and oral research that hold tremendous promise. These include studies of head and neck cancer, the development of saliva as a diagnostic fluid, more effective control of orofacial pain, and ongoing hands-on efforts in communities across the nation to help translate the fruits of our science into improved health care.

To tell these stories involves a new language of discovery. These include more familiar terms such as genomics, or the study of genes across species, and proteomics, the companion term for proteins. It also includes more recently minted biological pursuits such as the interactome, the complete set of possible protein interactions within a cell, and the microbiome, the complete set of microorganisms that inhabit distinct parts of the body, such as the mouth, and greatly influence our health and susceptibility to disease over time.

These and other terms represent the need for conceptual distinctions in science. While organizationally helpful, they are in many ways artificial. All human biology is one, from head to toe. As NIDCR-supported research unfolds in the years ahead, its lessons will have broad applications throughout science and, more importantly, in hospitals, clinics and dental offices across the land.

One final note: Although the scientists highlighted here are all outstanding, they represent just a cross section of a much larger community of NIDCR researchers and grantees who are making important contributions to their fields and the nation's public health.

Part I: Neural crest cells: The first mystery of craniofacial development

In 1868, the Swiss embryologist Wilhelm His spotted a thin band of previously undetected cells bunched between fetal ectoderm and the inchoate neural tube of a developing chick. Dr. His called his find the Zwischenstrang, or "the intermediate cord." By the end of the century, the German word Zwischenstrang had been scrapped for the more descriptive English term "neural crest cells," denoting the geographic crest of the neural tube as their site of origin. The cells also had become a topic of controversy. Reports had begun to trickle into the scientific literature that neural crest cells in some fish gave rise to neurons and nerve fibers of the cranium, while those in certain salamanders were proposed to produce cartilage of the head and dentin forming cells of the teeth. Many biologists claimed this was preposterous.

"One hundred years ago, claiming that an ectodermal derivative such as the neural crest was in any way involved with the formation of skeletal structures was the embryological and evolutionary equivalent of nailing an additional thesis to the cathedral door," wrote Langille and Hall in the early 1990s, referring to Martin Luther's famous Protestant rebellion. "That skeletal structures were mesodermal in origin was dogma, known and accepted by all; an ectodermal origin was heresy."

Today, the controversy has ebbed. Scientists have solidly established that these short-lived precursor cells come in four distinct types, all of which are programmed to migrate throughout the body and seed new tissue. Among them are the cranial neural crest cells that, as mentioned above, help to generate most of the distinctive skeletal structures of the head and

face. Although the mystery of neural crest cells historically has attracted anatomists and evolutionary biologists, the last few decades have brought more molecular and cell biologists to the field. The prospect of increased collaboration among the scientific disciplines coupled with the rapid progress in research technology promises to herald a new era of discovery in craniofacial development.

To take a closer look at neural crest cells, craniofacial development and the research job ahead, two NIDCR grantees offer their thoughts. We start with Dr. Marianne Bronner-Fraser, a biologist at the California Institute of Technology in Pasadena, and Dr. Paul Trainor, a scientist at the Stowers Institute for Medical Research in Kansas City, Missouri.

Marianne Bronner-Fraser

Dr. Marianne Bronne-Fraser
California Institute of Technology
Pasadena, California

In studying craniofacial development, why go all the way back to neural crest cells? In other words, why study the Book of Genesis? Why not just cut to the Book of Revelations and fully formed human tissues?

Think of it like this. You wouldn't understand the meaning of Tolstoy's "War and Peace" by flipping to the last few chapters. The same is true here. You've got to start as close to the beginning as possible to follow the biological narrative and, hopefully, tease out its underlying molecular logic and morphological patterns.

What are some of the early features of the narrative?

Well, the initial generation of neural crest cells. It's really quite fascinating. The early embryo forms as three distinct layers of tissue – the exterior ectoderm, the middle mesoderm, and the internal endoderm. By day 19, the interaction of ectoderm and mesoderm produces the neural plate, a precursor of the central nervous system.

And neural crest cells form along the neural plate?

Exactly. The interaction between ectoderm and mesoderm is a classic mode of embryonic tissue formation. That's why the lessons learned here will have relevance to understanding tissue formation elsewhere in the body. That's also why it's essential to define the molecular machinery within the neural crest cells that prompt them to migrate. In other words, which molecular gears and sprockets turn on and off to enable neural crest cells to transition from ectoderm to mesoderm? How does this transition enable them to loosen from the neural tube and migrate, for example, to the heart or the cranium? And, of course, in the context of cranial neural crest cells, it's essential to understand how these cells at first produce what appears to be the same generic, undifferentiated facial primordial in vertebrate species. And yet, neural crest and the surrounding ectodermal cells generate these dynamic development programs that produce vertebrate structures as distinct as the beak of a toucan, the tusk of a boar or the venom-producing salivary gland of a rattle snake.

Your group is looking comparatively up and down the evolutionary ladder not only at vertebrates but also invertebrates, or more primitive creatures that lack a spinal column and thus a head. Why?

That's where many of the answers to vertebrate evolution will be found. People tend to be so human-centric, and because of that, vertebrate-centric. But if you can piece together and understand some of the evolutionary changes that occurred through the millennia from invertebrate to vertebrate, they can be extremely informative in telling us how something as complex as a human head is assembled. In short, we need to listen to the biology, not impose our own mechanistic thoughts and metaphors upon it.

And by listening to the biology, it also will help to explain where things go wrong to cause a malformation, say a cleft lip?

Sure. I think a lot of the answers to birth defects will lie in the early stages of craniofacial development. One of the things that I'm very interested in is the gene regulatory networks in neural crest cells that help to initiate this self-assembly machine that forms a head. You start development in all species with a single-cell fertilized egg. Without giving any obvious hints, that lone cell gives rise to structures and faces as diverse as those of a human being, a finch and a giraffe.

And yet, as complex as craniofacial development is, it usually is completed without a hitch.

That's right, craniofacial development goes right in the vast majority of cases. What's interesting is the same genes that are used early in development also are used later in the process and at multiple times. It seems to be a reiterative process during which you have important genes that are first used to specify a cell type and then later that same gene might be used to tell it to differentiate into a tooth, bone or jaw cell. It's fascinating to realize that the toolkit is not as vast as we once thought it must be. It's the way that toolkit is deployed that proves to be especially important.

Has the genetic activity of neural crest cells during the developmental process been catalogued?

We're getting there. With the full genetic complement, or genomes, of various species now determined, the rate of identifying genes involved in making a head or a heart has increased exponentially. It's left us wading through a huge amount of data. That's exciting in that so many

more pieces to the puzzle are spread out on the table. The problem is this heavy volume makes assembling the puzzle more complex. If I'm dealing with 500 or 1,000 genes to figure out how they work together to create a cell type or render a neural crest cell migratory, I might be really puzzled. If I have a colleague who is looking at it from an opposite approach but happens to identify a subset of those genes, we can take those 1,000 candidate genes and cut them to 10 and begin thinking about communication nodes and signaling networks. What I'm hoping is that by putting these groups together, we can narrow down the key players more quickly. There's going to be common themes running through this. Unless you do something comparatively, you can't see those threads.

And these multidisciplinary groups have been formed and continue to be?

That's right. For example, the NIDCR soon will launch its FaceBase Project. It will bring together scientists of various research backgrounds and establish collaborative consortia that focus on specific sequences of craniofacial development. That will be very constructive. Our best science is ahead of us, and as more of the metaphorical pieces to the neural-crest puzzle are discovered, I think fitting them into a coherent biological picture of craniofacial development will have profound implications for human health and disease.

Paul Trainor

Dr. Paul Trainor
Stowers Institute for Medical Research
Kansas City, Missouri

Has the push for multidisciplinary research been a positive change in the field?

Oh, definitely. I would say that combining disciplines has helped in the last few years to increase the scale and pace of the research in general. You can see that with the NIH Roadmap Initiative. The Roadmap, because of its larger scope, interdisciplinary emphasis and often the uniqueness of its projects, can open up very rapidly numerous unexpected avenues of study and thus a number of spinoff projects. In neural crest research, we're already benefiting from this progress.

How so?

People no longer look at neural crest cells and facial development from a single-gene perspective. That had long been the norm, largely because of technological limitations. People now have a better set of investigative tools at their disposal and are broadly defining the different gene regulatory changes in neural crest cells. They also are taking the next biological step and defining the protein signaling networks that must be activated for initial crest cell formation, and those that subsequently trigger their three-dimensional patterning. That will also impact our understanding of neural crest cell evolution.

But neural crest cells are only a part of the developmental story. There are also the pharyngeal arches that they colonize and which serve as an inductive template – the facial primordial – for craniofacial development.

That's right, it's a two-way street. The neural crest cells certainly don't exist in a vacuum. Historically, we used to think that neural crest cells did a lot, basically by themselves. I'd say that if one thing has changed dramatically in the field over the last five to 10 years, it's been this recognition that, yes, neural crest cells have species-specific programming information. But a lot of what they do is dependent on which tissues they contact during their migration and which signals are received when they reach their final resting place.

A VIEW OF THE NEURAL PLATE AND MIGRATING CRANIAL CREST CELLS

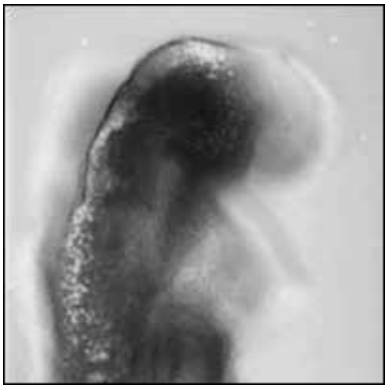
What I'm wondering, though, does the facial primordial serve as the rough blueprint of a vertebrate head?

That may well be. If you look at the different vertebrate species, there are tremendous similarities in the initial formation and migration of neural crest cells into the pharyngeal arches, also called the branchial arches in fish. There's no doubt about that. But if you fast-forward the developmental process and look at the diversity of facial structures that arise across the vertebrate spectrum, they are dramatically different from the trunk of an elephant to the tusks of a boar. So what is highly conserved in nature is this early basic blueprint of facial development, and then the diversification themes that take place beyond that time point.

When you say "diversification themes," that's where neural crest cells play such a major role?

Correct. Neural crest cells are endowed with an innate plasticity, or an ability to make developmental modifications. That means the genetic program wired into the neural crest cells don't need to be modified at the neural tube, that is, the very front end of their life cycle. The

modifications can occur throughout the developmental process. By that, I mean cranial neural crest cells can be influenced by molecular factors during their migration to the pharyngeal arches. Or they might arrive there at a new position, where they have multiple tissue interactions that modify their location, orientation, and ultimately their fates.



Neural crest cells are endowed with innate plasticity.

And it's this plasticity that leads to diversity?

That's right. The plasticity allows such tremendous variation. If everything was rigidly preprogrammed when neural crest cells form in the neural tube, you certainly wouldn't have the same flexibility. Vertebrate faces might look relatively the same from one species to the next.

When can neural crest cells first be detected during embryonic development?

In the mouse and chick, we've gone all the way back to the induction process, which is the earliest relevant time point [embryonic Day 8 in the mouse, 1.5 in the chick]. If we went back any further, we'd see things that might influence neural crest formation secondarily, such as the development of the neural plate. But they may not be particularly relevant to the actual induction process.

So, in these species, you can track neural crest cells from just about point A to Z of the developmental process?

Yes, exactly. What we can't do is tackle that in humans for technical and ethical reasons. What we do know, if you think about the different phases of neural crest development – whether it be the formation, migration, differentiation phase – if an anomaly arises in any one of those phases, you can end up with a very different craniofacial malformation.

For example?

Well, there a number of recognized neural crest-derived malformation syndromes. I'm talking about potentially devastating conditions such as Treacher-Collins syndrome or DiGeorge syndrome. What's clear is these often-severe syndromes arise within the first eight weeks of pregnancy. At the moment, there is no way that we can detect or visualize them in people during these early, in utero stages based on morphology alone. I think an exceptionally skilled sonographer, even if he or she was specially trained to detect craniofacial anomalies, wouldn't necessarily be able to do it with 100 percent accuracy, even at 22 to 25 weeks of pregnancy.

I should note that even if one could recognize a potential problem, many craniofacial syndromes are quite similar and overlap in their phenotypes, or visible manifestations. They also vary in severity from child to child. So the precise identification of a specific condition still requires genetic confirmation. It can't be done visually.

What needs to change to turn back the detection clock?

Well, it's incredibly complex. I guess one is the development of improved technologies to visualize the very early embryo. But that's only part of it. I think it is very difficult at that early stage to say whether something looked unusual and needed to be monitored and then to try to figure out what it was that was abnormal. It's not always so clear.

How will clarity be reached?

I would say the key is building on the recent progress in biology. If we have good mouse models of individual diseases, we can take all of the genetic network information for neural crest patterning, migration and differentiation and pinpoint the origin of the problem. If it turns out to be something as simple as a wave of cell death – the neural crest cells are being killed off – then the simplistic idea is to find something that will keep them alive, in the same way that some people have found that folic acid somehow confers better viability to neuroepithelial cells and can reduce the incidence of neural tube defects. That's the approach that we're taking. We start with a specific syndrome, try to identify its cause, test whether the mechanism may be common to similar syndromes, and see if we can find a way either to chemically or genetically rescue the problem.

But, as you said, nobody will get anywhere without doing the biology?

That's right. Every day presents a new challenge, but we enjoy our work and its potential to benefit substantial numbers of people worldwide.

Part II: Tooth development

Is it possible to build a tooth? That's a question that many giants of 20th century dental research no doubt considered, and it's a conceptual puzzle that continues to capture the imaginations of the nation's oral health scientists. But there is a key difference between the musings of then and now. Today's scientists possess for the first time the needed laboratory tools to plumb the molecular depths and developmental biology of tooth formation, and some already have begun to do so in earnest.

The research follows two broad but complementary tracks. One seeks to define in sequential detail the genetic programs underlying tooth development and, moving to the next biological level of activity, to map the protein biocircuitry in tooth-forming cells that carry out the genetic program. This research delves into the genetics of the initial tooth placode, a thickened patch of ectoderm near the fetal head that arises as migratory neural crest cells arrive early in development. It then tracks the sequential development of the tooth cap, the tooth bud and ultimately, the maturation of the individual dental tissues therein, from enamel down to the cementum of the tooth root.

The other path aims to take this fundamental information and, like a minimalist artist, deconstruct the complexity of tooth development and define its essential molecular requirements. By stripping away the redundancies and other non-essential molecular chaff of the process, scientists hope to match or possibly improve upon nature's instructions to engineer replacement tissues for damaged teeth.

Offering their perspectives on the road ahead are two NIDCR grantees. They are: Dr. Richard Maas, a scientist at Brigham and Women's Hospital and Harvard Medical School in Boston, and Dr. Malcolm Snead, a scientist at the University of Southern California in Los Angeles.

Richard Maas

Dr. Richard Maas
Brigham and Women's Hospital and
Harvard Medical School
Boston, Massachusetts

As a part of the NIH Roadmap Initiative, you have begun a project that, in part, explores tooth development. Could you tell us about it?

Sure. The project focuses on three structures: the tooth, pancreatic islet cells and the heart valve. The central premise here is we now know enough about organ development to use this knowledge base as a template to assemble a far more comprehensive biological picture of the process.

You want to put more meat on the bone?

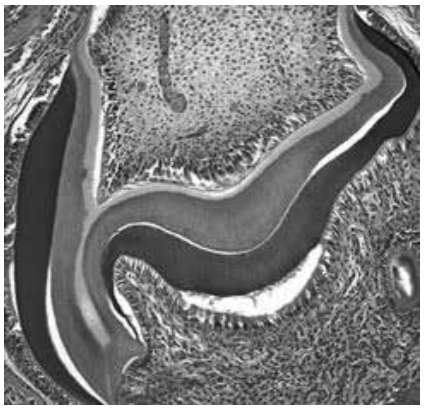
Exactly. The program's acronym is SysCODE, which stands for Systems-Based Consortium for Organ Design and Engineering.

So, it's a systems biology approach, or studying the cell as an integrated system of biological circuits and data processing?

Well, the systems-based aspect arises because we want to integrate the various data sets that the new generation of research tools now can generate. These include comprehensive gene expression profiles; extensive catalogues of protein expression, chip-on-chip analyses to figure out where transcription factors bind in the genome; assembling data on common inherited genetic alterations; and logging the results of RNAi experiments to inhibit an individual gene's expression and thus study its function and thereby dissect relevant signaling pathways within the system.

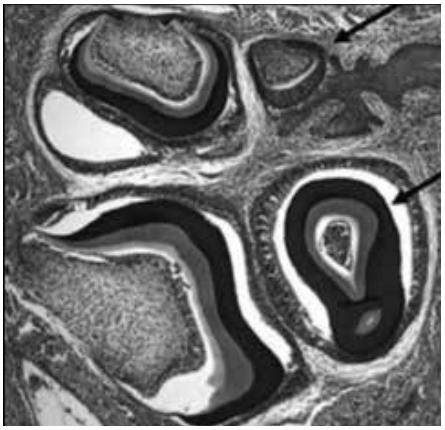
What will this data integration produce?

Our hope is a coherent molecular blueprint to build a tooth. The idea is, once the computer and genome scientists have assembled this large body of information in an intelligent format, it will be amenable for tissue engineers to use.



Extra (supernumerary) teeth can be induced to grow in the jaws of mice by selective inactivation of a single gene, Apc. A normal mouse first molar is shown (above) for comparison.

Below, the two left teeth are normal upper and lower first molars, while the two right teeth (arrows) are supernumeraries.



The magnification in the first image is twice that in the second image. From: Kuaraguchi, Wang et al., PLoS Genetics 2, e146, 2006.

How would they use it?

The set of instructions would be in a user-friendly format for a scientist to say, "Okay, I need to add Factors A, B and C in this particular sequence, at these concentrations and in this particular combination. I need to couple them with a scaffold of extracellular matrix materials X, Y and Z. And because this information is based on how the tooth normally forms, if we reconstruct those types of parameters as best we're able in vitro, we should have a molecular blueprint that will yield a structure that approximates a tooth."

How does a tissue engineer do it today?

Well, the current paradigm for tissue engineering consists of taking Factor X, adding it to some cells and seeing what happens. I'm in no way denigrating that approach. I think it's been incredibly successful. But it is empiric, and a molecular blueprint would be extremely helpful.

In generating the data sets, on one level, wouldn't you want to tease out the evolutionary biology of the tooth? By analogy, a mechanic needs to know the make and model of an automobile before lifting up the hood.

You certainly want to know how a tooth or any part of the body came to be. So, yes, I agree. And that information is being assembled. From DNA sequencing projects to improved mouse models, a major investment has been made over the last decade or so to perform comparative analyses among species and tease out these evolutionary motifs.

But on another level, how detailed will the instruction manual need to be? In other words, do we need to recapitulate all of the moving parts and redundancies that are built into the system? Or can the process be streamlined in the laboratory and remain functional?

The answer is we probably don't need to know all of the moving parts. There are some basic organizing principles at work in the tooth bud that can be mastered and hopefully exploited to form enamel, dentin, cementum and the other constituent parts of the tooth. That means we don't need to account for every last gene in the human genome to build a tooth, and that's why this is a doable task.

How many genes would make the cut?

Well, although there are roughly 23,500 genes in the human genome, probably only one-tenth of those are expressed during tooth development. That is to say, they satisfy the condition of being necessary or sufficient.

And of those genes, some likely will be more critical than others?

That's right. Not all genes and proteins that plug into a developmental pathway are of equal importance. This relates to the structure of networks.

How so?

There are two general types of biological networks hard-wired into our cells. One is called a universal random network, where every component, or node, is equal in importance and weight. The second and more prevalent form is called a scale-free network. In scale-free networks, not all nodes are created the same. There are very important centralized nodes, or hubs, that act as convergence points and processing centers for incoming biological information. Think of the spoke-and-hub system in aviation. If you identify the hubs – the Chicago O'Hares – it's possible to predict the behavior of the system to a large extent and without needing to piece together every single element of the network.

What might these predictions reveal?

Let me give you an example. We study a gene that, if inactivated, results in the formation of supernumary, or extra, teeth in the mouse. That suggests that this gene and its protein product are very high up in the regulatory cascade that controls tooth development. By manipulating that protein, you wouldn't necessarily have to control all of the others that are activated subsequently, or downstream, of it. Because that master gene would take care of them for you. You see? So, this shows the great simplification that is possible as some genes turn on entire programs of downstream events.

Are there other basic organizing principles?

There's a corollary principle. I would call it the principle of autonomy. By that I mean, if an early tooth germ reaches a certain developmental stage, it will continue to develop all the way to the latter stages of mineralization. There's actually a precedent for that. Dr. Paul Sharp and colleagues at Guy's Hospital in the UK showed some time ago that if they took what's called a cap-stage tooth germ and grafted it into the jaw of an adult mouse, it will in fact develop much, much further.

That would mean we don't necessarily have to worry about mastering all stages of tooth development. Once a developmental tipping point is reached, biology could take care of the rest?

That's right.

What impact might this and related work have on practitioners in the coming years?

Well, let me just say that current prosthetics work relatively well. But millions of Americans still lose a significant number of teeth during their lives, and dental disease remains a significant health problem. So, if we could generate a biomimetic substitute, it would be welcomed. To do that at a reasonable cost and with good efficiency, I think you're looking at least a decade into the future. On the other hand, if you could generate enamel matrix in a test tube from cells that you've programmed, that would be very exciting. How that would figure into clinical practice, dentists no doubt would decide. But, clearly it would be a wonderful natural product.

Malcolm Snead

Dr. Malcolm Snead
University of Southern California
Los Angeles, California

The term "building a tooth" suggests creating a bicuspid or incisor from scratch. But that's not the focus in your laboratory?

Most of my interest and expertise developmentally lie downstream of those initiating events. In other words, I'm not interested in day six or seven of gestation, although I think early development is very interesting. My research focus is on the problems of tissue specification during late gestation and early postnatal development.

How do these problems flow into building a tooth?

In our case, the focus is on learning to engineer new tissue to replace damaged or diseased tooth structures.

Why engineer?

Let me back up a bit. Biology is now in a golden age of discovery. We can knock out a specific gene, modify another gene and ask a variety of profound questions about the circuitry of the cell that just weren't on the table a decade or so ago.

For example?

Well, you could ask what happens systemically if you remove 100 percent of transforming growth factor X? Does the cell – the biological system – have the ability to compensate for the loss via a redundant signal? If there's no compensation and thus the effect is uniform within the system, what then happens downstream when the circuit is shut down? It's kind of like a caveman holding a pocket watch. You smash the pocket watch and say, "Great, look at all of these parts in it." But can you reassemble the watch? The next step is to go back and say, "Okay, I know that I need this piece, but how far can I turn it down," so that it functions at 10 percent of its normal level and still get an outcome?

Addition by subtraction?

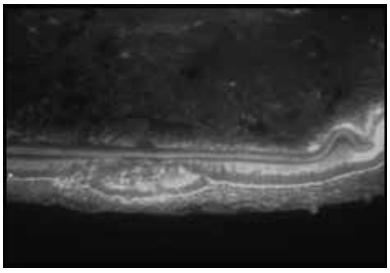
Right. If you've identified all of the pieces, can you also define the ones that you don't need? It's a matter of relevance, and that is now framed within the context of our expanded scale of discovery. What I mean is we used to try to understand how the cell worked at some level when we could perform a Western blot assay and detect a protein. We advanced to a Northern blot assay to process RNA, and that gave us greater sensitivity to quantify gene expression. In the 1990s, we entered a PCR state of affairs that enabled us to look at five or six molecules in a cell. Now you must start pulling out the noise, the chaff in the system. What is relevant? And what is spurious noise? It's oftentimes a matter of understanding what you know relative to how you think you need to know it.

What about something as complex as a tooth root?

It's a four-in-one proposition with dentin, pulp, cementum and the periodontal ligament. It represents a challenge, but certainly a worthy one. If you can understand root formation, you have a much better handle on regenerating a major cause of tooth loss in adults, which is the loss of supporting bone and ligament. But what's important here is this is a challenge that we now can productively wrap our minds around.

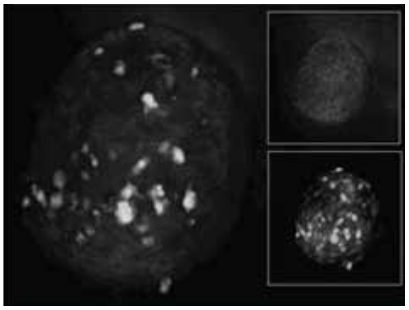
What about regenerating enamel, one of your major research interests?

Enamel is a tissue in which the whole is greater structurally than its individual parts, in this case, elongated hydroxyapatite crystals. Enamel is a fascinating tissue. I'm actually sitting here holding a chunk of hydroxyapatite in my hand. If you made a tooth out of what's in my hand, it would fracture and fall apart in a matter of days. Its toughness, hardness and elasticity really are quite different than the hydroxyapatite in the enamel of my teeth. Some of that has to do with the nanoscale that nature works to weave hydroxyapatite crystals into the patterned structure that we know as tooth enamel. Another small part of that is some residual amount of protein. It's maybe 5/10 of a percent of protein dry weight. It's likely retained for a very specific function.

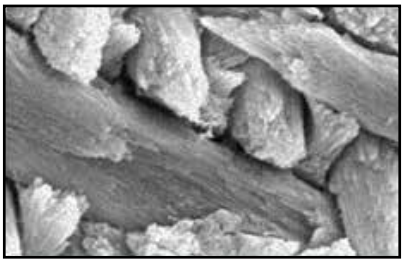


After injecting artificial bioactive nanostructures, the mouse incisor expresses the protein integrin alpha-6.

The protein helps to form the enamel matrix.



Green fluorescent proteins illuminate cells in a three-dimensional matrix of artificial bioactive nanostructures.



A close-up of dental enamel.

And it's retained as a remnant of the original protein matrix?

Right. Even though the tooth erupts into the oral cavity as a white, mineralized fossil, it was not a fossil during its formation. Before the hydroxyapatite crystals elongated, properly oriented themselves and formed mature enamel, they were seeded in an extensive protein matrix that served as a developmental lattice. All of the rules that apply to changes in gene expression, control of protein expression, response of different signaling molecules through membrane-mediated receptor events and secondary signals. All of those are happening. Enamel is very, very much alive as it's being made.

But can you go all the way back and track the assembly of, say, the amelogenins in forming the protein matrix?

Absolutely. In fact, there are 16 different isoforms, or types, of amelogenin. We work on them in in vitro analyses, and that tells you certain things. But then you must go back to the organism and say, "In the context of the organism, does it work this way?" That's where the approach to genetic engineering – the simplicity of a biological outcome as your measure – is very appealing. If you can achieve the same outcome with a lot less moving parts, it suggests that you understand how the system works and know its critical parts.

How's the engineering going?

Quite well. Right now, I'm working on a manuscript that produces almost a two-order reduction in simplicity in proteins of the amelogenin class. We used a genetic knock-in strategy that ends up producing an enamel that has essentially characteristics that are within 20 percent of the natural enamel. So I've made this enamel with 16 times less alternative proteins participating. And yet, the system seems to work adequately. Or within 95 percent of the expected values. So this question of how far can we go is one that I share with the rest of the team here at University Southern California and numerous labs around the world.

And across scientific disciplines.

Yeah, I think a lot of people in materials sciences, engineering and nanotechnology get quite excited about it. Making a mineral that behaves in the way that enamel does, or the way that the enamel actually bonds to the underlying dentin and dentinoenamel junction, or DEJ, is a phenomenal piece of engineering. When I speak to my engineering colleagues, they would like to know about the DEJ. How does the enamel stay on the dentin?

That's of real significance to dentists.

Right, if you could replicate a dentino enamel junction (DEJ), it would be a first step toward ensuring that fillings lasted longer. Restorations usually fail at the interface. It's not the filling itself. It's the bond of the materials… If a glass-like ceramic material is more compatible with the dentin, you can increase the longevity of the restoration.

And the data are pouring in.

Yes, that's right. It's pouring in all areas of biology. I was at a nanotechnology meeting recently where people were talking about monitoring 40 channels of data coming out of the cell simultaneously, from their cell receptors to their oxidative state. You could see lots of different parameters of cell biology being reported in a single cell instead of looking at a thousand cells and averaging their behavior for one parameter. You can just imagine what that means. Suddenly you must ask, "Have we been measuring the tops of mountains? There's a much wider range to how cells respond. We have to see a 20-fold change in its activity before we even start to say we can measure and work with it. When, in fact, the system is much more sensitive, it may be changes of 20 percent that make differences. But we ignore them because we don't see them.

Part III: Periodontal disease: Engineering the future of care

In the 1950s, soon after NIDCR's founding, millions of Americans often flipped on their black-and-white tube televisions and watched commercials that warned of a tongue-twisting condition called gingivitis. As the ads warned, gingivitis was step one on the road to chronic gum, or periodontal, disease and tooth loss.

Today, researchers now know that gingivitis does not necessarily lead to advanced periodontal disease. However, a confluence of factors can induce chronic inflammation of the gingiva in some people. These factors include lifestyle choices, such as an addiction to tobacco; diabetes or another underlying health condition that compromises one's ability to heal; a susceptibility to gingival infections; and an over-reactive immune system. If left unchecked, periodontal disease gradually will degrade the four tooth-supporting tissues of the periodontium – gingiva, periodontal ligament, cementum and bone. As the television ads from the 1950s correctly concluded, advanced periodontal disease will lead to tooth loss.

Today, a trip to the periodontist typically entails anti-infective therapy coupled with scaling and planing of the inflamed tissue. Periodontists also have been eager to employ the latest biological discoveries to regenerate damaged gingiva, ligament and bone. Although today's tissue regeneration techniques remain works in progress, research on this front continues to progress nicely. Most scientists are optimistic that tissue engineering, with its sophisticated mix of biology and chemistry, will be more predictable in the years ahead. To present the research themes are two scientists with long and productive records of accomplishment in this area: Dr. William Giannobile, a researcher at the University of Michigan in Ann Arbor; and Dr. Pamela Robey, an NIDCR scientist in Bethesda, Maryland.

William Giannobile

Dr. William Giannobile
University of Michigan
Ann Arbor, Michigan

You oversee your own laboratory and head a clinical research center at the University of Michigan. You also find the time to see patients in private practice?

I'm also a practicing periodontist, and I continue to treat patients in a private practice here in Ann Arbor.

So you've both seeded and directly benefited from the research?

I think so. As a practitioner, I've benefited from the advancements in biomaterials research, various bone grafting techniques, guiding tissue membranes that separate the bone from the connective tissues, and anti-infective therapies to arrest or slow the progression of periodontal disease.

But there's still a ways to go?

That's right. Periodontal treatment today remains fairly unpredictable in terms of getting stable, long-term results. That's especially true for regenerating the various parts of the periodontium – bone, ligament, cementum and mucosa. But there have been some nice advancements, and I just mentioned a few of them. I think the combination of research progress and the continued unpredictability of treatment has to some extent shifted the periodontist's role.

How so?

About 20 years ago, most restorative dentists would contact periodontists with referrals and say, "Do your best. See how many more years you can help this patient eke out of these teeth." Now, the mindset is much different. Because of the unpredictability of regeneration and the challenge of controlling a chronic immune response, many practitioners don't want to run the risk of trying to save the teeth. Many advise their patient to have the questionable teeth removed and replaced with dental implants. But as a researcher and periodontist, I continue to support the profession's founding principles to save teeth whenever possible. For that reason, I see a lot of areas that we need to explore to develop more predictable therapies, especially when regenerating the damaged tissues of the periodontium.

And the biology is there for the taking?

I think so. You can see it in the scientific literature each and every month. You also can see it in the ideas that have entered the clinical research pipeline.

For example?

Growth factors. Platelet-derived growth factor and the bone morphogenetic proteins are both now FDA approved and have entered into the clinical arena over the last two years. Although these growth factors have sometimes incorrectly been held up as a panacea for tissue regeneration, they are indeed very important advancements. Keep in mind, though, periodontal disease is an extremely complex condition. Tissue regeneration alone is not the answer. We need to push the anti-infective/host modulation side, too, and ensure that the patient's immune response doesn't turn chronic and self-destructive.

And that self destruction is of an inherently complex environment?

Absolutely. We essentially have a tooth – an avascular mineralized tissue – that protrudes through soft tissue, where it is under constant microbial attack. The tooth is rooted in bone, anchored by ligament and dependent on supportive tissues in dentin and cementum. These tissues have specific geographic junctions, abilities to bond and load-bearing qualities. What's more, all of this complexity is rolled into a tight biologic space. We often talk about millimeters of eroded bone. Millimeters are very significant in the support of teeth. There's just nothing like the periodontium in the rest of the body.

So how do you make tissue regeneration more predictable?

I think interdisciplinary collaborations hold the key. We need scientists with training in infectious disease, biomedical engineering, molecular and cell biology, genetics, and clinical research.

What about technology development?

Discovery and technology development typically go hand in hand. A good example is the progress in high-resolution imaging techniques. They produce images of bone damage that are so accurate, we literally can construct in the laboratory three dimensional scaffolds that fit perfectly into the periodontal pockets and deliver regenerative factors. This hasn't yet reached the clinic, but it suggests the following scenario: The surgeon images the tooth/bone lesion and says to the patient, "Okay, I can quickly make this scaffold, and it will be loaded with growth factors or stem cells. We'll just drop it into the defect." Right now, the surgeons carve out the decalcified bone as best they can. It's just not efficient.

We talked a moment ago about the biology being there for the taking. If the scaffolds and their growth-promoting cargo now can be more precisely delivered, that raises the issue of timing. You need to drop in the growth factors at the right time and in the right sequence.

That's an important point. Researchers have begun to look at the process in a more systematic way. But defining the biology is even more important from a diagnostic standpoint. Patients respond differently to treatment.

Dr. Pamela Robey
National Institutes of Health
Bethesda, Maryland

It's kind of like reading the Old Testament Book of Numbers. The patriarchal stem cell begets sublineages of cells.

Right, and this theme is played out throughout the body. We see it in bone and cartilage, and we see it in dentin.

What's the second defining feature?

Self renewal. A stem cell must have the ability to produce a new generation of stem cells. So, as they churn out more mature daughter cells, they also must produce daughter cells that remain stem cells. Without self-renewal, the stem cells that populate a given tissue would die their natural deaths and take with them the ability to regenerate and maintain a tissue structure.

Why has the definition of stem cell gone off in all directions?

It's the vagaries of cell culture. Because scientists typically study stem cells in a culture dish, they describe certain characteristics that may or may not relate back to what they do in the body.

For example?

Well, two things come to mind. A lot of people say that a stem cell is undifferentiated. But that may be a misnomer. It may be that a fully differentiated cell, under the proper conditions, can revert back and become a precursor cell or maybe even a more primitive cell like a stem cell. As long as a cell has an intact nucleus, nothing is really impossible.

Secondly, some say a stem cell has the ability to replicate almost endlessly. But that isn't necessarily the case either. Even the best characterized stem cell – the hematopoietic stem cell – is not immortal. Each hematopoietic stem cell can only repopulate blood cells a certain number of times before it becomes exhausted.

How does this "stemness" translate to the periodontium?

Let's start with the periodontal ligament. We know it's possible to take a periodontal ligament and treat it with enzymes to release single cells. Within that mixed population of cells, there are some that have the ability to recreate fibers that look a lot like those in a periodontal ligament. Whether the fibers can function as a periodontal ligament I think is still on the table for discussion. These cells were discovered here at NIDCR about four years ago, and this lead is actively being pursued. It's also not clear where the cell comes from and whether it's truly a stem cell or a useful progenitor cell. I think that is something that we need to take into consideration.

What do you mean?

Having a stem cell is a wonderful thing. Knowing about it is a wonderful thing. But the slightly more committed progenitors also can do wonderful things. We shouldn't dismiss them just because they don't self-renew or produce more than one cell type. If they're useful in a regenerative way, that's fine. They don't have to be a stem cell to be useful.

What about alveolar bone of the tooth socket?

Stem cells that make bone have been under study since the mid-1990s. Obviously, they have great potential in regenerating the periodontium. If you don't have bone to anchor the tooth, you have no foundation. But we really need to know how this type of bone is different from the axial and appendicular bone found elsewhere in the body. They have a different embryonic origin, and their properties appear to be slightly different. We don't know how that is. Nor do we know the impact of these differences. For example, can we take bone marrow from the iliac crest of the pelvis and use it to regenerate craniofacial bone, including alveolar bone? That's a huge question, and we really don't know much about it.

What about cementum?

Well, I just mentioned the periodontal ligament cells. They also make something that appears to be cementum. In addition, a number of years ago, we took shavings of cementum and could get cells to grow out of those shavings in a clonal fashion. When we took the cells, put

them in a scaffold and transplanted them into our animal model, they formed tissue that resembled cementum. The problem is we don't really know whether cementum is different from bone. We don't have any cementum specific markers to answer that question. It may just be that osteogenic cells form a cementum-like structure when they are in close proximity to dentin-forming cells. So, the boundaries between cementum and bone are very subtle. It's difficult to say that they are truly two distinct entities. Maybe under the right conditions, cells that normally would be osteogenic could be made to be cementogenic.

Are you confident that the regeneration of the periodontium will be doable down the road?

I am confident. We have cells that we can begin to use to try and create a viable tooth. But there is a major hurdle in trying to construct a root that will provide a solid anchor within the jawbone. And we need to bring a broader array of expertise to the table. We need more people with expertise in bioinformatics, biomaterials, biochemistry and clinicians to help us design appropriate animal models that mimic a given human disease. There can be a perception that it's only a tooth. It's not going to kill you if you don't have one. But try to tell that to somebody with a bad toothache. Dental problems cut across all aspects of society, and their solutions potentially benefit everyone. Secondly, the mouth is readily accessible, unlike the body's internal organs such as the pancreas or liver. The lessons learned in the oral cavity might not be a perfect fit in learning to better treat osteoporosis or kidney cancer, but they will have applicability.

Conclusion

There are many challenges in the study of the human molecular genetics, and researchers are involved from many different areas and in many different ways. We have learned from the interviews above that researchers are confident that eventually they will be able to engineer or build a tooth, and will see tissue regeneration or self renewal from stem cells that have the ability to produce a new generation of stem cells. Additional scientific progress in the neurosciences will have broad implications for the diagnosis and treatment of diseases and disorders of the craniofacial system. The approach to design and fabricate bioceramics to be used in the replacement of human enamel or dentin of the surfaces of teeth is another scientific breakthrough. The continued study of the fabrication of biomaterials to be used in the advances toward repair and regeneration of cartilage, bone, muscle, teeth, (cementum, dentin, enamel and periodontal ligament) offers another focus. Dental professionals are indeed ready to face the future.

Reference

- www..nidcr.nih.gov/facingthe future.htm National Institute of Dental and Craniofacial Research

FACING THE FUTURE: NIDCR RESEARCHERS OFFER THEIR VISION OF THE FUTURE

Final Examination Questions

Select the best answer questions 1 through 5 and mark your answers on the Final Examination Answer Sheet found on page 148, or for faster service complete your test online at **Dental.EliteCME.com.**

1. The interaction between ectoderm and mesoderm is a classic mode of embryonic tissue formation.
   - ○ True
   - ○ False
2. Neural crest and the surrounding ectodermal cells generate the development programs that produce vertebrate structures as distinct as the beak of a toucan, the tusk of a boar or the venom-producing salivary gland of a rattlesnake.
   - ○ True
   - ○ False
3. Neural crest cells are endowed with an innate plasticity, but cannot make developmental modifications.
   - ○ True
   - ○ False
4. Dr. Paul Sharp and colleagues at Guy's Hospital in the UK showed some time ago that if they took what's called a cap-stage tooth germ and grafted it into the jaw of an adult mouse, it will in fact develop much, much further.
   - ○ True
   - ○ False
5. Tooth enamel is an organism that is already dead when it is being made.
   - ○ True
   - ○ False

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Chapter 2: Guidelines for Hand Hygiene in Health Care Settings

2 CE Hours

By: Elite Staff

Learning objectives

- Explain the normal bacterial skin flora.
- Describe the relation of hand hygiene and acquisition of health care-associated pathogens.
- List methods used to evaluate hand hygiene products.
- Review trial data for soaps and alcohol-based products.
- Discuss irritant contact dermatitis resulting from hand-hygiene measures.

Introduction

This course provides health-care personnel (HCPs) with a review of data regarding the relation of hand hygiene and acquisition of health care-associated pathogens and the methods used to evaluate the efficacy of hand-hygiene products. In addition, it provides specific data and study information regarding the review of preparation used for hand hygiene plain nonantimicrobial soap and alcohol-based antiseptics. This course provides recommendations to promote improved hand-hygiene practices and reduce transmission of pathogenic microorganisms to patients and personnel in health-care settings. It provides information regarding the contact dermatitis resulting from hand-hygiene measures and factors to consider when selecting hand hygiene products.

Historical perspective

For generations, handwashing with soap and water has been considered a measure of personal hygiene. The concept of cleansing hands with an antiseptic agent probably emerged in the early 19th century. As early as 1822, a French pharmacist demonstrated that solutions containing chlorides of lime or soda could eradicate the foul odors associated with human corpses and that such solutions could be used as disinfectants and antiseptics.

In 1961, the U. S. Public Health Service produced a training film that demonstrated handwashing techniques recommended for use by health-care personel. At the time, recommendations directed that personnel wash their hands with soap and water for 1–2 minutes before and after patient contact. Rinsing hands with an antiseptic agent was believed to be less effective than handwashing and was recommended only in emergencies or in areas where sinks were unavailable.

In 1975 and 1985, formal written guidelines on handwashing practices in hospitals were published by CDC. These guidelines recommended handwashing with nonantimicrobial soap between the majority of patient contacts and washing with antimicrobial soap before and after performing invasive procedures or caring for patients at high risk. Use of waterless antiseptic agents (e.g., alcohol-based solutions) was recommended only in situations where sinks were not available.

In 1988 and 1995, guidelines for handwashing and hand antisepsis were published by the Association for Professionals in Infection Control (APIC). Recommended indications for handwashing were similar to those listed in the CDC guidelines. The 1995 APIC guideline included more detailed discussion of alcohol-based hand rubs and supported their use in more clinical settings than had been recommended in earlier guidelines. In 1995 and 1996, the Health Care Infection Control Practices Advisory Committee (HICPAC) recommended that either antimicrobial soap or a waterless antiseptic agent be used for cleaning hands upon leaving the rooms of patients with multidrug-resistant pathogens (e.g., vancomycin-resistant enterococci [VRE] and methicillin-resistant staphylococcus aureus [MRSA]). These guidelines also provided recommendations for handwashing and hand antisepsis in other clinical settings, including routine patient care. Although the APIC and HICPAC guidelines have been adopted by the majority of hospitals, adherence of health-care personnel to recommended handwashing practices has remained low.

Recent developments in the field have stimulated a review of the scientific data regarding hand hygiene and the development of new guidelines designed to improve hand-hygiene practices in health care facilities. This literature review and accompanying recommendations have been prepared by a Hand Hygiene Task Force, composed of representatives from HICPAC, the Society for Healthcare Epidemiology of America (SHEA), APIC, and the Infectious Diseases Society of America (IDSA).

Normal bacterial skin flora

To understand the objectives of different approaches to hand cleansing, a knowledge of normal bacterial skin flora is essential. Normal human skin is colonized with bacteria; different areas of the body have varied total aerobic bacterial counts (e.g., 1 x 106 colony forming units (CFUs)/cm2 on the scalp, 5 x 105 CFUs/cm2 in the axilla, 4 x 104 CFUs/cm2 on the abdomen, and 1 x 104 CFUs/cm2 on the forearm). Total bacterial counts on the hands of medical personnel have ranged from 3.9 x 104 to 4.6 x 106. In 1938, bacteria recovered from the hands were divided into two categories: transient and resident. Transient flora, which colonize the superficial layers of the skin, are more amenable to removal by routine handwashing. They are often acquired by health-care personnel during direct contact with patients or contact with contaminated environmental surfaces within close proximity of the patient. Transient flora are the organisms most frequently associated with health care-associated infections. Resident flora, which are attached to deeper layers of the skin, are more resistant to removal. In addition, resident flora (e.g., coagulase-negative staphylococci and diphtheroids) are less likely to be associated with

such infections. The hands of health-care personnel may become persistently colonized with pathogenic flora (e.g., S. aureus), gram-negative bacilli, or yeast. Investigators have documented that, although the number of transient and resident flora varies considerably from person to person, it is often relatively constant for any specific person.

Relation of hand hygiene and acquisition of health care-associated pathogens

Trials have studied the effects of handwashing with plain soap and water versus some form of hand antisepsis on health care-associated infection rates. Health care-associated infection rates were lower when antiseptic handwashing was performed by personnel. In another study, antiseptic handwashing was associated with lower health care-associated infection rates in certain intensive-care units, but not in others.

Health care associated infection rates were lower after antiseptic handwashing using a chlorhexidine-containing detergent compared with handwashing with plain soap or use of an alcohol-based hand rinse. However, because only a minimal amount of the alcohol rinse was used during periods when the combination regimen also was in use and because adherence to policies was higher when chlorhexidine was available, determining which factor (i.e., the hand-hygiene regimen or differences in adherence) accounted for the lower infection rates was difficult. Investigators have determined also that health care-associated acquisition of MRSA was reduced when the antimicrobial soap used for hygienic handwashing was changed.

Increased handwashing frequency among hospital staff has been associated with decreased transmission of Klebsiella spp. among patients; these studies, however, did not quantitate the level of handwashing among personnel. In a recent study, the acquisition of various health care-associated pathogens was reduced when hand antisepsis was performed more frequently by hospital personnel; both this study and another documented that the prevalence of health care-associated infections decreased as adherence to recommended hand-hygiene measures improved.

Outbreak investigations have indicated an association between infections and understaffing or overcrowding; the association was consistently linked with poor adherence to hand hygiene. During an outbreak investigation of risk factors for central venous catheter-associated bloodstream infections, after adjustment for confounding factors, the patient-to-nurse ratio remained an independent risk factor for bloodstream infection, indicating that nursing staff reduction below a critical threshold may have contributed to this outbreak by jeopardizing adequate catheter care. The understaffing of health-care personnel can facilitate the spread of MRSA in health-care settings through relaxed attention to basic control measures (e.g., hand hygiene). In an outbreak of Enterobacter cloacae in a neonatal intensive-care unit, the daily number of hospitalized children was above the maximum capacity of the unit, resulting in an available space per child below current recommendations. In parallel, the number of staff members on duty was substantially less than the number necessitated by the workload, which also resulted in relaxed attention to basic infection-control measures. Adherence to hand-hygiene practices before device contact was only 25 percent during the workload peak, but increased to 70 percent after the end of the understaffing and overcrowding period. Surveillance documented that being hospitalized during this period was associated with a fourfold increased risk of acquiring a health care-associated infection. This study not only demonstrates the association between workload and infections, but it also highlights the intermediate cause of antimicrobial spread: poor adherence to hand-hygiene policies.

METHODS USED TO EVALUATE THE EFFICACY OF HAND-HYGIENE PRODUCTS

Current methods

Investigators use different methods to study the in vivo efficacy of handwashing, antiseptic handwash, and surgical hand antisepsis protocols. Differences among the various studies include 1) whether hands are purposely contaminated with bacteria before use of test agents, 2) the method used to contaminate fingers or hands, 3) the volume of hand-hygiene product applied to the hands, 4) the time the product is in contact with the skin, 5) the method used to recover bacteria from the skin after the test solution has been used, and 6) the method of expressing the efficacy of the product (i.e., either percent reduction in bacteria recovered from the skin or log reduction of bacteria released from the skin). Despite these differences, the majority of studies can be placed into one of two major categories: studies focusing on products to remove transient flora and studies involving products that are used to remove resident flora from the hands. The majority of studies of products for removing transient flora from the hands of health-care personnel involve artificial contamination of the volunteer's skin with a defined inoculum of a test organism before the volunteer uses a plain soap, an antimicrobial soap, or a waterless antiseptic agent. In contrast, products tested for the preoperative cleansing of surgeons' hands (which must comply with surgical hand antisepsis protocols) are tested for their ability to remove resident flora without artificially contaminating the volunteers' hands.

In the United States, antiseptic handwash products intended for use by health-care personnel are regulated by FDA's Division of Over-the-Counter Drug Products (OTC). Requirements for in vitro and in vivo testing of health-care handwash products and surgical hand scrubs are outlined in the FDA Tentative Final Monograph for Healthcare Antiseptic Drug Products (TFM). Products intended for use as health-care personnel handwashes are evaluated by using a standardized method. Tests are performed in accordance with use directions for the test material. Before baseline bacterial sampling and before each wash with the test material, 5 ml of a standardized suspension of Serratia marcescens are applied to the hands and then rubbed over the surfaces of the hands. A specified volume of the test material is dispensed into the hands and is spread over the hands and lower one third of the forearms. A small amount of tap water is added to the hands, and hands are completely lathered for a specified time, covering all surfaces of the hands and the lower third of the forearms. Volunteers then rinse hands and forearms under 40 degrees C tap water for 30 seconds. Ten washes with the test formulation are required. After the first, third, seventh, and tenth washes, rubber gloves or polyethylene bags used for sampling are placed on the right and left hands, and 75 ml of sampling solution is added to each glove; gloves are secured above the wrist. All surfaces of the hand are massaged for 1 minute, and samples are obtained aseptically for quantitative culture. No neutralizer of the antimicrobial is routinely added to the sampling solution, but if dilution of the antimicrobial in the sampling fluid does not result in demonstrable neutralization, a neutralizer specific for the test formulation is added to the sampling solution. For waterless formulations, a similar procedure is used. TFM criteria for efficacy are as follows: a 2-log-10 reduction of the indicator organism on each hand within 5 minutes after the first use, and a 3-log-10 reduction of the indicator organism on each hand within 5 minutes after the tenth use.

Products intended for use as surgical hand scrubs have been evaluated also by using a standardized method. Volunteers clean under

fingernails with a nail stick and clip their fingernails. All jewelry is removed from hands and arms. Hands and two thirds of forearms are rinsed with tap water (38 degrees C to 42 degrees C) for 30 seconds, and then they are washed with a non-antimicrobial soap for 30 seconds and are rinsed for 30 seconds under tap water. Baseline microbial hand counts can then be determined. Next, a surgical scrub is performed with the test formulation using directions provided by the manufacturer. If no instructions are provided with the formulation, two 5-minute scrubs of hands and forearms followed by rinsing are performed. Reduction from baseline microbial hand counts is determined in a series of 11 scrubs conducted during 5 days. Hands are sampled at 1 minute, 3 hours, and 6 hours after the first scrubs on day 1, day 2, and day 5. After washing, volunteers wear rubber gloves; 75 ml of sampling solution are then added to one glove, and all surfaces of the hands are massaged for 1 minute. Samples are then taken aseptically and cultured quantitatively. The other glove remains on the other hand for 6 hours and is sampled in the same manner. TFM requires that formulations reduce the number of bacteria 1-log-10 on each hand within 1 minute of product application and that the bacterial cell count on each hand does not subsequently exceed baseline within 6 hours on day 1; the formulation must produce a 2-log-10 reduction in microbial flora on each hand within 1 minute of product application by the end of the second day of enumeration and a 3-log-10 reduction of microbial flora on each hand within 1 minute of product use by the end of the fifth day when compared with the established baseline.

Because of different standards for efficacy, criteria cited in FDA TFM and the European EN 1500 document for establishing alcohol-based hand rubs vary. Alcohol-based hand rubs that meet TFM criteria for efficacy may not necessarily meet the EN 1500 criteria for efficacy. In addition, scientific studies have not established the extent to which counts of bacteria or other microorganisms on the hands need to be reduced to minimize transmission of pathogens in health care facilities; whether bacterial counts on the hands must be reduced by 1 log-10 (90 percent reduction), 2 log-10 (99 percent), 3 log-10 (99.9 percent), or 4 log-10 (99.99 percent) is unknown. Several other methods also have been used to measure the efficacy of antiseptic agents against various viral pathogens.

Shortcomings of traditional methodologies

Accepted methods of evaluating hand-hygiene products intended for use by health-care personnel require that test volunteers wash their hands with a plain or antimicrobial soap for 30 seconds or 1 minute, despite the observation in the majority of studies that the average duration of handwashing by hospital personnel is less than 15 seconds. A limited number of investigators have used 15-second handwashing or hygienic hand-wash protocols. Therefore, almost no data exist regarding the efficacy of plain or antimicrobial soaps under conditions in which they are actually used by health-care personnel. Similarly, certain accepted methods for evaluating waterless antiseptic agents for use as antiseptic hand rubs require that 3 ml of alcohol be rubbed into the hands for 30 seconds, followed by a repeat application for the same duration. This type of protocol also does not reflect actual usage patterns among health-care personnel. Furthermore, volunteers used in evaluations of products are usually surrogates for health-care personnel, and their hand flora may not reflect flora found on the hands of personnel working in health-care settings. Further studies should be conducted among practicing health-care personnel using standardized protocols to obtain more realistic views of microbial colonization and risk of bacterial transfer and cross-transmission.

REVIEW OF PREPARATIONS USED FOR HAND HYGIENE

Plain (non-anti-microbial) soap

Soaps are detergent-based products that contain esterified fatty acids and sodium or potassium hydroxide. They are available in various forms including bar soap, tissue, leaflet, and liquid preparations. Their cleaning activity can be attributed to their detergent properties, which result in removal of dirt, soil, and various organic substances from the hands. Plain soaps have minimal, if any, antimicrobial activity. However, handwashing with plain soap can remove loosely adherent transient flora. For example, handwashing with plain soap and water for 15 seconds reduces bacterial counts on the skin by 0.6–1.1-log-10, whereas washing for 30 seconds reduces counts by 1.8–2.8-log-10. However, in several studies, handwashing with plain soap failed to remove pathogens from the hands of hospital personnel. Handwashing with plain soap can result in paradoxical increases in bacterial counts on the skin. Nonantimicrobial soaps may be associated with considerable skin irritation and dryness, although adding emollients to soap preparations may reduce their propensity to cause irritation. Occasionally, plain soaps have become contaminated, which may lead to colonization of hands of personnel with gram-negative bacilli.

Alcohols

The majority of alcohol-based hand antiseptics contain either isopropanol, ethanol, n-propanol, or a combination of two of these products. The majority of studies of alcohols have evaluated individual alcohols in varying concentrations. Other studies have focused on combinations of two alcohols or alcohol solutions containing limited amounts of hexachlorophene, quaternary ammonium compounds, povidone-iodine, triclosan, or chlorhexidine gluconate.

The anti-microbial activity of alcohols can be attributed to their ability to denature proteins. Alcohol solutions containing 60 percent–95 percent alcohol are most effective, and higher concentrations are less potent because proteins are not denatured easily in the absence of water. The alcohol content of solutions may be expressed as percent by weight (w/w), which is not affected by temperature or other variables, or as percent by volume (vol/vol), which can be affected by temperature, specific gravity, and reaction concentration (123). For example, 70 percent alcohol by weight is equivalent to 76.8 percent by volume if prepared at 15 degrees C, or 80.5 percent if prepared at 25 degrees C. Alcohol concentrations in antiseptic hand rubs are often expressed as percent by volume. Alcohols have excellent in vitro germicidal activity against gram-positive and gram-negative vegetative bacteria, including multidrug-resistant pathogens (e.g., MRSA and VRE), Mycobacterium tuberculosis, and various fungi. Certain enveloped (lipophilic) viruses (e.g., herpes simplex virus, human immunodeficiency virus [HIV], influenza virus, respiratory syncytial virus, and vaccinia virus) are susceptible to alcohols when tested in vitro. Hepatitis B virus is an enveloped virus that is somewhat less susceptible but is killed by 60 percent–70 percent alcohol; hepatitis C virus also is likely killed by this percentage of alcohol. In a porcine tissue carrier model used to study antiseptic activity, 70 percent ethanol and 70 percent isopropanol were found to reduce titers of an enveloped bacteriophage more effectively than an anti-microbial soap containing 4 percent chlorhexidine gluconate. Despite its effectiveness against these organisms, alcohols have very poor activity against bacterial spores, protozoan oocysts, and certain nonenveloped (nonlipophilic) viruses.

Numerous studies have documented the in vivo anti-microbial activity of alcohols. Alcohols effectively reduce bacterial counts on the hands. Typically, log reductions of the release of test bacteria from artificially

contaminated hands average 3.5-log-10 after a 30-second application and 4.0–5.0-log-10 after a 1-minute application. In 1994, the FDA TFM classified ethanol 60 percent–95 percent as a Category I agent (i.e., generally safe and effective for use in antiseptic handwash or health care hand-wash products) (19). Although TFM placed isopropanol 70 percent–91.3 percent in category IIIE (i.e., insufficient data to classify as effective), 60 percent isopropanol has subsequently been adopted in Europe as the reference standard against which alcohol-based hand-rub products are compared. Alcohols are rapidly germicidal when applied to the skin, but they have no appreciable persistent (i.e., residual) activity.

However, regrowth of bacteria on the skin occurs slowly after use of alcohol-based hand antiseptics, presumably because of the sublethal effect alcohols have on some of the skin bacteria. Addition of chlorhexidine, quaternary ammonium compounds, octenidine, or triclosan to alcohol-based solutions can result in persistent activity. Alcohols, when used in concentrations present in alcohol-based hand rubs, also have in vivo activity against several nonenveloped viruses. For example, 70 percent isopropanol and 70 percent ethanol are more effective than medicated soap or nonmedicated soap in reducing rotavirus titers on fingerpads. A more recent study using the same test methods evaluated a commercially available product containing 60 percent ethanol and found that the product reduced the infectivity titers of three nonenveloped viruses (i.e., rotavirus, adenovirus, and rhinovirus) by greater than 3 logs. Other nonenveloped viruses such as hepatitis A and enteroviruses (e.g., poliovirus) may require 70 percent–80 percent alcohol to be reliably inactivated. However, both 70 percent ethanol and a 62 percent ethanol foam product with emollients reduced hepatitis A virus titers on whole hands or fingertips more than nonmedicated soap; both were equally as effective as anti-microbial soap containing 4 percent chlorhexidine gluconate in reducing reduced viral counts on hands. In the same study, both 70 percent ethanol and the 62 percent ethanol foam product demonstrated greater virucidal activity against poliovirus than either nonanti-microbial soap or a 4 percent chlorhexidine gluconate-containing soap. However, depending on the alcohol concentration, the amount of time that hands are exposed to the alcohol, and viral variant, alcohol may not be effective against hepatitis A and other nonlipophilic viruses. The inactivation of nonenveloped viruses is influenced by temperature, disinfectant-virus volume ratio, and protein load. Ethanol has greater activity against viruses than isopropanol. Further in vitro and in vivo studies of both alcohol-based formulations and anti-microbial soaps are warranted to establish the minimal level of virucidal activity that is required to interrupt direct contact transmission of viruses in health care settings.

Alcohols are not appropriate for use when hands are visibly dirty or contaminated with proteinaceous materials. However, when relatively small amounts of proteinaceous material (e.g., blood) are present, ethanol and isopropanol may reduce viable bacterial counts on hands more than plain soap or antimicrobial soap. Alcohol can prevent the transfer of health care-associated pathogens. In one study, gram-negative bacilli were transferred from a colonized patient's skin to a piece of catheter material via the hands of nurses in only 17 percent of experiments after antiseptic hand rub with an alcohol-based hand rinse. In contrast, transfer of the organisms occurred in 92 percent of experiments after handwashing with plain soap and water. This experimental model indicates that when the hands of health-care personnel are heavily contaminated, an antiseptic hand rub using an alcohol-based rinse can prevent pathogen transmission more effectively than can handwashing with plain soap and water.

Alcohol-based products are more effective for standard handwashing or hand antisepsis than soap or antimicrobial soaps. In all but two of the trials that compared alcohol-based solutions with antimicrobial soaps or detergents, alcohol reduced bacterial counts on hands more than washing hands with soaps or detergents containing hexachlorophene, povidone-iodine, 4 percent chlorhexidine, or triclosan. In studies examining antimicrobial-resistant organisms, alcohol-based products reduced the number of multidrug-resistant pathogens recovered from the hands of health-care personel more effectively than did handwashing with soap and water.

Alcohols are effective for preoperative cleaning of the hands of surgical personnel. In multiple studies, bacterial counts on the hands were determined immediately after using the product and again 1–3 hours later; the delayed testing was performed to determine if regrowth of bacteria on the hands is inhibited during operative procedures. Alcohol-based solutions were more effective than washing hands with plain soap in all studies, and they reduced bacterial counts on the hands more than antimicrobial soaps or detergents in the majority of experiments. In addition, the majority of alcohol-based preparations were more effective than povidone-iodine or chlorhexidine. The efficacy of alcohol-based hand-hygiene products is affected by several factors, including the type of alcohol used, concentration of alcohol, contact time, volume of alcohol used, and whether the hands are wet when the alcohol is applied.

Applying small volumes (i.e., 0.2–0.5 ml) of alcohol to the hands is not more effective than washing hands with plain soap and water. One study documented that 1 ml of alcohol was substantially less effective than 3 ml (91). The ideal volume of product to apply to the hands is not known and may vary for different formulations. However, if hands feel dry after rubbing hands together for 10–15 seconds, an insufficient volume of product likely was applied. Because alcohol-impregnated towelettes contain a limited amount of alcohol, their effectiveness is comparable to that of soap and water. Alcohol-based hand rubs intended for use in hospitals are available as low viscosity rinses, gels, and foams. Limited data are available regarding the relative efficacy of various formulations.

One field trial demonstrated that an ethanol gel was slightly more effective than a comparable ethanol solution at reducing bacterial counts on the hands. However, a more recent study indicated that rinses reduced bacterial counts on the hands more than the gels tested. Further studies are warranted to determine the relative efficacy of alcohol-based rinses and gels in reducing transmission of health care-associated pathogens. Frequent use of alcohol-based formulations for hand antisepsis can cause drying of the skin unless emollients, humectants, or other skin-conditioning agents are added to the formulations. The drying effect of alcohol can be reduced or eliminated by adding 1 percent–3 percent glycerol or other skin conditioning agents.

Moreover, in several recent prospective trials, alcohol-based rinses or gels containing emollients caused substantially less skin irritation and dryness than the soaps or anti-microbial detergents tested. These studies, which were conducted in clinical settings, used various subjective and objective methods for assessing skin irritation and dryness. Further studies are warranted to establish whether products with different formulations yield similar results. Even well-tolerated alcohol hand rubs containing emollients may cause a transient stinging sensation at the site of any broken skin (e.g., cuts and abrasions). Alcohol-based hand-rub preparations with strong fragrances may be poorly tolerated by health-care personnel with respiratory allergies. Allergic contact dermatitis or contact urticaria syndrome caused by hypersensitivity to alcohol or to various additives present in certain alcohol hand rubs occurs only rarely.

Alcohols are flammable. Flash points of alcohol-based hand rubs range from 21 degrees C to 24 degrees C, depending on the type and concentration of alcohol present. As a result, alcohol-based hand rubs should be stored away from high temperatures or flames in accordance with National Fire Protection Agency recommendations.

Frequency and pathophysiology of irritant contact dermatitis

In certain surveys, approximately 25 percent of nurses report symptoms or signs of dermatitis involving their hands, and as many as 85 percent give a history of having skin problems. Frequent and repeated use of hand-hygiene products, particularly soaps and other detergents, is a primary cause of chronic irritant contact dermatitis among health-care personnel. The potential of detergents to cause skin irritation can vary considerably and can be ameliorated by the addition of emollients and humectants. Irritation associated with antimicrobial soaps may be caused by the antimicrobial agent or by other ingredients of the formulation. Affected persons often complain of a feeling of dryness or burning; skin that feels "rough;" and erythema, scaling, or fissures. Detergents damage the skin by causing denaturation of stratum corneum proteins, changes in intercellular lipids (either depletion or reorganization of lipid moieties), decreased corneocyte cohesion, and decreased stratum corneum water-binding capacity.

Damage to the skin also changes skin flora, resulting in more frequent colonization by staphylococci and gram-negative bacilli. Although alcohols are among the safest antiseptics available, they can cause dryness and irritation of the skin. Ethanol is usually less irritating than n-propanol or isopropanol. Irritant contact dermatitis is more commonly reported with iodophors. Other antiseptic agents that can cause irritant contact dermatitis (in order of decreasing frequency) include chlorhexidine, PCMX, triclosan, and alcohol-based products. Skin that is damaged by repeated exposure to detergents may be more susceptible to irritation by alcohol-based preparations. The irritancy potential of commercially prepared hand hygiene products, which is often determined by measuring transepidermal water loss, may be available from the manufacturer. Other factors that can contribute to dermatitis associated with frequent handwashing include using hot water for handwashing, low relative humidity (most common in winter months), failure to use supplementary hand lotion or cream, and the quality of paper towel. Shear forces associated with wearing or removing gloves and allergy to latex proteins may also contribute to dermatitis of the hands of health-care personnel.

Allergic contact dermatitis associated with hand-hygiene products

Allergic reactions to products applied to the skin (i.e., contact allergies) may present as delayed type reactions (i.e., allergic contact dermatitis) or less commonly as immediate reactions (i.e., contact urticaria). The most common causes of contact allergies are fragrances and preservatives; emulsifiers are less common causes. Liquid soaps, hand lotions or creams, and "udder ointments" may contain ingredients that cause contact allergies among health-care personnel.

Allergic reactions to antiseptic agents, including quaternary ammonium compounds, iodine or iodophors, chlorhexidine, triclosan, PCMX, and alcohols have been reported. Allergic contact dermatitis associated with alcohol-based hand rubs is uncommon. Surveillance at a large hospital in Switzerland, where a commercial alcohol hand rub has been used for more than 10 years, failed to identify a single case of documented allergy to the product. In late 2001, a Freedom of Information request for data in the FDA's Adverse Event Reporting System regarding adverse reactions to popular alcohol hand rubs in the United States yielded only one reported case of an erythematous rash reaction attributed to such a product (John M. Boyce, M.D., Hospital of St. Raphael, New Haven, Connecticut, personal communication, 2001). However, with increasing use of such products by health-care personnel, true allergic reactions to such products likely will be encountered.

Allergic reactions to alcohol-based products may represent true allergy to alcohol, allergy to an impurity or aldehyde metabolite, or allergy to another constituent of the product. Allergic contact dermatitis or immediate contact urticarial reactions may be caused by ethanol or isopropanol. Allergic reactions can be caused by compounds that may be present as inactive ingredients in alcohol-based hand rubs, including fragrances, benzyl alcohol, stearyl or isostearyl alcohol, phenoxyethanol, myristyl alcohol, propylene glycol, parabens, and benzalkonium chloride.

Proposed methods for reducing adverse effects of agents

Potential strategies for minimizing hand-hygiene–related irritant contact dermatitis among health-care personnel include reducing the frequency of exposure to irritating agents (particularly anionic detergents), replacing products with high irritation potential with preparations that cause less damage to the skin, educating personnel regarding the risks of irritant contact dermatitis, and providing caregivers with moisturizing skin-care products or barrier creams. Reducing the frequency of exposure to hand-hygiene products would prove difficult and is not desirable because of the low levels of adherence to hand-hygiene policies in the majority of institutions. Although hospitals have provided personnel with nonantimicrobial soaps in hopes of minimizing dermatitis, frequent use of such products may cause greater skin damage, dryness, and irritation than antiseptic preparations. One strategy for reducing the exposure of personnel to irritating soaps and detergents is to promote the use of alcohol-based hand rubs containing various emollients. Several recent prospective, randomized trials have demonstrated that alcohol-based hand rubs containing emollients were better tolerated than washing hands with nonantimicrobial soaps or antimicrobial soaps. Routinely washing hands with soap and water immediately after using an alcohol hand rub may lead to dermatitis. Therefore, personnel should be reminded that it is neither necessary nor recommended to routinely wash hands after each application of an alcohol hand rub.

Hand lotions and creams often contain humectants and various fats and oils that can increase skin hydration and replace altered or depleted skin lipids that contribute to the barrier function of normal skin. Several controlled trials have demonstrated that regular use (e.g., twice a day) of such products can help prevent and treat irritant contact dermatitis caused by hand-hygiene products. In one study, frequent and scheduled use of an oil-containing lotion improved skin condition, and thus led to a 50 percent increase in handwashing frequency by health-care personnel. Reports from these studies emphasize the need to educate personnel regarding the value of regular, frequent use of hand-care products.

Recently, barrier creams have been marketed for the prevention of hand-hygiene–related irritant contact dermatitis. Such products are absorbed to the superficial layers of the epidermis and are designed to form a protective layer that is not removed by standard handwashing. Two recent randomized, controlled trials that evaluated the skin condition of caregivers demonstrated that barrier creams did not yield better results than did the control lotion or vehicle used. As a result, whether barrier creams are effective in preventing irritant contact dermatitis remains unknown. In addition to evaluating the efficacy and acceptability of hand-care products, product-selection committees should inquire about the potential deleterious effects that oil-containing products may have on the integrity of rubber gloves and on the efficacy of antiseptic agents used in the facility.

Factors to consider when selecting hand-hygiene products

When evaluating hand-hygiene products for potential use in health-care facilities, administrators or product-selection committees must consider factors that can affect the overall efficacy of such products, including the relative efficacy of antiseptic agents against various pathogens and acceptance of hand-hygiene products by personnel. Soap products that are not well-accepted by health-care personnel can be a deterrent to frequent handwashing. Characteristics of a product (either soap or alcohol-based hand rub) that can affect acceptance by personnel include its smell, consistency (i.e., "feel"), and color. For soaps, ease of lathering also may affect user preference.

Because health-care personnel may wash their hands from a limited number of times per shift to as many as 30 times per shift, the tendency of products to cause skin irritation and dryness is a substantial factor that influences acceptance, and ultimate usage. For example, concern regarding the drying effects of alcohol was a primary cause of poor acceptance of alcohol-based hand-hygiene products in hospitals in the United States. However, several studies have demonstrated that alcohol-based hand rubs containing emollients are acceptable to users. With alcohol-based products, the time required for drying may also affect user acceptance.

Studies indicate that the frequency of handwashing or antiseptic handwashing by personnel is affected by the accessibility of hand-hygiene facilities. In contrast to sinks used for handwashing or antiseptic handwash, dispensers for alcohol-based hand rubs do not require plumbing and can be made available in patient care areas. Pocket carriage of alcohol-based hand-rub solutions, combined with availability of bedside dispensers, has been associated with substantial improvement in adherence to hand-hygiene protocols. To avoid any confusion between soap and alcohol hand rubs, alcohol hand-rub dispensers should not be placed adjacent to sinks. Health-care personnel should be informed that washing hands with soap and water after each use of an alcohol hand rub is not necessary and is not recommended, because it may lead to dermatitis. However, because personnel feel a "build-up" of emollients on their hands after repeated use of alcohol hand gels, washing hands with soap and water after 5–10 applications of a gel has been recommended by certain manufacturers.

Automated handwashing machines have not been demonstrated to improve the quality or frequency of handwashing. Although technologically advanced automated handwashing devices and monitoring systems have been developed recently, only a minimal number of studies have been published that demonstrate that use of such devices results in enduring improvements in hand-hygiene adherence among health-care personnel. Further evaluation of automated handwashing facilities and monitoring systems is warranted. Dispenser systems provided by manufacturers or vendors also must be considered when evaluating hand-hygiene products. Dispensers may discourage use by health-care personnel when they 1) become blocked or partially blocked and do not deliver the product when accessed by personnel, and 2) do not deliver the product appropriately onto the hands. In one hospital where a viscous alcohol-based hand rinse was available, only 65 percent of functioning dispensers delivered product onto the caregivers' hands with one press of the dispenser lever, and 9 percent of dispensers were totally occluded. In addition, the volume delivered was often suboptimal, and the product was sometimes squirted onto the wall instead of the caregiver's hand.

Hand-hygiene practices among health-care personnel

In observational studies conducted in hospitals, health-care personnel washed their hands an average of five times per shift to as many as 30 times per shift; certain nurses washed their hands less than 100 times per shift. Hospitalwide surveillance of hand hygiene reveals that the average number of handwashing opportunities varies markedly between hospital wards. For example, nurses in pediatric wards had an average of eight opportunities for hand hygiene per hour of patient care compared with an average of 20 for nurses in intensive-care units. The duration of handwashing or hygienic handwash episodes by health-care personnel has averaged 6.6–24.0 seconds in observational studies. In addition to washing their hands for limited time periods, personnel often fail to cover all surfaces of their hands and fingers.

OTHER POLICIES RELATED TO HAND HYGIENE

Fingernails and artificial nails

Studies have documented that subungual areas of the hand harbor high concentrations of bacteria, most frequently coagulase-negative staphylococci, gram-negative rods (including pseudomonas spp.), corynebacteria, and yeasts. Freshly applied nail polish does not increase the number of bacteria recovered from periungual skin, but chipped nail polish may support the growth of larger numbers of organisms on fingernails. Even after careful handwashing or the use of surgical scrubs, personnel often harbor substantial numbers of potential pathogens in the subungual spaces.

Whether artificial nails contribute to transmission of health care-associated infections is unknown. However, people who wear artificial nails are more likely to harbor gram-negative pathogens on their fingertips than are those who have natural nails, both before and after handwashing. Whether the length of natural or artificial nails is a substantial risk factor is unknown, because the majority of bacterial growth occurs along the proximal 1 mm of the nail adjacent to subungual skin. Recently, an outbreak of P. aeruginosa in a neonatal intensive care unit was attributed to two nurses (one with long natural nails and one with long artificial nails) who carried the implicated strains of pseudomonas spp. on their hands. Patients were substantially more likely than controls to have been cared for by the two nurses during the exposure period, indicating that colonization of long or artificial nails with pseudomonas spp. may have contributed to causing the outbreak. Personnel wearing artificial nails also have been epidemiologically implicated in several other outbreaks of infection caused by gram-negative bacilli and yeast. Although these studies provide evidence that wearing artificial nails poses an infection hazard, additional studies are warranted.

Gloving policies

CDC has recommended that health-care personnel wear gloves to 1) reduce the risk of acquiring infections from patients, 2) prevent health care personnel flora from being transmitted to patients, and 3) reduce transient contamination of the hands of personnel by flora that can be transmitted from one patient to another. Before the emergence of the acquired immunodeficiency syndrome (AIDS) epidemic, gloves were worn primarily by those caring for patients colonized or infected with certain pathogens or by those exposed to patients with a high risk of hepatitis B. Since 1987, a dramatic increase in glove use has occurred in an effort to prevent transmission of HIV and other blood-borne pathogens from patients to health-care personnel. The Occupational Safety and Health Administration (OSHA) mandates that gloves be

worn during all patient-care activities that may involve exposure to blood or body fluids that may be contaminated with blood.

Having more than one type of glove available is desirable, because it allows personnel to select the type that best suits their patient-care activities. Although recent studies indicate that improvements have been made in the quality of gloves, hands should be decontaminated or washed after removing gloves. Gloves should not be washed or reused. Use of petroleum-based hand lotions or creams may adversely affect the integrity of latex gloves. After use of powdered gloves, certain alcohol hand rubs may interact with residual powder on the hands of personnel, resulting in a gritty feeling on the hands. In facilities where powdered gloves are commonly used, various alcohol-based hand rubs should be tested after removal of powdered gloves to avoid selecting a product that causes this undesirable reaction. Personnel should be reminded that failure to remove gloves between patients may contribute to transmission of organisms.

Jewelry

Several studies have demonstrated that skin underneath rings is more heavily colonized than comparable areas of skin on fingers without rings. One study found that 40 percent of nurses harbored gram-negative bacilli (e.g., E. cloacae, klebsiella, and acinetobacter) on skin under rings and that certain nurses carried the same organism under their rings for several months. In a more recent study involving more than 60 intensive care unit nurses, multivariable analysis revealed that rings were the only substantial risk factor for carriage of gram-negative bacilli and S. aureus and that the concentration of organisms recovered correlated with the number of rings worn. Whether the wearing of rings results in greater transmission of pathogens is unknown. Two studies determined that mean bacterial colony counts on hands after handwashing were similar among persons wearing rings and those not wearing rings. Further studies are needed to establish if wearing rings results in greater transmission of pathogens in health care settings.

Conclusion

The results of these studies demonstrated that the use of alcohol-based products containing emollients were better tolerated by health-care personnel than washing hands with nonantimicrobial soaps or antimicrobial soap and that these products are more effective in reducing the bacterial counts on the hands. One of the most important factors it also highlights is the intermediate cause of antimicrobial spread: poor adherence to hand-hygiene policies. This must be an immediate focus in all health care facilities to prevent the transmission of pathogens from patient to health care provider to patient.

References

- www.ccd.gov, "Guidelines for Hand Hygiene in Health-Care Settings", 10/25/2002/,Vol.51 No.RR-16

GUIDELINES FOR HAND HYGIENE IN HEALTH CARE SETTINGS

Final Examination Questions

Select the best answer questions 6 through 10 and mark your answers on the Final Examination Answer Sheet found on page 148, or for faster service complete your test online at **Dental.EliteCME.com.**

6. Transient flora, which colonize the superficial layers of the skin, are more amenable to removal by routine handwashing.
   - ○ True
   - ○ False

7. Hepatitis B virus is an enveloped virus that is somewhat less susceptible but is killed by 20 percent–30 percent alcohol; hepatitis C virus also is likely killed by this percentage of alcohol.
   - ○ True
   - ○ False

8. In all but two of the trials that compared alcohol-based solutions with antimicrobial soaps or detergents, alcohol reduced bacterial counts on hands more than washing hands with soaps or detergents containing hexachlorophene, povidone- iodine, 4 percent chlorhexidine, or triclosan.
   - ○ True
   - ○ False

9. Health-care personnel who wear artificial nails are more likely to harbor gram-negative pathogens on their fingertips than are those who have natural nails, both before and after handwashing.
   - ○ True
   - ○ False

10. Hands need not be decontaminated or washed after removing gloves.
    - ○ True
    - ○ False

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Chapter 3: Guidelines for Infection Control in Dental Health Care Settings

5 CE Hours

By: Elite Staff

Learning objectives

- Establish the process of educating and protecting dental health care personnel.
- Explain the prevention of transmission of blood-borne pathogens.
- Discuss the concerns of hand hygiene.
- Identify protection against spatter by use of personal protective equipment.
- Learn how to avoid the risk of contact dermatitis and latex hypersensitivity.
- Emphasize the importance of sterilization and disinfection of patient-care items.
- List the special considerations that should be taken with dental handpieces, waterlines, water quality, biofilm, radiology, oral surgical procedures and dental laboratories.

Introduction

Infection control and health care epidemiology is the discipline concerned with preventing the spread of infections within the health care setting. As such, it is a practical (rather than an academic) subdiscipline of epidemiology. It is an essential (though often underrecognized and undersupported) part of the infrastructure of health care. Infection control and hospital epidemiology are akin to public health practice, practiced within the confines of a particular health care delivery system rather than directed at society as a whole.

Infection control concerns itself both with prevention (hand hygiene/ hand-washing, cleaning/disinfection/ sterilization, vaccination, surveillance) and with investigation and management of a demonstrated or suspected spread of infection within a particular health care setting (e.g., outbreak investigation). It is on this basis that the common title being adopted within health care is "infection prevention and control."

This course consolidates recommendations for preventing and controlling infectious diseases and managing personnel health and safety concerns related to infection control in dental settings. It:

- Updates and revises previous CDC recommendations regarding infection control in dental settings.
- Incorporates relevant infection control measures from other CDC guidelines.
- Discusses concerns not addressed in previous recommendations for dentistry. These updates and additional topics include:
    - Application of standard precautions rather than universal precautions.
    - Work restrictions for health care personnel (HCP) infected with or occupationally exposed to infectious diseases.
    - Management of occupational exposures to blood-borne pathogens, including post-exposure prophylaxis (PEP) for work exposures to hepatitis B virus (HBV), hepatitis C virus (HCV); and human immunodeficiency virus (HIV).
    - Selection and use of devices with features designed to prevent sharps injury.
    - Hand-hygiene products and surgical hand antisepsis.
    - Contact dermatitis and latex hypersensitivity.
    - Sterilization of unwrapped instruments.
    - Dental water-quality concerns (e.g., dental unit waterline biofilms; delivery of water of acceptable biological quality for patient care; usefulness of flushing waterlines; use of sterile irrigating solutions).
    - Oral surgical procedures; handling of community boilwater advisories.
    - Dental radiology.
    - Aseptic technique for parenteral medications.
    - Preprocedural mouth rinsing for patients.
    - Oral surgical procedures.
    - Laser/electrosurgery plumes.
    - Tuberculosis (TB).
    - Creutzfeldt-Jakob disease (CJD) and other prion-related diseases.
    - Infection control program evaluation.
    - Research considerations.

These guidelines were developed by CDC staff members in collaboration with other authorities on infection control. Draft documents were reviewed by other federal agencies and professional organizations from the fields of dental health care, public health and hospital epidemiology and infection control. A Federal Register notice elicited public comments that were considered in the decision-making process. Existing guidelines and published research pertinent to dental infection control principles and practices were reviewed. Wherever possible, recommendations are based on data from well-designed scientific studies. However, only a limited number of studies have characterized risk factors and the effectiveness of prevention measures for infections associated with dental health care practices.

Some infection control practices routinely used by health care practitioners cannot be rigorously examined for ethical or logistical reasons. In the absence of scientific evidence for such practices, certain recommendations are based on strong theoretical rationale, suggestive evidence or opinions of respected authorities based on clinical experience, descriptive studies or committee reports. In addition, some recommendations are derived from federal regulations. No recommendations are offered for practices for which insufficient scientific evidence or lack of consensus supporting their effectiveness exists.

Background

In the United States, an estimated 9 million persons work in health care professions, including approximately 168,000 dentists, 112,000 registered dental hygienists, 218,000 dental assistants and 53,000 dental laboratory technicians. In this report, dental health care personnel (DHCP) refers to all paid and unpaid personnel in the dental health care setting who might be occupationally exposed to infectious materials, including body substances and contaminated supplies, equipment, environmental surfaces, water, or air. Dental health care personnel includes dentists, dental hygienists, dental assistants, dental laboratory technicians (in-office and commercial), students and trainees, contractual personnel and other

persons not directly involved in patient care but potentially exposed to infectious agents (e.g., administrative, clerical, housekeeping, maintenance or volunteer personnel).

Recommendations in this report are designed to prevent or reduce potential for disease transmission from patient to dental health care personnel, from dental workers to patient, and from patient to patient. Although these guidelines focus mainly on outpatient, ambulatory dental health care settings, the recommended infection control practices are applicable to all settings in which dental treatment is provided. Dental patients and workers can be exposed to pathogenic microorganisms including cytomegalovirus (CMV), HBV, HCV, herpes simplex virus types 1 and 2, HIV, Mycobacterium tuberculosis, staphylococci, streptococci and other viruses and bacteria that colonize or infect the oral cavity and respiratory tract. These organisms can be transmitted in dental settings through:

- Direct contact with blood, oral fluids or other patient materials.
- Indirect contact with contaminated objects (e.g., instruments, equipment or environmental surfaces).
- Contact of conjunctival, nasal or oral mucosa with droplets (e.g., spatter) containing microorganisms generated from an infected person and propelled a short distance (e.g., by coughing, sneezing or talking).
- Inhalation of airborne microorganisms that can remain suspended in the air for long periods.

Infection through any of these routes requires that all of the following conditions be present:

- A pathogenic organism of sufficient virulence and in adequate numbers to cause disease.
- A reservoir or source that allows the pathogen to survive and multiply (e.g., blood).
- A mode of transmission from the source to the host.
- A portal of entry through which the pathogen can enter the host.
- A susceptible host (i.e., one who is not immune).

Occurrence of these events provides the chain of infection. Effective infection control strategies prevent disease transmission by interrupting one or more links in the chain.

Previous CDC recommendations regarding infection control for dentistry focused primarily on the risk of transmission of blood-borne pathogens among dental care personnel and patients and use of universal precautions to reduce that risk. Universal precautions were based on the concept that all blood and body fluids that might be contaminated with blood should be treated as infectious because patients with blood-borne infections can be asymptomatic or unaware they are infected. Preventive practices used to reduce blood exposures, particularly percutaneous exposures, include:

- Careful handling of sharp instruments.
- Use of rubber dams to minimize blood spattering.
- Hand washing.
- Use of protective barriers (e.g., gloves, masks, protective eyewear and gowns).

The relevance of universal precautions to other aspects of disease transmission was recognized, and in 1996, CDC expanded the concept and changed the term to standard precautions. Standard precautions integrate and expand the elements of universal precautions into a standard of care designed to protect health care personnel and patients from pathogens that can be spread by blood or any other body fluid, excretion or secretion.

Standard precautions apply to contact with:

- Blood.
- All body fluids, secretions and excretions (except sweat), regardless of whether they contain blood.
- Nonintact skin.
- Mucous membranes.

Saliva has always been considered a potentially infectious material in dental infection control; thus, no operational difference exists in clinical dental practice between universal precautions and standard precautions.

In addition to standard precautions, other measures (e.g., expanded or transmission-based precautions) might be necessary to prevent potential spread of certain diseases (e.g., TB, influenza and varicella) that are transmitted through airborne, droplet or contact transmission (e.g., sneezing, coughing and contact with skin). When acutely ill with these diseases, patients do not usually seek routine dental outpatient care. Nonetheless, a general understanding of precautions for diseases transmitted by all routes is critical because:

- Some dental health workers are hospital-based or work part-time in hospital settings.
- Patients infected with these diseases might seek urgent treatment at outpatient dental offices.
- Dental workers might become infected with these diseases.

Necessary transmission-based precautions might include patient placement (e.g., isolation), adequate room ventilation, respiratory protection (e.g., N-95 masks) for workers, or postponement of nonemergency dental procedures.

Dental health care personnel should be familiar also with the hierarchy of controls that categorizes and prioritizes prevention strategies. For blood-borne pathogens, engineering controls that eliminate or isolate the hazard (e.g., puncture-resistant sharps containers or needle retraction devices) are the primary strategies for protecting dental workers and patients. Where engineering controls are not available or appropriate, work-practice controls that result in safer behaviors (e.g., one-hand needle recapping or not using fingers for cheek retraction while using sharp instruments or suturing), and use of personal protective equipment (PPE) (e.g., protective eyewear, gloves and mask) can prevent exposure. In addition, administrative controls (e.g., policies, procedures and enforcement measures targeted at reducing the risk of exposure to infectious persons) are a priority for certain pathogens (e.g., M. tuberculosis), particularly those spread by airborne or droplet routes.

