Tactile agnosia
Casuistic evidence and theoretical remarks on modality-specific meaning representations and sensorimotor integration

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Summary

Somaesthetic, motor and cognitive functions were studied in a man with impaired tactile object-recognition (TOR) in his left hand due to a right parietal convexity meningeoma which had been surgically removed. Primary motor and somatosensory functions were not impaired, and discriminative abilities for various tactile aspects and cognitive skills were preserved. Nevertheless, the patient could often not appreciate the object's nature or significance when it was placed in his left hand and was unable to name or to describe or demonstrate the use of these objects. Therefore, he can be regarded as an example of associative tactile agnosia. The view is taken and elaborated that defective modality-specific meaning representations account for associative tactile agnosia. These meaning representations are conceptualized as learned unimodal feature-entity relationships which are thought to be defective in tactile agnosia. In line with this hypothesis, tactile feature analysis and cross-modal matching of features were largely preserved in the investigated patient, while combining features to form entities was defective in the tactile domain. The alternative hypothesis of agnosia as deficit of cross-modal association of features was not supported. The presumed distributed functional network responsible for TOR is thought to involve perception of features, object recognition and related tactile motor behaviour interactively. A deficit leading primarily to impaired combining features to form entities can therefore be expected to result in additional minor impairment of related perceptual-motor processes. Unilaterality of the gnostic deficit can be explained by a lateralized organization of the functional network responsible for tactile recognition of objects.

Keywords: agnosia; apraxia; knowledge; sensorimotor integration; somaesthesis

Abbreviation: TOR = tactile object-recognition

Introduction

According to Bauer (1993) patients 'with tactile agnosia cannot appreciate the nature or significance of objects placed in the affected hand despite elementary somotosensory function, intellectual ability, attentional capacity and linguistic skill adequate to the task of object identification'. Early authors developed theoretical concepts of tactile agnosia with a remarkable distinction of related cognitive processes. Impaired TOR with relatively intact sensation of touch, pain and temperature was recognized as early as 1844 by Puchelt who observed this phenomenon in three reported cases. Hoffmann used the term 'astereognosis' in 1885 to describe an inability to distinguish between different three-dimensional forms which he explained in terms of impairment of spatial discrimination. Wernicke (1895) described two cases of 'tactile paresis' ('Tastlähmung') with unilaterally impaired

TOR, which in his opinion was not attributable to coexisting (minor) somatosensory deficits. He thought this syndrome was caused by unilateral destruction of the middle third of the central gyri leading to loss of 'tactile images' ('Tastvorstellungen'). The latter were viewed by Wernicke as a specific category of memorized representations. Each single representation would reflect a specific combination of tactile perceptual features of a corresponding object. Delay (1935) postulated three mechanisms involved in impaired object recognition: 'amorphognosia', the inability to recognize size and shape of objects, 'ahylognosia', the failure to discriminate distinctive qualities of objects like weight, texture and thermal properties, and 'tactile asymboly', a form of impaired object recognition in the absence of ahylognosia and amorphognosia.

Positive empirical evidence for tactile agnosia as a modality-specific impairment of object recognition in the absence of primary or discriminative somatosensory dysfunction is still scant. Only a few clinical case reports of tactile agnosia (without concomitant sensory or higher-order perceptual deficits severe enough to explain impaired TOR) have been documented in the literature (Wernicke, 1895; Raymond and Egger, 1906; Bonhoeffer, 1918; Campora, 1925; Delay, 1935; Hécean and David, 1945; Newcombe and Ratcliff, 1974; Caselli, 1991; Endo *et al.*, 1992). Several authorities in the field, including Strümpell (1918), von Monakow (1914) and Dejerine, Revesz and Foerster (*see* Bay, 1944), argued that the reported cases of 'tactile agnosia' could be explained by elementary somatosensory dysfunction. Bay (1944) described four patients with 'normal' primary somatosensory function on neurological examination, but major impairment of TOR. However, he also found that the impaired discriminative abilities and lability of somatosensory thresholds with repeated stimulation, a phenomenon known as 'Funktionswandel' (change of function) according to the school of von Weizäcker (*see* Cohen, 1926), was in proportion with TOR. Bay also claimed that many previous cases of 'tactile agnosia', including Wernicke's original cases could be explained in this way. More recently, it was pointed out that impaired TOR could also be mediated by supramodal spatial deficits (Semmes, 1965), hemineglect (Caselli, 1991) or a modality-specific anomia (Geschwind and Kaplan, 1962). Tactile anomia is phenomenologically similar to tactile agnosia. It has been reported mainly in patients with callosal lesions and is then characterized by an inability, in the absence of somaesthetic dysfunction, to name or describe objects held in the left hand (Bogen, 1993). In contrast to tactile agnosia, tactually anomic patients recognize objects by touch and can demonstrate recognition non-verbally (Endo *et al.*, 1992). Hence, this deficit is not an agnosia in its true sense, but represents a disconnection of intact tactile recognition mechanisms from speech processes.

