G R A V I T Y A N D M A N

PART I. STUDIES ON THE EFFECT OF INCREASED GRAVITATIONAL FORCE ON MAN.

PART II. THE EFFECTS OF OTHER PHYSIOLOGICAL STRESSES ON MAN'S TOLERANCE TO INCREASED GRAVITATIONAL FORCE.

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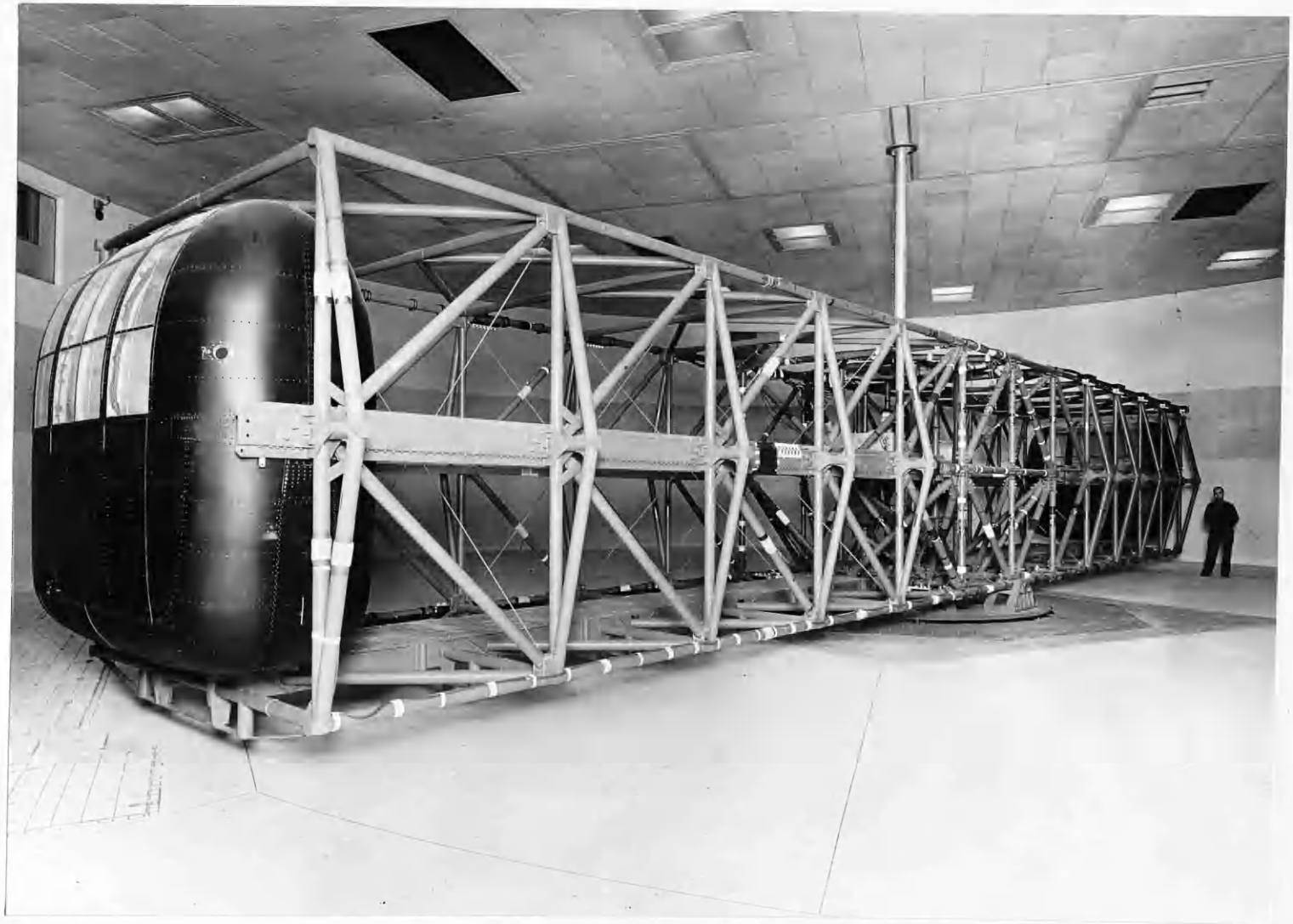


Frontispiece: The Human Centrifuge at the R.A.F. Institute of Aviation Medicine, Farnborough.

INTRODUCTION.

The immediate human problems of high speed flight are acceleration, environmental temperature and mental stress. All occur to some extent in every sortie, their relative importance depending on the flight pattern. In order to predict the human limitations it is necessary to determine man's tolerance to these stresses and to examine any other factors which may influence their effect.

This thesis presents the results of a number of experiments designed to examine human tolerance to positive acceleration. This type of acceleration, which will be more closely defined in the following paragraphs, may be considered as that which produces a force acting from the head to the feet, i.e. an increase in gravitational force. The simplest subjective example is the impression of increased weight which is felt when ascending in an express lift. The latter part of this thesis is the results of studies on the influence of other physiological factors which commonly occur in flight on human tolerance to increased gravitational force. These factors are heat, hypoglycaemia, hyperventilation, breathing pure oxygen, the after effects of alcohol and the degree of filling of the stomach. It is appreciated that most of the experiments performed deserve fuller examination and that many other physiological parameters require to be measured. This work however was of an applied nature and the requirement was for an urgent

survey of the whole problen rather than a detailed analysis of any one facet.