Dental practices should develop a written infection control program to prevent or reduce the risk of disease transmission. Such a program should include establishment and implementation of policies, procedures and practices (in conjunction with selection and use of technologies and products) to prevent work-related injuries and illnesses among dental care workers as well as health care-associated infections among patients. The program should embody principles of infection control and occupational health, reflect current science and adhere to relevant federal, state and local regulations and statutes. An infection control coordinator (e.g., dentist or other dental health worker) knowledgeable or willing to be trained should be assigned responsibility for coordinating the program. The effectiveness of the infection control program should be evaluated on a day-to-day basis and over time to help ensure that policies, procedures and practices are useful, efficient, and successful.

Although the infection control coordinator remains responsible for overall management of the program, creating and maintaining a safe work environment ultimately requires the commitment and accountability of all dental workers. This course is designed to provide guidance to workers for preventing disease transmission in dental health care settings, for promoting a safe working environment and for assisting dental practices in developing and implementing infection control programs. These programs should be followed in addition to practices and procedures for worker protection required by the Occupational Safety and Health Administration's (OSHA) standards for occupational exposure to blood-borne pathogens, including instituting controls to protect employees from exposure to blood or other potentially infectious materials (OPIM), and requiring implementation of a written exposure-control plan, annual

employee training, HBV vaccinations and post-exposure follow-up. Interpretations and enforcement procedures are available to help dental workers apply this OSHA standard in practice. Also, manufacturers' Material Safety Data Sheets (MSDS) should be consulted regarding correct procedures for handling or working with hazardous chemicals.

REVIEW OF SCIENCE RELATED TO DENTAL INFECTION CONTROL

Personnel health elements of an infection control program

A protective health component for dental health care personnel is an integral part of a dental practice infection control program. The objectives are to educate workers about the principles of infection control, identify work-related infection risks, institute preventive measures and ensure prompt exposure management and medical follow-up. Coordination between the dental practice's infection control coordinator and other qualified health care professionals is necessary to provide dental workers with appropriate services. Dental programs in institutional settings, (e.g., hospitals, health centers and educational institutions) can coordinate with departments that provide personnel health services.

However, the majority of dental practices are in ambulatory, private settings that do not have licensed medical staff and facilities to provide complete on-site health service programs. In such settings, the infection control coordinator should establish programs that arrange for site-specific infection control services from external health care facilities and providers before workers are placed at risk for exposure. Referral arrangements can be made with qualified health care professionals in an occupational health program of a hospital, with educational institutions or with health care facilities that offer personnel health services.

Education and training

Personnel are more likely to comply with an infection control program and exposure control plan if they understand its rationale. Clearly written policies, procedures and guidelines can help ensure consistency, efficiency and effective coordination of activities. Personnel subject to occupational exposure should receive infection control training on initial assignment, when new tasks or procedures affect their occupational exposure, and at a minimum, annually. Education and training should be appropriate to the assigned duties of specific workers (e.g., techniques to prevent cross-contamination or instrument sterilization). For dental workers who perform tasks or procedures likely to result in occupational exposure to infectious agents, training should include:

- A description of their exposure risks.
- Review of prevention strategies and infection control policies and procedures.
- Discussion regarding how to manage work-related illness and injuries, including post-exposure prophylaxis.
- Review of work restrictions for an exposure or infection.

Inclusion of dental workers with minimal exposure risks (e.g., administrative employees) in education and training programs might enhance facility-wide understanding of infection control principles and the importance of the program. Educational materials should be appropriate in content and vocabulary for each person's educational level, literacy and language, as well as be consistent with existing federal, state and local regulations.

Immunization programs

Dental workers are at risk for exposure to, and possible infection with, infectious organisms. Immunizations substantially reduce both the number of workers susceptible to these diseases and the potential for disease transmission to other workers and patients. Thus, immunizations are an essential part of prevention and infection control programs for dental health care personnel, and a comprehensive immunization policy should be implemented for all dental health care facilities. The Advisory Committee on Immunization Practices (ACIP) provides national guidelines for immunization of health care personnel, which includes dental workers.

Dental practice immunization policies should incorporate current state and federal regulations as well as recommendations from the U.S. Public Health Service and professional organizations.

On the basis of documented health care-associated transmission, health care workers are considered to be at substantial risk for acquiring or transmitting hepatitis B, influenza, measles, mumps, rubella and varicella. All of these diseases are vaccine-preventable. ACIP recommends that all health care personnel be vaccinated or have documented immunity to these diseases. ACIP does not recommend routine immunization of workers against TB (i.e., inoculation with bacille Calmette-Guérin vaccine) or hepatitis A. No vaccine exists for HCV. ACIP guidelines also provide recommendations regarding immunization of workers with special conditions (e.g., pregnancy, HIV infection or diabetes).

Immunization of workers before they are placed at risk for exposure remains the most efficient and effective use of vaccines in health care settings. Some educational institutions and infection control programs provide immunization schedules for students and workers. OSHA requires that employers make hepatitis B vaccination available to all employees who have potential contact with blood or OPIM. Employers are also required to follow CDC recommendations for vaccinations, evaluation and follow-up procedures. Nonpatient care staff (e.g., administrative or housekeeping) might be included, depending on their potential risk of coming into contact with blood or OPIM.

Employers are also required to ensure that employees who decline to accept hepatitis B vaccination sign an appropriate declination statement. Dental workers unable or unwilling to be vaccinated as required or recommended should be educated regarding their exposure risks, infection control policies and procedures for the facility, and the management of work-related illness and work restrictions (if appropriate) for exposed or infected workers.

Exposure prevention and post-exposure management

Avoiding exposure to blood and OPIM, as well as protection by immunization, remain primary strategies for reducing occupationally acquired infections, but occupational exposures can still occur. A combination of standard precautions, engineering, work practice and administrative controls is the best means to minimize occupational exposures. Written policies and procedures to facilitate prompt reporting, evaluation, counseling, treatment and medical follow-up of all occupational exposures should be available to all dental health care personnel. Written policies and procedures should be consistent with federal, state and local requirements addressing education and training, post-exposure management and exposure reporting.

Dental health personnel who have contact with patients can also be exposed to persons with infectious TB, and should have a baseline

tuberculin skin test (TST), preferably by using a two-step test at the beginning of employment. Thus, if an unprotected occupational exposure occurs, TST conversions can be distinguished from positive TST results caused by previous exposures. The facility's level of TB risk will determine the need for routine follow-up TSTs.

Medical conditions, work-related illness and work restrictions

Dental workers are responsible for monitoring their own health status. Those who have acute or chronic medical conditions that render them susceptible to opportunistic infection should discuss with their personal physicians or other qualified authority whether the condition might affect their ability to safely perform their duties.

However, under certain circumstances, health care facility managers might need to exclude dental health personnel from work or patient contact to prevent further transmission of infection. Decisions concerning work restrictions are based on the mode of transmission and the period of infectivity of the disease (Table 1). Exclusion policies should:

- Be written.
- Include a statement of authority that defines who can exclude dental workers (e.g., personal physicians).
- Be clearly communicated through education and training.

Policies should also encourage workers to report illnesses or exposures without jeopardizing wages, benefits or job status. With increasing concerns regarding blood-borne pathogens and introduction of universal precautions, use of latex gloves among health care workers has increased markedly. Increased use of these gloves has been accompanied by increased reports of allergic reactions to natural rubber latex among workers and patients, as well as increased reports of irritant and allergic contact dermatitis from frequent and repeated use of hand-hygiene products, exposure to chemicals and glove use.

Dental health workers should be familiar with the signs and symptoms of latex sensitivity. A physician should evaluate workers exhibiting symptoms of latex allergy, because further exposure could result in a serious allergic reaction. A diagnosis is made through medical history, physical examination and diagnostic tests. Procedures should be in place for minimizing latex-related health problems among DHCP and patients while protecting them from infectious materials. These procedures should include:

- Reducing exposures to latex-containing materials by using appropriate work practices.
- Training and educating dental workers on monitoring symptoms.
- Substituting nonlatex products where appropriate.

Maintenance of records, data management and confidentiality

The health status of dental workers can be monitored by maintaining records of work-related medical evaluations, screening tests, immunizations, exposures and post-exposure management. Such records must be kept in accordance with all applicable state and federal laws. Examples of laws that might apply include the Privacy Rule of the Health Insurance Portability and Accountability Act (HIPAA) of 1996, 45 CFR 160 and 164, and the OSHA Occupational Exposure to Blood-borne Pathogens; Final Rule 29 CFR 1910. 1030(h)(1)(i–iv). The HIPAA Privacy Rule applies to covered entities, including certain defined health providers, health care clearinghouses and health plans. OSHA requires employers to ensure that certain information contained in employee medical records is:

- Kept confidential.
- Not disclosed or reported without the employee's express written consent to any person within or outside the workplace except as required by the OSHA standard.
- Maintained by the employer for at least the duration of employment plus 30 years.

Dental practices that coordinate their infection control program with off-site providers might consult OSHA's blood-borne pathogen standard and employee access to medical and exposure records standard, as well as other applicable local, state and federal laws, to determine a location for storing health records.

Preventing transmission of blood-borne pathogens

Although transmission of blood-borne pathogens (e.g., HBV, HCV and HIV) in dental health care settings can have serious consequences, such transmission is rare. Exposure to infected blood can result in transmission from patient to dental workers, from workers to patients, and from one patient to another. The opportunity for transmission is greatest from patients to dental workers, who frequently encounter patient blood and blood-contaminated saliva during dental procedures. Since 1992, no HIV transmission from dental care personnel to patients has been reported, and the last HBV transmission from dental workers to patients was reported in 1987. HCV transmission from workers to patients has not been reported. The majority of dental care workers infected with a blood-borne virus do not pose a risk to patients because they do not perform activities meeting the necessary conditions for transmission. For workers to pose a risk for blood-borne virus transmission to patients, the worker must:

- Be viremic, i.e., have infectious virus circulating in the bloodstream.
- Be injured or have a condition (e.g., weeping dermatitis) that allows direct exposure to their blood or other infectious body fluids.
- Enable their blood or infectious body fluid to gain direct access to a patient's wound, traumatized tissue, mucous membranes or similar portal of entry. Although an infected worker might be viremic, unless the second and third conditions are also met, transmission cannot occur.

The risk of occupational exposure to blood-borne viruses is largely determined by their prevalence in the patient population and the nature and frequency of contact with blood and body fluids through percutaneous or permucosal routes of exposure. The risk of infection after exposure to a blood-borne virus is influenced by inoculum size, route of exposure and susceptibility of the exposed health care personnel. The majority of attention has been placed on the blood-borne pathogens HBV, HCV and HIV, and these pathogens present different levels of risk to dental care workers.

Hepatitis B virus

HBV is a well-recognized occupational risk for health care workers. HBV is transmitted by percutaneous or mucosal exposure to blood or body fluids of a person with either acute or chronic HBV infection. Persons infected with HBV can transmit the virus for as long as they are HBsAg-positive. The risk of HBV transmission is highly related to the HBeAg status of the source person. In studies of health care personnel who sustained injuries from needles contaminated with blood containing HBV, the risk of developing clinical hepatitis if the blood was positive for both HBsAg and HBeAg was 22 percent to 31 percent; the risk of developing serologic evidence of HBV infection

was 37–62 percent. By comparison, the risk of developing clinical hepatitis from a needle contaminated with HBsAg-positive, HBeAg-negative blood was 1-6 percent, and the risk of developing serologic evidence of HBV infection, 23-37 percent.

Blood contains the greatest proportion of HBV infectious particle titers of all body fluids and is the most critical vehicle of transmission in the health care setting. HBsAg is also found in multiple other body fluids, including breast milk, bile, cerebrospinal fluid, feces, nasopharyngeal washings, saliva, semen, sweat and synovial fluid. However, the majority of body fluids are not efficient vehicles for transmission because they contain low quantities of infectious HBV, despite the presence of HBsAg. The concentration of HBsAg in body fluids can be 100-1,000 times greater than the concentration of infectious HBV particles.

Although percutaneous injuries are among the most efficient modes of HBV transmission, these exposures probably account for only a minority of HBV infections among health care workers. In multiple investigations of nosocomial hepatitis B outbreaks, the majority of infected health care workers could not recall an overt percutaneous injury, although in certain studies, approximately one-third of infected workers recalled caring for a patient who was HBsAg-positive. In addition, HBV has been demonstrated to survive in dried blood at room temperature on environmental surfaces for one week. Thus, HBV infections that occur in workers with no history of nonoccupational exposure or occupational percutaneous injury might have resulted from direct or indirect blood or body fluid exposures that inoculated HBV into cutaneous scratches, abrasions, burns, other lesions or on mucosal surfaces. The potential for HBV transmission through contact with environmental surfaces has been demonstrated in investigations of HBV outbreaks among patients and health care personnel in hemodialysis units.

Since the early 1980s, occupational infections among health workers have declined because of vaccine use and adherence to universal precautions. Among U. S. dentists, more than 90 percent have been vaccinated, and serologic evidence of past HBV infection decreased from prevaccine levels of 14 percent in 1972, to approximately 9 percent in 1992. During 1993–2001, levels remained relatively unchanged. Infection rates can be expected to decline further as vaccination rates remain high among young dentists and as older dentists with lower vaccination rates and higher rates of infection retire.

Although the potential for transmission of blood-borne infections from dental care workers to patients is considered limited, precise risks have not been quantified by carefully designed epidemiologic studies. Reports published during 1970-1987 describe nine clusters in which patients were thought to be infected with HBV through treatment by an infected dental worker. However, transmission of HBV from dentist to patient has not been reported since 1987, possibly reflecting such factors as:

- Adoption of universal precautions.
- Routine glove use.
- Increased levels of immunity as a result of hepatitis B vaccination of dental care workers.
- Implementation of the 1991 OSHA blood-borne pathogen standard.
- Incomplete ascertainment and reporting.

Standard precautions are strategies used to reduce the risk of infection from exposure to blood, all body fluids and secretions (except sweat), non-intact skin and mucous membranes.

Only one case of patient-to-patient transmission of HBV in the dental setting has been documented (CDC, unpublished data, 2003). In this case, appropriate office infection control procedures were being followed, and the exact mechanism of transmission was undetermined.

Because of the high risk of HBV infection to health care and dental workers who perform tasks that might involve contact with blood, blood-contaminated body substances, other body fluids or sharps, such workers should be vaccinated. Vaccination can protect both dental care workers and patients from HBV infection and, whenever possible, should be completed when dentists or other dental staff are in training and before they have contact with blood.

Prevaccination serological testing for previous infection is not indicated, although it can be cost-effective where prevalence of infection is expected to be high in a group of potential vacinees (e.g., persons who have emigrated from areas with high rates of HBV infection). Dental workers should be tested for anti-HBs 1-2 months after completion of the three-dose vaccination series. Those who do not develop an adequate antibody response (i.e., anti-HBs less than 10 mIU/mL) to the primary vaccine series should complete a second three-dose vaccine series or be evaluated to determine if they are HBsAg-positive. Revaccinated persons should be retested for anti-HBs at the completion of the second vaccine series. Approximately half of nonresponders to the primary series will respond to a second three-dose series. If no antibody response occurs after the second series, testing for HBsAg should be performed.

Persons who prove to be HBsAg-positive should be counseled regarding how to prevent HBV transmission to others and regarding the need for medical evaluation. Nonresponders to vaccination who are HBsAg-negative should be considered susceptible to HBV infection and should be counseled regarding precautions to prevent HBV infection and the need to obtain HBIG prophylaxis for any known or probable parenteral exposure to HBsAg-positive blood.

Vaccine-induced antibodies decline gradually over time, and 60 percent of persons who initially respond to vaccination will lose detectable antibodies over 12 years. Even so, immunity continues to prevent clinical disease or detectable viral infection. Booster doses of vaccine and periodic serologic testing to monitor antibody concentrations after completion of the vaccine series are not necessary for vaccine responders.

Hepatitis D virus

An estimated 4 percent of persons with acute HBV infection are also infected with hepatitis delta virus (HDV). Discovered in 1977, HDV is a defective blood-borne virus requiring the presence of HBV to replicate. Patients co-infected with HBV and HDV have substantially higher mortality rates than those infected with HBV alone. Because HDV infection is dependent on HBV for replication, immunization to prevent HBV infection, through either pre- or post-exposure prophylaxis, can also prevent HDV infection.

Hepatitis C virus

Hepatitis C virus appears not to be transmitted efficiently through occupational exposures to blood. Follow-up studies of HCP exposed to HCV-infected blood through percutaneous or other sharps injuries have determined a low incidence of seroconversion (mean: 1.8 percent; range, 0 percent-7 percent). One study determined transmission occurred from hollow-bore needles but not other sharps. Although these studies have not documented seroconversion associated with mucous membrane or nonintact skin exposure, at least two cases of HCV transmission from a blood splash to the conjunctiva and one case of simultaneous transmission of HCV and HIV after nonintact skin exposure have been reported.

Data are insufficient to estimate the occupational risk of HCV infection among health care workers, but the majority of studies indicate the prevalence of HCV infection among dentists, surgeons and hospital-based workers is similar to that among the general population, approximately 1-2 percent. In a study that evaluated risk factors for infection, a history of unintentional needlesticks was the only occupational risk factor independently associated with HCV infection.

No studies of transmission from HCV-infected dental workers to patients have been reported, and the risk for such transmission appears limited. Multiple reports have been published describing transmission from HCV-infected surgeons, which apparently occurred during performance of invasive procedures; the overall risk for infection averaged 0.17 percent.

Human immunodeficiency virus

In the United States, the risk of HIV transmission in dental settings is extremely low. As of December 2001, a total of 57 cases of HIV seroconversion had been documented among health care workers, but none among dental care workers, after occupational exposure to a known HIV-infected source. Transmission of HIV to six patients of a single dentist with AIDS has been reported, but the mode of transmission could not be determined. As of Sept. 30, 1993, CDC had information regarding test results of more than 22,000 patients of 63 HIV-infected health care workers, including 33 dentists or dental students. No additional cases of transmission were documented.

Prospective studies worldwide indicate the average risk of HIV infection after a single percutaneous exposure to HIV-infected blood is 0.3 percent (range: 0.2-0.5 percent). After an exposure of mucous membranes in the eye, nose or mouth, the risk is approximately 0.1 percent. The precise risk of transmission after skin exposure remains unknown, but is believed to be even smaller than that for mucous membrane exposure.

Certain factors affect the risk of HIV transmission after an occupational exposure. Laboratory studies have determined that if needles that pass through latex gloves are solid rather than hollow-bore, or are of small gauge (e.g., anesthetic needles commonly used in dentistry), they transfer less blood. In a retrospective case-control study of health care personnel, an increased risk for HIV infection was associated with exposure to a relatively large volume of blood, as indicated by a deep injury with a device that was visibly contaminated with the patient's blood, or a procedure that involved a needle placed in a vein or artery. The risk was also increased if the exposure was to blood from patients with terminal illnesses, possibly reflecting the higher titer of HIV in late-stage AIDS.

Exposure prevention methods

Avoiding occupational exposures to blood is the primary way to prevent transmission of HBV, HCV and HIV, to workers in health care settings. Exposures occur through percutaneous injury (e.g., a needlestick or cut with a sharp object), as well as through contact between potentially infectious blood, tissues or other body fluids and mucous membranes of the eye, nose, mouth or nonintact skin (e.g., exposed skin that is chapped, abraded or shows signs of dermatitis).

Observational studies and surveys indicate that percutaneous injuries among general dentists and oral surgeons occur less frequently than among general and orthopedic surgeons and have decreased in frequency since the mid-1980s. This decline has been attributed to safer work practices, safer instrumentation or design, and continued dental care workers education. Percutaneous injuries among DHCP usually:

- Occur outside the patient's mouth, thereby posing less risk for recontact with patient tissues.
- Involve limited amounts of blood.
- Are caused by burs, syringe needles, laboratory knives and other sharp instruments.

Injuries among oral surgeons might occur more frequently during fracture reductions using wires. Experience, as measured by years in practice, does not appear to affect the risk of injury among general dentists or oral surgeons.

The majority of exposures in dentistry are preventable, and methods to reduce the risk of blood contacts have included use of standard precautions, use of devices with features engineered to prevent sharp injuries and modifications of work practices. These approaches might have contributed to the decrease in percutaneous injuries among dentists during recent years. However, needlesticks and other blood contacts continue to occur, which is a concern because percutaneous injuries pose the greatest risk of transmission.

Standard precautions include use of personal protective equipment (e.g., gloves, masks, protective eyewear or face shield, and gowns) intended to prevent skin and mucous membrane exposures. Other protective equipment (e.g., finger guards while suturing) might also reduce injuries during dental procedures.

Engineering controls are the primary method to reduce exposures to blood and OPIM from sharp instruments and needles. These controls are frequently technology-based and often incorporate safer designs of instruments and devices (e.g., self-sheathing anesthetic needles and dental units designed to shield burs in handpieces) to reduce percutaneous injuries.

Work-practice controls establish practices to protect dental workers whose responsibilities include handling, using, assembling or processing sharp devices (e.g., needles, scalers, laboratory utility knives, burs, explorers and endodontic files) or sharps disposal containers. Work-practice controls can include removing burs before disassembling the handpiece from the dental unit, restricting use of fingers in tissue retraction or palpation during suturing and administration of anesthesia and minimizing potentially uncontrolled movements of such instruments as scalers or laboratory knives.

As indicated, needles are a substantial source of percutaneous injury in dental practice, and engineering and work-practice controls for needle handling are of particular importance. In 2001, revisions to OSHA's blood-borne pathogens standard as mandated by the Needlestick Safety and Prevention Act of 2000 became effective. These revisions clarify the need for employers to consider safer needle devices as they become available and to involve employees directly responsible for patient care (e.g., dentists, hygienists and dental assistants) in identifying and choosing such devices. Safer versions of sharp devices used in hospital settings have become available (e.g., blunt suture needles, phlebotomy devices and butterfly needles), and their impact on reducing injuries has been documented. Aspirating anesthetic syringes that incorporate safety features have been developed for dental procedures, but the low injury rates in dentistry limit assessment of their effect on reducing injuries among dental care workers.

Work-practice controls for needles and other sharps include placing used disposable syringes and needles, scalpel blades and other sharp items in appropriate puncture-resistant containers located as close as feasible to where the items were used. In addition, used needles should never be recapped or otherwise manipulated by using both hands or any other technique that involves directing the point of a needle toward any part of the body. A one-handed scoop technique,

a mechanical device designed for holding the needle cap to facilitate one-handed recapping, or an engineered sharps injury protection device (e.g., needles with resheathing mechanisms) should be employed for recapping needles between uses and before disposal. Dental care workers should never bend or break needles before disposal because this practice requires unnecessary manipulation. Before attempting to remove needles from nondisposable aspirating syringes, they should recap them to prevent injuries. For procedures involving multiple injections with a single needle, the practitioner should recap the needle between injections by using a one-handed technique or use a device with a needle-resheathing mechanism. Passing a syringe with an unsheathed needle should be avoided because of the potential for injury.

Additional information for developing a safety program and for identifying and evaluating safer dental devices is available at:

- *http://www.cdc.gov/OralHealth/infectioncontrol/ forms.htm* (forms for screening and evaluating safer dental devices).
- *http://www.cdc.gov/niosh/topics/bbp* (state legislation on needlestick safety).

Post-exposure management and prophylaxis

Post-exposure management is an integral component of a complete program to prevent infection after an occupational exposure to blood. During dental procedures, saliva is predictably contaminated with blood. Even when blood is not visible, it can still be present in limited quantities and therefore is considered a potentially infectious material by OSHA. A qualified health care professional should evaluate any occupational exposure incident to blood or OPIM, including saliva, regardless of whether blood is visible, in dental settings.

Dental practices and laboratories should establish written, comprehensive programs that include hepatitis B vaccination and post-exposure management protocols that:

- Describe the types of contact with blood or OPIM that can place dental care workers at risk for infection.
- Describe procedures for promptly reporting and evaluating such exposures.
- Identify a health care professional who is qualified to provide counseling and perform all medical evaluations and procedures in accordance with current recommendations of the U. S. Public Health Service (PHS), including prophylaxis with chemotherapeutic drugs when indicated.

Dental workers, including students, who might reasonably be considered at risk for occupational exposure to blood or OPIM should be taught strategies to prevent contact with blood or OPIM and the principles of post-exposure management, including prophylaxis options, as part of their job orientation and training. Educational programs for dental workers and students should emphasize reporting all exposures to blood or OPIM as soon as possible, because certain interventions have to be initiated promptly to be effective.

Policies should be consistent with the practices and procedures for worker protection required by OSHA and with current Public Health Service recommendations for managing occupational exposures to blood.

After an occupational blood exposure, first aid should be administered as necessary. Puncture wounds and other injuries to the skin should be washed with soap and water; mucous membranes should be flushed with water. No evidence exists that using antiseptics for wound care or expressing fluid by squeezing the wound further reduces the risk of blood-borne pathogen transmission; however, use of antiseptics is not contraindicated. The application of caustic agents (e.g., bleach) or the injection of antiseptics or disinfectants into the wound is not recommended. Exposed workers should immediately report the exposure to the infection control coordinator or other designated person, who should initiate referral to the qualified health care professional and complete necessary reports.

Because multiple factors contribute to the risk of infection after an occupational exposure to blood, the following information should be included in the exposure report, recorded in the exposed person's confidential medical record and provided to the qualified health care professional:

- Date and time of exposure.
- Details of the procedure being performed, including where and how the exposure occurred and whether the exposure involved a sharp device, the type and brand of device, and how and when during its handling the exposure occurred.
- Details of the exposure, including its severity and the type and amount of fluid or material.
    - For a percutaneous injury, severity might be measured by the depth of the wound, gauge of the needle and whether fluid was injected.
    - For a skin or mucous membrane exposure, the estimated volume of material, duration of contact and the condition of the skin (e.g., chapped, abraded or intact) should be noted.
    - Details regarding whether the source material was known to contain HIV or other blood-borne pathogens.
    - If the source was infected with HIV, the stage of disease, history of antiretroviral therapy and viral load, if known.
- Details regarding the exposed person (e.g., hepatitis B vaccination and vaccine-response status).
- Details regarding counseling, post-exposure management and follow-up.

Each occupational exposure should be evaluated individually for its potential to transmit HBV, HCV and HIV, based on the following:

- The type and amount of body substance involved.
- The type of exposure (e.g., percutaneous injury, mucous membrane or nonintact skin exposure, or bites resulting in blood exposure to either person involved).
- The infection status of the source.
- The susceptibility of the exposed person.

All of these factors should be considered in assessing the risk for infection and the need for further follow-up (e.g. post-exposure prophylaxis, or PEP).

During 1990-1998, the Public Health Service published guidelines for PEP and other management of health care worker exposures to HBV, HCV or HIV. In 2001, these recommendations were updated and consolidated into one set of Public Health Service guidelines. The new guidelines reflect the availability of new anti-retroviral agents, new information regarding the use and safety of HIV PEP, and considerations regarding employing HIV prophylaxis when resistance of the source patient's virus to anti-retroviral agents is known or suspected. In addition, the 2001 guidelines provide guidance to clinicians and exposed workers regarding when to consider HIV prophylaxis and recommendations for treatment regimens.

Hand hygiene

Hand hygiene (e.g., hand-washing, hand antisepsis or surgical-hand antisepsis) substantially reduces potential pathogens on the hands and is considered the single most critical measure for reducing the risk of transmitting organisms to patients and health care personnel. Hospital-based studies have demonstrated that noncompliance with hand hygiene practices is associated with health care-associated infections and the spread of multiresistant organisms. Noncompliance also has been a major contributor to outbreaks. The prevalence of health care-

associated infections decreases as adherence of health care workers to recommended hand hygiene measures improves.

The microbial flora of the skin, first described in 1938, consist of transient and resident microorganisms. Transient flora, which colonize the superficial layers of the skin, are easier to remove by routine hand-washing. They are often acquired by workers during direct contact with patients or contaminated environmental surfaces; these organisms are most frequently associated with health care-associated infections. Resident flora attached to deeper layers of the skin are more resistant to removal and less likely to be associated with such infections.

The preferred method for hand hygiene depends on the type of procedure, the degree of contamination and the desired persistence of antimicrobial action on the skin (Table 2). For routine dental examinations and nonsurgical procedures, hand-washing and hand antisepsis is achieved by using either a plain or antimicrobial soap and water. If the hands are not visibly soiled, an alcohol-based hand rub is adequate.

The purpose of surgical hand antisepsis is to eliminate transient flora and reduce resident flora for the duration of a procedure to prevent introduction of organisms in the operative wound, if gloves become punctured or torn. Skin bacteria can rapidly multiply under surgical gloves if hands are washed with soap that is not antimicrobial. Thus, an antimicrobial soap or alcohol hand rub with persistent activity should be used before surgical procedures.

Agents used for surgical hand antisepsis should substantially reduce microorganisms on intact skin, contain a nonirritating antimicrobial preparation, have a broad spectrum of activity, be fast-acting and have a persistent effect. Persistence (i.e., extended antimicrobial activity that prevents or inhibits survival of microorganisms after the product is applied) is critical because microorganisms can colonize on hands in the moist environment underneath gloves.

Alcohol hand rubs are rapidly germicidal when applied to the skin, but should include such antiseptics as chlorhexidine, quaternary ammonium compounds, octenidine or triclosan to achieve persistent activity. Factors that can influence the effectiveness of the surgical hand antisepsis in addition to the choice of antiseptic agent include duration and technique of scrubbing, as well as condition of the hands, and techniques used for drying and gloving. CDC's 2002 guideline on hand hygiene in health care settings provides more complete information.

Selection of antiseptic agents

Selecting the most appropriate antiseptic agent for hand hygiene requires consideration of multiple factors. Essential performance characteristics of a product (e.g., the spectrum and persistence of activity and whether or not the agent is fast acting) should be determined before selecting a product. Delivery system, cost per use, reliable vendor support and supply are also considerations. Because worker acceptance is a major factor regarding compliance with recommended hand hygiene protocols, considering their needs is critical and should include possible chemical allergies, skin integrity after repeated use, compatibility with lotions used and offensive agent ingredients (e.g., scent). Discussing specific preparations or ingredients used for hand antisepsis is beyond the scope of this report. Health care workers should choose from commercially available health care worker hand washes when selecting agents for hand antisepsis or surgical hand antisepsis.

Storage and dispensing of hand-care products

Hand-washing products, including plain (i.e., non-antimicrobial) soap and antiseptic products, can become contaminated or support the growth of microorganisms. Liquid products should be stored in closed containers and dispensed from either disposable containers or containers that are washed and dried thoroughly before refilling. Soap should not be added to a partially empty dispenser, because this practice of topping off might lead to bacterial contamination. Store and dispense products according to manufacturers' directions.

Lotions

The primary defense against infection and transmission of pathogens is healthy, unbroken skin. Frequent hand-washing with soaps and antiseptic agents can cause chronic irritant contact dermatitis among workers. Damage to the skin changes skin flora, resulting in more frequent colonization by staphylococci and gram-negative bacteria. The potential of detergents to cause skin irritation varies considerably, but can be reduced by adding emollients. Lotions are often recommended to ease the dryness resulting from frequent hand-washing and to prevent dermatitis from glove use. However, petroleum-based lotion formulations can weaken latex gloves and increase permeability. For that reason, lotions that contain petroleum or other oil emollients should only be used at the end of the work day. Dental practitioners should obtain information from lotion manufacturers regarding interaction between lotions, gloves, dental materials and antimicrobial products.

Fingernails and artificial nails

Although the relationship between fingernail length and wound infection is unknown, keeping nails short is considered key because the majority of flora on the hands are found under and around the fingernails. Fingernails should be short enough to allow dental workers to thoroughly clean underneath them and prevent glove tears. Sharp nail edges or broken nails are also likely to increase glove failure. Long artificial or natural nails can make donning gloves more difficult and can cause gloves to tear more readily.

Hand carriage of gram-negative organisms has been determined to be greater among wearers of artificial nails than among nonwearers, both before and after hand-washing. In addition, artificial fingernails or extenders have been epidemiologically implicated in multiple outbreaks involving fungal and bacterial infections in hospital intensive-care units and operating rooms. Freshly applied nail polish on natural nails does not increase the microbial load from periungual skin if fingernails are short; however, chipped nail polish can harbor added bacteria.

Jewelry

Studies have demonstrated that skin underneath rings is more heavily colonized than comparable areas of skin on fingers without rings. In a study of intensive-care nurses, multivariable analysis determined rings were the only substantial risk factor for carriage of gram-negative bacilli and Staphylococcus aureus, and the concentration of organisms correlated with the number of rings worn. However, two other studies demonstrated that mean bacterial colony counts on hands after hand-washing were similar among persons wearing rings and those not wearing rings. Whether wearing rings increases the likelihood of transmitting a pathogen is unknown; further studies are needed to

establish whether rings result in higher transmission of pathogens in health care settings. However, rings and decorative nail jewelry can make donning gloves more difficult and cause gloves to tear more readily. Thus, jewelry should not interfere with glove use (e.g., impair ability to wear the correct-sized glove or alter glove integrity).

Personal protective equipment

PPE is designed to protect the skin and the mucous membranes of the eyes, nose and mouth of dental care workers from exposure to blood or OPIM. Use of rotary dental and surgical instruments (e.g., handpieces or ultrasonic scalers) and air-water syringes creates a visible spray that contains primarily large particle droplets of water, saliva, blood, microorganisms and other debris. This spatter travels only a short distance and settles out quickly, landing on the floor, nearby operatory surfaces, dental care workers or the patient. The spray also might contain certain aerosols (i.e., particles of respirable size, less than 10 μm). Aerosols can remain airborne for extended periods and can be inhaled. However, they should not be confused with the large-particle spatter that makes up the bulk of the spray from handpieces and ultrasonic scalers. Appropriate work practices, including use of dental dams and high-velocity air evacuation, should minimize dissemination of droplets, spatter and aerosols.

Protective clothing

Protective clothing and equipment (e.g., gowns, lab coats, gloves, masks and protective eyewear or face shield) should be worn to prevent contamination of street clothing and to protect the skin of dental workers from exposures to blood and body substances. Uniforms/scrubs are not considered personal protective equipment when anticipating spatter of blood or body fluids. OSHA blood-borne pathogens standard requires sleeves to be long enough to protect the forearms when the gown is worn as personal protective equipment (i.e., when spatter and spray of blood, saliva or OPIM to the forearms is anticipated). Dental personnel should change protective clothing when it becomes visibly soiled and as soon as feasible if penetrated by blood or other potentially infectious fluids. All protective clothing should be removed before leaving the work area.

Gloves and gloving

DHCP wear gloves to prevent contamination of their hands when touching mucous membranes, blood, saliva or OPIM, and also to reduce the likelihood that microorganisms present on the workers' hands will be transmitted to patients during surgical or other patient-care procedures. Medical gloves, both patient examination and surgeon's gloves, are manufactured as single-use disposable items that should be used for only one patient, then discarded. Gloves should be changed between patients and when torn or punctured.

Wearing gloves does not eliminate the need for hand-washing. Hand hygiene should be performed immediately before donning gloves. Gloves can have small, unapparent defects or can be torn during use, and hands can become contaminated during glove removal. These circumstances increase the risk of operative wound contamination and exposure of the worker's hands to microorganisms from patients. FDA regulates the medical glove industry, which includes gloves marketed as sterile surgeon's and sterile or nonsterile patient examination gloves. General-purpose utility gloves are also used in dental health care settings but are not regulated by FDA because they are not promoted for medical use. More rigorous standards are applied to surgeon's gloves than to examination gloves. FDA has identified acceptable quality levels (e.g., maximum defects allowed) for glove manufacturers, but even intact gloves eventually fail with exposure to mechanical (e.g., sharps, fingernails or jewelry) and chemical (e.g., dimethy-acrylates) hazards and over time. These variables can be controlled, ultimately optimizing glove performance, by:

- Maintaining short fingernails.
- Minimizing or eliminating hand jewelry.
- Using engineering and work-practice controls to avoid injuries with sharps.

Sterile surgeon's gloves and double-gloving during oral surgical procedures

Certain limited studies have determined no difference in postoperative infection rates after routine tooth extractions when surgeons wore either sterile or nonsterile gloves. However, wearing sterile surgeon's gloves during surgical procedures is supported by a strong theoretical rationale. Sterile gloves minimize transmission of microorganisms from the hands of surgical dental care personnel to patients and prevent contamination of the hands of the workers with the patient's blood and body fluids. In addition, sterile surgeon's gloves are more rigorously regulated by FDA and therefore, might provide an increased level of protection for the provider if exposure to blood is likely.

Although the effectiveness of wearing two pairs of gloves in preventing disease transmission has not been demonstrated, the majority of studies among health care workers have demonstrated a lower frequency of inner glove perforation and visible blood on the surgeon's hands when double gloves are worn. In one study evaluating double gloves during oral surgical and dental hygiene procedures, the perforation of outer latex gloves was greater during longer procedures (i.e., more than 45 minutes), with the highest rate (10 percent) of perforation occurring during oral surgery procedures. Based on these studies, double-gloving might provide additional protection from occupational blood contact. Double-gloving does not appear to substantially reduce either manual dexterity or tactile sensitivity. Additional protection might also be provided by specialty products (e.g., orthopedic surgical gloves and glove liners).

Contact dermatitis and latex hypersensitivity

Occupationally related contact dermatitis can develop from frequent and repeated use of hand hygiene products, exposure to chemicals, and glove use. Contact dermatitis is classified as either irritant or allergic. Irritant contact dermatitis is common, nonallergic and develops as dry, itchy, irritated areas on the skin around the area of contact. By comparison, allergic contact dermatitis (type IV hypersensitivity) can result from exposure to accelerators and other chemicals used in the manufacture of rubber gloves (e.g., natural rubber latex, nitrile and neoprene), as well as from other chemicals found in the dental practice setting (e.g., methacrylates and glutaraldehyde). Allergic contact dermatitis often manifests as a rash beginning hours after contact and, similar to irritant dermatitis, is usually confined to the area of contact.

Latex allergy (type I hypersensitivity to latex proteins) can be a more serious systemic allergic reaction, usually beginning within minutes of exposure, but sometimes occurring hours later and producing varied symptoms. More common reactions include runny nose, sneezing, itchy eyes, scratchy throat, hives and itchy, burning skin sensations.

More severe symptoms include asthma marked by difficult breathing, coughing spells and wheezing; cardiovascular and gastrointestinal ailments; and in rare cases, anaphylaxis and death. The American Dental Association (ADA) began investigating the prevalence of type I latex hypersensitivity among dental care personnel at the ADA annual meeting in 1994. In 1994 and 1995, approximately 2,000 dentists, hygienists and assistants volunteered for skin-prick testing. Data demonstrated that 6.2 percent of those tested were positive for type I latex hypersensitivity. Data from the subsequent five years of this ongoing cross-sectional study indicated a decline in prevalence from 8.5 percent to 4.3 percent. This downward trend is similar to that reported by other studies and might be related to use of latex gloves with lower allergen content.

Natural rubber latex proteins responsible for latex allergy are attached to glove powder. When powdered latex gloves are worn, more latex protein reaches the skin. In addition, when powdered latex gloves are donned or removed, latex protein/powder particles become aerosolized and can be inhaled, contacting mucous membranes. As a result, allergic patients and dental workers can experience cutaneous, respiratory and conjunctival symptoms related to latex protein exposure. Dental care workers can become sensitized to latex protein with repeated exposure. Work areas where only powder-free, low-allergen latex gloves are used demonstrate low or undetectable amounts of latex allergy-causing proteins and fewer symptoms among workers related to natural rubber latex allergy. Because of the role of glove powder in exposure to latex protein, NIOSH recommends that if latex gloves are chosen, workers should be provided with reduced protein, powder-free gloves. Nonlatex (e.g., nitrile or vinyl) powder-free and low-protein gloves are also available. Although rare, potentially life-threatening anaphylactic reactions to latex can occur, and dental practices should be appropriately equipped and have procedures in place to respond to such emergencies.

Dental care personnel and dental patients with latex allergy should not have direct contact with latex-containing materials and should be in a latex-safe environment with all latex-containing products removed from their vicinity. Dental patients with histories of latex allergy can be at risk from dental products (e.g., prophylaxis cups, rubber dams, orthodontic elastics and medication vials). Any latex-containing devices that cannot be removed from the treatment environment should be adequately covered or isolated. Persons might also be allergic to chemicals used in the manufacture of natural rubber latex and synthetic rubber gloves, as well as metals, plastics or other materials used in dental care.

Taking thorough health histories for both patients and dental workers, followed by avoidance of contact with potential allergens, can minimize the possibility of adverse reactions. Certain common predisposing conditions for latex allergy include previous history of allergies, a history of spina bifida, urogenital anomalies or allergies to avocados, kiwis, nuts or bananas. The following precautions should be considered to ensure safe treatment for patients who have possible or documented latex allergy:

- Be aware that latent allergens in the ambient air can cause respiratory or anaphylactic symptoms among persons with latex hypersensitivity. Patients with latex allergy can be scheduled for the first appointment of the day to minimize their inadvertent exposure to airborne latex particles.
- Communicate with other dental workers regarding patients with latex allergy (e.g., by oral instructions, written protocols and posted signage) to prevent them from bringing latex-containing materials into the treatment area.
- Frequently clean all working areas contaminated with latex powder or dust.
- Have emergency treatment kits with latex-free products available at all times.
- If latex-related complications occur during or after a procedure, manage the reaction and seek emergency assistance as indicated. Follow current medical emergency response recommendations for management of anaphylaxis.

Sterilization and disinfection of patient-care items

Patient-care items (dental instruments, devices and equipment) are categorized as critical, semicritical or noncritical, depending on the potential risk for infection associated with their intended use (Table 1). Critical items used to penetrate soft tissue or bone have the greatest risk of transmitting infection and should be sterilized by heat. Semicritical items touch mucous membranes or nonintact skin and have a lower risk of transmission; because the majority of semicritical items in dentistry are heat-tolerant, they also should be sterilized by using heat. If a semicritical item is heat-sensitive, it should, at a minimum, be processed with high-level disinfection.

Noncritical patient-care items pose the least risk of transmission of infection, contacting only intact skin, which can serve as an effective barrier to microorganisms. In the majority of cases, cleaning, or if visibly soiled, cleaning followed by disinfection with an EPA-registered hospital disinfectant is adequate. When the item is visibly contaminated with blood or OPIM, an EPA-registered hospital disinfectant with a tuberculocidal claim (i.e., intermediate-level disinfectant) should be used. Cleaning or disinfection of certain noncritical patient-care items can be difficult or damage the surfaces; therefore, use of disposable barrier protection of these surfaces might be a preferred alternative.

FDA-cleared sterilant/high-level disinfectants and EPA registered disinfectants must have clear label claims for intended use, and manufacturer instructions for use must be followed.

TABLE 1. Infection control categories of patient-care instruments

Category	Definition	Dental instrument/ item
Critical	Penetrates soft tissue, contacts bone, enters into or contacts the bloodstream or other normally sterile tissue.	Surgical instruments, periodontal scalers, scalpel blades, surgical dental burs.
Semi-critical	Contacts mucous membranes or nonintact skin; will not penetrate soft tissue, contact bone, enter into or contact the bloodstream or other normally sterile tissue.	Dental mouth mirror, amalgam condenser, reusable dental impression trays, dental handpieces*.
Non-critical	Contacts intact skin.	Radiograph head/cone, blood pressure cuff, facebow, pulse oximeter

* Although dental handpieces are considered a semicritical item, they should always be heat-sterilized between uses and not high-level disinfected. See Dental handpieces and other devices attached to air or waterlines for detailed information.

Three levels of disinfection, high, intermediate and low, are used for patient-care devices that do not require sterility; and two levels, intermediate and low, for environmental surfaces. The intended use of the patient-care item should determine the recommended level of disinfection. Dental practices should follow the product manufacturer's directions regarding concentrations and exposure time for disinfectant activity relative to the surface to be disinfected.

Transporting and processing contaminated critical and semi-critical patient-care items

Dental workers can be exposed to microorganisms on contaminated instruments and devices through percutaneous injury, contact with nonintact skin on the hands or contact with mucous membranes of the eyes, nose or mouth. Contaminated instruments should be handled carefully to prevent exposure to sharp instruments that can cause a percutaneous injury. Instruments should be placed in an appropriate container at the point of use to prevent percutaneous injuries during transport to the instrument processing area.

Instrument processing requires multiple steps to achieve sterilization or high-level disinfection. Sterilization is a complex process requiring specialized equipment, adequate space, qualified dental workers who are provided with ongoing training, and regular monitoring for quality assurance. Correct cleaning, packaging, sterilizer-loading procedures, sterilization methods or high-level disinfection methods should be followed to ensure that an instrument is adequately processed and safe for reuse on patients.

Instrument processing area

Dental workers should process all instruments in a designated central processing area to more easily control quality and ensure safety. The central processing area should be divided into sections for:

- Receiving, cleaning and decontamination.
- Preparation and packaging.
- Sterilization.
- Storage.

Ideally, walls or partitions should separate the sections to control traffic flow and contain contaminants generated during processing. When physical separation of these sections cannot be achieved, adequate spatial separation might be satisfactory if the dental workers who process instruments are trained in work practices to prevent contamination of clean areas. Space should be adequate for the volume of work anticipated and the items to be stored.

Receiving, cleaning and decontamination

Reusable instruments, supplies and equipment should be received, sorted, cleaned and decontaminated in one section of the processing area. Cleaning should precede all disinfection and sterilization processes; it should involve removal of debris, as well as organic and inorganic contamination. Removal of debris and contamination is achieved either by scrubbing with a surfactant, detergent and water, or by an automated process (e.g., ultrasonic cleaner or washer-disinfector) using chemical agents. If visible debris, whether inorganic or organic matter, is not removed, it will interfere with microbial inactivation and can compromise the disinfection or sterilization process. After cleaning, instruments should be rinsed with water to remove chemical or detergent residue. Splashing should be minimized during cleaning and rinsing. Before final disinfection or sterilization, instruments should be handled as though contaminated.

Considerations in selecting cleaning methods and equipment include:

- Efficacy of the method, process and equipment.
- Compatibility with items to be cleaned.
- Occupational health and exposure risks.

Use of automated cleaning equipment (e.g., ultrasonic cleaner or washer-disinfector) does not require presoaking or scrubbing of instruments and can increase productivity, improve cleaning effectiveness and decrease worker exposure to blood and body fluids. Thus, using automated equipment can be safer and more efficient than manually cleaning contaminated instruments.

If manual cleaning is not performed immediately, placing instruments in a puncture-resistant container and soaking them with detergent, a disinfectant/detergent or an enzymatic cleaner will prevent drying of patient material and make cleaning easier and less time-consuming. Use of a liquid chemical sterilant/high-level disinfectant (e.g., glutaraldehyde) as a holding solution is not recommended.

Using work-practice controls (e.g., a long-handled brush) to keep the scrubbing hand away from sharp instruments is recommended. To avoid injury from sharp instruments, workers should wear puncture-resistant, heavy-duty utility gloves when handling or manually cleaning contaminated instruments and devices. Employees should not reach into trays or containers holding sharp instruments that cannot be seen (e.g., sinks filled with soapy water in which sharp instruments have been placed). Work-practice controls should include use of a strainer-type basket to hold instruments and forceps to remove the items. Because splashing is likely to occur, a mask, protective eyewear or face shield, and gown or jacket should be worn.

Preparation and packaging

In another section of the processing area, cleaned instruments and other dental supplies should be inspected, assembled into sets or trays, and wrapped, packaged or placed into container systems for sterilization. Hinged instruments should be processed open and unlocked. An internal chemical indicator should be placed in every package. In addition, an external chemical indicator (e.g., chemical indicator tape) should be used when the internal indicator cannot be seen from outside the package. For unwrapped loads, at a minimum, an internal chemical indicator should be placed in the tray or cassette with items to be sterilized (see Sterilization of unwrapped instruments). Dental practices should refer to the manufacturer's instructions regarding use and correct placement of chemical indicators (see Sterilization monitoring). Critical and semicritical instruments that will be stored should be wrapped or placed in containers (e.g., cassettes or organizing trays) designed to maintain sterility during storage.

Packaging materials (e.g., wraps or container systems) allow penetration of the sterilization agent and maintain sterility of the processed item after sterilization. Materials for maintaining sterility of instruments during transport and storage include wrapped perforated instrument cassettes, peel pouches of plastic or paper and sterilization wraps (woven and nonwoven). Packaging materials should be designed for the type of sterilization process being used.

Sterilization

The sterilization section of the processing area should include the sterilizers and related supplies, with adequate space for loading, unloading and cool down. The area can also include incubators for analyzing spore tests and enclosed storage for sterile items and disposable (single-use) items. Manufacturer and local building

code specifications will determine placement and room ventilation requirements.

Sterilization procedures – Heat-tolerant dental instruments usually are sterilized by:

- Steam under pressure (autoclaving).
- Dry heat.
- Unsaturated chemical vapor.

All sterilization should be performed by using medical sterilization equipment cleared by FDA. The sterilization times, temperatures and other operating parameters recommended by the manufacturer of the equipment used, as well as instructions for correct use of containers, wraps and chemical or biological indicators, should always be followed.

Items to be sterilized should be arranged to permit free circulation of the sterilizing agent (e.g., steam, chemical vapor or dry heat); manufacturer's instructions for loading the sterilizer should be followed. Instrument packs should be allowed to dry inside the sterilizer chamber before removing and handling. Packs should not be touched until they are cool and dry because hot packs act as wicks, absorbing moisture, and hence, bacteria from hands. The ability of equipment to attain physical parameters required to achieve sterilization should be monitored by mechanical, chemical and biological indicators. Sterilizers vary in their types of indicators and their ability to provide readings on the mechanical or physical parameters of the sterilization process (e.g., time, temperature and pressure). Consult with the sterilizer manufacturer regarding selection and use of indicators.

Steam sterilization – Among sterilization methods, steam sterilization, which is dependable and economical, is the most widely used for wrapped and unwrapped critical and semicritical items that are not sensitive to heat and moisture. Steam sterilization requires exposure of each item to direct steam contact at a required temperature and pressure for a specified time needed to kill microorganisms. Two basic types of steam sterilizers are the gravity displacement and the high-speed prevacuum sterilizer.

The majority of tabletop sterilizers used in a dental practice are gravity displacement sterilizers, although prevacuum sterilizers are becoming more widely available. In gravity displacement sterilizers, steam is admitted through steam lines, a steam generator or self-generation of steam within the chamber.

Unsaturated air is forced out of the chamber through a vent in the chamber wall. Trapping of air is a concern when using saturated steam under gravity displacement; errors in packaging items or overloading the sterilizer chamber can result in cool air pockets and items not being sterilized.

Prevacuum sterilizers are fitted with a pump to create a vacuum in the chamber and ensure air removal from the sterilizing chamber before the chamber is pressurized with steam. Relative to gravity displacement, this procedure allows faster and more positive steam penetration throughout the entire load. Prevacuum sterilizers should be tested periodically for adequate air removal, as recommended by the manufacturer. Air not removed from the chamber will interfere with steam contact. If a sterilizer fails the air removal test, it should not be used until inspected by sterilizer maintenance personnel and it passes the test. Manufacturer's instructions, with specific details regarding operation and user maintenance information, should be followed.

Unsaturated chemical-vapor sterilization – Unsaturated chemical-vapor sterilization involves heating a chemical solution of primarily alcohol with 0.23 percent formaldehyde in a closed pressurized chamber. Unsaturated chemical vapor sterilization of carbon steel instruments (e.g., dental burs) causes less corrosion than steam sterilization because of the low level of water present during the cycle. Instruments should be dry before sterilizing. State and local authorities should be consulted for hazardous waste disposal requirements for the sterilizing solution.

Dry-heat sterilization – Dry heat is used to sterilize materials that might be damaged by moist heat (e.g., burs and certain orthodontic instruments). Although dry heat has the advantages of low operating cost and being noncorrosive, it is a prolonged process and the high temperatures required are not suitable for certain patient-care items and devices. Dry-heat sterilizers used in dentistry include static-air and forced-air types:

- The static-air type is commonly called an oven-type sterilizer. Heating coils in the bottom or sides of the unit cause hot air to rise inside the chamber through natural convection.
- The forced-air type is also known as a rapid heat-transfer sterilizer. Heated air is circulated throughout the chamber at a high velocity, permitting more rapid transfer of energy from the air to the instruments, thereby reducing the time needed for sterilization.

Sterilization of unwrapped instruments – An unwrapped cycle (sometimes called flash sterilization) is a method for sterilizing unwrapped patient-care items for immediate use. The time required for unwrapped sterilization cycles depends on the type of sterilizer and the type of item (i.e., porous or nonporous) to be sterilized. The unwrapped cycle in tabletop sterilizers is preprogrammed by the manufacturer to a specific time and temperature setting and can include a drying phase at the end to produce a dry instrument with much of the heat dissipated. If the drying phase requirements are unclear, the operation manual or manufacturer of the sterilizer should be consulted. If the unwrapped sterilization cycle in a steam sterilizer does not include a drying phase or has only a minimal drying phase, items retrieved from the sterilizer will be hot and wet, making aseptic transport to the point of use more difficult. For dry-heat and chemical-vapor sterilizers, a drying phase is not required.

Unwrapped sterilization should be used only under certain conditions:

- Thorough cleaning and drying of instruments precedes the unwrapped sterilization cycle.
- Mechanical monitors are checked and chemical indicators used for each cycle.
- Care is taken to avoid thermal injury to dental care personnel or patients.
- Items are transported aseptically to the point of use to maintain sterility.

Because all implantable devices should be quarantined after sterilization until the results of biological monitoring are known, unwrapped or flash sterilization of implantable items is not recommended.

Critical instruments sterilized unwrapped should be transferred immediately by using aseptic technique, from the sterilizer to the actual point of use. Critical instruments should not be stored unwrapped. Semicritical instruments that are sterilized unwrapped on a tray or in a container system should be used immediately or within a short time. When sterile items are open to the air, they will eventually become contaminated. Storage, even temporary, of unwrapped semicritical instruments is discouraged because it permits exposure to dust, airborne organisms and other unnecessary contamination before use on a patient. A carefully written protocol for minimizing the risk of contaminating unwrapped instruments should be prepared and followed.

Other sterilization methods – Heat-sensitive critical and semicritical instruments and devices can be sterilized by immersing them in liquid chemical germicides registered by FDA as sterilants. When using a liquid chemical germicide for sterilization, certain post-sterilization procedures are essential. Items need to be:

- Rinsed with sterile water after removal to remove toxic or irritating residues.
- Handled using sterile gloves and dried with sterile towels.
- Delivered to the point of use in an aseptic manner.

If an instrument is stored before use, the instrument should not be considered sterile and should be sterilized again just before use. In addition, the sterilization process with liquid chemical sterilants cannot be verified with biological indicators.

Because of these limitations and because liquid chemical sterilants can require approximately 12 hours of complete immersion, they are almost never used to sterilize instruments. Rather, these chemicals are more often used for high-level disinfection. Shorter immersion times (12-90 minutes) are used to achieve high-level disinfection of semicritical instruments or items. These powerful, sporicidal chemicals (e.g., glutaraldehyde, peracetic acid and hydrogen peroxide) are highly toxic. Manufacturer instructions (e.g., regarding dilution, immersion time and temperature) and safety precautions for using chemical sterilants/high-level disinfectants must be followed precisely.

These chemicals should not be used for applications other than those indicated in their label instructions. Misapplications include use as an environmental surface disinfectant or instrument-holding solution.

When using appropriate precautions (e.g., closed containers to limit vapor release, chemically resistant gloves and aprons, goggles and face shields), glutaraldehyde-based products can be used without tissue irritation or adverse health effects. However, dermatologic, eye irritation, respiratory effects and skin sensitization have been reported. Because of their lack of chemical resistance to glutaraldehydes, medical gloves are not an effective barrier. Other factors might apply (e.g., room exhaust ventilation or 10 air exchanges/hour) to ensure workers' safety. For all of these reasons, using heat-sensitive semicritical items that must be processed with liquid chemical germicides is discouraged; heat-tolerant or disposable alternatives are available for the majority of such items.

Low-temperature sterilization with ethylene oxide gas (ETO) has been used extensively in larger health care facilities. Its primary advantage is the ability to sterilize heat- and moisture-sensitive patient-care items with reduced deleterious effects. However, extended sterilization times of 10-48 hours and potential hazards to patients and workers requiring stringent health and safety requirements make this method impractical for private-practice settings. Handpieces cannot be effectively sterilized with this method because of decreased penetration of ETO gas flow through a small lumen.

Other types of low-temperature sterilization (e.g., hydrogen peroxide gas plasma) exist but are not yet practical for dental offices. Bead sterilizers have been used in dentistry to sterilize small metallic instruments (e.g., endodontic files). FDA has determined that a risk of infection exists with these devices because of their potential failure to sterilize dental instruments and has required their commercial distribution cease unless the manufacturer files a premarket approval application. If a bead sterilizer is employed, DHCP assume the risk of employing a dental device FDA has deemed neither safe nor effective.

Sterilization monitoring – Monitoring of sterilization procedures should include a combination of process parameters, including mechanical, chemical and biological. These parameters evaluate both the sterilizing conditions and the procedure's effectiveness.

Mechanical techniques for monitoring sterilization include assessing cycle time, temperature and pressure by observing the gauges or displays on the sterilizer and noting these parameters for each load. Some tabletop sterilizers have recording devices that print out these parameters. Correct readings do not ensure sterilization, but incorrect readings can be the first indication of a problem with the sterilization cycle.

Chemical indicators, internal and external, use sensitive chemicals to assess physical conditions (e.g., time and temperature) during the sterilization process. Although chemical indicators do not prove sterilization has been achieved, they allow detection of certain equipment malfunctions, and they can help identify procedural errors. External indicators applied to the outside of a package (e.g., chemical indicator tape or special markings) change color rapidly when a specific parameter is reached, and they verify that the package has been exposed to the sterilization process. Internal chemical indicators should be used inside each package to ensure the sterilizing agent has penetrated the packaging material and actually reached the instruments inside. A single-parameter internal chemical indicator provides information regarding only one sterilization parameter (e.g., time or temperature). Multiparameter internal chemical indicators are designed to react to more than two parameters (e.g., time and temperature; or time, temperature and the presence of steam) and can provide a more reliable indication that sterilization conditions have been met. Multiparameter internal indicators are available only for steam sterilizers (i.e., autoclaves).

Because chemical indicator test results are received when the sterilization cycle is complete, they can provide an early indication of a problem and where in the process the problem might exist. If either mechanical indicators or internal or external chemical indicators indicate inadequate processing, items in the load should not be used until reprocessed.

Biological indicators (BIs) (i.e., spore tests) are the most accepted method for monitoring the sterilization process because they assess it directly by killing known highly resistant microorganisms (e.g., Geobacillus or Bacillus species), rather than merely testing the physical and chemical conditions necessary for sterilization. Because spores used in BIs are more resistant and present in greater numbers than the common microbial contaminants found on patient-care equipment, an inactivated BI indicates other potential pathogens in the load have been killed.

Correct functioning of sterilization cycles should be verified for each sterilizer by the periodic use (at least weekly) of BIs. Every load containing implantable devices should be monitored with such indicators, and the items quarantined until BI results are known. However, in an emergency, placing implantable items in quarantine until spore tests are known to be negative might be impossible.

Manufacturer's directions should determine the placement and location of BI in the sterilizer. A control BI, from the same lot as the test indicator and not processed through the sterilizer, should be incubated with the test BI; the control BI should yield positive results for bacterial growth.

In-office biological monitoring is available; mail-in sterilization monitoring services (e.g., from private companies or dental schools) can also be used to test both the BI and the control. Although some dental care personnel have expressed concern that delays caused by mailing specimens might cause false negatives, studies have determined that mail delays have no substantial effect on final test results.

Procedures to follow in the event of a positive spore test have been developed. If the mechanical (e.g., time, temperature and pressure) and chemical (i.e., internal or external) indicators demonstrate that the sterilizer is functioning correctly, a single positive spore test probably does not indicate sterilizer malfunction. Items other than implantable devices do not necessarily need to be recalled; however, the spore test should be repeated immediately after correctly loading the sterilizer and using the same cycle that produced the failure. The sterilizer should be removed from service, and all records reviewed of chemical and mechanical monitoring since the last negative BI test.

Also, sterilizer operating procedures should be reviewed, including packaging, loading and spore testing, with all persons who work with the sterilizer to determine whether operator error could be responsible. Overloading, failure to provide adequate package separation and incorrect or excessive packaging material are all common reasons for a positive BI in the absence of mechanical failure of the sterilizer unit. A second monitored sterilizer in the office can be used, or a loaner from a sales or repair company obtained, to minimize office disruption while waiting for the repeat BI.

If the repeat test is negative and chemical and mechanical monitoring indicates adequate processing, the sterilizer can be put back into service. If the repeat BI test is positive, and packaging, loading and operating procedures have been confirmed as performing correctly, the sterilizer should remain out of service until it has been inspected, repaired and rechallenged with BI tests in three consecutive empty chamber sterilization cycles. When possible, items from suspect loads dating back to the last negative BI should be recalled, rewrapped and resterilized.

A more conservative approach has been recommended in which any positive spore test is assumed to represent sterilizer malfunction and requires that all materials processed in that sterilizer, dating from the sterilization cycle having the last negative biologic indicator to the next cycle indicating satisfactory biologic indicator results, should be considered nonsterile and retrieved, if possible, and reprocessed or held in quarantine until the results of the repeat BI are known. This approach is considered conservative because the margin of safety in steam sterilization is sufficient enough that infection risk associated with items in a load indicating spore growth is minimal, particularly if the item was properly cleaned and the temperature was achieved (e.g., as demonstrated by acceptable chemical indicator or temperature chart). Published studies are not available that document disease transmission through a nonretrieved surgical instrument after a steam sterilization cycle with a positive biological indicator. This more conservative approach should always be used for sterilization methods other than steam (e.g., dry heat, unsaturated chemical vapor, ETO or hydrogen peroxide gas plasma).

Results of biological monitoring should be recorded and sterilization monitoring records (i.e., mechanical, chemical and biological) retained long enough to comply with state and local regulations. Such records are a component of an overall dental infection control program (See program evaluation).

Storage of sterilized items and clean dental supplies

The storage area should contain enclosed storage for sterile items and disposable (single-use) items. Storage practices for wrapped sterilized instruments can be either date- or event-related. Packages containing sterile supplies should be inspected before use to verify barrier integrity and dryness.

Although some health care facilities continue to date every sterilized package and use shelf-life practices, other facilities have switched to event-related practices. This approach recognizes that the product should remain sterile indefinitely unless an event causes it to become contaminated (e.g., torn or wet packaging). Even for event-related packaging, minimally, the date of sterilization should be placed on the package, and if multiple sterilizers are used in the facility, the sterilizer used should be indicated on the outside of the packaging material to facilitate the retrieval of processed items in the event of a sterilization failure. If packaging is compromised, the instruments should be recleaned, packaged in new wrap and sterilized again.

Clean supplies and instruments should be stored in closed or covered cabinets, if possible. Dental supplies and instruments should not be stored under sinks or in other locations where they might become wet.

Environmental infection control

In the dental operatory, environmental surfaces (i.e., a surface or equipment that does not contact patients directly) can become contaminated during patient care. Certain surfaces, especially ones touched frequently (e.g., light handles, unit switches and drawer knobs) can serve as reservoirs of microbial contamination, although they have not been associated directly with transmission of infection to either dental workers or patients. Transfer of microorganisms from contaminated environmental surfaces to patients occurs primarily through dental care personnel hand contact. When these surfaces are touched, microbial agents can be transferred to instruments, other environmental surfaces or to the nose, mouth or eyes of workers or patients. Although hand hygiene is key to minimizing this transferal, barrier protection or cleaning and disinfecting of environmental surfaces also protects against health care-associated infections.

Environmental surfaces can be divided into clinical contact surfaces and housekeeping surfaces. Because housekeeping surfaces (e.g., floors, walls and sinks) have limited risk of disease transmission, they can be decontaminated with less rigorous methods than those used on dental patient-care items and clinical contact surfaces. Strategies for cleaning and disinfecting surfaces in patient-care areas should consider the following:

- Potential for direct patient contact.
- Degree and frequency of hand contact.
- Potential contamination of the surface with body substances or environmental sources of microorganisms (e.g., soil, dust or water).

Cleaning is the necessary first step of any disinfection process. Cleaning is a form of decontamination that renders the environmental surface safe by removing organic matter, salts and visible soils, all of which interfere with microbial inactivation. The physical action of scrubbing with detergents and surfactants and rinsing with water removes substantial numbers of microorganisms. If a surface is not cleaned first, the success of the disinfection process can be compromised. Removal of all visible blood and inorganic and organic matter can be as critical as the germicidal activity of the disinfecting agent. When a surface cannot be cleaned adequately, it should be protected with barriers.

Clinical contact surfaces

Clinical contact surfaces can be directly contaminated from patient materials by direct spray or spatter generated either during dental procedures or by contact with dental care personnel's gloved hands. These surfaces can subsequently contaminate other instruments, devices, hands or gloves. Examples of such surfaces include:

- Light handles.
- Switches.
- Dental radiograph equipment.
- Dental chair-side computers.
- Reusable containers of dental materials.
- Drawer handles.
- Faucet handles.
- Countertops.
- Pens.
- Telephones.
- Doorknobs.

Barrier protection of surfaces and equipment can prevent contamination of clinical contact surfaces, but is particularly effective for those that are difficult to clean. Barriers include clear plastic wrap, bags, sheets, tubing and plastic-backed paper or other materials impervious to moisture. Because such coverings can become contaminated, they should be removed and discarded between patients while dental workers are still gloved. After removing the barrier, examine the surface to make sure it did not become soiled inadvertently. The surface needs to be cleaned and disinfected only if contamination is evident. Otherwise, after removing gloves and performing hand hygiene, dental workers should place clean barriers on these surfaces before the next patient. If barriers are not used,

surfaces should be cleaned and disinfected between patients by using an EPA-registered hospital disinfectant with an HIV, HBV claim (i.e., low-level disinfectant) or a tuberculocidal claim (i.e., intermediate-level disinfectant). Intermediate-level disinfectant should be used when the surface is visibly contaminated with blood or OPIM. Also, general cleaning and disinfection are recommended for clinical contact surfaces, dental unit surfaces and countertops at the end of daily work activities and are required if surfaces have become contaminated since their last cleaning. To facilitate daily cleaning, treatment areas should be kept free of unnecessary equipment and supplies.

Manufacturers of dental devices and equipment should provide information regarding material compatibility with liquid chemical germicides, whether equipment can be safely immersed for cleaning, and how it should be decontaminated if service is required. Because of the risks associated with exposure to chemical disinfectants and contaminated surfaces, dental workers who perform environmental cleaning and disinfection should wear gloves and other protective equipment to prevent occupational exposure to infectious agents and hazardous chemicals. Chemical- and puncture-resistant utility gloves offer more protection than patient examination gloves when using hazardous chemicals.

Housekeeping surfaces

Evidence does not support that housekeeping surfaces (e.g., floors, walls and sinks) pose a risk for disease transmission in dental health care settings. Actual physical removal of microorganisms and soil by wiping or scrubbing is probably as critical, if not more so, than any antimicrobial effect provided by the agent used. The majority of housekeeping surfaces need to be cleaned only with a detergent and water or an EPA-registered hospital disinfectant/detergent, depending on the nature of the surface and the type and degree of contamination. Schedules and methods vary according to the area (e.g., dental operatory, laboratory, bathrooms or reception rooms), surface and amount and type of contamination.

Floors should be cleaned regularly, and spills should be cleaned up promptly. An EPA-registered hospital disinfectant/detergent designed for general housekeeping purposes should be used in patient-care areas if uncertainty exists regarding the nature of the soil on the surface (e.g., blood or body fluid contamination versus routine dust or dirt). Unless contamination is reasonably anticipated or apparent, cleaning or disinfecting walls, window drapes and other vertical surfaces is unnecessary. However, when housekeeping surfaces are visibly contaminated by blood or OPIM, prompt removal and surface disinfection is appropriate infection control practice and required by OSHA.

Part of the cleaning strategy is to minimize contamination of cleaning solutions and cleaning tools (e.g., mop heads or cleaning cloths). Mops and cloths should be cleaned after use and allowed to dry before re-use, or single-use, disposable mop heads and cloths should be used to avoid spreading contamination.

Cost, safety, product-surface compatibility and acceptability by housekeepers can be key criteria for selecting a cleaning agent or an EPA-registered hospital disinfectant/detergent. Protective equipment used during cleaning and housekeeping procedures should be appropriate to the task.

Another reservoir for microorganisms can be solutions of detergents or disinfectants, especially if prepared in dirty containers, stored for long periods of time or prepared incorrectly. Manufacturers' instructions for preparation and use should be followed. Making fresh cleaning solution each day, discarding any remaining solution and allowing the container to dry will minimize bacterial contamination. Preferred cleaning methods produce minimal mists and aerosols or dispersion of dust in patient care areas.

Cleaning and disinfection strategies for blood spills

The majority of blood contamination events in dentistry result from spatter during dental procedures using rotary or ultrasonic instrumentation. Although no evidence supports that HBV, HCV or HIV has been transmitted from a housekeeping surface, prompt removal and surface disinfection of an area contaminated by either blood or OPIM are appropriate infection control practices and required by OSHA.

Strategies for decontaminating spills of blood and other body fluids differ by setting and volume of the spill. Blood spills on either clinical contact or housekeeping surfaces should be contained and managed as quickly as possible to reduce the risk of contact by patients and workers. The person assigned to clean the spill should wear gloves and other protective equipment as needed. Visible organic material should be removed with absorbent material (e.g., disposable paper towels discarded in a leak-proof, appropriately labeled container). Nonporous surfaces should be cleaned and then decontaminated with either an EPA-registered hospital disinfectant effective against HBV and HIV or an EPA-registered hospital disinfectant with a tuberculocidal claim (i.e., intermediate-level disinfectant). If sodium hypochlorite is chosen, an EPA-registered sodium hypochlorite product is preferred. However, if such products are unavailable, a 1:100 dilution of sodium hypochlorite (e.g., approximately ¼ cup of 5.25 percent household chlorine bleach to 1 gallon of water) is an inexpensive and effective disinfecting agent.

Carpeting and cloth furnishings

Carpeting is more difficult to clean than nonporous hard surface flooring, and it cannot be reliably disinfected, especially after spills of blood and body substances. Studies have documented the presence of diverse microbial populations, primarily bacteria and fungi, in carpeting. Cloth furnishings pose similar contamination risks in areas of direct patient care and places where contaminated materials are managed (e.g., dental operatory, laboratory, or instrument processing areas). For these reasons, use of carpeted flooring and fabric-upholstered furnishings in these areas should be avoided.

Nonregulated and regulated medical waste

Studies have compared microbial load and diversity of microorganisms in residential waste with waste from multiple health care settings. General waste from hospitals or other health care facilities (e.g., dental practices or clinical/research laboratories) is no more infective than residential waste. The majority of soiled items in dental offices are general medical waste, and thus can be disposed of with ordinary waste. Examples include used gloves, masks, gowns, lightly soiled gauze or cotton rolls, and environmental barriers (e.g., plastic sheets or bags) used to cover equipment during treatment.

Although any item that has had contact with blood, exudates or secretions might be infective, treating all such waste as infective is neither necessary nor practical. Infectious waste that carries a substantial risk of causing infection during handling and disposal is regulated medical waste. A complete definition of regulated waste

is included in OSHA's blood-borne pathogens standard. Regulated medical waste is only a limited subset of waste: 9-15 percent of total waste in hospitals and 1-2 percent of total waste in dental offices.

Regulated medical waste requires special storage, handling, neutralization and disposal and is covered by federal, state and local rules and regulations. Examples of regulated waste found in dental-practice settings are solid waste soaked or saturated with blood or saliva (e.g., gauze saturated with blood after surgery), extracted teeth, surgically removed hard and soft tissues, and contaminated sharp items (e.g., needles, scalpel blades and wires). Regulated medical waste requires careful containment for treatment or disposal. A single leak-resistant biohazard bag is usually adequate for containment of nonsharp regulated medical waste, provided the bag is sturdy and the waste can be discarded without contaminating the bag's exterior. Exterior contamination or puncturing of the bag requires placement in a second biohazard bag. All bags should be securely closed for disposal. Puncture-resistant containers with a biohazard label, located at the point of use (i.e., sharps containers), are used as containment for scalpel blades, needles, syringes and unused sterile sharps.

Dental health care facilities should dispose of medical waste regularly to avoid accumulation. Any facility generating regulated medical waste should have a plan for its management that complies with federal, state and local regulations to ensure health and environmental safety.

Discharging blood or other body fluids to sanitary sewers or septic tanks

All containers with blood or saliva (e.g., suctioned fluids) can be inactivated in accordance with state-approved treatment technologies, or the contents can be carefully poured down a utility sink, drain or toilet. Appropriate protective equipment (e.g., gloves, gown, mask and protective eyewear) should be worn when performing this task. No evidence exists that blood-borne diseases have been transmitted from contact with raw or treated sewage. Multiple blood-borne pathogens, particularly viruses, are not stable in the environment for long periods, and the discharge of limited quantities of blood and other body fluids into the sanitary sewer is considered a safe method for disposing of these waste materials. State and local regulations vary and dictate whether blood or other body fluids require pretreatment or if they can be discharged into the sanitary sewer and in what volume.

Dental unit waterlines, biofilm, and water quality

Studies have demonstrated that dental unit waterlines (i.e., narrow-bore plastic tubing that carries water to the high-speed handpiece, air/water syringe and ultrasonic scaler) can become colonized with microorganisms, including bacteria, fungi and protozoa. Protected by a polysaccharide slime layer known as a glycocalyx, these microorganisms colonize and replicate on the interior surfaces of the waterline tubing and form a biofilm, which serves as a reservoir that can amplify the number of free-floating (i.e., planktonic) microorganisms in water used for dental treatment. Although oral flora and human pathogens (e.g., Pseudomonas aeruginosa, Legionella species, and nontuberculous Mycobacterium species), have been isolated from dental water systems, the majority of organisms recovered from dental waterlines are common heterotrophic water bacteria. These exhibit limited pathogenic potential for immuno-competent persons.

Dental unit water quality

Research has demonstrated that microbial counts can reach 200,000 colony-forming units (CFU)/mL within five days after installation of new dental unit waterlines, and levels of microbial contamination 106 CFU/mL of dental unit water have been documented. These counts can occur because dental unit waterline factors (e.g., system design, flow rates and materials) promote both bacterial growth and development of biofilm.

Although no epidemiologic evidence indicates a public health problem, the presence of substantial numbers of pathogens in dental unit waterlines generates concern. Exposing patients or dental workers to water of uncertain microbiological quality, despite the lack of documented adverse health effects, is inconsistent with accepted infection control principles. Thus, in 1995, ADA addressed the dental water concern by asking manufacturers to provide equipment with the ability to deliver treatment water with less than 200 CFU/mL of unfiltered output from waterlines. This threshold was based on the quality assurance standard established for dialysate fluid, to ensure that fluid delivery systems in hemodialysis units have not been colonized by indigenous waterborne organisms.

Standards also exist for safe drinking water quality as established by EPA, the American Public Health Association (APHA) and the American Water Works Association (AWWA); they have set limits for heterotrophic bacteria of less than 500 CFU/mL of drinking water. Thus, the number of bacteria in water used as a coolant/irrigant for nonsurgical dental procedures should be as low as reasonably achievable and, at a minimum, less than 500 CFU/mL, the regulatory standard for safe drinking water established by EPA and APHA/AWWA.

SPECIAL CONSIDERATIONS

Dental handpieces and other devices attached to air and waterlines

Multiple semicritical dental devices that touch mucous membranes are attached to the air or waterlines of the dental unit. Among these devices are high- and low-speed handpieces, prophylaxis angles, ultrasonic and sonic scaling tips, air abrasion devices and air and water syringe tips. Although no epidemiologic evidence implicates these instruments in disease transmission, studies of high-speed handpieces using dye expulsion have confirmed the potential for retracting oral fluids into internal compartments of the device. This determination indicates that retained patient material can be expelled intraorally during subsequent uses. Studies using laboratory models also indicate the possibility for retention of viral DNA and viable virus inside both high-speed handpieces and prophylaxis angles. The potential for contamination of the internal surfaces of other devices (e.g., low-speed handpieces and ultrasonic scalers), has not been studied, but restricted physical access limits their cleaning. Accordingly, any dental device connected to the dental air/water system that enters the patient's mouth should be run to discharge water, air or a combination for a minimum of 20-30 seconds after each patient. This procedure is intended to help physically flush out patient material that might have entered the turbine and air and waterlines.

Heat methods can sterilize dental handpieces and other intraoral devices attached to air or waterlines. For processing any dental device that can be removed from the dental unit air or waterlines, neither surface disinfection nor immersion in chemical germicides is an acceptable method. Ethylene oxide gas cannot adequately sterilize

internal components of handpieces. In clinical evaluations of high-speed handpieces, cleaning and lubrication were the most critical factors in determining performance and durability. Manufacturer's instructions for cleaning, lubrication and sterilization should be followed closely to ensure both the effectiveness of the process and the longevity of handpieces.

Some components of dental instruments are permanently attached to dental unit waterlines, and although they do not enter the patient's oral cavity, they are likely to become contaminated with oral fluids during treatment procedures. Such components (e.g., handles or dental unit attachments of saliva ejectors, high-speed air evacuators and air/water syringes) should be covered with impervious barriers that are changed after each use. If the item becomes visibly contaminated during use, dental care personnel should clean and disinfect with an EPA-registered hospital disinfectant (intermediate-level) before use on the next patient.

Saliva ejectors

Backflow from low-volume saliva ejectors occurs when the pressure in the patient's mouth is less than that in the evacuator. Studies have reported that backflow in low-volume suction lines can occur, and microorganisms can be present in the lines retracted into the patient's mouth when a seal around the saliva ejector is created (e.g., by a patient closing their lips around the tip of the ejector, creating a partial vacuum). This backflow can be a potential source of cross-contamination; occurrence is variable because the quality of the seal formed varies between patients. Furthermore, studies have demonstrated that gravity pulls fluid back toward the patient's mouth whenever a length of the suction tubing holding the tip is positioned above the patient's mouth, or during simultaneous use of other evacuation (high-volume) equipment. Although no adverse health effects associated with the saliva ejector have been reported, practitioners should be aware that in certain situations, backflow could occur when using a saliva ejector.

Dental radiology

When taking radiographs, the potential to cross-contaminate equipment and environmental surfaces with blood or saliva is high if aseptic technique is not practiced. Gloves should be worn when taking radiographs and handling contaminated film packets. Other protective equipment (e.g., mask, protective eyewear and gowns) should be used if spattering of blood or other body fluids is likely. Heat-tolerant versions of intraoral radiograph accessories are available, and these semicritical items (e.g., film-holding and positioning devices) should be heat-sterilized before patient use. After exposure of the radiograph and before glove removal, the film should be dried with disposable gauze or a paper towel to remove blood or excess saliva and placed in a container (e.g., disposable cup) for transport to the developing area. Alternatively, if FDA-cleared film barrier pouches are used, the film packets should be carefully removed from the pouch to avoid contamination of the outside film packet and placed in the clean container for transport to the developing area. Various methods have been recommended for aseptic transport of exposed films to the developing area, and for removing the outer film packet before exposing and developing the film. Other information regarding dental radiography infection control is available.

Oral surgical procedures

The oral cavity is colonized with numerous microorganisms. Oral surgical procedures present an opportunity for entry of microorganisms (i.e., exogenous and endogenous) into the vascular system and other normally sterile areas of the oral cavity (e.g., bone or subcutaneous tissue); therefore, an increased potential exists for localized or systemic infection. Oral surgical procedures involve the incision, excision or reflection of tissue that exposes the normally sterile areas of the oral cavity. Examples include biopsy, periodontal surgery, apical surgery, implant surgery and surgical extractions of teeth (e.g., removal of erupted or nonerupted tooth requiring elevation of mucoperiosteal flap, removal of bone or section of tooth, and suturing if needed) (see Hand hygiene, PPE, single-use or disposable devices, and Dental unit water quality).

Handling of biopsy specimens

To protect persons handling and transporting biopsy specimens, each specimen must be placed in a sturdy, leak-proof container with a secure lid for transportation. Care should be taken when collecting the specimen to avoid contaminating the outside of the container. If the outside of the container becomes visibly contaminated, it should be cleaned and disinfected or placed in an impervious bag. The container must be labeled with the biohazard symbol during storage, transport, shipment and disposal.

Handling of extracted teeth disposal

Extracted teeth that are being discarded are subject to the containerization and labeling provisions outlined by OSHA's blood-borne pathogens standard. OSHA considers extracted teeth to be potentially infectious material that should be disposed in medical waste containers. Extracted teeth sent to a dental laboratory for shade or size comparisons should be cleaned, surface-disinfected with an EPA-registered hospital disinfectant with intermediate-level activity (i.e., tuberculocidal claim), and transported in a manner consistent with OSHA regulations. However, extracted teeth can be returned to patients on request, at which time provisions of the standard no longer apply. Extracted teeth containing dental amalgam should not be placed in a medical waste container that uses incineration for final disposal. Commercial metal recycling companies also might accept extracted teeth with metal restorations, including amalgam. State and local regulations should be consulted regarding disposal of the amalgam.

M. tuberculosis

Patients infected with M. tuberculosis occasionally seek urgent dental treatment at outpatient dental settings. Understanding the pathogenesis of the development of TB will help dental care workers determine how to manage such patients. M. tuberculosis is a bacterium carried in airborne infective droplet nuclei that can be generated when persons with pulmonary or laryngeal TB sneeze, cough, speak or sing. These small particles (1–5 μm) can stay suspended in the air for hours. Infection occurs when a susceptible person inhales droplet nuclei containing M. tuberculosis,

which then travel to the alveoli of the lungs. Usually within two to 12 weeks after initial infection with M. tuberculosis, immune response prevents further spread of the TB bacteria, although they can remain alive in the lungs for years, a condition termed latent TB infection. Persons with latent TB infection usually exhibit a reactive tuberculin skin test (TST), have no symptoms of active disease and are not infectious. However, they can develop active disease later in life if they do not receive treatment for their latent infection.

Any dental worker with a persistent cough (i.e., lasting more than three weeks), especially in the presence of other signs or symptoms compatible with active TB (e.g., weight loss, night sweats, fatigue, bloody sputum, anorexia or fever), should be evaluated promptly. The person should not return to the workplace until a diagnosis of TB has been excluded or he or she is on therapy and has been determined noninfectious by a physician.

Creutzfeldt-Jakob disease and other prion diseases

Creutzfeldt-Jakob disease (CJD) belongs to a group of rapidly progressive, invariably fatal, degenerative neurological disorders, transmissible spongiform encephalopathies (TSEs) that affect both humans and animals and are thought to be caused by infection with an unusual pathogen called a prion. Prions are isoforms of a normal protein, capable of self-propagation although they lack nucleic acid. Prion diseases have an incubation period of years and are usually fatal within one year of diagnosis. Among humans, TSEs include CJD, Gerstmann-Straussler-Scheinker syndrome, fatal familial insomnia, kuru and variant CJD (vCJD). Occurring in sporadic, familial and acquired (i.e., iatrogenic) forms, CJD has an annual incidence in the United States and other countries of approximately one case per million population. In approximately 85 percent of affected patients, CJD occurs as a sporadic disease with no recognizable pattern of transmission. A smaller proportion of patients (5-15 percent) experience familial CJD because of inherited mutations of the prion protein gene.

Program evaluation

The goal of a dental infection control program is to provide a safe working environment that will reduce the risk of health care-associated infections among patients and occupational exposures among workers. Medical errors are caused by faulty systems, processes and conditions that lead persons to make mistakes or fail to prevent errors being made by others. Effective program evaluation is a systematic way to ensure procedures are useful, feasible, ethical and accurate. Program evaluation is an essential organizational practice; however, such evaluation is not practiced consistently across program areas, nor is it sufficiently well-integrated into the day-to-day management of the majority of programs.

A successful infection control program depends on developing standard operating procedures, evaluating practices, routinely documenting adverse outcomes (e.g., occupational exposures to blood) and work-related illnesses in dental workers, and monitoring health care-associated infections in patients. Strategies and tools to evaluate the infection control program can include periodic observational assessments, checklists to document procedures and routine review of occupational exposures to blood-borne pathogens. Evaluation offers an opportunity to improve the effectiveness of both the infection control program and dental practice protocols. If deficiencies or problems in the implementation of infection control procedures are identified, further evaluation is needed to eliminate the problems. Examples of infection control program evaluation activities are provided on (Table 2).

TABLE 2. Examples of methods for evaluating infection control programs

Program element	Evaluation activity
Appropriate immunization of dental health care personnel (DHCP).	Conduct annual review of personnel records to ensure up-to-date immunizations.
Assessment of occupational exposures to infectious agents.	Report occupational exposures to infectious agents. Document the steps that occurred around the exposure and plan how such exposure can be prevented in the future.
Comprehensive postexposure management plan and medical follow-up program after occupational exposures to infectious agents.	Ensure the postexposure management plan is clear, complete, and available at all times to all DHCP. All staff should understand the plan, which should include toll-free phone numbers for access to additional information.
Adherence to hand hygiene before and after patient care.	Observe and document circumstances of appropriate or inappropriate handwashing. Review findings in a staff meeting.
Proper use of personal protective equipment to prevent occupational exposures to infectious agents.	Observe and document the use of barrier precautions and careful handling of sharps. Review findings in a staff meeting.
Routine and appropriate sterilization of instruments using a biologic monitoring system.	Monitor paper log of steam cycle and temperature strip with each sterilization load, and examine results of weekly biologic monitoring. Take appropriate action when failure of sterilization process is noted.
Evaluation and implementation of safer medical devices.	Conduct an annual review of the exposure control plan and consider new developments in safer medical devices.
Compliance of water in routine dental procedures with current U.S. Environmental Protection Agency drinking water standards (fewer than 500 CFU of heterotrophic water bacteria).	Monitor dental water quality as recommended by the equipment manufacturer, using commercial self-contained test kits, or commercial water-testing laboratories.
Proper handling and disposal of medical waste.	Observe the safe disposal of regulated and nonregulated medical waste and take preventive measures if hazardous situations occur.
Health care–associated infections.	Assess the unscheduled return of patients after procedures and evaluate them for an infectious process. A trend might require formal evaluation.