Several contemporary authors who investigated TOR with relatively large cohorts of brain-damaged subjects could not support the concept of agnosia (Semmes, 1965; Corkin *et al.*, 1970; Roland, 1976). However, more recently Caselli (1991) reported seven patients with tactile agnosia 'defined as a modality-specific disturbance of somaesthetically mediated TOR that was not caused by more basic somatosensory imperception, a supramodal disorder of spatial perception, the inability to explore an object tactually, or the inability to demonstrate intact recognition (for example, aphasia).' Further casuistic evidence for the existence of tactile agnosia (and tactile anomia) was provided by Endo *et al.* (1992).

The author recently took care of a patient with whom he found evidence of tactile agnosia. Sensibility and motor evaluation will be described and discussed in detail and complemented with neuroanatomical, routine clinical and psychological data. Theoretical considerations concerning the nature of tactile agnosia and its relation to sensorimotor integration in the described case are presented.

Case report

Patient H.K., a 51-year-old man, attended medical care due to paraesthesias on the left side of his body. Cranial computerized tomography and MRI revealed a right parietal tumour. Craniotomy was performed a few days later for removal of the tumour, which was histologically classified as meningeoma. His postoperative clinical and neurological status was unchanged. Four weeks postoperatively he was admitted to our department for functional neurological evaluation and rehabilitation, since he subjectively reported deficits of fine motor control of his left hand even though no overt paresis could be documented.

His neurodevelopmental history was unremarkable. He had received the minimal required school education and served an apprenticeship as optician. He has been working as a technical expert for a television company for >20 years .

Neurological and psychometric examination

Neurological examination on admission to our department revealed no cranial nerve damage. Signs of paresis, central or peripheral, were absent. The strength of all muscle groups was full, tendon reflexes were equally brisk on both sides, pathological reflexes or cloni could not be elicited, and muscle tone and bulk as well as passive range of motion were unremarkable. Clinical tests of co-ordination for upper and lower limbs as well as during stance and gait could be performed without signs of impairment. Routine testing of sensibility revealed normal sensation of light touch, pinprick, position sense and vibration throughout his body. Sensation of temperature seemed to be slightly impaired for his left hand. Without visual clues he recognized numbers written on his palm flawlessly on both sides. However, he seemed unable to recognize common objects when they were put in his left hand even though he could name these objects instantly when put in his right hand afterwards. Clinically, vigilance, orientation, attention, memory, right–left differentiation, body schema, calculation, phasia, buccofacial, ideomotor, ideational and constructional praxia were found to be unimpaired.

Psychometric evaluation of Patient H.K. revealed above-average fluent intelligence (Standard Progressive Matrices, percentile 100; Raven *et al.*, 1987). Visual spatial performance was also above average (Block Design subtest from the WAIS, percentile 98; Wechsler, 1981). Mnesic deficits were excluded with an average performance in the Benton Visual Retention Test (Benton, 1974); both correct score and error score were in the range of scores expected from the estimated premorbid IQ and age. Attention functions were above average (Trail Making Test, percentile 62; Reitan, 1958). Psychomotor speed was unimpaired (single and choice reaction times ranged between 180 and 450 ms). The results of a motor performance series (Schoppe, 1974) measuring steadiness, aiming, tapping, precision and speed showed a right–left difference with somewhat lower performance of the left hand according to speed parameters.
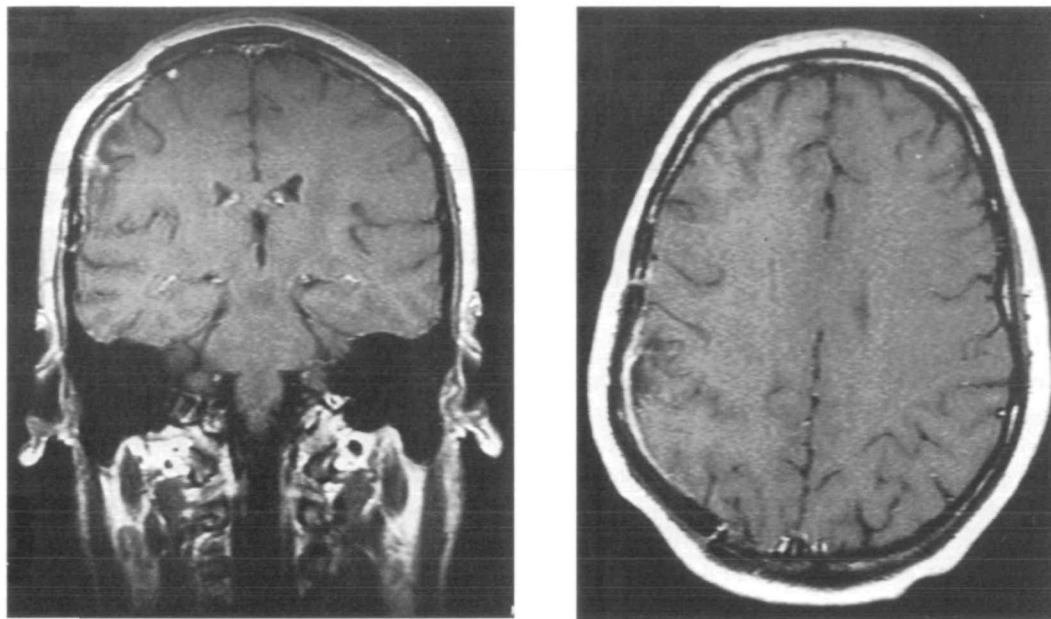
Fig. 1 Delayed postoperative contrast-enhanced T1-weighted MRI of the head of Patient H. K. showing the right parietal cortical lesion in coronal (*left*) and transversal (*right*) projection. Courtesy of Professor Felix, Dept of Radiology, Virchow-Klinikum, Humboldt-Universität, Berlin.