Infection control research considerations

Although the number of published studies concerning dental infection control has increased in recent years, questions regarding infection control practices and their effectiveness remain unanswered. Multiple concerns were identified by the working group for this report, as well as by others during the public comment period on Table 6. This list is not exhaustive and does not represent a CDC research agenda, but rather is an effort to identify certain concerns, stimulate discussion, and provide direction for determining future action by clinical, basic science and epidemiologic investigators, as well as health and professional organizations, clinicians and policy makers.

Infection control research considerations

Although the number of published studies concerning dental infection control has increased in recent years, questions regarding infection control practices and their effectiveness remain unanswered. Multiple concerns were identified by the working group for a CDC report, as well as by others during a public comment period.

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Education and promotion

- Design strategies to communicate, to the public and providers, the risk of disease transmission in dentistry.
- Promote use of protocols for recommended postexposure management and follow-up.
- Educate and train dental health care personnel (DHCP) to screen and evaluate safer dental devices by using tested design and performance criteria.

Laboratory-based research

- Develop animal models to determine the risk of transmitting organisms through inhalation of contaminated aerosols (e.g., influenza) produced from rotary dental instruments.
- Conduct studies to determine the effectiveness of gloves (i.e., material compatibility and duration of use).
- Develop devices with passive safety features to prevent percutaneous injuries.
- Study the effect of alcohol-based hand-hygiene products on retention of latex proteins and other dental allergens (e.g., methyl methacrylate, glutaraldehyde, thiurams) on the hands of workers after latex glove use.
- Investigate the applicability of other types of sterilization procedures (e.g., hydrogen peroxide gas plasma) in dentistry. Encourage manufacturers to determine optimal methods and frequency for testing dental-unit waterlines and maintaining dental-unit water-quality standards.
- Determine the potential for internal contamination of low-speed handpieces, including the motor, and other devices connected to dental air and water supplies, as well as more efficient ways to clean, lubricate, and sterilize handpieces and other devices attached to air or waterlines.
- Investigate the infectivity of oral tissues in Creutzfeldt-Jakob disease (CJD) or variant CJD patients.
- Determine the most effective methods to disinfect dental impression materials.
- Investigate the viability of pathogenic organisms on dental materials (e.g., impression materials, acrylic resin, or gypsum materials) and dental laboratory equipment.
- Determine the most effective methods for sterilization or disinfection of digital radiology equipment.
- Evaluate the effects of repetitive reprocessing cycles on burs and endodontic files.
- Investigate the potential infectivity of vapors generated from the various lasers used for oral procedures.

Clinical and population-based epidemiologic research and development

- Continue to characterize the epidemiology of blood contacts, particularly percutaneous injuries, and the effectiveness of prevention measures.
- Further assess the effectiveness of double gloving in preventing blood contact during routine and surgical dental procedures.
- Continue to assess the stress placed on gloves during dental procedures and the potential for developing defects during different procedures.
- Develop methods for evaluating the effectiveness and costeffectiveness of infection control interventions.
- Determine how infection control guidelines affect the knowledge, attitudes, and practices of dental workers.

Selected definitions

Alcohol-based hand rub – An alcohol-containing preparation designed for reducing the number of viable microorganisms on the hands.

Antiseptic – A germicide used on skin or living tissue for the purpose of inhibiting or destroying microorganisms (e.g., alcohols, chlorhexidine, chlorine, hexachlorophene, iodine, chloroxylenol [PCMX], quaternary ammonium compounds, and triclosan).

Bead sterilizer – A device using glass beads 1.2–1.5 mm diameter and temperatures 217ºC–232ºC for brief exposures (e.g., 45 seconds) to inactivate microorganisms. (This term is actually a misnomer because it has not been cleared by the Food and Drug Administration [FDA] as a sterilizer).

Bioburden – Microbiological load (i.e., number of viable organisms in or on an object or surface) or organic material on a surface or object before decontamination, or sterilization. Also known as bioload or microbial load.

Colony-forming unit (CFU) – The minimum number (i.e., tens of millions) of separable cells on the surface of or in semisolid agar medium that give rise to a visible colony of progeny. CFUs can consist of pairs, chains, clusters, or as single cells and are often expressed as colony-forming units per milliliter (CFUs/mL).

DHCP – Dental health care personnel/professionals (DHCP) include dentists, dental hygienists, dental assistants, dental laboratory technicians (in-office and commercial), students and trainees, contractual personnel, and other persons not directly involved

in patient care but potentially exposed to infectious agents (e.g., administrative, clerical, housekeeping, maintenance, or volunteer personnel). Working in a dental health care facility.

Dental treatment water – Nonsterile water used during dental treatment, including irrigation of nonsurgical operative sites and cooling of high-speed rotary and ultrasonic instruments.

Droplet nuclei – Particles <5 μm in diameter formed by dehydration of airborne droplets containing microorganisms that can remain suspended in the air for long periods of time.

Endotoxin – The lipopolysaccharide of gram-negative bacteria, the toxic character of which resides in the lipid protein. Endotoxins can produce pyrogenic reactions in persons exposed to their bacterial component.

HCP – Health care personnel/professionals include doctors, nurses, radiologist, laboratory technicians, pharmacists, assistants, (in-office and commercial), students and trainees, contractual personnel, and other persons not directly involved in patient care but potentially exposed to infectious agents (e.g., administrative, clerical, housekeeping, maintenance, or volunteer personnel) working in a health care facility.

HCW – Health care worker includes anyone working in a health care facility of any kind whenever there is potential contact for spattering of blood or OPIM.

Hepatitis B immune globulin (HBIG) – Product used for prophylaxis against HBV infection. HBIG is prepared from plasma containing high titers of hepatitis B surface antibody (anti-HBs) and provides protection for 3–6 mos.

Hepatitis B surface antigen (HBsAg) – Serologic marker on the surface of HBV detected in high levels during acute or chronic hepatitis. The body normally produces antibodies to surface antigen as a normal immune response to infection.

Hepatitis B e-antigen (HBeAg) – Secreted product of the nucleocapsid gene of HBV found in serum during acute and chronic HBV infection. Its presence indicates that the virus is replicating and serves as a marker of increased infectivity.

Hepatitis B surface antibody (anti-HBs) – Protective antibody against HBsAg. Presence in the blood can indicate past infection with, and immunity to, HBV, or immune response from hepatitis B vaccine.

Heterotrophic bacteria – Those bacteria requiring an organic carbon source for growth (i.e., deriving energy and carbon from organic compounds).

Iatrogenic – Induced inadvertently by HCP, medical (including dental) treatment, or diagnostic procedures. Used particularly in reference to an infectious disease or other complication of treatment.

Nosocomial – Infection acquired in a hospital as a result of medical care.

OPIM – Other potentially infectious materials. OPIM is an OSHA term that refers to: 1.) Body fluids including semen, vaginal secretions, cerebrospinal fluid, synovial fluid, pleural fluid, pericardial fluid, peritoneal fluid, amniotic fluid, saliva in dental procedures; any body fluid visibly contaminated with blood; and all body fluids in situations where differentiating between body fluids is difficult or impossible; 2.) Any unfixed tissue or organ (other than intact skin) from a human (living or dead); and 3.) HIV-containing cell or tissue cultures, organ cultures; HIV- or HBV-containing culture medium or other solutions; and blood, organs, or other tissues from experimental animals infected with HIV or HBV.

Prion – Protein particle lacking nucleic acid that has been implicated as the cause of certain neurodegenerative diseases (e.g., scrapie, CJD, and bovine spongiform encephalopathy [BSE]).

Retraction – Entry of oral fluids and microorganisms into waterlines through negative water pressure.

Seroconversion – The change of a serological test from negative to positive indicating the development of antibodies in response to infection or immunization.

Sterile – Free from all living microorganisms; usually described as a probability (e.g., the probability of a surviving microorganism being 1 in 1 million).

Sterilization – Use of a physical or chemical procedure to destroy all microorganisms including substantial numbers of resistant bacterial spores.

Conclusion

Dental health care professionals are at risk everyday, but here we have seen many infection control practices a dentist, his staff and his patients can take to reduce those risks By taking the sterilization precautions, developing a written plan for the key elements of an infection control process, maintaining the necessary records, evaluating the plan on a routine basis and making changes to keep the processes up-to-date, the goal of minimizing the risk of disease transmission in the dental office can be met.

References

- CDC. Guidelines for Infection Control in Dental Health care Setings-2003. MMWR Dec 19, 2003/ Vol. 52/ No. RR-17.
- Provisions Applicable To All Licensees, Article 1. Section 1005. Minimum Standards for Infection Control.

Final Examination Questions

Select the best answer questions 11 through 15 and mark your answers on the Final Examination Answer Sheet found on page 148, or for faster service complete your test online at **Dental.EliteCME.com.**

11. Avoiding exposure to blood and OPIM as well as protection by immunization remain primary strategies for reducing occupationally acquired infections, but occupational exposures can still occur.
    - ○ True
    - ○ False

12. Standard precautions are strategies used to reduce the risk of infection from exposure to blood, all body fluids and secretions (except sweat), nonintact skin and mucous membranes.
    - ○ True
    - ○ False

13. There is evidence that shows using antiseptics for wound care or expressing fluid by squeezing the wound further reduces the risk of blood-borne pathogen transmission.
    - ○ True
    - ○ False

14. Chemical sterilization monitoring assesses physical conditions, such as time and temperature, during the sterilization process, but it cannot prove sterilization has been achieved.
    - ○ True
    - ○ False

15. Sterilization is the use of a physical or chemical procedure to destroy all microorganisms, including substantial numbers of resistant bacterial spores.
    - ○ True
    - ○ False

Chapter 4: Nitrous Oxide – N_2O

6 CE Hours

By: Elite Staff

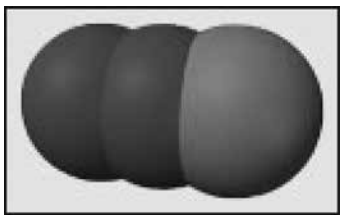
Learning objectives

- Review the history of nitrous oxide.
- Describe the production of nitrous oxide.
- List the uses of nitrous oxide.
- Explain the use of nitrous oxide in dental operatories.
- Describe the hazards in the workplace.
- Review the methods of engineering control and training.

Introduction

Sedation dentistry, sometimes called relaxation dentistry, refers to the way dentists manage pain and anxiety during dental appointments.

Conscious sedation is defined as a minimally depressed level of consciousness that retains the patient's ability to independently and continuously maintain an airway and respond appropriately to physical stimulation and verbal command that is produced by pharmacological or nonpharmacologic method or a combination of both. Nitrous oxide is only one of the 14 different ways that sedation drugs can be administered. There are three primary ways that sedation is administered in the dental office: IV sedation, enteral conscious sedation and inhalation conscious sedation or nitrous oxide.



N^2O

Inhalation conscious sedation or the use of nitrous oxide, commonly known as laughing gas, is a chemical compound with the formula N_2O. It is an oxide of nitrogen. At room temperature, it is a colorless non-flammable gas with a pleasant, slightly sweet odor and taste. It is used in surgery and dentistry for its anesthetic and analgesic effects. It is known as "laughing gas" because of the euphoric effects of inhaling it, a property that has led to its recreational use as a dissociative hallucinogen. It is also used as an oxidizer in rocketry and in motor racing to increase the power output of engine. At elevated temperatures, nitrous oxide is a powerful oxidizer similar to molecular oxygen. For example, nitrous oxide in a test tube will re-ignite a smoldering splint.

Nitrous oxide reacts with ozone and is the main naturally occurring regulator of stratospheric ozone. It is also a major greenhouse gas and air pollutant. Considered over a 100-year period, it has 298 times more impact per unit weight than carbon dioxide.

History

The gas was first synthesized by English chemist and Unitarian minister Joseph Priestley in 1772, who called it phlogisticated nitrous air. Priestley published his discovery in the book "Experiments and Observations on Different Kinds of Air" (1775), where he described how to produce the preparation of "nitrous air diminished" by heating iron filings dampened with nitric acid.

Early use (1794-1843)

The first important use of nitrous oxide was made possible by Thomas Beddoes and the renowned engineer James Watt, who worked together to publish the book "Considerations on the Medical Use and on the Production of Factitious Airs" (1794). This book was important for two reasons. First, James Watt had invented a novel machine to produce "factitious airs" (i.e. nitrous oxide) and a novel "breathing apparatus" to inhale the gas. Second, the book also presented the new medical theories by Thomas Beddoes, that tuberculosis and other lung diseases could be treated by inhalation of factitious airs.

The machine to produce factitious airs was comprised of three parts: a furnace to burn the needed material, a vessel with water where the produced gas passed through in a spiral pipe (in order for impurities to be "washed off"), and finally the gas cylinder with a gasometer where the produced air could be tapped into portable air bags (made of airtight oily silk). The breathing apparatus was one of the portable air bags connected with a tube to a mouthpiece. With this new equipment engineered and produced already in 1794, the way was now paved for clinical trials, which began when Thomas Beddoes in 1798 established the Pneumatic Institution for Relieving Diseases by Medical Airs in Clifton (Bristol). In the basement of the building, a large-scale machine was producing the gases under the supervision of a young Humphry Davy, who was encouraged to experiment with new gases for patients to inhale. The first important work of Davy was to examine the nitrous oxide, with the results being published in his book: "Researches, Chemical and Philosophical" (1800).

Despite the valuable finding made by Davy, that inhalation of nitrous oxide could relieve a conscious person from pain, another 44 years would elapse before doctors attempted to use it for anesthesia.

Anesthetic use

At a "popular science" exhibition in Hartford, Connecticut, where volunteers inhaled nitrous oxide, local dentist Horace Wells noted one of them, a man who had injured his leg, seemed unaware of any pain from the injury. Thus was the born the first use of nitrous oxide as anesthetic drug. Wells himself, with assistance by Gardner Quincy Colton and John Mankey Riggs, demonstrated insensitivity to pain

from a dental extraction in December 1844. In the following weeks, Wells treated the first 12-15 patients with nitrous oxide in Hartford, and according to his own record, only failed in two cases. In spite of these convincing results reported by Wells to the medical society in Boston in December 1844, this new method was not immediately adopted by other dentists. This probably was because in January 1845, Wells had been partly unsuccessful at his first public demonstration of the use of nitrous oxide for the medical faculty in Boston, leaving his colleagues doubtful regarding its efficacy and safety.

The method did not come into general use until 1863, when Colton successfully started to use it in all his Colton Dental Association clinics, which he just had established in New Haven and New York City. Over the following three years, Colton and his associates successfully administered nitrous oxide to more than 25,000 patients. With its efficacy and safety now demonstrated by large numbers, the usage of nitrous oxide rapidly became the preferred anesthetic method in dentistry. Because the gas is mild enough to keep a patient in a conscious and conversational state but in most cases is strong enough to suppress the pain caused by dental work, it remains the preferred agent in dentistry today.

In hospitals, however, nitrous oxide was found not to be a strong enough for use in large operations. A stronger and more potent anesthetic, sulfuric ether, was instead demonstrated and accepted for use in October 1846, along with chloroform in 1847. When Joseph Thomas Clover invented the "gas-ether inhaler" in 1876, it became a common practice at hospitals to initiate all anesthetic treatments with a mild flow of nitrous oxide, and then gradually increase the anesthesia with the stronger ether/chloroform. Clover's gas-ether inhaler was designed to supply the patient with nitrous oxide and ether at the same time, with the exact mixture controlled by the operator of the device. It remained in use by many hospitals until the 1930s. Although hospitals today are using a more advanced anesthetic machine, these machines still use the same principle launched with Clover's gas-ether inhaler, to initiate the anesthesia with nitrous oxide before the administration of a more powerful anesthetic.

Production

Nitrous oxide is most commonly prepared by careful heating of ammonium nitrate, which decomposes into nitrous oxide and water vapor. The addition of various phosphates favors formation of a purer gas at slightly lower temperatures. One of the earliest commercial producers was George Poe in Trenton, New Jersey.

- NH_4NO_3 (s) → 2 H_2O (g) + N_2O (g)
  - This reaction occurs between 170-240 degrees C, temperatures where ammonium nitrate is a moderately sensitive explosive and a very powerful oxidizer. Above 240 degrees C, the exothermic reaction may accelerate to the point of detonation, so the mixture must be cooled to avoid such a disaster. Superheated steam is used to reach reaction temperature in some turnkey production plants.

Downstream, the hot, corrosive mixture of gases must be cooled to condense the steam and filtered to remove higher oxides of nitrogen. Ammonium nitrate smoke, as an extremely persistent colloid, will also have to be removed. The cleanup is often done in a train of three gas washes, base, acid and base again. However, significant amounts of nitric oxide (NO) may not necessarily be absorbed directly by the base (sodium hydroxide) washes.

The nitric oxide impurity is sometimes chelated out with ferrous sulfate, reduced with iron metal or oxidized and absorbed in base as a higher oxide. The first base wash may (or may not) react out much of the ammonium nitrate smoke. However, this reaction generates ammonia gas, which may have to be absorbed in the acid wash.

Other routes

The direct oxidation of ammonia may someday rival the ammonium nitrate pyrolysis synthesis of nitrous oxide mentioned above. This capital-intensive process, which originates in Japan, uses a manganese dioxide-bismuth oxide catalyst:

- 2 NH_3 + 2 O_2 → N_2O + 3 H_2O
  - Higher oxides of nitrogen are formed as impurities. In comparison, uncatalyzed ammonia oxidation (i.e. combustion or explosion) goes primarily to N_2 and H_2O.

Nitrous oxide can be made by heating a solution of sulfamic acid and nitric acid. Many gases are made this way in Bulgaria.

- HNO_3 + NH_2SO_3H → N_2O + H_2SO_4 + H_2O
  - There is no explosive hazard in this reaction if the mixing rate is controlled. However, as usual, toxic higher oxides of nitrogen are formed.

Nitrous oxide is produced in large volumes as a byproduct in the synthesis of adipic acid, one of the two reactants used in nylon manufacture. This might become a major commercial source, but will require the removal of higher oxides of nitrogen and organic impurities. Currently, much of the gas is decomposed before release for environmental protection. Greener processes may prevail that substitute hydrogen peroxide for nitric acid oxidation; hence no generation of oxide of nitrogen by-products.

Hydroxylammonium chloride can react with sodium nitrite to produce N_2O as well:

- $NH_3OH^+Cl^-$ + $NaNO_2$ → N_2O + NaCl + 2 H_2O
  - If the nitrite is added to the hydroxylamine solution, the only remaining byproduct is salt water. However, if the hydroxylamine solution is added to the nitrite solution (nitrite is in excess), then toxic higher oxides of nitrogen are also formed.

APPLICATIONS

Rocket motors

Nitrous oxide can be used as an oxidizer in a rocket motor. This has the advantages over other oxidizers that it is non-toxic, and because of its stability at room temperature, easy to store and relatively safe to carry on a flight. As a secondary benefit, it can be readily decomposed to form breathing air. Its high density and low storage pressure enable it to be highly competitive with stored high-pressure gas systems.

In a 1914 patent, American rocket pioneer Robert Goddard suggested nitrous oxide and gasoline as possible propellants for a liquid-fueled rocket. Nitrous oxide has been the oxidizer of choice in several hybrid rocket designs (using solid fuel with a liquid or gaseous oxidizer). The combination of nitrous oxide with hydroxyl-terminated polybutadiene fuel has been used by SpaceShipOne and others. It is also notably used in amateur and high power rocketry with various plastics as the fuel.

Nitrous oxide can also be used in a monopropellant rocket. In the presence of a heated catalyst, N_2O will decompose exothermically into nitrogen and oxygen, at a temperature of approximately 1,300 degrees C. Because of the large heat release, the catalytic action rapidly becomes secondary as thermal autodecomposition becomes dominant.

In a vacuum thruster, this can provide a monopropellant specific impulse (Isp) of as much as 180s. While noticeably less than the Isp available from hydrazine thrusters (monopropellant or bipropellant with nitrogen tetroxide), the decreased toxicity makes nitrous oxide an option worth investigating.

Specific impulse (I_{sp}) can be improved by blending a hydrocarbon fuel with the nitrous oxide inside the same storage tank, becoming a nitrous oxide fuel blend (NOFB) monopropellant. This storage mixture does not incur the danger of spontaneous ignition because N_2O is chemically stable. When the nitrous oxide decomposes by a heated catalyst, high-temperature oxygen is released and rapidly ignites the hydrocarbon fuel blend. NOFB monopropellants are capable of I_{sp} greater than 300 seconds, while avoiding the toxicity associated with hypergolic propulsion systems. The low freezing point of NOFB eases thermal management compared to hydrazine and dinitrogen tetroxide – a valuable property for space storable propellants.

Internal combustion engine

In vehicle racing, nitrous oxide (often referred to as just "nitrous" or as NOS after the name of the brand Nitrous Oxide Systems) allows the engine to burn more fuel and air, resulting in a more powerful combustion. The gas itself is not flammable, but it delivers more oxygen than atmospheric air by breaking down at elevated temperatures.

Nitrous oxide is stored as a compressed liquid; the evaporation and expansion of liquid nitrous oxide in the intake manifold causes a large drop in intake charge temperature, resulting in a denser charge, further allowing more air/fuel mixture to enter the cylinder. Nitrous oxide is sometimes injected into (or prior to) the intake manifold, whereas other systems directly inject right before the cylinder (direct port injection) to increase power.

The technique was used during World War II by Luftwaffe aircraft with the GM-1 system to boost the power output of aircraft engines. Originally meant to provide the Luftwaffe standard aircraft with superior high-altitude performance, technological considerations limited its use to extremely high altitudes. Accordingly, it was only used by specialized planes like high-altitude reconnaissance aircraft, high-speed bombers and high-altitude interceptor aircraft.

One of the major problems of using nitrous oxide in a reciprocating engine is that it can produce enough power to damage or destroy the engine. Very large power increases are possible, and if the mechanical structure of the engine is not properly reinforced, the engine may be severely damaged or destroyed during this kind of operation. It is very important with nitrous oxide augmentation of internal combustion engines to maintain proper operating temperatures and fuel levels to prevent "pre-ignition" or "detonation" (sometimes referred to as "knocking" or "pinging"). Most problems that are associated with nitrous do not come from mechanical failure due to the power increases. Since nitrous allows a much denser charge into the cylinder, it dramatically increases cylinder pressures. The increased pressure and temperature can cause problems, such as melting the piston or valves. It may also crack or warp the piston or head and cause pre-ignition due to uneven heating.

Aerosol propellant

The gas is approved for use as a food additive (also known as E942), specifically as an aerosol spray propellant. Its most common uses in this context are in aerosol whipped cream canisters, cooking sprays and as an inert gas used to displace oxygen and inhibit bacterial growth when filling packages of potato chips and other similar snack foods.

The gas is extremely soluble in fatty compounds. In aerosol whipped cream, it is dissolved in the fatty cream until it leaves the can, when it becomes gaseous and thus creates foam. Used in this way, it produces whipped cream four times the volume of the liquid, whereas whipping air into cream only produces twice the volume. If air were used as a propellant, oxygen would accelerate rancidification of the butterfat; nitrous oxide inhibits such degradation. Carbon dioxide cannot be used for whipped cream because it is acidic in water, which would curdle the cream and give it a seltzer-like "sparkling" sensation.

However, the whipped cream produced with nitrous oxide is unstable and will return to a liquid state within half an hour to one hour. Thus, the method is not suitable for decorating food that will not be immediately served.

Similarly, cooking spray, which is made from various types of oils combined with lecithin (an emulsifier), may use nitrous oxide as a propellant; other propellants used in cooking spray include food-grade alcohol and propane.

Users of nitrous oxide for recreational use as a euphoria-inducing inhalant drug, often obtain it from whipped cream dispensers that use nitrous oxide as a propellant. It is not harmful in small doses, but risks due to lack of oxygen do exist (see Recreational section).

Recreational use

Nitrous oxide (N_2O) is a dissociative drug that can cause analgesia, depersonalization, dizziness, euphoria and some sound distortion. Research has also found that it increases suggestibility and imagination. Inhalation of nitrous oxide for recreational use to cause euphoria and slight hallucinations began as a phenomenon for the British upper class in 1799 at "laughing gas parties." When equipment became more widely available for dentistry and hospitals, most countries also restricted the legal access to buy pure nitrous oxide gas cylinders to those sectors. A low availability of equipment to produce the gas combined with a low usage of the gas for medical purposes meant recreational use was a relatively rare phenomenon that mainly took place among students at medical universities. That apparently continued into the 20th century. A poll taken in 1979 indicated that between 1 and 2 percent of medical and dental students used nitrous oxide for recreational purposes, according to Theodore J. Jastak in a 1991 article in the Journal of the American Dental Association.

In the 1960 and '70s, the recreational use of inhalants became somewhat fashionable again, according to a Consumers Union report in 1972 based on reports of use in Maryland and Vancouver and a survey at the University of Michigan in 1970.

According to the Michigan survey: "It was not uncommon [in the interviews] to hear from individuals who had been to parties where a professional (doctor, nurse, scientist, inhalation therapist, researcher) had provided nitrous oxide. There also were those who work in restaurants who used the N_2O stored in tanks for the preparation of whip cream. Reports were received from individuals who used the gas contained in aerosol cans both of food and non-food products. At a rock festival, nitrous oxide was widely sold for 25 cents a balloon. Contact was made with a 'mystical-religious' group that used the gas to accelerate arriving at their transcendental-meditative state of choice. Although a few more sophisticated users employed nitrous oxide-oxygen mixes with elaborate equipment, most users employed balloons or plastic bags. They either held a breath of N_2O or rebreathed

the gas. There were no adverse effects reported in the more than 100 individuals surveyed."

Although recreational use is believed to be somewhat limited today, government data on substance abuse of youths shows that inhalants, including nitrous oxide, are being used by young people.

The Substance Abuse and Mental Health Services Administration (SAMHSA) said in its 2007 report on trends in drug use that almost 1 million youth had used inhalants within the past year. The percentage of young people aged 12-17 who had used all inhalants within the past year was lower in 2007 (3.9 percent) than in 2003 (4.5 percent), in 2004 (4.6 percent), and in 2005 (4.5 percent). Among first-time users, the rate of use of nitrous oxide, or "whippets" – usually canisters of the propellants to create whipped cream – declined between 2002 and 2007 among males (40.2 percent to 20.2 percent) and females (22.3 percent to 21.2 percent).

However, an investigation in 2009 by the Bristol (Va.) Herald Courier reported that the records of 46 health care professionals in the area – including doctors, nurses, pharmacists and dentists – were "marred by substance abuse, and in some cases, criminal convictions."

The Herald Courier told the story of a Big Stone Gap, Va., dentist who "huffed nitrous oxide in the mid-1970s and quit only after a temporary loss of feeling in his hands." From there, the dentist descended into alcoholism and took Valium and Hydrocodone from his office, the newspaper said.

A grassroots drug-recovery group of Virginia dentists directed the man to a rehab program. The state board of dentistry got an anonymous call about his situation. Instead of disciplinary action, the board helped monitor his recovery. According to the newspaper, keeping addictions confidential is at the discretion of either a Department of Health Professions investigator or a licensing board.

A board of medicine official said the policy protected the public by "making sure the individual is identified and investigated, set for an evaluation and treatment, and continue with monitoring. They (the Virginia monitoring program) will not OK a doctor to go back into practice until he or she is believed to be safe."

That is a common practice. The Federation of State Physician Health Programs Inc. (FSPHP) evolved in 1990 from an initiative of the American Medical Association and individual state physician health programs that focus upon rehabilitation and monitoring of physicians with psychoactive substance abuse disorders as well as mental and physical illness. The nonprofit organization includes members from 42 state programs.

FSPHP serves as a resource for state programs; helps to establish monitoring standards; serves as an informational source; advocates for physicians and their health issues at local state and national levels; and helps states in their quest to protect the public. The organization promotes confidentiality for health care professionals who chose to address their substance problems and submit to rigorous monitoring of their progress.

A 2003 report in the Journal of the California Dental Association [Malamed and Clark] cited concerns about abuse of nitrous oxide by health care professionals. The authors said nitrous oxide causes euphoria and can include "sexual phenomena," including increased feelings of sexuality and arousal, and therefore has the potential for abuse. "This abuse is usually not as addictive as some drugs, but nonetheless can be a steppingstone to other drugs and can cause incapacitation of the affected person. Nitrous oxide should be given the same respect as all drugs."

The typical abuser of nitrous oxide is older and middle- or upper class, they said. If the abuser has an inhalation sedation unit available, it may have been altered to deliver a higher concentrate of gas, they said.

The authors noted there have been reports of sexual abuse of patients under anesthetics, including nitrous oxide. They noted there are three elements that put a practitioner at risk: treating a patient without an assistant in the operatory, high concentrations of nitrous oxide, and failure to titrate the patient to avoid extension of therapeutic sedation.

"Nitrous oxide should be employed with confidence. Employing simple guidelines will ensure there are no difficulties with sexual issues and the administrator of nitrous oxide," they said.

In medicine

Nitrous oxide has been used for anesthesia in dentistry since December 1844, when Horace Wells made the first dental operations with the gas in Hartford. Its debut as a generally accepted method came in 1863, when Gardner Quincy Colton introduced it more broadly at all the Colton Dental Association clinics. The first devices used in dentistry to administer the gas, known as nitrous oxide inhalers, were designed in a very simple way, with the gas stored and breathed through a breathing bag made of rubber cloth, without a scavenger system and flow meter, and with no addition of oxygen/air.

Today these simple and somewhat unreliable inhalers, of course, have been replaced by the more modern relative analgesia machine, which is an automated machine designed to deliver a precisely dosed and breath-actuated flow of nitrous oxide mixed with oxygen for the patient to inhale safely. The machine used in dentistry is designed as a more simplified version of the larger anesthetic machine used by hospitals, and it doesn't feature the additional anesthetic vaporizer and medical ventilator. The machine allows for a more simple design, because it only delivers a mixture of nitrous oxide and oxygen for the patient to inhale to depress the feeling of pain while keeping the patient in a conscious state.

The relative analgesia machine typically features a constant-supply flow meter, which allows the proportion of nitrous oxide and the combined gas flow rate to be individually adjusted. The gas is administered by dentists through a demand-valve inhaler over the nose, which will only release gas when the patient inhales through the nose. Because nitrous oxide is minimally metabolized in humans (with a rate of 0.004 percent), it retains its potency when exhaled into the room by the patient and can pose an intoxicating and prolonged exposure hazard to the clinic staff if the room is poorly ventilated. Where nitrous oxide is administered, a continuous-flow fresh-air ventilation system or nitrous scavenger system is used to prevent a waste-gas buildup.

Hospitals are administering nitrous oxide as one of the anesthetic drugs delivered by anesthetic machines. Nitrous oxide is a weak general anesthetic, and so is generally not used alone in general anesthesia. In general anesthesia it is used as a carrier gas in a 2:1 ratio with oxygen for more powerful general anesthetic, drugs such as sevoflurane or desflurane. It has a MAC (minimum alveolar concentration) of 105 percent and a blood gas partition coefficient of 0.46.

When nitrous oxide is inhaled as the only anesthetic drug, it is normally administered as a mixture with 30 percent gas and 70 percent oxygen.

Neuropharmacology

The pharmacological mechanism of action of N_2O in medicine is not fully known. However, it has been shown to directly modulate a broad range of ligand-gated ion channels, and this likely plays a major role in many of its effects. It moderately blocks NMDA and β_2-subunit-

containing nACh channels; weakly inhibits AMPA, kainate, $GABA_C$ and $5\text{-}HT_3$ receptors; and slightly potentiates $GABA_A$ and glycine receptors. It has also been shown to activate two-pore-domain K^+ channels. While N_2O affects quite a few ion channels, its anesthetic, hallucinogenic and euphoriant effects are likely caused predominantly or fully via inhibition of NMDAR-mediated currents. In addition to its effects on ion channels, N_2O may act to imitate nitric oxide (NO) in the central nervous system as well, and this may relate to its analgesic and anxiolytic properties.

Anxiolytic effect

In behavioral tests of anxiety, a low dose of N_2O is an effective anxiolytic, and this anti-anxiety effect is associated with enhanced activity of $GABA_A$ receptors as it is partially reversed by benzodiazepine receptor antagonists. Mirroring this, animals that have developed tolerance to the anxiolytic effects of benzodiazepines are partially tolerant to N_2O. Indeed, in humans given 30 percent N_2O, benzodiazepine receptor antagonists reduced the subjective reports of feeling "high," but did not alter psychomotor performance in human clinical studies.

Analgesic and anti-nociceptive effect

The analgesic effects of N_2O are linked to the interaction between the endogenous opioid system and the descending noradrenergic system. When animals are given morphine chronically, they develop tolerance to its pain-killing effects, and this also renders the animals tolerant to the analgesic effects of N_2O. Administration of antibodies that bind and block the activity of some endogenous opioids (not β-endorphin) also block the anti-nociceptive effects of N_2O. Drugs that inhibit the breakdown of endogenous opioids also potentiate the anti-nociceptive effects of N_2O. Several experiments have shown that opioid receptor antagonists applied directly to the brain block the anti-nociceptive effects of N_2O, but these drugs have no effect when injected into the spinal cord.

Conversely, α_2-adrenoceptor antagonists block the anti-nociceptive effects of N_2O when given directly to the spinal cord, but not when applied directly to the brain. Indeed, α_{2B}-adrenoceptor knockout mice or animals depleted in norepinephrine are nearly completely resistant to the anti-nociceptive effects of N_2O. It seems N_2O-induced release of endogenous opioids causes disinhibition of brain stem noradrenergic neurons, which release norepinephrine into the spinal cord and inhibit pain signaling. Exactly how N_2O causes the release of endogenous opioid peptides is still uncertain.

Euphoric effect

In rats, N_2O stimulates the mesolimbic reward pathway via inducing dopamine release and activating dopaminergic neurons in the ventral tegmental area and nucleus accumbens, presumably through antagonization of NMDA receptors localized in the system. This action has been implicated in its euphoric effects, and notably, appears to augment its analgesic properties as well.

However, it is remarkable that in mice, N_2O blocks amphetamine-induced and dopamine release in the nucleus accumbens and behavioral sensitization, abolishes the conditioned place preference (CPP) of cocaine and morphine, and does not produce reinforcing (or aversive) effects of its own. Studies on CPP of N_2O in rats is mixed, consisting of reinforcement, aversion and no change. In contrast, it is a positive reinforcer in squirrel monkeys, and is well known as a drug of abuse in humans. These discrepancies in response to N_2O may reflect specie variations or methodological differences. It is noteworthy that in human clinical studies, N_2O was found to produce mixed responses similarly to rats, reflecting high subjective individual variability.

Neurotoxicity

Similarly to other NMDA antagonists like ketamine, N_2O has been demonstrated to produce neurotoxicity in the form of Olney's lesions (damage to the posterior cingulate and retrosplenial cortices) in rodents upon prolonged (e.g., several hours) exposure. However, it also simultaneously exerts widespread neuroprotective effects via inhibiting glutamate-induced and it has been argued that on account of its very short duration under normal circumstances, N_2O may not share the neurotoxicity of other NMDA antagonists. Indeed, in rodents, short-term exposure results in only mild injury that is rapidly reversible, and permanent neuronal death only occurs after constant and sustained exposure.

Safety

The major safety hazards of nitrous oxide come from the fact that it is a compressed liquefied gas, an asphyxiation risk and a dissociative anesthetic. Exposure to nitrous oxide causes short-term decreases in mental performance, audiovisual ability and manual dexterity. Long-term exposure can cause vitamin B_{12} deficiency, numbness, reproductive side effects and other problems.

The National Institute for Occupational Safety and Health recommends that workers' exposure to nitrous oxide should be controlled during the administration of anesthetic gas in medical, dental and veterinary operators.

Chemical/physical

At room temperature (20 degrees C), the saturated vapor pressure is 58.5 bar, rising up to 72.45 bar at 36.4 degrees C – the critical temperature. The pressure curve is thus unusually sensitive to temperature. Liquid nitrous oxide acts as a good solvent for many organic compounds; liquid mixtures may form shock-sensitive explosives.

As with many strong oxidizers, contamination of parts with fuels have been implicated in rocketry accidents, where small quantities of nitrous/fuel mixtures explode due to "water hammer-like" effects (sometimes called "dieseling" – heating caused by adiabatic compression of gases that can reach decomposition temperatures). Some common building materials, such as stainless steel and aluminum, can act as fuels with strong oxidizers such as nitrous oxide, as can contaminants, which can ignite due to adiabatic compression.

There have also been accidents where nitrous oxide decomposition in plumbing has led to the explosion of large tanks.

Biological

Nitrous oxide inactivates the cobalamin form of vitamin B_{12} by oxidation. Symptoms of vitamin B_{12} deficiency, including sensory neuropathy and encephalopathy, can occur within days or weeks of exposure to nitrous oxide anesthesia in people with subclinical vitamin B_{12} deficiency. Symptoms are treated with high doses of vitamin B_{12}, but recovery can be slow and incomplete. People with normal vitamin B_{12} levels have stores to make the effects of nitrous oxide insignificant, unless exposure is repeated and prolonged (nitrous oxide abuse). Vitamin B_{12} levels should be checked in people with risk factors for vitamin B_{12} deficiency prior to using nitrous oxide anesthesia.

A study of workers and several experimental animal studies indicate that adverse reproductive effects for pregnant females may also result from chronic exposure to nitrous oxide.

Flammability

Nitrous oxide is a non-flammable gas at room temperature.

The National Fire Protection Association has not assigned a flammability rating to nitrous oxide:

- Flash point: Not applicable.
- Autoignition temperature: Not applicable.
- Flammable limits in air: Not applicable.
- Extinguishant: For small fires, use dry chemical or carbon dioxide. Use water spray, fog or standard foam to fight large fires involving nitrous oxide.

Fires involving nitrous oxide should be fought upwind from the maximum distance possible. Keep unnecessary people away; isolate the hazard area and deny entry. Isolate the area for ½-mile in all directions if a tank, rail car or tank truck is involved in the fire. For a massive fire in a cargo area, use unmanned hose holders or monitor nozzles; if this is impossible, withdraw from the area and let the fire burn. Emergency personnel should stay out of low areas and ventilate closed spaces before entering. Vapors are an explosion hazard indoors, outdoors or in sewers. Containers of nitrous oxide may explode in the heat of the fire and should be moved from the fire area if it is possible to do so safely. If this is not possible, cool fire-exposed containers from the sides with water until well after the fire is out. Stay away from the ends of containers. Firefighters should wear a full set of protective clothing and self-contained breathing apparatus when fighting fires involving nitrous oxide.

Environmental

Nitrous oxide is a greenhouse gas, accounting for about 6 percent of the heating effect of greenhouse gases in the atmosphere. According to 2006 data from the United States Environmental Protection Agency, industrial sources make up only about 20 percent of all anthropogenic sources, and include the production of nylon and the burning of fossil fuel in internal combustion engines. Human activity is thought to account for 30 percent; tropical soils and oceanic release account for 70 percent. However, a 2008 study by Nobel Laureate Paul Crutzen suggests that the amount of nitrous oxide release attributable to agricultural nitrate fertilizers has been seriously underestimated, most of which would presumably come under soil and oceanic release in the Environmental Protection Agency data. Nitrous oxide also causes ozone depletion. A recent study suggests that N_2O emission currently is the single most important ozone-depleting substance (ODS) emission and is expected to remain the largest throughout the 21st century.

Legality

In the United States, possession of nitrous oxide is legal under federal law and is not subject to DEA purview. It is, however, regulated by the Food and Drug Administration under the Food Drug and Cosmetics Act; prosecution is possible under its "misbranding" clauses, prohibiting the sale or distribution of nitrous oxide for the purpose of human consumption.

Many states have laws regulating the possession, sale and distribution of nitrous oxide. Such laws usually ban distribution to minors or limit the amount of nitrous oxide that may be sold without special license.

In the state of California, possession for recreational use is prohibited and qualifies as a misdemeanor.

In some countries, it is illegal to have nitrous oxide systems plumbed into an engine's intake manifold. These laws are ostensibly used to prevent street racing and to meet emission standards. Nitrous oxide is entirely legal to possess and inhale in the United Kingdom, although supplying it to others to inhale, especially minors, is more likely to end up with a prosecution under the Medicines Act.

In New Zealand, the Ministry of Health has warned that nitrous oxide is a prescription medicine, and its sale or possession without a prescription is an offense under the Medicines Act. This statement would seemingly prohibit all non-medicinal uses of the chemical, though it is implied that only recreational use will be legally targeted.

In India, for general anesthesia purposes, nitrous oxide is available as nitrous oxide IP. India's gas cylinder rules (1985) permit the transfer of gas from one cylinder to another for breathing purposes. This law benefits remote hospitals, which would otherwise suffer because of India's geographic immensity. Nitrous oxide IP is transferred from bulk cylinders (17,000 liters capacity gas) to smaller pin-indexed valve cylinders (1,800 liters of gas), which are then connected to the yoke assembly of Boyle's machines. Because India's Food and Drug Authority (FDA-India) rules state that transferring a drug from one container to another (refilling) is equivalent to manufacturing, anyone found doing so must possess a drug-manufacturing license.

Nitrous oxide in dental operatories

The Engineering Control Technology Branch (ECTB) of the Division of Physical Sciences and Engineering studies the aspects of health hazard prevention and control in the workplace. Nitrous oxide (N_2O) mixed with oxygen has been used in dentistry as an analgesic and as a sedative for more than 100 years. Today, more than 424,000 workers who practice dentistry (such as dentists, dental assistants and dental hygienists) in the United States are potentially exposed to N_2O.

In a technical report published in 1977, the National Institute for Occupational Safety and Health recommended controlling exposure limits of nitrous oxide waste to 25 parts per million parts (ppm) of air during dental surgery. The report presented methods for limiting the waste during administration, based on the technical feasibility of existing controls. Since publication of this technical report, data collected by NIOSH have shown occupational exposures as high as 300 ppm in hospital operating rooms and exposures higher than 1,000 ppm in dental operatories equipped with scavenging systems (properly operating scavenging systems have been shown to reduce N_2O concentrations by more than 70 percent). The scavenging systems use local exhaust ventilation to collect waste gases from anesthetic breathing systems and remove them from the workplace.

Effects of exposure to high concentrations

Animal studies have shown adverse reproductive effects in female rats exposed to airborne concentrations of N_2O. Data from these studies indicate that exposure to N_2O during gestation can produce adverse health effects in the offspring.

Several studies of workers have shown that occupational exposure to N_2O causes adverse effects such as reduced fertility, spontaneous abortions and neurologic, renal and liver disease. A recent study reported that female dental assistants exposed to unscavenged N_2O for five or more hours per week had a significant risk of reduced fertility compared with unexposed female dental assistants. The exposed assistants had a 59 percent decrease in probability of conception for any given menstrual cycle compared with the unexposed assistants. For dental assistants who used scavenging systems during N_2O administration, the probability of conception was not significantly different from that of the unexposed assistants. Because environmental exposures were not measured during these epidemiologic studies, no dose-effect relationship could be established.

Exposure to high concentrations of waste anesthetic gases – even for a short time – may cause the following health effects:

- Headache.
- Irritability.
- Fatigue.
- Nausea.
- Drowsiness.
- Difficulties with judgment and coordination.
- Liver and kidney disease.

Workers exposed

In 1983, the American Dental Association (ADA) reported that 35 percent of all dentists used N_2O to control pain and anxiety in their patients [ADA 1983]. The ADA 1991 Survey of Dental Practice indicated that 58 percent of dentists reported having N_2O anesthetic equipment, and 64 percent of those practitioners also reported having a scavenging system. The percentage of pediatric dentists using N_2O increased from 65 percent in 1980 to 88 percent in 1988.

Occupational exposure limits

The Occupational Safety and Health Administration (OSHA) does not currently have a standard for N_2O.

The NIOSH recommended exposure limit (REL) for N_2O is 25 ppm as a time-weighted average (TWA) during the period of anesthetic administration [NIOSH 1977b]. This REL is intended to prevent decreases in mental performance, audiovisual ability and manual dexterity during exposures to N_2O. A recommended exposure limit to prevent adverse reproductive effects cannot be established until more data are available.

The American Conference of Governmental Industrial Hygienists' (ACGIH) threshold limit value (TLV) for N_2O is 50 ppm as an eight-hour time-weighted average [ACGIH 1993]. The 1991 Documentation of the Threshold Limit Values and Biological Exposure Indices states that "control to this level should prevent embryo-fetal toxicity in humans and significant decrements in human psychomotor and cognitive functions or other adverse health effects in exposed personnel" [ACGIH 1991].

Medical surveillance

OSHA is currently developing requirements for medical surveillance. When these requirements are promulgated, readers should refer to them for additional information and to determine whether employers whose employees are exposed to nitrous oxide are required to implement medical surveillance procedures.

Medical screening

Workers who may be exposed to chemical hazards should be monitored in a systematic program of medical surveillance that is intended to prevent occupational injury and disease. The program should include education of employers and workers about work-related hazards, early detection of adverse health effects and referral of workers for diagnosis and treatment. The occurrence of disease or other work-related adverse health effects should prompt immediate evaluation of primary preventive measures (e.g., industrial hygiene monitoring, engineering controls, and personal protective equipment). A medical surveillance program is intended to supplement, not replace, such measures. To detect and control work-related health effects, medical evaluations should be performed (1) before job placement, (2) periodically during the term of employment, and (3) at the time of job transfer or termination.

Preplacement medical evaluation

Before a worker is placed in a job with a potential for exposure to nitrous oxide, a licensed health care professional should evaluate and document the worker's baseline health status with thorough medical, environmental and occupational histories, a physical examination, and physiologic and laboratory tests appropriate for the anticipated occupational risks. These should concentrate on the function and integrity of the respiratory, reproductive, central nervous and hematological systems. Medical surveillance for respiratory disease should be conducted using the principles and methods recommended by the American Thoracic Society. A preplacement medical evaluation is recommended to assess medical conditions that may be aggravated or may result in increased risk when a worker is exposed to nitrous

oxide at or below the prescribed exposure limit. The health care professional should consider the probable frequency, intensity and duration of exposure as well as the nature and degree of any applicable medical condition. Such conditions (which should not be regarded as absolute contraindications to job placement) include a history and other findings consistent with diseases of the respiratory, reproductive, central nervous or hematological systems.

Periodic medical evaluations

Occupational health interviews and physical examinations should be performed at regular intervals during the employment period, as mandated by any applicable federal, state or local standard. Where no standard exists and the hazard is minimal, evaluations should be conducted every three to five years or as frequently as recommended by an experienced occupational health physician. Additional examinations may be necessary if a worker develops symptoms attributable to nitrous oxide exposure. The interviews, examinations and medical screening tests should focus on identifying the adverse effects of nitrous oxide on the respiratory, reproductive, central nervous or hematological systems. Current health status should be compared with the baseline health status of the individual worker or with expected values for a suitable reference population.

Termination medical evaluations

The medical, environmental and occupational history interviews; the physical examination; and selected physiologic or laboratory tests that were conducted at the time of placement should be repeated at the time of job transfer or termination to determine the worker's medical status at the end of his or her employment. Any changes in the worker's health status should be compared with those expected for a suitable reference population.

Biological monitoring

Biological monitoring involves sampling and analyzing body tissues or fluids to provide an index of exposure to a toxic substance or metabolite. No biological monitoring test acceptable for routine use has yet been developed for nitrous oxide.

Workplace monitoring and measurement

Determination of a worker's exposure to airborne nitrous oxide can be made using one of the following techniques:

- A Landauer Passive Dosimeter badge, which can be used for a minimum sampling duration of one hour (maximum duration 40 hours). Analysis is performed by the manufacturer of the badge as described in the OSHA Computerized Information System.
- An ambient air or bag sample with a minimum collection volume of two spectrophotometer cell volumes. Analysis is conducted using a long-path-length portable infrared spectrophotometer as described in NIOSH Method No. 6600.

Personal hygiene procedures

If liquid nitrous oxide contacts the skin, workers should flush the affected areas immediately with tepid water to reduce the likelihood of frostbite.

A large population of health care workers is potentially exposed to N_2O, and NIOSH has documented cases in which exposures substantially exceed existing recommended exposure limits. NIOSH has concluded that exposure to N_2O causes decreases in mental performance, audiovisual ability and manual dexterity. Data from animal studies demonstrate that exposure to N_2O may cause adverse reproductive effects. Studies of workers exposed to N_2O have reported adverse health effects such as reduced fertility, spontaneous abortion, and neurological, renal, and liver disease. The recommendations in a 1994 NIOSH alert should therefore be followed to minimize worker exposures.

Recommendations

Engineering controls, work practices and respirators (when necessary) should be used to minimize the exposure of workers to N_2O. Employers should ensure that their workers are adequately protected from N_2O exposure by taking the following steps:

- Monitor airborne concentrations of N_2O.
- Implement appropriate engineering controls, work practices and maintenance procedures.
- Institute a worker education program that:
    - Describes standard operating procedures for all tasks that may expose workers to N_2O.
    - Informs workers about proper work practices, controls, equipment and protective gear that should be used when working with N_2O.
- Use the guidelines in the following section to minimize worker exposures to N_2O.

GUIDELINES FOR MINIMIZING WORKER EXPOSURES

Exposure monitoring

Exposure monitoring should be the first step in developing work practices and worker education programs, because measurements of N_2O are needed to determine the type and extent of controls that are necessary. Follow the guidelines below to minimize worker exposures:

- Monitor for N_2O when the anesthetic equipment is installed and every three months thereafter. Include the following types of monitoring:
    - Leak testing of equipment.
    - Monitoring of air in the worker's personal breathing zone.
    - Environmental (room air) monitoring.
- Prepare a written monitoring and maintenance plan for each facility that uses N_2O . This plan should be developed by knowledgeable persons who consider the equipment manufacturers' recommendations, frequency of use and other circumstances that might affect the equipment.
- Perform air monitoring by gasbag sampling or real-time sampling.

- When real-time sampling is conducted to obtain personal exposure data, attach the sampling train to the lapel of the worker on the side closest to the patient; N_2O concentrations in this location are most representative of those in the worker's breathing zone. Diffusive samplers (referred to as passive dosimeters) are commercially available and may be useful as initial indicators of exposures.

Engineering controls and maintenance procedures

The following engineering controls and maintenance procedures have been shown to be feasible and effective in reducing exposure to N_2O during anesthetic administration.

Anesthetic delivery. Excessive exposure to N_2O may occur as a result of leaks from the anesthetic delivery system during administration. The rubber and plastic components of the anesthetic equipment are potential sources of N_2O leakage because they may be degraded by the N_2O and the oxygen as well as by repeated sterilization.

Take the following steps to control N_2O exposure from anesthetic delivery systems:

- Use connection ports with different-diameter hoses for N_2O and O2 to reduce the possibility of incorrectly connecting the gas delivery and scavenging hoses.
- Check all rubber hoses, connections, tubing and breathing bags daily and replace them when damaged or when recommended by the manufacturer.
- Following visual inspection, perform leak testing of the equipment and connections by using a soap solution to check for bubbles at high-pressure connections. For a more thorough inspection of all connectors, use a portable infrared spectrophotometer (such as a Miran 1A or 1B) calibrated for N_2O detection.
- Check both high- and low-pressure connections (such as O-rings) regularly, as they may become worn; replace them periodically, according to the manufacturer's recommendations.
- Evaluate the N_2O and oxygen mixing system for leaks when it is first installed and periodically thereafter, according to the manufacturer's recommendations.
- Ensure that gas cylinders are safely handled, used and stored as specified by the National Research Council and as required by OSHA Federal Code Rule Title 29, 1910.101.
    - Sec. 1910.101 Compressed gases (general requirements).
    - (a) Inspection of compressed gas cylinders. Each employer shall determine that compressed gas cylinders under his control are in a safe condition to the extent that this can be determined by visual inspection. Visual and other inspections shall be conducted as prescribed in the Hazardous Materials Regulations of the Department of Transportation (49 CFR parts 171-179 and 14 CFR part 103). Where those regulations are not applicable, visual and other inspections shall be conducted in accordance with Compressed Gas Association Pamphlets C-6-1968 and C-8-1962, which is incorporated by reference as specified in Sec. 1910.6.

Scavenging systems. Control of N_2O at the scavenging mask is the next priority after control of N_2O leakage from the anesthetic equipment. Leakage from the scavenging mask can be one of the most significant sources of N_2O exposure because the breathing zone of a dentist or dental assistant is within inches of the mask. NIOSH research has reported breathing-zone concentrations of N_2O above 1,000 ppm.

Take the following steps to control N_2O exposure from anesthetic scavenging systems:

- Supply scavenging masks in a variety of sizes so that the mask always fits comfortably and securely over the patient's nose or face.
- Use an automatic interlock system to assure that the N_2O cannot be turned on unless the scavenging system is also activated. N_2O should never be used without a properly operating scavenging system.
- Make sure that the scavenging system exhaust rates (flow rates) are approximately 45 liters per minute (L/min) to minimize leakage of N_2O. Flow rates of less than 40 L/min may result in significant leakage around the mask. Monitor the flow rate with a flow meter that is:
    - Validated to measure airflow within 5 percent of actual airflow.
    - Permanently connected to the scavenging system vacuum line.
    - Positioned so that it is always visible to the operator.
- Maintain the flow meter by cleaning and recalibrating it according to the manufacturer's recommendations.
- Use scavenging vacuum pumps that are powerful enough to maintain a scavenging flow rate of at least 45 L/min at each nasal mask, regardless of the number of scavenging units in use at one time.
- Vent N_2O from all scavenging vacuum pumps to the outside of the building away from fresh air intakes, windows or walkways. Scavenging system exhaust should not be vented into a recirculating ventilation system.

Room ventilation. Take the following steps to assure that the ventilation system effectively removes waste N_2O:

- If concentrations of N_2O are above 25 ppm in work areas, increase the airflow into the room or increase the percentage of outside air to allow for more air mixing and further dilution of the anesthetic gas. Maintain a balanced air supply and exhaust system so that N_2O does not contaminate adjacent areas.
- If concentrations of N_2O are still above 25 ppm, use supplementary local ventilation in conjunction with a scavenging system to reduce N_2O exposure in the operatory. The effectiveness of this ventilation depends on its location with respect to the patient and the airflow rates. Do not work between the patient and the exhaust duct, where contaminated air would be drawn through the worker's breathing zone.
- Dilute N_2O and remove contaminated air from the work area by placing fresh-air vents in the ceiling; direct the supply of fresh air toward the floor and the operating area. Place exhaust-air vents at or near the floor.

Work practices

Use the following work practices to control N_2O exposures:

- Inspect the anesthetic delivery systems and all connections before starting anesthetic gas administration. Make sure that breathing bags, hoses and clamps are in place before turning on the anesthetic machine.
- Connect the scavenging mask properly to the gas delivery hose and the vacuum system.
- Do not turn on the machine delivering N_2O until:
    - The vacuum system scavenging unit is operating at the recommended flow rate of 45 L/min.
    - The scavenging mask is secured over the patient's nose or face.
- Fasten the mask according to the manufacturer's instructions to prevent leaks around the mask during gas delivery.
- Do not fill the breathing bag to capacity with N_2O; an overinflated bag can cause excessive leakage from the scavenging mask. The breathing bag should collapse and expand as the patient breaths. This bag activity shows that the proper amounts of N_2O and air are being delivered to the patient.

- Flush the system of N_2O after surgery by administering oxygen to the patient through the anesthetic equipment for at least five minutes before disconnecting the gas delivery system.
- Encourage patients to minimize talking and mouth-breathing during dental surgery. When mouth-breathing is apparent, avoid the patient's breathing zone to the extent possible.

Respiratory protection

Workers should wear respiratory protection when N_2O concentrations are not consistently below 25 ppm; however, practical considerations may prevent them from wearing such protection. Therefore, it is essential that employers use the engineering controls and work practices described in a 1994 NIOSH alert to reduce N_2O concentrations below 25 ppm.

When N_2O concentrations are not consistently below 25 ppm, workers should take the following steps to protect themselves:

- Wear air-supplied respirators. Air-purifying respirators (that is, respirators that remove N_2O from the air rather than supply air from a clean source) should not be used because respirator filters do not efficiently remove N_2O.
- As specified by the NIOSH respirator standards, the minimum level of protection for an air-supplied respirator is provided by a half-mask respirator operated in the demand or continuous-flow mode. More protective air-supplied respirators are described in the NIOSH respirator decision logic.
- When respirators are used, the employer must establish a comprehensive respiratory protection program as outlined in the NIOSH Guide to Industrial Respiratory Protection [NIOSH 1987a] and as required by the OSHA respiratory protection standard [29 CFR 1910.134]. Important elements of this standard are:
  - An evaluation of the worker's ability to perform the work while wearing a respirator.
  - Regular training of personnel.
  - Periodic environmental monitoring.
  - Respirator fit testing.
  - Maintenance, inspection, cleaning and storage.
  - Selection of proper NIOSH-approved respirators.
- The respiratory protection program should be evaluated regularly by the employer.

Signs and symptoms of exposure

- **Acute exposure**: The signs and symptoms of acute exposure to nitrous oxide include dizziness, difficult breathing, headache, nausea, fatigue and irritability. Acute exposure to nitrous oxide concentrations of 400,000 to 800,000 ppm may cause loss of consciousness.
- **Chronic exposure**: The signs or symptoms of chronic overexposure to nitrous oxide may include tingling, numbness, difficulty in concentrating, interference with gait, and reproductive effects.

Storage

Nitrous oxide should be stored in a cool, dry, well-ventilated area in tightly sealed containers that are labeled in accordance with OSHA's Hazard Communication Standard [29 Code of Federal Regulations (CFR)1910.1200]. Containers of nitrous oxide should be protected from physical damage and should be stored separately from cylinders containing oxygen. Nitrous oxide should also be stored separately from aluminum, boron, hydrazine, lithium hydride, phenyllithium, phosphine, sodium, tungsten carbide, hydrogen, hydrogen sulfide, organic peroxides, ammonia and carbon monoxide.

Spills and leaks

In the event of a spill or leak involving nitrous oxide (liquid or gas), persons not wearing protective equipment and clothing should be restricted from contaminated areas until cleanup has been completed. The following steps should be undertaken following a spill or leak:

- Do not touch the spilled material; stop the leak if it is possible to do so without risk.
- Use water spray to protect persons attempting to stop the leak.
- Notify safety personnel of large spills or leaks.
- Minimize all sources of ignition because a fire may cause nitrous oxide to accelerate the burning of other combustibles; keep combustible materials (wood, paper, oil, etc.) away from the spilled material.
- Isolate the area until the gas has dispersed.

Special requirements

United States Environmental Protection Agency (EPA) requirements for emergency planning, reportable quantities of hazardous releases, community right-to-know and hazardous waste management may change over time. Users are therefore advised to determine periodically whether new information is available.

Emergency planning requirements

Nitrous oxide is not subject to EPA emergency planning requirements under the Superfund Amendments and Reauthorization Act (SARA) (Title III) in 42 CFR 11022.

Reportable quantity requirements for hazardous releases

Employers are not required by the emergency release notification provisions in 40 CFR Part 355.40 to notify the National Response Center of an accidental release of nitrous oxide; there is no reportable quantity for this substance.

Community right-to-know requirements

Employers are not required by EPA in 40 CFR Part 372.30 to submit a Toxic Chemical Release Inventory form (Form R) to EPA reporting the amount of nitrous oxide emitted or released from their facility annually.

Hazardous waste management requirements

EPA considers a waste to be hazardous if it exhibits any of the following characteristics: ignitability, corrosivity, reactivity or toxicity as defined in 40 CFR 261.21-261.24. Under the Resource Conservation and Recovery Act (RCRA) [40 USC 6901 et seq.], EPA has specifically listed many chemical wastes as hazardous. Although nitrous oxide is not specifically listed as a hazardous waste under RCRA, EPA requires employers to treat waste as hazardous if it exhibits any of the characteristics discussed above. Providing detailed information about the removal and disposal of specific chemicals is beyond the scope of this guideline. The U.S. Department of Transportation, EPA, and state and local regulations should be followed to ensure that removal, transport and disposal of this substance are conducted in accordance with existing regulations. To be certain that chemical waste disposal meets EPA regulatory requirements, employers should address any questions to the RCRA hotline at (703) 412-9810 (in the Washington, D.C. area) or toll-free at 800-424-9346 (outside Washington, D.C.). In addition, relevant state and local authorities should be contacted for information on any requirements they may have for the waste removal and disposal of this substance.

RESPIRATORY PROTECTION

Conditions for respirator use

Good industrial hygiene practice requires that engineering controls be used where feasible to reduce workplace concentrations of hazardous materials to the prescribed exposure limit. However, some situations may require the use of respirators to control exposure. Respirators must be worn if the ambient concentration of nitrous oxide exceeds prescribed exposure limits. Respirators may be used (1) before engineering controls have been installed, (2) during work operations such as maintenance or repair activities that involve unknown exposures, (3) during operations that require entry into tanks or closed vessels, and (4) during emergencies. Workers should only use respirators that have been approved by NIOSH and the Mine Safety and Health Administration (MSHA).

Respiratory protection program

Employers should institute a complete respiratory protection program that, at a minimum, complies with the requirements of OSHA's Respiratory Protection Standard [29 CFR 1910.134]. Such a program must include respirator selection, an evaluation of the worker's ability to perform the work while wearing a respirator, the regular training of personnel, respirator fit testing, periodic workplace monitoring, and regular respirator maintenance, inspection and cleaning. The implementation of an adequate respiratory protection program (including selection of the correct respirator) requires that a knowledgeable person be in charge of the program and that the program be evaluated regularly. For additional information on the selection and use of respirators and on the medical screening of respirator users, consult the latest edition of the NIOSH Respirator Decision Logic [NIOSH 1987b] and the NIOSH Guide to Industrial Respiratory Protection [NIOSH 1987a].

Personal protective equipment

Workers should use appropriate personal protective clothing and equipment that must be carefully selected, used and maintained to be effective in preventing skin contact with liquid nitrous oxide. The selection of the appropriate personal protective equipment (PPE) (e.g., gloves, sleeves, encapsulating suits) should be based on the extent of the worker's potential exposure to liquid nitrous oxide and the PPE material's ability to protect workers from frostbite. There are no published reports on the resistance of various materials to permeation by liquid nitrous oxide.

To evaluate the use of PPE materials with liquid nitrous oxide, users should consult the best available performance data and manufacturers' recommendations. Significant differences have been demonstrated in the chemical resistance of generically similar PPE materials (e.g., butyl) produced by different manufacturers. In addition, the chemical resistance of a mixture may be significantly different from that of any of its neat components.

Any chemical-resistant clothing that is used should be periodically evaluated to determine its effectiveness in preventing dermal contact.

Safety showers and eye wash stations should be located close to operations that involve nitrous oxide.

Splash-proof chemical safety goggles or face shields (20 to 30 cm long, minimum) should be worn during any operation in which a solvent, caustic or other toxic substance may be splashed into the eyes.

In addition to the possible need for wearing protective outer apparel (e.g., aprons, encapsulating suits), workers should wear work uniforms, coveralls or similar full-body coverings that are laundered each day. Employers should provide lockers or other closed areas to store work and street clothing separately. Employers should collect work clothing at the end of each work shift and provide for its laundering. Laundry personnel should be informed about the potential hazards of handling contaminated clothing and instructed about measures to minimize their health risk.

Protective clothing should be kept free of oil and grease and should be inspected and maintained regularly to preserve its effectiveness.

Protective clothing may interfere with the body's heat dissipation, especially during hot weather or during work in hot or poorly ventilated work environments.

More on personal protective equipment

- Personal protective equipment should not be used as a substitute for engineering, work practice and/or administrative controls in anesthetizing locations and post anesthesia care units (PACUs). In fact, exposure to waste gases is not effectively reduced by gloves, goggles and surgical masks. A negative-pressure, high-efficiency particulate air (HEPA) filter used for infection control is also not appropriate to protect workers from waste gases. Air-supplied respirators with self-contained air source are ideal for eliminating exposure but are not a practical alternative.
- During cleanup and containment of spills of liquid anesthetic agents, personal protective equipment should be used in conjunction with engineering, work practice and administrative controls to provide for employee safety and health. Gloves, goggles, face shields and chemical protective clothing (CPC) are recommended to ensure worker protection. Respirators, where

needed, should be selected based on the anticipated contamination level.

- When selecting gloves and chemical protective clothing, some of the factors to be considered include material chemical resistance, physical strength and durability, and overall product integrity. Permeation, penetration and degradation data should be consulted if available. Among the most effective types of gloves and body protection are those made from Viton®, neoprene and nitrile. Polyvinyl alcohol (PVA) is also effective, but it should not be exposed to water or aqueous solutions.
- When the gloves and the CPC being used have not been tested under the expected conditions, they may fail to provide adequate protection. In this situation, the wearer should observe the gloves and the chemical protective clothing during use and treat any noticeable change (e.g., color, stiffness, chemical odor inside) as a failure until proved otherwise by testing. If the work must continue, new CPC should be worn for a shorter exposure time, or be of a different generic material. The same thickness of a generic material such as neoprene or nitrile supplied by different manufacturers may provide significantly different levels of protection because of variations in the manufacturing processes or in the raw materials and additives used in processing.
- Professional judgment must be used in determining the type of respiratory protection to be worn. For example, where spills of halogenated anesthetic agents are small, exposure time brief and sufficient ventilation present, NIOSH-approved chemical cartridge respirators for organic vapors should provide adequate protection during cleanup activities.
- Where large spills occur and there is insufficient ventilation to adequately reduce airborne levels of the halogenated agent, respirators designed for increased respiratory protection should be used. The following respirators, to be selected for large spills, are ranked in order from minimum to maximum respiratory protection:
  - Any type C supplied-air respirator with a full facepiece, helmet or hood operated in continuous-flow mode.
  - Any type C supplied-air respirator with a full facepiece operated in pressure-demand or other positive-pressure mode.
  - Any self-contained breathing apparatus with a full facepiece operated in pressure-demand or other positive-pressure mode.

Workplace exposures

Workplace exposures to anesthetic gases occur in hospital-based and stand-alone operating rooms, recovery rooms, dental operatories and veterinary facilities. Engineering, work practice and administrative controls that help reduce these exposures in all anesthetizing locations are identified and discussed. Sources of leaks in anesthesia equipment systems, components, and accessories are identified, and appropriate methods are described that limit excessive leaks.

Inhaled anesthetic agents include two different classes of chemicals: nitrous oxide and halogenated agents. Halogenated agents currently in use include halothane (Fluothane®), enflurane (Ethrane®), isoflurane (Forane®), desflurane (Suprane®), and sevoflurane (Ultane®). Methoxyflurane (Penthrane®), once in general use, is now only infrequently used, primarily in veterinary procedures. At present, OSHA has no permissible exposure limits regulating these agents.

The basic anesthesia machine

An anesthesia machine is an assembly of various components and devices that include medical gas cylinders in machine hanger yokes, pressure regulating and measuring devices, valves, flow controllers, flow meters, vaporizers, CO_2 absorber canisters, and breathing circuit assembly. The basic two-gas anesthesia machine has more than 700 individual components.

The anesthesia machine is a basic tool of the anesthesiologist/anesthetist and serves as the primary work station. It allows the anesthesia provider to select and mix measured flows of gases, to vaporize controlled amounts of liquid anesthetic agents, and thereby to administer safely controlled concentrations of oxygen and anesthetic gases and vapors to the patient via a breathing circuit. The anesthesia machine also provides a working surface for placement of drugs and devices for immediate access and drawers for storage of small equipment, drugs, supplies and equipment instruction manuals. Finally, the machine serves as a frame and source of pneumatic and electric power for various accessories such as a ventilator, and monitors that observe or record vital patient functions or that are critical to the safe administration of anesthesia.

Gas flow in the anesthesia machine and breathing system

The internal piping of a basic two-gas anesthesia machine is shown in **Figure 1** (located at end of this chapter). The machine has many connections and potential sites for leaks. Both oxygen and N_2O may be supplied from two sources (**Figure 2,** located at end of this chapter): a pipeline supply source (central piping system from bulk storage) and a compressed gas cylinder supply source. In hospitals, the pipeline supply source is the primary gas source for the anesthesia machine. Pipeline-supplied gases are delivered through wall outlets at a pressure of 50-55 psig through diameter indexed safety system (DISS) fittings or through quick-connect couplings that are gas-specific within each manufacturer's patented system.

Because pipeline systems can fail and because the machines may be used in locations where piped gases are not available, anesthesia machines are fitted with reserve cylinders of oxygen and N_2O. The oxygen cylinder source is regulated from approximately 2,200 psig in the tanks to approximately 45 psig in the machine high-pressure system, and the N_2O cylinder source is regulated from 745 psig in the tanks to approximately 45 psig in the machine high-pressure system.

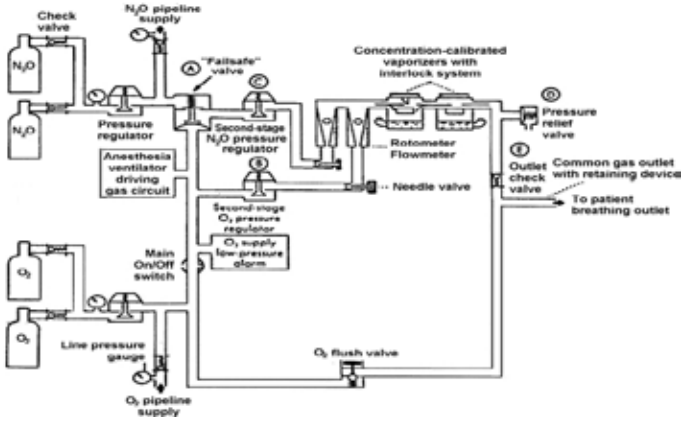


Figure 1 – The flow arrangement of a basic two-gas anesthesia machine. A, The fail-safe valve in Ohmeda machines is termed a pressure sensor shut-off valve; in Dräger machines it is the oxygen failure protection device (OFPD). B, Second-stage oxygen pressure regulator is used in Ohmeda (but not Dräger Narkomed) machines. C, Second-stage nitrous oxide pressure regulator is used in Ohmeda Modulus machines having the Link 25 Proportion Limiting System;

not used in Dräger machines. D, Pressure relief valve used in certain Ohmeda mchines; not used in Dräger machines. E, Outlet check valve used in Ohmeda machines except Modulus II Plus and Modulus CD models; not used in Dräger machines. The oxygen take-off for the anesthesia ventilator driving gas circuit is downstream of the main on/off switch in Dräger machines, as shown here. In Ohmeda machines, the take-off is upstream of the main on/off switch. (Adapted from Check-out: a guide for preoperative inspection of an anesthesia machine, ASA, 1987. Reproduced by permission of the American Society of Anesthesiologists, 520 N. Northwest Highway, Park Ridge, Ill.)

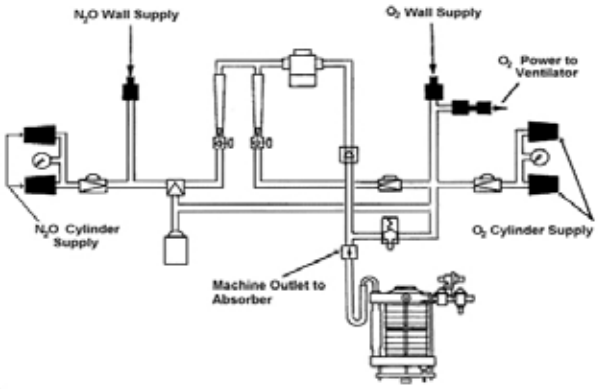


Figure 2 – The supply of nitrous oxide and oxygen may come from two sources: the wall (pipeline) supply and the reserve cylinder supply. (Reproduced by permission of Datex·Ohmeda, Madison, Wisconsin). Compressed gas cylinders of oxygen, N_2O, and other medical gases are attached to the anesthesia machine through the hanger yoke assembly. Each hanger yoke is equipped with the pin index safety system, a safeguard introduced to eliminate cylinder interchanging and the possibility of accidentally placing the incorrect gas tank in a yoke designed for another gas tank.

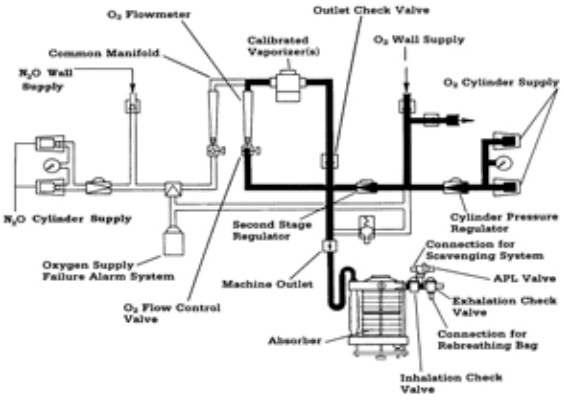
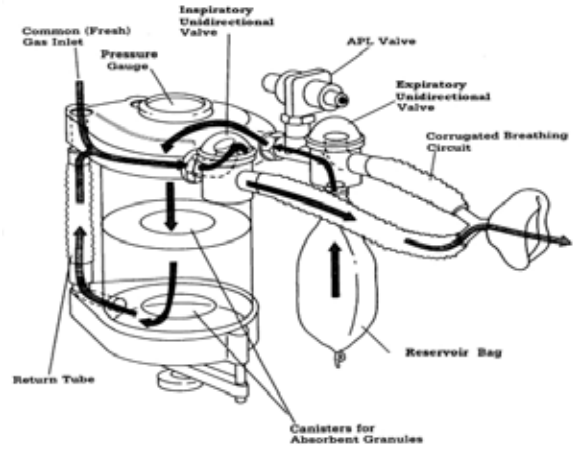
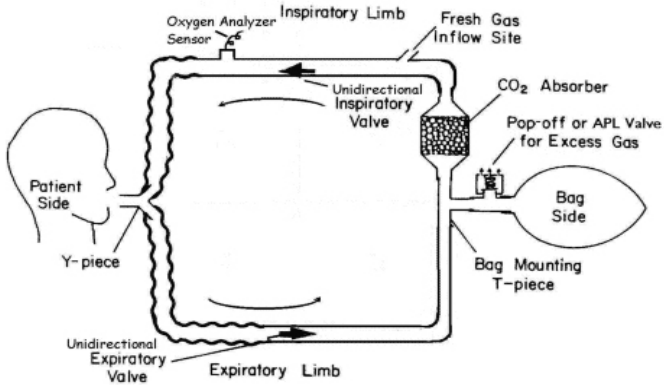


Figure 3 – shows the oxygen pathway through the flow meter, the agent vaporizer, and the machine piping, and into the breathing circuit. Oxygen from the wall outlet or cylinder pressurizes the anesthesia delivery system. Compressed oxygen provides the needed energy for a pneumatically powered ventilator, if used, and it supplies the oxygen flush valve used to supplement oxygen flow to the breathing circuit. Oxygen also "powers" an in-line pressure-sensor shutoff valve ("fail-safe" valve) for other gases to prevent their administration if the O_2 supply pressure in the O_2 high-pressure system falls below a threshold value.

Oxygen and N_2O flow from their supply sources via their flow control valves, flow meters and common manifold to the concentration-calibrated vaporizer and then via the machine common gas outlet to the breathing system. The high pressure system of the anesthesia machine comprises those components from the compressed gas supply source to the gas (O_2 and N_2O) flow control valves. The low pressure system of the anesthesia machine comprises those components downstream of the gas flow control valves.

Once the flows of oxygen, N_2O, and other medical gases (if used) are turned on at their flow control valves, the gas mixture flows into the common manifold and through a concentration-calibrated agent-specific vaporizer where a potent inhaled volatile anesthetic agent is added. The mixture of gases and vaporized anesthetic agent then exits the anesthesia machine low pressure system through the common gas outlet and flows to the breathing system.





The circle system shown in **Figure 4** is the breathing system most commonly used in operating rooms (ORs). It is so named because its components are arranged in a circular manner. The essential components of a circle breathing system **(Figure 5)** include a site for inflow of fresh gas (common [fresh] gas inlet), a carbon dioxide absorber canister (containing soda lime or barium hydroxide lime) where exhaled carbon dioxide is absorbed; a reservoir bag; inspiratory and expiratory unidirectional valves; flexible corrugated breathing tubing; an adjustable pressure-limiting (APL) or "pop-off" valve for venting excess gas; and a Y piece that connects to a face mask, tracheal tube, laryngeal mask airway (LMA) or other airway management device.

Once inside the breathing system, the mixture of gases and vapors flows to the breathing system's inspiratory unidirectional valve, then on toward the patient. Exhaled gases pass through the expiratory unidirectional valve and enter the reservoir bag. When the bag is full, excess gas flows through the APL (or pop-off) valve and into the scavenging system that removes the waste gases. On the next inspiration, gas from the reservoir bag passes through the carbon dioxide absorber prior to joining the fresh gas from the machine on its way to the patient. The general use of fresh gas flow rates into anesthetic systems in excess of those required to compensate for uptake, metabolism, leaks, or removal of exhaled carbon dioxide results in variable volumes of anesthetic gases and vapors exiting the breathing system through the APL valve.

When an anesthesia ventilator is used, the ventilator bellows functionally replaces the circle system reservoir bag and becomes a part of the breathing circuit. The APL valve in the breathing circuit is either closed or excluded from the circuit using a manual ("bag")/automatic (ventilator) circuit selector switch. The ventilator incorporates a pressure-relief valve that permits release of excess anesthetic gases from the circuit at end-exhalation. These gases should also be scavenged.

Sources of leaks within the anesthesia machine and breathing system

No anesthesia machine system is totally leak-free (Emergency Care Research Institute 1991). Leakage may originate from both the high-pressure and low-pressure systems of the anesthesia or analgesia machine.

The high-pressure system consists of all piping and parts of the machine that receive gas at cylinder or pipeline supply pressure. It extends from the high-pressure gas supply (i.e., wall supply or gas cylinder) to the flow control valves. Leaks may occur from the high-pressure connections where the supply hose connects to the wall outlet or gas cylinder and where it connects to the machine inlet. Therefore, gas-supply hoses should be positioned to prevent strain on the fittings (ASTM Standard F1161-88; Dorsch and Dorsch 1994) and constructed from supply-hose materials designed for high-pressure gas flow and minimal kinking (Bowie and Huffman 1985). High-pressure leakage may also occur within the anesthesia machine itself. Other potential sources of leaks include quick-connect fittings, cylinder valves, absent or worn gaskets, missing or worn yoke plugs in a dual yoke assembly, and worn hoses.

The low-pressure system of the anesthesia machine (in which the pressure is slightly above atmospheric) consists of components downstream of the flow-control valves. It therefore includes the flow meter tubes, vaporizers, common gas outlet and breathing circuit, (i.e., from the common gas outlet to the patient). Low-pressure system leaks may occur from the connections and components anywhere between the anesthesia gas flow control valves and the airway. This leakage may occur from loose-fitting connections, defective and worn seals and gaskets, worn or defective breathing bags, hoses, and tubing, loosely assembled or deformed slip joints and threaded connections, and the moisture drainage port of the CO_2 absorber, which may be in the "open" position.

Low-pressure system leaks also may occur at the gas analysis sensor (i.e., circuit oxygen analyzer) and gas sampling site(s), face mask, the tracheal tube (especially in pediatric patients where a leak is required around the uncuffed tracheal tube), laryngeal mask airway (over the larynx), and connection points for accessory devices such as a humidifier, temperature probe, or positive end-expiratory pressure (PEEP) valve. Inappropriate installation of a calibrated vaporizer(s) or misalignment of a vaporizer on its manifold can also contribute to anesthetic gas leakage.

Minute absorbent particles that may have been spilled on the rubber seal around the absorber canister(s) may also prevent a gas-tight seal when the canister(s) in the carbon dioxide absorber is (are) reassembled). The exhaust from a sidestream sampling respiratory gas analyzer and/or capnograph should also be connected to the waste gas scavenging system because the analyzed gas sample may contain N_2O or halogenated vapors.

Checking anesthesia machines

Prior to induction of anesthesia, the anesthesia machine and its components/accessories should be made ready for use. All parts of the machine should be in good working order with all accessory equipment and necessary supplies on hand. The waste gas disposal system should be connected, hoses visually inspected for obstructions or kinks, and proper operation determined. Similarly, the anesthesia breathing system should be tested to verify that it can maintain positive pressure. Leaks should be identified and corrected before the system is used. The ability of the anesthesia system to maintain constant pressure is tested not only for the safety of the patient dependent on a generated positive pressure ventilation but also to test for leaks and escape of anesthetic gases, which may expose health-care personnel to waste anesthetic gases.

General workplace controls

Occupational exposures can be controlled by the application of a number of well-known principles, including engineering and work practice controls, administrative controls, personal protective equipment and monitoring. These principles may be applied at or near the hazard source, to the general workplace environment, or at the point of occupational exposure to individuals. Controls applied at the source of the hazard, including engineering and work practice controls, are generally the preferred and most effective means of control. In anesthetizing locations and PACUs, where employees are at risk of exposure to waste anesthetic gases, exposure may be controlled by some or all of the following: (1) effective anesthetic gas scavenging systems that remove excess anesthetic gas at the point of origin; (2) effective general or dilution ventilation; (3) good work practices on the part of the health care workers, including the proper use of controls; (4) proper maintenance of equipment to prevent leaks; and (5) periodic personnel exposure and environmental monitoring to determine the effectiveness of the overall waste anesthetic gas control program.

The following is a general discussion of engineering controls, work practices, administrative controls, and personal protective equipment that can reduce worker exposure to waste anesthetic gases. However, not every control listed in this section may be feasible in all settings.

Engineering controls

The collection and disposal of waste anesthetic gases in operating rooms and non-operating room settings is essential for reducing occupational exposures. Engineering controls such as an appropriate anesthetic gas scavenging system are the first line of defense and the preferred method of control to protect employees from exposure to anesthetic gases. An effective anesthetic gas scavenging system traps waste gases at the site of overflow from the breathing circuit and disposes of these gases to the outside atmosphere. The heating, ventilating and air conditioning (HVAC) system also contributes to the dilution and removal of waste gases not collected by the scavenging system or from other sources such as leaks in the anesthetic apparatus or improper work practices.

The exhalation of residual gases by patients in the PACU may result in significant levels of waste anesthetic gases when appropriate work practices are not used at the conclusion of the anesthetic or inadequate ventilation exists in the PACU. A nonrecirculating ventilation system can reduce waste gas levels in this area. Waste gas emissions to the outside atmosphere must meet local, state, and Environmental Protection Agency (EPA) regulatory requirements.

A scavenging system consists of five basic components (ASTM, F 1343 - 91):

- A gas collection assembly, such as a collection manifold or a distensible bag (i.e., Jackson-Rees pediatric circuit), which

captures excess anesthetic gases at the site of emission, and delivers it to the transfer tubing.
- Transfer tubing, which conveys the excess anesthetic gases to the interface.
- The interface, which provides positive (and sometimes negative) pressure relief and may provide reservoir capacity. It is designed to protect the patient's lungs from excessive positive or negative scavenging system pressure.
- Gas disposal assembly tubing, which conducts the excess anesthetic gases from the interface to the gas disposal assembly.
- The gas disposal assembly, which conveys the excess gases to a point where they can be discharged safely into the atmosphere. Several methods in use include a nonrecirculating or recirculating ventilation system, a central vacuum system, a dedicated (single-purpose) waste gas exhaust system, a passive duct system and an adsorber.

In general, a machine-specific interface must be integrated with a facility's system for gas removal. The interface permits excess gas to be collected in a reservoir (bag or canister) and limits the pressure within the bag or canister. A facility's gas disposal system receives waste anesthetic gases from the interface and should vent the waste gases outside the building and away from any return air ducts or open windows, thus preventing the return of the waste gases back into the facility.

Removal of excess anesthetic gases from the anesthesia circuit can be accomplished by either active or passive scavenging. When a vacuum or source of negative pressure is connected to the scavenging interface, the system is described as an active system. When a vacuum or negative pressure is not used, the system is described as a passive system. With an active system, there will be a negative pressure in the gas disposal tubing. With a passive system, this pressure will be increased above atmospheric (positive) by the patient exhaling passively, or manual compression of the breathing system reservoir bag.

Use of a central vacuum system is an example of an active system: The waste anesthetic gases are moved along by negative pressure. Venting waste anesthetic gas via the exhaust grille or exhaust duct of a nonrecirculating ventilation system is an example of a passive system: The anesthetic gas is initially moved along by the positive pressure from the breathing circuit until it reaches the gas disposal assembly.

Active systems

Excess anesthetic gases may be removed by a central vacuum system (servicing the ORs in general) or an exhaust system dedicated to the disposal of excess gases. When the waste anesthetic gas scavenging system is connected to the central vacuum system (which is shared by other users, e.g., surgical suction), exposure levels may be effectively controlled. The central vacuum system must be specifically designed to handle the large volumes of continuous suction from OR scavenging units. If a central vacuum system is used, a separate, dedicated gas disposal assembly tubing should be used for the scavenging system, distinct from the tubing used for patient suctioning (used for oral and nasal gastric sources as well as surgical suctioning).

Similarly, when a dedicated exhaust system (low velocity) is used, excess gases can also be collected from one or more ORs and discharged to the outdoors. The exhaust fan must provide sufficient negative pressure and air flow so that cross-contamination does not occur in the other ORs connected to this system. Active systems are thought to be more effective than passive systems at reducing excess waste anesthetic gas concentrations because leaks in the scavenging system do not result in an outward loss of gas.

Passive systems

HVAC systems used in health-care facilities are of two types: nonrecirculating and recirculating. Nonrecirculating systems, also termed "one-pass" or "single-pass" systems, take in fresh air from the outside and circulate filtered and conditioned air (i.e., controlled for temperature and humidity) through the room. Whatever volumes of fresh air are introduced into the room are ultimately exhausted to the outside. Waste anesthetic gases can be efficiently disposed of via this nonrecirculating system.

When a nonrecirculating ventilation system serves through large-diameter tubing and terminating the tubing at the room's ventilation exhaust as the disposal route for excess anesthetic gases, disposal involves directing the waste gases grille. The sweeping effect of the air flowing into the grille carries the waste gases away. Because all of the exhausted air is vented to the external atmosphere in this type of system, the excess anesthetic gases can be deposited into the exhaust stream either at the exhaust grille or further downstream in the exhaust duct.

Concern for fuel economy has increased the use of systems that recirculate air. Recirculating HVAC/ventilation systems return part of the exhaust air back into the air intake and recirculate the mixture through the room. Thus, only a fraction of the exhaust air is disposed of to the outside. To maintain minimal levels of anesthetic exposure, air that is to be recirculated must not contain anesthetic gases. Consequently, recirculating systems employed as a disposal pathway for waste anesthetic gases must not be used for gas waste disposal. The exception is an arrangement that transfers waste gases into the ventilation system at a safe distance downstream from the point of recirculation to ensure that the anesthetic gases will not be circulated elsewhere within the building.

Under certain circumstances, a separate duct for venting anesthetic gases directly outside the building without the use of a fan may be an acceptable alternative. By this technique, excess anesthetic gases may be vented through the wall, window, ceiling or floor, relying only on the slight positive pressure of the gases leaving the gas collection assembly to provide the flow. However, several limitations are apparent. A separate line would be required for each OR to prevent the cross-contamination with anesthetic gases among the ORs. A safe disposal site would be necessary. The possible effects of variations in wind velocity and direction would require a means for preventing a reverse flow in the disposal system. Occlusion of the outer portion of such a passive system by ice or by insect or bird nests is also possible. The outside opening of a through-wall, window, ceiling or floor disposal assembly should be directed downward, shielded and screened to prevent the entrance of foreign matter or ice buildup. Despite these limitations, the separate duct without the use of a fan may be ideal in older facilities constructed with windows that cannot be opened and in the absence of nonrecirculating air conditioning.

Absorbers can also trap most excess anesthetic gases. Canisters of varying shapes and capacities filled with activated charcoal have been used as waste gas disposal assemblies by directing the gases from the gas disposal tubing through them. Activated charcoal canisters will effectively adsorb the vapors of halogenated anesthetics, but not N_2O. The effectiveness of individual canisters and various brands of charcoal vary widely. Different potent inhaled volatile agents are adsorbed with varying efficiencies. The efficiency of adsorption also depends on the rate of gas flow through the canister. The canister is used where portability is necessary. The disadvantages are that they are expensive and must be changed frequently. Canisters must be used and discarded in the appropriate manner, as recommended by the manufacturer.

General or dilution ventilation

An effective room HVAC system when used in combination with an anesthetic gas scavenging system should reduce, although not entirely eliminate, the contaminating anesthetic gases. If excessive concentrations of anesthetic gases are present, then airflow should be increased in the room to allow for more air mixing and further dilution of the anesthetic gases. Supply register louvers located in the ceiling should be designed to direct the fresh air toward the floor and toward the health care workers to provide dilution and removal of the contaminated air from the operatory or PACU. Exhaust register louvers should be properly located (usually low on the wall near the floor level) in the room to provide adequate air distribution. They should not be located near the supply air vents because this will short-circuit the airflow and prevent proper air mixing and flushing of the contaminants from the room.

Work practices

Work practices, as distinct from engineering controls, involve the way in which a task is performed. OSHA has found that appropriate work practices can be a vital aid in reducing the exposures of OR personnel to waste anesthetic agents. In contrast, improper anesthetizing techniques can contribute to increased waste gas levels. These techniques can include an improperly selected and fitted face mask, an insufficiently inflated tracheal tube cuff, an improperly positioned laryngeal mask, or other airway, and careless filling of vaporizers and spillage of liquid anesthetic agents.

General work practices recommended for anesthetizing locations include the following:

- A complete anesthesia apparatus checkout procedure should be performed each day before the first case. An abbreviated version should be performed before each subsequent case. The FDA Anesthesia Apparatus Checkout Recommendations should be considered in developing inspection and testing procedures for equipment checkout prior to administering an anesthetic.
- If a face mask is to be used for administration of inhaled anesthetics, it should be available in a variety of sizes to fit each patient properly. The mask should be pliable and provide as effective a seal as possible against leakage into the surrounding air.
- Tracheal tubes, laryngeal masks, and other airway devices should be positioned precisely and the cuffs inflated adequately.
- Vaporizers should be filled in a well-ventilated area and in a manner to minimize spillage of the liquid agent. This can be accomplished by using a specialized "key-fill" spout to pour the anesthetic into the vaporizer instead of pouring from a bottle into a funnel-fill vaporizer. When feasible, vaporizers should be filled at the location where the anesthetic will be administered and, when filled electively, with the fewest possible personnel present in the room. Vaporizers should be turned off when not in use.
- Spills of liquid anesthetic agents should be cleaned up promptly.
- Before extubating the patient's trachea or removing the mask or other airway management device, one should administer non-anesthetic gases/agents so that the washed-out anesthetic gases can be removed by the scavenging system. The amount of time allowed for this should be based on clinical assessment and may vary from patient to patient. When possible, flushing of the breathing system should be achieved by exhausting into the scavenging system rather than into the room air.
- Work practices performed by biomedical engineers and technicians also contribute significantly to the efficacy of managing waste gas exposure. It is, therefore, important for this group of workers to do the following:
    - Monitor airborne concentrations of waste gases by sampling, measuring, and reporting data to the institution's administration. Air monitoring for waste anesthetic gases should include both personal sampling (i.e., in a health-care worker's breathing zone) and area sampling.
    - Assist in identifying sources of waste/leaking gases and implementing corrective action.
    - Determine whether the scavenging system is designed and functioning properly to remove the waste anesthetic gases from the breathing circuit, and ensure that the gases are vented from the workplace in such a manner that occupational re-exposure does not occur (e.g., smoke trail tests of exhaust grilles used with passive scavenging systems).
    - Ensure that operatory and PACU ventilation systems provide sufficient room air exchange to reduce ambient waste gas levels.

Administrative controls

Administrative controls represent another approach for reducing worker exposure to waste gases other than through the use of engineering controls, work practices, or personal protective equipment. Administrative controls may be thought of as any administrative decision that results in decreased anesthetic-gas exposure. For workers potentially exposed to waste anesthetic gases, the program administrator should establish and implement policies and procedures to:

- Institute a program of routine inspection and regular maintenance of equipment in order to reduce anesthetic gas leaks and to have the best performance of scavenging equipment and room ventilation. Preventive maintenance should be performed by trained individuals according to the manufacturer's recommendations and at intervals determined by equipment history and frequency of use. Preventive maintenance includes inspection, testing, cleaning, lubrication and adjustment of various components. Worn or damaged parts should be repaired or replaced. Such maintenance can result in detection of deterioration before an overt malfunction occurs. Documentation of the maintenance program should be kept indicating the nature and date of the work performed, as well as the name of the trained individual servicing the equipment.
- Implement a monitoring program to measure airborne levels of waste gases in the breathing zone or immediate work area of those most heavily exposed (e.g., anesthesiologist, nurse anesthetist, oral surgeon) in each anesthetizing location and PACU. Periodic monitoring (preferably at least semiannually) of waste gas concentrations is needed to ensure that the anesthesia delivery equipment and engineering/environmental controls work properly and that the maintenance program is effective. Monitoring may be performed effectively using conventional time-weighted average air sampling or real-time air sampling techniques.
- Encourage or promote the use of scavenging systems in all anesthetizing locations where inhaled agents are used, recognizing that a waste gas scavenging system is the most effective means of controlling waste anesthetic gases.
- Implement an information and training program for employees exposed to anesthetic agents that complies with OSHA's Hazard Communication Standard (29 CFR 1910.1200) so that employees can meaningfully participate in, and support, the protective measures instituted in their workplace.
- Define and implement appropriate work practices to help reduce employee exposure. Training and educational programs covering appropriate work practices to minimize levels of anesthetic gases

in the operating room should be conducted at least annually. Employers should emphasize the importance of implementing these practices and should ensure that employees are properly using the appropriate techniques on a regular basis.
- Implement a medical surveillance program for all workers exposed to waste gases.
- Ensure the proper use of personal protective equipment during cleanup and containment of major spills of liquid anesthetic agents.
- Manage disposal of liquid agents, spill containment, and air monitoring for waste gases following a spill.
- Comply with existing federal, state, and local regulations and guidelines developed to minimize personnel exposure to waste anesthetic gases, including the proper disposal of hazardous chemicals.

Location-specific workplace controls

This section describes engineering and work practice controls specific to hospital ORs, PACUs, dental operatories and veterinary clinics and hospitals. Operational procedures relating to engineering controls are also discussed where appropriate.

Hospital operating rooms

For years, anesthesia providers tolerated exposure to waste anesthetic gases and regarded it as an inevitable consequence of their work. Since the 1970s, anesthesiologists have steadily worked to improve equipment and technique to reduce workplace exposures to waste anesthetic gases, and significant progress has been made. In early delivery equipment, waste gases were exhausted through the APL or "pop-off" valve into the face of the anesthesia provider and were distributed into the room air. Present practice, which utilizes an efficient scavenging system, avoids this type of contamination by collecting the excess gases immediately at the APL valve.

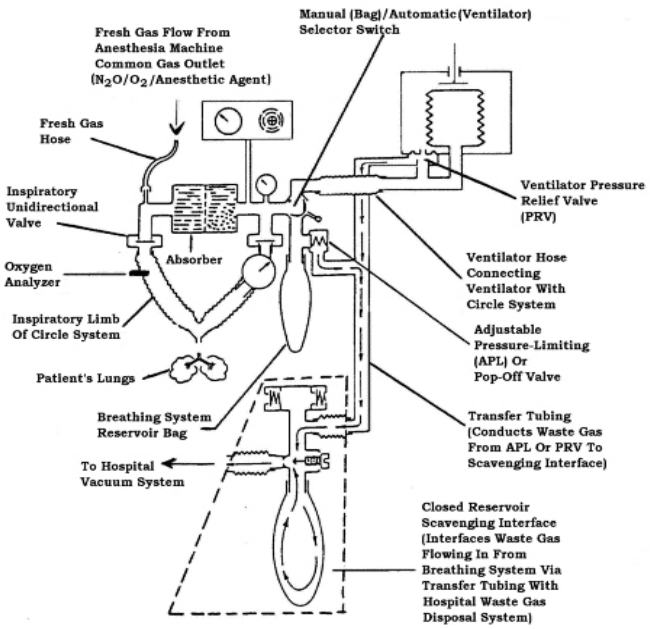
Engineering controls

Waste gas evacuation is required for every type of breathing circuit configuration with the possible exception of a closed circuit, because most anesthesia techniques typically use more fresh gas flow than is required. Appropriate waste gas evacuation involves collection and removal of waste gases, detection and correction of leaks, consideration of work practices, and effective room ventilation. To minimize waste anesthetic gas concentrations in the operating room, the recommended air exchange rate (room dilution ventilation) is a minimum total of 15 air changes per hour with a minimum of 3 air changes of outdoor air (fresh air) per hour. Operating room air containing waste anesthetic gases should not be recirculated to the operating room or other hospital locations.

Work practices

In most patients, a circle absorption system is used and can be easily connected to a waste gas scavenging system. In pediatric anesthesia, systems other than those with a circle absorber may be used. Choice of the breathing circuit that best meets the needs of pediatric patients may alter a clinician's ability to scavenge waste gas effectively. Breathing circuits frequently chosen for neonates, infants and small children are usually valveless, have low resistance and limit rebreathing. The Mapleson D system and the Jackson-Rees modification of the Ayre's T-piece are examples of limited rebreathing systems that require appropriate scavenging equipment.

The following work practices may be employed with any of the above breathing circuits:
- Empty the contents of the reservoir bag directly into the anesthetic gas scavenging system and turn off the flow of N_2O and any halogenated anesthetic agent prior to disconnecting the patient circuit.
- Turn off the flow of N_2O and the vaporizer, if appropriate, when the patient circuit is disconnected from the patient, for example, for oral or tracheal suctioning.
- Test daily for low-pressure leaks throughout the entire anesthesia system. All leaks should be minimized before the system is used. Starting anesthetic gas flow before the actual induction of anesthesia begins is not acceptable. For techniques to rapidly induce anesthesia using inhaled agents (single-breath mask induction), the patient connector should be occluded when filling the breathing circuit with nitrous oxide or halogenated agent prior to applying the mask to the patient's face.



If the circle absorber system (Figure 6) is used, the following additional work practices can be employed:
- Adjust the vacuum needle valve as needed to regulate the flow of waste anesthetic gases into the vacuum source in an active scavenging system. Adjustments prevent the bag from overdistending by maintaining the volume in the scavenging system reservoir bag between empty and half-full. In machines that use an open reservoir to receive waste gas, a flow meter is used to adjust the rate of gas flow to the vacuum system.
- Cap any unused port in a passive waste gas scavenging configuration.

Postanesthesia care in hospitals and stand-alone facilities

Because the patient is the main source of waste anesthetic gases in the PACU, it becomes more difficult to control health-care workers' exposures to waste anesthetic gases. The unique PACU environment coupled with the patient's immediate condition upon arrival from surgery require different work practices than those routinely used in ORs. Patients undergoing general anesthesia usually have their airways secured using a tracheal tube with an inflatable cuff that seals the tube within the trachea. The seal between the tracheal tube cuff and the trachea (or between the face mask and the face) is essential for maintaining a gas-tight system that permits effective scavenging in the OR. The tracheal tube connects the patient with the breathing circuit that is connected to the scavenging system in the OR. Once the patient reaches the PACU, scavenging systems such as those used in the OR are no longer effective, since the patient is no longer connected to the breathing circuit. Other less-effective methods of waste gas removal are thus relied upon.

Engineering controls

As a result of using appropriate anesthetic gas scavenging in ORs, the levels of contamination have been decreased. In the PACU, however, the principle of scavenging as practiced in the OR is not widely accepted due to medical considerations and consequently is infrequently employed as a source-control method for preventing the release of waste anesthetic gases into the PACU environment. Most PACUs provide care to multiple patients in beds without walls between them, and convective currents move the gases from their source to other areas. Therefore, in the PACU, a properly designed and operating dilution ventilation system should be relied upon to minimize waste anesthetic gas concentrations. This system should provide a recommended minimum total of 6 air changes per hour with a minimum of 2 air changes of outdoor air per hour to adequately dilute waste anesthetic gases. Room exhaust containing waste anesthetic gases should not be recirculated to other areas of the hospital.

Work practices

PACU managers should consider:

- Periodic exposure monitoring with particular emphasis on peak gas levels in the breathing zone of nursing personnel working in the immediate vicinity of the patient's head. Methods using random room sampling to assess ambient concentrations of waste anesthetic gases in the PACU are not an accurate indicator of the level of exposure experienced by nurses providing bedside care. Because of the closeness of the PACU nurse to the patient, such methods would consistently underestimate the level of waste anesthetic gases in the breathing zone of the bedside nurse.
- Application of a routine ventilation system maintenance program to keep waste gas exposure levels to a minimum.

Dental operatory

Mixtures of N_2O and oxygen have been used in dentistry as general anesthetic agents, analgesics and sedatives for more than 100 years. The usual analgesia equipment used by dentists includes a N_2O and O_2 delivery system, a gas mixing bag and a nasal mask with a positive pressure relief valve. The analgesia machine is usually adjusted to deliver more of the analgesic gas mixture than the patient can use.

Analgesia machines for dentistry are designed to deliver up to 70 percent (700,000 ppm) N_2O to a patient during dental surgery. The machine restricts higher concentrations of N_2O from being administered to protect the patient from hypoxia. In most cases, patients receive between 30 and 50 percent N_2O during surgery. The amount of time N_2O is administered to a patient depends on the dentist's judgment of patient needs and the complexity of the surgery. The most common route of N_2O delivery and exhaust is through a nasal scavenging mask applied to the patient.

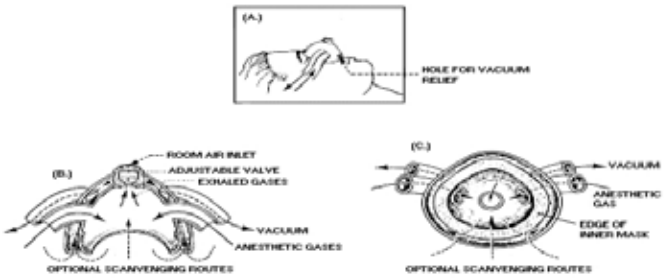
Some dentists administer N_2O at higher concentrations at the beginning of the operation, then decrease the amount as the operation progresses. Others administer the same amount of N_2O throughout the operation. When the operation is completed, the N_2O is turned off. Some dentists turn the N_2O on only at the beginning of the operation, using N_2O as a sedative during the administration of local anesthesia, and turn it off before operating procedures. Based on variations in dental practices and other factors in room air, N_2O concentrations can vary considerably for each operation and also vary over the course of the operation.

Unless the procedure is performed under general anesthesia in an OR, halogenated anesthetics are not administered, nor does the patient undergo laryngoscopy and tracheal intubation. In the typical dental office procedure, the nasal mask is placed on the patient, fitted and adjusted prior to administration of the anesthetic agent. The mask is designed for the nose of the patient because access to the patient's mouth is essential for dental procedures.

A local anesthetic, if needed, is typically administered after the N_2O takes effect. The patient's mouth is opened and the local anesthetic is injected. The dental procedure begins after the local anesthetic takes effect. The patient opens his/her mouth but is instructed to breathe through the nose. Nonetheless, a certain amount of mouth breathing frequently occurs. The dentist may periodically stop the dental procedure for a moment to allow the patient to close the mouth and breathe deeply to re-establish an appropriate concentration of N_2O in the patient's body before resuming the procedure. Depending on the nature of the procedure, high velocity suction is regularly used to remove intraoral debris and, when used, creates a negative air flow and captures some of the gas exhaled by the patient.

At the end of the procedure, the nosepiece is left on the patient while the N_2O is turned off and the oxygen flow is increased. The anesthetic mixture diffuses from the circulating blood into the lungs and is exhaled. Scavenging is continued while the patient is eliminating the N_2O.

The dental office or operatory should have a properly installed N_2O delivery system. This includes appropriate scavenging equipment with a readily visible and accurate flow meter (or equivalent measuring device), a vacuum pump with the capacity for up to 45 L/min of air per workstation, and a variety of sizes of masks to ensure proper fit for individual patients.



A common nasal mask, shown in Figure 7 (located at the end of this chapter), consists of an inner and a slightly larger outer mask component. The inner mask has two hoses connected that supply

anesthetic gas to the patient. A relief valve is attached to the inner mask to release excess N_2O into the outer mask. The outer mask has two smaller hoses connected to a vacuum system to capture waste gases from the patient and excess gas supplied to the patient by the analgesia machine. The nasal mask should fit over the patient's nose as snugly as possible without impairing the vision or dexterity of the dentist. Gases exhaled orally are not captured by the nasal mask. A flow rate of approximately 45 L/min has been recommended as the optimum rate to prevent significant N_2O leakage into the room air.

A newer type of mask is a frequent choice in dental practice, a single-patient-use nasal hood. This mask does not require sterilization after surgery because it is used by only one patient and is disposable.

In a dental operatory, a scavenging system is part of a high-volume evacuation system used with a dental unit. The vacuum system may dispose of a combination of waste gases, oral fluid and debris, and is not limited to waste gas removal. The exhaust air of the evacuation system should be vented outside the building and away from fresh-air inlets and open windows to prevent re-entry of gas into the operatory.

The general ventilation should provide good room air mixing. In addition, auxiliary (local) exhaust ventilation used in conjunction with a scavenging system has been shown to be effective in reducing excess N_2O in the breathing zone of the dentist and dental assistant, from nasal mask leakage and patient mouth breathing. This type of ventilation captures the waste anesthetic gases at their source. However, there are practical limitations in using it in the dental operatory. These include proximity to the patient, interference with dental practices, noise, and installation and maintenance costs. It is most important that the dentist not work between the patient and a free-standing local exhaust hood. Doing so will cause the contaminated air to be drawn through the dentist's breathing zone. These auxiliary ventilation systems are not now commercially available. The Academy of General Dentistry also emphasizes properly installed and maintained analgesia delivery systems.

Work practices

- Prior to first use each day of the N_2O machine and every time a gas cylinder is changed, the low-pressure connections should be tested for leaks. High-pressure line connections should be tested for leaks quarterly. A soap solution may be used to test for leaks at connections. Alternatively, a portable infrared spectrophotometer can be used to detect an insidious leak.
- Prior to first use each day, inspect all N_2O equipment (e.g., reservoir bag, tubing, mask, connectors) for worn parts, cracks, holes, or tears. Replace as necessary.
- Connect mask to the tubing and turn on vacuum pump. Verify appropriate flow rate (i.e., up to 45 L/min or manufacturer's recommendations).
- A properly sized mask should be selected and placed on the patient. A good, comfortable fit should be ensured. The reservoir (breathing) bag should not be over- or underinflated while the patient is breathing oxygen (before administering N_2O).
- Encourage the patient to minimize talking, mouth breathing and facial movement while the mask is in place.
- During N_2O administration, the reservoir bag should be periodically inspected for changes in tidal volume, and the vacuum flow rate should be verified.
- On completing anesthetic administration and before removing the mask, non-anesthetic gases/agents should be delivered to the patient for a sufficient time based on clinical assessment that may vary from patient to patient. In this way, both the patient and the system will be purged of residual N_2O. Do not use an oxygen flush.

Cleanup and disposal of liquid anesthetic agent spills

Small volumes of liquid anesthetic agents such as halothane, enflurane, isoflurane, desflurane, and sevoflurane evaporate readily at normal room temperatures, and may dissipate before any attempts to clean up or collect the liquid are initiated. However, when large spills occur, such as when one or more bottles of a liquid agent break, specific cleaning and containment procedures are necessary and appropriate disposal is required. The recommendations of the chemical manufacturer's material safety data sheet (MSDS) that identify exposure reduction techniques for spills and emergencies should be followed.

In addition, OSHA Standard for Hazardous Waste Operations and Emergency Response would apply if emergency response efforts are performed by employees. The employer must determine the potential for an emergency in a reasonably predictable worst-case scenario, and plan response procedures accordingly. Only adequately trained and equipped workers may respond to spills. When the situation is unclear or data are lacking on the exposure level, the response needs to be the same as for high levels of exposure. Responses to incidental releases of liquid anesthetic agents where the substance can be absorbed, neutralized or otherwise controlled at the time of release by employees in the immediate release area, or by maintenance personnel do not fall within the scope of this standard.

Because of the volatility of liquid anesthetics, rapid removal by suctioning in the OR is the preferred method for cleaning up spills. Spills of large volumes in poorly ventilated areas or in storage areas should be absorbed using an absorbent material, sometimes called a sorbent, that is designed for cleanup of organic chemicals. "Spill pillows" commonly used in hospital laboratories, vermiculite, and carbon-based sorbents are some of the materials commercially available and regularly used for this purpose. Caution should be exercised if broken glass bottles pose a hazard.

Both enflurane and desflurane are considered hazardous wastes under the EPA regulations because these chemicals contain trace amounts of chloroform (a hazardous substance), a byproduct of the manufacturing process. Consequently, sorbents that have been saturated with enflurane or desflurane should be managed as an EPA hazardous waste material due to the trace concentrations of chloroform present. Isoflurane and halothane do not contain trace amounts of chloroform or any other regulated substance and are therefore not considered hazardous wastes by EPA.

To minimize exposure to all liquid anesthetic agents during cleanup and to limit exposure during disposal procedures, the following general guidelines are recommended. The waste material should be placed in a container, tightly sealed, properly labeled, and disposed of with other chemical wastes sent to a facility's incinerator or removed by a chemical waste contractor. After a large spill has occurred and the appropriate response action taken, airborne monitoring should be conducted to determine whether the spill was effectively contained and cleaned up.

Determination of appropriate disposal procedures for each facility is the sole responsibility of that facility. Empty anesthetic bottles are not considered regulated waste and may be discarded with ordinary trash or recycled. Furthermore, the facility as well as the waste handling contractor must comply with all applicable federal, state, and local regulations.

To minimize exposure to waste liquid anesthetic agents during cleanup and disposal, the following general guidelines are recommended by the manufacturers of liquid anesthetic agents:

- Wear appropriate personal protective equipment. Where possible, ventilate area of spill or leak. Appropriate respirators should be worn.
- Restrict persons not wearing protective equipment from areas of spills or leaks until cleanup is complete.
- Collect the liquid spilled and the absorbent materials used to contain a spill in a glass or plastic container. Tightly cap and seal the container and remove it from the anesthetizing location. Label the container clearly to indicate its contents.
- Transfer the sealed containers to the waste disposal company that handles and hauls waste materials.
- Health-care facilities that own or operate medical waste incinerators may dispose of waste anesthetics by using an appropriate incineration method after verifying that individual incineration operating permits allow burning of anesthetic agents at each site.

Air monitoring

Air monitoring is one of the fundamental tools used to evaluate workplace exposures. Accordingly, this section presents some of the appropriate methods that can be used to detect and measure the concentration of anesthetic gases that may be present in the health care environment. The data provided by monitoring are necessary to establish proper engineering, work practice, and administrative controls to ensure the lowest reasonably achievable gas levels in the operatory and PACU room air.

OSHA recommends that air sampling for anesthetic gases be conducted every six months to measure worker exposures and to check the effectiveness of control measures. Furthermore, OSHA recommends that only the agents most frequently used need to be monitored, since proper engineering controls, work practices and control procedures should reduce all agents proportionately. However, the decision to monitor only selected agents could depend not only on the frequency of their use, but on the availability of an appropriate analytical method and the cost of instrumentation. ASA emphasizes regular maintenance of equipment and scavenging systems, daily check-out procedures for anesthesia equipment, and education to ensure use of appropriate work practices. It does not believe that a routine monitoring program is necessary when these actions are being carried out. ASA prefers to use monitoring when indicated, such as in the event of known or suspected equipment malfunction.

Three fundamental types of air samples can be taken in order to evaluate the workplace: personal, area and source samples. Personal samples give the best estimate of a worker's exposure level because they represent the actual airborne contaminant concentration in the worker's breathing zone during the sampling period. This is the preferred method for determining a worker's time-weighted average (TWA) exposure and should be used to assess personal exposures during anesthetic administration and in the PACU. Where several health care workers perform the same job, on the same shift, and in the same work area, and the length, duration, and level of waste gas exposures are similar, an employer may sample a representative fraction of the employees instead of all employees.

Area sampling is useful for evaluating overall air contaminant levels in a work area and for investigating cross-contamination with other areas in the health care facility. Source sampling can be used to detect leaks in the anesthesia delivery and scavenging systems as well as ineffective capture by the scavenging system. Thus, how samples are taken is a critical point in any safety program.

The OSHA Chemical Information Manual contains current sampling technology for several of the anesthetic gases that may be present in anesthetizing locations and PACUs. Some of the sampling methods available are summarized below.

Time-integrated sampling

- **Nitrous oxide**
  Personal N_2O exposures can be determined by using the VAPOR-TRAK nitrous oxide passive monitor (sometimes called a "passive dosimeter" or "diffusive sampler") as referenced in the 2000 OSHA Chemical Information Manual under IMIS:1953. The minimum sampling duration for the dosimeter is 15 minutes; however, it can be used for up to 16 hours of passive sampling. This sampler has not been validated by OSHA. Other dosimeters are commercially available and can be used. Although not validated by OSHA at this time, they may be validated in the future. Five liter, 5-layer aluminized gas sampling bags can also be used to collect a sample.
- **Halogenated agents**
  Three chlorofluorocarbon-based anesthetic agents (halothane, enflurane, and isoflurane) and one fluorocarbon-based agent (desflurane) are listed in the Chemical Information Manual. The OSHA sampling procedure for halothane is listed under IMIS:0395; for enflurane, under IMIS:1038; for isoflurane, under IMIS:F118; and for desflurane, under IMIS:R218.

  The current recommended media sampling for halothane, enflurane and isoflurane requires an Anasorb 747 tube (140/70 mg sections) or an Anasorb CMS tube (150/75 mg. sections). The sample can be taken at a flow rate of 0.5 L/min. Total sample volumes not exceeding 12 liters are recommended. The current recommended sampling media for desflurane requires an Anasorb 747 tube (140/70 mg sections). The sample can be taken at a flow rate of 0.05 L/min. Total sample volumes not exceeding 3 liters are recommended. All four sampling methodologies are fully validated analytical procedures.

Real-time sampling

Sampling that provides direct, immediate, and continuous (real-time) readout of anesthetic gas concentrations in ambient air utilizes a portable infrared spectrophotometer. Since this method provides continuous sampling and instantaneous feedback, sources of anesthetic gas leakage and effectiveness of control measures can be immediately determined.

Additional sampling guidelines

If it should ever be necessary to enter an operating room to conduct air sampling, the following guidelines provide the information needed. Individuals performing air sampling should be familiar with and follow all OR procedures for access into and out of the surgical suite with particular attention to sterile and nonsterile areas. The patient is the center of the sterile field, which includes the areas of the patient, operating table, and furniture covered with sterile drapes and the personnel wearing sterile attire. Sampling in the breathing zone of surgeons and other nursing or technical personnel who work in the sterile field must conform to the principles of sterile field access. Strict adherence to sound principles of sterile technique and recommended practices is mandatory for the safety of the patient.

Generally speaking, each hospital has its own guidelines for proper OR attire and other safety procedures. These rules should be strictly followed by anyone entering the OR. There are standard uniform guidelines that apply to all hospitals. Only clean and/or freshly laundered OR attire is worn in the OR. Proper attire consists of body covers such as a two-piece pantsuit (scrub suit), head cover (cap or hood), mask, and shoe covers. A sterile gown is worn over the scrub suit to permit the wearer to come within the sterile field. Other attire such as gloves and eyewear may be required. Some hospitals, but not all, may allow persons coming into the OR to wear a clean gown (in addition to the cap, the mask, and the shoe covers) over their street clothes if they are not going to remain in the OR for longer than 10-15 minutes.

In regard to decontaminating outside equipment, each hospital has its own policy. However, the common practice is to "wipe off" all surfaces with a chemical disinfectant. Most hospitals use Wescodyne or other phenolic solutions. Good physical cleaning before disinfection helps reduce the number of microorganisms present and enhances biocidal action.

Any person not familiar with the OR is usually instructed by a scrub nurse on all the safety procedures pertaining to the hospital. The scrub nurse will also provide instructions on hand scrubbing and other procedures that may be necessary. Persons entering the OR must follow these guidelines and instructions.

In addition, it should be recognized that the patient's welfare, safety, and rights of privacy are paramount.

Hazard communication

In accordance with the Hazard Communication Standard (29 CFR 1910,1200), employers in health care facilities must develop, implement and maintain at the workplace a written, comprehensive hazard communication program that includes provisions for container labeling, collection and availability of material safety data sheets (MSDSs), and an employee training and information program. The standard also requires a list of hazardous chemicals in the workplace as part of the written hazard communication program.

Any chemicals subject to the labeling requirements of the FDA are exempt from the labeling requirements under the Hazard Communication Standard. This includes such chemicals as volatile liquid anesthetics and compressed medical gases. However, containers of other chemicals not under the jurisdiction of the FDA must be labeled, tagged or marked with the identity of the material and must show appropriate hazard warnings as well as the name and address of the chemical manufacturer, importer, or other responsible party. The hazard warning can be any type of message – words, pictures, or symbols – that conveys the hazards of the chemical(s) in the container. Labels must be legible, in English (plus other languages if desired), and prominently displayed.

Each MSDS must be in English, although the employer may maintain copies in other languages as well, and must include information regarding the specific chemical identity of the anesthetic gases or hazardous chemical and its common names. In addition, information must be provided on the physical and chemical characteristics of the hazardous chemical, known acute and chronic health effects and related health information, primary route(s) of entry, exposure limits, precautionary measures, emergency and first-aid procedures, and the identification of the organization responsible for preparing the sheet. As a source of detailed information on hazards, copies of the MSDS for each hazardous chemical must be readily accessible during each work shift to employees when they are in their work area(s).

Employers must prepare a list of all hazardous chemicals in the workplace, and the list should be checked to verify that MSDSs have been received for each chemical. If there are hazardous chemicals used for which no MSDS has been received, the employer must contact the supplier, manufacturer or importer to obtain the missing MSDS.

Health care employers must establish a training and information program for all personnel who are involved in the handling of, or who have potential exposure to, anesthetic gases and other hazardous chemicals to apprise them of the hazards associated with these chemicals in the workplace. Training relative to anesthetic gases should place an emphasis on reproductive risks. Training and information must take place at the time of initial assignment and whenever a new hazard is introduced into the work area. At a minimum, employees must be informed of the following:

- The Hazard Communication Standard (29 CFR 1910.1200) and its requirements.
- Any operations and equipment in the work area where anesthetic agents and hazardous chemicals are present.
- Location and availability of the written hazard communication program including the required lists of hazardous chemicals and the required MSDS forms.

The employee training program must consist of the following elements:

- How the hazard communication program is implemented in the workplace, how to read and interpret information on the MSDS and label of each hazardous chemical, and how employees can obtain and use the available hazard information.
- The physical and health hazards of the chemicals in the work area.
- Measures employees can take to protect themselves from these hazards, including specific procedures put into effect by the employer to provide protection such as engineering controls, appropriate work practices, emergency procedures for spill containment, and the use of personal protective equipment.
- Methods and observations that may be used to detect the presence or release of anesthetic gases and other hazardous chemicals in the work area (such as monitoring conducted by the employer, continuous monitoring devices, and the appearance or odor of chemicals when released).

Personnel training records are not required to be maintained, but such records would assist employers in monitoring their programs to ensure that all employees are appropriately trained. Employers can provide employees information and training through whatever means are found appropriate and protective. Although there would always have to be some training on-site (such as informing employees of the location and availability of the written program and MSDSs), employee training may be satisfied in part by general training about the requirements of the hazard communication standard and about chemical hazards on the job which is provided by, for example, professional associations, colleges, universities, and training centers. In addition, previous training, education and experience of a worker may relieve the employer of some of the burdens of informing and training that worker. The employer, however, maintains the responsibility to ensure that employees are adequately trained and are equipped with the knowledge and information to do their jobs safely.

Step-by-step approach for controlling N_2O		
Step	Procedure	Control
1	Visually inspect all N_2O equipment (reservoir bag, hoses, mask, connectors) for worn parts, cracks, holes, or tears.	Replace defective equipment and/or parts.
2	Turn on the N_2O tank and check all high- to low-pressure connections for leaks. Use a non-oil-based soap solution to check for bubbles at high pressure connectors, or use a portable infrared gas analyzer.	Determine leak source and fix. If tank valve leaks, replace tank; if O-rings, gaskets, valves, hoses, or fittings, replace. Contact the manufacturer for parts replacement. For threaded pipe fittings, use Teflon tape. Do not use this tape on compression fittings.
3	Select scavenging system and mask. Mask should come in various sizes to patients. Scavenging systems should operate at air flow rate of 45 lpm.	Provide a range of mask sizes for patients. Check to see that noise levels at the mask are acceptable when the scavenging system exhaust rate is operated at 45 lpm.
4	Connect mask to hose and turn on vacuum pump before turning on N_2O. Scavenging system vacuum pump must have capacity to scavenge 45 lpm per dental operation.	Determine proper vacuum pump size for maintaining 45 lpm flow rates, especially when interconnected with other dental scavenging systems. If undersized, replace pump.
5	Place mask on patient and assure a good, comfortable fit. Make sure reservoir bag is not over- or under-inflated while the patient is breathing.	Secure mask with "slip" ring for "good activity" from patient breathing.
6	Check general ventilation for good room air mixing. Exhaust vents should not be close to air supply vents (use smoke tubes to observe air movement in room.)	If smoke from smoke tubes indicate room air mixing is poor, then increase the airflow or redesign. If exhaust vents are close to air supply vents, relocate (check with ventilation engineers to make adjustments).
7	Conduct personal sampling of dentist and dental assistant for N_2O exposure. Use diffusive sampler or infrared gas analyzer (see sampling methods).	If personal exposures exceed 150 ppm during administration, improve mask fit and make sure it is secure over the patient's nose. Minimize patient talking while N_2O is administered.
8	Repeat procedure in step 7.	If personal exposures are less than 150 ppm but greater than 25 ppm, implement auxiliary exhaust ventilation near the patient's mouth. Capture distance should no greater than 10 inches from the patient's nose and mouth area and exhaust no less than 250 cfm at the hood opening. Avoid getting between the auxiliary exhaust hood and patient's mouth and nose area.

Conclusion

Nitrous oxide is used in many dental and medical offices and should be used with care. It is the most frequently used sedation method used in dentistry. All bodily functions remain normal, and the patient is able to breathe on his/her own. The patient will not fall asleep and will not have memory loss. It is best used for mildly anxious patients who wish only a small amount of sedation to "take the edge off" or to make them less nervous. The patient is able to respond appropriately to physical stimulation and verbal commands. It is a way for the dentist to manage pain and anxiety during dental appointments, but they must also administer it wisely and with caution. Each dental or medical office should establish its own comprehensive training program on how to maintain the equipment and proper safety standards. This course should help you to review your office routines and make sure you are setting the way to achieve the best safety standards for your patients and for your employees.

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Final Examination Questions

Select the best answer questions 16 through 20 and mark your answers on the Final Examination Answer Sheet found on page 148, or for faster service complete your test online at **Dental.EliteCME.com.**

16. Long-term exposure to nitrous oxide can cause adverse reproductive effects for pregnant females.
    - ○ True
    - ○ False

17. Inhaled anesthetic agents include two different classes of chemicals: nitrous oxide and halogenated agents.
    - ○ True
    - ○ False

18. Venting waste anesthetic gas via the exhaust grille or exhaust duct of a non-recirculating ventilation system is an example of an active system.
    - ○ True
    - ○ False

19. Analgesia machines for dentistry are designed to deliver up to 95 percent N_2O to a patient during dental surgery. In most cases, patients receive between 60-90 percent N_2O during surgery.
    - ○ True
    - ○ False

20. Three fundamental types of air samples can be taken to evaluate the workplace; they are personal, area and source samples.
    - ○ True
    - ○ False

Chapter 5: Oral Complications with Diseases

3 CE Hours

By: Elite Staff

Learning objectives

- Identify oral complications with diabetes.
- List ways to prevent complications with diabetes.
- Apply your understanding of cancer treatment and oral health.
- Prepare your patients for oral care after hematopoietic stem cell transplantation.
- Assess patients before and after radiation and chemotherapy.
- Discuss the role of cancer pretreatment oral care.
- Review the complications of the heart with infective endocarditis.
- Describe the oral problems caused by Sjögren's syndrome.
- Explain the treatment that can be done for xerostomia/salivary gland dysfunction.

Introduction

To achieve and maintain good oral health, people with diseases often require a special approach to dental care. With the help of their dentist, patients who practice good daily dental care can prevent bleeding, pain and even the spread of germs or bacteria to other parts of their bodies. Few people even realize that side effects of many diseases affect the mouth. But these complications may require special care and create needs that interfere with a patient's quality of life. This course is designed to help dental professionals better understand some of these special needs so they can include some of the strategies in their general practice setting and help their patients cope with these diseases.

Diabetes and oral health

How does diabetes affect the mouth?

People who have diabetes know the disease can harm the eyes, nerves, kidneys, heart and other important systems in the body. But it also can cause problems in the mouth.

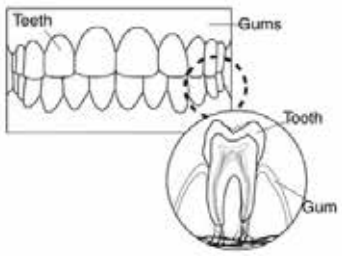
People with diabetes are at special risk for periodontal disease, which can lead to painful chewing difficulties and tooth loss for the patient. Dry mouth, often a symptom of undetected diabetes, can cause soreness, ulcers, infections and tooth decay. Smoking makes these problems worse.

What can be done for this problem?

Good blood glucose control is key to controlling and preventing mouth problems. People with poor blood glucose control get gum disease more often and more severely than people whose diabetes is well controlled. Daily brushing and flossing, regular dental check-ups and good blood glucose control are the best defense against the oral complications of diabetes.

What are diabetes problems?

Too much glucose in the blood for a long time can cause diabetes problems. This high blood glucose can damage many parts of the body, such as the heart, blood vessels, eyes and kidneys. Heart and blood vessel disease can lead to heart attacks and strokes. But a lot can be done to prevent or slow down diabetes problems.



Guide your patients to a healthier lifestyle and give them these suggestions.

- Work with a dietitian to design a healthy eating plan.
- Create an exercise program that works with daily activities. Discuss this with a medical doctor. Being active at least 30 minutes every day will make a big difference.
- Always take medication as directed.
- Check blood glucose every day or as directed by a medical doctor.
- Keep a record of blood glucose; write it in a book or on a notepad.
- Check feet every day for cuts, blisters, sores, swelling, redness or sore toenails.
- Brush and floss teeth daily.
- Control blood pressure and cholesterol.
- Don't smoke.

How can diabetes hurt the teeth and gums?

High blood glucose helps bacteria grow in the mouth, which can cause the patient to develop red, sore and swollen gums that bleed when they brush their teeth.

People with diabetes can have tooth and gum problems more often if their blood glucose stays high, and it can make tooth and gum problems worse or cause tooth loss.

Smoking makes it more likely for the patient to get a bad case of gum disease, especially for a person with diabetes or one who is age 45 or older. Red, sore and bleeding gums are the first sign of gum disease.

Some diabetes medicine can cause low blood glucose, called hypoglycemia. A dentist might want to talk with a patient's medical doctor before the person's visit about the best way to take care of his or her blood glucose during the dental work. The patient may need to bring some diabetes medicine and food to your office to help maintain a level blood glucose.

If the mouth is sore after the dental work, the patient may not be able to eat or chew for several hours or days. For guidance on how to adjust their normal routine while their mouth is healing, check with the medical doctor or dietitian and plan the following:

- What foods and drinks the patient should have.
- Whether the patient should change diabetes medicine routines.
- How often the patient should check his or her blood glucose during the day of and the days following the dental procedure.

Diabetes can cause serious problems in the mouth

If you have a patient with diabetes, discuss this with the person and make sure he or she stays on a routine schedule with your office. People with diabetes are at risk for mouth infections, especially periodontal disease, which can lead to painful chewing problems.

Other problems diabetes can cause are dry mouth and a fungal infection called thrush. Dry mouth happens when a person does not have enough saliva. Diabetes may also cause the glucose level in saliva to increase. Together, these problems may lead to thrush, which causes painful white patches in the mouth.

Impress upon your diabetic patients that by controlling their blood glucose, brushing and flossing every day and visiting a dentist regularly, they can prevent periodontal disease.

Cancer treatment and oral health

Most people are aware of common side effects of cancer treatment like nausea and hair loss. But many don't realize that more than one-third of people treated for cancer develop complications that affect the mouth. These problems may interfere with cancer treatment and diminish the patient's quality of life.

Oral complications of cancer treatment

With more than 1.4 million new cases of cancer diagnosed each year and a shift to outpatient management, you will likely see some of these patients in your practice. Because cancer treatment can affect the oral tissues, you need to know about potential oral side effects. Preexisting or untreated oral disease can also complicate cancer treatment. Your role in patient management can extend benefits beyond the oral cavity.

Oral complications from radiation to the head and neck or chemotherapy for any malignancy can compromise patients' health and quality of life, and affect their ability to complete planned cancer treatment. For some patients, the complications can be so debilitating that they may tolerate only lower doses of therapy, postpone scheduled treatments or discontinue treatment entirely. Oral complications can also lead to serious systemic infections. Medically necessary oral care before, during and after cancer treatment can prevent or reduce the incidence and severity of oral complications, enhancing both patient survival and quality of life.

Oral complications related to cancer treatment

Oral complications of cancer treatment arise in various forms and degrees of severity, depending on the individual and the cancer treatment. Chemotherapy often impairs the function of bone marrow, suppressing the formation of white blood cells, red blood cells and platelets (myelosuppression). Some cancer treatments are described as stomatotoxic because they have toxic effects on the oral tissues. Both chemotherapy and radiation therapy come with specific complications. You will need to consider the possibility of these complications each time you evaluate a patient with cancer.

Head and neck radiation, chemotherapy, and blood and marrow transplantation can cause oral complications ranging from dry mouth to life-threatening infections.

Head and neck radiation therapy

Patients receiving radiation therapy to the head and neck are at risk for developing oral complications. Because of the risk of osteonecrosis in irradiated fields, oral surgery should be performed before radiation treatment begins.

Before head and neck radiation therapy

- Conduct a pretreatment oral health examination and prophylaxis.
- Schedule dental treatment in consultation with the radiation oncologist.
- Extract teeth in the proposed radiation field that may be a problem in the future.
- Prevent tooth demineralization and radiation caries:
  - Fabricate custom gel-applicator trays for the patient.
  - Prescribe a 1.1 percent neutral pH sodium fluoride gel or a 0.4 percent stannous, unflavored fluoride gel (not fluoride rinses).
  - Use a neutral fluoride for patients with porcelain crowns or resin or glass ionomer restorations.
  - Be sure that the trays cover all tooth structures without irritating the gingival or mucosal tissues.
  - Instruct the patient in home application of fluoride gel. Several days before radiation therapy begins, the patient should start a daily 10-minute application.
  - Have patients brush with a fluoride gel if using trays is difficult.
- Allow at least 14 days of healing for any oral surgical procedures.
- Conduct prosthetic surgery before treatment, because elective surgical procedures are contraindicated on irradiated bone.

During radiation therapy

- Monitor the patient's oral hygiene.
- Watch for muscositis and infection.
- Advise against wearing removable appliances during treatment.

After radiation therapy

- Recall the patient for prophylaxis and home-care evaluation every four to eight weeks or as needed for the first six months after cancer treatment.
- Reinforce the importance of optimal oral hygiene.
- Monitor the patient for trismus; check for pain or weakness in masticating muscles in the radiation field. Instruct the patient to

exercise three times a day, opening and closing the mouth as far as possible without pain; repeat 20 times.
- Consult with the oncology team about use of dentures and other appliances after muscositis subsides. Patients with friable tissues and xerostomia may not be able to wear them again.
- Watch for demineralization and caries. Lifelong, daily applications of fluoride gel are needed for patients with xerostomia.
- Advise against elective oral surgery on irradiated bone because of the risk of osteonecrosis. Tooth extraction, if unavoidable, should be conservative, using antibiotic coverage and possibly hyperbaric oxygen therapy.

Chemotherapy

The oral complications of chemotherapy depend upon the drugs used, the dosage, the degree of dental disease and the use of radiation. Chemoradiation therapy carries a significant risk for mucositis.

Before chemotherapy
- Conduct a pretreatment oral health examination and prophylaxis.
- Schedule dental treatment in consultation with the oncologist.
- Schedule oral surgery at least seven to 10 days before myelosuppresive therapy begins.
- Consult the oncologist before conducting any oral procedures in patients with hematologic cancers; do not conduct procedures in patients who are immunosuppressed or have thrombocytopenia.

During chemotherapy
- Consult the oncologist before any dental procedure, including prophylaxis.
- Ask the oncologist to order blood work 24 hours before oral surgery or other invasive procedures. Postpone when:
  - The platelet count is less than 75,000/mm3 or abnormal clotting factors are present.
  - Absolute neutrophil count is less than 1,000/mm3 or consider prophylactic antibiotics. (American Heart Association)
- Check for oral source of viral, bacterial or fungal infection in patients with fever of unknown origin.
- Encourage consistent oral hygiene measures.
- Consult the oncologist about the need for antibiotic prophylaxis before any dental procedures in patients with central venous catheters.

Normal complete blood count	
Red blood cells	Male: 4.7 – 6.1 million cells/mcL Female: 4.2 – 5.4 million cells/mcL
Hemoglobin	Male 13.8 – 17.2 gm/dL Female: 12.1 – 15.1 gm/dL
Hematocrit	Males 40.7 – 50.3 percent Female: 36.1 – 44.3 percent
Platelets	150,000 – 400,000/mm3
White blood cells	4,500 – 10,000 cells/mcL

Differential white blood cell (WBC) count		
Neutrophils (PMNs)	40 – 60 percent	(3000 – 6000/mm3)
Neutrophils (Bands)	0 – 3 percent	(0 – 300/mm3)
Eosinophils	1 – 4 percent	(50 – 400/mm3)
Basophils	0.5 – 1 percent	(15 – 15/mm3)
Lymphocytes	20 – 40 percent	(1200 – 3000/mm3)
Monocytes	2 – 8 percent	(100 – 600/mm3)
Absolute neutrophil count = WBC x (percent PMNs + percent bands). Source: A.D.A.M. Medical Encyclopedia (Internet) *http://www.nimnih.gov/medlineplus/ency/article/003643.htm*		

After chemotherapy
- Place the patient on a dental recall schedule when chemotherapy is completed and all side effects, including immunosuppression, have resolved.
- Confirm normal hematologic status prior to dental treatment.
- Ask whether the patient has received intravenous bisphosphonate therapy.

Oral complications common to both chemotherapy and radiation

- **Oral mucositis**: Inflammation and ulceration of the mucous membranes; can increase the risk for pain, oral and systemic infection, and nutritional compromise. Culture lesions to identify secondary infection. Prescribe topical anesthetics and system analgesics. Consult the oncologist about prescribing antimicrobial agents for known infections. Have the patient avoid rough-textured foods and report oral problems early.
- **Infection**: Viral, bacterial and fungal; results from myelosuppression, xerostomia and/or damage to the mucosa from chemotherapy or radiotherapy.
- **Xerostomia/salivary gland dysfunction**: Dryness of the mouth due to thickened, reduced or absent salivary flow; increases the risk of infection and compromises speaking, chewing and swallowing. Medications other than chemotherapy can also cause salivary gland dysfunction. Persistent dry mouth increases the risk for dental caries. Advise the patient to soften or thin foods with liquid, chew sugarless gum, or suck ice chips or sugar-free hard candies. Suggest using commercial saliva substitutes or prescribe a saliva stimulant.
- **Functional disabilities**: Impaired ability to eat, taste, swallow and speak because of mucositis, dry mouth, trismus and infection.
- **Taste alterations**: Changes in taste perception of foods, ranging from unpleasant to tasteless. Refer to a dietitian.
- **Nutritional compromise**: Poor nutrition from eating difficulties caused by mucositis, dry mouth, dysphagia and loss of taste.
- **Abnormal dental development**: Altered tooth development, craniofacial growth, or skeletal development in children secondary to radiotherapy and/or high doses of chemotherapy before age 9.
- **Etched enamel**: Advise the patient to rinse the mouth with water and baking soda solution after vomiting to protect enamel.

Other complications of chemotherapy

Neurotoxicity: Persistent, deep aching and burning pain that mimics a toothache, but for which no dental or mucosal source can be found. This complication is a side effect of certain classes of drugs, such as the vinca alkaloids. Provide analgesics or systemic pain relief.

Bleeding: Oral bleeding from the decreased platelets and clotting factors associated with the effects of therapy on bone marrow. Advise the patient to clean teeth thoroughly with a toothbrush softened in warm water; to avoid flossing the areas that are bleeding but to keep flossing the other teeth.

Other complications of radiation therapy

Demineralization and radiation caries: Prescribe daily fluoride gel applications before treatment starts. Continue for the patient's lifetime if changes in quality or quantity of saliva persist.

Radiation caries: Lifelong risk of rampant dental decay that may begin within three months of completing radiation treatment if changes in either the quality or quantity of saliva persist.

Trimus/tissue fibrosis: Loss of elasticity of masticatory muscles that restrict normal ability to open the mouth. Instruct the patient on stretching exercises for the jaw to prevent or reduce the severity of fibrosis.

Osteonecrosis: Blood vessel compromise and necrosis of bone exposed to high-dose radiation therapy; results in decreased ability to heal if traumatized. Avoid invasive procedures involving irradiated bone, particularly the mandible.

Who has oral complications?

Oral complications occur in virtually all patients receiving radiation for head and neck malignancies, in approximately 80 percent of hematopoietic (blood-forming) stem cell transplant recipients, and in nearly 40 percent of patients receiving chemotherapy. Risk for oral complications can be classified as low or high:

- **Lower risk**: Patients receiving minimally myelosuppressive or nonmyelosuppressive chemotherapy.
- **Higher risk**: Patients receiving stomatotoxic chemotherapy resulting in prolonged myelosuppression, including patients undergoing hematopoietic stem cell transplantation; and patients undergoing head and neck radiation for oral, pharyngeal and laryngeal cancer.

Some complications occur only during treatment; others, such as xerostomia, may persist for years. Unfortunately, patients with cancer often do not receive oral care until serious complications develop.

The role of pretreatment oral care

A thorough oral evaluation by a knowledgeable dentist before cancer treatment begins is important to the success of the regimen. Pretreatment oral care achieves the following:

- Reduces the risk and severity of oral complications.
- Allows for prompt identification and treatment of existing infections or other problems.
- Improves the likelihood that the patient will successfully complete planned cancer treatment.
- Prevents, eliminates or reduces oral pain.
- Minimizes oral infections that could lead to potentially serious systemic infections.
- Prevents or minimizes complications that compromise nutrition.
- Prevents or reduces later incidence of bone necrosis.
- Preserves or improves oral health.
- Provides an opportunity for patient education about oral hygiene during cancer therapy.
- Improves the quality of life.
- Decreases the cost of care.

With a pretreatment oral evaluation, the dental team can identify and treat problems such as infection, fractured teeth or restorations, or periodontal disease that could contribute to oral complications when cancer therapy begins. The evaluation also establishes baseline data for comparing the patient's status in subsequent examinations.

Before the exam, you will need to obtain the patient's cancer diagnosis and treatment plan, medical history and dental history. Open communication with the patient's oncologist is essential to ensure that each provider has the information necessary to deliver the best possible care.

Patient evaluation

Ideally, a comprehensive oral evaluation should take place one month before cancer treatment starts to allow adequate time for recovery from any required invasive dental procedures. The pretreatment evaluation includes a thorough examination of hard and soft tissues as well as appropriate radiographs to detect possible sources of infection and pathology. Also take the following steps before cancer treatment begins:

- Identify and treat existing infections, carious and other compromised teeth, and tissue injury or trauma.
- Stabilize or eliminate potential sites of infection.
- Extract teeth in the radiation field that are nonrestorable or may pose a future problem to prevent later extraction-induced osteonecrosis.
- Conduct a prosthodontic evaluation if indicated. If a removable prosthesis is worn, make sure that it is clean and well adapted to the tissue. Instruct the patient not to wear the prosthesis during treatment, if possible; or at the least, not to wear it at night.
- Perform oral prophylaxis if indicated.
- Time oral surgery to allow at least two weeks for healing before radiation therapy begins. For patients receiving radiation treatment, this is the best time to consider surgical procedures. Oral surgery should be performed at least seven to 10 days before the patient receives myelosuppressive chemotherapy. Medical consultation is indicated before invasive procedures.
- Remove orthodontic bands and brackets if highly stomatotoxic chemotherapy is planned or if the appliances will be in the radiation field.
- Consider extracting highly mobile primary teeth in children, and teeth that are expected to exfoliate during treatment.
- Prescribe an individualized oral hygiene regimen to minimize oral complications. Patients undergoing head and neck radiation therapy should be instructed on the use of supplemental fluoride.

Supplemental fluoride

Fluoride rinses are not adequate to prevent tooth demineralization. Instead, a high-potency fluoride gel, delivered via custom gel-applicator trays, is recommended. Several days before radiation therapy begins, patients should start a daily 10-minute application of a 1.1 percent neutral pH sodium fluoride gel or a 0.4 percent stannous fluoride (unflavored) gel. Patients with porcelain crowns or resin or glass ionomer restorations should use a neutral pH fluoride. Be sure that the trays cover all tooth structures without irritating the gingival or mucosal tissues.

For patients reluctant to use a tray, a high-potency fluoride gel should be brushed on the teeth following daily brushing and flossing. Either 1.1 percent neutral pH sodium or 0.4 percent stannous fluoride gel is recommended, based on the patient's type of dental restorations.

Questions to ask the medical oncologist

1. What is the patient's complete blood count, including absolute neutrophil and platelet counts?
2. If an invasive dental procedure needs to be done, are there adequate clotting factors?
3. Does the patient have a central venous catheter?
4. What is the scheduled sequence of treatments so that safe dental treatment can be planned?
5. Is radiation therapy also planned?

Questions to ask the radiation oncologist

1. What parts of the mandible/maxilla and salivary glands are in the field of radiation?
2. What is the total dose of radiation the patient will receive, and what will be the impact on these areas?
3. Has the vascularity of the mandible been previously compromised by surgery?
4. How quickly does the patient need to start radiation treatment?
5. Will there be induction chemotherapy with the radiation treatment?

Hematopoietic stem cell transplantation

Most stem cell transplant patients develop acute oral complications, especially patients with graft-versus-host disease.

Before transplantation

- Conduct a pretreatment oral health examination and prophylaxis.
- Consult the oncologist about scheduling dental treatment.
- Schedule oral surgery at least seven to 10 days before myelosuppressive therapy begins.
- Prevent tooth demineralization and radiation caries:
  - Instruct the patient in home application of fluoride gel (not fluoride rinses).
  - Explain the necessary oral hygiene regimen to the patient.

After transplantation

- Consult the oncologist before any dental procedure, including prophylaxis.
- Monitor the patient's oral health for plaque control, tooth demineralization, dental caries and infection.
- Watch for infections on the tongue and oral mucosa. Herpes simplex and Candida albicans are common oral infections.
- Delay elective oral procedures for one year.
- Follow patients for long-term oral complications. Such problems are strong indicators of chronic graft-versus-host disease.
- Monitor transplant patients carefully for second malignancies in the oral region.

Advice for your patients

- Brush teeth, gums and tongue gently with an extra-soft toothbrush and fluoride toothpaste after every meal and at bedtime. If brushing hurts, soften the bristles in warm water.
- Floss teeth gently every day. If gums bleed and hurt, avoid the areas that are bleeding or sore but keep flossing your other teeth.
- Follow instructions for fluoride gel applications.
- Avoid mouthwashes containing alcohol.
- Rinse the mouth several times a day with a baking soda and salt solution, followed by a plain water rinse. Use ¼ teaspoon each of baking soda and salt in 1 quart of warm water. Omit salt during mucositis.
- Try the following if dry mouth is a problem:
  - Sip water frequently.
  - Suck ice chips or use sugar-free gum or candy.
  - Use saliva substitute spray or gel or a prescribed saliva stimulant if appropriate.
  - Avoid glycerin swabs.
- Exercise the jaw muscles three times a day to prevent and treat jaw stiffness from radiation treatment.
- Avoid candy, gum and soda unless they are sugar-free.
- Avoid spicy or acidic foods, toothpicks, tobacco products and alcohol.

Special care for children

Children receiving chemotherapy and/or radiation therapy are at risk for the same oral complications as adults. Other actions to consider in managing pediatric patients include:

- **Before the cancer treatment begins**, extract loose primary teeth and teeth expected to exfoliate during cancer treatment.
  - Remove orthodontic bands and brackets if highly stomatotoxic chemotherapy is planned or if the appliances will be in the radiation field.
- **Monitor craniofacial and dental structures** for abnormal growth and development.

Oral complications and the heart with infective endocarditis

Endocarditis is a sometimes life-threatening infection of the inner surface of the heart and/or its valves. Of the approximately 15,000 cases of endocarditis reported each year in the United States, many likely arise when bacteria that naturally attach to our teeth are displaced and pass into the bloodstream during a dental procedure, flossing, or even chewing food.

These microbes, while relatively harmless in the mouth, have an affinity for damaged endothelial cells or blood clots in the heart, where they attach, multiply and form larger bacterial colonies that trigger the endocarditis. Scientists have shown that immune cells called monocytes are prominently found in early inflammatory lesions linked to endocarditis. What's been puzzling is the monocytes tend to disappear from the lesions over time without becoming macrophages, a scavenging immune cell formed from monocytes that removes debris from tissues, such as the damaged, bacteria-laden cells linked to endocarditis.

In a report in the journal Infection and Immunity, NIDCR grantees show that the usual monocyte-macrophage transformation rarely occurs because monocytes infected in studies with the well-known oral bacterium Streptococcus mutans instead become dendritic cells, a type of immune cell that initiates an inflammation-producing immune response upon interaction with this bacterium. This finding indicates that oral streptococci-mediated changes in a person's normal immune response can contribute to endocarditis. It also suggests that an effective future strategy to treat endocarditis might involve learning to turn off the destructive immune response and/or reprogram the monocytes to produce macrophages to clear away the disease-causing bacterial colonies from the heart.

Endocarditis is an infection of the inner lining of the heart chambers and valves. This lining is called the endocardium. The condition also is called infective endocarditis (IE).

Infective endocarditis occurs if bacteria, fungi, or other germs invade the bloodstream and attach to abnormal areas of the heart. The infection can damage the heart and cause serious and sometimes fatal complications. It can develop quickly or slowly; it depends on what

type of germ is causing it and whether the patient has an underlying heart problem. When it develops quickly, it is called acute infective endocarditis. When it develops slowly, it is called subacute infective endocarditis.

The disease mainly affects people who have:
- Damaged or artificial (man-made) heart valves.
- Congenital heart defects (defects present at birth).
- Implanted medical devices in the heart or blood vessels.

People who have normal heart valves also can have this disease. However, the condition is much more common in people who have abnormal hearts.

Certain factors make it easier for bacteria to enter the bloodstream. These factors put the patient at higher risk for infective endocarditis. For example, poor dental hygiene and unhealthy teeth and gums increase the risk for the infection.

Other risk factors include using intravenous (IV) drugs, having a catheter (tube) or another medical device in the body for long periods, and having a history of infective endocarditis.

Common symptoms of infective endocarditis are fever and other flu-like symptoms. Because the infection can affect people in different ways, the signs and symptoms vary. The disease may cause problems in many other parts of the body besides the heart.

What causes endocarditis?

The disease occurs if bacteria, fungi or other germs invade the bloodstream and attach to abnormal areas of the heart. A common underlying factor in infective endocarditis is a structural heart defect, especially faulty heart valves. Usually the immune system will kill germs in the bloodstream. If the heart has a rough lining or abnormal valves, the invading germs can attach and multiply in the heart.

Other factors also can play a role in causing the disease. Common activities, such as brushing the teeth or having certain dental procedures, can allow bacteria to enter the bloodstream. This is even more likely to happen if the teeth and gums are in poor condition.

Having a catheter, tube or other medical device inserted through the skin, especially for long periods, can allow bacteria to enter the bloodstream. People who use intravenous (IV) drugs also are at risk for infective endocarditis because of the germs on needles and syringes.

Bacteria may spread to the blood and heart from infections in other parts of the body such as the gut, skin or genitals.

Endocarditis complications

- This disease can cause many complications; the most common is problems with the heart. They occur in one-third to one-half of all people who have the infection. These problems may include:
  - A heart murmur.
  - Heart failure.
  - Heart valve damage.
  - Heart block.
  - Heart attack.
- Complications of the central nervous system occur in as many as 20 to 40 percent of people who have infective endocarditis. The central nervous system complications most often occur when bits of the vegetation called emboli break away and lodge in the brain. The emboli can cause local infections called brain abscesses. They also can cause a more widespread brain infection called meningitis.
  - Emboli can cause strokes or seizures. This happens if they block blood vessels or affect the brain's electrical signals. These complications can cause long-term brain damage or even be fatal.
- Infective endocarditis can affect other organs in the body, such as the lungs, kidneys and spleen.
  - The lungs are at risk when the endocarditis affects the right side of the heart.
  - The kidneys can become abscessed and the infection can inflame the internal filtering structures of the kidneys.
  - The spleen can become enlarged, especially in people with long-term infective endocarditis. Sometimes emboli can damage the spleen.

How can endocarditis be prevented?

To help your patients prevent endocarditis, always take steps to maintain infection control in your office and advise your patients to:
- Let you know if they are at risk for endocarditis. (These patients may need an antibiotic before routine dental exams and certain other dental and medical procedures.)
- Brush and floss their teeth regularly.
- Have regular dental checkups.
- Avoid body piercing, tattoos and other procedures that may allow germs to enter the bloodstream.
- Be alert to the signs and symptoms of infective endocarditis. (Advise the patient to see a medical doctor if any of these symptoms persist.)
  - Flu-like symptoms, such as fever, chills, fatigue (tiredness), aching muscles and joints, night sweat and headaches.
  - Shortness of breath or a cough that won't go away.
  - A new heart murmur or a change in an existing heart murmur.
  - Skin changes such as:
    - Overall paleness.
    - Small, painful, red purplish bumps under the skin on the fingers or toes.
    - Tiny spots under the fingernails, on the whites of the eyes, on the roof of the mouth and inside of the cheeks, or on the chest. These spots are from broken blood vessels.
  - Nausea, vomiting, a decrease in appetite, a sense of fullness with discomfort on the upper left side of the abdomen or weight loss with or without a change in appetite.
  - Blood in the urine.
  - Swelling in the feet, legs or abdomen.

Sjögren's syndrome

In the early 20th century, Swedish physician Henrik Sjögren (SHOW-gren) first described a group of women whose chronic arthritis was accompanied by dry eyes and dry mouth. Today rheumatologists know more about the syndrome that is named for Sjögren and, most significantly for patients, can provide advice about how to live with it.
- Sjögren's syndrome sometimes develops as a complication of another autoimmune disorder.

- Symptoms vary in type and intensity, but many people with Sjögren's are able to live normal lives.
- Although serious complications are rare, regular medical care is important.

What is Sjögren's syndrome?

Sjögren's syndrome is an inflammatory disease that can affect many different parts of the body, but most often affects the tear and saliva glands. Patients with this condition may notice irritation, a gritty feeling or painful burning in the eyes. Dry mouth or difficulty eating dry foods and swelling of the glands around the face and neck are also common. Some patients experience dryness of other mucous membranes (such as the nasal passages, throat and vagina) and skin.

"Primary" Sjögren's syndrome occurs in people with no other rheumatologic disease. "Secondary" Sjögren's occurs in people who do have another rheumatologic disease, most often lupus and rheumatoid arthritis.

Most of the complications of Sjögren's syndrome occur because of decreased tears and saliva. Patients with dry eyes are at increased risk for infections around the eye and may have damage to the cornea. Dry mouth may cause an increase in dental decay, gingivitis (gum inflammation), and oral yeast infections (thrush) that may cause pain and burning. Some patients have episodes of painful swelling in the saliva glands around the face.

Complications in other parts of the body occur rarely in patients with Sjögren's syndrome. Pain and stiffness in the joints with mild swelling may occur in some patients, even in those without rheumatoid arthritis or lupus. Rashes on the arms and legs related to inflammation in small blood vessels (vasculitis) and inflammation in the lungs, liver, and kidney may occur rarely and be difficult to diagnose. Neurological complications that cause symptoms such as numbness, tingling and weakness have also been described in some patients.

What causes Sjögren's syndrome?

The cause of Sjögren's syndrome is not known, but it is considered an autoimmune disorder. People with this disease have abnormal proteins in their blood suggesting that their immune system, which normally functions to protect the body against cancers and invading infections, is reacting against their own tissue. The decreased production of tears and saliva seen in Sjögren's syndrome occurs when the glands that produce these fluids are damaged by inflammation. Research suggests that genetic factors and possibly viral infections (as yet unidentified) may predispose people to developing this condition.

Who gets Sjögren's syndrome?

Between 400,000 and 3.1 million adults have Sjögren's syndrome. This condition can affect people of any age, but symptoms usually appear between the ages of 45 and 55. It affects 10 times as many women as men. About half of affected patients also have rheumatoid arthritis or other connective tissue diseases, such as lupus.

How is Sjögren's syndrome diagnosed?

Diagnosis depends on a combination of symptoms, physical findings, blood tests and sometimes special studies. Dry eyes and mouth may be early signs of the condition but require further investigation because these symptoms can be caused by many other conditions or medications. Special tests may be used to assess any decrease in tear or saliva production (an example would be the Schirmer test for tear production). An eye examination is helpful in detecting any eye changes seen in Sjögren's. Blood tests can determine the presence of antibodies (immune system cells that help destroy foreign invaders) typical of the disease, including anti-nuclear antibodies (ANA), anti-SSA and SSB antibodies, or rheumatoid factor. Biopsies of saliva glands around the face or under the surface of the inner lip may also sometimes be used to establish a diagnosis.

How is Sjögren's syndrome treated?

Treatment is designed to lessen the most bothersome symptoms. Dry eyes usually respond to the use of artificial tears applied regularly during the day or to gels applied at night. Other measures, such as plugging or blocking tear ducts, can be used in more severe cases. Eye drops that reduce inflammation in the glands around the eyes (cyclosporine-Restasis) may be used to increase tear production. Dry mouth can be relieved by drinking water, chewing gum or using saliva substitutes. Some patients benefit from using prescription medications that stimulate saliva flow, such as pilocarpine (Salagen) or cevimuline (Evoxac). If patients develop yeast infections, these can be relieved by anti-fungal therapies. The currently available treatments may help relieve some of the dryness, but usually some dryness persists.

All patients should receive regular dental care in order to prevent cavities and tooth loss that may occur as a complication of the disorder. Patients with dry eyes should see an ophthalmologist regularly for signs of damage to the cornea. Patients with excessive redness and pain in the eyes should be evaluated for infections.

Hydroxychloroquine (Plaquinel), an antimalarial drug used in lupus and rheumatoid arthritis, may be helpful in some patients with Sjögren's syndrome by reducing joint pain and rash experienced by some patients. Patients with rare but serious systemic symptoms, such as fever, rashes, abdominal pain or lung or kidney problems, may require treatment with corticosteroids such as prednisone (Deltasone and others) and/or immunosuppressive agents, such as methotrexate (Rheumatrex), azathioprine (Imuran), mycophenolate (CellCept), cyclophosphamide (Cytoxan). In addition, rituximab (Rituxan) and other biological therapies (as used in rheumatoid arthritis) are undergoing evaluation for treating patients with severe systemic manifestations of disease.

Broader health impact of Sjögren's syndrome

A vast majority of patients with Sjögren's syndrome remain very healthy, without any serious complications. Patients should be aware that they do face an increased risk for infections in and around the eyes and an increased risk for dental problems – both of which are due to the long-term reduction in tears and saliva.

Rarely, patients may have complications related to inflammation in other body systems, including:

- Joint and muscle pain with fatigue.
- Lung problems that may mimic pneumonia.
- Abnormal liver and kidney function tests.

- Skin rashes related to inflammation of small blood vessels.
- Neurologic problems causing weakness and numbness.

In a small number of people, Sjögren's syndrome may be associated with lymphoma, a cancer of the lymph glands.

Living with Sjögren's syndrome

People with Sjögren's syndrome are usually able to live normal lives with very few adjustments. When a diagnosis is made, many patients must focus a great deal of attention dealing with dry eyes and dry mouth, but these symptoms tend to subside with time. Any pain or redness in the eyes should be evaluated promptly, because this may signal an infection. To reduce risk for cavities and other dental problems, patients must pay close attention to proper oral hygiene and regular dental care.

Patients should see their physician regularly for general health screening and should pay close attention to any abnormal swelling in the glands around the face or neck, under the arms or in the groin areas because this may be a sign of lymphoma.

Sjögren's syndrome is an autoimmune condition that can occur at any age, but is most common in older women. Many patients develop Sjögren's syndrome as a complication of another autoimmune disease, such as rheumatoid arthritis or lupus.

Most of the treatment for Sjögren's syndrome is aimed at relieving symptoms of dry eyes and mouth and preventing and treating long-term complications such as infection and dental disease. Currently available treatments often do not completely eliminate the symptoms of dryness in some patients.

Most patients with Sjögren's syndrome remain healthy, but a number of rare complications have been described, including an increased risk for cancer of the lymph glands (lymphoma). Thus, regular medical care and follow-up is important for all patients.

Mouth problems with human immunodeficiency virus (HIV)

Human immunodeficiency virus is a lentivirus (a member of the retrovirus family) that causes acquired immunodeficiency syndrome (AIDS), a condition in humans in which the immune system begins to fail, leading to life-threatening opportunistic infections. Infection with HIV occurs by the transfer of blood, semen, vaginal fluid, pre-ejaculate or breast milk. Within these bodily fluids, HIV is present as both free virus particles and virus within infected immune cells.

HIV infection in humans is considered pandemic by the World Health Organization (WHO). Nevertheless, complacency about HIV may play a key role in HIV risk. From its discovery in 1981 to 2006, AIDS killed more than 25 million people. HIV infects about 0.6 percent of the world's population. In 2005 alone, AIDS claimed an estimated 2.4 million to 3.3 million lives, of which more than 570,000 were children.

Oral problems are very common in people with HIV. More than a third of people living with HIV have oral conditions that arise because of their weakened immune system. And even though combination antiretroviral therapy has made some oral problems less common, others are occurring more often with this type of treatment. These problems can be very painful, annoying and lead to other problems. Oral problems can also lead to trouble with eating, as well as cause discomfort and embarrassment. If the mouth is in pain and has tenderness, it becomes difficult to chew and swallow, and the patient may not eat enough, causing weight loss and other complications. The body may not have enough energy to deal with HIV.

Some of the most common oral problems linked with HIV can be treated; they include:

- **Apthous ulcers (canker sores)**: Red sores that might also have a yellow-gray film on top. They are usually on the moveable parts of the mouth, such as the tongue or inside of the cheeks and lips.
- **Herpes**: A viral infection, red sores usually on the roof of the mouth. They are sometimes on the outside of the lips, where they are called fever blisters.
- **Hairy leukoplakia**: This is caused by the Epstein-Barr virus. They are white patches that do not wipe away and are sometimes very thick and hairlike. They usually appear on the side of the tongue or sometimes on the cheeks and lower lip.
- **Candidiasis**: This is a fungal yeast infection that produces white or yellowish patches (sometimes red). If wiped away, there will be redness or bleeding underneath. They can appear anywhere in the mouth.
- **Warts**: Small, white, gray or pinkish rough bumps that look like cauliflower. They can appear inside the lips and on other parts of the mouth.
- **Dry mouth xerostomia/salivary gland dysfunction**: This happens when the patient does not have enough saliva to keep the mouth wet. Without enough saliva, the patient could develop tooth decay or other infections and might have trouble chewing and swallowing. This can cause the mouth to be very dry, with a burning feeling, cracked and chapped lips. (See the section below on xerostomia).

Treatment for these include:

- **Infection control.**
  - Brush and floss regularly with a soft bristle toothbrush.
- **Apthous ulcers or canker sores**: An over-the-counter cream or prescription mouthwash that contains corticosteroids; for more severe cases, use corticosteroids pills.
- **Herpes**: Antiviral medications can reduce the healing time and frequency of outbreaks.
- **Hairy leukoplakia**: Antivirals for the more severe cases may reduce symptoms. In some cases, a pain reliever may be required.
- **Candidiasis**: A mild prescription for an antifungal lozenge or mouthwash. An antifungal pill might be necessary for the more severe cases.
- **Warts in the mouth**: These can be removed surgically or by cryosurgery. A prescription cream may be used for treatment. The warts may return after treatment.

Oral complications with xerostomia/salivary gland dysfunction or dry mouth

Dry mouth is a complication of many of these diseases, but it is also a symptom of a gland dysfunction.

Dry mouth is the feeling that there is not enough saliva in the mouth. Everyone has dry mouth occasionally, often when they are nervous, upset, under stress or taking certain medications. Many older adults have dry mouth, but it is not a normal part of aging.

Saliva does more than keep the mouth wet. It protects teeth from decay, it helps heal sores in the mouth and prevents infection by controlling bacteria, viruses and fungi in the mouth.

Saliva helps digest food and helps us chew and swallow. Saliva is involved in taste perception as well. Each of these functions of saliva is hampered when a person has dry mouth. It can be very uncomfortable. Some people notice a sticky, dry feeling in the mouth. Others notice a burning feeling or difficulty while eating. It may cause the throat to feel dry, making swallowing difficult and choking

common. People with dry mouth may get sores, cracked lips and a dry, rough tongue.

People get dry mouth when the glands in the mouth that make saliva are not working properly and do not produce enough saliva to keep the mouth healthy. There are several reasons why salivary glands might not work right.

More than 400 medicines, including some over-the-counter medications, can cause the salivary glands to make less saliva, or to change the composition of the saliva so that it can't perform the functions it should. As an example, medicines for urinary incontinence, allergies, high blood pressure and depression often cause dry mouth.

Some diseases can affect the salivary glands. Dry mouth can occur in patients with diabetes and Parkinson's disease. Dry mouth is the hallmark symptom of the fairly common autoimmune disease Sjögren's syndrome.

Sjögren's syndrome can occur either by itself or with another autoimmune disease like rheumatoid arthritis or lupus. Salivary and tear glands are the major targets of the syndrome, and the result is a decrease in production of saliva and tears. The disorder can occur at any age, but the average person with the disorder at the Sjögren's Syndrome Clinic of the National Institute of Dental and Craniofacial Research (NIDCR) is in his or her late 50s. Women with the disorder outnumber men 9 to 1.

Certain cancer treatments can affect the salivary glands. Head and neck radiation therapy can cause the glands to produce little or no saliva. Chemotherapy may cause the salivary glands to produce thicker saliva, which makes the mouth feel dry and sticky.

Injury to the head or neck can damage the nerves that tell salivary glands to make saliva.

How is dry mouth treated?

- First try to determine the cause.
- Change the patient's medication or dosage if this is the cause.
- Prescribe a medicine for the salivary glands, if this is the cause.
- Suggest the use of artificial saliva to keep the mouth wet.
- Suggest the patient drink lots of water and sugarless drinks.
- Instruct patients to avoid caffeine drinks such as coffee, tea and some sodas. (Caffeine can dry out the mouth.)
- Tell patients to avoid tobacco or alcohol, which will dry out the mouth.
- Suggest that the patient chew sugarless gum or suck on sugarless hard candy to stimulate saliva flow.
- Tell the patient to stay away from spicy or salty foods because they will cause pain in a dry mouth.
- Suggest using a humidifier at night to promote moisture in the air while the patient is sleeping.

Scientists are exploring the potential use of gene therapy – replacing, manipulating or supplementing nonfunctional genes with healthy genes – to treat salivary gland dysfunction. The idea is to transfer additional or replacement genes into the salivary glands of people with Sjögren's syndrome and cancer patients whose salivary glands are damaged by radiation treatment. The hope is that these genes will increase the production of saliva and eliminate the chronic parched sensation that bothers people with dry mouth conditions.

Research efforts are also under way to develop an artificial salivary gland for patients who have lost all salivary gland function. The first-generation artificial gland will be a tiny tube lined with cells that have been engineered to produce saliva-like fluid. Made of biodegradable material, the tube would be inserted into the inside of the cheek. All the components for the artificial gland have been developed with the goal of producing a prototype within a few years.

Advise patients to take these steps to prevent xerostomia/salivary gland dysfunction or dry mouth:

- Brush teeth several times a day with an extra-soft toothbrush, at least after every meal and at bedtime. If brushing hurts, soften the bristles in warm water.
- Floss teeth gently every day. If gums bleed, avoid the areas that are bleeding and sore.
- Always use toothpaste with fluoride.
- Avoid sticky, sugary foods. Brush immediately eating.
- Do not use mouthwashes with alcohol in them. Alcohol can dry out the mouth.
- If necessary, prescribe a fluoride gel to help prevent dental decay.

Conclusion

There are many causes for oral complications, and the best way to help your patients is to stay informed about the types of diseases, treatments and ways to reduce the risk and impact of these often painful side effects that diminish their quality of life. Good oral health not only makes them feel better, it also makes them look better and elevates their self-esteem, a very important factor.

But the most important fact to remember is that good health begins in the mouth, and that saliva carries germs through the bloodstream, which can cause complications. Patients with these diseases have been through so much that they tend to ignore or "let go" their dental care. As a dental professional, you can help to prevent these complications with a health care strategy and advice on necessary home-care treatment, thus removing at least one problem for people facing such serious diseases.

ORAL COMPLICATIONS WITH DISEASES

Final Examination Questions

Select the best answer questions 21 through 25 and mark your answers on the Final Examination Answer Sheet found on page 148, or for faster service complete your test online at **Dental.EliteCME.com.**

21. People with diabetes are not at risk for mouth infections such as thrush or dry mouth.
    - ○ True
    - ○ False

22. Supplemental fluoride rinses are adequate to prevent tooth demineralization for patients undergoing cancer treatments.
    - ○ True
    - ○ False

23. After hematopoietic stem cell transplantation, you should monitor patients carefully for second malignancies in the oral region.
    - ○ True
    - ○ False

24. Infective endocarditis prevention includes avoiding body piercing or tattoos that can allow germs to enter the bloodstream.
    - ○ True
    - ○ False

25. Saliva protects teeth from decay, heals sores in the mouth and prevents infection by controlling bacteria, viruses and fungi in the mouth.
    - ○ True
    - ○ False

DOH03OCE15

Chapter 6: Oral Diseases, Infections and Craniofacial Disorders

9 CE Hours

By: Elite Staff

Learning objectives

- Explain new research and findings regarding the connection between oral health and health.
- Learn about the six major dental diseases.
- Discuss the connection between heart disease, diabetes and oral infections.
- Review some effective disease preventative measures.
- Learn about craniofacial disorders.
- List factors affecting future dental health care practices.

Introduction

The realization that oral health can have a significant impact on the overall health and well-being of the nation's population has become a major issue in the world of science and research. Realizing the gains that have been made in disease prevention, while acknowledging that there are populations that suffer disproportionately from oral health problems, the purpose of this course is to help "define, describe and evaluate the interaction between oral health and health and well-being [quality of life], through the life span in the context of changes in society." Key elements to be addressed are the determinants of health and disease, with a primary focus on prevention and "producing health" rather than "restoring health"; a description of the burden of oral diseases and disorders in the nation; and the evidence for actions to improve oral health to be taken across the life span.

Oral health

The mouth includes not only the teeth and the gums (gingiva) and their supporting tissues, but also the hard and soft palate, the mucosal lining of the mouth and throat, the tongue, the lips, the salivary glands, the chewing muscles, and the upper and lower jaws. Equally important are the branches of the nervous, immune and vascular systems that animate, protect and nourish the oral tissues, as well as provide connections to the brain and the rest of the body. The genetic patterning of development in utero further reveals the intimate relationship of the oral tissues to the developing brain and to the tissues of the face and head that surround the mouth, structures whose location is captured in the word craniofacial.

A key element to discuss is that oral health means much more than healthy teeth. It means being free of chronic oral-facial pain conditions, oral and pharyngeal (throat) cancers, oral soft tissue lesions, birth defects such as cleft lip and palate, and scores of other diseases and disorders that affect the oral, dental and craniofacial tissues, collectively known as the craniofacial complex. These are tissues whose functions we often take for granted, yet they represent the very essence of our humanity. They allow us to speak and smile; sigh and kiss; smell, taste, touch, chew and swallow; cry out in pain; and convey a world of feelings and emotions through facial expressions. They also provide protection against microbial infections and environmental insults.

The craniofacial tissues also provide a useful means to understanding organs and systems in less accessible parts of the body. The salivary glands are a model of other exocrine glands, and an analysis of saliva can provide telltale clues of overall health or disease. The jawbones and their joints function like other musculoskeletal parts. The nervous system apparatus underlying facial pain has its counterpart in nerves elsewhere in the body. A thorough oral examination can detect signs of nutritional deficiencies as well as a number of systemic diseases, including microbial infections, immune disorders, injuries and some cancers. Indeed, the phrase the mouth is a mirror has been used to illustrate the wealth of information that can be derived from examining oral tissues.

New research is pointing to associations between chronic oral infections and heart and lung diseases, stroke and low-birth-weight, premature births. Associations between periodontal disease and diabetes have long been noted. Scientific reports assess these associations and explore mechanisms that might explain the oral-systemic disease connections.

The broadened meaning of oral health parallels the broadened meaning of health. In 1948, the World Health Organization expanded the definition of health to mean "a complete state of physical, mental and social well-being, and not just the absence of infirmity." It follows that oral health must also include well-being. Just as we now understand that nature and nurture are inextricably linked, and mind and body are both expressions of our human biology, so, too, we must recognize that oral health and general health are inseparable. We ignore signs and symptoms of oral disease and dysfunction to our detriment. Consequently, a second element of the course is that oral health is integral to general health. You cannot be healthy without oral health. Oral health and general health should not be interpreted as separate entities. Oral health is a critical component of health and must be included in the provision of health care and the design of community programs.

The wider meanings of oral and health in no way diminish the relevance and importance of the two leading dental diseases, caries and the periodontal diseases. They remain common and widespread, affecting nearly everyone at some point in the life span. What has changed is what we can do about them.

Researchers in the 1930s discovered that people living in communities with naturally fluoridated water supplies had fewer dental caries than people drinking unfluoridated water. But not until the end of World War II were the investigators able to design and implement the community clinical trials that confirmed their observations and launch a better approach to the problem of dental caries: prevention. Soon after, adjusting the fluoride content of community water supplies was pursued as an important public health measure to prevent dental caries.

Although this measure has not been fully implemented, the results have been dramatic. Dental caries began to decline in the 1950s among children who grew up in fluoridated cities, and by the late 1970s, decline in decay was evident for many Americans. The application of science to improve diagnostic, treatment and prevention strategies has saved billions of dollars per year in the nation's annual health bill. Even more significant, the result is that far fewer people are edentulous (toothless) today than a generation ago.

The theme of prevention gained momentum as pioneering investigators and practitioners in the 1950s and 1960s showed that not only dental caries but also periodontal diseases are bacterial infections. The researchers demonstrated that the infections could be prevented by increasing host resistance to disease and reducing or eliminating the suspected microbial pathogens in the oral cavity. The applications of research discoveries have resulted in continuing improvements in the oral health of Americans, new approaches to the prevention and treatment of dental diseases, and the growth of the science.

The significant role that scientists, dentists, dental hygienists and other health professionals have played in the prevention of oral disease and disability leads to a third theme of this course: safe and effective disease prevention measures exist that everyone can adopt to improve oral health and prevent disease. These measures include daily oral hygiene procedures and other lifestyle behaviors, community programs such as community water fluoridation and tobacco cessation programs, and provider-based interventions such as the placement of dental sealants and examinations for common oral and pharyngeal cancers.

General health risk factors, such as tobacco use and poor dietary practices, also affect oral and craniofacial health. The evidence for an association between tobacco use and oral diseases has been clearly delineated in almost every surgeon general's report on tobacco since 1964, and the oral effects of nutrition and diet are presented in the surgeon general's report on nutrition (1988). All the dental professions can play a role in reducing the burden of disease in America by calling attention to these and other risk factors and suggesting appropriate actions.

Clearly, promoting health and preventing diseases are concepts the American people have taken to heart. As a nation, we hope to eliminate disparities in health and prevent oral diseases, cancer, birth defects, AIDS and other devastating infections; mental illness and suicide; and the chronic diseases of aging. To live well into old age free of pain and infirmity and with a high quality of life is the American dream.

Scientists today take that dream seriously in researching the intricacies of the craniofacial complex. They are using an ever-growing array of sophisticated analytic tools and imaging systems to study normal function and diagnose disease. They are completing the mapping and sequencing of human, animal, microbial and plant genomes, the better to understand the complexities of human development, aging and pathological processes. They are growing cell lines, synthesizing molecules and using a new generation of biomaterials to revolutionize tissue repair and regeneration. More than ever before, they are working in multidisciplinary teams to bring new knowledge and expertise to the goal of understanding complex human diseases and disorders.