Summarizing the results of routine clinical and psychometric testing revealed largely preserved cognitive and sensorimotor abilities. Thermaesthesia seemed slightly reduced for the left hand. However, the most striking finding was that TOR was severely impaired when performed with the left hand.

In order to evaluate the reasons for the impaired TOR, sensory and motor functions were specifically studied in more detail. The patient was informed about the purpose of the investigation and gave informed consent to participate in the evaluation procedure.

Neuroanatomy of the lesion

Preoperative MRI of the head in sagittal, horizontal and frontal planes showed a well-defined tumour with a base close to the skull (dura mater) and protruding with a spherical shape into the middle and lower third of the right parietal cortex. The maximal diameter measured approximately 4.5 cm in the longitudinal, 2.6 cm in the lateral and 3 cm in the vertical dimension. The tumour's localization consistently corresponded to the postcentral and supramarginal gyrus in an unaffected brain. An MRI of the head was also done 10 months postoperatively and revealed cortical damage of the right postcentral gyrus, but especially of the right supramarginal gyrus (Fig. 1) when compared with a neuroanatomical atlas (Kretschmann and Weinrich, 1991).

Experimental methods
Somatosensory evaluation methods

The following sensibility and perceptual modalities were tested: light touch, pain, temperature, vibration, position sense, two-point discrimination, kinaesthesia, simultaneous double stimulation, discrimination of weight, texture, size, two-dimensional and three-dimensional form, thermal properties and TOR. Sensibility testing was performed separately for the right and left hand, usually at the tip of the index finger but if the task demanded with the whole hand (e.g. for TOR). Except for the subtest 'tactile-visual matching of objects' the patient was blindfolded throughout sensibility evaluation.

Light touch was assessed using a cotton wool string measuring ~1 mm in diameter. A sharp needle was used for pinprick testing. For evaluation of temperature sensation two test-tubes were filled with either warm (~45°C) or cold water (~20°C) and the patient had to give his impression spontaneously. Vibration was measured semi-quantitatively with a tuning fork scaled from 1 to 8 reflecting amplitude of vibration. The patient's vibration sensation was first measured with the largest, and then the smallest, discernible amplitude. Proprioception was tested with very small vertical passive movements of the index finger. Static two-point discrimination used a spacing of 0.3 cm. Kinaesthesia was tested by either lengthwise or sideways movements of a cotton wool string over the finger tip. Extinction was investigated by touching the right or left hand (or both together) and with the patient reporting which side was touched.

For weight discrimination three wooden plates of equal size, shape and texture weighing 14, 20 and 28 g were presented to the patient. He first had to rank them according to their weight (heavy–medium–light). Afterwards he was presented with another plate weighing either 14, 20 or 28 g and had to match it tactually to the corresponding plate among the set of three plates. The order of presentation was changed for right and left hand for this and all other matching

tasks. For texture discrimination, a cube was used; it had 4.5 cm sides and four different textures on its four horizontal sides. Curtain material, a smooth surface of PVC, felt and fine-grain sandpaper had to be identified tactually. Then each texture had to be matched tactually with the corresponding texture of an identically structured second cube. Size discrimination was tested by ranking and matching iron nuts sized 5, 7.5, 9 and 12.5 mm in diameter. This had to be performed by touch only, without lifting the objects. Form discrimination was assessed both two- and three-dimensionally. A square-shaped metal frame with 7.5 cm sides contained either a vertically or diagonally oriented rod or a rectangularly bent rod, which had to be identified and afterwards tactually matched. For three dimensional form analysis, two sets of two arbitrarily formed plastic objects (one for each hand) had to be described verbally while they were manipulated with either right or left hand. Their length varied between 1.5 and 5.7 cm, and their width between 1.5 and 2.9 cm. It was judged whether principal form properties and their spatial relationships with each other within a single object were appropriately verbalized (e.g. plate, cylinder, cone and ring). For the subsequent tactile matching the two sets had been exchanged. For discrimination of thermal properties the patient's index finger was put on four different plates consisting of either metal ('cool'), stone ('moderately cool'), wood ('indifferent') or felt ('warm') without being allowed to explore the plates by finger movements; he had to name the thermal property according to given categories. Afterwards he had to match thermal properties.

For TOR tests, 25 common objects were presented, 17 to the patient's left (affected) hand and eight to his right (normal) hand. Objects were presented in groups of four (or five). Within each group the patient was first asked to identify single manipulated objects verbally. If the patient failed to recognize a given object tactually, he was immediately given a tactile selection task; the unidentified object was again presented, but together with other objects. The patient was then asked to explore the group of four (or five) objects tactually and to select the unidentified object. Finally, tactile–visual matching was tested; the patient was allowed to explore the unidentified object tactually (without vision of the object or of exploring hand) and was then shown a group of four or five objects among which there was a second identical exemplar of the unidentified object. The task was then to show which of the visually presented objects matched the tactually explored object. The following objects were presented: a pencil, screw, comb, lighter, rubber, coin, battery, paper-clip, cotton bud ('Q-tip'), key, screw-top, ink catridge for a pen, safety-pin, clothes-pin, candle for a teapot-heater ('Teelicht'), thimble, nail-clipper for the left hand, candle, nail, ball-pen, screwdriver, toothbrush, button, match and a thumb-tack for the right hand.

The patient's performance during sensibility evaluation was videotaped. It was rated by two independent raters, one being the author, the other a medical student who was not informed about the purpose of the study and questions raised

by it. Performance was rated qualitatively as either correct or incorrect for each single required response. Assessment was conservative in the sense that patient's approach to the task and the time needed to complete it was not scored, only his final solution on a pass/fail basis. Inter-rater agreement was high. Disagreement occurred in only one out of 128 ratings, where the author's judgement had been more conservative than the medical student's during the subsession related to TOR.

Motor evaluation methods

Maximal fist closure was measured with a hand dynamometer in bar. The fastest repetitive vertical movements of the index finger and manipulative movements of thumb, index and middle finger were analysed three-dimensionally using an optoelectronic system while the hand and pronated arm rested comfortably on a desk. The patient was instructed to make repetitive vertical tapping movements with his index finger as fast as possible and without concomitant horizontal movements. A total of 50 consecutive taps were analysed on each side. Mean and standard deviation of movement time of single taps and of a laterality quotient reflecting the ratio between the amplitude of the horizontal movement component and the vertical movement component were automatically calculated. Thus, both temporal and spatial aspects of these simple finger movements were assessed. Mean values of the left hand were considered pathological if they deviated >2 SD from the corresponding mean of the right hand. The rationale for this approach was to analyse the principle capacity to make fast, partially constrained finger movements quantitatively; the next task assessed spontaneously evoked finger movements qualitatively, during tactile object manipulation.

For recording the manipulative behaviour the arm rested on a desk in a supine position, parallel to one coordinate of the optoelectronic system. After being blindfolded, the patient held his fingers in a semiflexed position and the object, a dice, was put into a precision grip between the thumb, and index and middle fingers. Shortly afterwards the patient was ask to manipulate the object until further notice in order to recognize it tactually. Recording lasted 25 s starting from object placement into precision grip. During manipulation it was arranged that if the dice was dropped it would be caught softly without eliciting a characterizing sound, to prevent auditory clues.

Three-dimensional motion analysis was performed with a conventional optoelectronic system (SELSPOT II) with two infrared-sensitive cameras and one (tapping) or three (manipulation) light-emitting diodes. A standard calibration procedure allowed a spatial resolution of <1 mm. The sampling frequency was 100 Hz. The three-dimensional position data describing manipulative behaviour were low-pass-filtered using a fast digital response filter with a cut-off at 20% of sampling frequency.

Table 1 *Results of somaesthetic tests in Patient H.K.*

(A) Basic and intermediate somatosensory functions
Correct responses: Right and left index finger and hand: correct responses to light touch, pain, temperature, vibration, position changes, one or two-point stimuli, kinaesthetic stimuli, unilateral or simultaneous double stimulation; appropriate categorical discrimination and tactile matching of weight, texture, size and two dimensional form as well as tactile matching of thermal properties; correct verbal characterization of three dimensional form of arbitrary objects with geometrical constituents and tactile matching of these objects.

Incorrect responses: Left index finger: Categorization of thermal properties was rated incorrect with one out of four stimuli (by one rater).

B) Tactile object-recognition (TOR)
Right hand: eight out of eight common objects immediately recognized.

Left hand: eight out of 17 common objects not recognized; tactile selection of individual objects from a group of four to five objects (previously tactually unrecognized) was unimpaired with one exception; tactile-visual matching was performed flawlessly and was correct for all tactually unrecognized objects.

Results

Results of somatosensory evaluation

Somaesthetic analysis was primarily based on a pass-fail evaluation. Results are summarized in Table 1. Patient H.K. responded correctly to all stimuli testing basic and intermediate somatosensory functions with either hand with one exception: the patient categorized one out of four thermal stimuli incorrectly when he touched them with his left finger. The touched stone plate was reported to have 'indifferent' thermal properties instead of 'moderately cool' thermal properties. The fact that his responses were almost always correct showed largely preserved discriminative somaesthetic abilities. Nevertheless, it often took Patient H.K. longer to respond to stimuli when his left hand was tested possibly reflecting a reduced efficiency of otherwise preserved discriminative abilities.