Oral diseases and disorders in and of themselves affect health and well-being throughout life.

The burden of oral problems is extensive and may be particularly severe in vulnerable populations. It includes the common dental diseases and other oral infections such as cold sores and candidiasis that can occur at any stage of life, as well as birth defects in infancy and the chronic facial pain conditions and oral cancers seen in later years. Many of these conditions and their treatments may undermine self-image and self-esteem, discourage normal social interaction, cause other health problems, and lead to chronic stress and depression as well as incur great financial cost. They may also interfere with vital functions such as breathing, food selection, eating, swallowing and speaking and with activities of daily living such as work, school and family interactions.

Safe and effective measures exist to prevent the most common dental diseases – dental caries and periodontal diseases. Community water fluoridation is safe and effective in preventing dental caries in both children and adults. Water fluoridation benefits all residents served by community water supplies regardless of their social or economic status. Professional and individual measures, including the use of fluoride mouth rinses, gels, dentifrices and dietary supplements and the application of dental sealants, are additional means of preventing dental caries. Gingivitis can be prevented by good personal oral hygiene practices, including brushing and flossing.

Lifestyle behaviors that affect general health such as tobacco use, excessive alcohol use and poor dietary choices affect oral and craniofacial health as well.

These individual behaviors are associated with increased risk for craniofacial birth defects, oral and pharyngeal cancers, periodontal disease, dental caries and candidiasis, among other oral health problems. Opportunities exist to expand the oral disease prevention and health promotion knowledge and practices of the public through community programs and in health care settings. All health care providers can play a role in promoting healthy lifestyles by incorporating tobacco cessation programs, nutritional counseling and other health promotion efforts into their practices.

There are profound and consequential oral health disparities within the U.S. population. Disparities for various oral conditions may relate to income, age, sex, race or ethnicity, or medical status. Although common dental diseases are preventable, not all members of society are informed about or able to avail themselves of appropriate oral-health-promoting measures. Similarly, not all health providers may be aware of the services needed to improve oral health. In addition, oral health care is not fully integrated into many care programs. Social, economic and cultural factors and changing population demographics affect how health services are delivered and used, and how people care for themselves. Reducing disparities requires wide-ranging approaches that target populations at highest risk for specific oral diseases and involves improving access to existing care. One approach includes making dental insurance more available to Americans. Public coverage for dental care is minimal for adults, and programs for children have not reached the many eligible beneficiaries.

The mouth reflects general health and well-being. The mouth is a readily accessible and visible part of the body and provides health care providers and individuals with a window on their general health status. As the gateway of the body, the mouth senses and responds to the external world and at the same time reflects what is happening deep inside the body. The mouth may show signs of nutritional deficiencies and serve as an early warning system for diseases such as HIV infection and other immune system problems. The mouth can also show signs of general infection and stress. As the number of substances that can be reliably measured in saliva increases, it may well become the diagnostic fluid of choice, enabling the diagnosis of specific disease as well as the measurement of the concentration of a variety of drugs, hormones and other molecules of interest. Cells and fluids in the mouth may also be used for genetic analysis to help uncover risks for disease and predict outcomes of medical treatments.

Oral diseases and conditions are associated with other health problems. Oral infections can be the source of systemic infections in people with weakened immune systems, and oral signs and symptoms often are part of a general health condition. Associations between chronic oral infections and other health problems, including diabetes, heart disease, and adverse pregnancy outcomes, have also been reported. Ongoing research may uncover mechanisms that strengthen the current findings and explain these relationships.

Touch, temperature and pain

The mouth also contains large numbers of nerve endings, similar to those found elsewhere in the body, that are sensitive to touch (mechanoreceptors), hot and cold temperatures (thermoreceptors) and pain (nociceptors). The dense concentration of these receptors in the facial skin, joints, muscle and oral soft tissues, relayed to an image of the body mapped onto the sensory cortex of the brain, accounts for the finesse with which we can discriminate the qualities and precise location of these sensations. In particular, the periodontal ligament, which anchors the teeth in the jaws, is a tactilely sensitive tissue providing important feedback with regard to mastication and dental occlusion. As a test of this sensibility, a human hair placed between the tips of the fingers will rarely be sufficient to stimulate the nerve endings, but the same hair placed between the lips or incisors will instantly be felt.

Pain and thermal sensitivity in the teeth are transmitted through nerve endings in the pulp. Because the pulp is in a narrow canal composed of connective tissue, blood vessels and nerves and surrounded by hard tissue, any infection or inflammation that would normally cause tissue to swell creates pressure on the pulpal nerves. That pressure, along with bacterial or immune system products that stimulate the nerve endings, produces the severe pain of pulpal infections.

Neuroscientists have long studied oral-facial pain, not only because of its importance in oral disease, but also because it provides an accessible model of pain elsewhere in the body. These investigations have greatly enriched our understanding of the basic mechanisms of pain perception and modulation. They have helped delineate the complex pathways and multiple transmitters that convey pain signals to the brain and spinal cord, as well as the mechanisms and molecules that can modulate and inhibit nociceptive input. These studies have also exploited new brain-imaging techniques to confirm the wide distribution of pain pathways and relay centers in the cerebral hemispheres and cerebellum.

This research has generated new approaches to the control of acute and chronic pain. These approaches include the use of nonsteroidal, anti-inflammatory drugs and long-acting local anesthetics for acute oral and dental pain, and the use of more potent drugs, drug combinations and other kinds of therapies to treat chronic pain.

Researchers have emphasized the importance of adequate pain control in patients with chronic pain conditions.

Otherwise, the constant barrage of signals can effect long-term changes in the brain that actually worsen the pain (producing hyperalgesia) and cause normally nonpainful stimuli to be perceived as painful (a condition called allodynia). Unrelieved chronic pain may also suppress the immune system.

Recently, investigators discovered a link between certain taste sensations, pain and temperature. Their findings indicate that capsaicin, the ingredient that makes hot peppers taste hot, binds to a receptor on the surface of nociceptors that also responds to noxious heat. The researchers have cloned the gene for the capsaicin receptor (called vanilloid receptor 1); they believe it is involved in several chronic pain conditions, especially those where inflammation plays a role, such as viral and diabetic neuropathy, rheumatoid arthritis and oral mucositis pain caused by cancer chemotherapy or radiation.

There is evidence that the prevalence of a number of pain conditions varies by gender and that men and women respond differently to different analgesic drugs. These findings have prompted studies aimed at determining whether there are sex differences in pain anatomy and neurochemistry and whether (and how) nociception is affected by sex hormones.

Speech

Human speech and language are the faculties that most distinguish us from other higher primates; they are also the links that bind people together in diverse social groups and cultures.

Central to speech are laryngeal mechanisms involving the vocal cords. Equally critical are the respiratory system, the pharynx and the nasal and oral cavities. The tongue is the most important structure of the peripheral speech mechanisms, working in conjunction with the lips, teeth and palate to produce a rich repertoire of sounds.

Abnormalities in oral structures, from missing or malformed teeth and malocclusion to cleft lip and palate, can seriously affect articulation. The movements of speech are orchestrated by brain centers that coordinate the muscles of mastication, facial expression and jaw movements.

Hearing impairments can also affect speech. To learn to speak, children must be able to hear others and monitor the feedback from their own voices. Congenital deafness and the serious hearing defects associated with some craniofacial syndromes can severely compromise speech acquisition.

The oral cavity

The mouth is the gateway to the body, performing dozens of functions that place high demands on its unique hard and soft tissues. The point of entry is the lips, which open into the oral cavity. The cheeks form the sides of the cavity, and the roof is formed by the palate, which separates the mouth from the nose above and the pharynx (throat) behind. The anterior palate is hard, formed by underlying bone, and serves as a shield against trauma to the face and head. The posterior palate is soft, composed of muscles and connective tissue that blend into the walls of the pharynx. Hanging from the rear of the soft palate is the uvula, a mass of muscle and connective tissue. Under the tongue is the floor of the mouth, composed primarily of muscle and salivary glands. The paired tonsils and adenoids, important components of the immune system, lie at the sides of the palate and within the nasopharynx, respectively.

The pharynx opens into channels leading either to the lungs for respiration or the esophagus for further digestion and passage to the stomach. This is a point of vulnerability: Should food or some other obstruction lodge in the airway, it could lead to death by asphyxiation.

Externally, the oral cavity is bounded by the maxilla (the upper jaw bone), attached to the cranium, and the mandible (the lower jaw), attached to the temporal bone of the skull by the temporomandibular joint.

Oral invaders

As the gateway to the body, the mouth is challenged by a constant barrage of invaders – bacteria, viruses, parasites, fungi. Thus infectious diseases, notably dental caries and periodontal diseases, predominate among the ills that can compromise oral health. Injuries take their toll as well, with the face and head particularly vulnerable to sports injuries, motor vehicle crashes, violence and abuse. Less common but very serious are oral and pharyngeal cancers, with a five-year survival rate of hardly better than 50 percent. Birth defects and developmental disorders frequently affect the craniofacial complex. These appear most commonly as isolated cases of cleft lip or palate, but clefting or other craniofacial defects can also be part of complex hereditary diseases or syndromes. Additionally, acute and chronic pain can affect the oral-facial region, particularly in and around the temporomandibular (jaw) joint, and accounts for a disproportionate amount of all types of pain that drive individuals to seek health care.

Many systemic diseases such as diabetes, arthritis, osteoporosis and AIDS as well as therapies for systemic diseases can directly or indirectly compromise oral tissues. The World Health Organization's International Classification of Diseases and Stomatology currently lists more than 120 specific diseases, distributed in 10 or more classes that have manifestations in the oral cavity.

Dental and periodontal infections

The most common oral diseases are dental caries and the periodontal diseases. Individuals are vulnerable to dental caries throughout life, with 85 percent of adults aged 18 and older affected. Periodontal diseases are most often seen in maturity, with the majority of adults experiencing some signs and symptoms by the mid-30s. Certain rare forms of periodontal disease affect young people. The major oral health success story of the past half century is that both caries and periodontal diseases can be prevented by a combination of individual, professional, and community measures.

Dental caries

The word caries derives from the Latin for rotten, and many cultures early on posited a tooth worm as the cause of this rottenness. By the 20th century, caries came to describe the condition of having holes in the teeth – cavities. This description, although not incorrect, is misleading. In actuality, a cavity is a late manifestation of a bacterial infection.

The bacteria colonizing the mouth are known as the oral flora. They form a complex community that adheres to tooth surfaces in a gelatinous mat, or biofilm, commonly called dental plaque. A cariogenic biofilm at a single tooth site may contain one-half billion bacteria, of which species of mutans streptococci are critical components.

These bacteria are able to ferment sugars and other carbohydrates to form lactic and other acids. Repeated cycles of acid generation can result in the microscopic dissolution of minerals in tooth enamel and the formation of an opaque white or brown spot under the enamel surface. Frequency of carbohydrate consumption, physical characteristics of food (e.g., stickiness), and timing of food intake also play a role.

The essential role of bacteria in caries initiation was established in landmark experiments in the 1950s. Investigators observed that germ-free animals fed high-sugar diets remained caries-free until the introduction of mutans streptococci (a particular group of bacterial strains having a number of common characteristics and which adhere tightly to the tooth). Later experiments demonstrated the transmissibility of the bacteria from mother to litter and from caries-infected to uninfected cage-mates. Species of Lactobacillus, Actinomyces and other acid-producing streptococci within the plaque may also contribute to the process.

If the caries infection in enamel goes unchecked, the acid dissolution can advance to form a cavity that can extend through the dentin (the component of the tooth located under the enamel) to the pulp tissue, which is rich in nerves and blood vessels. The resulting toothache can be severe and often is accompanied by sensitivity to temperature and sweets. Treatment requires endodontic (root canal) therapy. If untreated, the pulp infection can lead to abscess, destruction of bone, and spread of the infection via the bloodstream.

Dental caries can occur at any age after teeth erupt. Particularly damaging forms can begin early, when developing primary teeth are especially vulnerable. This type of dental caries is called early childhood caries (ECC). Some six out of 10 children in the United States have one or more decayed or filled primary teeth by age 5. ECC may occur in children who are given pacifying bottles of juice, milk or formula to drink during the day or overnight. The sugar contents pool around the upper front teeth, mix with cariogenic bacteria and give rise to rapidly progressing destruction. Other risk factors for ECC include arrested development of tooth enamel, chronic illness, altered salivary composition and volume (resulting from the use of certain medications or malnourishment), mouth breathing and blockage of saliva flow in a bottle-fed infant.

Although there have been continuing reductions in dental caries in permanent teeth among children and adolescents over the past few decades, caries prevalence in the primary dentition may have stabilized or increased slightly in some population groups. Reductions in caries in permanent teeth also have been proportionately greater on the smooth surfaces rather than on the pit-and-fissure surfaces characteristic of chewing surfaces. The gingival tissues tend to recede over time, exposing the tooth root to cariogenic bacteria that can cause root caries. An important risk factor for root caries in older people is the use of medications that inhibit salivary flow, leading to dry mouth (xerostomia).

Saliva contains components that can directly attack cariogenic bacteria, and it is also rich in calcium and phosphates that help to remineralize tooth enamel. Demineralization of enamel occurs when pH levels fall as a result of acid production by bacteria. It can be reversed at early stages if the local environment can counteract acid production, restoring pH to neutral levels. Remineralization can occur through the replacement of lost mineral (calcium and phosphates) from the stores in saliva. Fluoride in saliva and dental plaque and the buffering capacity of saliva also contribute to this process. Indeed, it is now believed that fluoride exerts its chief caries-preventive effect by facilitating remineralization. Several studies have demonstrated that remineralization results in an increase in tooth hardness and mineral content, rendering the tooth surface more resistant to subsequent acid attack. Overt caries lesions develop when there is insufficient time for remineralization between periods of acidogenesis or when the saliva production is compromised. Over 400 medications list dry mouth as a side effect, notably some antidepressants, antipsychotics, antihistamines, decongestants, antihypertensives, diuretics and anti-Parkinsonian drugs. The effects of xerostomia may be particularly severe in cancer patients receiving radiation to the head or neck because the rays can destroy salivary gland tissue rather than simply inhibiting salivary secretion.

The professional application of dental sealants (plastic films coated onto the chewing surfaces of teeth) is an important caries-preventive measure that complements the use of fluorides. The films prevent decay from developing in the pits and fissures of teeth, channels that are often inaccessible to brushing and where fluoride may be less effective.

The rate of caries progression through enamel is relatively slow and may be slower in patients who have received regular fluoride treatment or who consume fluoridated water. Because a large percentage of enamel lesions remain unchanged over periods of three to four years, and because progression rates through dentin are comparably slow, the application of infection control and monitoring procedures to assess caries risk status, lesion activity status, evidence of lesion arrest and evidence of lesion remineralization over extended periods of time is recommended.

Experts believe that the earlier mutans streptococci are acquired in infancy, the higher the caries risk. Most studies indicate that infants are infected before their first birthday, around the time the first incisors emerge. However, one study found the median age of acquisition to be 26 months, coinciding with the emergence of the primary molars. DNA fingerprinting has demonstrated that the source of transmission is usually the mother.

It is not clear why some individuals are more susceptible and others more resistant to caries. Genetic differences in the structure and biochemistry of enamel proteins and crystals as well as variations in the quality and quantity of saliva and in immune defense mechanisms are among the factors under study. Analysis of mutans streptococci genomes may also shed light, indicating which species are particularly virulent and which genes contribute to that virulence.

Even the most protective genetic endowment and developmental milieu are unlikely to confer resistance to decay in the absence of positive personal behaviors. These include sound dietary habits and good oral hygiene, including the use of fluorides, and seeking professional care. There are indications, however, that some destructive oral habits are on the rise, such as the use of smokeless (spit) tobacco products by teenage boys. Although the chief concern here lies in the long-term risk for oral cancers, spit tobacco that contains high levels of sugar is also associated with increased levels of decay of both crown and root surfaces.

Periodontal diseases

Like dental caries, the periodontal diseases are infections caused by bacteria in the biofilm (dental plaque) that forms on oral surfaces. The basic division in the periodontal diseases is between gingivitis, which affects the gums, and periodontitis, which may involve all of the soft tissue and bone supporting the teeth. Gingivitis and milder forms of periodontitis are common in adults. The percentage of individuals with moderate to severe periodontitis, in which the destruction of supporting tissue can cause the tooth to loosen and fall out, increases with age.

Signs and symptoms

In the early stages, periodontitis has very few symptoms, and in many individuals the disease has progressed significantly before they seek treatment. Symptoms may include the following:

- Redness or bleeding of gums while brushing teeth, using dental floss or biting into hard food (such as apples) (though this may occur even in gingivitis, where there is no attachment loss).
- Gum swelling that recurs.
- Halitosis, or bad breath, and a persistent metallic taste in the mouth.
- Gingival recession, resulting in apparent lengthening of teeth. (This may also be caused by heavy-handed brushing or with a stiff toothbrush.)
- Deep pockets between the teeth and the gums (pockets are sites where the attachment has been gradually destroyed by collagen-destroying enzymes, known as collagenases).
- Loose teeth, in the later stages (though this may occur for other reasons as well).
- Patients should realize that the gingival inflammation and bone destruction are largely painless. Hence, people may wrongly assume that painless bleeding after teeth cleaning is insignificant, although this may be a symptom of progressing periodontitis in that patient.
- Certain factors increase the risk for periodontal disease:
    - Smoking.
    - Diabetes.
    - Poor oral hygiene.
    - Stress.
    - Heredity.
    - Crooked teeth.
    - Underlying immunodeficiencies – e.g., AIDS.
    - Fillings that have become defective.
    - Taking medications that cause dry mouth.
    - Bridges that no longer fit properly.
    - Female hormonal changes, such as with pregnancy or the use of oral contraceptives.

Prevention

Daily oral hygiene measures to prevent periodontal disease include:

- Brushing properly on a regular basis (at least twice daily), with the patient attempting to direct the toothbrush bristles underneath the gumline, to help disrupt the bacterial growth and formation of subgingival plaque.
- Flossing daily and using interdental brushes (if there is a sufficiently large space between teeth), as well as cleaning behind the last tooth, the third molar, in each quarter.
- Using an antiseptic mouthwash. Chlorhexidine gluconate based mouthwash in combination with careful oral hygiene may cure gingivitis, although they cannot reverse any attachment loss due to periodontitis.
- Using a “soft” toothbrush to prevent damage to tooth enamel and sensitive gums.
- Using periodontal trays to maintain dentist-prescribed medications at the source of the disease. The use of trays allows the medication to stay in place long enough to penetrate the biofilms where the bacteria are found.
- Regular dental check-ups and professional teeth cleaning as required. Dental check-ups serve to monitor the person’s oral hygiene methods and levels of attachment around teeth, identify any early signs of periodontitis, and monitor response to treatment.

Typically dental hygienists (or dentists) use special instruments to clean (debride) teeth below the gumline and disrupt any plaque growing below the gumline. This is a standard treatment to prevent any further progress of established periodontitis. Studies show that after such a professional cleaning (periodontal debridement), bacteria and plaque tend to grow back to pre-cleaning levels after about three

to four months. Hence, in theory, cleanings every three to four months might be expected to also prevent the initial onset of periodontitis. However, analysis of published research has reported little evidence either to support this or the intervals at which this should occur. Instead, it is advocated that the interval between dental check-ups should be determined specifically for each patient between every three to 24 months.

Nonetheless, the continued stabilization of a patient's periodontal state depends largely, if not primarily, on the patient's oral hygiene at home as well as on the go. Without daily oral hygiene, periodontal disease will not be overcome, especially if the patient has a history of extensive periodontal disease.

A contributing cause may be low selenium in the diet: "Results showed that selenium has the strongest association with gum disease, with low levels increasing the risk by 13-fold."

The cornerstone of successful periodontal treatment starts with establishing excellent oral hygiene. This includes twice daily brushing with daily flossing. Also the use of an interdental brush (called a proxi-brush) is helpful if space between the teeth allows. Persons with dexterity problems such as arthritis may find oral hygiene to be difficult and may require more frequent professional care and the use of a powered toothbrush. Persons with periodontitis must realize that it is a chronic inflammatory disease and a lifelong regimen of excellent hygiene and professional maintenance care with a dentist/hygienist or periodontist is required to maintain affected teeth.

Initial therapy

Removal of bacterial plaque and calculus is necessary to establish periodontal health. The first step in the treatment of periodontitis involves nonsurgical cleaning below the gumline with a procedure called scaling and debridement. In the past, root planing was used (removal of cemental layer as well as calculus). This procedure involves use of specialized curettes to mechanically remove plaque and calculus from below the gumline, and may require multiple visits and local anesthesia to adequately complete. In addition to initial scaling and root planing, it may also be necessary to adjust the occlusion (bite) to prevent excessive force on teeth with reduced bone support. Also it may be necessary to complete any other dental needs such as replacement of rough, plaque retentive restorations, closure of open contacts between teeth and any other requirements diagnosed at the initial evaluation.

Re-evaluation

Multiple clinical studies have shown that nonsurgical scaling and root planing is usually successful in periodontal pocket depths no greater than 4-5mm. It is necessary for the dentist or hygienist to perform a re-evaluation four to six weeks after the initial scaling and root planing to determine whether the treatment was successful in reducing pocket depths and eliminating inflammation. It has been found that pocket depths which remain after initial therapy of greater than 5-6 mm with bleeding upon probing are indicative of continued active disease and will very likely show further bone loss over time. This is especially true in molar tooth sites where furcations (areas between the roots) have been exposed.

Periodontal surgery

If nonsurgical therapy is found to have been unsuccessful in managing signs of disease activity, periodontal surgery may be needed to stop progressive bone loss and regenerate lost bone where possible. There are many surgical approaches used in treatment of advanced periodontitis, including open flap debridement, osseous surgery, guided tissue regeneration and bone grafting. The goal of periodontal surgery is access for definitive calculus removal and surgical management of bony irregularities that have resulted from the disease process to reduce pockets as much as possible. Long-term studies have shown that in moderate to advanced periodontitis, surgically treated cases often have less further breakdown over time and when coupled with a regular post-treatment maintenance regimen are successful in nearly halting tooth loss in nearly 85 percent of patients.

Maintenance

Once successful periodontal treatment has been completed, with or without surgery, an ongoing regimen of "periodontal maintenance" is required. This involves regular checkups and detailed cleanings every three months to prevent repopulation of periodontitis-causing bacteria, and to closely monitor affected teeth so that early treatment can be rendered if disease recurs. Usually periodontal disease exists due to poor plaque control, so if the brushing techniques are not modified, a periodontal recurrence is probable.

Assessment and prognosis

Dentists and dental hygienists "measure" periodontal disease using a device called a periodontal probe. This is a thin "measuring stick" that is gently placed into the space between the gums and the teeth and slipped below the gumline. If the probe can slip more than 3 millimetres length below the gumline, the patient is said to have a "gingival pocket" around that tooth. This is somewhat of a misnomer, as any depth is in essence a pocket, which in turn is defined by its depth, i.e., a 2 mm pocket or a 6 mm pocket. However, it is generally accepted that pockets are self-cleansable (at home, by the patient, with a toothbrush) if they are 3 mm or less in depth. This is important because if there is a pocket which is deeper than 3 mm around the tooth, at-home care will not be sufficient to cleanse the pocket, and professional care should be sought. When the pocket depths reach 6 and 7 mm in depth, the hand instruments and cavitrons used by the dental professionals may not reach deeply enough into the pocket to clean out the bacterial plaque that cause gingival inflammation. In such a situation the bone or the gums around that tooth should be surgically altered or it will always have inflammation, which will likely result in more bone loss around that tooth. An additional way to stop the inflammation would be for the patient to receive subgingival antibiotics (such as minocycline) or undergo some form of gingival surgery to access the depths of the pockets and perhaps even change the pocket depths so that they become 3 mm or less in depth and can once again be properly cleaned by the patient at home with his or her toothbrush.

If a patient has 7 mm or deeper pockets around the teeth, then he or she would likely risk eventual tooth loss over the years. If this periodontal condition is not identified and the patient remains unaware of the progressive nature of the disease then, years later, they may be surprised that some teeth will gradually become loose and may need to be extracted, sometimes due to a severe infection or even pain.

According to the Sri Lankan Tea Labourer study, in the absence of any oral hygiene activity, approximately 10 percent will suffer from severe periodontal disease with rapid loss of attachment (more than 2 mm/year). Eighty percent will suffer from moderate loss (1-2 mm/year), and the remaining 10 percent will not suffer any loss.

Alternative treatments

Periodontitis has an inescapable relationship with subgingival calculus (tartar). The first step in any procedure is to eliminate calculus under the gumline, as it houses destructive anaerobic bacteria that consume bone, gum and cementum (connective tissue) for food.

Most alternative "at-home" gum disease treatments involve injecting antimicrobial solutions, such as hydrogen peroxide, into periodontal pockets via slender applicators or oral irrigators. This process disrupts anaerobic bacteria colonies and is effective at reducing infections and inflammation when used daily. There are any number of potions and elixirs that are commercially available which are functionally equivalent to hydrogen peroxide; but at substantially higher cost. These treatments, however, do not address calculus formations and are therefore short-lived, as anaerobic bacteria colonies quickly regenerate in and around calculus.

In a new field of study, calculus formations are addressed on a more fundamental level. At the heart of the formation of subgingival calculus, growing plaque formations starve out the lowest members of the community, which calcify into calcium phosphate salts of the same shape and size of the original, organic bacilli. Calcium phosphate salts (unlike calcium phosphate; the primary component in teeth) are ionic and adhere to tooth surfaces via electrostatic attraction. Smaller, free floating calcium phosphate salt particles are equally attracted to the same areas, as are additional calcified bacteria, growing calculus formations as unorganized, yet strong, "brick and mortar" matrices. The microscopic voids in calculus formations house new anaerobic bacteria, as does the top "diseased layer."

Because the root cause of subgingival calculus development is ionic attraction, it was hypothesized that the introduction of oppositely charged particles around the formations might chelate calcium phosphate salt components away from the matrix, thus actually reducing the size of subgingival calculus formations.

To accomplish this, a sequestering agent solution comprised partly of sodium tripolyphosphate (STPP) and sodium fluoride (charge-1) was tested on a patient with burnished and new subgingival calculus at a depth of 6 mm. The patient delivered the solution using an oral irrigator, once a day for 60 days. The results of this test were the successful elimination of all calculus formations studied. This test was conducted using a subgingival endoscopic camera (perioscope) by an independent periodontist.

This alternative treatment keeps subgingival calculus at bay, in concert with traditional periodontal treatments. In this way, periodontitis may be controlled by the patient, with complete restoration of dental health being a collaborative effort between the patient and the dental professional.

Gingivitis

Gingivitis is an inflammation of the gums characterized by a change in color from normal pink to red, with swelling, bleeding and often sensitivity and tenderness. These changes result from an accumulation of biofilm along the gingival margins and the immune system's inflammatory response to the release of destructive bacterial products. The early changes of gingivitis are reversible with thorough toothbrushing and flossing to reduce plaque. Without adequate oral hygiene, however, these early changes can become more severe, with infiltration of inflammatory cells and establishment of a chronic infection. Biofilm on tooth surfaces opposite the openings of the salivary glands often mineralizes to form calculus or tartar, which is covered by unmineralized biofilm – a combination that can exacerbate local inflammatory responses. A gingival infection may persist for months or years, yet never progress to periodontitis.

Gingival inflammation does not appear until the biofilm changes from one composed largely of gram-positive streptococci (which can live with or without oxygen) to one containing gram-negative anaerobes (which cannot live in the presence of oxygen). Numerous attempts have been made to pinpoint which microorganisms in the supragingival (above the gumline) plaque are the culprits in gingivitis. Frequently mentioned organisms include Fusobacterium nucleatum, Veillonella parvula, and species of Campylobacter and Treponema. But as Ranney (1989) notes, "The complexity of the results defies any attempt to define a discrete group clearly and consistently associated with gingivitis."

Gingival inflammation may be influenced by steroid hormones, occurring as puberty gingivitis, pregnancy gingivitis, and gingivitis associated with birth control medication or steroid therapy. The presence of steroid hormones in tissues adjacent to biofilm apparently encourages the growth of certain bacteria and triggers an exaggerated response to biofilm accumulation. Again, thorough oral hygiene can control this response.

Certain prescription drugs can also lead to gingival overgrowth and inflammation. These include the antiepileptic drug phenytoin (Dilantin); cyclosporin, used for immunosuppressive therapy in transplant patients; and various calcium channel blockers used in heart disease. Treatment often requires surgical removal of the excess tissue followed by appropriate personal and professional oral health care.

A form of gingivitis common 50 years ago but relatively rare today is acute necrotizing ulcerative gingivitis, also known as Vincent's infection or trench mouth. This aggressive infection is characterized by destruction of the gingiva between the teeth, spontaneous bleeding, pain and oral odor. People under extreme stress have an increased susceptibility. Spirochetes and other bacteria have been found in the connective tissue of those affected. An association between smoking

and this type of gingivitis is well recognized and was demonstrated as early as 1946. This condition has been seen in some HIV-positive patients. Treatment requires a combination of professional periodontal treatment and antibacterial therapy along with professional smoking cessation assistance as appropriate.

Adult periodontitis

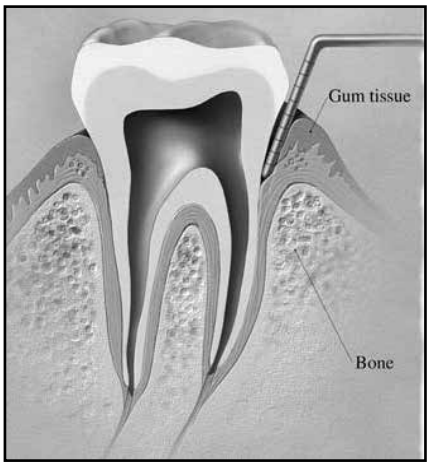
The most common form of adult periodontitis is described as general and moderately progressing; a second form is described as rapidly progressing and severe, and is often resistant to treatment. The moderately progressive adult form is characterized by a gradual loss of attachment of the periodontal ligament to the gingiva and bone along with loss of the supporting bone. It is most often accompanied by gingivitis. It is not necessarily preceded by gingivitis, but the gingivitis-related biofilm often seeds the subgingival plaque. The destruction of periodontal ligament and bone results in the formation of a pocket between the tooth and adjacent tissues, which harbors subgingival plaque. The calculus formed in the pocket by inflammatory fluids and minerals in adjacent tissues is especially damaging.

The severity of periodontal disease is determined through a series of measurements, including the extent of gingival inflammation and bleeding, the probing depth of the pocket to the point of resistance, the clinical attachment loss of the periodontal ligament measured from a fixed point on the tooth (usually the cemento-enamel junction), and the loss of adjacent alveolar bone as measured by x-ray. Severity is determined by the rate of disease progression over time and the response of the tissues to treatment.

Adult periodontitis often begins in adolescence but is usually not clinically significant until the mid-30s. Prevalence and severity increase but do not accelerate with age. One view proposes that destruction occurs at a specific site during a defined period, after which the disease goes into remission. The current view is that the disease process may not be continuous but rather progresses in random bursts in which short periods of breakdown of periodontal ligament and bone alternate with periods of quiescence. These episodes occur randomly over time and at random sites in the mouth. Part of the difficulty in determining the pattern of progression reflects variation in the sensitivity of the instruments used to measure the loss of soft tissue and bone. The latest generation of probes finds evidence of both continuous and multiple-burst patterns of loss in different patients and at different times.

Most researchers agree that periodontitis results from a mixed infection but that a particular group of gram-negative bacteria are key to the process and markedly increase in the subgingival plaque. The bacteria most frequently cited are Porphyromonas gingivalis, Prevotella intermedia, Bacteroides forsythus, Treponema denticola, and Actinobacillus actinomycetemcomitans. Their role in disease initiation and progression is determined in part by their "virulence factors." These include the ability to colonize subgingival plaque, generate products that can directly injure tissues, and elicit an inflammatory or immune response. The potentially noxious bacterial products include hydrogen sulfide, polyamines, the fatty acids butyrate and propionate, lipopolysaccharide (also known as endotoxin), and a number of destructive enzymes. The interaction of this arsenal with the host response is at the core of periodontal pathology, (Socransky and Haffajee 1991, 1992). Sequencing of the genomes of several key periodontal pathogens is under way and should provide further insight into these pathogens as well as catalyze new treatment approaches.

This drawing shows a dentist using a probe to check for inflammatory pockets.



Delicate balances

Neutrophils (a type of white blood cell) and antibodies are the major immune defenses against bacterial attack. Neutrophils move to the site of infection, where they engulf bacteria and elaborate antibacterial agents and enzymes to destroy bacteria. Although stimulation of the immune system to attack the offending bacteria is generally protective, immune hyper-responsiveness and hypersensitivity can be counterproductive, leading to the destruction of healthy tissue. Nevertheless, the neutrophil/antibody axis is critical for full protection against periodontal diseases.

Also important is the release of certain potent molecules called cytokines and prostaglandins, especially prostaglandin E2 (PGE2) which can contribute to tissue destruction. Cytokines are proteins secreted by immune cells that help regulate immune responses and also affect bone, epithelial and connective tissues. Most prominent in periodontal diseases are interleukin 1 (IL-1), tumor necrosis factor alpha (TNF-gamma) and interferon gamma (IFN-gamma). These cytokines mediate the processes of bone resorption and connective tissue destruction.

Susceptibility and resistance

PGE2 may play a central role in the tissue destruction that occurs in periodontal diseases. Levels of PGE2 in periodontal tissue are low or undetectable in health, increase in gingivitis, and rise significantly in periodontitis. Now there is increasing evidence that the level of PGE2 produced in response to bacterial challenge (especially by endotoxin) can be used as a measure of susceptibility.

Presumably, the level of PGE2 production is subject to genetic influence. Studies of identical and fraternal twins, either reared together or apart, provide evidence that genetic factors may indeed influence susceptibility or resistance to the common adult form of periodontitis. Recently, a commercial test for a genetic marker of susceptibility has been introduced. The marker is associated with increased production of a particular form of interleukin 1-beta (IL-1-beta) when stimulated by periodontopathic bacteria. In 1996, it was found that nonsmoking adults who are positive for the marker are 6 to 19 times more likely to develop severe periodontitis.

Susceptibility to adult periodontitis has also been explored in relation to a variety of behavioral and demographic variables as well as to the presence of other diseases. One of the strongest behavioral associations is with tobacco use. The risk of alveolar bone loss for heavy smokers is seven times greater than for those who have not smoked. Cigarette smoking also may impair the normal host response in neutralizing infection, resulting in the destruction of healthy periodontal tissues adjacent to the site of infection. Smokers also have decreased levels of salivary and serum immunoglobulins to Prevotella intermedia and Fusobacterium nucleatum and depressed numbers of helper T cells as well. Finally, smoking alters the cells that engulf and dispose of bacteria – neutrophils and other phagocytes – affecting their ability to clear pathogens.

Epidemiologic studies have found that such additional factors as increasing age, infrequent dental visits, low education level, low income, comorbidities and inclusion in certain racial or ethnic populations are associated with increased prevalence of periodontitis. It is important that epidemiologic studies also take into consideration the fact that tobacco use, oral hygiene, professional prophylaxis, and routine dental care are correlated to socioeconomic status, as are race and ethnicity. Sex is another factor. Males tend to have higher levels of periodontal diseases, presumably because of a history of greater tobacco use and differences in personal care and frequency of dental visits. However, female hormones may play a protective role (as they do in protecting against osteoporosis).

Certain systemic diseases heighten susceptibility. Epidemiological studies have confirmed that patients with diabetes mellitus, both type 1 and type 2, are more susceptible to periodontal diseases. Measures such as the gingival index, pocket depth and loss of attachment are more severe if the diabetic patients are smokers. The likelihood of periodontal disease increases markedly when diabetes is poorly controlled. In contrast, periodontal diseases respond well to therapy and can be managed successfully in patients with well-controlled diabetes. Such therapy can result in improvements in the diabetic condition itself. There is some evidence that osteoporosis may be a risk factor for periodontal disease. More clinical attachment loss and edentulousness have been reported in osteoporotic than in nonosteoporotic women. Two studies in 1996 showed that estrogen replacement therapy in postmenopausal women not only gives protection against osteoporosis, but also results in fewer teeth lost to periodontal disease.

The less common rapidly progressive form of adult periodontitis typically affects people in their early 20s and 30s. It is characterized by severe gingival inflammation and rapid loss of connective tissue and bone. Many patients have an inherent defect in neutrophil response to infection. Several systemic diseases have been associated with this form of periodontal disease, including type 1 diabetes, Down syndrome, Papillon-Lefevre syndrome, Chediak-Higashi syndrome and HIV infection. Specific bacteria associated with rapidly progressive disease include Porphyromonas gingivalis, Prevotella intermedia, Eikenella corrodens, and Wolinella recta. Most recently, mutations in the cathepsin C gene have been associated with the Papillon-Lefevre syndrome and how the defect can result in periodontal disease.

Refractory periodontitis

Refractory periodontitis is not a specific form of disease, but refers to cases in which patients continue to exhibit progressive disease at multiple sites despite aggressive mechanical therapy to remove biofilm and calculus, along with the use of antibiotics. Refractory sites exhibit elevated levels of a number of different bacteria, with the dominant species different in different subjects. It is not known whether variations in pathogenicity of the bacteria, defects in the subject's defense systems, or combinations of these factors are responsible for the refractory nature of the disease. The adoption of new diagnostic technology to detect predominant bacterial species, followed by selective antibiotic treatment, may help resolve infection and disease in these patients.

Early-onset periodontitis

The forms of periodontitis occurring in adolescents and young adults generally involve defects in neutrophil function. Localized juvenile periodontitis (LJP) mainly affects the first molar and incisor teeth of teenagers and young adults, with rapid destruction of bone but almost no telltale signs of inflammation and very little supragingival plaque or calculus. Actinobacillus actinomycetemcomitans has been isolated at 90 to 100 percent of diseased sites in these patients, but is absent or appears in very low frequency in healthy or minimally diseased sites. It is possible that the bacteria are transmitted among family members through oral contacts such as kissing or sharing utensils, because the same bacterial strain appears in affected family members. However, evidence of a neutrophil defect argues for a genetic component. Another organism frequently associated with LJP is Capnocytophaga ochracea. Neither of these bacteria dominate in the generalized adult form of the disease, where Porphyromonas gingivalis is considered of greatest significance.

Prepubertal periodontitis is rare and can be either general or localized. The generalized form begins with the eruption of the primary teeth and proceeds to involve the permanent teeth. There is severe inflammation, rapid bone loss, tooth mobility and tooth loss. The localized form of the disease is less aggressive, affecting only some primary teeth. The infection involves many of the organisms associated with periodontitis, but the mix may differ somewhat, with Actinobacillus actinomycetemcomitans, Prevotella intermedia, Eikenella corrodens and several species of Capnocytophaga implicated. Defects in neutrophil function noted in both forms of the may explain why patients are highly susceptible to other infections as well.

Selected mucosal infections and conditions

Like the skin, the mucosal lining of the mouth serves to protect the body from injury. This lining is itself subject to a variety of infections and conditions, ranging from benign canker sores to often fatal cancers.

Oral candidiasis

Chronic hyperplastic candidiasis is a red or white lesion that may be flat or slightly elevated and may adhere to soft or hard tissue surfaces, including dental appliances. It is caused by species of Candida, especially Candida albicans, the most common fungal pathogen

isolated from the oral cavity. Normally, the fungi are present in relatively low numbers in up to 65 percent of healthy children and adults and cause no harm. Problems arise when there is a change in oral homeostatis – the normal balance of protective mechanisms and resident oral flora that maintain the health of the oral cavity, so that defense mechanisms are compromised. Under these circumstances, the fungal organisms can overgrow to cause disease. A primary disruption in homeostasis occurs with the use of antibiotics and corticosteroids, which can markedly change the composition of the oral flora.

Deficiencies in the immune and endocrine systems are also important. Indeed, the diagnosis of candidiasis in an otherwise seemingly healthy young adult may be the first sign of HIV infection. Other causes of candidiasis include cancer chemotherapy or radiotherapy to the head and neck, xerostomia resulting from radiation to the head and neck, medications, chronic mucosal irritation, certain blood diseases and other systemic conditions. Also, tobacco use has been identified as a cofactor.

Candidiasis often causes symptoms of burning and soreness as well as sensitivity to acidic and spicy foods. Patients may complain of a foul taste in the mouth. However, it can also be asymptomatic. Genomic analysis of the Candida albicans genome is helping investigators identify numerous genes that code for virulence factors, including enzymes that can facilitate adhesion to and penetration of mucous membranes. At the same time, researchers are exploring novel gene technologies to increase production of a family of native salivary proteins, the histatins that have known anticandidal and other antimicrobial effects.

The most common form of oral candidiasis is denture stomatitis. It occurs when tissues are traumatized by continued wearing of ill-fitting or inadequately cleaned dental appliances and is described as chronic erythematous candidiasis. Another form, candidal angular cheilosis, occurs in the folds at the angles of the mouth and is closely associated with denture sore mouth. Other common forms of Candida infection are pseudomembranous candidiasis (thrush), which may affect any of the mucosal surfaces, and acute erythematous candidiasis, a red and markedly painful variant commonly seen in AIDS patients.

In most cases, Candida infection can be controlled with antifungal medications used locally or systemically. Control is difficult, however, in patients with immune dysfunction, as in AIDS, or other chronic debilitating diseases. Often the organisms become resistant to standard therapy, and aggressive approaches are necessary. The spread of oral candidiasis to the esophagus or lungs can be life-threatening and is one of the criteria used to define frank AIDS.

Herpes simplex virus infections

In any given year, about a half-million Americans will experience their first encounter with the herpes simplex virus type 1 (HSV-1), the cause of cold sores. That first encounter usually occurs in the oral region and may be so mild as to go unnoticed. But in some people, particularly young children and young adults, infection may take the form of primary herpetic stomatitis, with symptoms of malaise, muscle aches, sore throat and enlarged and tender lymph nodes before the appearance of the familiar cold sore blisters. These blisters usually show up on the lips, but any of the mucosal surfaces can be affected. Bright-red ulcerated areas and marked gingivitis may also be seen.

Herpes viruses also cause genital infections, which are transmitted sexually. Both HSV-1 and HSV-2 have been found in oral and genital infections, with HSV-1 predominating in oral areas and HSV-2 in genital areas. Herpes viruses have also been implicated as cofactors in the development of oral cancers. Crowded living conditions can result in greater contact with infected individuals, which aids in transmission of HSV.

Normally, the immune system mounts a successful attack on the viruses, with symptoms abating by the time neutralizing antibodies appear in the bloodstream, in about 10 days. However, herpes viruses are notorious for their ability to avoid immune detection by taking refuge in the nervous system, where they can remain latent for years. In oral herpes, the virus commonly migrates to the nearby trigeminal ganglion, the cluster of nerve cells whose fibers branch out to the face and mouth. In about 20 to 40 percent of people who are virus-positive, the virus may reactivate, with infectious virus particles moving to the oral cavity to cause recurrent disease.

The usual site of a recurrent lesion is on or near the lips. Recurrences are rarely severe, and lesions heal in seven to 10 days without scarring. The recurrences may be provoked by a wide range of stimuli, including sunlight, mechanical trauma and mild fevers such as occur with a cold. Emotional factors may play a role as well.

Oral human papillomavirus infections

There are more than 100 recognized strains of oral human papillomavirus (HPV), a member of the papovavirus family, implicated in a variety of oral lesions. Most common are papillomas (warts) found on or around the lips and in the mouth. HPV is found in 80 percent of these oral squamous papillomas. The virus has also been identified in 30 to 40 percent of oral squamous cell carcinomas and has been implicated in cervical cancer as well. Whether a cancer or nonmalignant wart develops may depend on which virus is present or on which viral genes are activated.

Oral warts are most often found in children, probably as a result of chewing warts on the hands. In adults, sexual transmission from the anogenital region can occur.

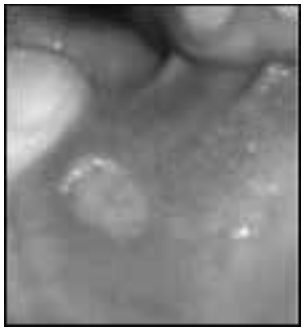
In general, viral warts spontaneously regress after one or two years. The immune system normally keeps HPV infections under control, as evidenced by the increased prevalence of HPV-associated lesions in HIV-infected patients and others with immunodeficiency.

Recurrent aphthous ulcers or canker sores

Recurrent aphthous ulcers (RAU), also referred to as recurrent aphthous stomatitis, is the technical term for canker sores, the most common and generally mild oral mucosal disease. Between 5 and 25 percent of the general population is affected, with higher numbers in selected groups, such as health professional students. The disease takes three clinical forms: RAU minor, RAU major and herpetiform RAU. The minor form accounts for 70 to 87 percent of cases. The sores are small, discrete, shallow ulcers surrounded by a red halo appearing at the front of the mouth or the tongue. The ulcers, which usually last up to two weeks, are painful and may make eating or speaking difficult.

About half of RAU patients experience recurrences every one to three months; as many as 30 percent report continuous recurrences.

RAU major accounts for 7 to 20 percent of cases and usually appears as one to 10 larger coalescent ulcers at a time, which can persist for weeks or months. Herpetiform RAU has been reported as occurring in 7 to 10 percent of RAU cases. The ulcers appear in crops of 10 to 100 at a time, concentrating in the back of the mouth and lasting for seven to 14 days.



This picture shows a canker sore in the mouth on the upper lip.

RAU can begin in childhood, but the peak period for onset is the second decade. About 50 percent of close relatives of patients with RAU also have the condition, and a high correlation of RAU has been noted in identical but not fraternal twins. Associations have been found between RAU and specific genetic markers.

RAU has also been associated with hypersensitivities to some foods, food dyes and food preservatives. Nutritional deficiencies – especially in iron, folic acid, various B vitamins or combinations thereof – have also been reported, and improvements noted with suitable dietary supplements.

The two factors that have been found to have the strongest association with RAU are immunologic abnormality, possibly involving autoimmunity and trauma.

Volunteers with and without a history of RAU were studied for their reaction to the trauma of a needle prick to the inner cheek tissue. No ulcers developed in non-RAU subjects, but nearly half of those prone to canker sores had a recurrence.

RAU also can occur in a number of systemic diseases, including HIV infection, ulcerative colitis, Crohn's disease and Behçet's disease. In general, people who are immunocompromised are more susceptible to RAU, as are people with a variety of blood diseases.

RAU itself does not give rise to other illnesses but is uncomfortable. Symptomatic treatment includes topical analgesics, antibacterial rinses, topical corticosteroids and a new prescription medication that reduces pain and healing time.

Developmental disorders

The importance of the face as the bearer of identity, character, intelligence and beauty is universal. Craniofacial birth defects, which can include such manifestations as cleft lip or palate, eyes too closely or widely spaced, deformed ears, eyes mismatched in color and facial asymmetries, can be devastating to the parents and child affected. Surgery, dental care, psychological counseling and rehabilitation may help to ameliorate the problems but often at great cost and over many years.

Although each developmental craniofacial disease or syndrome is relatively rare, the number of children affected worldwide is in the millions. In addition, craniofacial defects form a substantial component of many other developmental birth defects, largely because they occur very early in gestation, when many of the same genes that orchestrate the development of the brain, head, face and mouth are also directing the development of the limbs and many vital internal organs, such as the heart, lungs and liver.

By about the third week after fertilization, the three germ layers of the embryo – the ectoderm, endoderm and mesoderm – have formed, as well as the first of four sets of paired swellings – the branchial arches – that appear at the sides of the head end of the embryo. Some craniofacial defects result from failure of the arches to complete their morphogenetic development. Other craniofacial defects are the result of the abnormal differentiation of cells derived from the ectoderm and endoderm or from ectomesenchyme cells, which originate in a part of the ectoderm (the neural crest), in interaction with future connective tissue (the mesenchyme).

CRANIOFACIAL ANOMALIES CAUSED BY ALTERED BRANCHIAL ARCH MORPHOGENESIS

Cleft lip/palate and cleft palate

The most common of all craniofacial anomalies and among the most common of all birth defects, are clefts of the lip with or without cleft palate and cleft palate alone; these occur at a rate of 1 to 2 out of 1,000 births, resulting in more than 8,000 affected newborns every year. Cleft lip/palate and cleft palate are distinct conditions with different patterns of inheritance and embryological origins. The male to female ratio of cleft lip/palate is 2 to 1; the ratio for cleft palate alone is just the reverse, 1 to 2.

These anomalies result from the failure of the first branchial arches to complete fusion processes. Clefting can occur independently or as part of a larger syndrome that may include mental retardation and defects of the heart and other organs. Not all cases of clefting are inherited; a number of teratogens (environmental agents that can cause birth defects) have been implicated, as well as defects in essential nutrients such as folic acid. Smoking by the mother during pregnancy also increases the risk. It is becoming increasingly evident that most diseases and disorders, not just craniofacial anomalies, result from interactions involving multiple genes and environmental factors.

Infants with clefts have difficulty with vital oral functions such as feeding, breathing, speaking and swallowing. They are also susceptible to repeated respiratory infections. As these children grow, they must cope with the social consequences of a facial deformity, delayed and altered speech, frequent illness and repeated surgeries that may persist through late adolescence.

Current molecular epidemiology investigations have examined both syndromic and nonsyndromic (isolated) cleft lip/palate and cleft palate. Linkage studies have identified a number of candidate genes, including MSX1, RAR, an X-linked locus and the genes for TGF-beta-3 and TGF-alpha. The pattern of inheritance in cleft lip/palate and cleft palate suggests that between 2 and 20 genes may be involved, with one gene representing a major component in the development of the cleft. One of the common syndromic forms of cleft lip/palate, the Van der Woude syndrome, is caused by an autosomal dominant form of inheritance at a locus on chromosome 1. Future molecular genetic studies will be needed to provide the information necessary for prenatal diagnosis, calculation of risk, and potential gene therapy.

The Treacher Collins syndrome – mandibulofacial dysostosis

Children with the Treacher Collins syndrome have downward-sloping eyelids; depressed cheekbones; a large fishlike mouth; deformed ears with conductive deafness; a small, receding chin and lower jaw; a highly arched or cleft palate; and severe dental malocclusion. These defects result from defective cranial neural crest cell differentiation, migration and proliferation. Consequently, the first branchial arch structures are deficient, and all derivative craniofacial components are affected.

The underdeveloped facial structures can contribute to airway blockage and repeated upper respiratory infections, either of which can be fatal. The faulty development of the ears leads to a conductive deafness. The severe facial deformities exacerbate the psychological difficulties these youngsters face.

Investigators have identified the gene involved in an autosomal dominant form of the syndrome. The function of the gene is not yet known, but its identity will provide opportunities for prenatal diagnosis, gene therapy and further understanding of craniofacial development.

The Pierre Robin syndrome

Deficient development of the first-branchial-arch-derived mandibular portion results in the lower jaw being set far back in relation to the forehead. As a result, the tongue is set back and may obstruct the posterior airway, compromising respiration and, in severe cases, leading to inadequate aeration and failure to thrive. The infant is also at risk for the development of cor pulmonale, an enlargement of the right ventricle of the heart that occurs secondarily to a chronic lung condition. Cleft palate may be another consequence.

The DiGeorge/Velocardiofacial syndrome

The primary defect in the DiGeorge syndrome results from altered development of the fourth branchial arch and the third and fourth pharyngeal pouches. Deficiencies affecting the thymus, parathyroid glands and the great vessels that derive from these structures result. The facial features are subtle and include a squared-off nasal tip, small mouth and widely spaced eyes. Similar facial features, along with heart defects, are seen in the velocardiofacial syndrome. Both syndromes are associated with deletions on the long arm of chromosome 22 (22q11). Further characterization of this chromosomal deletion region will provide information on the specific gene(s) affected and its function in craniofacial development.

The thymus defects severely compromise cellular immunity, depriving the body of thymus-derived T cells and paving the way for severe infectious disease. Inadequate or missing parathyroid glands cause severe hypocalcemia (low blood calcium levels) and seizures. The great vessel abnormalities alter cardiac output and lead to compromised circulation to heart tissues.

Cranial bone and dental anomalies

Defects in the timing of developmental events can cause premature fusion of cranial bones. Impairments of tooth development can result from interruptions of the developmental sequence at several different stages.

This picture is of a child with a facial deformity caused by a cleft lip/palate.

Craniosynostoses

Some craniofacial anomalies are associated with so-called master genes that orchestrate a program by which the embryo assumes its basic shape. Craniosynostosis, which occurs in approximately 1 out of 3,000 births, is one such anomaly. It results in the premature fusion of the cranial sutures, a dangerous condition that puts pressure on the developing brain. A number of diseases and syndromes, including Crouzon's, Apert's Boston-type craniosynostosis, Pfeiffer's and Saethre-Chotzen, share this anomaly, but differ in other features, which can include structural defects such as webbing of the hands and feet as well as mental retardation. Boston-type craniosynostosis has been linked to MSX2, one of the master genes. Several of the other syndromes involve point mutations at one or another locus in genes that code for fibroblast growth factor receptors (FGFR 1, 2 and 3). Collectively, these genes are associated with cell regulation, either through mediating growth factor effects or by serving as transcription factors.

Hereditary hypodontia or anodontia

Conditions of underdeveloped teeth (hypodontia) or their complete absence (anodontia) have been correlated with specific genes, such as MSX1 and LEF1. The complete absence of teeth alters the bony development of the mandible and maxilla.

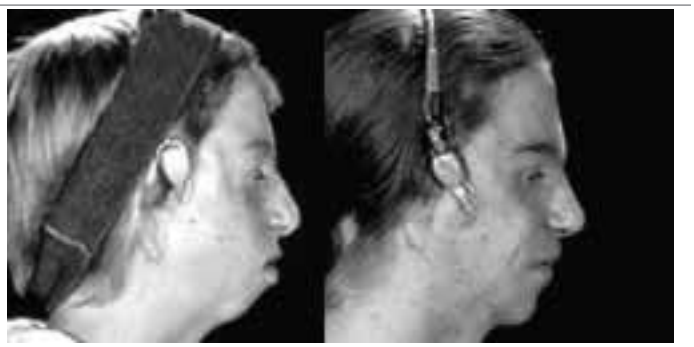
Amelogenesis imperfecta and dentinogenesis imperfecta

Amelogenesis imperfecta and dentinogenesis imperfecta are linked to defects in structural genes that code for proteins essential to the development of tooth enamel (amelogenesis imperfecta) or dentin (dentinogenesis imperfecta). The teeth are weak and extremely sensitive to temperature and pressure. The ordinary forces of chewing are painful and can lead to further wear and pain.

The enamel matrix genes include tuftelin, ameloblastin, and amelogenin; researchers have begun to link mutations in these genes with amelogenesis imperfecta. Similarly, genes labeled DSP and DPP have been characterized for dentin matrix and are associated with the inheritance of dentinogenesis imperfecta.

Craniofacial defects secondary to other developmental disorders

A number of genetic diseases occur in which craniofacial defects are secondary to a more generalized structural defect.



This picture shows Treacher Collins Syndrome patients with jaw deformity.

Osteogenesis imperfecta

Inherited mutations of collagen genes lead to a number of "brittle bone" diseases characterized by defects in mineralized tissues that form from a collagen-rich matrix. Osteogenesis imperfecta presents a spectrum of deficiencies that includes fragile bones, clear or blue sclera, deafness, loose ligaments, and painful dentinogenesis imperfecta-like changes in the teeth.

Epidermolysis bullosa – recessive dystrophic type

The gene defect in epidermolysis bullosa – recessive dystrophic type – manifests as blisters or bullae that appear shortly after birth on skin areas following minor trauma. Mutations in keratin genes that contribute to the epithelial cell cytoskeleton have been correlated with this condition. The oral manifestations include both mucosal bullae and altered teeth. Altered teeth with hypoplastic enamel develop and exhibit an increased susceptibility to caries. Oral bullae develop from even the slightest mucosal trauma. The condition is painful and dangerous because of the constant risk that the bullae will become infected.

Craniofacial manifestations of single-gene defects

In many craniofacial defects, mutations within a single gene manifest as complex syndromes with varied organ and limb defects as well as facial anomalies.

Ectodermal dysplasias

The ectodermal dysplasias (EDs) are a family of hereditary diseases first observed by Charles Darwin over a century ago. They involve defects in two or more tissues derived from the ectoderm – skin, hair, teeth, nails and sweat glands. The ectodermal structures fail to differentiate properly owing to altered epithelial-mesenchymal signaling. A gene, EDA, at an X-linked locus has been linked to the syndrome, and ongoing research is aimed at determining the function of the gene and the molecular mechanism of the syndromes. More recently, investigators have discovered genes linked to autosomal (i.e., non-sex-linked) forms of ED, displaying both dominant and recessive inheritance. Oral manifestations of the ectodermal dysplasias are associated with the teeth. Alterations in tooth development can include hypodontia, anodontia and conically shaped teeth.

The Waardenburg syndrome

The Waardenburg syndrome has been subdivided into several types. All involve a variety of abnormalities in the position and appearance of the nose and eyes, with pigment changes that may cause one eye to differ in color from the other. Other signs include deafness, a mildly protruding jaw, cleft lip and palate, and skeletal deformities. The syndrome is inherited in an autosomal dominant manner with complete penetrance and variable expression. Specific genes associated with this syndrome are members of the homeobox family that regulate the transcription of other genes: Waardenburg type 1 with PAX3; Waardenburg type 2 with MITF, 3q14.1; and Waardenburg type 3 with PAX3, 2q35.

Cleidocranial dysplasia

The inheritance of a regulatory gene defect in cleidocranial dysplasia leads to features that include delayed tooth eruption, supernumerary teeth, altered or missing collarbones, short stature and possible failure of cranial suture closure. The exact mechanism of the associated gene, CBFA1, located on chromosome 6, has not been determined but appears to be essential for bone development.

Injury

The common perception is that injuries are random occurrences that are unpredictable and hence unpreventable. In actuality, experts in the field make the point that there are no basic scientific distinctions between injury and disease. Injuries have been categorized as "intentional" and "unintentional." People identified as being at risk for certain injuries, as well as the causes of those injuries, can be targeted for appropriate

prevention strategies. Such an approach is broadly applicable to sports, falls and motor vehicle injuries (unintentional) as well as to injuries caused by abusive and violent behaviors (intentional).

Injuries are a major public health problem, outranking cancer and heart disease as a leading cause of death in some age groups of the population. Cranial injuries in particular are a leading cause of mortality. Oral-facial injuries can bring disfigurement and dysfunction, greatly diminishing the quality of life and contributing to social and economic burdens.

The leading causes of oral and craniofacial injuries are sports, violence, falls and motor vehicle collisions. Oral cavity injuries may also be caused by foreign objects in food.

Sports

Craniofacial sports injuries occur not only in contact sports, but also in individual activities such as bicycling, skating, and gymnastics, especially on trampolines. Each sport predisposes its participants to a specific array of extrinsic risk factors. These include physical contact, projectiles such as balls and pucks, and the quality of the playing field and equipment. In contact sports the absence of protective equipment such as headguards and mouthguards is a major risk factor. In a recent survey of school-aged children in organized sports, football was the only sport in which the majority of participants used mouthguards and headgear.

There are intrinsic risk factors as well, relating to characteristics of the individual participant. These include age, sex, injury history, body size, aerobic fitness and muscle strength, central motor control, and general mental ability.

Falls

Falls are a major cause of trauma to teeth, primarily to incisors. Unlike bone fractures, fractures of the crowns of the teeth do not heal or repair, and affected teeth often have an uncertain prognosis. Problems may develop later due to damage to the pulp.

Motor vehicle collisions

The effects of motor vehicle collisions may range from minor and reversible effects to long-term medical, surgical and rehabilitative consequences. Post-traumatic headaches and chronic oral-facial pain can occur. Neuromuscular and glandular damage may cause short- or long-term problems with chewing, swallowing and tearing or result in facial tics or paralysis.

Violence

The family is the single most frequent locus of violence in Western society. Domestic violence includes child abuse, spousal and elder abuse, and abuse of the disabled. Child abuse is of particular concern to the oral health community because 65 percent of cases involve head and oral-facial trauma and dentists are required to report suspected cases of child abuse. In the young child, head injury is the most common cause of death.

Psychological trauma from abuse can result in sleep disturbances, eating disorders, developmental growth failure in young children and nervous habits such as lip and fingernail biting and thumb sucking. Effects may also include chronic underachievement in school and poor peer relationships. In abusive families, physical neglect is commonplace, with inadequate provision of basic needs, including medical and oral health care.

Selected chronic pain conditions

Oral, dental or craniofacial signs and symptoms play a critical role in autoimmune disorders such as Sjögren's syndrome and in a number of chronic and disabling pain conditions.

Sjögren's syndrome

Sjögren's syndrome is one of several autoimmune disorders in which the body's own cells and tissues are mistakenly targeted for destruction by the immune system. Like other autoimmune conditions, Sjögren's syndrome is more prevalent among women. The ratio of females to males affected is 9 to 1, with symptoms usually developing in middle age. There are an estimated 1 million to 2 million individuals in the United States with Sjögren's syndrome.

The disease occurs in two forms. Primary Sjögren's involves the salivary and lacrimal (tear) glands. In secondary Sjögren's the glandular involvement is accompanied by the development of a connective tissue or collagen disease, most often rheumatoid arthritis, lupus erythematosis, scleroderma or biliary cirrhosis.

The glandular involvement causes a marked reduction in fluid secretion, resulting in xerostomia and xerophthalmia (dry eyes). The constant oral dryness causes difficulty in speaking, chewing and swallowing; the dry eyes often itch and feel gritty. There is no cure for Sjögren's, and patients often carry eyedrops and water bottles or saliva substitutes in an attempt to provide symptomatic relief. Clinically, the reduction in salivary flow changes the bacterial flora, which, in addition to the reduction in salivary protective components, increases the risk of caries and candidiasis. Recent studies have indicated that there is a reduction in masticatory function and an increased prevalence of periodontal disease. In advanced stages, the salivary glands may swell because of obstruction and infection or lymphatic infiltration. In both forms of the disease, other systems may eventually become affected. Nasal, laryngeal and vaginal dryness may occur, as well as abnormalities in internal organs.

Diagnosis is difficult in the early stages, and women often report that it took many years and consultations with many specialists before they received the correct diagnosis. Diagnosis involves demonstration of specific antibodies in the blood characteristic of an autoimmune disorder, a labial (minor) salivary gland biopsy, and a series of eye tests to measure flow rate and tissue characteristics. Confirmatory tests include an evaluation of salivary flow and chemistry.

Patients with Sjögren's syndrome are at some risk of developing diseases such as non-Hodgkin's lymphoma; clinical data indicate that such lymphomas develop in 5 percent of patients with Sjögren's syndrome.

Histological examination shows that immune cells infiltrate the glands and cluster around the secretory elements, resulting in a breakdown of the normal structure of the gland. The mechanisms by which this occurs involve immune-cell-mediated inflammation and stimulation of the salivary gland cells themselves to produce tissue-destructive molecules such as cytokines. Another hypothesis is that a viral

infection of the glands may trigger the immune response that leads to autoimmunity, whereas genetic or regulatory alterations might lead to abnormalities in apoptosis.

In addition to saliva substitutes and artificial tears, some medications, such as pilocarpine and cevimeline, are prescribed to increase salivary flow from the residual healthy gland tissue, again providing symptomatic relief only. The problems that develop in the other organ systems are also treated symptomatically. At advanced stages, steroids are employed intermittently to alleviate problems.

Acute and chronic oral-facial pain

Since the 19th century when two dentists, Horace Wells and Frederick Morton, demonstrated the analgesic powers of nitrous oxide and ether, oral health investigators have been recognized leaders in the field of pain management worldwide. Their analyses of the cells, pathways and molecules involved in the transmission and modulation of pain have given rise to a growing variety of medications, often combined with other approaches, that can control acute and chronic pain. Pain researchers today stress that chronic pain can become a disease in itself, causing long-term detrimental changes in the nervous system. These changes may affect resistance to other diseases as well as effectively destroy quality of life. Most people have experienced acute pain involving teeth and the oral tissues at one time or another.

Atypical facial pain

Atypical facial pain is characterized by a continuous dull ache on one or both sides, most frequently in the region of the maxilla (the upper jaw). The pain tends to be episodic and is aggravated by fatigue, worry or emotional upset. It is often accompanied by pain elsewhere in the body and depression. Once a dental cause can be ruled out, pain resolution depends on the successful use of antidepressants, psychotherapy or both.

Tic douloureux

The oral-facial region is also subject to pain that can be paroxysmal or continuous along a distinct nerve distribution. The most frequently encountered of these oral facial neuralgias is tic douloureux, or trigeminal neuralgia, a disease of unknown etiology affecting one, two or all three branches of the trigeminal nerve. The pain is highly intense and of a stabbing nature, and lasts for a few seconds. This transient attack may be repeated every few minutes or several hours. There may be no precipitating factor, or it may occur in response to a gentle touch or a breeze wafting across the face – a condition experts call allodynia, the feeling of pain in response to a normally nonpainful stimulus. On other occasions, there may be a specific trigger zone. Although spontaneous remission for weeks or months may occur, it is rarely permanent. Given the unknown, unpredictable nature of tic douloureux, it is not surprising that fear of pain comes to dominate these patients' lives, as they avoid doing anything that might trigger an attack.

Trigeminal neuralgia generally occurs in later life, but also occurs in younger individuals affected by multiple sclerosis, where it is assumed to be associated with lesions (multiple sclerosis "plaques") in the brain stem. Medical treatment depends largely on the use of a drug that has become a virtual specific, the antiepileptic drug carbamazepine. For those patients with no consequential adverse effects, it can control the disease. An alternative for chronic sufferers is the surgical removal of a small vein or artery that may be exerting pressure on the nerve root or the selective destruction of the nerve fibers themselves using chemical or electrical methods. In many cases, these procedures can produce complete relief from pain.

Temporomandibular disorders

Various etiological factors, including trauma, can give rise to pain and dysfunction in the temporomandibular joint and surrounding muscles, conditions collectively called temporomandibular disorders (TMDs). The pain may be localized or radiate to the teeth, head, ears, neck, and shoulders. Abnormal grating, clicking or crackling sounds, known as crepitus, in the joint often accompany localized pain. Pain is also found in response to clinical palpation of the affected structures. TMDs are common, occurring in as many as 10 million Americans. Although surveys indicate that both sexes are affected, the majority of individuals seeking treatment are women of childbearing age, a phenomenon suggesting that hormonal influences should be investigated.

Several factors can contribute to the onset or exacerbation of TMD symptoms. These factors include:

- Certain developmental anomalies.
- Injury to the jaw from accidents or abuse.
- Oral habits that greatly stress the joint and musculature, such as tooth grinding (bruxism).
- Jaw manifestations of systemic diseases such as fibromyalgia and arthritic diseases; and some irreversible treatments for initial signs and symptoms.

The multiplicity of factors that may cause or contribute to TMDs has unfortunately led to a multiplicity of treatments. Most of these treatments have not been tested in randomized controlled clinical trials. During the 1970s and 1980s, many individuals underwent surgery that proved unsuccessful in many cases.

Leading investigators have proposed standardized research diagnostic criteria to clarify the kinds of pathology that can give rise to TMDs and to classify the most common forms of TMDs. Such criteria could be used in designing clinical trials and could ultimately lead to better diagnostics, treatments and prevention.

The criteria use two dimensions or axes:

- Axis I delineates various forms of joint or muscle pathology.
- Axis II explores pain-related disability and psychological status.

The approach requires detailed clinical examinations and patient histories.

A mirror, a model and a better understanding of diseases and disorders

Studying the diseases and disorders that affect craniofacial tissues can provide scientists with models of systemic pathology. Because some craniofacial tissues, such as bones, mucosa, muscles, joints and nerve endings, have counterparts in other parts of the body and these tissues are often more accessible to research analysis than deeper-lying tissues, researchers studying craniofacial tissues can gain valuable insights into how cancer develops, the role of inflammation in infection and pain, the effects of diet and smoking, the consequences of depressed immunity and the changes that can arise from a mutated gene.

Other craniofacial tissues, teeth, gingiva, tongue, salivary glands and the organs of taste and smell, are unique to the craniofacial complex. Study of the diseases affecting these tissues has revealed a wealth of information about their special nature as well as the molecules and mechanisms that normally operate for the protection, maintenance and repair of all the oral, dental and craniofacial tissues. When factors perturb these nurturing elements, the oral health scale can tip toward disease. When those factors stem from systemic diseases or disorders, the mouth can sometimes mirror the body's ill health. Similarly underscoring the connection between oral and general health are studies suggesting that poor dental health, mainly due to chronic dental infections, may heighten the risk for both cardiovascular disease and stroke independently of factors such as social class and established cardiovascular risk factors.

Infection and disorder highlights

- Microbial infections, including those caused by bacteria, viruses and fungi, are the primary cause of the most prevalent oral diseases. Examples include dental caries, periodontal diseases, herpes labialis and candidiasis.
- The etiology and pathogenesis of diseases and disorders affecting the craniofacial structures are multifactorial and complex, involving an interplay among genetic, environmental and behavioral factors.
- Many inherited and congenital conditions affect the craniofacial complex, often resulting in disfigurement and impairments that may involve many body organs and systems and affect millions of children worldwide.
- Tobacco use, excessive alcohol use and inappropriate dietary practices contribute to many diseases and disorders. In particular, tobacco use is a risk factor for oral cavity and pharyngeal cancers, periodontal diseases, candidiasis and dental caries, among other diseases.
- Some chronic diseases, such as Sjögren's syndrome, present with primary oral symptoms.
- Oral-facial pain conditions are common and often have complex etiologies.

The range of oral, dental and craniofacial diseases and conditions that take a toll on the U.S. population is extensive. This course provides highlights of diseases and conditions affecting Americans using available national and state data to describe the burden of disease in the United States. There is no single measure of oral health or the burden of oral diseases and conditions, just as there is no single measure of overall health or overall disease. The relationship of oral health to the use of dental services is described.

However, the effects of health care visits and of specific services rendered need further study.

Physical signs and symptoms of disease and risk factors

A number of signs and symptoms of disease, lifestyle behaviors and exposure to toxins can be detected in or around the craniofacial complex. Pathogens entering the mouth may proliferate locally with oral and pharyngeal signs and symptoms; other pathogens may enter the bloodstream directly or through lymphatic channels and cause generalized disease. Oral signs suspected to be indications of systemic illness may be confirmed by the presence of rash, fever, headache, malaise, enlarged lymph nodes or lesions elsewhere on the body.

Swollen parotid glands are a cardinal sign of infection with the mumps virus and can also be seen in individuals with Sjögren's syndrome and HIV. The salivary glands are also frequently involved in tuberculosis and histoplasmosis infections. Oral signs of infectious mononucleosis, caused by Epstein-Barr virus, include sore throat, gingival bleeding and multiple pinpoint-sized hemorrhagic spots (pettechiae) on the oral mucosa. There can be a large overlap in the clinical appearance of oral manifestations of various diseases with different etiologies, and the clinical diagnosis often involves ancillary procedures, which may include laboratory tests, diagnostic imaging and biopsy.

Oral tissues may also reflect immune deficiency. For example, nearly all HIV-infected individuals develop oral lesions at some time during their illness. Other immunosuppressed individuals may have the same lesions. However, the presentation and the extent, severity and management of some of these lesions may reflect nuances due to variation in the underlying systemic condition. For example, the linear gingival erythema and necrotizing ulcerative periodontitis sometimes seen in HIV infection have been difficult to resolve with routine dental curettage and prophylaxis.

The appearance of soft or hard tissue pigmentation is associated with a number of diseases and treatments. Malignant melanoma can appear in the mouth as brown or black flat or raised spots. Kaposi's sarcoma can appear as a flat or raised pigmented lesion. Addison's disease causes blotches or spots of bluish-black or dark brown pigmentation to occur early in the disease. Congenital discrete brown or black patches (nevi) can appear in any part of the mouth. Pigmentation of the tooth crowns may be seen in children with cystic fibrosis and porphyria and those exposed to tetracycline during tooth development.

The oral tissues can also reflect nutritional status and exposure to risk factors such as tobacco. The tongue appears smooth in pernicious anemia. Group B vitamin deficiency is associated with oral mucositis and ulcers, glossitis, and burning sensations of the tongue. Scurvy, caused by severe vitamin C deficiency, is associated with gingival swelling, bleeding, ulceration and tooth loosening. Lack of vitamin D in utero or infancy impairs tooth development. Enamel hypoplasia may result from high levels of fluoride or from disturbances in calcium and phosphate metabolism, which can occur in hypoparathyroidism, gastroenteritis, and celiac disease. The mouth also can reflect the effects of tobacco use, perhaps providing the only visible evidence of its adverse effects.

Oral manifestations of HIV infection and of osteoporosis

The mouth can serve as an early warning system, diagnostic of systemic infectious disease and predictive of its progression, such as with HIV infection. In the case where oral cells and tissues have counterparts in other parts of the body, oral changes may indicate a common pathological process. During routine oral examinations and perhaps in future screening tests, radiographic or magnetic resonance imaging of oral bone may be diagnostic of early osteoporotic changes in the skeleton. The following sections provide details.

HIV infection

The progressive destruction of the body's immune system by HIV leads to a number of oral lesions, such as oral candidiasis and oral hairy leukoplakia, that have been used not only in diagnosis but also in determining specific stages of HIV infection. Oral candidiasis is rarely seen in previously healthy young adults who have not received prior medical therapy such as cancer chemotherapy or treatment with other immunosuppressive drugs. It was associated with AIDS as early as 1981 in the first report of the syndrome and was frequently

noted among otherwise asymptomatic HIV-positive populations. Oral candidiasis may be the first sign of HIV infection and often occurs as part of the initial phase of infection, the acute HIV syndrome. It tends to increase in prevalence with progression of HIV infection when CD4 lymphocyte counts fall. It also appears to be the most common oral manifestation in pediatric HIV infection and has been demonstrated to proceed to esophageal candidiasis, a sign of overt AIDS. Both the pseudomembranous and the erythematous forms of candidiasis appear to be important predictors of progression of HIV infection.

Like oral candidiasis, oral hairy leukoplakia in HIV-positive persons heralds more rapid progression to AIDS. Oral hairy leukoplakia is an oral lesion first reported in the early days of the AIDS epidemic. Since its discovery, hairy leukoplakia has been found in HIV-negative persons with other forms of immunosuppression, such as organ or bone marrow recipients and those on long-term steroid therapy and less frequently among immunocompetent persons.

Osteoporosis, a degenerative disease characterized by the loss of bone mineral and associated structural changes, has long been suspected as a risk factor for oral bone loss. In addition, measures of oral bone loss have been proposed as potential screening tests for osteoporosis. Osteoporosis affects over 20 million people in the United States, most of whom are women, and results in nearly 2 million fractures per year. The disease is more prevalent in white and Asian American women than in black women. Oral bone loss has been reported to be more prevalent in women than in men.

Oral-fluid-based diagnostics: The example of saliva

The diagnostic value of salivary secretions to detect systemic diseases has long been recognized, and oral fluids and tissues (buccal cells) are increasingly being used to diagnose a wide range of conditions. Saliva- and oral-based diagnostics use readily available samples and do not require invasive procedures. Researchers have detected antibodies in saliva that are directed against viral pathogens such as human immunodeficiency virus and hepatitis A virus or B virus. Saliva is being used to detect antibodies, drugs, hormones, and environmental toxins. The simplest tests are those that detect the presence or absence of a substance in the saliva, such as various drugs. Greater technical challenges are presented for tests that will be used for therapeutic monitoring since accurate levels of a substance and/or its metabolites are needed. In these instances the saliva/plasma concentration ratio must be determined experimentally. Most recently, oral fluids have been used as a source of microbial or host DNA.

Saliva has the potential of replacing blood, the current standard for testing many diseases and conditions (e.g., diabetes, infectious disease, Parkinson's disease, alcoholic cirrhosis, Sjögren's syndrome and cystic fibrosis sarcoidosis). Important goals for the future are the development of new diagnostic tests for early disease detection, defining individual patient risk of adverse response to drugs, monitoring therapeutic progress and determining outcomes of treatment. Key issues in the development of a new generation of saliva diagnostics include their selectivity, sensitivity, response time, dynamic range (values of interest), representative sampling and, perhaps most important, their reliability or stability as well as their ability to assess multiple substances simultaneously.

For the clinician, the mouth and face provide ready access to physical signs and symptoms of local and generalized disease and risk factor exposure. These signs and symptoms augment other clinical features of underlying conditions. Comprehensive care of the patient requires knowledge of these signs and symptoms, their role in the clinical spectrum of general diseases and conditions, and their appropriate management. Oral biomarkers and surrogate measures are also being explored as means of early diagnosis. With further development and refinement, oral-based diagnostics such as salivary tests can become widely used and acceptable tools for individuals, health care professionals, researchers and community programs. The continued refinement of imaging techniques also has the potential of using oral imaging to identify early signs of skeletal bone degeneration.

The mouth as a portal of entry for infection

More than 500 bacterial strains have been identified in dental biofilm, and more than 150 bacterial strains have been isolated from dental pulp infections. More recently, 37 unique and previously unknown strains of bacteria were identified in dental plaque (biofilm). Most oral lesions are opportunistic infections – that is, they are caused by microorganisms commonly found in the mouth, but normally kept in check by the body's defense mechanisms. These microorganisms can induce extensive localized infections that compromise general well-being in and of themselves.

However, they also may spread to other parts of the body if normal barriers are breached. The oral mucosa is one such barrier that provides critical defense against pathogens and other challenges. Salivary secretions are a second major line of defense. Damage to the oral mucosa from mechanical trauma, infection or salivary dysfunction with resulting derangements in lubricatory and antimicrobial functions of saliva as a result of chemotherapy, radiation and medications causing hyposalivation, allows a portal of entry for invading pathogens.

Oral infections and bacteremia

Oral microorganisms and cytotoxic byproducts associated with local infections can enter the bloodstream or lymphatic system and cause damage or potentiate an inappropriate immune response elsewhere in the body. Dissemination of oral bacteria into the bloodstream (bacteremia) can occur after most invasive dental procedures, including tooth extractions, endodontic therapy, periodontal surgery and scaling and root planing. Even routine oral hygiene procedures such as daily toothbrushing, subgingival irrigation and flossing may cause bacteremia. However, these distant infections have been seen more often in high-risk patients such as those who are immunocompromised.

Oral bacteria have several mechanisms by which they invade mucosal tissues, perhaps contributing to their ability to cause bacteremias. For example, oral bacteria and their products may invade the periodontal tissues directly. Actinobacillus actinomycetemcomitans has been found in gingival connective tissue in patients with localized juvenile periodontitis. Invasion of tissue by Porphyromonas gingivalis has also been described in vivo and in vitro. Although oral bacteria can enter the blood through injured or ulcerated tissue, bacterial invasion of periodontal tissues represents another possible mechanism.

In the immunocompetent individual, bacteremia originating from the oral cavity is usually transient and harmless. However, if the individual's immune system is compromised, the normally harmless oral bacteria may pose a significant risk. The morbidity and mortality associated with oral foci of infections are hard to assess. This is due to the formidable task of tracking the source of an infection unless the responsible pathogen is indigenous to a specific anatomic location.

Viridans group streptococci (VGS) have a low degree of virulence but can be associated with morbidity and mortality under certain circumstances. Increased pathogenicity of Streptococcus viridans is most prominent in individuals with neutropenia (low blood counts of circulating white blood cells called neutrophils) and has been

associated with a toxic-shock-like syndrome (TSLS) or viridans streptococcal shock syndrome (VSSS), as well as with adult respiratory distress syndrome (ARDS).

Although a high degree of morbidity is associated with viridans streptococcal bacteremia, a low incidence of mortality has been reported. Several studies have shown that under adverse circumstances, oral flora and oral infections are associated with increased incidence of morbidity. Reduction of oral foci of infection decreases systemic complications, specifically in severely neutropenic patients undergoing chemotherapy. In addition, hospital stays for patients with oral mucositis undergoing autologous bone marrow transplants were longer than for those without oral mucositis.

Other cohorts identified at increased risk for systemic complications due to oral bacteria include hospitalized patients unable to perform adequate oral hygiene, those receiving saliva-reducing medications and those taking antibiotics that alter the oral flora. A positive dental plaque culture for aerobic pathogens was significantly associated with the development of hospital-acquired pneumonia and bacteremia in a study of individuals in an intensive care unit.

In addition, several case reports have been published implicating indigenous oral flora in the development of brain abscesses. This serious condition is associated with a mortality rate of almost 20 percent, and full recovery in only slightly more than 50 percent of all patients. These data are based on single case reports and most probably represent rare events. However, they provide additional examples that point to the potential pathogenicity of the normal oral flora during special adverse circumstances.

ORAL INFECTIONS AS A RESULT OF THERAPY

Chemotherapy

Oral mucositis can be a major dose-limiting problem during chemotherapy with some anticancer drugs, such as 5-fluorouracil, methotrexate, and doxorubicin. It is estimated that approximately 400,000 patients undergoing cancer therapy each year will develop oral complications (NIH 1990). Infection of ulcerated mucous membranes often occurs after chemotherapy, especially since patients are usually immunocompromised. Bacterial, fungal, and viral causes of mucositis have been identified.

The mechanism by which cancer-chemotherapy-induced mucositis occurs is likely associated with the rapid rate of turnover of oral epithelial cells. In addition, other components likely include upregulation of pro-inflammatory cytokines and metabolic by-products of colonizing oral microflora. Chemotherapy alters the integrity of the mucosa and contributes to acute and chronic changes in oral tissue and physiologic processes. The ulcerated mucosa is susceptible to infection by microbial flora that normally inhabit the oral cavity, as well as by exogenous organisms, and exacerbates the existing mucositis. Further, these microflora can disseminate systemically. Compromised salivary function can further elevate risk for systemic infection of oral origin.

Both indigenous oral flora and hospital-acquired pathogens have been associated with bacteremias and systemic infection. Changes in infection profiles in myelosuppressed (immunosuppressed) cancer patients tend to occur in cyclic fashion over many years. This evolving epidemiology is caused by multiple factors including use of antibiotics. Gram-positive organisms including viridans streptococci and enterococci are currently associated with systemic infection of oral origin in myelosuppressed cancer patients. In addition, gram-negative pathogens including P. aeruginosa, Neisseria spp. and Escherichia coli remain of concern.

Cancer patients undergoing bone marrow radiation who have chronic periodontal disease may also develop acute periodontal infections with systemic complications. The extensive ulceration of gingival sulcular epithelium associated with periodontal disease is not directly observable clinically, yet may represent a source for disseminated infection by an extensive array of organisms. Inflammatory signs may be masked due to the underlying bone marrow suppression.

Viruses are also associated with clinically important oral disease in patients receiving chemotherapy. Infections caused by herpes simplex virus, varicella-zoster virus and Epstein-Barr virus typically result from reactivation of a latent virus, whereas cytomegalovirus infections can result via reactivation of a latent virus or a newly acquired virus. The severity of the infection, including fatal outcome, depends on the degree of immunocompromise.

Radiation therapy

Radiation therapy disrupts cell division in healthy tissue as well as in tumors and also affects the normal structure and function of craniofacial tissues, including the oral mucosa, salivary glands and bone. Oral-facial complications are common after radiation therapy to the head and neck. The most frequent, and often the most distressing, complication is mucositis, but adverse reactions can affect all oral-facial tissues.

Radiation can cause irreversible damage to the salivary glands, resulting in dramatic increases in dental caries. Oral mucosal alterations may become portals for invasion by pathogens, which may be life-threatening to immunosuppressed or bone-marrow-suppressed patients. A less common but very serious adverse consequence is destruction of bone cells and bone death, called osteoradionecrosis (ORN). ORN can result in infection of the bone and soft tissue and can require surgery to excise the dead tissue, which can in turn leave the face badly disfigured as well as functionally impaired. The likelihood of ORN is increased with trauma to the bone, including that caused by tooth extraction. The risk is especially marked when the trauma occurs near the time of radiation. Management includes elimination of acute or potential dental and periodontal foci of disease, increased patient participation in oral hygiene, use of oral topical fluorides for caries prevention and use of antiviral, antifungal or antimicrobial therapy for management of infections associated with mucositis.

Combined cancer therapies

Rapid developments have occurred in the use of blood cell growth factors for treatment of various conditions, including the anemia of end-stage renal disease, the neutropenia occurring with cancer care and the bone marrow toxicity and mucositis that can follow aggressive chemotherapy or radiation therapy. Researchers have found that topical application of transforming growth factor beta (TGF-B) in the hamster model of oral mucositis significantly reduced basal cell proliferation and reduced the severity of mucositis associated with 5-fluorouracil treatment.

Other growth factors considered for use in reducing mucositis include granulocyte-monocyte colony-stimulating factor and granulocyte colony-stimulating factor. Bone morphogenetic proteins are also in development for alleviating the toxicity and mucositis that follow chemotherapy and radiation therapy. Other approaches to reducing

mucositis and adverse oral effects of chemotherapy and radiation therapy include fractionating the dose of radiation and combining chemotherapy with growth factors or with less toxic oncostatic agents.

Although the oral mucositis occurring in chemotherapy and in head and neck radiation patients shares many characteristics, distinct differences also exist. For example, in contrast to chemotherapy-associated lesions, radiation damage is anatomically site-specific; toxicity is localized to irradiated tissue volumes. The degree of damage depends on treatment-regimen-related factors, including the type of radiation used, the total dose administered, the fractionation, and field size. Thus, research involving both cohorts of cancer patients remains essential to enhancing patient management.

Development of new technologies to prevent cancer-therapy-induced oral mucositis could substantially reduce the risk for oral and systemic infections, oral pain and the number of hospital days. Improvement in quality of life and reduction in health costs are also likely and desirable outcomes.

The new technologies could also provide a setting in which novel classes of chemotherapeutic drugs, used at increased doses, could be implemented. These advances in turn could lead to enhanced cancer patient survival and lengthen the duration of disease remission.

Pharmaceuticals

A number of medications used to treat systemic diseases can cause oral complications, ranging from xerostomic effects to alterations in the surface structure of the enamel or mucosa. More than 400 over-the-counter and prescription drugs have xerostomic side effects. These include tricyclic antidepressants, antihistamines and diuretics. The dimensions and impact of these side effects vary depending on the response of the individual patient and the duration of medication use.