During the TOR test the patient could not recognize the following eight (out of 17) common objects with his left hand: the lighter, battery, paper-clip, screw-top, ink catridge for a pen, safety-pin, candle for a teapot-heater and the nail-clip. His recognition time was often prolonged (with the left hand) and seemed to involve conscious analysis of object properties and exclusion strategies. For every tactually unrecognized object, tactile selection and tactile–visual matching were tested. Tactile selection tested the ability to select the unrecognized object tactually when a group of four (to five) different objects was presented including the unrecognized item. The patient was able to select unidentified objects with one exception: tactile selection of the safety-pin was rated inappropriate by one rater. Tactile–visual matching, when the patient had the unrecognized object in his left hand without seeing it and was allowed to view an array of objects including the identical equivalent of the unrecognized object, was performed flawlessly and was correct for all tactually unrecognized objects. Usually the patient was astonished that he had not recognized the object tactually, since all of them were instantly recognized and named by him when he had the chance to see an identical copy of the tactually unrecognized object among other objects. All eight of these objects were immediately and correctly recognized when put

Table 2 *Motion analysis of 50 repetitive, fastest vertical index finger movements in Patient H.K.*

	Right index finger Mean±SD	Left index finger Mean±SD
Movement duration (ms)	250±20	280±40
Laterality ratio* (%)	4±3	6±4

*Laterality ratio = 100×(horizontal movement amplitude/vertical movement amplitude).

in the right hand of the blindfolded patient, as indicated with an appropriate verbal response.

Results of motor evaluation

The maximal power of fist closure was 0.78 bar for the right hand and 0.7 bar for the left hand. This minor difference can be attributed to right-handedness of the patient. Thus, in agreement with clinical examination absence of unilateral paresis of the left hand can be assumed confidently.

The 50 repetitive, fastest vertical index finger movements were analysed by three-dimensional motion analysis. Results presented in Table 2 show that movement time was slightly longer with the left index finger and that spatial task requirements were slightly less well fulfilled. However, for both parameters, the mean values for the left index finger had been within a range of <2 SD from the corresponding values of the right hand. Therefore, they could not be classified 'pathological' according to *a priori* criteria.

Manipulation movements were recorded when the blinded patient explored a dice tactually between thumb, index and middle finger. The dice was first put into his left hand. The first striking finding was that he lost the dice ~11 s after starting to manipulate the object. Higher amplitude finger movements occurred at the time when the dice slipped of his fingers, perhaps reflecting an attempt to regain control over the object (Fig. 2). After loss of the dice, movements stopped. The patient had not recognized the object during this period. The dice was then put into his right hand. After
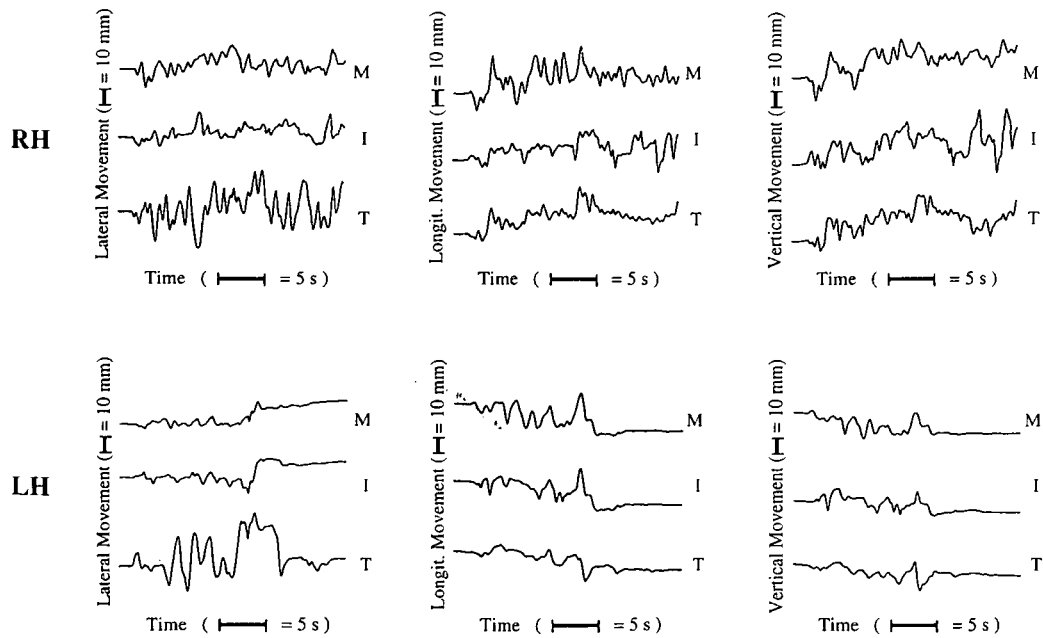
Fig. 2 Manipulative finger movements. Each set of nine traces reflect lateral, longitudinal and vertical positional changes (movement) of the thumb (T), index (I) and middle finger (M), while the subject was exploring a dice with these fingers, with his the left (LH) or right hand (RH). All traces are of 25 s duration (time 0 at left of each trace).

the patient had started exploratory movements he recognized the object immediately. According to the task instruction he continued exploratory movements (Fig. 2) until further notice was given after 25 s recording time. For both right and left hands, predominant directions of movement were recognizable in the thumb and middle finger; the greatest movement amplitudes were recorded in the lateral coordinate for thumb movements and less markedly in the longitudinal coordinate for movements of the middle finger. Even though this seems to reflect a common exploratory movement pattern for both hands, there are also right–left differences. Compared with the right side, movements of the left thumb were less frequent. In addition, the amplitude of index and middle finger motion was reduced on the left side.

Follow-up

The patient was investigated again clinically 19 months after his surgery. Neurological examination of cranial nerves, motor system and sensibility (light touch, pinprick, temperature, position sense and vibration) was unremarkable. The follow-up investigation of object recognition using the same set of items showed that he could still not recognize, name or demonstrate the use of four out of the 17 common objects. Nevertheless, he spontaneously described features of unrecognized objects (material, shape, size and weight in comparison with other items) adequately and again showed intact tactile selection and tactile–visual matching for unrecognized objects.

Discussion

Tactile agnosia

Does the reported case, H.K., resemble an example of tactile agnosia? Clinically and psychometrically it was clear that orientation, attention functions, psychomotor speed, visual spatial performance, mnesic abilities, linguistic skills and intellectual abilities were all adequate to the task of object identification. Basic somaesthetic functions were intact with the exception of a very mild discriminative deficit of thermaesthesia, which would certainly not explain his deficit in TOR. Bilateral simultaneous stimulation did not cause extinction. Further, it was shown that H.K. could tactually discriminate object characteristics like size, shape, weight and texture and could use these skills for tactile selection and tactile–visual matching of unrecognized objects. However, in spite of his cognitive, primary somatosensory and tactile discriminative abilities H.K. could often not appreciate an object's nature or significance when it was placed in his left hand and he was then unable to name or to describe or demonstrate the use of these objects. He therefore can be regarded as an example of tactile agnosia. His deficit was shown to persist, at least partially, for >1 year.

While Patient H.K. could be considered agnosic, he also seemed to have subtle perceptual changes, i.e. the time he needed to make tactile discriminative decisions was often longer for his agnosic left hand.

The old postulate that tactile perception and tactile gnosis are distinct consecutive stages in the process of TOR has encouraged the search for 'purely' agnosic patients who do

not show perceptual deficits. However, this aim has not been achieved. While tactile agnosia has been shown to exist phenomenologically, all 'agnosic' patients seem to have at least minor perceptual deficits, if they are investigated carefully enough. For some, this observation is evidence enough to argue that tactile agnosia does not exist. Others would point out that these minor perceptual deficits are not always severe enough to explain impaired TOR and hence, that there are cases of tactile agnosia. Indeed, within the conceptual framework of stages of processing both positions had to be taken and neither position could easily be invalidated. As a consequence, the question arises whether the clinical evidence is at all compatible with the notion of perception and recognition as completely separate stages of processing.

An alternative concept for tactile agnosia is proposed here which not only allows for associated perceptual deficits: it expects them. It is argued that TOR involves distributed perceptual-motor processes and is not a separate stage following perception. Perceptual deficits (aperceptive agnosia) and associative agnosia are not viewed as phenomena that reflect impairment of completely distinct underlying processes, but as two overlapping phenomena which are observable with damage to the system involved in TOR. A postulated unimodal knowledge system guaranteeing TOR would not memorize entities completely separately from their features. Instead, both perception and memory for perceptual features, and for their combinations, representing entities (feature–entity relationships) are functions of a single distributed network. Aperceptive and gnostic processes overlap and run interactively. Clinical evidence for tactile agnosia with largely intact, but presumably never completely intact, perceptual abilities arise as follows: deficient representations of feature–entity relationships within the distributed network lead primarily to an agnosic deficit, but due to the interactive nature of the network they also lead to alteration of feature analysis.

Object manipulation

As outlined above, a variety of primary and intermediate (i.e. discriminative) somatosensory as well as supramodal cognitive deficits were excluded as possible causes of impaired TOR in the case of Patient H.K. apart from tactile agnosia. In addition, it seemed warranted to investigate motor processes and their relationship to impaired TOR.

Tactile agnosia was called 'Tastlähmung' (tactile paresis) by Wernicke (1895) and it was thus terminologically related to active touch. Active touch isolates and enhances the components of stimulation which specify the characteristics of the object being touched (Gibson, 1962). Both non-specific motor dysfunction (central paresis) and motor impairment specifically related to active touch, i.e. 'tactile apraxia' (Delay, 1935), can affect manipulative motor behaviour.