Staining of the teeth or mucosa is associated with a variety of drugs, including tranquilizers, oral contraceptives and antimalarials. The antibiotic tetracycline can cause enamel hypoplasia when taken by the mother during pregnancy and by children during tooth development. The antimicrobial mouthrinse agent chlorhexidine also can stain the teeth, but this staining is external and can be removed by dental prophylaxis.

Other drugs have been associated with gingival overgrowth, including cyclosporin, which has been used as an immunosuppressant in the United States since 1984 to prevent rejection of transplanted organs and bone marrow. This drug has also been used in other countries for treatment of type 2 diabetes, rheumatoid arthritis, psoriasis, multiple sclerosis, malaria, sarcoidosis and several other diseases with an immunological basis. Other drugs that cause gingival overgrowth include calcium ion channel blocking agents used in the treatment of angina pectoris and postmyocardial syndrome, such as nifedipine and verapamil, and phenytoin (sodium 5,5-phenylhydantoin), used in the treatment of epilepsy and also for management of other neurological disorders. Treatment often consists of using an alternate drug, although this is not always possible. Conservative periodontal therapy can reduce the inflammatory component of enlargement, but surgery is often required. Drugs that cause systemic bone marrow suppression, oral mucosal injury or salivary compromise collectively promote the risk for clinical infection. In addition, antibiotics and concurrent steroid therapy often alter oral flora, thereby creating an environment for fungal overgrowth. In high-risk cancer patients, fungal infection can cause severe morbidity and even death.

Infective endocarditis

The purported connection between oral infection and a specific heart disease, infective endocarditis, has a long history. Endocarditis is caused by bacteria that adhere to damaged or otherwise receptive surfaces of the tissue that lines heart valves (the endocardium). Dental and other surgical procedures may predispose susceptible patients to infective endocarditis by inducing bacteremias. However, bacteremias from oral infections that occur frequently during normal daily activities, coincidental even with chewing food, toothbrushing and flossing, contribute more substantially to the risk of infective endocarditis. Oral organisms are common etiologic agents of infective endocarditis. For example, strains of S. sanguis, as well as gram-negative oral bacteria including Haemophilus aphrophilus, A. actinomycetemcomitans, E. corrodens, Capnocytophaga spp., and Fusobacterium nucleatum, have been associated with bacterial endocarditis.

Infective endocarditis occurs with different incubation periods, which differ in causative bacteria and signs and symptoms. For example, Staphylococcus aureus endocarditis may have a rapid onset and fatal course if it affects the left side of the heart. With a more indolent course, patients may often be unaware of infection and may experience fever, night chills, myalgia and arthralgia for a considerable period of time before diagnosis. The infection is often curable if diagnosed and treated early.

The classic risk factors for endocarditis include cardiac valve disorders (valvulopathies) that include rheumatic and congenital heart disease, complex cyanotic heart disease in children, and mitral valve prolapse with regurgitation. Recent studies indicate that the use of certain diet drugs (fenfluramine and dexfenfluramine) has induced cardiac valvulopathy, which may in some cases be transient. Among at-risk persons, bacteremias are more likely to occur in those with periodontal disease. However, the oral pathogens causing periodontitis have only rarely been shown to cause endocarditis.

Prevention of infective endocarditis from oral bacteria depends on limiting the entry and dissemination of bacteria through the bloodstream and lymphatic circulation. Antibiotic prophylaxis for dental procedures that are likely to provoke bacteremia has historically been recommended. A recent study, however, suggests that receiving dental treatment does not significantly increase the risk of infective endocarditis, even in patients with valvular abnormalities. Further research is necessary to determine whether some heart or valvular conditions or certain dental procedures, such as surgery or scaling, would require coverage with pre-procedural antibiotics and others would be precluded.

Oral infections and respiratory disease

Pathogens in the oral cavity can also gain access to the airway, sometimes with serious consequences. In adults, bacterial pneumonias are strongly associated with aspiration of bacteria into the lower respiratory tract, which is normally sterile. Common respiratory pathogens such as Streptococcus pneumoniae, Streptococcus pyogenes, Mycoplasma pneumoniae and Haemophilus influenzae can colonize the oropharynx and the lower airway. In addition, oral bacteria including A. actinomycetemcomitans, Actinomyces israelii, Capnocytophaga spp., Eikenella corrodens, Prevotella intermedia and Streptococcus constellatus can be aspirated into the lower airways.

Chronic obstructive pulmonary disease, characterized by obstruction of airflow due to chronic bronchitis or emphysema and by recurrent episodes of respiratory infection, has been associated with poor oral health status. Although oral bacteria, including periodontal pathogens, have the potential for causing respiratory infections, the frequency and nature of such infections are not known and merit further study.

Oral transmission of infections

Besides being a portal of entry for infections, the mouth is an important source of potentially pathogenic organisms and is often the vehicle by which infection is delivered to the bodies of others. Microorganisms were not discovered in the mouth until the 17th century, when van Leewenhoek examined dental plaque using a microscope he had constructed. In 1884, Koch demonstrated that tuberculosis could be transmitted by airborne droplets from the mouth and respiratory tract. Since that time, we have learned that many common respiratory infections, such as influenza, the common cold, pneumonia and tuberculosis, can be transmitted from oral secretions. Before the development of effective vaccines, orally transmitted diseases such as chickenpox, measles, mumps, polio and diphtheria were a major source of morbidity and mortality in childhood. Viral diseases such as hepatitis B, herpes labialis, acute herpetic gingivostomatitis, cytomegalovirus and infectious mononucleosis may also originate from oral contact.

Disease-causing microorganisms can be spread by direct contact (with saliva or blood from the mouth) or indirect contact (with saliva- or blood-contaminated surfaces, including hands or lips), droplet infection (from coughing, sneezing or even normal speech), or by aerosolized organisms. These organisms can be inhaled, ingested or taken in through mucous membranes in the eyes, nose or mouth or through breaks in the skin. A number of diseases can be spread via oral sexual contact, including gonorrhea, syphilis, trichomoniasis, chlamydia and mononucleosis.

As mentioned earlier, the oral mucosa and saliva provide significant defense against disease transmission. Epidemiological and animal studies are providing evidence, however, that the oral cavity may be the site for transmission of serious systemic infections despite the protective factors in saliva. Infection with HIV provides a case in point.

Early in the 1980s, when AIDS was first identified in the United States, concern was expressed about casual (i.e., nonsexual) transmission of HIV. Detailed household studies did not demonstrate transmission of HIV, even when family members shared eating utensils and toothbrushes with an HIV-affected member. Similarly, surveillance data collected over time showed no evidence of casual transmission.

Only one nonoccupational episode of HIV transmission has been attributed to blood-contaminated saliva; this incident involved intimate kissing between sexual partners. There have been a few cases of HIV transmission from performing oral sex on a person infected with HIV, and it is also possible to become infected with HIV by receiving oral sex. In the San Francisco Options Study of men who have sex with men identified within 12 months of HIV seroconversion, oral transmission represented 7.8 percent of primary HIV infections. Rothenberg et al. (1998) reviewed epidemiologic studies and reports of 38 cases of oral transmission of HIV in the literature. They concluded that although oral-genital contact may be less efficient than needle-sharing or anal intercourse for the transmission of HIV, its increased use by men who have sex with men and in crack cocaine smokers may increase its contribution to HIV transmission over time. Several studies provide evidence that when the oral environment is compromised, the mouth can be a potential site of transmission of infectious microbes. Data suggest that there is a positive association between the presence of oral lesions resulting from crack cocaine use, receptive oral intercourse and HIV transmission. A case report has documented the passage of HIV from a partner who is HIV-positive to one who is HIV-negative in the presence of periodontal disease but in the absence of other risk factors. Because the type, duration and frequency of oral contact in past studies may not have been specified, the risk could be somewhat higher for oral transmission of HIV than previously reported. The risk might also vary depending on factors such as viral load, infectious dose, area of exposure and presence or absence of oral lesions. Additional studies are needed to evaluate the risk of oral-genital transmission of HIV; some are under way.

Other sexually transmitted diseases (STDs) can occur through oral contact. For example, pharyngeal infection with Chlamydia trachomatis has been found in 3 to 6 percent of men and women attending STD clinics. Most infections are asymptomatic. Another common sexually transmitted infection, herpes simplex virus, commonly infects the pharynx and is seen in 20 percent of patients with primary genital herpes. The painless chancre of primary syphilis can be found in the oral cavity; however, there are no data on the prevalence of this site of infection for Treponema pallidum. Among persons with gonorrhea, pharyngeal infection occurs in 3 to 7 percent of heterosexual men, 10 to 20 percent of heterosexual women and 10 to 25 percent of men who have sex with men. Gonococcal infection can cause acute pharyngitis, but is usually asymptomatic. The transmission of pharyngeal gonorrhea to sex partners had been thought to be rare. However, in one study, 17 of 66 men who had sex with men who had urethral gonorrhea reported insertive oral sex as their only risk factor in the past two months.

The role of the mouth as a portal of entry for infection presents ever-new challenges for study. Although oral tissues and fluids normally provide significant barriers and protection against microbial infections, at times these infections can not only cause local disease but also, under certain circumstances, can disseminate to cause infections in other parts of the body. The control of existing oral infections is clearly of intrinsic importance and a necessary precaution to prevent systemic complications.

Associations among oral infections and diabetes, heart disease/stroke and adverse pregnancy outcomes

Recent studies have reported associations between oral infections, primarily periodontal infections, and diabetes, heart disease and stroke, and adverse pregnancy outcomes, but sufficient evidence does not yet exist to conclude that one leads to the other. This section characterizes the nature of these associations by describing the quality of the evidence supporting the reports. Both observational and experimental studies were accepted as admissible evidence. Where there are operative mechanisms proposed that support an association between oral infectious agents and the systemic conditions in question, they are introduced at the outset.

The periodontal disease-diabetes connection

There is growing acceptance that diabetes is associated with increased occurrence and progression of periodontitis, so much so that periodontitis has been called the “sixth complication of diabetes.” The risk is independent of whether the diabetes is type 1 or type 2. Type 1 diabetes is the condition in which the pancreas produces little or no insulin. It usually begins in childhood or adolescence. In type 2 diabetes,

secretion and utilization of insulin are impaired; onset is typically after age 30. Together, these two types of diabetes affect an estimated 15.7 million people in the United States and represent the seventh leading cause of death. The goal of diabetic care is to lower blood glucose levels to recommended levels. Some investigators have reported a two-way connection between diabetes and periodontal disease, proposing that not only are diabetic patients more susceptible to periodontal disease, but also the presence of periodontal disease affects glycemic control.

Effects of diabetes on periodontitis prevalence and severity

Several reviews have described candidate mechanisms to explain why individuals with diabetes may be more susceptible to periodontitis. These include vascular changes, alterations in gingival crevicular fluid, alterations in connective tissue metabolism, altered host immunological and inflammatory response, altered subgingival microflora and hereditary patterns. Studies were classified by type of diabetes and age of study population.

Type 1 diabetes

Ten reports focused principally on children and adolescents with type 1 diabetes, comparing them with groups of similar ages without diabetes. All but one of the studies reported greater prevalence, extent or severity of at least one measure or index of periodontal disease (e.g., gingival inflammation, probing pocket depth, loss of periodontal attachment or radiographic evidence of alveolar bone loss) among subjects with diabetes, even though these investigations were conducted in a variety of countries across several continents.

Another set of studies on the relationship between type 1 diabetes and periodontal disease included subjects with and without diabetes between the ages of 15 and 35. All six studies reported greater prevalence, extent or severity of at least one measure or index of periodontal disease. A third set of studies conducted in Scandinavia looked at the relationship between periodontal disease and type 1 diabetes (or diabetes reported as requiring insulin therapy without specification of diabetes type) in adults between 20 and 70 years old. Three of the four studies were cross-sectional, and one was a treatment follow-up study. All four studies reported greater prevalence, extent or severity of at least one measure of periodontal disease.

Type 2 diabetes

There are fewer reports on the relationship between type 2 diabetes and periodontal disease, particularly where type 2 diabetes is explicitly identified or discernible from the ages of subjects. Seven studies limited to subjects with type 2 diabetes included a comparison group without diabetes. Two of these studies included only adult subjects; the remaining five were large population-based studies of diabetes and periodontal disease in Pima Indians, a group with the highest known prevalence of type 2 diabetes in the world. The Pima Indian studies included subjects aged 5 years and older or 15 and older. All seven studies reported greater prevalence, extent or severity of periodontal disease among subjects with diabetes for at least one measure of periodontal disease. Three of these studies were longitudinal and showed that the progression of periodontal disease was greater in diabetes patients than in individuals without diabetes.

In addition to finding significant differences in various measures of periodontal status between subjects with and without type 2 diabetes, a number of these reports also provide estimates of association and risk. Using periodontal attachment loss as the measure estimated that people with type 2 diabetes were 2.8 times more likely to have destructive periodontal disease. When they used radiographic bone loss as the measure and controlled for other important covariates, the estimate rose to 3.4.

Diabetes type not specified

The final set of reports on the association between diabetes and periodontal diseases consists of seven cross-sectional studies in which the type of diabetes was not specified and was not easily determined from other information provided. Four of the seven studies included only adults. In the other three studies, subjects ranged in age from childhood to older adulthood. All seven studies found subjects with diabetes to have increased prevalence, extent, and severity of periodontal disease.

Diabetes is a risk factor for the occurrence and prevalence of periodontal diseases. Although there is insufficient evidence of a causal association, the findings of greater prevalence, severity, or extent of at least one manifestation of periodontal disease in individuals with diabetes is remarkably consistent in the overwhelming majority of studies. Furthermore, there are no studies with superior design features in the literature to refute this assessment. The studies were conducted in distinctly different settings, with subjects from different ethnic populations and of different ages, and with a variety of measures of periodontal status. This inevitable variation in methodology and study populations limits the possibility that the same biases apply in all the studies. There is a need for further research using stronger designs that also control for confounding variables such as socioeconomic status.

Glycemic control

Several lines of evidence support the plausibility that periodontal infections contribute to problems with glycemic control, thus compromising the health of diabetic patients. It has been reported that the chronic release of tumor necrosis factor alpha (TNF-alpha) and other cytokines such as those associated with periodontitis interferes with the action of insulin and leads to metabolic alterations. Other studies have noted relationships between insulin resistance and active inflammatory connective tissue diseases, other clinical diseases, acute infections and periodontal disease.

The body of literature concerning the relationship between periodontal infection and impaired glycemic control is varied in the strength, quantity, breadth and consistency of evidence presented. The preliminary evidence, while encouraging, does not support a clear-cut conclusion that treating periodontal infection can contribute to management of glycemic control in type 1 or type 2 diabetes. Only studies using systemic antibiotic treatment affected glycemic control favorably. The results suggest that infections other than periodontitis may be implicated or that intensive treatment of periodontal infections with systemic antibiotics is necessary to affect glycemic control favorably. Further rigorous controlled studies in diverse populations are warranted.

The oral infection, heart disease and stroke connection

During the past decade, infectious agents have become recognized as causes of systemic diseases, without fever or other traditional signs of infection. Helicobacter pylori is associated with peptic ulcers and, along with Chlamydia pneumoniae and cytomegalovirus, is now thought to be associated with increased risk for cardiovascular disease as well as malignancies (Wu et al. 2000). Studies investigating the relationship between oral and dental infections and the risk for cardiovascular disease suggest that there is potential for oral microorganisms, such as periodontopathic bacteria, and their effects to be linked with heart disease.

Mechanisms of action

Infectious agents are thought to affect the risk of heart disease through several possible mechanisms. Bacteria or viruses originating in tissues such as the lungs or oral mucosa may directly infect blood vessel walls. Such infection may be largely asymptomatic, but may cause local vascular inflammation and injury, which would contribute to the development of lipid-rich plaques and atherosclerosis. Bacteria or viruses may also interact with white blood cells or platelets, both of which integrate into the developing atherosclerotic plaque. Cells of the blood vessel wall and white blood cells and platelets can release prostaglandins (especially PGE2), interleukins (especially IL-1), thromboxane B2 (TBX2) and tumor necrosis factor alpha (TNF-alpha). Bacterial products in the blood may also stimulate liver production of other pro-inflammatory or pro-coagulant molecules such as C-reactive protein and fibrinogen.

Microbes may also stimulate expression of tissue factor, which would activate coagulation. During the process of coagulation, platelets would become trapped in the growing clot or thrombus. Microthrombus formation is one of the key factors in the development of atherosclerotic plaques. As atherosclerotic plaques enlarge, the lumen of the coronary blood vessels narrows and the blood supply to the heart muscle becomes reduced. A frank heart attack or myocardial infarction results when a larger part of the coronary artery lumen becomes occluded. Failing to receive enough blood, the heart muscle dies, resulting in an infarct.

- The oral cavity is a portal of entry as well as the site of disease for microbial infections that affect general health status.
- The oral cavity and its functions can be adversely affected by many pharmaceuticals and other therapies commonly used in treating systemic conditions.
- The oral complications of these therapies can compromise patient compliance with treatment. Individuals such as immunocompromised and hospitalized patients are at greater risk for general morbidity due to oral infections.
- Individuals with diabetes are at greater risk for periodontal diseases.
- Animal and population-based studies have demonstrated an association between periodontal diseases and diabetes, cardiovascular disease, stroke and adverse pregnancy outcomes. Further research is needed to determine the extent to which these associations are causal or coincidental.

Prevention and control of dental caries

Although many caries prevention strategies, notably community water fluoridation and use of a fluoride-containing dentifrice, benefit adults and children alike, most of our understanding of the effectiveness of these strategies comes from the study of children, during a life stage when caries incidence is high. Caries prevention programs have been designed and evaluated for children and have used a variety of fluoride and dental sealant strategies applied separately and together. Because these strategies are complementary, their use in combination has the potential of virtually eliminating dental caries in all children.

However, dental caries is a problem for all ages. Although direct evidence of caries preventive strategies in adults is limited, the evidence that does exist is consistent with expected effects based on experience with children. The Centers for Disease Control and Prevention (CDC) recently convened an expert work group to develop recommendations for modalities to prevent and control dental caries based on a review of publications selected by the work group and other experts.

Fluoride

Fluoride reduces the incidence of dental caries and slows or reverses the progression of existing lesions (i.e., helps prevent cavities). Today, all Americans are exposed to fluoride to some degree, and there is little doubt that widespread use of fluoride has been a major factor in the overall decline in recent decades in the prevalence and severity of dental caries in the United States and other economically developed countries. Fluoride is the ionic form of the element fluorine, the 13th most abundant element in the crust of the Earth. Because of its high affinity for calcium, fluoride is mainly associated with calcified tissues (i.e., bones and teeth). The ability of fluoride to inhibit, and even reverse, the initiation and progression of dental caries is well known. Fluoride's mechanisms of action include incorporation of fluoride into enamel pre-eruptively, inhibition of demineralization, enhancement of remineralization and inhibition of bacterial activity in dental plaque.

A variety of theories regarding fluoride's mechanisms of action account for the range of fluoride products available. The initial theory of action was based on the belief that incorporation of fluoride into the hydroxyapatite of developing tooth enamel in the pre-eruptive phase reduced the mineral's solubility, thereby increasing enamel resistance. Because of the length of time the tooth is at risk of caries during the post-eruptive phase, however, the topical effects of fluoride are considered to predominate. These effects are based on fluoride's role in the aqueous phase around the tooth, both in saliva and in dental biofilm (plaque). Fluoride in plaque contributes to the remineralization of demineralized enamel when bound fluoride is released during an acid challenge, resulting in a more acid-resistant enamel surface structure. Fluoride also has been shown to inhibit the process of glycolysis by which fermentable carbohydrates are metabolized by cariogenic bacteria to produce acid. All these effects occur after the tooth erupts, while it is functioning in the mouth, enabling fluoride to prevent caries over a lifetime in both children and adults.

The first use of fluoride for caries prevention was in 1945 in the United States and Canada, when the fluoride concentration was adjusted in the drinking water supplying four communities. This public health approach followed a long period of epidemiologic studies of the effects of naturally occurring fluoride in drinking water.

The success of the community water fluoridation trials in reducing dental caries led to the development of other important fluoride-containing products, such as dietary supplements and, most notably, fluoride-containing dentifrices, in the early 1960s. Fluoride-containing gels, solutions, pastes and varnishes were also developed for topical use, either applied by professionals or self-applied at home or in other settings. All of these products were tested for safety and effectiveness in reducing caries. Products designed for professional use generally

have higher concentrations and are used at less frequent intervals than those designed for self-application.

Controlled clinical trials from the 1940s through the 1970s documented the benefits of professionally applied fluoride in reducing dental caries, and several excellent reviews are available. Professional application of fluoride is inherently more expensive than self-applied methods, so the use of such an approach for groups and individuals at low risk of dental caries is unlikely to be cost-effective. For patients at high risk of dental caries, however, professionally applied fluoride is still considered cost-effective. It is not clear whether fluoride varnishes and gels would be most efficiently used in clinical programs targeting groups at high risk of dental caries or whether they should be reserved for individual high-risk patients.

The U.S. Preventive Services Task Force and the Canadian Task Force on Periodic Health Examination affirm that there is strong evidence to support the major methods for providing fluoride to prevent dental caries.

The safety of fluoride is well documented and has been reviewed comprehensively by several scientific and public health organizations (Institute of Medicine (IOM) 1997, National Research Council (NRC) 1993, Newbrun 1996, U.S. Department of Health and Human Services (USDHHS) 1991, World Health Organization (WHO) 1984). When used appropriately, fluoride has been demonstrated to be both safe and effective in preventing and controlling dental caries. The IOM (1997) classified fluoride as a micronutrient, citing it, along with calcium, phosphorus, magnesium and vitamin D, as an important constituent in maintaining health.

Appropriate use of fluoride products can minimize the potential for enamel fluorosis, a broad term applied to certain visually detectable changes in the opacity of tooth enamel associated with areas of fluoride-related developmental hypomineralization. There are also many developmental changes in enamel that are not fluoride-related. Most enamel fluorosis seen today is of the mildest form, which affects neither aesthetics nor dental function. Cosmetically objectionable enamel fluorosis can occur when young children ingest higher than optimal amounts of fluoride from any source while tooth enamel is forming (up to age 6). Its occurrence appears to be most strongly associated with the total cumulative fluoride intake during the period of enamel development, but the condition's severity depends on the dose, duration and timing of fluoride intake. Specific recommendations have been made to control fluoride intake by children during the years of tooth development.

Fluoridation of drinking water

For more than half a century, community water fluoridation has been the cornerstone of caries prevention in the United States; indeed, CDC has recognized water fluoridation as one of the great public health achievements of the 20th century. All water contains at least trace amounts of fluoride. Water fluoridation is the controlled addition of a fluoride compound to a public water supply to achieve a concentration optimal for dental caries prevention. In the 1940s, it was concluded that 1 ppm (part per million) fluoride was the optimal concentration for climates similar to that of the Chicago area; this concentration would significantly reduce the prevalence of dental caries with an acceptably low prevalence of enamel fluorosis.

Current U.S. Public Health Service (USPHS) recommendations for fluoride use include an optimally adjusted concentration of fluoride in drinking water ranging from 0.7 to 1.2 ppm, depending on the mean maximum daily air temperature of the area. A lower fluoride concentration is recommended for communities in warmer climates than cooler climates because it is assumed that persons living in warmer climates drink more tap water.

Effectiveness

Numerous studies in naturally fluoridated areas preceded the field trials. There are no randomized, double-blind, controlled trials of water fluoridation because its community-wide nature does not permit randomization of people to study and control groups. Similar results have been derived from numerous well-conducted field studies by various investigators on thousands of subjects in different parts of the world. Conducting a study in which individuals are randomized to receive or not receive fluoridated water is unnecessary and is not feasible.

In 1945, Grand Rapids, Mich., became the first city in the United States to fluoridate its water supply; the oral health of its schoolchildren was periodically compared with that of schoolchildren in the control city, Muskegon, Mich. Dramatic declines in dental caries among children in Grand Rapids and three other cities conducting studies shortly thereafter led to fluoridation in many other cities. In an extensive review of 95 studies conducted between 1945 and 1978 reported the modal caries reduction following water fluoridation to be between 40 and 50 percent for primary teeth and 50 and 60 percent for permanent teeth. Fluoridation also benefits middle-aged and older adults. Benefits to adults include reductions in both coronal and root caries.

These benefits are important because older people typically experience gingival recession, which results in exposed root surfaces, which are susceptible to caries. In addition, tooth retention in older U.S. cohorts has increased in recent decades, so that the number of teeth at risk for caries in older age groups is also increasing.

Finally, many medications used to treat chronic diseases common in aging have the side effect of diminished salivary flow, depriving teeth of the many protective factors in saliva. Other evidence of the benefits of fluoridation comes from studies of populations where fluoridation has ceased. Examples in the United States, Germany and Scotland have shown that when fluoridation is withdrawn and there are few other fluoride exposures, the prevalence of caries increases. In Wick, Scotland, which began water fluoridation in 1969 but stopped it in 1979, the caries prevalence in 5- to 6-year-olds with limited exposure to other sources of fluoride increased by 27 percent between 1979 and 1984. This was despite a national decline in caries and increased availability of fluoride-containing dentifrices.

Fluoride mouth rinses

Several different formulations of fluoride mouthrinses are available, differing in the amount of fluoride and suggested frequency of use. Rinses with low fluoride concentrations (0.05 percent neutral sodium fluoride or 0.1 percent stannous fluoride) are designed for daily use and are available over-the-counter. Higher-concentration rinses (0.2 percent sodium fluoride) are designed for weekly use and are available only by prescription or in public programs.

Dental sealants

The pits and fissures that characterize the biting surfaces of posterior teeth provide a haven for food debris and decay-causing bacteria. Not surprisingly, these sites are often the first and most frequent to be affected by decay in children and adolescents. The width of most pits and fissures is narrower than a single toothbrush bristle, making cleaning of their deepest recesses almost impossible. According to national estimates, as much as 90 percent of all dental caries in schoolchildren occurs in pits and fissures. The teeth at highest risk by far are permanent first and second molars.

Enamel bonding, a technology introduced in the mid-1950s, led to the development of sealants. These are clear or opaque plastic resinous materials designed for professional application to the pit-and-fissure surfaces of teeth. The material hardens within 60 seconds or so into a thin, hard, protective coating. Sealants were introduced in the late 1960s and received the American Dental Association Seal of Approval in 1976. Most of the dozen products approved by the ADA do not contain a therapeutic agent, but work by providing a physical barrier that prevents microorganisms and food particles from collecting in the pits and fissures. First-generation sealants used ultraviolet light to harden or "cure" the material; improved second- and third-generation sealants cure by chemical or visible light activation, respectively.

Sealant placement requires meticulous attention to technique, but they can be successfully provided in "field" settings using portable dental equipment. To be most effective, sealants should be placed on teeth soon after they erupt, but they can be applied across a wide age range. Not only does the risk for caries continue across the life span, but an individual's risk also can increase for any number of reasons. Sealants are particularly helpful for persons with medical conditions associated with higher caries rates, children who have experienced extensive caries in their primary teeth, and children who already have incipient caries in a permanent molar tooth.

Efficacy

Initial clinical trials using a random half-mouth design and first- or second-generation sealant materials established their efficacy. Several comprehensive reviews and a meta-analysis of the amount of caries prevented testify to the utility of these materials a systematic process to select and review studies of one-time sealant placement on permanent teeth in subjects unexposed to other preventive measures. Pooled results from 17 studies meeting their selection criteria found that second-generation sealants reduced caries over 70 percent.

These early trials firmly established retention as essential to preventing caries; a sealant is virtually 100 percent effective if it is fully retained on the tooth.

Effectiveness

Administrators of school-linked sealant programs and of school-based programs with either fixed clinics or portable equipment reported on their experiences with these programs. These studies, using second-generation sealants, have shown effectiveness results comparable to those of clinical trials, regardless of the physical delivery site or personnel used for sealant application. Complete retention after approximately one year varied from 83 to 94 percent.

A Consensus Development Conference sponsored by the National Institutes of Health concluded that "an extensive body of knowledge has firmly established the scientific basis for the use of sealants." The panel urged the development of educational materials to enhance public and professional acceptance as well as third-party reimbursement. Consensus on the value of sealants is reflected by the inclusion of sealant objectives in Healthy People 2000 and Healthy People 2010. In addition, sealant placement is supported in federally funded programs for women and children, and sealants are covered services in all state Medicaid programs. A workshop on guidelines for sealant use has made recommendations for sealant use in both community and individual care programs.

Community dental sealant programs

Several community-based public health initiatives have arisen to promote sealant use among private practitioners and through community-based programs. These activities include reaching dentists through continuing education courses; directing large-scale promotional activities to consumers, community leaders and third-party payers; and providing sealants directly to children in school programs.

Prevention and control of periodontal diseases

Periodontal diseases, caused by specific bacteria in dental plaque, affect most adults at some point in their lives. The mildest and most common form of periodontal disease is gingivitis. Over time, periodontitis, the more severe form of periodontal disease, can lead to the destruction of the soft tissue and bone that anchor the teeth into the jaw. Lacking support, teeth can loosen and be lost. Periodontal diseases can be prevented and controlled through an array of mechanical and chemical means. Conscientious oral hygiene and professional oral cleanings to reduce plaque can reverse gingivitis. Methods for personal oral hygiene include toothbrushing and flossing, which may be augmented by over-the-counter and prescription mouthrinses with antimicrobial action.

Community programs to prevent gingivitis

With the confirmation of specific bacteria in dental plaque as the cause of gingivitis, public health officials began to seek ways to educate the public about plaque control in community settings, primarily in schools. These efforts have had equivocal results. Although knowledge and attitudes were enhanced in demonstration programs, improvements in plaque levels and gingivitis were short-lived in clinical trials.

Prevention of periodontitis

Tobacco use is a major risk factor for the development and progression of periodontal diseases, and proven strategies aimed at reducing tobacco use should aid in the prevention of periodontitis. The following section on oral and pharyngeal cancers includes a discussion of such intervention strategies. Until recently, most interest in controlling tobacco use reflected concerns about oral cancers. As appreciation of the role of tobacco in the progression of periodontal diseases and tooth loss increases, attention to these oral health effects may increase attention to tobacco cessation in primary oral health care. Periodontitis can also be a complication of poorly controlled diabetes.

Some efforts have been directed at alerting dental practitioners to the need to educate patients about diseases affecting the periodontal tissues. These efforts have met with some success, but they tend to reach only those people who already use dental services. Currently, there are no broad community-based intervention programs that address periodontal diseases.

Gingivitis can be controlled with available methods, and its control is the principal way to prevent periodontitis. However, the currently available methods are individually or professionally based and require conscientious oral hygiene practices and regular dental visits. Although some schools instruct children in proper methods of oral hygiene, no community methods, other than programs designed to discourage tobacco use, are available for preventing gingivitis or periodontitis in the general population.

Prevention and control of craniofacial birth defects

The causes of craniofacial birth defects are often complex and multifactorial, the result of gene-environment interactions occurring from the time of conception to birth. Even when a mutation in a single gene has been discovered as the cause of a particular syndrome, there can be considerable variation in susceptibility, with some infants showing little or no sign of a problem and others experiencing multiple organ defects.

The work to complete the mapping and sequencing of the human genome will undoubtedly shed light on the hundreds of genes involved in craniofacial development and provide details on when and how they function in development. This knowledge may in turn lead to gene therapies that restore or "rescue" the function of a defective gene and thus prevent the anomaly.

Craniofacial defects also may occur because the susceptible embryo or fetus was exposed to an environmental teratogen, a diminished oxygen supply or a deficit in an essential nutrient. An association may exist between low-birth-weight, premature babies who may show other subtle craniofacial anomalies and mothers with chronic oral infectious disease.

In addition, diets poor in folic acid increase the risk of spina bifida and possibly clefting syndromes. Clinical trials using vitamin supplementation with varying levels of folic acid are under development to determine whether they can lower the risk of clefts in high-risk pregnancies. Outcomes of clinical trials of nutrient supplementation in pregnancy may lead to new nutritional guidelines and the development of enriched food products, which can form the basis for community-wide health promotion and disease prevention programs.

Given the array of variables affecting prenatal growth and development, the key to public health programs aimed at preventing birth defects lies primarily in health promotion and education campaigns. Individuals need to be made aware of known risk and protective factors in pregnancy. Such programs should emphasize the importance of good nutrition, avoidance of tobacco and alcohol use, and prenatal care. Education includes knowledge about the teratogenic effects of prescription drugs, such as the antiepileptic drug phenytoin and the retinoic acid drugs used to treat cystic acne.

As information from developmental biology, genetics and epidemiologic and clinical studies accrues, dental care providers are better positioned to provide counseling. The public is best served by health promotion and disease prevention campaigns that communicate findings about risk and protective factors in pregnancy.

Prevention and control of intentional and unintentional injury

Intentional and unintentional injuries are related to behaviors and are thus amenable to prevention. As studies of motor vehicle and sports injuries have demonstrated, injuries are frequently due to a sequence of predictable events, and a public health approach can be successful in injury prevention and control.

The interventions that have proved to be most effective in controlling injuries have been passive; that is, they do not require the individual to participate. Examples include the use of environmental controls such as vehicle and roadway design, speed limits, passenger restraints and airbags to prevent injuries from motor vehicle collisions. Passive measures such as these are more easily implemented at the state or federal level. However, many preventive measures for oral-facial injuries have been directed at the individual and professional health service levels, rather than at the population at large.

Craniofacial injuries

The principal causes of craniofacial injuries are motor vehicle collisions, falls, assaults and sporting activities. Except in relation to sports, injuries to the craniofacial region have received little attention. These injuries are hardly insignificant, however, and efforts to prevent them are gaining acceptance. For example, to increase public awareness of the importance of facial protection, the inaugural National Facial Protection Month was celebrated in April 2000. This national campaign, providing information to the media and the public, was sponsored by the American Association of Oral and Maxillofacial Surgeons (AAOMS 2000).

Motor vehicle collisions are the leading cause of death during the first three decades of life in the United States and the leading cause of death from injury over most of the life span. Data from multiple sources indicate that craniofacial injuries account for a substantial subset of these injuries annually. Even though it is likely that passive measures enacted to reduce fatalities have reduced nonfatal craniofacial injuries, no supporting data exist.

Various sources report the number of motorcycle- and pedal-cycle-related craniofacial injuries. Data from the National Electronic Injury Surveillance System indicate that head injuries account for 50 percent of all pedal-cycle-related injuries; of those, bicycle-related events accounted for 19 percent of all facial injuries within the study period. In similar studies, tricycle-related incidents were found to be responsible for up to 61 percent of injuries to the head, face or mouth. Motorcycle injuries are a major source of fatal and nonfatal head trauma in the United States.

Helmet use reduces head and facial injuries among bicyclists and motorcyclists by up to 50 percent. Health promotion efforts have increased acceptance at the community level for helmet use by bicyclists; however, helmet use regulations vary by state and with the public whim. Over a dozen states currently have bicycle helmet laws, and half of the states have motorcycle helmet laws.

Many authors have described craniofacial injuries related to sports. Information is usually obtained from community or regional surveys of injuries or mouthguard use and effectiveness. Craniofacial injuries sustained during sporting activities are a major source of nonfatal injury and disability, possibly accounting for up to one third of all sports injuries. The increasing participation of women in competitive sports means that young women should be alerted to the risks and advised of the need for additional protective gear as appropriate.

Health professionals are in an ideal position to provide up-to-date health information and care to their patients. They also have an opportunity to enhance their knowledge and practices as well as increase their communication to patients about the procedures they provide and the reasons for these procedures.

Daily hygiene and dental caries prevention

The use of a fluoride-containing dentifrice is critical for dental caries prevention. Even more beneficial than the physical removal of plaque in toothbrushing is the delivery of a small amount of fluoride to the tooth surfaces. Investigators have conducted numerous clinical trials on fluoride dentifrices using rigorous designs and including randomized groups, double-blind designs and placebo controls. All together, these studies provide strong evidence that using a fluoride dentifrice is effective. Fluoride dentifrices account for more than 90 percent of the market in the United States, Canada and other developed countries.

A fluoride dentifrice is an effective means of reducing the prevalence of dental caries for all persons. Although children's teeth should be cleaned daily from the time they erupt, parents and caregivers should consult a dentist or other health care provider about the use of a fluoride dentifrice for children under the age of 2. For children under 6, fluoride dentifrices should be used in small amounts to minimize swallowing of the product. Mild enamel fluorosis can result from excessive dentifrice use, because children under 6 do not have adequate control of the swallowing reflex or may intentionally swallow a flavored dentifrice. Experts recommend that for children under 6, the parent or caregiver should supervise toothbrushing, apply a pea-sized amount (0.25 gram) of dentifrice to the toothbrush, and encourage the child to spit out the excess.

Because the topical benefits of fluoride have been shown to be highly effective and daily exposure to small amounts of fluoride can reduce the risk of dental caries in all age groups, experts recommend that all persons drink water with an optimal fluoride concentration in addition to brushing daily with a fluoride dentifrice. This combination provides a cost-effective and easy way to prevent dental caries and is an excellent example of the individual-community partnership. For persons at low risk of dental caries, these two exposures to fluoride may be the only ones necessary. For persons at moderate or high risk of dental caries, additional fluoride may be helpful and can come from daily use of another fluoride product. These can include mouth rinses, prescribed supplements and professionally applied topical fluoride products.

Daily hygiene and the prevention of periodontal diseases

Toothbrushing and flossing also play a critical role in the prevention of periodontal diseases. Unlike dental caries prevention, prevention and control of gingivitis and periodontitis are achieved directly through the mechanical removal and disruption of dental plaque. Some dentifrices also contain chemical therapeutics to control the formation of tartar (calculus) and to reduce plaque formation and gingival inflammation. Both manual and electric toothbrushing are effective at removing plaque and preventing gingivitis.

Interproximal (between the teeth) cleaning is also important in maintaining gingival health. In one short-term evaluation of adults, the addition of flossing to the daily regimen of brushing resulted in an almost twofold reduction in gingival inflammation.

Because preventive measures in periodontics rely mainly on the removal of bacterial plaque and calculus, methods typically include personal oral hygiene measures combined with professional diagnostic and prophylactic measures (i.e., regular exam and cleaning). Periodic professional care for removal of plaque and calculus deposits has also been demonstrated to improve the periodontal health of participants.

Healthy lifestyles

There is more to the individual's role in promoting oral health and hygiene than brushing and flossing. Other behaviors that have an influence on oral health include use of tobacco and/or alcohol products, diet, oral habits such as bruxing and clenching the teeth, and use of helmets, mouthguards or other protective devices.

Individuals can obtain credible information regarding oral health from various sources, including health care providers, professional organizations, government agencies and patient advocacy groups. Increasingly, the World Wide Web is a source for health care information. For example, the National Oral Health Information Clearinghouse offers information on oral health, with an emphasis on special-care patients and their health care providers.

Provider-based care

The range of conditions and diseases that affect the craniofacial complex is extensive and can provide clinicians with important indications about the patient's general as well as oral health status. Management of the oral health, general health interface calls for interdisciplinary and coordinated care and an enhanced role for primary care providers. Dentists, oncologists, dermatologists, infectious disease specialists, hematologists, endocrinologists, plastic surgeons and rheumatologists are just a few of the specialists who may be involved in the diagnosis and management of conditions affecting the craniofacial complex.

Dentists, their allied staff and medical and nursing personnel are in a unique position to incorporate new approaches for prevention, diagnostic and treatment strategies in their practices. Advances in oral science are providing the basis for a shift in emphasis from the repair and restoration of damaged tissues to earlier diagnoses, control of infections and remineralization and regeneration of lost tissues. The application of risk-assessment strategies and interventions tailored to individuals and groups is expanding with the increased understanding of risk factors and the development of biomarkers that signal host resistance, susceptibility, and the presence and progression of disease.

The changing demographics of the U.S. population and a greater understanding of the relationship between oral health and general health are presenting new challenges. Making clinical decisions for patients requires integrating a range of interacting biological, psychological, social, cultural and environmental factors. In order for disease to manifest, the etiologic agent(s) must be present, the host must be susceptible, the environment conducive and sufficient time available for the factors to interact.

Early diagnosis and prompt treatment require an understanding of the pathology and of the diagnostic, prevention and treatment modalities available. As genetic information accumulates, clinical judgments will increasingly be informed by knowledge of an individual's genetic

susceptibility or resistance to particular diseases and disorders. The development of tailored treatment plans will require incorporating all these factors together with input from the patient's health providers, taking into consideration the patient's interests and needs.

Changing approaches to selected diseases and conditions

The science and technology base is providing new approaches to risk assessment, diagnosis, prevention and treatment. Highlights of selected diseases and conditions follow.

Dental caries

Dental caries is caused by a transmissible microbial infection that affects tooth mineral. A number of factors play a role in the initiation and progression of the disease, including bacterial biofilm, specifically the presence of mutans streptococci and species of lactobacilli; the frequency of simple sugars in the diet; the flow and composition of saliva; the availability of fluoride; the structure of tooth mineral in a given individual; and oral hygiene behaviors. Sound caries management takes all these factors into account. Today there is the prospect that clinicians will be able to balance protective and pathologic factors and work with the patient to control disease.

Risk assessment

Reviews of caries risk prediction models conclude that clinical variables, especially past caries experience, are the best predictors of new caries experience. At the time of initial tooth eruption, the presence of mutans streptococci appears to be the primary predictor of future caries. With continued tooth eruption, this variable disappears as a primary predictor and is replaced by the status of the most recently exposed or erupted tooth surface. For example, the presence of carious lesions in the primary incisors has been found to be the best predictor of caries in the later-erupting primary molars.

Despite recent declines, dental caries is a prevalent disease, with some age and population groups particularly vulnerable. A guide for the identification of vulnerable patients and the treatment of caries as an infectious disease developed by the American Dental Association proposes questions to be considered at an initial examination. These questions, together with information gathered at recall examinations, allow classification of child and adult patients into high-, moderate- and low-risk disease categories. This approach has been incorporated in a variety of caries risk assessment forms adopted by some dental schools and managed care programs. Studies are needed to determine the validity and reliability of such approaches for different patient populations and practice settings.

The use of tests to assess caries risk to determine the activity status of preclinical disease is becoming more widespread. A range of diagnostic aids for caries activity testing are available. Microbial tests can detect the presence and quantify the levels of lactobacilli and mutans streptococci. The development and use of these tests are based on studies that have associated these microbes individually and together with different types of carious lesion development. Measurements of plaque and salivary pH have been used to evaluate the oral environment overall and to note the changes in pH that occur after eating various foods. Salivary flow and composition analyses add another dimension. Decreased flow has been related to caries susceptibility, as have increases in viscosity. These factors warrant further study to determine their sensitivity and specificity.

Diagnosis

Clinical signs, patient-derived history and radiographic images remain the primary means of dental caries diagnosis. Tooth surface pitting and cavitation, white and/or brown spots, areas soft to tactile probing and radiolucencies are used to detect the effects of this disease. The most common diagnostic approaches include visual inspection, the use of an explorer (a probelike instrument) to determine the integrity of the tooth surface, the use of a light source to detect difference in reflectance across tooth structure (transillumination) and radiographs.

The most basic diagnostic methods – visual alone and visual examination with an explorer – have limited sensitivity but excellent specificity. The visual examination may be combined with a radiographic series for the initial assessment. Bite-wing radiographs are frequently used to diagnose interproximal caries (between teeth) and for these surfaces provide excellent sensitivity and specificity. Radiographic examination allows examination of otherwise inaccessible areas. Specifically, the depth of a lesion and its relationship to the pulp chamber can be evaluated for interproximal lesions. However, radiographs are of little value in detecting caries on the occlusal surfaces of the teeth. For these surfaces, a negative radiographic diagnosis does not imply lack of a carious lesion in enamel.

Precavitated carious lesions and caries in restored teeth pose an additional diagnostic challenge. A review of the literature on the clinical diagnosis of precavitated carious lesions concluded that visual detection of these lesions has low sensitivity and moderate specificity. It is difficult with these lesions to determine whether there are no caries or whether only the enamel or outer layer of dentin is involved. Carious lesions forming around restorations are seen more frequently at the approximal and cervical margins of these restorations. Distinctive color changes around a restoration alone are not diagnostic of active caries. Currently, the progression of carious lesions is the most definitive diagnostic parameter for disease activity. Progression can be determined over specific time intervals only by professional assessment.

Prevention

The primary prevention of dental caries starts with adequate prenatal and perinatal nutrition to ensure normal development of the teeth and supporting structures. It continues with interventions aimed at preventing transmission of cariogenic microbes from caregivers to infants, and proceeds with specific strategies employed across the life span. These approaches include the provision of sufficient fluoride, the use of dental sealants, the adoption of healthy behaviors, including avoiding unhealthy dietary practices and practicing appropriate oral hygiene, and the timely use of care services. Although many factors are brought to bear on the primary prevention of dental caries, the combination of fluoride in its multiple forms and dental sealants is the foundation.

Fluoride is available in a variety of products that can be used by health professionals, individuals and public programs. Topical solutions and gels, mouth rinses and dentifrices are available for daily, weekly or as-prescribed frequency. In addition, fluoride-containing prophylactic pastes are available for professional application. Clinical judgment of risk factors determines the type and frequency of interventions

needed. Although there is general agreement on the overall value of topical fluorides in reducing dental caries, comparative clinical trials are needed to determine which of the existing fluoride formulations (acidulated phosphate fluoride, stannous fluoride, amino-fluoride or sodium fluoride) and which delivery system (gel, varnish, dentifrice or solution) are most efficacious.

A second line of defense is through control of the etiologic agent. Chemotherapeutic agents (including the antimicrobial mouthrinse agent chlorhexidine and fluoride) can be used to reduce plaque. Dietary measures aimed at reducing the frequency and quantity of sugars and the substitution of sugars by sugar-free sweeteners may effectively starve the bacteria.

The process of tooth demineralization and re-mineralization has received significant attention over the past four decades. Investigators are studying the effectiveness of therapeutic agents for arresting carious lesions and remineralizing enamel in populations at high risk for dental caries. For example, a combined chlorhexidine-fluoride solution can enhance remineralization of incipient lesions and arrest caries in patients who suffer from radiation-induced caries. The use of a twice-daily rinse with 0.05 percent sodium fluoride to prevent demineralization and induce remineralization in subjects with radiation-induced hyposalivation has also been found to be effective.

This study also addressed the effects of chlorhexidine use alone, which has been associated with tooth staining, alterations in taste and potential hypersensitivity reactions. It showed that the application of 40 percent by weight chlorhexidine varnish every three months enhanced remineralization of root caries more than fluoride varnish, although both treatments were associated with fewer filled root surfaces than the control group after one year. A chlorhexidine varnish has not yet been approved in the United States, and large-scale, double-blind, placebo-controlled clinical trials are not yet available to test the effects of specific regimens in relation to caries risk.

Studies also are evaluating interventions to prevent mutans streptococci transmission. Findings from cross-sectional studies indicate that infants are initially infected by their parents, specifically mothers, around the time the teeth erupt. A longitudinal study using DNA fingerprinting demonstrated that mothers were the source of the bacteria in their infants and the degree of matching to maternal strains was higher for female infants than for males.

Based on a study of child-mother pairs (with the child initially at 1 year of age), the application of a 1.0 percent chlorhexidine rinse alternated with a 0.2 percent sodium fluoride gel to the mother's teeth (three times per day on two consecutive days, twice per year for three years) delayed, and in some cases prevented, the colonization of their children's teeth by mutans streptococci. Timing of colonization has been shown to be correlated with caries prevalence. In a longitudinal study that followed children in four-month intervals from 15 months to 4 years of age, children who were infected earlier had a higher caries prevalence than those in whom the infection was detected at later ages. Studies also have been aimed at reducing the levels of cariogenic bacteria in the infants themselves.

Work continues on the development of a caries vaccine. One approach focuses on the production and release of antibodies against cariogenic bacteria antigens. Specific antigens have been purified and synthesized. Another approach involves biological replacement therapy, where nonpathogenic bacteria instilled in the mouth prevent pathogenic bacteria from colonizing. Yet another approach employs passive immunization in which antibodies, produced outside the body (in cultures, animals, eggs, or plants), are applied to the teeth and oral tissues to protect against disease. A recent study indicated that "plantibodies" painted on the teeth could prevent mutans streptococci colonization for 120 days, the period of the experiment.

Treatment

Prompt treatment of early carious lesions permits the preservation of tooth structure through conservative approaches. A 10-year study reported that caries did not progress under a dental sealant placed over cavitated lesions where the lesions were no more than halfway through the dentin.

Materials that can bond to enamel and to dentin continue to be refined and improved. Glass ionomer cements have contributed to materials that can bond to enamel and dentin, release fluoride and increase remineralization in adjacent teeth. These cements, together with polymeric resin composites and hybrids of these two materials, are now available for tooth restoration with other materials. Based on the available materials and emerging techniques, such as air abrasion and laser ablation, restoration procedures are more conservative than ever before.

A proposed categorization of carious lesions for the purpose of conservative management places lesions into three categories: lesions where no treatment is advised, lesions where preventive care is advised and lesions where restorative treatment is advised. This approach, using caries as an infectious disease paradigm, resulted in a marked reduction of operative procedures in Danish schoolchildren and has been proposed as a means to preserve tooth structure and maximize appropriate care in the United States.

New imaging and laser technologies are emerging as tools for early diagnosis and prompt treatment of dental caries. For example, quantitative light-induced fluorescence is showing promise for dental caries diagnosis. Two different methods, the quantitative infrared laser fluorescence method and electrical conductance measurements, are currently commercially available. At present, these methods are being used to augment conventional diagnostic tools but are not yet part of routine practice. However, they could potentially be used for close monitoring of the lesions and for patient motivation. Laser treatments for soft tissue surgery have been used in dentistry in recent years. Currently, in vitro studies are under way for the application of lasers for hard tissues, specifically to prevent dental caries by altering tooth mineral and inhibiting progression of artificial caries-like lesions.

Despite the best efforts of the individual and health care provider, caries may progress. Advances in materials science over the last two decades have fortunately led to major improvements in dental restorative materials, resulting in a wide range of aesthetically pleasing, longer-lasting restorations that can be placed with less trauma. Traditional materials such as amalgam fillings and gold crowns are now augmented by aesthetic materials, including bonded composite resins, porcelain fused to metal crowns and facings.

When teeth have been lost, the options for rehabilitation include a range of prosthetic devices. Removable full and partial dentures and fixed bridges provide aesthetic and serviceable restorations for many patients. Still another option is the use of dental implants. These are used not only in patients who have lost teeth due to caries and periodontal diseases, but also to restore form and function in patients treated for trauma, craniofacial cancers, hereditary tooth defects and other abnormalities.

The evidence base for the survival of the endosseous dental implants, an implant that is placed directly into a tooth socket, is extensive and has been recently reviewed. The predictability of endosseous dental implants in fully and partially edentulous patients has been clearly demonstrated in longitudinal studies. Many implant designs and surfaces have shown high success rates (often exceeding 95 percent in good-quality bone and 85 percent in poorer-quality bone, such as the posterior maxilla).

Rehabilitation of lost tooth structure or even the whole tooth itself may be revolutionized in the next century, based on discoveries of the natural repair and regeneration mechanisms the body uses. The new sciences of biomimetics and tissue engineering combine engineering principles and materials science with rapidly growing knowledge of the progenitor cells and molecules that give rise to specific tissues such as skin, bone, teeth and cartilage. Already it is possible to generate new cartilage and bone of a prescribed shape to replace tissue lost from injury or disease. Eventually, it may be possible to use a patient's own oral cells and cell products to generate new tooth enamel, dentin and cementum for the natural repair of carious lesions.

Periodontal diseases

Periodontal diseases are caused by microbial infections, and are plaque-related complex diseases like dental caries, presenting as several clinical variants. The mildest form is gingivitis, characterized by inflammation of the gingiva with a marked loss of gingival collagenous material. In a more advanced disease, periodontitis, there is involvement of the soft tissue and bone that support the teeth. If untreated, periodontitis may progress and result in abscesses, mobile teeth and tooth loss. Periodontitis also may be associated with certain systemic diseases and conditions.

Gram-negative anaerobic bacteria in plaque are implicated as causative agents in periodontitis. However, host immune system factors, specifically, a chronic inflammatory response, are now considered to be the primary determinants of disease progression and outcome. The disease process is very similar across the different types of periodontal disease and involves interactions between infectious agents and their virulence factors and host defense mechanisms, operating within a context of environmental, acquired and genetic risk factors specific to a given individual. *S*ufficient knowledge of demographic and systemic risk factors and indicators has been acquired to guide clinical decisions in the management of periodontal diseases. The presence of pathogenic bacteria, poor oral hygiene, tobacco smoking, diabetes mellitus and pre-existing periodontal disease are some of the factors that contribute to the likelihood of disease presence, progression and treatment outcomes.

A systematic identification of risk factors, indicators and predictors has been proposed as the first step in diagnosing and managing periodontal diseases. Clinicians can weigh the known risks for individual patients and devise treatment plans appropriate to their risk category. These same factors and the outcomes of treatment can also be used to assess prognosis upon completion of therapy. Studies are under way to determine the feasibility and validity of assessing a complex of risk factors to predict states of periodontal health and disease.

Most recently, putative genetic markers for susceptibility for oral disease have been studied. In particular, a specific genotype of the polymorphic IL-1 gene cluster has been shown to be associated with severe periodontitis in nonsmokers. IL-1-beta is of interest because the proinflammatory cytokines are key regulators of the host immune response to microbial infection and extracellular matrix catabolism and bone resorption. Functionally, this polymorphism is associated with high levels of IL-1 production, and high levels of IL-1 have been associated with progressive periodontal breakdown.

A consensus has been reached by a specialty organization that all patients in general and specialty care should be screened for periodontal disease. The recommended approach is to apply the periodontal screening and recording examination (PSR). Related screening tests include the community periodontal index of treatment needs (CPITN) and the basic periodontal examination.

Diagnosis

Most diagnostic tests for periodontal diseases rely on a physical examination to note any swelling, redness, gingival bleeding or tooth mobility. Periodontal probing, radiographs and microbiologic and histological examinations of biopsied tissue provide important additional information. These tests indicate the presence, extent and severity of gingival and periodontal tissue destruction; they do not indicate the cause of disease or whether it is quiescent or actively progressing.

Gingival inflammation may be assessed using a variety of methods, including bleeding on probing and the use of indices such as the gingival index to grade redness and bleeding. In adult periodontitis, the absence of inflammation is associated with a lack of disease progression, but the presence of inflammation does not indicate inevitable progression to destruction. Longitudinal studies have also been conducted in patients who participate in maintenance programs.

The absence of gingival bleeding, especially at recall visits, has been shown to be a valid indicator of gingival health in these patients. Measurement of probing depths (also termed pocket depths) is an integral part of the periodontal examination. Longitudinal studies have shown that shallow probing depths and minimal loss of attachment are associated with lack of disease progression. The mere presence of a pocket does not herald progressive periodontitis at that site.

Although teeth with moderate to deep probing depths are at higher risk for additional destruction, a single examination cannot determine the fate of the tooth with certainty. Radiographs are used to obtain a visual image of the bony support around a tooth or dental implant. They are an essential tool in planning complexprosthetic reconstructions, as well as a necessary diagnostic aid in assessing periodontal progression.

Prevention

Because periodontal diseases are plaque-associated infections, prevention and management of the early signs of these diseases depend on effective plaque control. This can be accomplished using both mechanical and chemotherapeutic approaches. The prophylaxis performed in the dental office on periodontally healthy patients reduces plaque and removes stains and calculus. How often patients should be recalled for such preventive procedures is based on an assessment of risk factors, such as the patient's age, oral hygiene, personal habits (e.g., smoking and diet) and a medical history indicating a heightened risk of infection (such as noted with diabetes or HIV infection).

Chemical plaque control has become an important part of the clinician's armamentarium and may be prescribed for patient care at home. Significant reductions in gingival inflammation have been demonstrated for chlorhexidine, triclosan co-polymer when used in conjunction with a fixed combination of essential oils and stannous fluoride. The magnitude of gingival inflammation reduction was greatest for chlorhexidine. The evidence supporting these effects includes multiple randomized, double-blind controlled clinical trials.

Treatment

Once periodontal disease is established, the resultant bone and connective tissue loss may be quiescent or actively progressing. The goal of treatment is to determine whether the disease is active in order to prevent further tissue loss. This entails professional plaque removal and careful instruction of the patient on scrupulous self-care.

The concept of management of a patient's risk factors as part of treatment is reasonably well documented for individuals who smoke and those who are diabetic and may be important for other risk factors such as stress and low dietary calcium. Several studies have shown that treatment of periodontal disease in smokers is not as successful as in nonsmokers. Thus, the management of smoking as a risk factor will contribute to the success of periodontal therapy. Furthermore, it appears that treatment of diabetic patients with periodontal disease may require more intense therapy since several studies have shown that antibiotic therapy is successful not only in reducing periodontal disease, but also in reducing glycated hemoglobin.

Professional plaque removal typically employs scaling and root planing, in which hardened deposits of plaque and other debris are removed from the periodontal pocket and the tooth root surface is smoothed over. The effectiveness of scaling and root planing has been demonstrated repeatedly in longitudinal, cohort and randomized clinical trials and was reviewed by Cobb. Demonstrated benefits include decreased gingival inflammation, decreased probing depth and facilitation of maintenance of clinical attachment level. The evidence indicates that similar results may be obtained with ultrasonic and sonic instruments as with manual instruments. Regardless of the methods used, meticulous attention to detail is required to achieve optimal results.

Topical administration of antimicrobial agents contributes to the control of gingival inflammation. Supragingival irrigation (e.g., applying a jet of water under pressure) may be used as an adjunct to toothbrushing and has been shown to aid in the reduction of gingival inflammation. However, no clear substantial long-term benefits for the treatment of periodontitis have been shown if irrigation is applied subgingivally. Surgical therapy is employed to provide access to root surfaces and bony defects for debridement and root planing. Surgery can facilitate regeneration, augment the gingiva and promote root coverage. It is also necessary in placing dental implants.

Palcanis reviewed the evidence regarding surgical therapy. The overall goal is to make plaque control easier for the patient, thereby reducing disease progression. Many surgical techniques are available. Extensive randomized clinical trials and longitudinal studies form the basis of the evidence for the efficacy of these procedures. All procedures decrease pocket depth, and, with the exception of gingivectomy, all increase clinical attachment level. A caveat to be noted, however, is that procedures designed to reduce probing depth may increase gum recession, exposing the root and possibly compromising aesthetics. Thus, selection of a particular surgical procedure must always be based on the individual needs of the patient. Regardless of the approach selected, maintenance is important to long-term success.

Systemic administration of antibiotics, including the tetracyclines, 3 metronidazole, spiromycin and clindamycin, has been extensively studied and reviewed. The risk of generating antibiotic resistance in bacteria precludes the use of systemic agents in treating simple gingivitis. Similarly, systemic antibiotics should not be used for the routine first-line treatment of common forms of adult periodontitis. The preponderance of evidence from well-controlled, randomized, blinded clinical trials indicates that the agents do not offer sufficient benefit to overcome risks of either drug sensitivity or the emergence of antibiotic-resistant pathogens.

The situation is different in cases of aggressive forms of periodontitis, such as early-onset, rapidly progressive or refractory periodontitis, which affect less than 10 percent of periodontitis patients. Randomized, double-blind clinical trials as well as longitudinal assessments indicate that the use of systemic antibiotics can slow disease progression in these patients.

To circumvent the problems of systemic therapy, investigators have applied antimicrobial agents directly into the pocket. Antimicrobials incorporated into either resorbable and nonresorbable interpocket delivery systems have been studied in randomized, double-blind, controlled clinical trials and are now FDA-approved and on the market. When used as an adjunct to scaling and root planing, gains in clinical attachment level and decreases in probing depth and gingival bleeding were demonstrated. Because these delivery systems are relatively new, there is a paucity of evidence addressing their long-term effectiveness.

For patients who have lost significant bone and/or connective tissue, there are a number of regeneration procedures to facilitate the growth of new periodontal ligament, cementum and alveolar bone over previously diseased root surfaces. The evidence base for bone-grafting techniques using either natural or synthetic bone materials has been reviewed by Garrett (1996). Natural bone grafts may use autografts, in which bone is transferred from one site to another in the same patient; allografts, which use bone grafts from a human donor; and xenografts, which use tissues from other species. Limited case-report evidence shows that extraoral autogenous bone, such as hip grafts, has high potential for bone growth. Extraoral sites require a second surgical site, and in some cases fresh grafts may be associated with root resorption. Case report evidence indicates bone fill exceeding 50 percent of the osseous defect may be achieved. Controlled studies comparing grafted to nongrafted sites report significant improvements in clinical attachment levels and bone gain, but the magnitude of gain is less than that indicated in case reports.

Freeze-dried demineralized bone represents one of the most frequently used and well-studied bone graft materials in periodontics. Freeze-dried demineralized bone is an allograft material, harvested, prepared and demineralized prior to grafting. The demineralization step is important because it retains the activity of bone morphogenetic proteins, compounds in the graft material found to be essential for new bone formation. Case reports and controlled clinical trials have demonstrated the bone-forming potential of such material, with some variability in the amount of bone fill achieved. Because allografts are derived from donor tissues, proper collection, handling and storage are essential to ensure viability and prevent contamination with viruses or other pathogens.

Alloplasts represent a class of synthetic resorbable or nonresorbable graft materials. When evaluated in controlled clinical trials, they demonstrated improvements in probing depth and attachment level. Histology, however, indicates that, in general, synthetic grafts act primarily as space fillers, with little, if any, regeneration.

Beginning in the 1980s, a number of investigators explored a procedure called guided tissue regeneration. The idea was to employ either a resorbable or nonresorbable membrane at the diseased site that would selectively allow passage of cells able to regenerate periodontal attachment apparatus and bone while prohibiting migration of nonregenerative cells such as fibroblasts. The evidence for the efficacy of guided tissue regeneration ranged from randomized controlled clinical trials to case reports. Although less evidence is available for resorbable membranes than for nonresorbable membranes, significant improvements in clinical attachment levels have been shown compared to debridement alone. Most favorable results are reported for bone loss between the roots of mandibular tooth defects (Class II furcations). Less favorable results were reported in maxillary molar and Class III (through and through) furcation defects (Garrett 1996).

Birth defects

There are hundreds of genetic diseases and syndromes as well as congenital anomalies that affect the craniofacial, oral and dental tissues. However, some craniofacial anomalies may be spontaneous and manifest only at the time of birth. Rapidly advancing knowledge of the genetics of development and of mutations associated with specific birth defects is aiding in the development of screening tests for genetic disorders and identifying high-risk individuals and families.

A complete diagnosis of the craniofacial disorder may involve a multidisciplinary team of experts in imaging, genetics and other areas. Similarly, long-term management of the disorder, often extending to adulthood, generally calls for a team of specialists, including physicians and dentists, surgeons, nurses, rehabilitation experts, speech pathologists, psychologists and social workers. Quality of life considerations, including social and psychological effects of birth defects such as cleft lip and palate, are taken into account.

Prevention

Primary prevention involves minimizing exposure to known teratogens, and genetic counseling as appropriate. The importance of educating parents or potential parents on behavioral risk factors, especially tobacco and alcohol use, the teratogenic potential of certain prescription drugs and the need for adequate nutrition in the perinatal period is emphasized. A study performed in 1995, supplementation of the diet by multivitamins and folic acid during the periconceptional period (i.e., before, during and after conception) markedly diminished the occurrence of cleft lip and palate in a high-risk group. Unfortunately, only about 29 percent of women of childbearing age consume recommended amounts of these essential nutrients.

The evidence associating moderate to severe periodontal disease in pregnant women with low-weight preterm births warrants attention to the importance of maintaining optimal oral health in pregnancy. The oral care clinician can contribute to birth defect prevention not only by treating oral disease, but also by providing educational messages to patients to promote the birth of healthy, full-term babies.

Treatment

A number of birth defects may not be apparent at birth because they are not manifested until later in development. One example is the ectodermal dysplasias (ED), disorders characterized by abnormalities of skin, hair, sweat glands and teeth. Dentists are essential in the management of care for children with these disorders, who must be repeatedly fitted with dentures throughout childhood.

More recently, clinical studies have demonstrated that fitting ectodermal dysplasia patients as young as 12 years old with dental implants not only is effective, but also provides greater functional utility and satisfaction. As with other complex craniofacial anomalies, management by a multidisciplinary team is the best approach, with experts able to advise on the various oral, skin and sweat gland complications.

Mutations have recently been identified for several forms of ED, including the anhydrotic form (absence of sweat glands). Ultimately, the development of genetic diagnostic tests can confirm the diagnosis in the child and permit counseling of parents.

Chronic craniofacial pain and sensorimotor conditions

A variety of problems involving pain and other sensorimotor abnormalities affect the craniofacial complex. These conditions can include burning mouth syndrome, trigeminal neuralgia, various facial palsies, postherpetic neuralgia affecting branches of the trigeminal nerve, temporomandibular disorders, fibromyalgia and disorders of taste or olfaction. Some of these are infectious in origin (e.g., postherpetic neuralgia and some taste disorders); some are traumatic (e.g., some cases of temporomandibular disorder); and for others, the cause or causes are unknown. Patients with facial palsies and trigeminal neuralgia are generally referred to neurologists for treatment. Disorders of taste and smell also require neurological consultation as well as brain imaging because they can be symptomatic of brain tumors.

Pain relief may also improve function and can be combined with adjunctive measures such as the use of hot or cold compresses and behavioral treatments such as relaxation and imaging therapy to reduce muscle tension. The variety of pain medications has greatly increased in recent years. They include aspirin and other nonsteroidal anti-inflammatory drugs, tricyclic antidepressants, new antiepileptic drugs, the selective serotonin re-uptake inhibitors and the more potent opiate family of drugs.

If the pain problem has recently developed, providers can take steps to prevent the pain from becoming chronic. This will entail a general health assessment to determine whether there are co-morbidities, including other pain problems, as well as patient questionnaires to provide information on how the pain problem is affecting overall health and well-being. The data collected will record the extent to which the problem interferes with work, social interaction and sleep, whether the patient is experiencing mood changes and symptoms of depression, and what coping skills are manifest. Such patient profiles allow for more selective treatment tailored to the needs of the individual patient.

Patients in whom pain has become chronic and intractable may be referred to an established pain clinic for multidisciplinary treatment and may also be alerted to patient organizations where individuals with similar pain problems can find information and support.

Temporomandibular disorders

Among the common types of craniofacial pain likely to be seen by oral care providers are temporomandibular disorders, characterized by symptoms of pain and dysfunction in and around the temporomandibular joints or the masticatory muscles.

Temporomandibular disorders may occur as a result of injury, arthritis or fibromyalgia or for unknown reasons. Approaches used to obtain a differential diagnosis of these conditions can range from a physical examination that may include palpation and measuring the mouth opening, to the use of complex imaging and instrumentation, including procedures such as arthroscopy.

Diagnosis of temporomandibular disorders is based on the physical examination and a complete medical and dental history, including information about hearing, speech and swallowing problems, as well as pain and dysfunction. This information can be complemented by data from imaging and other diagnostic tests. Evaluation encompasses examination of oral-facial tissues, musculature and neurological function.

Particular attention is paid to measures of the range of motion, mouth opening, existence of any parafunctional conditions (e.g., clenching, grinding), and the presence of joint or muscle tenderness

and cutaneous hyperalgesia. Features of the reliability studies on the examination methods have been reviewed. Psychosocial assessments using validated instruments can determine the extent to which pain and dysfunction diminish the patient's quality of life and can suggest appropriate treatments.

The evidence base for the efficacy of treatment modalities is severely limited and has resulted in a wide range of diagnostics and therapies. Treatments range from conservative and reversible approaches to joint surgical procedures. At present the evidence is insufficient to warrant prophylactic intervention for management of these disorders.

Currently available epidemiological evidence suggests that temporomandibular disorders can frequently resolve over time and that conservative, reversible approaches are the treatments of choice. Ideally, the practitioner and the patient should work together to develop a treatment plan that is evidence-based and patient-centered, taking into consideration all etiologic factors, the level of pain and dysfunction the patient is experiencing, and their impact on the patient's quality of life.

Factors affecting future dental health care practices

The last decades of the 20th century were witness to major improvements in the prevention, diagnosis and treatment of oral diseases, a trend that will continue to accelerate the paradigm shift in the management of oral diseases from repair of damaged tissues to the control of infections. In addition, modification of risk factor exposures will result in improvements in health and in the management of disease.

A closer look into factors that will affect the future of oral health care requires an overview of the current state of guidelines for oral care and the status of evidence-based practice. The approaches used to determine the evidence for practice and the development of guidelines for care are an emerging field of activity. Education in the health professions is already emphasizing the importance of relying on randomized, controlled clinical trials, the gold standard for judging the merits of proposed interventions, wherever possible.

Evidence-based practice

During the 1990s, "evidence-based medicine" emerged as both popular phraseology and practice philosophy. The origins of evidence-based medicine go back to mid-19th century Paris and earlier, yet the approach is still a relatively young discipline that is now rapidly evolving. Evidence-based medicine has been defined as the integration of "individual clinical expertise with the best available external clinical evidence from systematic research" and with patients' choices. The skills required include defining a clinical problem, critically appraising the relevant literature, and deciding whether and how to integrate this information into practice (Evidence-Based Medicine Working Group 1992).

Evidence-based medicine is neither a "cookbook" nor an ivory tower approach. The philosophy is being adopted across a range of disciplines, leading to the terms "evidence-based dentistry" and "evidence-based nursing," among others. The practice of evidence-based dentistry "incorporates the judicious use of the best evidence available from systematic reviews, when possible, with knowledge of patients' preferences and clinicians' experiences to make recommendations for the provision of the right care, for the right patient, and at the right time."

The reliance on evidence using systematic reviews of the literature has led to initiatives in the United States, Canada and Europe to enhance the conduct and use of systematic reviews. The Agency for Healthcare Research and Quality (AHRQ) created 12 evidence-based practice centers in 1997 to conduct systematic reviews and develop evidence reports. The Cochrane Collaboration and the Centre for Reviews and Dissemination at the University of York are examples of prominent activities in the United Kingdom to support systematic reviews. The Cochrane Oral Health Review Group, one of 50 specialty review groups within the Cochrane Collaboration, has a number of systematic reviews completed or under way of interest to oral health practitioners. In Canada, considerable contributions to the field have been made by McMaster University and the Canadian Coordinating Office for Health Technology Assessment.

In the United States, the National Institute of Dental and Craniofacial Research joined efforts with AHRQ in 1999 to designate one of AHRQ's Evidence-based Practice Centers to conduct reviews on oral, dental and craniofacial diseases and disorders. The work of this center should significantly strengthen the scientific base of knowledge related to the diagnosis and management of oral, dental, and craniofacial conditions. Examples of topics that will be reviewed include the management of dental caries and dental care of medically compromised patients, including patients with HIV disease.

Conclusion

During the past several decades, there have been major improvements in the prevention, diagnosis and treatment of oral diseases. Enhanced disease prevention and health promotion will require the participation of all health professions, especially in addressing common risk factors such as tobacco, alcohol and inappropriate dietary practices. The field of diagnostic tests for oral diseases should continue to expand, enabling clinicians to analyze the risk of disease and disease progression for individual patients. Full assessment of the strengths and weaknesses of new diagnostic tests and evaluation of when they are best used will be key to proper interpretation of the results, permitting tailored referrals and treatments.

Treatment options for individual patients are increasing, including the recent efforts to understand and define early childhood caries and other periodontal infections. The increased knowledge of risk factors, the importance of monitoring disease progression and treatment effects, and the ability to diagnose conditions and intervene earlier will necessitate increased involvement of all health professionals in oral health care and may reflect changes in care provision and referral patterns.

Management of conditions such as oral and pharyngeal cancers, cleft lip/palate, and chronic pain requires multidisciplinary teams. The promotion of oral health and the prevention of oral disease are at a turning point. A systematic approach to integrate the scientific findings into evidence-based assessments will provide clearer guidance to all health care professions and the public. To capitalize on the rapidly emerging science base, the active participation of a full range of dentists and additional health care providers with individuals in the community is needed.

References

- American Surgeon General World Health Organization

Final Examination Questions

Select the best answer questions 26 through 30 and mark your answers on the Final Examination Answer Sheet found on page 148, or for faster service complete your test online at **Dental.EliteCME.com.**

26. The periodontal ligament, which anchors the teeth in the jaws, is a tactilely sensitive tissue providing important feedback with regard to mastication and dental occlusion.
    - ○ True
    - ○ False

27. The effects of xerostomia may be particularly severe in cancer patients receiving radiation to the head or neck because the rays can destroy salivary gland tissue rather than simply inhibiting salivary secretion.
    - ○ True
    - ○ False

28. Disease-causing microorganisms cannot be spread to the mouth by indirect contact.
    - ○ True
    - ○ False

29. Patients with diabetes mellitus are less susceptible to periodontal diseases.
    - ○ True
    - ○ False

30. Temporomandibular disorders may occur as a result of injury, arthritis or fibromyalgia or for unknown reasons.
    - ○ True
    - ○ False

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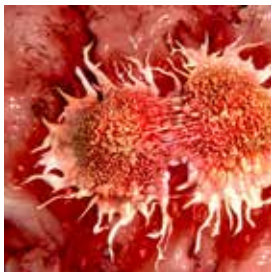


Chapter 7: Oral Health and Oral Pharyngeal Cancers

5 CE Hours

By: Elite Staff

Learning objectives

- Define the term oral health.
- Study the parts of the mouth affected by oral cancer.
- Review the different types of cancer, benign and malignant.
- List the major risk factors of oral cancer.
- Identify the signs and symptoms.
- Describe the possible treatments and side effects.

Introduction

In 2008, in the United States alone, about 34,000 individuals were diagnosed with oral cancer. Statistics show that 66 percent of the time, these will be found as a late stage three or four disease. The term oral cancer includes cancers of the mouth and the pharynx, part of the throat. About two-thirds of oral cancers occur in the mouth and about one-third are found in the pharynx. Oral cancer will be diagnosed in an estimated 35,000 Americans this year and will cause approximately 7,500 deaths. Oral cancer can spread quickly. On average, 60 percent of those with the disease will survive more than five years. Oral cancer most often occurs in people over the age of 40 and affects more than twice as many men as women. Low public awareness of the disease is a significant factor, but these cancers could be found at early, highly survivable stages through a simple, painless, five-minute examination by a trained medical or dental professional.

Scientists are studying oral cancer to learn more about this disease, and doctors are exploring new ways to treat it. This research keeps increasing our knowledge about oral cancer.

Oral health means more than healthy teeth and the absence of disease. It involves the ability of individuals to carry out essential functions such as eating and speaking as well as to contribute fully to society. The meaning of oral health has developed in tandem with progress in understanding the two chief dental diseases – dental caries and periodontal diseases – which historically have been the major preoccupation of patients, providers and research investigators alike. There is a marvelous success story here regarding the prevention of oral cancer and how it can be detected early. These investigations were complemented by studies of the tissues of the mouth and adjacent areas – the craniofacial complex.

Great progress has been made in reducing the extent and severity of common oral diseases, and recent history has seen marked improvements in the nation's oral and dental health, thanks to successful prevention measures adopted by communities, individuals and oral health professionals. However, not everyone is experiencing the same degree of improvement. What amounts to a silent epidemic of dental and oral diseases is affecting some population groups – a burden of disease that restricts activities in school, work and home, and often significantly diminishes the quality of life.

The word oral, both in its Latin root and in common usage, refers to the mouth. The mouth includes not only the teeth and the gums (gingiva) and their supporting connective tissues, ligaments and bone, but also the hard and soft palate, the soft mucosal tissue lining of the mouth and throat, the tongue, the lips, the salivary glands, the chewing muscles and the upper and lower jaws, which are connected to the skull by the temporomandibular joints. Equally important are the branches of the nervous, immune and vascular systems that animate, protect and nourish the oral tissues, as well as provide the connections to the brain and the rest of the body. The genetic patterning of development in utero further reveals the intimate relationship of the oral tissues to the developing brain and to the tissues of the face and head that surround the mouth, structures whose location is captured in the word craniofacial.

A major theme of this course is that oral health means much more than healthy teeth. It means being free of chronic oral-facial pain conditions, oral and pharyngeal (throat) cancers, oral soft tissue lesions, birth defects such as cleft lip and palate, and scores of other diseases and disorders that affect the oral, dental and craniofacial tissues, collectively known as the craniofacial complex. These are tissues whose functions we often take for granted, yet they represent the very essence of our humanity. They allow us to speak and smile; sigh and kiss; smell, taste, touch, chew and swallow; cry out in pain; and convey a world of feelings and emotions through facial expressions. They also provide protection against microbial infections and environmental insults.

The craniofacial tissues also provide a useful means to understanding organs and systems in less accessible parts of the body. The salivary glands are a model of other exocrine glands, and an analysis of saliva can provide telltale clues of overall health or disease. The jawbones are examples of other skeletal parts. The nervous system apparatus underlying facial pain has its counterpart in nerves elsewhere in the body.

A thorough oral examination can detect signs of nutritional deficiencies as well as a number of systemic diseases, including microbial infections, immune disorders, injuries and some cancers. Indeed, the phrase "the mouth is a mirror" has been used to illustrate the wealth of information that can be derived from examining oral tissues.

New research is pointing to associations between chronic oral infections and heart and lung diseases, stroke, and low birth-weight and premature births. Associations between periodontal disease and diabetes have long been noted. This course assesses these associations and explores mechanisms that might explain these oral-systemic disease connections.

In parallel with the broadened meaning of oral health, the meaning of health has evolved. The standard definition of health, "freedom from disease, defect, or pain," defines what health is not, rather than what it is. A more positive definition, one that the World Health Organization established in 1948, states that health is a complete state of physical, mental, and social well-being, and not just the absence of infirmity.

The broadened meaning of oral health parallels the broadened meaning of health. In 1948, the World Health Organization expanded the definition of health to mean "a complete state of physical, mental and social well-being, and not just the absence of infirmity." It follows that oral health must also include well-being. Just as we now understand that nature and nurture are inextricably linked, and mind and body are both expressions of our human biology, so, too, we must recognize that oral health and general health are inseparable. We ignore signs and symptoms of oral disease and dysfunction to our detriment.

Consequently, a second theme of the report is that oral health is integral to general health. You cannot be healthy without oral health. Oral health and general health should not be interpreted as separate entities. Oral health is a critical component of health and must be included in the provision of health care and the design of community programs.

The wider meanings of oral and health in no way diminish the relevance and importance of the two leading dental diseases, caries (tooth decay) and the periodontal diseases. They remain common and widespread, affecting nearly everyone at some point in the life span. What has changed is what we can do about them.

At the start of the 20th century, most Americans expected to be toothless by age 45, and most were. Expectations have changed, and most people now assume that they will maintain their teeth over their lifetime and take active measures to do so. Researchers in the 1930s discovered that people living in communities with naturally fluoridated water supplies had fewer dental caries than people drinking unfluoridated water. But not until the end of World War II were the investigators able to design and implement the community clinical trials that confirmed their observations and launched a better approach to the problem of dental caries: prevention. Soon after, adjusting the fluoride content of community water supplies was pursued as an important public health measure to prevent dental caries.

Although this measure has not been fully implemented, the results have been dramatic. Dental caries began to decline in the 1950s among children who grew up in fluoridated cities, and by the late 1970s, declines in decay were evident for many Americans. The application of oral science to improved diagnostic, treatment and prevention strategies has saved billions of dollars per year in the nation's annual health bill. Even more significant, the result is that far fewer people are edentulous (toothless) today than a generation ago.

The theme of prevention gained momentum as pioneering investigators and practitioners in the 1950s and 1960s showed that not only dental caries but also periodontal diseases are bacterial infections. The researchers demonstrated that the infections could be prevented by increasing host resistance to disease and reducing or eliminating the suspected microbial pathogens in the oral cavity. The applications of research discoveries have resulted in continuing improvements in the oral health of Americans, new approaches to the prevention and treatment of dental diseases, and the growth of the science.

The significant role that scientists, dentists, dental hygienists and other health professionals have played in the prevention of oral disease and disability leads to a third theme of this report: safe and effective disease prevention measures exist that everyone can adopt to improve oral health and prevent disease. These measures include daily oral hygiene procedures and other lifestyle behaviors, community programs such as community water fluoridation and tobacco cessation programs, and provider-based interventions such as the placement of dental sealants and examinations for common oral and pharyngeal cancers. It is hoped that this surgeon general's report will facilitate the maturing of the broad field of craniofacial research so that gains in the prevention of craniofacial diseases and disorders can be realized that are as impressive as those achieved for common dental diseases.

At the same time, more needs to be done to ensure that messages of health promotion and disease prevention are brought home to all Americans. In this regard, a fourth theme of the report is that general health risk factors, such as tobacco use and poor dietary practices, also affect oral and craniofacial health. The evidence for an association between tobacco use and oral diseases has been clearly delineated in almost every surgeon general's report on tobacco since 1964, and the oral effects of nutrition and diet are presented in the surgeon general's report on nutrition (1988). All the health professions can play a role in reducing the burden of disease in America by calling attention to these and other risk factors and suggesting appropriate actions.

Clearly, promoting health and preventing disease are concepts the American people have taken to heart. For the third decade, the nation has developed a plan for the prevention of disease and the promotion of health, embodied in the U.S. Department of Health and Human Services (2000) document, Healthy People 2010. As a nation, we hope to eliminate disparities in health and prevent oral diseases, cancer, birth defects, AIDS and other devastating infections; mental illness and suicide; and the chronic diseases of aging. To live well into old age free of pain and infirmity and with a high quality of life is the American dream.

Scientists today take that dream seriously in pursuing the intricacies of the craniofacial complex. They are using an ever-growing array of sophisticated analytic tools and imaging systems to study normal function and diagnose disease. They are completing the mapping and sequencing of human, animal, microbial and plant genomes, the better to understand the complexities of human development, aging and pathological processes. They are growing cell lines, synthesizing molecules and using a new generation of biomaterials to revolutionize tissue repair and regeneration. More than ever before, they are working in multidisciplinary teams to bring new knowledge and expertise to the goal of understanding complex human diseases and disorders.

The mouth

The mouth is the gateway to the body, performing dozens of functions that place high demands on its unique hard and soft tissues. The point of entry is the lips, which open into the oral cavity. The cheeks form the sides of the cavity, and the roof is formed by the palate, which separates the mouth from the nose above and the pharynx (throat) behind. The anterior palate is hard, formed by underlying bone, and serves as a shield against trauma to the face and head. The posterior palate is soft, composed of muscles and connective tissue that blend into the walls of the pharynx. Hanging from the rear of the soft palate is the uvula, a mass of muscle and connective tissue. Under the tongue is the floor of the mouth, composed primarily of muscle and salivary glands. The paired tonsils and adenoids, important components of the immune system, lie at the sides of the palate and within the nasopharynx, respectively.

The pharynx opens into channels leading either to the lungs for respiration or the esophagus for further digestion and passage to the stomach. This is a point of vulnerability, where food or other obstructions can lodge in the airway and lead to death by asphyxiation.

Externally, the oral cavity is bounded by the maxilla (the upper jaw bone), attached to the cranium, and the mandible (the lower jaw), attached to the temporal bone of the skull by the temporomandibular joint.

The oral mucosa

Except for the teeth, the oral tissues are covered by a mucous membrane called the oral mucosa, which varies in color from pink to brownish purple, depending on an individual's skin color. Like skin, the oral mucosa acts as a major barrier against chemical irritants and mechanical forces; it can even withstand temperatures that would be painful to the skin. In areas subject to chewing forces and food movements, the surface layer is relatively hard, composed of epithelial cells filled with insoluble keratin, the fibrous protein found in skin, nails, hair, and animal horn. Elsewhere – in the mucosal lining of the cheeks, for example – the surface layers are softer and more flexible, enabling the mobility we need to speak, chew and make facial expressions. To aid in their barrier function, surface mucosal cells are square-shaped and closely juxtaposed, with specialized organelles and cell products that promote cell-cell adherence. The cells can also secrete sticky molecules to plug gaps between them and further impede penetration by damaging chemicals or microorganisms. Still another type of oral mucosa forms the pebbly surface of the back and sides of the tongue. Lining the depths of these surface "papillae" are the taste buds.

Interestingly, the epithelium that lines the gingival surface completely lacks a keratin layer, yet this "naked" epithelium lies next to one of the most dense concentrations of bacteria to be found in the body. Thus there is an opportunity for infectious agents or their byproducts to penetrate the naked epithelial barrier and initiate an inflammatory response, as happens in gingival disease.

Special cells in the basal layer of the oral mucosa generate replacements for surface cells as they wear out. The painful oral ulcers and oral mucositis that may develop in patients undergoing radiation or chemotherapy for head and neck cancer occur because these cancer-killing agents attack all cells undergoing rapid turnover, whether healthy or cancerous.

The teeth

The most prominent features of the oral cavity are the teeth. The 20 primary, or deciduous, teeth erupt generally between 6 months and 2 to 3 years of age and are succeeded by the permanent teeth beginning at about age 6. The primary teeth enable infants to eat solid foods, aid speech development, and serve as placeholders for the permanent dentition. Keeping primary teeth healthy is important, not only in sparing an infant pain and disease, but also in preserving the dimensions of the dental arches and lessening the risk of dental caries in the permanent teeth. A period of mixed primary and permanent dentition occurs from about ages 6 to 13. There are 28 to 32 permanent teeth, depending on whether the four wisdom teeth (third molars), which are last to erupt, are present. Teeth are anchored in the jaws by the periodontal ligament. This ligament connects the cervix (neck) of the tooth, at the junction between the crown and root, to the gingiva. Below that, the ligament connects the outer layer of the tooth root, the cementum, to the adjacent alveolar bone (the part of the jaw bone that supports the tooth roots).

The evolutionary forces that shaped the human mouth designed an apparatus for optimal food intake. The front four upper and lower incisor teeth are chisel-shaped for biting, cutting, and tearing and exert forces of 30 to 50 pounds. The canines, or cuspids, are larger and stronger and have deeper roots than the incisors; their conical cusps are effective for ripping and tearing. The premolars, or bicuspids, and the molars are designed for heavy grinding and chewing, exerting forces as high as 200-plus pounds. The temporomandibular joint, the most complex synovial joint in the body, equips the human jaw with extraordinary mobility, enabling movements in three dimensions. Its range of motion is controlled by three sets of muscles of mastication – the masseter, temporalis, and pterygoid muscles. Chewing reduces food to small particles and mixes it with saliva to form a bolus for swallowing.