Cases with tactile agnosia have been reported both with (Wernicke, 1895; Oppenheim, 1906; Bonhoeffer, 1918;

Campora, 1925; Hécaen and David, 1945) and without (Oppenheim, 1906; Patient H.K.) signs of minimal to moderate central paresis affecting the agnosic hand. In cases with central paresis fine finger movements were (Wernicke, 1895; Oppenheim, 1906; Hécaen and David, 1945) or were not (Bonhoeffer, 1918) impaired. In addition, Caselli's (1991) work showed that hemiparesis itself does not impede TOR. Thus, central paresis seems not to be a critical factor in tactile agnosia. This position is strengthened by the case of H.K. Not only were clinical signs of central paresis absent, but it was also shown kinematically that his ability to make fast repetitive finger movements was preserved.

In accordance with Caselli's (1991) report on tactile agnosic patients, Patient H.K.'s exploratory finger movements were not grossly deranged when observed clinically. However, kinematic analysis showed subtle changes of exploratory behaviour with smaller movements of index and middle finger and less frequent thumb movements compared with the non-agnosic hand (Fig. 2). In addition, H.K. lost the object during manipulation. One might therefore ask how these motor findings relate to TOR. Since an underlying non-specific motor impairment (e.g. reduced speed of motor execution) was excluded they could imply a function-specific deficit, namely an impairment of sensorimotor integration related to object manipulation. This interpretation is supported by the minor reduction of speed of performance for the agnosic hand reported in a motor performance study (Schoppe, 1974) where all tasks involve handling of objects (stylus, pegs) and by subjectively noted deficits of fine motor control while the clinical examination did not reveal basic motor deficits. Such minor changes of manipulative behaviour are presumably of little functional significance for TOR when discriminative tactile abilities are preserved, as with patient H.K. Equally, they are not specific for tactile agnosia, since impaired exploratory finger movements have been observed with more basic somatosensory deficits (Pause *et al.*, 1989). According to Liepmann's (1908, 1920) concept of apraxia this subtle deficit would represent motor consequences of the prevailing agnosic failure rather than an apraxic syndrome. It follows that deficient gnostic processes can be associated with function-specific deficits of related motor processes. This conclusion speaks against the notion that TOR is performed in independent stages from more basic to higher sensorimotor and cognitive functions and favours the proposed concept of an interactive mode of function between involved processes.

Neuroanatomical considerations

Preoperative MRI of the head showed a meningeoma with a base close to the skull which protruded with a spherical shape into the middle and lower third of the right parietal cortex in a location corresponding to the postcentral and supramarginal gyrus in an unaffected brain. The second MRI of the head performed 10 months after surgical removal of the tumour revealed cortical damage in the area of the

right postcentral and especially the right supramarginal gyrus (Fig. 1). Dysfunction of these areas might therefore have been crucial for tactile agnosia in this case. The middle third of the postcentral gyrus was believed to contain 'tactile images' ('Tastvorstellungen') by Wernicke (1895) and was later postulated to be associated with impaired TOR due to basic and intermediate somatosensory deficits (Bay, 1944; Corkin *et al.*, 1970). Caselli concluded (1991) that tactile agnosia in his patients resulted from unilateral damage to parietotemporal cortices, possibly including the second somatosensory cortex which can be thought of as a sensorimotor integration area (Corkin, 1978).

Modality-specific meaning representations

Agnosia in the sense of associative agnosia (Lissauer, 1890) relates to memory for facts (or semantic memory) and can be thought of as a modality-specific impairment in the recognition of normally perceived previously learned stimuli. The existence of visual, auditory and tactile agnosic syndromes (Damasio *et al.*, 1992) shows that recognition of entities might selectively be impaired within a given modality while at the same time the specific entity might be recognized through any other modality. It follows that a single modality is not necessarily involved in every instance of entity recognition. Accepting this notion, one might postulate that recognition can occur within any single modality and/or within a multimodal system. Accordingly, agnosia could be understood either as deficit of modality-specific meaning systems or as a modality-specific disconnection from a multimodal meaning store.

Lissauer's (1890) concept of associative agnosia uses the second of these possibilities to understand agnosia, namely as a modality-specific disconnection from multimodal associations, and this idea will be discussed first. Lissauer thought that recognition is a result of an interaction between perception and association. Modality-specific cortical areas allow perception (and memory) of features. Their association with other multimodal contents of memory leads to recognition. Failure of perception could result from localized cortical damage and would lead to apperceptive agnosia. Failure of association would have its cause in disruption of transcortical fibres and would clinically present as associative agnosia with largely reserved (but due to an interactive mode of function never completely preserved) perception.

Both mechanisms seemed unimpaired with Patient H.K. He could tactually discriminate and identify features. Equally, multimodal feature-related associations were not positively found to be impaired; i.e. he could describe arbitrary three-dimensional forms reasonably well verbally and his tactile-visual matching of unrecognized objects was performed flawlessly, implying a preserved ability to match across modalities at feature-level. These observations in Patient H.K. are at odds with Lissauer's assumption about the nature of associative agnosia.