The salivary glands

Saliva is the mixed product of multiple salivary glands that lie under the mucosa. The three major glands are the paired parotid, submandibular and sublingual glands. The parotids, near the ears, secrete a watery saliva into the mouth via ducts in the cheeks. The walnut-sized submandibular glands lie in the floor of the mouth and secrete a mucous fluid. The secretions of the almond-shaped sublingual glands, also in the floor of the mouth but near the front, usually join with those of the submandibular glands. Tiny minor salivary glands are scattered within the inner surfaces of the lips, cheeks and soft and hard palates; these secrete a mucinous saliva directly onto the soft tissue surfaces.

Saliva moistens food and provides mucinous proteins to lubricate the bolus for ease of swallowing. The combined movements of the tongue and cheeks move the bolus to the back of the mouth. Saliva also contains the enzyme amylase, which initiates the digestion of starch. By solubilizing food components and facilitating their interaction with the taste buds on the tongue and palate, saliva also contributes to taste enhancement.

Tissue protection

The main function of saliva is not – as is commonly believed – to aid digestion, but to protect the integrity of the oral tissues. The moment a baby passes through the birth canal and takes its first breath, microbes begin to take up residence in its mouth. Later, as the teeth erupt, additional bacteria establish colonies on tooth surfaces. Nearly 500 species of microbes in all, most of which are not harmful, will colonize the oral cavity. The microbes form a biofilm, in which their numbers greatly exceed the number of human inhabitants on Earth.

Millions of years before there were toothbrushes, dental floss and water irrigators, evolutionary forces generated protective mechanisms to combat potentially harmful microbes. The physical flow of saliva helps to dislodge pathogens (viruses, bacteria and yeast) from teeth and mucosal surfaces, just as tearing and blinking, sneezing and coughing and expectorating clear the eye, nose and throat. Saliva can also cause microbes to clump together so that they can be swallowed before they become firmly attached. Saliva can destroy orally shed infected white blood cells by virtue of its low salt content: the infected cells – of higher salt content – swell and burst when exposed to fluids of lower salinity.

Salivary secretions, like tears and other exocrine gland secretions, are rich in antimicrobial components. Certain molecules in saliva, such as lysozyme, lactoferrin, peroxidase and histatins, can directly kill or inhibit a variety of microbes; the histatins are particularly potent antifungal agents. Several salivary proteins exhibit antiviral properties, including secretory leukocyte protease inhibitor (SLPI), recently discovered to have the ability to inhibit HIV from invading cells.

The ability of saliva to limit the growth of pathogens, in some instances even preventing them from establishing a niche in the

biofilm community in the first place, is a major determinant of general as well as of oral health. When salivary flow is compromised, the gateway to the body can open wide to local as well as to systemic pathogens.

Barrier and buffering properties

Salivary components protect oral tissues in other ways as well. Mucins have unique properties that enable them to concentrate on mucosal surfaces and provide an effective barrier against drying and physical and chemical irritants. They act as natural waterproofing, control the permeability of the tissue surfaces and help limit penetration of potential irritants and toxins in foods and beverages, as well as toxic chemicals and potential carcinogens in tobacco and tobacco smoke and from other sources. This barrier function complements the barrier formed by the oral mucosa itself. The mucosa has a specific permeability coefficient that can change under various conditions of stress, nutritional status, and other challenges.

Saliva contains several effective buffering systems that can help maintain a normal pH when acidic foods and beverages are introduced, thereby protecting oral tissues against acidic attack. When swallowed, these buffers protect the esophagus, helping neutralize the reflux acids of conditions such as heartburn and hiatal hernia.

Wound healing

Saliva also contains molecules that nurture and preserve the oral tissues, even helping them to repair and regenerate. Experimental studies have shown that wound healing is significantly enhanced by saliva, in part because of the presence of a potent molecule, epidermal growth factor (EGF). When swallowed, EGF can also protect the tissue surfaces of the esophagus. Vascular endothelial growth factor (VEGF) has also been identified in saliva. VEGF stimulates blood vessels and may contribute to the remarkable healing capacity of oral tissues.

Caries protection

Saliva also guards against dental caries (tooth decay), the disease that has been the greatest threat to teeth. Caries is caused by bacteria that generate acids that attack tooth mineral. The buffering systems in saliva, augmented by the neutralizing components urea and ammonium, counter the acid formation. The physical flow of saliva also helps flush out sugars and food particles that are the bacterial food source. Mineral salts in saliva – calcium and phosphate – can remineralize tooth enamel, effectively reversing the decay process. This regenerative function is greatly enhanced by the presence of fluoride in saliva. Finally, saliva forms a film on teeth made up of selectively adsorbed proteins that have a high affinity for tooth mineral. This acquired pellicle is insoluble and limits the diffusion of acids into the teeth and the dissolution of tooth mineral.

The immune system

The salivary glands and the oral mucosa, along with the body's other mucosal linings and the lymphatic circulation, constitute a major component of the body's defense system – the mucosal immune system. When the area of the oral mucosa is combined with the areas of the mucosal linings and passageways of the respiratory, gastrointestinal, urinary, and genital tracts, the total represents the largest surface area of the body – nearly 400 square meters, or 200 times larger than the total skin area.

The great majority of infectious diseases affect or are acquired through mucosal surfaces. Immune cells that line the mucous membranes throughout the body secrete antibodies targeted to specific disease-causing microbes. The mucosal immune system works in concert with the blood-borne immune system to detect and dispose of foreign substances and invading microbes.

The two components of the immune system consist of molecules and cells that provide both broad and specific defense mechanisms. In the broad group are some circulating white blood cells (monocytes and granulocytes) associated with the inflammatory response. These cells migrate to a site of injury or infection and move into damaged tissues manifesting the four signs of inflammation: swelling, heat, redness and pain. The cells promote an increase in blood flow to begin the healing process, and they recruit other cells able to engulf and dispose of the offending organism.

The specific immune system is associated with two major classes of immune cells: T cells and B cells. T cells react to antigens (proteins associated with microbes or irritants) and can stimulate B cells to make antigen-specific antibodies. These are the Y-shaped molecules called immunoglobulins.

T cells are the instruments of cell-mediated immunity; they are able to detect telltale surface markers on diseased or foreign cells that distinguish them from normal body cells. Some T cells can kill infected cells and cancer cells directly. T cells are also involved in the rejection of organ transplants.

Certain T cells are memory cells, preserving the information from earlier encounters with specific pathogens. Thus they are able to initiate more rapid and effective responses in the event of a repeat encounter with the pathogen. Helper T cells assist in activating killer T and B cells. It is the loss of helper T cells that leads to the many infections that cause illness and death in HIV disease. Still another group of T cells, suppressor T cells, moderates the activities of both B and T lymphocytes.

Activated T cells generate and release cytokines – potent families of proteins, such as the interleukins, that can stimulate immune cells to divide, migrate, attack and engulf invaders or participate in the inflammatory response. Other cytokines include varieties of tumor necrosis factor and adhesins (proteins that facilitate the binding of immune cells to each other or to blood vessel linings). Feedback mechanisms provide a system of checks and balances to regulate cytokine production.

The immune system interacts with the nervous and endocrine systems. For example, immune cytokines secreted into the brain can induce the fever associated with infection: the high temperature may help destroy the infectious agent. The brain's response to stress also has repercussions for the immune system. The hypothalamus pituitary-adrenal axis is a major pathway activated in response to stress, which results in the secretion of cortisol, the stress hormone, from the adrenal glands. Cortisol promotes the body's fight-or-flight mechanisms, but via feedback loops, cortisol acts to depress immune reactions.

Much of what we know about the immune system has come from studies of serum factors, but research in the last two decades has generated much new information about mucosal immunity. The mucosal immune system can be divided into inductive and effector compartments. The nasal-associated lymphoreticular tissues (NALT)

in the nasopharyngeal area (which includes the tonsils) and the gut-associated lymphoreticular tissues (GALT) in gut tissue are inductive regions where foreign invaders are encountered. If, for example, infectious bacteria are swallowed, they can stimulate immune cells in GALT to circulate T and B cells through the lymph system to various effector sites in the gastrointestinal and upper respiratory tracts and in the salivary and other exocrine glands. The B cells in the gland produce antibodies, designated S-IgA, to which is attached a secretory component. These antibodies are the dominant type found in saliva, tears, breast milk and colostrum and in the gastrointestinal and genitourinary tracts.

The uses of the mucosal immune system extend beyond its normal surveillance and defense functions. The tissues can be used as routes for delivery of oral (swallowed) or nasal (inhaled) vaccines, as sites for gene transfer to augment host defenses, and as a means of invoking oral tolerance — the suppression of overactive or inappropriate immune responses that occur in chronic inflammatory and autoimmune diseases.

Craniofacial origins

The extraordinary successes of research in molecular genetics over the past decade, coupled with the National Institutes of Health's project to map and sequence the human genome, have proved to be a boon in understanding craniofacial development. The use of automated gene-sequencing equipment, the Internet availability of genome databases and the ability to transfer genes or create "knockout" animals – in which a gene of interest has been eliminated – have greatly facilitated progress. The events that govern the transformation of a fertilized human egg cell into a healthy newborn with all organs and systems in place are being unfolded at the molecular level. Families of master and regulatory genes have been identified, and their role in controlling how the body's general shape and specialized tissues and organs are formed is coming to light.

EARLY DEVELOPMENT

The three-germ cell layers

By the time the face and the mouth are ready to form, the human embryo is in the third week of development. The embryo has evolved from a sphere to an oval, two-layered disk with a head-to-tail orientation. The outer layer is the epiblast and will become the ectodermal germ layer. A narrow groove, called the primitive streak, extends from the tail toward the center of the disk, where it ends in a spot surrounding a small depression called the primitive pit. Epiblast cells migrate toward the streak and pit, detach from the surface and slip downward to form the two additional germ-cell layers, the mesoderm and, below that, the endoderm.

The ectodermal layer gives rise to tissues that relate the body to the outside world: the nervous system; the sensory epithelium of the ears, nose, and eyes; skin, hair, nails, salivary glands, tonsils and tooth enamel; and the pituitary, mammary and sweat glands. At the head end, the mesodermal layer gives rise to a primitive connective tissue, called mesenchyme, which will interact with the ectoderm to form parts of the head and mouth. The remaining mesoderm develops into the muscle, cartilage, bone and subcutaneous skin tissue of the rest of the body. The mesoderm is also the origin of the vascular and urogenital systems (except for the bladder), the spleen and the adrenal cortex. The innermost, endodermal layer provides the linings of the gut, the respiratory system, bladder, liver, pancreas, thyroid and parathyroid glands, and parts of the middle ear.

Neural tube and neural crest

Further migrations and descending movements of cells result in the formation of the notochord, a solid cord of cells along the midline that will become the backbone. The ectoderm above the notochord next thickens to form a neural plate. The sides of the plate curve up and inward to form a neural tube, beginning at the head, with fusion completed by the end of the fourth week. The tail end of the tube will form the spinal cord; the head end differentiates into the three parts of the primitive brain: the forebrain, midbrain and hindbrain.

What happens next is of central importance to the craniofacial complex: Cells that were at the edges of the neural plate break away to form neural crest cells, which migrate to the forebrain area and to the nearby branchial arches, a series of swellings on either side of the embryo adjacent to the hindbrain. The hindbrain becomes organized into eight rhombomeres, segments of future nerve tissue arranged in an orderly fashion so that the first two rhombomeres innervate branchial arch 1, and so on.

During the formation of the midbrain and hindbrain, cranial neural crest cells migrate into the developing facial areas and differentiate into neuronal and nonneuronal tissues. The neuronal tissues include the clusters of nerve cells (ganglia) that lie adjacent to the spinal cord, parts of the ganglia of four cranial nerves, and two of the meningeal layers of the brain. The nonneuronal tissues include major bones, cartilage, the dentin and cementum of teeth, and the various types of connective tissues of the craniofacial complex, as well as the muscles of the eye. The branchial arches give rise to the bones, cartilage, nerves, muscles, and blood supply of successive segments of the head and neck.

The face and mouth

The branchial arches play a key role in the formation of the facial structures. Toward the end of the fourth week of gestation, a primitive mouth appears. This "stomadeum" is flanked by a series of swellings, or prominences, derived from the first pair of branchial arches. A single frontonasal prominence forms the upper border of the stomadeum. On either side of this prominence are two thickened regions of ectoderm – the nasal placodes. At the sides of the stomadeum and below it are pairs of maxillary and mandibular prominences.

In the course of the next three weeks, differential growth and movements of the various prominences and fusions of tissues that come together at the midline will sculpt the bridge, crest, sides and tip of the nose, the upper and lower lips and the upper and lower jaws.

The external merger at the midline of a pair of prominences that helps to form the nose occurs inside the mouth as well, resulting in an intermaxillary segment that will contribute to the formation of the four upper incisors and parts of a small triangular-shaped primary palate and the upper jaw. The bulk of the palate, the secondary palate,

forms from shelflike outgrowths of the maxillary prominences. These growths appear in the sixth week, and in the following week fuse along the midline above the tongue. (The tongue appears at approximately four weeks, the front two-thirds forming from the first branchial arch and the posterior third from parts of the second, third and fourth branchial arches.) The palatal shelves also fuse with the primary palate along a triangular border called the incisive foramen. This border is considered the line of division among clefting abnormalities. Lateral cleft lip, cleft upper jaw, and clefts between the primary and secondary palates are associated with defects anterior to the incisive foramen. Cleft palate and cleft uvula occur because of defects affecting closure of the palatal shelves posterior to the foramen.

The teeth

Tooth development begins in the sixth week with the appearance of an epithelial band lining the upper and lower jaws. A part of the band develops into a dental lamina, which forms a series of projections into the jaw. These are the tooth buds and correspond to the sites of deciduous teeth. The epithelial tissue of the bud develops into an enamel organ that forms a cap over tissue that is differentiating in the jaw to become the dental papilla. The two structures – the enamel organ, derived from the epithelium, and the dental papilla, derived from neural crest mesenchyme – constitute the tooth germ.

With further development, the tooth germ assumes a bell shape and separates from the oral epithelium. At the same time, the internal epithelial layer of the enamel organ undergoes a series of infoldings that will shape the future crown of the tooth.

Mineralization of the tooth begins at the late bell stage. The first mineralized tissue to form is dentin, which provides the foundation for the deposition of enamel. The differentiation of the odontoblasts (the dentin-producing cells) depends on organizing influences from enamel organ cells. Thus the development of these two different hard tissues is a mutually dependent process.

As dentin is laid down, the odontoblasts move toward the center of the papilla, trailing thin cellular processes, which become embedded in the mineralized matrix. When dentin formation is completed, dentin completely surrounds the pulp, protecting it from injury. The enamel layer of the tooth starts to form soon after the first dentin appears, synthesized by special enamel-forming cells, or ameloblasts, which develop from the enamel organ. The tooth root and its outer layer of cementum form only after the crown erupts.

Genetic controls

Only in the last decade have scientists begun to understand how certain genes and gene families control embryonic development. Their findings have come from detailed studies of species ranging from fruit flies, nematodes and zebrafish to frog, chick, mouse and human embryos. In many cases, the simpler organism has been the source of discoveries of genes or developmental processes that are highly conserved in the course of evolution.

Research on the fruit fly, for example, has revealed that particular families of genes are responsible for the fundamental head to thorax to tail patterning of the fly's body. Another set of genes determines the back-to-front positioning of organs, and a third set subdivides this general body plan into a series of discrete segments. With further development, yet another family of genes confers a positional memory on the cells within a segment. These "homeotic selector" genes ensure that cells in one part of a particular segment "know" that they are destined to be wings and not legs, or to be eyes and not antennae. In flies the homeotic genes are known as hom genes. Their arrangement on the fly chromosome is ordered with genes at one end of the chromosome specifying the developmental destiny of cells in the most anterior segments of the fly's body and genes at the other end specifying the fate of cells in the most posterior segments.

In the course of evolution, mammals have developed four overlapping sets of positional memory gene clusters homologous to the fly's single hom complex. The four mammalian hox gene families are ordered in a similar anterior-posterior fashion along four different chromosomes. The mammalian genes appear to operate like the hom genes: they code for DNA-binding proteins that control gene expression. The similarity from fly to human is particularly evident when maps of the expression domains of hom genes in anterior segments of the fly embryo are compared to maps of hox gene expression as seen in the rhombomeres and branchial arches of mammals.

Molecular genetic studies of flies and other nonmammalian species show some variation in how and when the basic body patterns and repeating segments are formed. Sometimes the head-to-tail pattern is laid down in the egg cell before fertilization – dictated by egg polarity genes. Although egg polarity genes do not operate in humans, mutations have been found in a human gene homologous to the fly egg polarity gene and account for serious syndromes in which there are defects in anterior organs, such as the pituitary gland and heart.

None of these developmental controls work in isolation. Much remains to be understood about the genetic clock that determines when and where developmental genes act, how they interact, and what mechanisms are used to sustain as well as terminate their function. The systems that govern programmed cell death are also important: normal development depends as much on the elimination of cells as it does on the orderly movement, proliferation and differentiation of cells.

When it comes to processes that control the development of particular tissues or organs – bones, skin or heart – developmental biologists observe that there is often an "organizer," that is, a cell or set of cells that initiates the process. The organizer induces changes in the behavior of neighboring cells through cell-cell interactions, so that these cells develop into the specified type – bone or skin or heart muscle. The interaction with the neighboring cell is often in the form of a signaling molecule, such as a growth factor (e.g., transforming growth factor beta, epidermal growth factor, fibroblast growth factor) that attaches to a receptor on the surface membrane of the recipient cell. This interaction is translated to the interior of the cell, where a chain of molecular interactions eventually reaches the cell nucleus to effect gene expression. One of the more startling discoveries of the past decade has been the finding that a series of mutations, each associated with a change in only one nucleotide of the gene for the fibroblast growth factor receptor – also called point mutation – accounts for a range of organ defects seen in at least a half dozen craniofacial syndromes. Interestingly, all these syndromes include craniosynostosis, a premature closure of the bones that form the skull.

The aging of craniofacial tissues

Normal aging describes the developmental processes that begin at conception, continue in childhood and merge gradually into maturation and senescence. The milestones of development such as the age when children teethe, begin to walk, talk, enter puberty, attain their full height and so on, are under genetic and hormonal controls, subject to important environmental factors such as nutrition and exercise. Despite the complexity and interrelationships of the variables involved, a

reasonably accurate picture of normal age-related changes in the craniofacial complex is emerging.

Barring major illness or injury, destructive behaviors or severe or unusual environmental circumstances, the cells, tissues and fluids of the face and mouth are hardy survivors, eminently durable and functional over a long life span. For any given individual the combination of life experience and lifestyle (including medical and dental history) creates a unique craniofacial portrait, one that inspired George Orwell to remark, "By the age of 50, a man gets the face he deserves."

The teeth

One of the more dramatic discoveries in biomedical science in the 20th century has been the realization that tooth loss is not an inevitable consequence of aging, but the result of disease or injury. Aging does produce a number of other dental changes, however. Teeth change in form and color with age. Wear and attrition alter the biting and chewing surfaces, as do food choices and oral habits. The altered surface structure produces a different pattern of light reflection in older teeth, resulting in some yellowing and a general loss of translucency. Fully formed enamel is acellular, hence there is no metabolic activity or turnover as occurs in skin, for example. Dentin and cementum have limited cellular activity. In contrast, tooth pulp and periodontal ligament undergo relatively high levels of tissue turnover.

Tooth surfaces can be eroded by chemical dissolution from fruit acids and from acids from sugars in foods such as soft drinks and candies. This destructive process can occur at any age, resulting in loss of translucency as well as some tissue loss from demineralization. Countering the erosive forces are the natural components in saliva that help re-mineralize the enamel surface, a process that is enhanced when fluoride is present.

The cementum increases in thickness with age. Gingival recession caused by normal aging exposes the cementum to the oral environment (and is the origin of the expression "long in the tooth"). The exposed cementum can often be worn away mechanically, exposing the underlying dentin, which can then become hypersensitive. Dentin responds through a series of protective changes that work to close off the connections between dentin and nerves in the pulp, reducing transmission of painful stimuli.

The jaws

The bones of the maxilla and mandible that support the teeth, called the alveolar processes, are, like bone elsewhere in the body, subject to cellular turnover in a coordinated process of bone resorption and formation. Alveolar bone is well adapted to mechanical stresses and changes continuously during facial growth, tooth eruption, tooth wear and tooth loss. This lifelong adaptation makes orthodontic treatments to reposition teeth in adults possible.

Because the primary function of alveolar bone is to support the teeth, the loss of teeth will lead to bone atrophy, making prosthetic replacements difficult. The rate of bone loss is affected by both local disease such as periodontal disease and systemic conditions such as osteoporosis.

The oral mucosa

The oral mucosa appears to age in much the same way as skin does. The oral epithelium thins and becomes less hydrated, increasing vulnerability to injury. The rate of cell division is slower, but the basic cell architecture and patterning of cell types throughout the oral cavity are maintained. It is not certain to what extent these changes are a natural consequence of aging; they may be due to altered protein synthesis or lowered responsiveness to regulatory molecules. They may also be an effect of diminished vascularity, which could limit cellular access to oxygen and nutrients.

Overall immune system function deteriorates with age, and it is likely that mucosal immunity does as well. Such a decline could result in an increased risk of transmission of infectious agents across the mucosa and probably contributes to delayed wound healing in oral tissues with aging.

Sensory and motor functioning

The high density of sensory nerve endings in the craniofacial tissues and their functional abilities are well-preserved in aging. There may be minor increases in threshold detection levels or in judgments of intensity, but, for the most part, sensory cells can turn over or have a built-in reserve capacity that allows for near-optimal functioning in aging. The exception is olfaction, which declines in both men and women with age. This decrement in smell may lead to some dissatisfaction with how foods taste and increased use of flavor enhancers to compensate. But for most people, the ability to enjoy food is not appreciably diminished as time goes by. Any dramatic change in sensory function – complaints of a continued unpleasant taste or smell or the sudden complete loss of a sensory modality – should be taken seriously as a sign of possible oral or systemic disease or a side effect of medication and not dismissed as a natural byproduct of aging.

The distribution of motor fibers in the craniofacial tissues is also abundant and sufficiently fine-tuned to allow for a full range of movement of the tongue, jaws and oral-facial muscles. There is some loss of muscle tone in aging, along with changes in tongue shape and function in articulating specific speech sounds. Subtle changes may also occur in preparing food for swallowing. As with sensory changes, these developments do not seriously interfere with motor function in healthy older adults.

The salivary glands

Studies of normative aging indicate that individuals vary in the quantity of "whole" saliva they produce. Whole saliva consists of the secretions of the various salivary glands plus other oral contents, such as cells shed from the mucosa. These individual patterns are consistent across the life span. In healthy adults, there is no diminution in the production of saliva from the major salivary glands in the course of aging.

This constancy may seem surprising given the morphological changes documented in aging salivary glands. Both the parotid and the submandibular glands lose between 20 and 30 percent of their essential tissue volume in the course of aging. The loss primarily affects the acinar components, the cells that secrete saliva. Increases in the number of ductal cells and in fat, vascular, and connective tissues compensate for this loss, however – evidence of the remarkable functional reserve capacity of the glands, which enables them to maintain a stable salivary output across the life span.

In contrast, studies of age-related changes in the chemistry of salivary secretions suggest that there are significant reductions in the concentration of mucins from the submandibular gland, which could result in reduced lubrication and contribute to a sensation of mouth dryness. There are also subtle changes in the protective ability of salivary secretory IgA antibody.

Findings

Natural selection has served Homo sapiens well in evolving a craniofacial complex with remarkable functions and abilities to adapt, enabling the organism to meet the challenges of an ever-changing environment. An examination of the various tissues reveals elaborate designs that have evolved to serve the basic needs and functions of a complex mammal as well as those that are uniquely human, such as speech. The rich distribution of nerves, muscles and blood vessels in the region as well as extensive endocrine and immune system connections is an indication of the vital role of the craniofacial complex in adaptation and survival over a long life span. In particular, genes controlling the basic patterning and segmental organization of human development, and specifically the craniofacial complex, are highly conserved in nature. Mutated genes affecting human development have counterparts in many simpler organisms.

There is considerable reserve capacity or redundancy in the cells and tissues of the craniofacial complex, so that if they are properly cared for, the structures should function well over a lifetime.

The salivary glands and saliva subserve tasting and digestive functions and also participate in the mucosal immune system, a main line of defense against pathogens, irritants, and toxins.

Salivary components protect and maintain oral tissues through antimicrobial components, buffering agents and a process by which dental enamel can be remineralized.

Oral and pharyngeal cancers and precancerous lesions

In 2000, oral or pharyngeal cancer was diagnosed in an estimated 30,200 Americans and caused more than 7,800 deaths (Greenlee et al. 2000). Over 90 percent of these cancers are squamous cell carcinomas – cancers of the epithelial cells. The most common oral sites are on the tongue, the lips and the floor of the mouth. Oral cancer is the sixth most common cancer in U.S. males and takes a disproportionate toll on minorities; it now ranks as the fourth most common cancer among African American men. The prominent role of tobacco use, especially in combination with alcohol, in causing these cancers is a major incentive to develop effective health promotion and disease prevention efforts.

This course is about cancers that occur in the mouth (oral cavity) and the part of the throat at the back of the mouth (oropharynx).

The oral cavity and oropharynx have many parts:

Pictured below are the following parts:

- Lip.
- Tongue.
- Salivary glands (glands that make saliva).
- Floor of the mouth.
- Hard palate.
- Soft palate.
- Uvula.
- Oropharynx.
- Tonsils.
- Tongue.

Heightening the risk

Oral cancer develops as a clone from a single genetically altered cell. It generally has a long latency period and invariably develops from a precancerous lesion on the oral mucosa, such as a white leukoplakia, or more commonly, a reddish erythroplakia. Both kinds of lesions are usually induced by tobacco use alone or in combination with heavy use of alcohol. The development of squamous cell carcinoma from initial erythroplakia lesions has been well demonstrated experimentally. Reported rates of malignant transformation for leukoplakias are between 0.13 and 17.5 percent. However, there is considerable debate as to the actual malignant potential of the leukoplakia lesion associated with the use of smokeless (spit) tobacco. Meaningful data for determining a specific malignant transformation rate or relative risk of oral cancer due to smokeless tobacco use are difficult to obtain because of the confounding effects of other habits such as concurrent smoking and alcohol consumption and because of the variations in smokeless (spit) products and how they are used.

Another oral precancerous lesion that has received attention is submucous fibrosis. It is commonly seen in India and Southeast Asia and is related to betel nut use.

Early epidemiologic studies identified behaviors such as smoking and environmental factors such as exposure to solar radiation and x-rays as causes of intraoral and lip cancers. Researchers then sought experimentally to explain the mechanisms of initiation. In the 1980s and 1990s, investigators exploited the techniques of molecular biology and genetics to probe what was going on deep inside the cell. These studies revealed an abundance of systemic and local factors, including viral and fungal infections, that affect cell behavior. Some factors stimulate cell division and others inhibit it – even to the point of initiating a program of cell "suicide," called apoptosis. How a given cell behaves at any given time in its life cycle is the net result of the signals it receives from neighboring cells and molecules, from circulating factors in the blood or immune system and from its own internal controls. The following sections provide a brief description of these factors and how they may participate in enhancing the risk for the development of oral cancers.

Tobacco and alcohol

Tobacco and alcohol are the major risk factors for oral cancers, and their effects have been studied for many years. Tobacco contains substances that are frankly carcinogenic or act as initiators or promoters of carcinogenesis. Among these are N-nitrosonornicotine, 4-nitroquinoline-N-oxide and benzpyrene. The most damaging carcinogens are found in the tars of tobacco smoke, but many forms of smokeless (spit) tobacco, including snuff, have been implicated in the development of mouth cancer. Other habits that have been related to oral cancer include chewing betel nut in the presence of tobacco, as is done primarily in Southeast Asia, and, more recently, using marijuana.

The role of alcohol in oral carcinogenesis has been demonstrated experimentally and appears to be related to its damaging effect on the liver. Major metabolites of alcohol, such as acetaldehyde – a known carcinogen in animals – may also be important. Alcohol is also thought to act as a solvent that facilitates the penetration of tobacco carcinogens into oral tissues. That observation may partly explain why the combined use of tobacco and alcohol produces a greater risk for oral cancer than use of either substance alone. Indeed, tobacco and alcohol, working in tandem, are thought to account for 75 to 90 percent of all oral and pharyngeal cancers in the United States.

Viruses

The role of viruses in causing cancer in animals was established early in the century when Rous showed that a virus, later named the Rous sarcoma virus (RSV), caused tumors in chickens. The issue of whether viruses could cause cancer in humans remained unexplored until the mid-1970s, when Varmus and Bishop showed that RSV had a special gene, which they called src (for sarcoma), that could transform the cell it infected into a malignant cell. It was an oncogene, or cancer-causing gene. The researchers subsequently, and surprisingly, discovered that src was not native to the virus, but had been picked up by some ancestor virus from a chicken cell's own genome, where src had presumably played a role in the chicken cell's normal growth and development. Somehow RSV was able to subvert src when it infected a chicken cell to cause the cell to divide uncontrollably.

Varmus and Bishop called the normal cellular src gene a proto-oncogene, meaning that it had the potential to be converted to an oncogene. Subsequent research led to the discovery of other viruses that could cause tumors in animals and revealed the presence of proto-oncogenes in birds and mammals. These genes could also be converted to oncogenes, behaving exactly like those carried by cancer viruses. In 1982, an oncogene isolated from a human bladder cancer turned out to be virtually identical to ras, the oncogene found in a rat sarcoma virus.

Viruses that have been implicated in oral cancer include herpes simplex type 1 and human papillomavirus. Epstein-Barr virus, also a herpes virus, is now accepted as an oncogenic virus responsible for Burkitt's lymphoma, occurring primarily in Africa, and nasopharyngeal carcinoma, occurring primarily in China. HPV is a major etiologic agent in cervical cancer, and has been found in association with oral cancer as well (Sugerman and Shillitoe 1997). HPV DNA sequences have been found in oral precancerous lesions as well as in squamous cell carcinomas, and experimental evidence has shown that HPV-16 can be an important cofactor in oral carcinogenesis. Herpes simplex type 1 antibodies were demonstrated in patients with oral cancer, and herpes was found to induce dysplasia (abnormal cellular changes) in the lips of hamsters when combined with the application of tobacco tar condensate.

More recently, human herpes virus 8, a newly identified member of the herpes virus family, has been found in Kaposi's sarcoma, an otherwise rare cancer occurring in patients with AIDS. These tumors often appear initially within the oral cavity. Other uncommon oral malignant tumors, such as Hodgkin's lymphoma and non-Hodgkin's lymphoma, can also occur in the mouths of AIDS patients.

In addition to viruses, infection with strains of the fungus Candida albicans has been linked to the development of oral cancers via the fungal production of nitrosamines, which are known carcinogens.

Genetic derangements

Of the more than 50 known oncogenes, many have been reported to be present in oral cancer, and multiple oncogenes have been reported in oral and pharyngeal cancer. Some of these are Bcl-1, c-erb-B2, c-myc, ins-2 and members of the ras family.

The genetic derangements that can give rise to oral cancer, including many mutations associated with the transformation of proto-oncogenes, have received notable attention. In some instances, a change in a single nucleotide base – a point mutation – in a gene encoding a proto-oncogene is enough to transform it into an oncogene. Cancerous changes may also involve alterations, deletions and break points in chromosomes that affect the position of genes.

Mutations that disarm the cell's DNA repair mechanisms as well as mutations in tumor suppressor genes, which inhibit abnormal cell growth, play a major role in cancer development. If an individual inherits or acquires a mutation in one or more tumor-suppressor genes, for example, the loss of this protective mechanism reduces the number of other deleterious changes needed for cancer to develop.

Tumor suppressor genes suspected to be mutated in oral and pharyngeal cancers include those for Rb, p16 (MTS1, CDKN2, or IN4a), and p53. Of the group of tumor suppressor genes, that coding for p53 is considered of major importance, with mutations in the p53 gene detected in many types of cancer, including oral and pharyngeal. The p53 gene has been called the "guardian of the genome" because of its ability to recognize damage to a cell's DNA and stop the process of growth and division until the damage is repaired. If repair is not possible, p53 can trigger apoptosis. Mutations in the p53 gene in oral cancer have been linked to smoking and alcohol use.

Loss of immunosurveillance and control

The immune system can, as first noted by Paul Ehrlich in 1909, seek and destroy initial clones of transformed cancer cells. Ehrlich called this process immunosurveillance, and it has been confirmed in experimental animals and in humans with induced immunosuppression.

One mechanism of immunosurveillance involves stimulating cytotoxic macrophages and lymphocytes to migrate to the tumor site and release tumor necrosis factors alpha and beta. Another mechanism operative in oral cancer appears to be stimulation of Langerhans cells, a special group of immune cells, in the oral mucosa. Other immune cells implicated in tumor rejection are natural killer cells and lymphokine-activated killer cells.

There is an increased incidence of cancer in patients with AIDS or other immunodeficient conditions or with induced immunosuppression prior to organ transplantation.

In addition, there is evidence that smoking depresses the immune system, and this may be one of the ways in which smoking acts as a major risk factor in oral cancer.

Growth factors

Immune cells are potent generators of growth factors and other molecules that can stimulate other cells to migrate and proliferate. This capacity is important in normal cell growth and turnover, in wound healing and in coping with infection. Unfortunately, the release of growth factors can contribute to oral cancer by stimulating keratinocyte (oral epithelial cell) proliferation. Increased levels of transforming growth factor alpha (TGF-alpha) and epidermal growth factor have been found in oral and pharyngeal cancers and therefore could serve as markers for malignancy. Nicotine at high doses stimulates the release of growth hormones, among other endocrine effects.

Understanding cancer

Cancer begins in cells, the building blocks that make up tissues. Tissues make up the organs of the body. Normally, cells grow and divide to form new cells as the body needs them. When cells grow old, they die, and new cells take their place. Sometimes this orderly process goes wrong. New cells form when the body does not need them, and old cells do not die when they should. These extra cells can form a mass of tissue called a growth or tumor. Tumors can be benign or malignant:

- **Benign tumors**:
  Benign tumors are not cancer:
  - Benign tumors are rarely life-threatening.
  - Generally, benign tumors can be removed, and they usually do not grow back.
  - Cells from benign tumors do not invade the tissues around them.
  - Cells from benign tumors do not spread to other parts of the body.
- **Malignant tumors**:
  - Malignant tumors are cancer.
  - Malignant tumors are generally more serious than benign tumors.
  - They may be life-threatening.
  - Sometimes they grow back.
  - Cells from malignant tumors can invade and damage nearby tissues and organs.
  - Cells from malignant tumors can spread to other parts of the body.
    - The cells spread by breaking away from the original cancer (primary tumor) and entering the bloodstream or lymphatic system.
    - They invade other organs, forming new tumors and damaging these organs.
    - The spread of cancer is called metastasis.

What are benign tumors?

There are many forms of benign (noncancerous) tumors that can appear in the oral cavity or oropharynx (in addition to other sites in/on the body), including:

Benign Tumors	
Condyloma acuminatum (also known as a genital warts)	A small, moist, pink or red growth that grows alone or in cauliflower-like clusters.
Eosinophilic granuloma	A benign tumor that most often affects children and adolescents and is usually found in a bone or the lungs.
Fibroma	A benign tumor consisting of fibrous connective tissues.
Keratoacanthoma	A flesh-colored, fast-growing bump on the skin with a keratin plug in the center (keratin, the main component of the external layer of skin, hair and nails, is a tough substance).
Leiomyoma	A tumor of the smooth muscle, often found in the esophagus, small intestine, uterus or stomach.
Lipoma	A tumor made up of mature fat cells.
Neurofibroma	A fibrous tumor consisting of nerve tissue.
Odontogenic tumors	Tumors in the jaw.
Osteochondroma	A tumor made up of bone and cartilage.
Papilloma	A tumor that resembles a wart, growing on the epithelium (the cells that form the skin and mucous membranes).
Pyogenic granuloma	A small, round bump that often has an ulcerated surface.
Rhabdomyoma	A striated-muscle tumor that may appear on the tongue, pharynx, uterus, vagina or heart.
Schwannoma	A single tumor that grows in the neurilemma (Schwann's sheath) of nerves.
Verruca form xanthoma	Wart-shaped tumors.

Some benign tumors disappear on their own. Others may have to be removed surgically. Most benign tumors do not recur. Always consult your physician for a diagnosis.

What oral conditions may be precancerous?

Two conditions in the mouth – leukoplakia and erythroplakia – actually can be precursors to cancer. Often caused by smoking or chewing tobacco, these (initially) benign conditions can occur anywhere in the mouth. Only a biopsy can determine whether precancerous cells (dysplasia) or cancer cells are present in a leukoplakia or erythroplakia.

- **Leukoplakia** – a condition characterized by a whitish patch that develops inside the mouth or throat.
- **Erythroplakia** – a condition characterized by a red, raised patch that develops inside the mouth.

Treatment for leukoplakias or erythroplakias may include use of retinoids – medications related to vitamin A – to eliminate, reduce, and/or prevent dysplasia from forming.

What are malignant oral tumors?

Although there are several types of malignant oral cancers, more than 90 percent of all diagnosed oral cancers are squamous cell carcinoma.

Malignant tumors	
Squamous cell carcinoma	Also known as squamous cell cancer, this type of cancer originates in the squamous cell layer in the lining of the oral cavity and oropharynx. In the early stages, this cancer is present only in the lining layer of cells (called carcinoma in situ). When the cancer spreads beyond the lining, it is called invasive squamous cell cancer.
Verrucous carcinoma	Although also considered a type of squamous cell carcinoma, this low-grade cancer rarely metastasizes (spreads to distant sites). Comprising less than 5 percent of all diagnosed oral cancers, verrucous carcinoma can spread deeply into surrounding tissue, requiring surgical removal with a wide margin of surrounding tissue.
Minor salivary gland cancers	The lining of the oral cavity and oropharynx contains numerous salivary glands. Sometimes cancer will originate in a salivary gland. Treatment depends on the type and location of the salivary gland cancer, as well as the extent of spreading. According to the American Cancer Society, salivary gland cancers account for less than 1 percent of all cancers.

Oral cancer and squamous cells

Oral cancer is part of a group of cancers called head and neck cancers. Oral cancer can develop in any part of the oral cavity or oropharynx. Most oral cancers begin in the tongue and in the floor of the mouth.

Almost all oral cancers begin in the flat cells (squamous cells) that cover the surfaces of the mouth, tongue, and lips. These cancers are called squamous cell carcinomas. When oral cancer spreads (metastasizes), it usually travels through the lymphatic system. Cancer cells that enter the lymphatic system are carried along by lymph, a clear, watery fluid. The cancer cells often appear first in nearby lymph nodes in the neck.

Cancer cells can also spread to other parts of the neck, the lungs and other parts of the body. When this happens, the new tumor has the same kind of abnormal cells as the primary tumor. For example, if oral cancer spreads to the lungs, the cancer cells in the lungs are actually oral cancer cells. The disease is metastatic oral cancer, not lung cancer. It is treated as oral cancer, not lung cancer. Doctors sometimes call the new tumor "distant" or metastatic disease.

Oral cancer: Who's at risk?

Dentists and doctors cannot always explain why one person develops oral cancer and another does not. However, we do know that this disease is not contagious. Scientists have determined that one cannot "catch" oral cancer from another person. Research has shown that people with certain risk factors are more likely than others to develop oral cancer. A risk factor is anything that increases your chance of developing a disease.

The following are risk factors for oral cancer:

- **Tobacco:** Tobacco use accounts for most oral cancers. Smoking cigarettes, cigars or pipes; using chewing tobacco; and dipping snuff are all linked to oral cancer. The use of other tobacco products (such as bidis and kreteks) may also increase the risk of oral cancer. Heavy smokers who use tobacco for a long time are most at risk. The risk is even higher for tobacco users who drink alcohol heavily. In fact, three out of four oral cancers occur in people who use alcohol, tobacco, or both alcohol and tobacco.
- **Alcohol:** People who drink alcohol are more likely to develop oral cancer than people who don't drink. The risk increases with the amount of alcohol that a person consumes. The risk increases even more if the person both drinks alcohol and uses tobacco.
- **Sun:** Cancer of the lip can be caused by exposure to the sun. Using a lotion or lip balm that has a sunscreen can reduce the risk. Wearing a hat with a brim can also block the sun's harmful rays. The risk of cancer of the lip increases if the person also smokes.
- **A personal history of head and neck cancer:** People who have had head and neck cancer are at increased risk of developing another primary head and neck cancer. Smoking increases this risk. Some studies suggest that not eating enough fruits and vegetables may increase the chance of getting oral cancer. Scientists also are studying whether infections with certain viruses (such as the human papillomavirus) are linked to oral cancer.

Quitting tobacco reduces the risk of oral cancer. Also, quitting reduces the chance that a person with oral cancer will get a second cancer in the head and neck region. People who stop smoking can also reduce their risk of cancer of the lung, larynx, mouth, pancreas, bladder and esophagus. There are many resources to help smokers quit:

- Advise patients to call The Cancer Information Service at 1-800-4-CANCER where they can talk with callers about ways to quit smoking and about groups that offer help to smokers who want to quit. Groups offer counseling in person or by telephone.
- Help patients find a local smoking cessation program.
- Tell them about the medicines (bupropion, Chantix) or about nicotine replacement therapy, which comes as a patch, gum, lozenges, nasal spray or inhaler.
- Give them The "National Cancer Institute Information Resources" information about the federal government's smoking cessation website, *http://www.smokefree.gov.*

As a dental professional, you should discuss any concerns your patients share with you regarding cancer or any that you may have with them as soon as possible. Discuss an appropriate schedule for checkups. Alert your patients that not using tobacco and limiting their use of alcohol are the most important things they can do to prevent oral cancers. If they spend a lot of time in the sun, using a lip balm that contains sunscreen and wearing a hat with a brim will help protect their lips.

Regular checkups can detect the early stages of oral cancer or conditions that may lead to oral cancer. Check the tissues in the mouth as part of the routine examination.

Symptoms

Common symptoms of oral cancer include:

- Patches inside the mouth or on the lips that are white, a mixture of red and white, or red.White patches (leukoplakia) are the most common. White patches sometimes become malignant. Mixed red and white patches (erythroleukoplakia) are more likely than white patches to become malignant. Red patches (erythroplakia) are brightly colored, smooth areas that often become malignant.
- A sore on the lip or in the mouth that won't heal.
- Bleeding in the mouth.
- Loose teeth.
- Difficulty or pain when swallowing.
- Difficulty wearing dentures.
- A lump in the neck.
- An earache.

Most often, these symptoms do not mean cancer. An infection or another problem can cause the same symptoms.

Diagnosis

- If your patient comes to you with symptoms that suggest oral cancer, such as red or white patches, lumps, swelling or other problems, you should explain to him or her very carefully that you are doing a special exam.
- Discuss this with the patient and explain that the exam includes looking carefully at the roof of the mouth, back of the throat and insides of the cheeks and lips.
- Let the patient know that you will gently pull out the tongue so it can be checked on the sides and underneath. Tell them that you will check the floor of the mouth and lymph nodes in the neck.
- Explain each step carefully. This is not a routine exam for you, and it is definitely not a routine checkup for your patient.
- When the exam is complete, you will need to discuss the next step with the patient.

Early diagnosis of oral and pharyngeal cancers

Dentists and primary care providers can counsel patients about lifestyle behaviors that increase the risk for oral cancers. Dental as well as medical personnel have provided successful tobacco control programs in their offices. Generally, Americans are ill-informed about the risk factors as well as the signs and symptoms of oral cancers The mass media have paid little attention to the topic, and health education textbooks are nearly devoid of discussion. The scant attention that has been paid to oral cancers has focused on the role of spit tobacco.

At present, the principal test for oral and pharyngeal cancers is a comprehensive clinical examination that includes a visual/tactile examination of the mouth, full protrusion of the tongue with the aid of a gauze wipe and palpation of the tongue, floor of the mouth and lymph nodes in the neck. The U.S. Preventive Services Task Force concluded that there was insufficient evidence to recommend for or against routine screening for oral cancers, but noted that clinicians should remain vigilant for signs and symptoms of oral cancers and premalignancy in people who use tobacco or regularly use alcohol.

The Canadian Task Force on Periodic Health Examination states that although there is insufficient evidence to include or exclude screening for oral cancers from the periodic health examination for the general public, those at high risk (smokers and heavy drinkers) over 60 warrant an annual oral cancer exam by a physician or dentist. The American Cancer Society recommends annual examinations for individuals 40 and older and for individuals who are exposed to known risks. Nevertheless, a 1992 national survey showed that only 15 percent of U.S. adults reported ever having had an oral cancer examination.

There are large gaps in knowledge of the efficacy of oral cancer examinations and the effectiveness and cost-effectiveness of community approaches to early detection of oral cancers. Methodologies and settings differ across studies. Moreover, these studies do not provide definitive evidence supporting the oral cancer exam, and there have been no controlled clinical trials for defining the effectiveness of screening programs. Further research is thus needed.

Biopsy

If the exam shows an abnormal area, a small sample of tissue may be removed. Removing tissue to look for cancer cells is called a biopsy. Usually, a biopsy is done with local anesthesia. Sometimes, it is done under general anesthesia. A pathologist then looks at the tissue under a microscope to check for cancer cells.

A biopsy is the only sure way to know whether the abnormal area is cancerous. When discussing a biopsy with patients, it is always good to be prepared to answer these questions:

- Why do I need a biopsy?
- How much tissue do you expect to remove?
- How long will it take? Will I be awake? Will it hurt?
- How soon will I know the results?
- Are there any risks? What are the chances of infection or bleeding after the biopsy?
- How should I care for the biopsy site afterward? How long will it take to heal?
- Will I be able to eat and drink normally after the biopsy?
- If I do have cancer, who will talk with me about treatment? When?

If an exam shows an abnormal area, a small sample of tissue may be removed. Usually, a biopsy is done with local anesthesia. Sometimes, it is done under general anesthesia. A pathologist then looks at the tissue under a microscope to check for cancer cells.

Remember, your patients are scared, because cancer is very serious. They will not understand many things. They will need you to explain everything to them and their family. It is often a good idea to schedule a family appointment to discuss the type of cancer and the necessary treatment.

Staging cancer

Staging is the key to finding the cancer in the early development. When found early, oral cancers have an 80 to 90 percent survival rate. Unfortunately at this time, the majority are found as late-stage cancers, and this accounts for the very high death rate of about 45 percent at five years from diagnosis, and high treatment-related morbidity in survivors. Late-stage diagnosis is not occurring because these cancers are hard to discover, it is because of a lack of public awareness coupled with the lack of a national program for opportunistic screenings which would yield early discovery by medical and dental

professionals. Worldwide the problem is far greater, with new cases annually exceeding 481,000.

If the biopsy shows that cancer is present, you will need to know the stage (extent) of the disease to plan the best treatment. The stage is based on the size of the tumor, whether the cancer has spread and, if so, to what parts of the body. Staging may require lab tests. It also may involve endoscopy. This involves a thin, lighted tube (endoscope) to check the throat, windpipe and lungs. You will need to explain this procedure to the patient before scheduling the appointment and inform them that local anesthesia is used to ease the discomfort. This exam may be done in a dental office, an outpatient clinic or a hospital.

At this time the dental professional may order one or more imaging tests to learn whether the cancer has spread:

- Dental x-rays: An x-ray of the entire mouth can show whether cancer has spread to the jaw.
- Chest x-rays: Images of the chest and lungs can show whether cancer has spread to these areas.
- CT scan: An x-ray machine linked to a computer takes a series of detailed pictures of the body.
    - Depending on the type of tests, an injection of dye may be used.
    - Tumors in the mouth, throat, neck or elsewhere in the body show up on the CT scan.
- MRI: A powerful magnet linked to a computer is used to make detailed pictures. The dentist or doctor can view these pictures on a monitor and can print them on film. An MRI can show whether oral cancer has spread.

Treatment

Many people with oral cancer want to take an active part in making decisions about their medical care. Be prepared to discuss all options with the patient. However, shock and stress after the diagnosis can make it hard to think of everything they want to ask. It often helps to make a list of questions and answers, have this ready to give to the patient before an appointment. Specialists who treat oral cancer include oral and maxillofacial surgeons, otolaryngologists (ear, nose and throat doctors), medical oncologists, radiation oncologists and plastic surgeons. There may be a team that includes specialists in surgery, radiation therapy or chemotherapy. Other health care professionals who may work with the specialists as a team include a speech pathologist, nutritionist and mental health counselor.

Surgical treatment for oral and pharyngeal cancers

Surgical treatment for oral and pharyngeal cancers can result in functional impairment as well as permanent disfigurement. Problems may include the loss of part of the tongue, loss of taste, loss of chewing ability, difficulty in speaking and pain. Furthermore, in addition to concerns about their function and their future, oral and pharyngeal cancer patients must cope with an altered appearance. In a study of patients who were disease-free from six months to eight years following surgical tumor removal, those with more pronounced disfigurement had greater changes in self-image, a worsened relationship with their partner, reduced sexuality and increased social isolation. One study noted that 30 percent of oral and pharyngeal cancer patients were still experiencing psychological distress seven to 11 years after treatment. Depression, too, is frequent in cancer patients. Patients with oral and pharyngeal cancers are at an even greater risk for depression than other cancer patients, due to surgeries that alter their appearance. Because oral and pharyngeal cancers are also frequently associated with chronic alcohol and tobacco use, depression may be related to withdrawal from these substances or to pre-existing psychopathology. Persistent pain, as noted earlier, may also be a contributing factor to depression.

A study conducted a prospective analysis of changes in quality of life over time with the aim of identifying which factors might be predictive of future improvements or declines. Participants were 186 oral and pharyngeal cancer patients, all smokers or recent former smokers, diagnosed with primary carcinomas of the oral cavity, pharynx, or larynx.

The patients were tested at baseline, at one month after radiation and/or surgery, and one year later (for a subset of 105 patients available for follow-up). Measures used included the Karnofsky Performance Scale, which uses expert judgments of functional performance scored from 0 to 100; the Cancer Rehabilitation Evaluation System Short Form, in which patients rate their quality of life along physical, psychosocial, marital, sexual and medical interaction scales; the previously mentioned Performance Status Scale for Head and Neck Cancer Patients (which includes scales for eating and speaking); and the Profile of Mood States, in which patients rate their feelings over the previous week, yielding analyses that enable scaling along six mood states: tension-anxiety, depression- dejection, anger-hostility, confusion-bewilderment, and vigor-activity.

Results indicated that in spite of functional improvement on some scales over time, there was continued dysfunction in speech and eating. Patients also reported declines in marital and sexual functioning, as well as an increase in alcohol use. Interestingly, the best predictor of quality of life one year after treatment was the scores obtained after initial smoking cessation advice was given, while the patients were undergoing treatment and in recovery. Other predictors were treatment type (quality of life was generally poorer for radiation patients) and score on the vigor subscale of the Profile of Mood States. The investigators concluded that medical follow-up must integrate tailored psychological and behavioral interventions to achieve better quality of life for oral and pharyngeal cancer patients.

Type of procedure

Surgical excision (removal) of the tumor is usually recommended if the tumor is small enough, and if surgery is likely to result in a functionally satisfactory result. Radiation therapy is often used in conjunction with surgery or as the definitive radical treatment, especially if the tumor is inoperable. Surgeries for oral cancers include:

- Maxillectomy (can be done with or without orbital exenteration).
- Mandibulectomy (removal of the mandible or lower jaw or part of it).
- Glossectomy (tongue removal, can be total, hemi or partial).
- Radical neck dissection.
- Moh's procedure.
- Combinational e.g. glossectomy and laryngectomy done together.

Owing to the vital nature of the structures in the head and neck area, surgery for larger cancers is technically demanding. Reconstructive surgery may be required to give an acceptable cosmetic and functional result. Bone grafts and surgical flaps such as the radial forearm flap are used to help rebuild the structures removed during excision of the cancer. An oral prothesis may also be required.

Survival rates for oral cancer depend on the precise site, and the stage of the cancer at diagnosis. Overall, survival is around 50 percent at

five years when all stages of initial diagnosis are considered. Survival rates for stage 1 cancers are 90 percent, hence the emphasis on early detection to increase survival outcome for patients.

Following treatment, rehabilitation may be necessary to improve movement, chewing, swallowing and speech. Speech and language pathologists may be involved at this stage.

Chemotherapy is useful in oral cancers when used in combination with other treatment modalities such as radiation therapy. It is seldom used alone as a monotherapy. When cure is unlikely, it can also be used to extend life and can be considered palliative but not curative care. Biological agents such as Cetuximab have recently been shown to be effective in the treatment of squamous cell head and neck cancers and are likely to have an increasing role in the future management of this condition when used in conjunction with other treatments.

Treatment of oral cancer will usually be by a multidisciplinary team, with treatment professionals from the realms of radiation, surgery, chemotherapy, nutrition, dental professionals, and even psychology all possibly involved with diagnosis, treatment, rehabilitation, and patient care.

Complications may include:

- Postoperative disfigurement of the face, head and neck.
- Complications of radiation therapy, including dry mouth and difficulty swallowing.
- Other metastasis (spread) of the cancer.

Methods of treatment

Oral cancer treatment may include surgery, radiation therapy, or chemotherapy. Some patients have a combination of treatments. At any stage of disease, people with oral cancer may have treatment to control pain and other symptoms, to relieve the side effects of therapy, and to ease emotional and practical problems. This kind of treatment is called supportive care, symptom management or palliative care.

Radiation therapy

Radiation therapy (also called radiotherapy) is a type of local therapy. It affects cells only in the treated area. Radiation therapy is used alone for small tumors or for patients who cannot have surgery. It may be used before surgery to kill cancer cells and shrink the tumor. It also may be used after surgery to destroy cancer cells that may remain in the area. Radiation therapy uses high-energy rays to kill cancer cells. Doctors use two types of radiation therapy to treat oral cancer:

- **External radiation**: The radiation comes from a machine. Patients go to the hospital or clinic once or twice a day, generally five days a week for several weeks.
- **Internal radiation** (implant radiation): The radiation comes from radioactive material placed in seeds, needles, or thin plastic tubes put directly in the tissue. The patient stays in the hospital. The implants remain in place for several days. Usually they are removed before the patient goes home. Some people with oral cancer have both kinds of radiation therapy.

Chemotherapy

Chemotherapy uses anticancer drugs to kill cancer cells. It is called systemic therapy because it enters the bloodstream and can affect cancer cells throughout the body.

Chemotherapy is usually given by injection. It may be given in an outpatient part of the hospital, at the dentist/doctor's office, or at home. Rarely, a hospital stay may be needed.

Rehabilitation after oral cancer

Rehabilitation may vary from person-to-person depending on the type of oral cancer treatment and the location and extent of the cancer. Rehabilitation may include:

- **Dietary counseling**: Many patients recovering from oral cancer surgery have difficulty eating, so it is often recommended that they eat small meals consisting of soft, moist foods.
- **Surgery:** Some patients may benefit from reconstructive or plastic surgery to restore the bones or tissues of the mouth, returning a more normal appearance.
- **Prosthesis:** If reconstructive or plastic surgery is not an option, patients may benefit from dental or facial-part prosthesis to restore a more normal appearance. Special training may be needed to learn to use a prosthetic device.
- **Speech therapy:** If a patient experiences difficulty in speaking following oral cancer treatment, speech therapy may help the patient relearn the process.

Side effects of cancer treatment

The possible side effects should be explained to each patient before treatment begins. Because treatment often damages healthy cells and tissues, unwanted side effects are common. These side effects depend mainly on the location of the tumor and the type and extent of the treatment. Side effects may not be the same for each person, and they may even change from one treatment session to the next.

Conclusion

While the diagnosis of oral cancer remains a challenge, oral health has come a long way in the last several years. The dental professionals, researchers and clinicians provide the latest information in cancer prevention and will continue to promote access to cancer screening services and clinical trials through a number of outreach initiatives.

Definitions

Benign – Not cancerous. Benign tumors do not spread to tissues around them or to other parts of the body.

Bidi – A cigarette made by rolling tobacco by hand into a dried leaf. Most bidis come from India in a variety of flavors.

Biopsy – The removal of cells or tissues for examination under a microscope. When only a sample of tissue is removed, the procedure is called an incisional biopsy or core biopsy. When an entire lumpor suspicious area is removed, the procedure is called an excisional biopsy. When a sample of tissue or fluid is removed with a needle, the procedure is called a needle biopsy or fine-needle aspiration.

Bupropion – A substance that is used to treat depression and to help people quit smoking. It belongs to the family of drugs called antidepressants.

Cancer – A term for diseases in which abnormal cells divide without control. Cancer cells can invade nearby tissues and can spread through the bloodstream and lymphatic system to other parts of the body.

Cell – The individual unit that makes up all of the tissues of the body. All living things are made up of one or more cells.

Chemotherapy – Treatment with anticancer drugs.

Clinical trial – A type of research study that uses volunteers to test new methods of screening, prevention, diagnosis or treatment of a disease. The study may be carried out in a clinic or other medical facility. Also called a clinical study.

CT scan – Computed tomography scan. A series of detailed pictures of areas inside the body taken from different angles; the pictures are created by a computer linked to an x-ray machine. Also called computerized tomography and computerized axial tomography (CAT) scan.

Dental implant – A small metal pin placed inside the jawbone or oral tissue. Dental implants can be used to help anchor a false tooth or teeth, or a crown or bridge.

Dentist – A health professional who specializes in caring for the teeth, gums and oral tissues.

Endoscope – A thin, lighted tube used to look at tissues inside the body.

Endoscopy – The use of a thin, lighted tube (called an endoscope) to examine the inside of the body.

Erythroleukoplakia – A patch found in the mouth that is a mixture of red and white. It can develop into cancer.

Erythroplakia – A reddened patch with a velvety surface found in the mouth. It can develop into cancer.

External radiation – Radiation therapy that uses a machine to aim high – energy rays at the cancer. Also called external-beam radiation.

Fluoride – A mineral that helps prevent tooth decay. Fluoride may be present in drinking water. It may be applied to the teeth as a gel, in toothpaste, or as a rinse.

General anesthesia – Drugs that cause loss of feeling or awareness and put the person to sleep.

Gland – An organ that makes one or more substances, such as hormones, digestive juices, sweat, tears, saliva or milk. Endocrine glands release the substances directly into the bloodstream. Exocrine glands release the substances into a duct or opening to the inside or outside of the body.

Graft – Healthy skin, bone or other tissue taken from one part of the body and used to replace diseased or injured tissue removed from another part of the body.

Hard palate – The front, bony portion of the roof of the mouth.

Head and neck cancer – Cancer that arises in the head or neck region (in the nasal cavity, sinuses, lip, mouth, salivary glands, throat or larynx [voice box]).

Human papillomaviruses – HPVs. Viruses that cause abnormal tissue growth (warts). Some types of HPV are associated with cervical and certain other cancers.

Imaging – Tests that produce pictures of areas inside the body.

Implant radiation – A procedure in which radioactive material sealed in needles, seeds, wires or catheters is placed directly into or near a tumor. Also called brachytherapy, internal radiation or interstitial radiation therapy.

Internal radiation – A procedure in which radioactive material sealed in needles, seeds, wires or catheters is placed directly into or near a tumor. Also called brachytherapy, implant radiation or interstitial radiation therapy.

Kretek – A cigarette made of a mixture of tobacco and clove spices.

Leukoplakia – A white patch that may develop on mucous membranes such as the gums, the tongue or the inside of the cheeks, and may become cancerous.

Local anesthesia – Drugs that cause a temporary loss of feeling in one part of the body. The patient remains awake but has no feeling in the part of the body treated with the anesthetic.

Local therapy – Treatment that affects cells in the tumor and the area close to it.

Lymph – The clear fluid that travels through the lymphatic system and carries cells that help fight infections and other diseases. Also called lymphatic fluid.

Lymph node – A rounded mass of lymphatic tissue that is surrounded by a capsule of connective tissue. Lymph nodes filter lymph (lymphatic fluid), and they store lymphocytes (white blood cells). They are located along lymphatic vessels. Also called a lymph gland.

Lymphatic system – The tissues and organs that produce, store and carry white blood cells that fight infections and other diseases. This system includes the bone marrow, spleen, thymus, lymph nodes and lymphatic vessels (a network of thin tubes that carry lymph and white blood cells).

Malignant – Cancerous. Malignant tumors can invade and destroy nearby tissue and spread to other parts of the body.

Medical oncologist – A doctor who specializes in diagnosing and treating cancer using chemotherapy, hormonal therapy and biological therapy. A medical oncologist often is the main health care provider for a person who has cancer. A medical oncologist also may coordinate treatment provided by other specialists.

Mental health counselor – A specialist who can talk with patients and their families about emotional and personal matters, and can help them make decisions.

Metastasis – The spread of cancer from one part of the body to another. A tumor formed from cells that have spread is called a "metastatic tumor" or a "metastasis." The metastatic tumor contains cells that are like those in the original (primary) tumor. The plural form of metastasis is metastases.

MRI – Magnetic resonance imaging – A procedure in which radio waves and a powerful magnet linked to a computer are used to create detailed pictures of areas inside the body. These pictures can show the difference between normal and diseased tissue. MRI makes better images of organs and soft tissue than other scanning techniques, such as CT or X-ray. MRI is especially useful for imaging the brain, spine, the soft tissue of joints, and the inside of bones. Also called nuclear magnetic resonance imaging.

Nutritionist – A health professional with special training in nutrition who can offer help with the choice of foods a person eats and drinks. Sometimes called a dietitian.

Oral cavity – The mouth.

Oral and maxillofacial surgeon – A dentist who specializes in surgery of the mouth, face and jaw.

Radiation therapy – The use of high-energy radiation from x-rays, gamma rays, neutrons and other sources to kill cancer cells and shrink tumors. Radiation may come from a machine outside the body (external-beam radiation therapy), or from materials called radioisotopes. Radioisotopes produce radiation and can be placed in or near the tumor or in the area near cancer cells. This type of radiation treatment is called internal radiation therapy, implant radiation, interstitial radiation, or brachytherapy. Systemic radiation therapy uses a radioactive substance, such as a radiolabeled monoclonal antibody, that circulates throughout the body. Also called radiotherapy, irradiation, and x-ray therapy.

Radioactive – Giving off radiation.

Radiotherapy – The use of high-energy radiation from x-rays, gamma rays, neutrons and other sources to kill cancer cells and shrink tumors. Radiation may come from a machine outside the body (external-beam radiation therapy), or it may come from radioactive material placed in the body near cancer cells (internal radiation therapy, implant radiation or brachytherapy). Systemic radiation therapy uses a radioactive substance, such as a radiolabeled monoclonal antibody, that circulates throughout the body. Also called radiation therapy, irradiation, and x–ray therapy.

Saliva – The watery fluid in the mouth made by the salivary glands. Saliva moistens food to aid in digestion and protects the mouth against infections.

Side effects – Problems that occur when treatment affects tissues or organs other than the ones meant to be affected by the treatment. Some common side effects of cancer treatment are fatigue, pain, nausea, vomiting, decreased blood cell counts, hair loss and mouth sores.

Soft palate – The muscular (not bony) part at the back of the roof of the mouth.

Speech pathologist – A specialist who evaluates and treats people with communication and swallowing problems. Also called a speech therapist.

Squamous cell carcinoma – Cancer that begins in squamous cells, which are thin, flat cells that look like fish scales. Squamous cells are found in the tissue that forms the surface of the skin, the lining of the hollow organs of the body and the passages of the respiratory and digestive tracts. Also called epidermoid carcinoma.

Squamous cells – Flat cells that look like fish scales under a microscope. These cells cover internal and external surfaces of the body. They are found in the tissue that forms the surface of the skin, the lining of the hollow organs of the body and the passages of the respiratory and digestive tracts.

Stage – The extent of a cancer within the body. If the cancer has spread, the stage describes how far it has spread from the original site to other parts of the body.

Staging – Performing exams and tests to learn the extent of the cancer within the body, especially whether the disease has spread from the original site to other parts of the body. It is important to know the stage of the disease in order to plan the best treatment.

Tumor – A new mass of excess tissue that results from abnormal cell division. Tumors perform no useful body function. They may be benign (not cancerous) or malignant (cancerous).

Uvula – The soft flap of tissue that hangs down at the back of the mouth (at the edge of the soft palate).

X-ray – A type of high-energy radiation. In low doses, x-rays are used to diagnose diseases by making pictures of the inside of the body. In high doses, x-rays are used to treat cancer.

References

- ♦American Cancer Society
- ♦American Surgeon General
- ♦www.cdc.gov

ORAL HEALTH AND ORAL PHARYNGEAL CANCERS

Final Examination Questions

Select the best answer questions 31 through 35 and mark your answers on the Final Examination Answer Sheet found on page 148, or for faster service complete your test online at **Dental.EliteCME.com.**

31. The great majority of infectious diseases affect or are acquired through mucosal surfaces.
    - ○ True
    - ○ False

32. The canines, or cuspids, are designed for heavy grinding and chewing, exerting forces as high as 200-plus pounds.
    - ○ True
    - ○ False

33. Two conditions in the mouth – leukoplakia and erythroplakia – actually can be precursors to cancer.
    - ○ True
    - ○ False

34. If oral cancer spreads to the lungs, the cancer cells in the lungs are actually oral cancer cells. The disease is metastatic lung cancer.
    - ○ True
    - ○ False

35. Staging is the key to finding the cancer in the early development.
    - ○ True
    - ○ False

DOH05OHE15

Chapter 8: Practical Oral Care for People with Developmental Disabilities

7 CE Hours

By: Elite Staff

Learning objectives

- Define autism, the different types, signs and symptoms.
- Describe how to provide oral care for patients with autism.
- Define cerebral palsy, the different types, the causes and the prognosis.
- Describe how to provide oral care for patients with cerebral palsy.
- Define Down syndrome, the common features, cognitive development, fertility and treatment management.
- Describe how to provide oral care for patients with Down syndrome.
- Define intellectual disability, review signs and symptoms, IQ levels and treatment.
- Describe how to provide oral care for patients with intellectual disabilities.
- Learn how to transfer a patient from a wheelchair to a dental chair.

Introduction

Approximately 4.5 million individuals in the United States have developmental disabilities such as autism, cerebral palsy, intellectual disability (mental retardation), and Down syndrome. One in every 33 babies in the United States is born with a birth defect or developmental disability. Birth defects are one of the leading causes of death in the first year of life and can affect how a child's body looks, works, or both. While the causes of some birth defects are known, we do not know what causes most of them. These individuals and their caretakers are very special people who live and cope with these disabilities every day, all day long. Developmental disabilities are present during childhood or adolescence and last a lifetime. They affect the mind, the body and the skills people use in everyday life, the normal daily activities of living: thinking, talking, bathing, brushing their teeth and feeding themselves. People with disabilities often need extra help to achieve and maintain good health. Oral health is no exception.

Today, approximately 80 percent of those with developmental disabilities are living in community-based group residences or at home with their families. People with disabilities and their caregivers now look to providers in the community for dental services.

Providing oral care to patients with developmental disabilities requires adaptation of the skills you use every day. Most people with mild or moderate developmental disabilities can be treated successfully in the general practice setting. This course will help you to understand a few of the disabilities better and presents an overview of physical, mental and behavioral challenges common in these patients and offers strategies for providing oral care.

Autism is a brain disorder that is associated with a wide range of developmental problems, especially in communication and social interaction. According to the American Psychiatric Association, autism is classified as a type of autism spectrum disorder (ASD). These disorders are characterized by problems with communication, social interaction and unusual, repetitive behaviors.

Some professionals use a broader term, called pervasive development disorder (PDD), to describe autism. In addition to autism, there are four other disorders that qualify as PDDs:

- Asperger's syndrome.
- Childhood disintegrative disorder.
- Pervasive developmental disorder-not otherwise specified (PDD-NOS).
- Rett syndrome.

The cause of autism remains unknown. Although most children are not diagnosed with autism until they are about preschool age, the first signs of autism generally appear between 12 and 18 months of age. The severity of symptoms varies among patients. Some are able to live independently once they become adults, while others may need lifelong support.

A minority of autistic patients may also be considered savants. These patients are autistic but express extraordinary mental abilities in a very specific area. Autistic savants often have exceptional skills with numbers, art or music. Not all savants are autistic. However, the number of autistic savants far exceeds the number of nonautistic savants.

As many as 1.5 million Americans may have autism. Researchers estimate that one to six of every 1,000 children have autism. The number of children diagnosed with autism has increased over the years. However, it is unclear whether more children are developing the disorder or better diagnostic techniques have helped doctors identify the disorder in more patients.

For unknown reasons, boys are three to four times more likely to develop autism than girls are. In the 1940s, when autism was first described, most autistic patients were institutionalized. Today, however, most autistic patients are able to live with their families. Although there is currently no cure for autism, treatments and therapies have been shown to help autistic patients live healthy, relatively normal lives. Regardless of how severe the patient's symptoms are, appropriate treatment and education can help autistic patients become integrated into their communities.

Other types of pervasive developmental disorders (PDDs)

- **Asperger's syndrome:** Patients with Asperger's syndrome have many of the same symptoms as autistic patients, in terms of problems with social interaction and communication. However, patients with Asperger's syndrome have normal intelligence and verbal skills. Although these patients typically have strong verbal and grammar skills, they usually have other language

problems, such as being too literal and/or having difficulty understanding nonverbal communications, such as body language. Other symptoms may include motor skill problems (e.g. clumsy movements), obsessive or repetitive routines and schedules, and sensitivity to sensory information (e.g. sound, light or taste).

- **Childhood disintegrative disorder**: Childhood disintegrative disorder, also called Heller's syndrome, is a rare condition in which children develop normally until they are about 3 or 4 years old. However, as they get older, children begin to experience a dramatic loss of social and communication skills, as well as motor skills. Patients may also develop stereotypical movements, such as hand wringing or flapping. These patients may develop specific routines or rituals and they may not respond well to changes or transitions. Some patients may become catatonic, which means they maintain a fixed posture or body position. Childhood disintegrative disorder is sometimes confused with late-onset autism because both of these disorders involve normal development followed by a loss of learned skills. However, autism generally occurs at an earlier age. Also, patients with childhood disintegrative disorder typically suffer from a much more dramatic loss of skills and greater likelihood of mental retardation than autistic patients do.
- **Pervasive developmental disorder-not otherwise specified (PDD-NOS):** Pervasive developmental disorder-not otherwise specified is a term used to describe patients who meet most, but not all, of the criteria for a PDD. Although these patients have many of the same symptoms associated with a PDD, they cannot be definitively diagnosed with a specific type of PDD.
- **Rett syndrome:** Rett syndrome is a progressive brain disorder that mostly affects females. Infants with Rett syndrome appear to develop normally at first, but over time, they stop developing and lose most of their previously developed skills. Eventually, these patients become intellectually disabled. Symptoms of Rett syndrome become noticeable when the patient is between the ages of 3 months and 3 years old. Patients typically lose purposeful hand movements (e.g. reaching or grasping for things) and they are no longer able to speak. Patients have difficulty balancing and poor coordination. This often prevents the patient from walking on his/her own. Patients may develop stereotypical hand movements, such as hand wringing or clapping. Breathing problems, including hyperventilation, breath-holding or apnea, may develop. Patients may also develop anxiety and social behavioral problems.

Causes

No definite cause has been found for autism, but these are some common causes known to trigger or influence the signs and symptoms of the behavior of the disorder.

- **Genetics:** A person's genetic makeup may play a role in his/her risk of developing autism. Researchers have identified several gene abnormalities that are associated with autism. According to researchers, families with one autistic child have a 3 to 8 percent chance of having a second child with the disorder. Although an individual's genetic makeup may influence the likelihood that they will develop the disorder, many other factors are also involved.
- **Medical conditions:** In some patients, autism has been linked to other medical conditions. For instance, autistic patients are more likely to have Fragile X syndrome (which causes mental retardation), tuberous sclerosis (which causes tumors to grow in the brain), epilepsy (which causes seizures), and Tourette's syndrome (which causes involuntary body movements).
- **Vaccines:** It has also been suggested that vaccines, especially the measles-mumps-rubella (MMR) vaccine, as well as mercury-containing vaccines, may lead to autism. However, current scientific research has not found a link between vaccines and autism.
- **Emotional trauma:** In the past, it was suggested that emotional trauma early in life increased a child's risk of developing autism. Traumatic events, such as physical abuse or neglect before the age of 3, were thought to contribute to the development of the disorder. However, researchers no longer support this theory.

Signs and symptoms

General: Autistic patients generally experience developmental problems that affect their behavior, social skills and language. The severity of symptoms varies among patients. Some patients may be able to live independently as adults, while others may require lifelong support. Patients with severe autism may be unable to communicate or interact with other people. The most severe autism occurs when the patient is completely unable to communicate or interact with others.

Children with autism may develop normally during the first few months or years of life. Then, usually before the age of 3, patients become less responsive to others.

A person with an ASD might:

- Not respond to his or her name by 12 months.
- Not point at objects to show interest (point at an airplane flying over) by 14 months.
- Not play "pretend" games (pretend to feed a doll) by 18 months.
- Avoid eye contact and want to be alone.
- Have trouble understanding other people's feelings or talking about his or her own feelings.
- Have delayed speech and language skills.
- Repeat words or phrases over and over (echolalia).
- Give unrelated answers to questions.
- Get upset by minor changes.
- Have obsessive interests.
- Flap hands, rock the body or spin in circles.
- Have unusual reactions to the way things sound, smell, taste, look or feel.

Behavior: Autistic patients may move constantly or perform repetitive movements, such as spinning or rocking. Patients typically develop specific routines or rituals and become highly disturbed if their schedules are even slightly changed. Patients may develop very specific interests, such as calendar dates or numbers. They may also become preoccupied with certain parts of an object, especially if the object has a repetitive motion. Some patients may develop abnormal posture or may walk on their toes. Autistic patients may be unusually sensitive to touch, sound or light. Some patients may be aggressive towards others or engage in behavior that hurts them, such as hitting their heads against the wall. Patients (adults and children) may throw temper tantrums, which may include yelling, crying, hitting, stomping of the feet or flailing of the arms or legs. Patients may have short attention spans, abnormalities in eating or sleeping habits, or extreme overactivity or underactivity.

Communication/social skills: Autistic patients may appear deaf because they may not respond to their name or they may appear not to hear others talking. An autistic patient may avoid eye contact with others or be unable to properly use body language, facial expressions or gestures. Autistic patients may resist cuddling and holding, appear unaware of others' feelings, or seem to prefer playing alone.

Emotion: Emotional symptoms vary considerably among autistic patients. Some patients may be unaware of others' feelings or be unable to express their own emotions. Some patients may be noticeably anxious or become depressed or frustrated when they are

unable to communicate to others. Some patients who express affection towards others may express this feeling indiscriminately.

Intelligence: Most children with autism are slow to learn new things or develop new skills. An estimated 75 percent of autistic patients have lower-than-normal intelligence quotients (IQs). However, the remaining 25 percent of patients have normal to high intelligence. Autistic patients with normal to high intelligence are quick learners, but still have difficulty communicating to others and applying their knowledge to everyday life. In rare cases, autistic patients may also be considered savants and have exceptional skills, such as math or art.

Language: While most children begin talking around the age of 1, autistic patients usually begin speaking at a later age. Patients may lose the ability to say words or sentences they were able to say in early childhood. Some patients may speak with an abnormal tone or rhythm. For instance, patients may use speech that sounds like a song or like a robot. Patients may be unable to start or maintain conversations with others. Patients may repeat words or phrases, but be unable to understand how to use them.

Neurological function: About 25-35 percent of autistic children experience seizures that may be resistant to medication. In most cases, children begin to experience the most seizures during early childhood and then again during adolescence. Autistic children who are mentally retarded or have a family history of autism have an increased risk of experiencing seizures.

Early diagnosis and prompt treatment has been shown to help improve autistic patients' long-term prognoses. According to the National Institute of Child Health and Human Development (NICHHD), children who experience certain developmental problems should visit their doctor. Children who have not made gestures, such as waving or pointing by the age of 12 months, should visit their doctor. Children who have not said a single word by the age of 16 months should visit their doctor. Children who have not said two-word phrases by 24 months of age should visit their doctor. Children who experience any loss of language or social skills at any age should visit their doctor.

Diagnosis

General: There is currently no specific test designed to diagnose autism. Instead, a diagnosis is made after the health care provider evaluates the patient's signs and symptoms. The health care provider will also talk with family members about their observations and interactions with the child. It may be helpful for the patient's family members to record observations of behavior that seems abnormal.

In some cases, the health care provider may order tests to rule out other conditions that may have similar symptoms, such as mental retardation, genetic diseases or deafness. If a health care provider cannot make a clear diagnosis, he/she may recommend other professionals who specialize in developmental disorders. Specialists, such as developmental pediatricians, psychiatrists, psychologists and neurologists, may be recommended. In addition, other specialists, such as experts who test hearing (audiologists), speech and language pathologists, occupational therapists, social workers and physical therapists, may also help diagnose the patient. Although most signs of autism begin to develop when the child is 12-18 months old, most diagnoses are made when the child is 2 to 3 years old.

Physical examination: During a physical examination, the health care provider will observe specific behaviors. The health care provider typically looks to see how the child responds to commands or questions.

Screening tests: Some health care providers use screening tests, including the checklist for autism in toddlers (CHAT) or the autism-screening questionnaire to determine whether a patient has autism. CHAT is a 16-question survey in which parents or caregivers respond "yes" or "no" to questions about their children's behavior. This test helps health care providers diagnose autism in patients who are 18 months old or younger. The autism-screening questionnaire, also called the pervasive development disorder (PDD) assessment scale, is a brief survey in which parents or caregivers rate the patient's developmental difficulties as nonexistent, resolved, mild, moderate or severe. This test helps health care providers diagnose autism in patients who are 4 years old or older.

Treatment

General: Currently, there is no cure for autism. However, many treatments and therapies may help patients cope with the disorder. With proper therapy, some patients are able to live independently once they become adults, while others may need lifelong support. Many different specialists, including speech or language pathologists, social workers, psychologists, psychiatrists and neurologists, may help parents and caregivers decide the best treatment options for autistic patients. It is important to note that different professionals will have different philosophies for the treatment of the patient. Parents or caregivers may want to meet with several specialists to find the one that meets the individual needs of both the caregiver(s) and the patient.

In general, the traditional approach to treating autism includes behavioral therapy and special education. Many different programs are available to help address the social, language and behavioral problems associated with autism.

Although no medication is specifically designed for autism, some patients may benefit from medications. However, medications do not treat the underlying cause of autism. Instead, they help treat the symptoms of the disorder. For instance, some patients may benefit from medications to help treat hyperactivity, short attention span and seizures, which are often associated with autism. Parents and caregivers should talk with the patient's health care providers about the potential side effects and benefits of medications before starting treatment.

- **Anticonvulsants**: Anticonvulsants are often used to treat seizures in autistic patients. These drugs may also help improve a patient's mood and/or behavior. These drugs are typically taken once daily to help prevent seizures from occurring. Phenobarbital (Luminal® Sodium) is one of the oldest and safest anticonvulsants for children. Valproic acid (Depakene® or Depakote®) has also been shown to be a safe and effective treatment for seizures in children.
- **Antidepressants:** Antidepressants may help improve depression, obsessive-compulsive disorder (OCD), and anxiety in some autistic patients. They may reduce repetitive behaviors, tantrums, aggression and irritability in patients. Drugs called selective serotonin reuptake inhibitors (SSRIs) are the most common type of antidepressants used. Commonly prescribed SSRIs include fluoxetine (Prozac®), fluvoxamine (Luvox®), sertraline (Zoloft®), and paroxetine (Paxil®). Less commonly prescribed antidepressants include clomipramine (Anafranil®), mirtazapine (Remeron®), amitriptyline (Elavil®), and bupropion (Wellbutrin®).
- **Antipsychotic drugs**: Antipsychotic drugs have been used to help treat aggressive and repetitive behaviors, as well as hyperactivity in autistic patients. Commonly prescribed drugs include risperidone (Risperdal®), olanzapine (Zyprexa®), and quetiapine (Seroquel®).

Behavioral therapy: The foundation of autism treatment is behavioral therapy. For more than 30 years, several different types of behavioral therapy have helped autistic patients improve their communication and social skills as well as their learning abilities and adaptive behaviors. Behavioral therapy has been shown to reduce inappropriate behavior, including aggressive behavior, in autistic children. Evidence suggests that behavioral therapy is most effective if it is started early in life, when the patient is 3 to 4 years old or younger.

Applied behavior analysis (ABA) is a type of therapy used to improve the patient's behavior and teach skills to help the person handle specific situations. The therapist uses positive reinforcement, which means the patient is rewarded when he/she behaves appropriately. ABA is highly structured, and it usually requires 15-40 hours of therapy per week. The therapist usually works one-on-one with the patient and collaborates with the parents/caregivers, teachers and others in the patient's life to provide treatment that is individualized to meet the patient's needs.

Providing oral care to people with autism

Providing oral care to people with autism requires adaptation of the skills you use every day. In fact, most people with mild or moderate forms of autism can be treated successfully in the general practice setting. This booklet will help you make a difference in the lives of people who need professional oral care.

Autism is a complex developmental disability that impairs communication and social, behavioral and intellectual functioning. Some people with the disorder appear distant, aloof, or detached from other people or from their surroundings. Others do not react appropriately to common verbal and social cues, such as a parent's tone of voice or smile. Obsessive routines, repetitive behaviors, unpredictable body movements and self-injurious behavior may all be symptoms that complicate dental care.

Autism varies widely in symptoms and severity, and some people have coexisting conditions such as intellectual disability or epilepsy. They can be among the most challenging of patients, but following the suggestions in this booklet can help make their dental treatment successful. Making a difference in the oral health of a person with autism may go slowly at first, but determination can bring positive results and invaluable rewards.

Health challenges in autism and strategies for care

Before the appointment, obtain and review the patient's medical history. Consultation with physicians, family and caregivers is essential to assembling an accurate medical history. Also, determine who can legally provide informed consent for treatment.

Communication problems and mental capabilities are central concerns when treating people with autism. Talk with the parent or caregiver to determine your patient's intellectual and functional abilities, and then communicate with the patient at a level he or she can understand. Use a "tell-show-do" approach to providing care. Start by explaining each procedure before it occurs. Take the time to show what you have explained, such as the instruments you will use and how they work. Demonstrations can encourage some patients to be more cooperative.

Behavior problems – which may include hyperactivity and quick frustration – can complicate oral health care for patients with autism. The invasive nature of oral care may trigger violent and self-injurious behavior, such as temper tantrums or head banging. Plan a desensitization appointment to help the patient become familiar with the office, staff and equipment through a step-by-step process. These steps may take several visits to accomplish.

Have the patient sit alone in the dental chair to become familiar with the treatment setting. Some patients may refuse to sit in the chair and choose instead to sit on the operator's stool.

Once your patient is seated, begin a cursory examination using your fingers.

Next, use a toothbrush to brush the teeth and gain additional access to the patient's mouth. The familiarity of a toothbrush will help your patient feel comfortable and provide you with an opportunity to further examine the mouth. When the patient is prepared for treatment, make the appointment short and positive. Pay special attention to the treatment setting. Keep dental instruments out of sight and light out of your patient's eyes.

Praise and reinforce good behavior after each step of a procedure. Ignore inappropriate behavior as much as you can. Try to gain cooperation in the least restrictive manner. Some patients' behavior may improve if they bring comfort items such as a stuffed animal or a blanket. Asking the caregiver to sit nearby or hold the patient's hand may be helpful as well. Use immobilization techniques only when absolutely necessary to protect the patient and staff during dental treatment – not as a convenience. There are no universal guidelines on immobilization that apply to all treatment settings.

Before employing any kind of immobilization, it may help to consult available guidelines on federally funded care, your state department of mental health disabilities and your state dental practice act.

Guidelines on behavior management published by the American Academy of Pediatric Dentistry (*www.aapd.org*) may also be useful. Obtain consent from your patient's legal guardian and choose the least restrictive technique that will allow you to provide care safely. Immobilization should not cause physical injury or undue discomfort.

If all other strategies fail, pharmacological options are useful in managing some patients. Others need to be treated under general anesthesia. However, caution is necessary because some patients with developmental disabilities can have unpredictable reactions to medications.

People with autism often engage in perseveration, a continuous, meaningless repetition of words, phrases or movements. Your patient may mimic the sound of the suction, for example, or repeat an instruction over and again. Avoid demonstrating dental equipment if it triggers perseveration, and note this in the patient's record.

Unusual responses to stimuli can create distractions and interrupt treatment. People with autism need consistency and can be especially sensitive to changes in their environment. They may exhibit unusual sensitivity to sensory stimuli such as sound, bright colors and touch. Reactions vary: Some people with autism may overreact to noise and touch, while exposure to pain and heat may not provoke much reaction at all. Use the same staff, dental operatory and appointment time to sustain familiarity. These details can help make dental treatment seem less threatening.

Minimize the number of distractions. Try to reduce unnecessary sights, sounds, odors or other stimuli that might be disruptive. Use an operatory that is somewhat secluded instead of one in the middle of a busy office. Also, consider lowering ambient light and asking the patient's caregiver whether soft music would help. Allow time for your patient to adjust and become desensitized to the noise of a dental setting. Some patients may be hypersensitive to the sound of dental instruments. Talk to the caregiver to get a sense of the patient's level of tolerance. People with autism differ in how they accept physical contact. Some are defensive and refuse any contact in or around the mouth, or cradling of the head or face. Others find such cradling comforting.

Note your findings and experiences in the patient's chart.

Unusual and unpredictable body movements are sometimes observed in people with autism. These movements can jeopardize safety as well as your ability to deliver oral health care. Make sure the path from the reception area to the dental chair is clear. Observe the patient's movements and look for patterns. Try to anticipate the movements, either blending your movements with those of your patient or working around them.

Seizures may accompany autism but can usually be controlled with anticonvulsant medications. The mouth is always at risk during a seizure: Patients may chip teeth or bite the tongue or cheeks. People with controlled seizure disorders can easily be treated in the general dental office:

- Consult your patient's physician.
- Record information in the chart about the frequency of seizures and the medications used to control them. Determine before the appointment whether medications have been taken as directed.
- Know and avoid any factors that trigger your patient's seizures.
- Be prepared to manage a seizure.
    - If one occurs during oral care, remove any instruments from the mouth and clear the area around the dental chair. Attaching dental floss to rubber dam clamps and mouth props when treatment begins can help you remove them quickly. Do not attempt to insert any objects between the teeth during a seizure.
    - Stay with your patient, turn him or her to one side and monitor the airway to reduce the risk of aspiration.

Record in the patient's chart strategies that were successful in providing care. Note your patient's preferences and other unique details that will facilitate treatment, such as music, comfort items and flavor choices.