They are, however, in agreement with the alternative

explanation that defective modality-specific meaning representations account for associative tactile agnosia. The notion of modality-specific meaning representations assumes that not only feature analysis, but also identification of objects takes place primarily within a single modality. Multimodal associations at feature level are not a necessary prerequisite for object identification and equally not the usual mode of association leading to recognition. Recognition is usually based on learned unimodal feature–entity relationships mediated by unimodal associations of features. Associative tactile agnosia would then be the deficit to integrate perceived features to entities within the tactile modality. Patient H.K. showed a compatible constellation. Feature analysis and multimodal association at feature level were largely preserved and yet object identification in the tactile modality was impaired. It should be noted that related theoretical concepts for tactile agnosia date back to Wernicke who thought that 'tactile images' ('Tastvorstellungen') encoding tactile entities are implemented as interconnected functional groups of neurons individually representing tactile perceptual features (Wernicke, 1895). The basic distinction from Lissauer's concept is that association of features with entities is thought to happen primarily within a given modality.

The well-documented case of tactile agnosia reported by Endo *et al.* (1992) is also in agreement with the presented concept. Their case Y.K. could adequately describe features of unrecognized objects, perform visual–tactile matching of objects and showed only mild impairment when asked to select named objects by touch, while his ability to recognize objects by touch was severely impaired. All preserved functions could be explained by an ability to transfer information at feature level across modalities; his TOR could have been impaired because it relied on deficient unimodal feature–entity relationships within the tactile domain which could not easily be compensated for by preserved cross-modal associations. The alternative explanation suggested by Endo and co-workers seems weaker; it assumes a unidirectional disconnection of the information flow from the somatosensory association cortex (responsible for feature analysis) to a postulated multimodal memory system in the temporal lobe causing the agnosic deficit while retaining an intact reverse information flow between the same two brain areas allowing the selection of named objects.

Anatomical evidence in monkeys also favours a unimodal mode of tactile perceptual processing and does not suggest multimodal associations at feature level. Areas of the somatosensory cortex are densely interconnected with each other, with Brodman areas 4 and 6, and partially with area 5. However, no other cortical area is known to project into them (Jones and Powell, 1969*a*, *b*).

A recently developed theory which is compatible with the outlined ideas formulates that for a given modality both perception and knowledge are mediated by distributed time-locked activation within the same distributed neural ensembles (Damasio, 1989; Damasio, *et al.*, 1992). This neural network encodes both features and recipes for binding

features into entities. 'Local convergence zones' are located near early sensory and motor cortices and bind featural components of entities. Associative agnosia would result from their malfunction. 'Nonlocal convergence zones' are located in multimodal cortices and provide a basis for the (distributed) representation of events linked to entities.

The concept of modality-specific meaning representations in general is supported by neuropsychological evidence (Goodglass *et al.*, 1966; Warrington, 1975; Warrington and McCarthy, 1987; McCarthy and Warrington, 1988) and recent results of neural network modelling (Small *et al.*, 1995) suggesting that within-modality learned feature–entity relationships could be one organizational principle of human knowledge (*cf. Warrington and McCarthy*, 1992).

Two further aspects of the outlined concept of associative tactile agnosia might be noteworthy. The given concept merges perception and recognition and therefore seems suitable to explain an unilateral gnostic deficit as observed in the described case. It further provides an explanation for associated minor disturbances of both motor behaviour and perception with a clinically predominantly agnosic deficit. Interactions between perceptual and motor parts of the sensorimotor apparatus involved in TOR (*see* Roland and Larsen, 1976) lead to reciprocal relationships between subfunctions. Malfunction in any part of the network might alter processes in any other part. Therefore, the model overcomes long-standing obstacles against the acceptance of associative agnosias which had to be raised within the theoretical framework of stage models because of the frequent association of agnosia with (minor) perceptual deficits.

Conclusion

Along with other recently reported cases (Caselli, 1991; Endo *et al.*, 1992) the reported case confirms tactile agnosia as a clinical syndrome. The clinical diagnosis of tactile agnosia can only be made when many other functional causes of impaired TOR can confidently be ruled out. However, minor perceptual and motor deficits, when not severe enough to cause impaired TOR themselves, do not rule out the diagnosis; indeed, they might be expected as a consequence of the agnosic deficit.

Tactile agnosia is understood as an impairment of modality-specific meaning representations functionally caused by a disruption of feature–entity relationships within the tactile modality. This notion is supported by the casuistic demonstration of largely preserved tactile feature analysis and cross-modal matching of features while tactile identification of entities was impaired. The observed constellation speaks against the alternative explanation of agnosia proposed by Lissauer (1890) who viewed defective cross-modal associations at feature-level as a primary mechanism responsible for agnosia. The presumed interactive mode of function of the distributed network responsible for TOR, feature analysis and related motor behaviour allows for an association between minor perceptual-motor deficits

and tactile agnosia. The observed unilateral deficit speaks for a lateralized organization of the network responsible for TOR.

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