Oral health problems in autism and strategies for care

People with autism experience few unusual oral health conditions. Although commonly used medications and damaging oral habits can cause problems, the rates of caries and periodontal disease in people with autism are comparable to those in the general population.

Communication and behavioral problems pose the most significant challenges in providing oral care.

Damaging oral habits are common and include bruxism; tongue thrusting; self-injurious behavior, such as picking at the gingiva or biting the lips; and pica – eating objects and substances such as gravel, cigarette butts or pens. If a mouth guard can be tolerated, prescribe one for patients who have problems with self-injurious behavior or bruxism.

Dental caries risk increases in patients who have a preference for soft, sticky or sweet foods; damaging oral habits; and difficulty brushing and flossing. Recommend preventive measures such as fluorides and sealants.

Caution patients or their caregivers about medicines that reduce saliva or contain sugar. Suggest that patients drink water often, take sugar-free medicines when available and rinse with water after taking any medicine.

Advise caregivers to offer alternatives to cariogenic foods and beverages as incentives or rewards.

Encourage independence in daily oral hygiene. Ask patients to show you how they brush, and follow up with specific recommendations. Perform hands-on demonstrations to show patients the best way to clean their teeth. If appropriate, show patients and caregivers how a modified toothbrush or floss holder might make oral hygiene easier. Some patients cannot brush and floss independently. Talk to caregivers about daily oral hygiene and do not assume that they know the basics. Use your experiences with each patient to demonstrate oral hygiene techniques and sitting or standing positions for the caregiver. Emphasize that a consistent approach to oral hygiene is important: Caregivers should try to use the same location, timing and positioning.

Periodontal disease occurs in people with autism in much the same way it does in persons without developmental disabilities. Some patients benefit from the daily use of an antimicrobial agent such as chlorhexidine. Stress the importance of conscientious oral hygiene and frequent prophylaxis. Tooth eruption may be delayed due to phenytoin-induced gingival hyperplasia. Phenytoin is commonly prescribed for people with autism.

Trauma and injury to the mouth from falls or accidents occur in people with seizure disorders. Suggest a tooth-saving kit for group homes. Emphasize to caregivers that traumas require immediate professional attention and explain the procedures to follow if a permanent tooth is knocked out. Also, instruct caregivers to locate any missing pieces of a fractured tooth, and explain that radiographs of the patient's chest may be necessary to determine whether any fragments have been aspirated.

Cerebral palsy

Cerebral palsy (CP) is an umbrella term encompassing a group of nonprogressive, motor, noncontagious conditions that cause physical disability in human development.

Cerebral refers to the cerebrum, which is the affected area of the brain (although the disorder most likely involves connections between the cortex and other parts of the brain such as the cerebellum), and palsy refers to disorder of movement. CP is caused by damage to the motor control centers of the developing brain and can occur during pregnancy (about 75 percent), during childbirth (about 5 percent) or after birth (about 15 percent) up to about age 3.

Cerebral palsy causes different types of disabilities in each child. A child may simply be a little clumsy or awkward, or unable to walk at all. Often the problem happens before birth or soon after being born. Further research is needed on adults with CP as the current literature is highly focused on the pediatric patient.

Cerebral palsy describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to nonprogressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication and behavior; by epilepsy, and by secondary musculoskeletal problems.

There is no known cure for CP. Medical intervention is limited to the treatment and prevention of complications arising from CP's effects. A 2003 study put the economic cost for CP sufferers in the U.S. at $921,000 per case, including lost income. In another study,

the incidence in six countries surveyed was 2.12 to 2.45 per 1,000 live births, indicating a slight rise in recent years. Improvements in neonnatal nursing have helped reduce the number of babies who develop cerebral palsy, but the survival of babies with very low birth weights has increased and these babies are more likely to have cerebral palsy.

CP is divided into three major classifications to describe different movement impairments. These classifications also reflect the areas of the brain that are damaged. The three major classifications are:

- **Spastic cerebral palsy** is by far the most common type, occurring in 70 percent to 80 percent of all cases. Moreover, spastic CP accompanies one of the other types in 30 percent of all cases. People with this type are hypertonic and have a neuromuscular condition stemming from damage to the corticospinal tract or the motor cortex that affects the nervous system's ability to receive gamma amino butyric acid in the area(s) affected by the disability. Spastic CP is further classified by topography dependent on the region of the body affected, which includes:
    - **Spastic hemiplegia** (one side being affected). Generally, injury to muscle-nerves controlled by the brain's left side will cause a right body deficit and vice versa. Typically, people who have spastic hemiplegia are the most ambulatory, although they generally have dynamic equinus on the affected side and are primarily prescribed ankle-foot orthoses to prevent the equinus.
    - **Spastic diplegia** (the lower extremities are affected with little to no upper-body spasticity), the most common form of the spastic forms. Most people with spastic diplegia are fully ambulatory and have a scissors gait. Flexed knees and hips to varying degrees are common. Hip problems, dislocations, and in three-quarters of spastic diplegics, also strabismus (crossed eyes), can be present as well. In addition, these individuals are often nearsighted. The intelligence of a person with spastic diplegia is unaffected by the condition.
    - **Spastic tetraplegia** (all four limbs affected equally). People with spastic quadriplegia are the least likely to be able to walk, or if they can, to want to walk, because they are too tight and it is too much effort to do so. Some children with quadriplegia also have hemiparetic tremors, an uncontrollable shaking that affects the limbs on one side of the body and impairs normal movement. Occasionally, terms such as monoplegia, paraplegia, triplegia and pentaplegia may also be used to refer to specific manifestations of the spasticity.
- **Ataxia** (ICD-10 G80.4) symptoms can be caused by damage to the cerebellum. Forms of ataxia are less common types of cerebral palsy, occurring in at most 10 percent of all cases. Some of these individuals have hypotonia and tremors. Motor skills like writing, typing or using scissors might be affected, as well as balance, especially while walking. It is common for individuals to have difficulty with visual and/or auditory processing of objects.
- **Athetoid or dyskinetic – mixed muscle tone – and sometimes hypotonia.** Hypotonia will usually occur before a child's first birthday; the muscle tone will be increased with age and progress to hypertonia. People with athetoid CP have trouble holding themselves in an upright, steady position for sitting or walking and often show involuntary motions. For some people with athetoid CP, it takes a lot of work and concentration to get their hand to a certain spot (like scratching the nose or reaching for a cup). Because of their mixed tone and trouble keeping a position, they may not be able to hold onto objects (such as a toothbrush or pencil). About one quarter of all people with CP have athetoid CP. The damage occurs to the extrapyramidal motor system and/or pyramidal tract and to the basal ganglia. It occurs in 40 percent of all cases. In newborn infants, high bilirubin levels in the blood, if left untreated, can lead to brain damage in certain areas. This may also lead to athetoid cerebral palsy.

Incidence and prevalence

In the industrialized world, the incidence of cerebral palsy is about two per 1,000 live births. The incidence is higher in males than in females in the United States, where approximately 10,000 infants and babies are diagnosed with CP each year, and 1,200 to 1,500 are diagnosed at preschool age. Overall, advances in care of pregnant mothers and their babies have not resulted in a noticeable decrease in CP. This is generally attributed to medical advances in areas related to the care of premature babies (which results in a greater survival rate). Only the introduction of quality medical care to locations with less-than-adequate medical care has shown any decreases. The incidence of CP increases with premature or very low-weight babies regardless of the quality of care. Prevalence of cerebral palsy is best calculated around the school entry age of about 6 years, and the prevalence in the U.S. is estimated to be 2.4 of 1,000 children. Apgar scores have sometimes been used as one factor to predict whether an individual will develop CP.

Signs and symptoms

All types of CP are characterized by abnormal muscle tone (i.e. slouching over while sitting), reflexes or motor development and coordination. There can be joint and bone deformities and contractures (permanently fixed, tight muscles and joints). The classical symptoms are spasticities, spasms, other involuntary movements (e.g. facial gestures), unsteady gait, problems with balance and soft tissue findings consisting largely of decreased muscle mass. Scissor walking (where the knees come in and cross) and toe walking (which can contribute to a gait reminiscent of a marionette) are common among people with CP who are able to walk, but taken on the whole, CP symptomatology is very diverse. The effects of cerebral palsy fall on a continuum of motor dysfunction which may range from virtually unnoticeable to "clumsy" and awkward movements on one end of the spectrum to such severe impairments that coordinated movements are almost impossible on the other end of the spectrum.

Babies born with severe CP often have an irregular posture; their bodies may be either very floppy or very stiff. Birth defects, such as spinal curvature, a small jawbone or a small head sometimes occur along with CP. Symptoms may appear, change or become more severe as a child gets older. Some babies born with CP do not show obvious signs right away. Classically, CP becomes evident when the baby reaches the developmental stage at 6 to 12 months and is starting to mobilize, where preferential use of limbs, asymmetry or gross motor developmental delay is seen.

Secondary conditions can include seizures, epilepsy, speech (apraxia or dysarthria) or communication disorders, eating problems, sensory impairments, mental retardation, learning disabilities, and/or behavioral disorders.

Speech and language disorders are common in people with cerebral palsy. Overall language delay is associated with problems of mental retardation, hearing impairment and learned helplessness. Children with cerebral palsy are at risk of learned helplessness and becoming passive communicators, initiating little communication. Early intervention with this clientele often targets situations in which children communicate with others, so that they learn that they can control people and objects in their environment through this communication, including making choices, decisions and mistakes.

What are some of the signs of cerebral palsy?

The signs of cerebral palsy vary greatly because there are many different types and levels of disability. The main sign that a child might have cerebral palsy is a delay reaching the motor or movement milestones. Parents who see any of these signs should call the child's doctor or nurse.

A child over 2 months with cerebral palsy might:
- Have difficulty controlling his or her head when picked up.
- Have stiff legs that cross or "scissor" when picked up.

A child over 6 months with cerebral palsy might:
- Continue to have a hard time controlling his or her head when picked up.
- Reach with only one hand while keeping the other in a fist.

A child over 10 months with cerebral palsy might:
- Crawl by pushing off with one hand and leg while dragging the opposite hand and leg.
- Not sit by himself or herself.

A child over 12 months with cerebral palsy might:
- Not crawl.
- Not be able to stand with support.

A child over 24 months with cerebral palsy might:
- Not be able to walk.
- Not be able to push a toy with wheels.

Causes

Despite years of debate, the cause of the majority of cases of CP is uncertain. Cerebral palsy is caused by a problem in the brain that affects a child's ability to control his or her muscles. Problems in different parts of the brain cause problems in different parts of the body. There are many possible causes of problems, such as genetic conditions, problems with the blood supply to the brain before birth, infections, bleeding in the brain, lack of oxygen, severe jaundice and head injury. While in certain cases there is no identifiable cause, other etiologies include problems in intrauterine development (e.g. exposure to radiation, infection), asphyxia before birth, hypoxia of the brain, birth trauma during labor and delivery, and complications in the perinatal period or during childhood.

CP is also more common in multiple births. Studies at the University of Liverpool have led to the hypothesis that many cases of cerebral palsy, and other conditions that an infant has at birth, are caused by the death in very early pregnancy of an identical twin. This may occur when twins have a joint circulation through sharing the same placenta. Not all identical twins share the same blood supply (monochorionic twins), but if they do, the suggestion is that perturbations in blood flow between them can cause the death of one and damage to the development of the surviving fetus.

It is common knowledge among obstetricians and midwives that a small dead fetus (fetus papyraceus) may sometimes be found attached to a placenta following birth. In the past, this has not been considered important, and knowledge of the so-called "vanishing twin" has been suppressed to avoid triggering feelings of loss, grief or guilt in mothers.

The pathological consequences depend on the severity and the stage of development of the fetus when the imbalances in blood flow between the fetuses occur. It has been proposed that such pathology could account not just for cerebral palsy, but also for developmental abnormalities of the eye, heart and gut, and other specific brain abnormalities such as neuronal migration disorders, e.g. lissencephaly and holoprosencephaly, which occur during very early fetal development.

Between 40 percent and 50 percent of all children who develop cerebral palsy were born prematurely. Premature infants are vulnerable, in part because their organs are not fully developed, increasing the risk of hypoxic injury to the brain that may manifest as CP. A problem in interpreting this is the difficulty in differentiating between CP caused by damage to the brain that results from inadequate oxygenation and CP that arises from prenatal brain damage that then precipitates premature delivery.

Recent research has demonstrated that intrapartum asphyxia is not the most important cause, probably accounting for no more than 10 percent of all cases; rather, infections in the mother, even infections that are not easily detected, may triple the risk of the child developing the disorder, mainly as the result of the toxicity to the fetal brain of cytokines that are produced as part of the inflammatory response. Low birth weight is a risk factor for CP – and premature infants usually have low birth weights, less than 2.0 kg, but full-term infants can also have low birth weights.

Multiple-birth infants are also more likely than single-birth infants to be born early or with a low birth weight.

After birth, other causes include toxins, severe jaundice, lead poisoning, physical brain injury, shaken baby syndrome, incidents involving hypoxia to the brain (such as near drowning), and encephalitis or meningitis. The three most common causes of asphyxia in the young child are:
- Choking on foreign objects such as toys and pieces of food.
- Poisoning.
- Near drowning.

Some structural brain anomalies such as lissencephaly may present with the clinical features of CP, although whether that could be considered CP is a matter of opinion (some people say CP must be due to brain damage, whereas people with these anomalies didn't have a normal brain). Often this goes along with rare chromosome disorders, and CP is not genetic or hereditary.

Diagnosis

The diagnosis of cerebral palsy has historically rested on the patient's history and physical examination. Once diagnosed with cerebral palsy, further diagnostic tests are optional. The American Academy of Neurology published an article in 2004 reviewing the literature and evidence available on CT and MRI imaging. It suggested that neuroimaging with CT or MRI is warranted when the etiology of a patient's cerebral palsy has not been established – and an MRI is preferred over CT due to diagnostic yield and safety. When abnormal, the neuroimaging study can suggest the timing of the initial damage. The CT or MRI is also capable of revealing treatable conditions, such as hydrocephalus, porencephaly, arteriovenous malformation, subdural hematomas and hygromas, and a vermian tumor. Furthermore, an abnormal neuroimaging study indicates a high likelihood of associated conditions, such as epilepsy and mental retardation.

Presentation: Bones

In order for bones to attain their normal shape and size, they require the stresses from normal musculature. Osseous findings will therefore mirror the specific muscular deficits in a given person with CP. The shafts of the bones are often thin (gracile). When compared to these

thin shafts (diaphyses), the metaphyses often appear quite enlarged (ballooning). With lack of use, articular cartilage may atrophy, leading to narrowed joint spaces. Depending on the degree of spasticity, a person with CP may exhibit a variety of angular joint deformities. Because vertebral bodies need vertical gravitational loading forces to develop properly, spasticity and an abnormal gait can hinder proper and full bone and skeletal development. People with CP tend to be shorter in height than the average person because their bones are not allowed to grow to their full potential. Sometimes bones grow to different lengths, so the person may have one leg longer than the other.

Prognosis

CP is not a progressive disorder (meaning the actual brain damage neither improves nor worsens), but the symptoms can become worse over time due to subdural damage. A person with the disorder may improve somewhat during childhood if he or she receives extensive care from specialists, but once bones and musculature become more established, orthopedic surgery may be required for fundamental improvement. People who have CP tend to develop arthritis at a younger age than normal because of the pressure placed on joints by excessively toned and stiff muscles.

The full intellectual potential of a child born with CP will often not be known until the child starts school. People with CP are more likely to have some type of learning disability, but this is not related to a person's intellect or IQ level. Intellectual level among people with CP varies from genius to mentally retarded, as it does in the general population, and experts have stated that it is important to not underestimate a person with CP's capabilities and to give them every opportunity to learn.

The ability to live independently with CP also varies greatly depending on the severity of the disability. Some individuals with CP will require personal assistant services for all activities of daily living. Others can live semi-independently, needing support only for certain activities. Still others can live in complete independence. The need for personal assistance often changes with increasing age and associated functional decline. However, in most cases, persons with CP can expect to have a normal life expectancy; survival has been shown to be associated with the ability to ambulate, roll and self-feed. As the condition does not directly affect reproductive function, some persons with CP have children and parent successfully.

Providing oral care to people with cerebral palsy

Providing oral care to people with cerebral palsy requires adaptation of the skills you use every day. In fact, most people with mild or moderate forms of cerebral palsy can be treated successfully in the general practice setting. Cerebral palsy is a complex group of motor abnormalities and functional impairments that affect muscle coordination. This developmental disability may be associated with uncontrolled body movements, seizure disorders, balance-related abnormalities, sensory dysfunction and intellectual disability. For some, the disorder is mild, causing movements to appear merely clumsy or awkward. These patients may need little or no day-to-day supervision. Others, however, experience such severe forms of cerebral palsy that they require a wheelchair and a lifetime of personal care.

Cerebral palsy itself does not cause any unique oral abnormalities. However, several conditions are more common or more severe in people with cerebral palsy than in the general population.

Health challenges in cerebral palsy and strategies for care

People with cerebral palsy may present with physical and mental challenges that have implications for oral care. Before the appointment, obtain and review the patient's medical history.

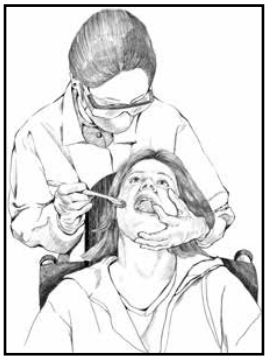
Consultation with physicians, family and caregivers is essential to assembling an accurate medical history. Also, determine who can legally provide informed consent for treatment.

The different types of cerebral palsy classified above according to associated motor impairments are:

- **Spastic palsy**, which presents with stiff or rigid muscles on one side of the body or in all four limbs, sometimes including the mouth, tongue and pharynx. People with this form of cerebral palsy may have legs that turn inward and scissor as they walk, or arms that are flexed and positioned against their bodies. Many also have intellectual disability, seizures and dysarthria (difficulty speaking).
- **Dyskinetic or athetoid palsy** is characterized by hypotonia and slow, uncontrolled writhing movements. People with this type of cerebral palsy experience frequent changes in muscle tone in all areas of their bodies; muscles may be rigid during waking hours and normal during sleep. Dysarthria is also associated with this type.
- **Ataxic palsy** is marked by problems with balance and depth perception, as well as an unsteady, wide-based gait. Hypotonia and tremors sometimes occur in people with this rare type of cerebral palsy.
- **Combined palsy** reflects a combination of these types. Everyone who has cerebral palsy has problems with movement and posture. Observe each patient, and then tailor your care accordingly. Maintain clear paths for movement and keep instruments and equipment out of the patient's way.

Some patients cannot be moved into the dental chair but instead must be treated in their wheelchairs. Some wheelchairs recline or are specially molded to fit people's bodies. Lock the wheels, and then slip a sliding board (also called a transfer board) behind the patient's back to support the head and neck. If you need to transfer your patient from a wheelchair to the dental chair, ask about special preferences such as padding, pillows or other things you can provide to ease the transition. The patient or caregiver can often explain how to make a smooth transfer.

Uncontrolled body movements are common in people with cerebral palsy. Their limbs move often, so providing oral care can be difficult. When patients with cerebral palsy attempt to move in order to help, their muscles often tense, increasing uncontrolled movements.



Positioning for treating a patient in a wheelchair. Note the support a sliding board can provide.

Make the treatment environment calm and supportive. Try to help your patient relax. Relaxation will not stop uncontrolled body movements, but it may reduce their frequency or intensity.

Place and maintain your patient in the center of the dental chair. Do not force arms and legs into unnatural positions, but allow the patient to settle into a position that is comfortable and will not interfere with dental treatment.

Observe your patient's movements and look for patterns to help you anticipate direction and intensity. Trying to stop these movements may only intensify the involuntary response. Try instead to anticipate the movements, blending your movements with those of your patient or working around them.

Softly cradle your patient's head during treatment. Be gentle and slow if you need to turn the patient's head. Exert gentle but firm pressure on your patient's arm or leg if it begins to shake.

Try to keep appointments short, take frequent breaks, and consider prescribing muscle relaxants when long procedures are needed.

People with cerebral palsy may need sedation, general anesthesia or hospitalization if extensive dental treatment is required.

Primitive reflexes are common in many people with cerebral palsy and may complicate oral care. These reflexes often occur when the head is moved or the patient is startled, and efforts to control them may make them more intense. Three types of reflexes are most commonly observed during oral care.

- **Asymmetric tonic neck reflex:** When a patient's head is turned, the arm and leg on that side stiffen and extend. The arm and leg on the opposite side flex.
- **Tonic labyrinthine reflex:** If the neck is extended while a patient is lying on his or her back, the legs and arms also extend, and the back and neck arch.
- **Startle reflex:** Any surprising stimuli, such as noises, lights, or a sudden movement on your part, can trigger uncontrolled, often forceful movements involving the whole body.

Be empathic about your patient's concerns and frustrations.

Minimize the number of distractions in the treatment setting.

Movements, lights, sounds or other stimuli can make it difficult for your patient to cooperate. Tell him or her about any such stimulus before it appears. For example, tell the patient before you move the dental chair.

Mental capabilities vary. Many people with cerebral palsy have mild or moderate intellectual disability, but only 25 percent have a severe form. Some have normal intelligence.

Talk with the parent or caregiver to determine your patient's intellectual and functional abilities, and then explain each procedure at a level the patient can understand. Allow extra time to explain oral health issues, instructions or procedures. Use simple, concrete instructions and repeat them often to compensate for any short-term memory problems. Speak slowly and give only one direction at a time.

Demonstrations can make patients more cooperative. For example, turn on the saliva ejector so the patient can hear it and feel it at the corner of the mouth. Then slowly introduce it inside the mouth, being careful not to trigger a gag reflex. Be consistent in all aspects of oral care. Use the same staff and dental operatory each time to help sustain familiarity. Consistency leads to improved cooperation.

Listen actively because communicating clearly is difficult for some; show your patient whether you understand. Be sensitive to the methods he or she uses to communicate, including gestures and verbal or nonverbal requests.

Seizures may accompany cerebral palsy, but can usually be controlled with anticonvulsant medications. The mouth is always at risk during a seizure: Patients may chip teeth or bite the tongue or cheeks. Patients with controlled seizure disorders can easily be treated in the general dental office.

- Consult your patient's physician.
- Record information in the chart about the frequency of seizures and the medications used to control them. Determine before the appointment whether medications have been taken as directed.
- Know and avoid any factors that trigger your patient's seizures.
- Be prepared to manage a seizure.
    - If one occurs during oral care, remove any instruments from the mouth and clear the area around the dental chair. Attaching dental floss to rubber dam clamps and mouth props when treatment begins can help you remove them quickly.
    - Do not attempt to insert any objects between the teeth during a seizure.
    - Stay with your patient, turn him or her to one side and monitor the airway to reduce the risk of aspiration.

Visual impairments affect a large number of people with cerebral palsy. The most common of these defects is strabismus, a condition in which the eyes are crossed or misaligned. People with cerebral palsy may develop visual motor skills, such as hand-eye coordination, later than other people.

Determine the level of assistance your patient requires to move safely through the dental office. Use your patients' other senses to connect with them, establish trust and make treatment a good experience. Tactile feedback, such as a warm handshake, can make your patients feel comfortable. Face your patients when you speak and keep them apprised of each upcoming step, especially when water will be used. Rely on clear, descriptive language to explain procedures and demonstrate how equipment might feel and sound.

Provide written instructions in large print (16 point or larger).

Hearing loss and deafness can be accommodated with careful planning. Patients with a hearing problem may appear to be stubborn because they don't seem to respond to a request. Patients may want to adjust their hearing aids or turn them off, since the sound of some instruments may cause auditory discomfort. If your patient reads lips, speak in a normal cadence and tone. If your patient uses a form of sign language, ask the interpreter to come to the appointment. Speak with this person in advance to discuss dental terms and your patient's needs.

Visual feedback is helpful. Maintain eye contact with your patient.

Before talking, eliminate background noise (turn off the radio and the suction). Sometimes people with a hearing loss simply need you to speak clearly in a slightly louder voice than normal. Remember to remove your facemask first or wear a clear face shield.

Dysarthria is common in people with cerebral palsy, due to problems involving the muscles that control speech and mastication.

Be patient. Allow time for your patient to express himself or herself. Remember that many people with dysarthria have normal intelligence. Consult with the caregiver if you have difficulty understanding your patient's speech.

Gastroesophageal reflux sometimes affects people with cerebral palsy, including those who are tube-fed. Teeth may be sensitive or display signs of erosion. Consult your patient's physician about the management of reflux.

Place patients in a slightly upright position for treatment. Talk with patients and caregivers about rinsing with plain water or a water and baking soda solution. Doing so at least four times a day can help

mitigate the effects of gastric acid. Stress that using a fluoride gel, rinse or toothpaste every day is essential.

Record in the patient's chart strategies that were successful in providing care. Note your patient's preferences and other unique details that will facilitate treatment, such as music, comfort items and flavor choices.

Oral health problems in cerebral palsy and strategies for care.

Cerebral palsy itself does not cause any unique oral abnormalities. However, several conditions are more common or more severe in people with cerebral palsy than in the general population.

Peridontal disease is common in people with cerebral palsy due to poor oral hygiene and complications of oral habits, physical abilities and malocclusion. Another factor is the gingival hyperplasia caused by medications.

Encourage independence in daily oral hygiene. Ask patients to show you how they brush, and follow up with specific recommendations on brushing methods or toothbrush adaptations. Involve your patients in hands-on demonstrations of brushing and flossing. Some patients cannot brush and floss independently because of impaired physical coordination or cognitive skills. Talk to caregivers about daily oral hygiene. Do not assume that all caregivers know the basics; demonstrate proper brushing and flossing techniques. A power toothbrush or a floss holder can simplify oral care. Also, use your experiences with each patient to demonstrate sitting or standing positions for the caregiver. Emphasize that a consistent approach to oral hygiene is important, and caregivers should try to use the same location, timing and positioning.

Explain that some patients benefit from the daily use of an antimicrobial agent such as chlorhexidine. Recommend an appropriate delivery method based on your patient's abilities. Rinsing, for example, may not work for a patient with swallowing difficulties or one who cannot expectorate. Chlorhexidine applied using a spray bottle or toothbrush is equally efficacious. If use of particular medications has led to gingival hyperplasia, monitor for possible delayed tooth eruption and emphasize the importance of daily oral hygiene and frequent professional cleanings.

Dental caries is prevalent among people with cerebral palsy, primarily because of inadequate oral hygiene. Other risk factors include mouth breathing, the effects of medication, enamel hypoplasia and food pouching.

Caution patients or their caregivers about medicines that reduce saliva or contain sugar. Suggest that patients drink water often, take sugar-free medicines when available and rinse with water after taking any medicine.

Advise caregivers to offer alternatives to cariogenic foods and beverages as incentives or rewards.

For people who pouch food, talk to caregivers about inspecting the mouth after each meal or dose of medicine. Remove food or medicine from the mouth by rinsing with water, sweeping the mouth with a finger wrapped in gauze or using a disposable foam applicator swab. Recommend preventive measures such as fluorides and sealants.

Malocclusion in people with cerebral palsy usually involves more than just misaligned teeth; it is also a musculoskeletal problem. An open bite with protruding anterior teeth is common, and is typically associated with tongue thrusting. The inability to close the lips because of an open bite also contributes to excessive drooling.

Unfortunately, correcting malocclusion is almost impossible in people with moderate or severe cerebral palsy. Orthodontic treatment may not be an option because of the risk of caries and enamel hypoplasia. However, a developmental disability in and of itself should not be perceived as a barrier to orthodontic treatment.

The ability of the patient or the caregiver to maintain good daily oral hygiene is critical to the feasibility and success of orthodontic treatment. Inform caregivers of emergency procedures for accidents involving oral trauma, since protruding anterior teeth are more likely to be displaced, fractured or avulsed.

Dysphagia, difficulty with swallowing, is often a problem in people with cerebral palsy. Food may stay in the mouth longer than usual, increasing the risk for caries. Additionally, the semi-soft foods caregivers may prepare for people with this problem tend to adhere to the teeth. Coughing, gagging, choking and aspiration are other related concerns. Keep the breathing passages open by placing your patient in a slightly upright position with the head turned to one side during oral care. Use suction frequently or as tolerated by the patient.

Use a rubber dam when indicated, but make sure you introduce it slowly, perhaps over a few appointments. Advise the caregiver to inspect the patient's mouth after eating and remove any residual food.

Drooling affects daily oral care as well as social interaction. Hypotonia contributes to drooling, as does an open bite and the inability to close the lips.

Bruxism is common in people with cerebral palsy, especially those with severe forms of the disorder. Bruxism can be intense and persistent and cause the teeth to wear prematurely. Before recommending mouth guards or bite splints, consider that gagging or swallowing problems may make them uncomfortable or unwearable.

Hyperactive bite and gag reflexes call for introducing instruments gently into the mouth. Consider using a mouth prop. A patient with a gagging problem benefits from an early morning appointment, before eating or drinking. Help minimize the gag reflex by placing your patient's chin in a neutral or downward position.

Trauma and injury to the mouth from falls or accidents occur in people with cerebral palsy. Suggest a tooth-saving kit for group homes. Emphasize to caregivers that traumas require immediate professional attention and explain the procedures to follow if a permanent tooth is knocked out. Also, instruct caregivers to locate any missing pieces of a fractured tooth, and explain that radiographs of the patient's chest may be necessary to determine whether any fragments have been aspirated.

Down syndrome

Down syndrome trisomy 21 or trisomy G is a chromosomal disorder caused by the presence of all or part of an extra 21st chromosome. It is named after John Langdon Down, the British doctor who described the syndrome in 1866. The disorder was identified as a chromosome 21 trisomy by Jérôme Lejeune in 1959. The condition is characterized by a combination of major and minor differences in structure. Often Down syndrome is associated with some impairment of cognitive ability and physical growth as well as facial appearance. Down syndrome in a baby can be identified with amniocentesis during pregnancy or at birth.

Individuals with Down syndrome tend to have a lower than average cognitive ability, often ranging from mild to moderate developmental disabilities. A small number have severe to profound mental disability. The incidence of Down syndrome is estimated at one per 800 to 1,000 births, although these statistics are heavily influenced by older mothers. Other factors may also play a role.

Many of the common physical features of Down syndrome may also appear in people with a standard set of chromosomes, including microgenia (an abnormally small chin) and unusually round face; macroglossia (protruding or oversized tongue); an almond-shape to the eyes caused by an epicanthic fold of the eyelid; upslanting palpebral fissures (the separation between the upper and lower eyelids); shorter limbs; a single transverse palmar crease (a single instead of a double crease across one or both palms, also called the simian crease); poor muscle tone; and a larger than normal space between the big and second toes. Health concerns for individuals with Down syndrome include a higher risk for congenital heart defects, gastroesophageal reflux disease, recurrent ear infections, obstructive sleep apnea and thyroid dysfunctions.

Some common physical signs of Down syndrome include:

- A flat face with an upward slant to the eye, a short neck, small ears and a large tongue.
- Tiny white spots on the iris (colored part) of the eye.
- Small hands and feet.
- A single crease across the palm of the hand.
- Small pinky fingers that sometimes curve toward the thumb.
- Poor muscle tone or loose ligaments.

Most individuals with Down syndrome have mental retardation in the mild (IQ 50 to 70) to moderate (IQ 35 to 50) range, with individuals having mosaic Down syndrome typically 10 to 30 points higher. In addition, individuals with Down syndrome can have serious abnormalities affecting any body system. They also may have a broad head and a very round face.

The medical consequences of the extra genetic material in Down syndrome are highly variable and may affect the function of any organ system or bodily process. The health aspects of Down syndrome encompass anticipating and preventing effects of the condition, recognizing complications of the disorder, managing individual symptoms and assisting the individual and his/her family in coping and thriving with any related disability or illnesses.

Down syndrome can result from several different genetic mechanisms. This results in a wide variability in individual symptoms due to complex gene and environment interactions. Prior to birth, it is not possible to predict the symptoms that an individual with Down syndrome will develop. Some problems are present at birth, such as certain heart malformations. Others become apparent over time, such as epilepsy.

The most common manifestations of Down syndrome are the characteristic facial features, cognitive impairment, congenital heart disease (typically a ventricular septal defect), hearing deficits (maybe due to sensory-neural factors, or chronic serous otitis media, also known as Glue-ear), short stature, thyroid disorders, and Alzheimer's disease. Other less common serious illnesses include leukemia, immune deficiencies and epilepsy.

However, health benefits of Down syndrome include greatly reduced incidence of many common malignancies except leukemia and testicular cancer – although it is, as yet, unclear whether the reduced incidence of various fatal cancers among people with Down syndrome is as a direct result of tumor-suppressor genes on chromosome 21 (such as Ets2), because of reduced exposure to environmental factors that contribute to cancer risk, or some other as-yet unspecified factor. In addition to a reduced risk of most kinds of cancer, people with Down syndrome also have a much lower risk of hardening of the arteries and diabetic retinopathy.

Cognitive development

Cognitive development in children with Down syndrome is quite variable. It is not currently possible at birth to predict the capabilities of any individual reliably, nor is the number or appearance of physical features predictive of future ability.

The identification of the best methods of teaching each particular child ideally begins soon after birth through early intervention programs. Since children with Down syndrome have a wide range of abilities, success at school can vary greatly, which underlines the importance of evaluating children individually. The cognitive problems that are found among children with Down syndrome can also be found among typical children. Therefore, parents can use general programs that are offered through the schools or other means.

Language skills show a difference between understanding speech and expressing speech, and commonly individuals with Down syndrome have a speech delay, requiring speech therapy to improve expressive language. Fine motor skills are delayed and often lag behind gross motor skills and can interfere with cognitive development. Effects of the disorder on the development of gross motor skills are quite variable. Some children will begin walking at around age 2, while others will not walk until age 4. Physical therapy, or participation in a program of adapted physical education (APE), may promote enhanced development of gross motor skills in Down syndrome children.

Individuals with Down syndrome differ considerably in their language and communication skills. It is routine to screen for middle ear problems and hearing loss; low gain hearing aids or other amplification devices can be useful for language learning. Early communication intervention fosters linguistic skills. Language assessments can help profile strengths and weaknesses; for example, it is common for receptive language skills to exceed expressive skills. Individualized speech therapy can target specific speech errors, increase speech intelligibility and in some cases encourage advanced language and literacy. Augmentative and alternative communication (AAC) methods, such as pointing, body language, objects or graphics are often used to aid communication. Relatively little research has focused on the effectiveness of communications intervention strategies.

In education, mainstreaming of children with Down syndrome is becoming less controversial in many countries. For example, there is a presumption of mainstream in many parts of the UK. Mainstreaming is the process whereby students of differing abilities are placed in classes with their chronological peers. Children with Down syndrome may not age emotionally/socially and intellectually at the same rates as children without Down syndrome, so over time the intellectual and emotional gap between children with and without Down syndrome may widen. Complex thinking as required in sciences but also in history, the arts, and other subjects can often be beyond the abilities of some, or achieved much later than in other children. Therefore, children with Down syndrome may benefit from mainstreaming provided that some adjustments are made to the curriculum.

Some European countries such as Germany and Denmark advise a two-teacher system, whereby the second teacher takes over a group of children with disabilities within the class. A popular alternative is cooperation between special schools and mainstream schools. In cooperation, the core subjects are taught in separate classes, which neither slows down the typical students nor neglects the students with disabilities. Social activities, outings, and many sports and arts activities are performed together, as are all breaks and meals.

Fertility

Fertility among both males and females is reduced; males are usually unable to father children, while females demonstrate significantly lower rates of conception relative to unaffected individuals. Approximately half of the offspring of someone with Down syndrome

also have the syndrome themselves. There have been only three recorded instances of males with Down syndrome fathering children.

Down syndrome is a chromosomal abnormality characterized by the presence of an extra copy of genetic material on the 21st chromosome, either in whole (trisomy 21) or part (such as due to translocations). The effects of the extra copy vary greatly among people, depending on the extent of the extra copy, genetic history and pure chance. Down syndrome occurs in all human populations, and analogous effects have been found in other species such as chimpanzees and mice. Recently, researchers have created transgenic mice with most of human chromosome 21 (in addition to the normal mouse chromosomes). The extra chromosomal material can come about in several distinct ways. A typical human karyotype is designated as 46,XX or 46,XY, indicating 46 chromosomes with an XX arrangement typical of females and 46 chromosomes with an XY arrangement typical of males.

Trisomy 21

Trisomy 21 (47,XX,+21) is caused by a meiotic nondisjunction event. With nondisjunction, a gamete (i.e., a sperm or egg cell) is produced with an extra copy of chromosome 21; the gamete thus has 24 chromosomes. When combined with a normal gamete from the other parent, the embryo now has 47 chromosomes, with three copies of chromosome 21. Trisomy 21 is the cause of approximately 95 percent of observed Down syndromes, with 88 percent coming from nondisjunction in the maternal gamete and 8 percent coming from nondisjunction in the paternal gamete.

Mosaicism

Trisomy 21 is usually caused by nondisjunction in the gametes prior to conception, and all cells in the body are affected. However, when some of the cells in the body are normal and other cells have trisomy 21, it is called mosaic Down syndrome (46,XX/47,XX,+21). This can occur in one of two ways: a nondisjunction event during an early cell division in a normal embryo leads to a fraction of the cells with trisomy 21; or a Down syndrome embryo undergoes nondisjunction and some of the cells in the embryo revert to the normal chromosomal arrangement. There is considerable variability in the fraction of trisomy 21, both as a whole and among tissues. This is the cause of 1 to 2 percent of observed Down syndromes.

Robertsonian translocation

The extra chromosome 21 material that causes Down syndrome may be due to a Robertsonian translocation in the karyotype of one of the parents. In this case, the long arm of chromosome 21 is attached to another chromosome, often chromosome 14 (45,XX, t(14;21q)) or itself (called an isochromosome, 45,XX, t(21q;21q)). A person with such a translocation is phenotypically normal. During reproduction, normal disjunctions leading to gametes have a significant chance of creating a gamete with an extra chromosome 21, producing a child with Down syndrome. Translocation Down syndrome is often referred to as familial Down syndrome. It is the cause of 2 to 3 percent of observed cases of Down syndrome. It does not show the maternal age effect, and is just as likely to have come from fathers as mothers.

Rarely, a region of chromosome 21 will undergo a duplication event. This will lead to extra copies of some, but not all, of the genes on chromosome 21 (46,XX, dup(21q)). If the duplicated region has genes that are responsible for Down syndrome physical and mental characteristics, such individuals will show those characteristics. This cause is very rare and no rate estimates are available.

Pregnant women can be screened for various complications during pregnancy. Many standard prenatal screens can discover Down syndrome. Genetic counseling along with genetic testing, such as amniocentesis, chorionic villus sampling (CVS), or percutaneous umbilical cord blood sampling (PUBS) are usually offered to families who may have an increased chance of having a child with Down syndrome, or where normal prenatal exams indicate possible problems. In the United States, ACOG guidelines recommend that noninvasive screening and invasive testing be offered to all women, regardless of their age, and most likely all physicians currently follow these guidelines. However, some insurance plans will only reimburse invasive testing if a woman is over 34 years old or if she has received a high-risk score from a noninvasive screening test.

Amniocentesis and CVS are considered invasive procedures, in that they involve inserting instruments into the uterus, and therefore carry a small risk of causing fetal injury or miscarriage. The risks of miscarriage for CVS and amniocentesis are often quoted as 1 percent and 0.5 percent respectively. There are several common noninvasive screens that can indicate a fetus with Down syndrome. These are normally performed in the late first trimester or early second trimester. Due to the nature of screens, each has a significant chance of a false positive, suggesting a fetus with Down syndrome when, in fact, the fetus does not have this genetic abnormality. Screen positives must be verified before a Down syndrome diagnosis is made. Common screening procedures for Down syndrome are given in Table 1.

Even with the best noninvasive screens, the detection rate is 90 percent to 95 percent, and the rate of false positive is 2 percent to 5 percent. Inaccuracies can be caused by undetected multiple fetuses (very rare with the ultrasound tests), incorrect date of pregnancy or normal variation in the proteins.

Confirmation of screen positive is normally accomplished with amniocentesis or chorionic villus sampling (CVS). Amniocentesis is an invasive procedure and involves taking amniotic fluid from the amniotic sac and identifying fetal cells. The lab work can take several weeks but will detect over 99.8 percent of all numerical chromosomal problems with a very low false positive rate.

Management

Treatment of individuals with Down syndrome depends on the particular manifestations of the disease. For instance, individuals with congenital heart disease may need to undergo major corrective surgery soon after birth. Other individuals may have relatively minor health problems requiring no therapy.

How often does Down syndrome occur?

Centers for Disease Control (CDC) estimates that each year about 3,357 babies in the United States are born with Down syndrome. In other words, about 13 of every 10,000 babies born in the United States each year is born with Down syndrome.

What problems do children with Down syndrome have?

Babies and adults with Down syndrome can have physical problems as well as intellectual disabilities. Every baby born with Down syndrome is different. In addition to the physical signs, some might have major birth defects or other medical problems. However, many people with Down syndrome live happy, productive lives well into adulthood.

Still, some physical problems associated with Down syndrome include:

- A birth defect of the heart.
- Stomach problems, such as a blocked small intestine.
- Celiac disease, a digestive disease that damages the small intestine so that nutrients from food are not absorbed well.
- Problems with memory, concentration and judgment, often called dementia.
- Hearing problems.
- Eye problems, such as cataracts or trouble seeing objects that are close by (far-sighted).
- Thyroid problems.
- Skeletal problems.

A person with Down syndrome can have an IQ in the mild-to-moderate range of mental retardation. He or she also might have delayed language development and difficulties with physical coordination.

Risk factors

Centers for Disease Control works with many researchers to study the risk factors that can increase the chance of having a baby with Down syndrome. Following are examples of what this research has found:

- The number of babies with Down syndrome seems to be increasing, especially among mothers older than 35 years of age.
- Certain factors seem to influence how long a person with Down syndrome will live, including ethnicity, low weight at birth, and whether the baby was born with a heart defect.
- Death rates among black or African-American infants with Down syndrome seem to be higher than death rates among white infants with Down syndrome.

Can Down syndrome be prevented?

There is no known way to prevent the Down syndrome. However, infants and children with Down syndrome often will benefit from special programs that help to improve their physical and mental limitations. These include speech therapy, occupational therapy and exercises for physical coordination. Children with Down syndrome usually also need extra help or attention in school.

While there is currently no way to prevent Down syndrome, mothers can take steps before and during pregnancy to have a healthy pregnancy. Steps include taking a daily multivitamin with folic acid (400 micrograms), not smoking and not drinking alcohol during pregnancy.

Early childhood intervention, screening for common problems, medical treatment where indicated, a conducive family environment, and vocational training can improve the overall development of children with Down syndrome. Although some of the physical genetic limitations of Down syndrome cannot be overcome, education and proper care will improve quality of life.

Providing oral care to people with Down syndrome

Providing oral care to people with Down syndrome requires adaptation of the skills you use every day. In fact, most people with mild or moderate Down syndrome can be successfully treated in the general practice setting. Down syndrome, a common genetic disorder, ranges in severity and is usually associated with medical and physical problems. For example, people with this developmental disability may have cardiac disorders, infectious diseases, hypotonia and hearing loss. Additionally, most people with this disorder have mild or moderate mental retardation, while small percentages are severely affected. Developmental delays, such as in speech and language, are common. Early professional treatment and daily oral care at home can allow people with Down syndrome to enjoy the benefits of a healthy mouth.

Health challenges in Down syndrome and strategies for care

People with Down syndrome may present with mental and physical challenges that have implications for oral care. Before the appointment, obtain and review the patient's medical history. Consultation with physicians, family and caregivers is essential to assembling an accurate medical history. Also, determine who can legally provide informed consent for treatment.

Intellectual disability and Down syndrome

Although the intellectual ability of people with Down syndrome varies widely, many have mild or moderate mental retardation that limits their ability to learn, communicate and adapt to their environment. Language development is often delayed or impaired in people with Down syndrome; they understand more than they can verbalize. Also, ordinary activities of daily living and understanding the behavior of others as well as their own can present challenges.

Listen actively, since speaking may be difficult for people with Down syndrome. Show your patient whether you understand. Talk with the parent or caregiver to determine your patient's intellectual and functional abilities, then explain each procedure at a level the patient can understand. Allow extra time to explain oral health issues or instructions and demonstrate the instruments you will use. Use simple, concrete instructions, and repeat them often to compensate for any short-term memory problems.

Behavior management is not usually a problem in people with Down syndrome because they tend to be warm and well behaved. Some can be stubborn or uncooperative, but most just need a little extra time and attention to feel comfortable. Gaining the patient's trust is the key to successful treatment. Talk to the caregiver or physician about techniques they have found to be effective in managing the patient's behavior. Share your ideas with them, and find out what motivates the patient. It may be that a new toothbrush at the end of each appointment is all it takes to ensure cooperation. Schedule patients with Down syndrome early in the day if possible. Early appointments can help ensure that everyone is alert and attentive and that waiting time is reduced. Set the stage for a successful visit by involving the entire dental team – from the receptionist's friendly greeting to the caring attitude of the dental assistant in the operatory.

Provide oral care in an environment with few distractions. Try to reduce unnecessary sights, sounds or other stimuli that might make it difficult for your patient to cooperate. Many people with Down syndrome, however, enjoy music and may be comforted by hearing it in the dental office during treatment. Plan a step-by-step evaluation, starting with seating the patient in the dental chair. If this is successful, perform an oral examination using only your fingers. If this, too, goes well, begin using dental instruments. Prophylaxis is the next step, followed by dental radiographs.

Several visits may be needed to accomplish these tasks. Try to be consistent in all aspects of providing oral health care. Use the same staff, dental operatory, appointment times and other details to help sustain familiarity. The more consistency you provide for your patients, the more likely that they will be cooperative. Comfort people who resist oral care and reward cooperative behavior with compliments throughout the appointment. Use immobilization techniques only when absolutely necessary to protect the patient and staff during dental treatment, not as a convenience. There are no universal guidelines on immobilization that apply to all treatment settings. Before employing any kind of immobilization, it may help to consult available guidelines on federally funded care, your state department of mental health disabilities and your state dental practice act. Guidelines on behavior management published by the American Academy of Pediatric Dentistry (*www.aapd.org*) may also be useful. Obtain consent from your patient's legal guardian and choose the least restrictive technique that will allow you to provide care safely. Immobilization should not cause physical injury or undue discomfort.

Medical conditions

Though their average life expectancy has risen to the mid-50s, people with Down syndrome are still at risk for problems in nearly every system in the body. Some problems are manifested in the mouth. For example, oral findings such as persistent gingival lesions, prolonged wound healing, or spontaneous gingival hemorrhaging may suggest an underlying medical condition and warrant consultation with the patient's physician.

Cardiac disorders are common in Down syndrome. In fact, mitral valve prolapse occurs in more than half of all adults with this developmental disability. Many others are at risk of developing valve dysfunction that leads to congestive heart failure, even if they have no known cardiac disease. Consult the patient's physician if you have questions about the medical history and the need for antibiotic prophylaxis (*www.americanheart.org*).

Compromised immune systems lead to more frequent oral and systemic infections and a high incidence of periodontal disease in people with Down syndrome. Aphthous ulcers, oral Candida infections and acute necrotizing ulcerative gingivitis are common. Chronic respiratory infections contribute to mouth breathing, xerostomia, and fissured lips and tongue. Treat acute necrotizing ulcerative gingivitis and other infections aggressively. Talk to patients and their caregivers about preventing oral infections with regular dental appointments and daily oral care. Stress the importance of using fluoride to prevent dental caries associated with xerostomia. Use lip balm during treatment to ease the strain on your patient's lips.

Hypotonia affects the muscles in various areas of the body, including the mouth and large skeletal muscles. When it involves the mouth, it leads to an imbalance of forces on the teeth and contributes to an open bite. If the muscles controlling facial expression and mastication are affected, problems with chewing, swallowing, drooling and speaking can result. A related problem is atlantoaxial instability, a spinal defect that increases the mobility of the cervical vertebrae and often leads to an unsteady gait and neck pain. Maintain a clear path for movement throughout the treatment setting. Determine the best position for your patient in the dental chair and the safest way to move his or her body, especially the head and neck. Talk with the physician or caregiver about ways to protect the spinal cord. Use pillows to stabilize your patient and make him or her more comfortable.

Seizures sometimes occur in this population, especially among infants, but can usually be controlled with anticonvulsant medications. The mouth is always at risk during a seizure: Patients may chip teeth or bite the tongue or cheeks. People with controlled seizure disorders can easily be treated in the general dental office.

- Consult your patient's physician.
- Record information in the chart about the frequency of seizures and the medications used to control them. Determine before the appointment whether medications have been taken as directed.
- Know and avoid any factors that trigger your patient's seizures.
- Be prepared to manage a seizure.
    - If one occurs during oral care, remove any instruments from the mouth and clear the area around the dental chair. Attaching dental floss to rubber dam clamps and mouth props when treatment begins can help you remove them quickly.
    - Do not attempt to insert any objects between the teeth during a seizure.
    - Stay with your patient, turn him or her to one side and monitor the airway to reduce the risk of aspiration.

Hearing loss and deafness may further complicate poor communication skills, but these, too, can be accommodated with planning. Patients with a hearing problem may appear to be stubborn because of their seeming lack of response to a request.

Patients may want to adjust their hearing aids or turn them off, since the sound of some instruments may cause auditory discomfort. If your patient reads lips, speak in a normal cadence and tone. If your patient uses a form of sign language, ask the interpreter to come to the appointment. Speak with this person in advance to discuss dental terms and your patient's needs. Visual feedback is helpful. Maintain eye contact with your patient. Before talking, eliminate background noise (turn off the radio and the suction). Sometimes people with a hearing loss simply need you to speak clearly in a slightly louder voice than normal. Remember to remove your facemask first or wear a clear face shield.

Visual impairments such as strabismus (crossed or misaligned eyes), glaucoma and cataracts can affect people with Down syndrome. Determine the level of assistance your patient requires to move safely through the dental office.

Use your patients' other senses to connect with them, establish trust and make treatment a better experience. Tactile feedback, such as a warm handshake, can make your patients feel comfortable.

Face your patients when you speak and keep them apprised of each upcoming step, especially when water will be used. Rely on clear, descriptive language to explain procedures and demonstrate how equipment might feel and sound. Provide written instructions in large print (16 point or larger). Record in the patient's chart strategies that were successful in providing care. Note your patient's preferences and other unique details that will facilitate treatment, such as music, comfort items and flavor choices.

Table 1: First and second trimester Down syndrome screens				
Screen	**When performed (weeks gestation)**	**Detection rate**	**False positive rate**	**Description**
Quad screen	15-20	81 percent	5 percent	This test measures the maternal serum alpha feto protein (a fetal liver protein), estriol (a pregnancy hormone), human chorionic gonadotropin (hCG, a pregnancy hormone), and inhibin-Alpha (INHA).
Nuchal translucency/free beta/PAPPA screen (aka "1st trimester combined test")	10-13.5	85 percent	5 percent	Uses ultrasound to measure nuchal translucency in addition to the free beta hCG and PAPPA (pregnancy-associated plasma protein A). NIH has confirmed that this first trimester test is more accurate than second trimester screening methods. Performing an NT ultrasound requires considerable skill; a combined test may be less accurate if there is operator error, resulting in a lower-than-advertised sensitivity and higher false-positive rate, possibly in the 5-10 percent range.
Integrated test	10-13.5 and 15-20	95 percent	5 percent	The integrated test uses measurements from both the first trimester combined test and the second trimester quad test to yield a more accurate screening result. Because all of these tests are dependent on accurate calculation of the gestational age of the fetus, the real-world false-positive rate is greater than 5 percent and maybe be closer to 7.5 percent.

Oral health problems in Down syndrome and strategies for care

People with Down syndrome have no unique oral health problems. However, some of the problems they have tend to be frequent and severe. Early professional treatment and daily care at home can mitigate their severity and allow people with Down syndrome to enjoy the benefits of a healthy mouth.

Periodontal disease is the most significant oral health problem in people with Down syndrome. Children experience rapid, destructive periodontal disease. Consequently, large numbers of them lose their permanent anterior teeth in their early teens. Contributing factors include poor oral hygiene, malocclusion, bruxism, conical-shaped tooth roots and abnormal host response because of a compromised immune system. Some patients benefit from the daily use of an antimicrobial agent such as chlorhexidine. Recommend an appropriate delivery method based on your patient's abilities. Rinsing, for example, may not work for a person who has swallowing difficulties or one who cannot expectorate. Chlorhexidine applied using a spray bottle or toothbrush is equally efficacious.

If use of particular medications has led to gingival hyperplasia, emphasize the importance of daily oral hygiene and frequent professional cleanings. Encourage independence in daily oral hygiene. Ask patients to show you how they brush, and follow up with specific recommendations on brushing methods or toothbrush adaptations. Involve patients in hands-on demonstrations of brushing and flossing. Some people with Down syndrome can brush and floss independently, but many need help. Talk to their caregivers about daily oral hygiene. Do not assume that all caregivers know the basics; demonstrate proper brushing and flossing techniques. A power toothbrush or a floss holder can simplify oral care. Also, use your experiences with each patient to demonstrate sitting or standing positions for the caregiver. Emphasize that a consistent approach to oral hygiene is important and that caregivers should try to use the same location, timing and positioning.

Dental caries

Children and young adults who have Down syndrome have fewer caries than people without this developmental disability. Several associated oral conditions may contribute to this fact: delayed eruption of primary and permanent teeth; missing permanent teeth; and small-sized teeth with wider spaces between them, which make it easier to remove plaque. Additionally, the diets of many children with Down syndrome are closely supervised to prevent obesity; this helps reduce consumption of cariogenic foods and beverages.

By contrast, some adults with Down syndrome are at an increased risk of caries due to xerostomia and cariogenic food choices. Also, hypotonia contributes to chewing problems and inefficient natural cleansing action, which allow food to remain on the teeth after eating. Advise patients taking medicines that cause xerostomia to drink water often. Suggest taking sugar-free medicines if available and rinsing with water after dosing. Recommend preventive measures such as topical fluoride and sealants. Suggest fluoride toothpaste, gel or rinse, depending on your patient's needs and abilities. Emphasize noncariogenic foods and beverages as snacks.

Advise caregivers to avoid using sweets as incentives or rewards.

Several orofacial features are characteristic of people with Down syndrome. The midfacial region may be underdeveloped, affecting the appearance of the lips, tongue and palate. The maxilla, the bridge of the nose, and the bones of the midface region are smaller than in the general population, creating a prognathic occlusal relationship.

Mouth breathing may occur because of smaller nasal passages, and the tongue may protrude because of a smaller midface region. People with Down syndrome often have a strong gag reflex due to placement of the tongue, as well as anxiety associated with any oral stimulation. The palate, although normal sized, may appear highly vaulted and narrow. This deceiving appearance is due to the unusual thickness of the sides of the hard palate. This thickness restricts the amount of space the tongue can occupy in the mouth and affects the ability to speak and

chew. The lips may grow large and thick. Fissured lips may result from chronic mouth breathing. Additionally, hypotonia may cause the mouth to droop and the lower lip to protrude. Increased drooling, compounded by a chronically open mouth, contributes to angular cheilitis. The tongue also develops cracks and fissures with age; this condition can contribute to halitosis.

Malocclusion is found in most people with Down syndrome because of the delayed eruption of permanent teeth and the underdevelopment of the maxilla. A smaller maxilla contributes to an open bite, leading to poor positioning of teeth and increasing the likelihood of periodontal disease and dental caries. Orthodontia should be carefully considered in people with Down syndrome. Some may benefit, while others may not. In and of itself, Down syndrome is not a barrier to orthodontic care. The ability of the patient or caregiver to maintain good daily oral hygiene is critical to the feasibility and success of treatment.

Tooth anomalies are common in Down syndrome. Congenitally missing teeth occur more often in people with Down syndrome than in the general population. Third molars, laterals and mandibular second bicuspids are the most common missing teeth. Delayed eruption of teeth, often following an abnormal sequence, affects some children with Down syndrome. Primary teeth may not appear until age 2, with complete dentition delayed until age 4 or 5. Primary teeth are then retained in some children until they are 14 or 15.

Irregularities in tooth formation, such as microdontia and malformed teeth, are also seen in people with Down syndrome. Crowns tend to be smaller, and roots are often small and conical, which can lead to tooth loss from periodontal disease. Severe illness or prolonged fevers can lead to hypoplasia and hypocalcification. Examine a child by his or her first birthday and regularly thereafter to help identify unusual tooth formation and patterns of eruption. Consider using a panoramic radiograph to determine whether teeth are congenitally missing. Patients often find this technique less threatening than individual films. Maintain primary teeth as long as possible.

Consider placing space maintainers where teeth are missing.

Trauma and injury to the mouth from falls or accidents occur in people with Down syndrome. Suggest a tooth-saving kit for group homes. Emphasize to caregivers that traumas require immediate professional attention and explain the procedures to follow if a permanent tooth is knocked out. Also, instruct caregivers to locate any missing pieces of a fractured tooth, and explain that radiographs of the patient's chest may be necessary to determine whether any fragments have been aspirated.

Making a difference in the oral health of a person with Down syndrome may go slowly at first, but determination can bring positive results – and invaluable rewards.

Intellectual disability

Mental retardation is a generalized disorder, characterized by sub-average cognitive functioning and deficits in two or more adaptive behaviors with onset before the age of 18. Once focused almost entirely on cognition, the definition now includes both a component relating to mental functioning and one relating to individuals' functional skills in their environment.

Alternative terms

The term "mental retardation" is a diagnostic term designed to capture and standardize a group of disconnected categories of mental functioning such as "idiot," "imbecile" and "moron" derived from early IQ tests, which acquired pejorative connotations in popular discourse over time. The term mental retardation has itself now acquired some pejorative and shameful connotations over the last few decades due to the use of "retarded" as an insult. This may in turn have contributed to its replacement with expressions such as "mentally challenged" or "intellectual disability**"** While "developmental disability**"** may be considered to subsume other disorders (see below), "developmental disability**"** or "developmental delay" (for children under age 18), are generally considered more acceptable terms than "mental retardation" by those with disabilities.

In North America, mental retardation is subsumed into the broader term developmental disability, which also includes epilepsy, autism, cerebral palsy and other disorders that develop during the developmental period (birth to age 18.) Because service provision is tied to the designation developmental disability, it is used by many parents, direct-support professionals and physicians. However, in school-based settings, the more specific term mental retardation is still typically used, and is one of 13 categories of disability under which children may be identified for special education services under Public Law 108-446.

The phrase intellectual disability is increasingly being used as a synonym for people with significantly below-average cognitive ability. These terms are sometimes used as a means of separating general intellectual limitations from specific, limited deficits as well as indicating that it is not an emotional or psychological disability. Intellectual disability may also be used to describe the outcome of traumatic brain injury or lead poisoning or dementing conditions such as Alzheimer's disease. It is not specific to congenital disorders such as Down syndrome.

The American Association on Mental Retardation continued to use the term mental retardation until 2006. In June 2006 its members voted to change the name of the organization to the American Association on Intellectual and Developmental Disabilities, rejecting the options to become the AAID or AADD. Part of the rationale for the double name was that many members worked with people with pervasive developmental disorders, most of whom do not have mental retardation.

In the UK, "mental handicap" had become the common medical term, replacing "mental subnormality" in Scotland and "mental deficiency" in England and Wales, until Stephen Dorrell, secretary of state for health for the United Kingdom from 1995-97, changed the NHS's designation to "learning disability." The new term is not yet widely understood, and is often taken to refer to problems affecting schoolwork (the American usage), which are known in the UK as "learning difficulties." British social workers may use "learning difficulty" to refer to both people with MR and those with conditions such as dyslexia.

In England and Wales between 1983 and 2008, the Mental Health Act 1983 defined "mental impairment" and "severe mental impairment" as a state of arrested or incomplete development of mind, which includes significant/severe impairment of intelligence and social functioning and is associated with abnormally aggressive or seriously irresponsible conduct on the part of the person concerned. As behavior was involved, these were not necessarily permanent conditions; they were defined for the purpose of authorizing detention in hospital or guardianship. The term mental impairment was removed from the act in November 2008, but the grounds for detention remained. However, English statute law uses "mental impairment" elsewhere in a less well-defined manner, e.g. to allow exemption from taxes, implying that mental retardation without any behavioral problems is what is meant.

Signs

Children with mental retardation may learn to sit up, to crawl or to walk later than other children, or they may learn to talk later. Both adults and children with mental retardation may also exhibit the following characteristics:

- Delays in oral language development.
- Deficits in memory skills.
- Difficulty learning social rules.
- Difficulty with problem solving skills.
- Delays in the development of adaptive behaviors, such as self-help or self-care skills.
- Lack of social inhibitors.

The limitations of cognitive functioning will cause a child with mental retardation to learn and develop more slowly than a typical child. Children may take longer to learn language, develop social skills and take care of their personal needs, such as dressing or eating. Learning will take them longer, require more repetition and skills may need to be adapted to their learning level. Nevertheless, virtually all children are able to learn, develop and become participating members of the community.

In early childhood, mild mental retardation (IQ 60 to 70) may not be obvious and may not be identified until children begin school. Even when poor academic performance is recognized, it may take expert assessment to distinguish mild mental retardation from learning disability or emotional/behavioral disorders. As individuals with mild mental retardation reach adulthood, many learn to live independently and maintain gainful employment.

Moderate mental retardation (IQ 50 to 60) is nearly always apparent within the first years of life. Children with moderate mental retardation will require considerable supports in school, at home and in the community in order to participate fully. As adults they may live with their parents, in a supportive group home or even semi-independently with significant supportive services to help them, for example, manage their finances.

A person with a more severe mental retardation will need more intensive support and supervision his or her entire life.

Diagnosis

According to the latest edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), three criteria must be met for a diagnosis of mental retardation:

- An IQ below 70.
- Significant limitations in two or more areas of adaptive behavior (as measured by an adaptive behavior rating scale, i.e. communication, self-help skills, interpersonal skills, and more).
- Evidence that the limitations became apparent before the age of 18.

It is formally diagnosed by professional assessment of intelligence and adaptive behavior.

IQ below 70

The first English-language IQ test, the Terman-Binet, was adapted from an instrument used to measure potential to achieve developed by Binet in France. Terman translated the test and employed it as a means to measure intellectual capacity based on oral language, vocabulary, numerical reasoning, memory, motor speed and analysis skills. The mean score on the currently available IQ tests is 100, with a standard deviation of 15 (WAIS/WISC-IV) or 16 (Stanford-Binet). Subaverage intelligence is generally considered to be present when an individual scores two standard deviations below the test mean. Factors other than cognitive ability (depression, anxiety, etc.) can contribute to low IQ scores; it is important for the evaluator to rule them out prior to concluding that measured IQ is "significantly below average."

The following ranges, based on standard scores of intelligence tests, reflect the categories of the American Association of Mental Retardation, the Diagnostic and Statistical Manual of Mental Disorders-IV-TR, and the International Classification of Diseases-10:

Class	IQ
Profound mental retardation	Below 20
Severe mental retardation	20-34
Moderate mental retardation	35-49
Mild mental retardation	50-69
Borderline intellectual functioning	70-79

Since the diagnosis is not based only on IQ scores, but must also take into consideration a person's adaptive functioning, the diagnosis is not made rigidly. It encompasses intellectual scores, adaptive functioning scores from an adaptive behavior rating scale based on descriptions of known abilities provided by someone familiar with the person, and also the observations of the assessment examiner who is able to find out directly from the person what he or she can understand, communicate, and the like.

Significant limitations in two or more areas of adaptive behavior

Adaptive behavior, or adaptive functioning, refers to the skills needed to live independently (or at the minimally acceptable level for age). To assess adaptive behavior, professionals compare the functional abilities of a child to those of other children of similar age. To measure adaptive behavior, professionals use structured interviews, with which they systematically elicit information about persons' functioning in the community from people who know them well. There are many adaptive behavior scales, and accurate assessment of the quality of someone's adaptive behavior requires clinical judgment as well. Certain skills are important to adaptive behavior, such as:

- Daily living skills.
  - Getting dressed.
  - Using the bathroom.
  - Feeding oneself.
- Communication skills.
  - Understanding what is said and being able to answer.
- Social skills with peers, family members, spouses, adults and others.

Evidence that the limitations became apparent in childhood

This third condition is used to distinguish it from dementing conditions such as Alzheimer's disease or due to traumatic injuries with attendant brain damage.

Causes

Down syndrome, fetal alcohol syndrome and Fragile X syndrome are the three most common inborn causes. However, doctors have found many other causes. The most common are:

- **Genetic conditions.** Sometimes disability is caused by abnormal genes inherited from parents, errors when genes combine, or other reasons. The most prevalent genetic conditions include Down syndrome, Klinefelter's syndrome, Fragile X syndrome, neurofibromatosis, congenital hypothyroidism, Williams syndrome, Phenylketonuria (PKU), and Prader-Willi syndrome. Other genetic conditions include Phelan-McDermid syndrome (22q13del), Mowat-Wilson syndrome, genetic ciliopathy. In the rarest of cases, abnormalities with the X or Y chromosome may also cause disability. 48, XXXX and 49, XXXXX syndrome affect a small number of girls worldwide, while boys may be affected by 47, XYY, 49, XXXXY, or 49, XYYYY.
- **Problems during pregnancy.** Mental disability can result when the fetus does not develop properly. For example, there may be a problem with the way the fetus's cells divide as it grows. A woman who drinks alcohol (see fetal alcohol syndrome) or gets an infection like rubella during pregnancy may also have a baby with mental disability.
- **Problems at birth.** If a baby has problems during labor and birth, such as not getting enough oxygen, he or she may have developmental disability due to brain damage.
- **Exposure to certain types of disease or toxins.** Diseases like whooping cough, measles or meningitis can cause mental disability if medical care is delayed or inadequate. Exposure to poisons like lead or mercury may also affect mental ability.
- **Iodine deficiency**, affecting approximately 2 billion people worldwide, is the leading preventable cause of mental disability in areas of the developing world where iodine deficiency is endemic. Iodine deficiency also causes goiter, an enlargement of the thyroid gland. More common than full-fledged cretinism, as retardation caused by severe iodine deficiency is called, is mild impairment of intelligence. Certain areas of the world due to natural deficiency and governmental inaction are severely affected. India is the most outstanding, with 500 million suffering from deficiency, 54 million from goiter and 2 million from cretinism. Among other nations affected by iodine deficiency, China and Kazakhstan have begun taking action, whereas Russia has not.
- **Malnutrition** is a common cause of reduced intelligence in parts of the world affected by famine, such as Ethiopia.

Treatment and assistance

By most definitions, mental retardation is more accurately considered a disability rather than a disease. MR can be distinguished in many ways from mental illness, such as schizophrenia or depression. Currently, there is no "cure" for an established disability, though with appropriate support and teaching, most individuals can learn to do many things.

There are thousands of agencies in the United States that provide assistance for people with developmental disabilities. They include state-run, for-profit, and nonprofit, privately run agencies. Within one agency there could be departments that include fully staffed residential homes, day habilitation programs that approximate schools, workshops wherein people with disabilities can obtain jobs, programs that assist people with developmental disabilities in obtaining jobs in the community, programs that provide support for people with developmental disabilities who have their own apartments, programs that assist them with raising their children, and many more. The Burton Blatt Institute at Syracuse University works to advance the civic, economic and social participation of people with disabilities. There are also many agencies and programs for parents of children with developmental disabilities.

Although there is no specific medication for mental retardation, many people with developmental disabilities have further medical complications and may take several medications. Beyond that there are specific programs that people with developmental disabilities can take part in wherein they learn basic life skills. These "goals" may take a much longer amount of time for them to accomplish, but the ultimate goal is independence. This may be anything from independence in tooth brushing to an independent residence. People with developmental disabilities learn throughout their lives and can obtain many new skills even late in life with the help of their families, caregivers, clinicians and the people who coordinate the efforts of all of these people.

Dental care for patients with intellectual disability

Providing oral care to people with intellectual disability requires adaptation of the skills you use every day. In fact, most people with mild or moderate intellectual disability can be treated successfully in the general practice setting. Intellectual disability is a disorder of mental and adaptive functioning, meaning that people who are affected are challenged by the skills they use in everyday life. Intellectual disability is not a disease or a mental illness; it is a developmental disability that varies in severity and is usually associated with physical problems. While one person with intellectual disability may have slight difficulty thinking and communicating, another may face major challenges with basic self-care and physical mobility. Data indicate that people with intellectual disability have more untreated caries and a higher prevalence of gingivitis and other periodontal diseases than the general population.

Health challenges in intellectual disability and strategies for care. Many people with intellectual disability also have other conditions, such as cerebral palsy, seizure or psychiatric disorders, attention deficit/hyperactivity disorder, or problems with vision, communication and eating. Though language and communication problems are common in anyone with intellectual disability, motor skills are typically more affected when a person has coexisting conditions.

Before the appointment, obtain and review the patient's medical history. Consultation with physicians, family and caregivers is essential to assembling an accurate medical history. Also, determine who can legally provide informed consent for treatment.

Mental challenges. People with intellectual disability learn slowly and often with difficulty. Ordinary activities of daily living, such as brushing teeth and getting dressed and understanding the behavior of others as well as their own, can all present challenges to a person with intellectual disability. Set the stage for a successful visit by involving the entire dental team, from the receptionist's friendly greeting to the caring attitude of the dental assistant in the operatory. All should be aware of your patient's mental challenges. Reduce distractions in the operatory, such as unnecessary sights, sounds, or other stimuli, to

compensate for the short attention spans commonly observed in people with intellectual disability.

Talk with the parent or caregiver to determine your patient's intellectual and functional abilities, then explain each procedure at a level the patient can understand. Allow extra time to explain oral health issues or instructions and demonstrate the instruments you will use. Address your patient directly and with respect to establish a rapport. Even if the caregiver is in the room, direct all questions and comments to your patient. Use simple, concrete instructions and repeat them often to compensate for any short-term memory problems. Speak slowly and give only one direction at a time.

Be consistent in all aspects of oral care, because long-term memory is usually unaffected. Use the same staff and dental operatory each time to help sustain familiarity. The more consistency you provide for your patients, the more likely they will cooperate. Listen actively, since communicating clearly is often difficult for people with intellectual disability. Show your patient whether you understand. Be sensitive to the methods he or she uses to communicate, including gestures and verbal or nonverbal requests.

Behavior challenges. While most people with intellectual disability do not pose significant behavior problems that complicate oral care, anxiety about dental treatment occurs frequently. People unfamiliar with a dental office and its equipment and instruments may exhibit fear. Some react to fear with uncooperative behavior, such as crying, wiggling, kicking, aggressive language, or anything that will help them avoid treatment. You can make oral health care a better experience by comforting your patients and acknowledging their anxiety. Talk to the caregiver or physician about techniques they have found to be effective in managing the patient's behavior.

Schedule patients with intellectual disability early in the day if possible. Early appointments can help ensure that everyone is alert and attentive and that waiting time is reduced. Keep appointments short and postpone difficult procedures until after your patient is familiar with you and your staff. Allow extra time for your patients to get comfortable with you, your office and the entire oral health care team. Invite patients and their families to visit your office before beginning treatment. Permit the parents or caregiver to come into the treatment setting to provide familiarity, help with communication and offer a calming influence by holding your patient's hand during treatment. Some patients' behavior may improve if they bring comfort items such as a stuffed animal or blanket. Reward cooperative behavior with compliments throughout the appointment.

Consider nitrous oxide/oxygen sedation to reduce anxiety and fear and improve cooperation. Obtain informed consent from the legal guardian before administering any kind of sedation.

Use immobilization techniques only when absolutely necessary to protect the patient and staff during dental treatment, not as a convenience. There are no universal guidelines on immobilization that apply to all treatment settings. Before employing any kind of immobilization, it may help to consult available guidelines on federally funded care, your state department of mental health disabilities and your state dental practice act. Guidelines on behavior management published by the American Academy of Pediatric Dentistry (*www.aapd.org*) may also be useful. Obtain consent from your patient's legal guardian and choose the least restrictive technique that will allow you to provide care safely. Immobilization should not cause physical injury or undue discomfort.

People with intellectual disability often engage in perseveration, a continuous, meaningless repetition of words, phrases or movements. Your patient may mimic the sound of the suction, for example, or repeat an instruction over and again. Avoid demonstrating dental equipment if it triggers perseveration and note this in the patient's record. Allow extra time for your patient to get comfortable with you, your office and the entire oral health care team.

Physical challenges. Intellectual disability does not always include a specific physical trait, although many people have distinguishing features such as orofacial abnormalities, scoliosis, unsteady gait or hypotonia due to coexisting conditions. Countering physical challenges requires attention to detail. Maintain clear paths for movement throughout the treatment setting. Keep instruments and equipment out of the patient's way. Place and maintain your patient in the center of the dental chair to minimize the risk of injury. Placing pillows on both sides of the patient can provide stability.

If you need to transfer your patient from a wheelchair to the dental chair, ask the patient or caregiver about special preferences such as padding, pillows, or other things you can provide to ease the transition. The patient or caregiver can often explain how to make a smooth transfer. Some patients cannot be moved into the dental chair but instead must be treated in their wheelchairs. Some wheelchairs recline or are specially molded to fit people's bodies. Lock the wheels, then slip a sliding board (also called a transfer board) behind the patient's back to provide support for the head and neck during care.

Cerebral palsy occurs in one-fourth of those who have intellectual disability and tends to affect motor skills more than cognitive skills. Uncontrolled body movements and reflexes associated with cerebral palsy can make it difficult to provide care. Place and maintain your patient in the center of the dental chair. Do not force arms and legs into unnatural positions, but allow your patient to settle into a position that is comfortable and will not interfere with dental treatment. Observe your patient's movements and look for patterns to help you anticipate direction and intensity. Trying to stop these movements may only intensify the involuntary response.

Try instead to anticipate the movements, blending your movements with those of your patient or working around them. Softly cradle your patient's head during treatment. Be gentle and slow if you need to turn the patient's head. Help minimize the gag reflex by placing your patient's chin in a neutral or downward position.

Stay alert and work efficiently in short appointments. Exert gentle but firm pressure on your patient's arm or leg if it begins to shake. Take frequent breaks or consider prescribing muscle relaxants when long procedures are needed. People with cerebral palsy may need sedation, general anesthesia or hospitalization if extensive dental treatment is required.

Cardiovascular anomalies such as heart murmurs and damaged heart valves occur frequently in people with intellectual disability, especially those with Down syndrome or multiple disabilities. Consult the patient's physician to determine whether antibiotic prophylaxis (*www.americanheart.org*) is necessary for dental treatment.

Seizures are common in this population but can usually be controlled with anticonvulsant medications. The mouth is always at risk during a seizure: Patients may chip teeth or bite the tongue or cheeks. Persons with controlled seizure disorders can easily be treated in the general dental office.

Consult your patient's physician:

- Record information in the chart about the frequency of seizures and the medications used to control them. Determine before the appointment whether medications have been taken as directed.
- Know and avoid any factors that trigger your patient's seizures.
- Be prepared to manage a seizure.
    - If one occurs during oral care, remove any instruments from the mouth and clear the area around the dental chair. Attaching dental floss to rubber dam clamps and mouth props when treatment begins can help you remove them quickly.
    - Do not attempt to insert any objects between the teeth during a seizure.
    - Stay with your patient, turn him or her to one side and monitor the airway to reduce the risk of aspiration.

Visual impairments, most commonly strabismus (crossed or misaligned eyes) and refractive errors can be managed with careful planning. Determine the level of assistance your patient requires to move safely through the dental office. Use your patients' other senses to connect with them, establish trust and make treatment a good experience. Tactile feedback, such as a warm handshake, can make your patients feel comfortable.

Face your patients when you speak and keep them apprised of each upcoming step, especially when water will be used. Rely on clear, descriptive language to explain procedures and demonstrate how equipment might feel and sound. Provide written instructions in large print (16 point or larger).

Hearing loss and deafness can also be accommodated with careful planning. Patients with a hearing problem may appear to be stubborn because they seem to not respond to a request. Patients may want to adjust their hearing aids or turn them off, since the sound of some instruments may cause auditory discomfort.

If your patient reads lips, speak in a normal cadence and tone. If your patient uses a form of sign language, ask the interpreter to come to the appointment. Speak with this person in advance to discuss dental terms and your patient's needs.

Visual feedback is helpful. Maintain eye contact with your patient. Before talking, eliminate background noise (turn off the radio and the suction). Sometimes people with a hearing loss simply need you to speak clearly in a slightly louder voice than normal. Remember to remove your facemask first or wear a clear face shield.

Record in the patient's chart strategies that were successful in providing care. Note your patient's preferences and other unique details that will facilitate treatment, such as music, comfort items and flavor choices.

Oral health problems in intellectual disability and strategies for care

In general, people with intellectual disability have poorer oral health and oral hygiene than those without this condition. Data indicate that people who have intellectual disability have more untreated caries and a higher prevalence of gingivitis and other periodontal diseases than the general population.

Periodontal disease. Medications, malocclusion, multiple disabilities and poor oral hygiene combine to increase the risk of periodontal disease in people with intellectual disability. Encourage independence in daily oral hygiene. Ask patients to show you how they brush, and follow up with specific recommendations on brushing methods or toothbrush adaptations. Involve your patients in hands-on demonstrations of brushing and flossing.

Some patients cannot brush and floss independently due to impaired physical coordination or cognitive skills. Talk to their caregivers about daily oral hygiene. Do not assume that all caregivers know the basics; demonstrate proper brushing and flossing techniques. A power toothbrush or a floss holder can simplify oral care. Also, use your experiences with each patient to demonstrate sitting or standing positions for the caregiver. Emphasize that a consistent approach to oral hygiene is important – caregivers should try to use the same location, timing and positioning.

Some patients benefit from the daily use of an antimicrobial agent such as chlorhexidine. Recommend an appropriate delivery method based on your patient's abilities. Rinsing, for example, may not work for a patient who has swallowing difficulties or one who cannot expectorate. Chlorhexidine applied using a spray bottle or toothbrush is equally efficacious. If use of particular medications has led to gingival hyperplasia, emphasize the importance of daily oral hygiene and frequent professional cleanings.

Dental caries. People with intellectual disability develop caries at the same rate as the general population. The prevalence of untreated dental caries, however, is higher among people with intellectual disability, particularly those living in noninstitutional settings. Emphasize noncariogenic foods and beverages as snacks. Advise caregivers to avoid using sweets as incentives or rewards. Advise patients taking medicines that cause xerostomia to drink water often. Suggest sugar-free medicine if available and stress the importance of rinsing with water after dosing. Recommend preventive measures such as fluorides and sealants.

Malocclusion. The prevalence of malocclusion in people with intellectual disability is similar to that found in the general population, except for those with coexisting conditions such as cerebral palsy or Down syndrome. A developmental disability in and of itself should not be perceived as a barrier to orthodontic treatment. The ability of the patient or caregiver to maintain good daily oral hygiene is critical to the feasibility and success of treatment.

Missing permanent teeth, delayed eruption and enamel hypoplasia are more common in people with intellectual disability and coexisting conditions than in people with intellectual disability alone. Examine a child by his or her first birthday and regularly thereafter to help identify unusual tooth formation and patterns of eruption.

Consider using a panoramic radiograph to determine whether teeth are congenitally missing. Patients often find this technique less threatening than individual films. Take appropriate steps to reduce sensitivity and risk of caries in your patients with enamel hypoplasia.

Damaging oral habits are a problem for some people with intellectual disability. Common habits include bruxism; mouth breathing; tongue thrusting; self-injurious behavior such as picking at the gingiva or biting the lips; and pica, which is eating objects and substances such as gravel, cigarette butts, or pens. If a mouth guard can be tolerated, prescribe one for patients who have problems with self-injurious behavior or bruxism.

Trauma and injury to the mouth from falls or accidents occur in people with intellectual disability. Suggest a tooth-saving kit for group homes. Emphasize to caregivers that traumas require immediate professional attention and explain the procedures to follow if a permanent tooth is knocked out. Also, instruct caregivers to locate any missing pieces of a fractured tooth, and explain that radiographs of the patient's chest may be necessary to determine whether any fragments have been aspirated.

Wheelchair transfer: a health care provider's guide

Some patients who use wheelchairs can transfer themselves into the dental chair, but others need assistance. The extent of your involvement will depend on the patient's or caregiver's ability to help. Most people can be transferred safely from wheelchair to dental chair and back by using the two-person method. The following outline describes a safe transfer with a minimum of apprehension for the patient and clinician. Practice these steps before doing an actual patient transfer.

Six steps to a safe wheelchair transfer

STEP 1: Determine the patient's needs.

- Ask the patient or caregiver about:
  - Preferred transfer method.
  - Patient's ability to help.
  - Use of special padding or a device for collecting urine.
  - Probability of spasms.

Reduce the patient's anxiety by announcing each step of the transfer before it begins.

STEP 2: Prepare the dental operatory.

- Remove the dental chair armrest or move it out of the transfer area.
- Relocate the hoses, foot controls, operatory light and bracket table from the transfer path.
- Position the dental chair at the same height as the wheelchair or slightly lower. Transferring to a lower level minimizes the amount of strength necessary during the lift.

STEP 3: Prepare the wheelchair.

- Remove the footrests.
- Position the wheelchair close to and parallel to the dental chair.
- Lock the wheels in place and turn the front casters forward.
- Remove the wheelchair armrest next to the dental chair.
- Check for any special padding or equipment.

STEP 4: Perform the two-person transfer.

- Support the patient while detaching the safety belt.
- Transfer any special padding or equipment from the wheelchair to the dental chair.
- First clinician: Stand behind the patient. Help the patient cross his arms across his chest. Place your arms under the patient's upper arms and grasp his wrists.
- Second clinician: Place both hands under the patient's lower thighs. Initiate and lead the lift at a prearranged count (1-2-3-lift).
- Both clinicians: Using your leg and arm muscles while bending your back as little as possible, gently lift the patient's torso and legs at the same time.
- Securely position the patient in the dental chair and replace the armrest.

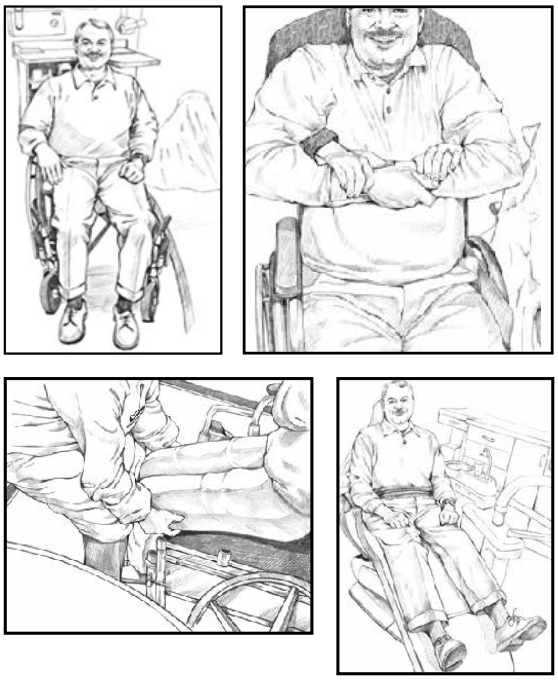
STEP 5: Position the patient after the transfer.

- Center the patient in the dental chair.
- Reposition the special padding and safety belt as needed for the patient's comfort.
- If a urine-collecting device is used, straighten the tubing and place the bag below the level of the bladder.

STEP 6: Transfer from the dental chair to the wheelchair.

- Position the wheelchair close to and parallel to the dental chair.
- Lock the wheels in place, turn the casters forward and remove the armrest.
- Raise the dental chair until it is slightly higher than the wheelchair and remove the armrest.
- Transfer any special padding.
- Transfer the patient using the two-person transfer (see step 4).
- Reposition the patient in the wheelchair.
- Attach the safety belt and check the tubing of the urine collecting device, if there is one, and reposition the bag.
- Replace the armrest and footrests.

This information can make a difference in your efforts to provide oral health care for patients who use a wheelchair. A skilled and sensitive dental staff can instill confidence during the transfer and encourage the patient to maintain a regular appointment schedule.



Two-person wheelchair transfer

1. Determine the patient's needs.
2. Prepare the dental chair and surrounding area.
3. Lock the wheelchair in place. Lower arm rest on wheelchair and raise the foot rest to clear the area.
4. Position one person behind the patient and one person in front of the patient.
5. Carefully follow all of the Steps 1-6 above to transfer the patient from the wheelchair to the dental chair.
6. Make sure the patient is positioned comfortably in the center of the chair.

Conclusion

Caring for individuals with developmental disabilities is very challenging, but it can also be very rewarding.

Making a difference in the oral health of a person with a developmental disability may go slowly at first, but determination can bring positive results – and invaluable rewards. By adopting the strategies discussed in this course, you can have a significant impact not only on your patients' oral health, but on their quality of life as well.

Physical abuse often presents as oral trauma. Abuse is more common in people with developmental disabilities than in the general population. If you suspect that a person is being abused or neglected, state laws require that you call your Child Protective Services agency. Assistance is also available from the Childhelp® USA National Child Abuse Hotline at (800) 422-4453 or the Child Welfare Information Gateway (www.childwelfare.gov).

References

- Centers for Disease Control
- The National Institute of Dental and Craniofacial Research Wikipedia

Final Examination Questions

Select the best answer questions 36 through 40 and mark your answers on the Final Examination Answer Sheet found on page 148, or for faster service complete your test online at **Dental.EliteCME.com.**

36. Cerebral palsy is divided into five major classifications to describe different movement impairments.
    - ○ True
    - ○ False

37. Behavior management during an exam is usually a severe problem in people with Down syndrome.
    - ○ True
    - ○ False

38. Children and young adults who have Down syndrome have more caries than people without this developmental disability.
    - ○ True
    - ○ False

39. Down syndrome, fetal alcohol syndrome and Fragile X syndrome are the three most common inborn causes of intellectual disability.
    - ○ True
    - ○ False

40. Abuse is more common in people with developmental disabilities than in the general population.
    - ○ True
    - ○ False

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2	○	○	10	○	○	18	○	○	26	○	○	34	○	○
3	○	○	11	○	○	19	○	○	27	○	○	35	○	○
4	○	○	12	○	○	20	○	○	28	○	○	36	○	○
5	○	○	13	○	○	21	○	○	29	○	○	37	○	○
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7	○	○	15	○	○	23	○	○	31	○	○	39	○	○
8	○	○	16	○	○	24	○	○	32	○	○	40	○	○

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