Force is defined by Newton's first law as that which modifies, or tends to modify, either the state of rest of a body or its uniform motion in a straight line. Newton's second law provides the fundamental basis for the measurement of force and, when the units of measurement have been suitably chosen, may be reduced to the equation:

$$F = m a \qquad (1)$$

where a is the acceleration produced in a body of mass m by the action of a force F.

This equation can be applied to calculate not only the forces producing linear accelerations, but also the forces involved when a body is made to traverse a curved path. Here, the direction of motion is being changed continually, and, by Newton's first law, a force is required to do this. When a body traverses a curve, radius r, with velocity v, it can be shown that the continuous change of direction is equivalent to an acceleration of amount v^2/r (called the centripetal acceleration) directed towards the centre of the curve; i.e. perpendicular to the direction of motion at any particular instant. Hence the force required to provide such an acceleration for a body of mass m is $m v^2/r$. This force acts in the same direction as the acceleration (towards the curve's centre) and is called the centripetal force.

The effect of inertia is to introduce a force exactly equal in magnitude, but opposite in direction, to the centripetal force. This opposing force, called the centrifugal force, would cause the body to fly off at a tangent to its curved path if the centripetal force were to be suddenly eliminated. It is this centrifugal force which is of importance in flight.

Forces due to acceleration, whether linear or centripetal, can never act in isolation except in the hypothetical case of an object removed to an infinite distance from all other matter. In practice, whatever other forces there may be, the force of gravity is always present.

The quantitative basis of Newton's work on gravitational attraction was his assumption that "every particle of matter in the universe attracts every other particle with a force inversely proportional to the square of the distance between the two particles". He also showed that a sphere of uniform density acts on an external body as if all its mass were concentrated at its centre. For the gravitational force, W, between the earth, mass M, and a comparatively small body, mass m, distant R from its centre, Newton's law of attraction gives the equation :-

$$W = G \frac{Mm}{R^2} \qquad (2)$$

where G is the 'constant gravitation'; when the other quantities are measured in c.g.s. units (i.e. M and m in g.,

R in cm. and W in dynes), G = 6,670 x 10^{-8}. The force with which any body is attracted by gravity towards the earth's centre is defined as the weight of that body. Equation (2), thus, gives the weight of a body of mass m, situated at the earth's surface, in terms of the earth's radius and mass.

It has been established that, if the effects of other forces such as air resistance can be excluded, all bodies fall to the ground with the same acceleration, irrespective of their mass. Thus it has been demonstrated that a very light body and a heavy one fall with the same acceleration when the experiment is done in vacuo: This acceleration for free fall is a characteristic of the locality at which it is measured. It is called the 'acceleration due to gravity' and is represented by the symbol $\underline{g}$. The conventional value of 32.2 ft./sec.^{2} is sufficiently accurate for normal use. By Newton's second law of motion (equation (1)) the weight W of a body of mass m may be expressed in terms of $\underline{g}$ as:

$$W = m\,\underline{g}.$$

Since $\underline{g}$ is also the force with which gravity acts upon unit mass, it is sometimes referred to as the 'intensity of gravity' at a given point.

If one equates the weight of unit mass in terms of the earth's mass and G (equation (2)) with the weight of unit mass in terms of $\underline{g}$, then at the surface of the earth:-

$$\underline{g} = G1 \frac{M}{R^2} \qquad (3)$$

R being the earth's radius. That $\underline{g}$ is the same for all bodies at the same locality, is therefore demonstrated to be a consequence of Newton's laws of motion and his law of universal gravitation.

Equation (3) is approximate to the extent that the distribution of the earth's mass is not completely symmetrical with respect to its centre. Also the relatively small centrifugal force due to the earth's rotation has been ignored. The magnitude of this force depends on latitude, but its effect never reduces true gravity by more than a few parts in a thousand.

The main purpose of this digression on gravity has been to explain what it meant by the term 'weight' in its usual sense and by 'acceleration due to gravity'. Also it will be clear from equation (2), that, with the range of altitudes possible at present with manned aircraft, overall variations in the force of gravity due to change of distance from the earth's centre cannot exceed 1 per cent and are therefore insignificant from a physiological point of view. For example, the error of assuming $\underline{g}$ to be that at the earth's surface for an aircraft flying at an altitude of 100 mls. would be less than one part in 1300 (Byford 1958).

By Newton's third law of motion, which states that action and reaction are equal and opposite, the weight of an object

such as the human body is opposed by an equal and opposite reactive force exerted by the floor, chair, or other support, preventing motion with acceleration g towards the earth's centre. It is this reactive force which produces the sensation of weight. Not only is it responsible for pressure on that part of the body in contact with the support, but, by preventing motion, it sets up stresses throughout the body. These stresses arise from the restraining forces exerted upon any particle by its neighbours. In a solid such forces are elastic in nature; in a liquid hydrostatic pressures are produced, calculable in terms of the weight of a vertical column per unit area (viz. pgh, where p is the liquid density, and h is the height of the column).

Consider now a case in which the stresses due to the action of gravity can be reduced to an abnormally low level. Imagine a passenger in a lift that can be given a constant downward acceleration, a (less than g), for an indefinite period by means of some device which introduces an increasing downward force to balance the increase of air resistance with speed. Applying Newton's second law of motion to the passenger, mass m, the resultant force acting upon him must be $m \times a$. The two components of this resultant are the man's weight, mg, acting downward and the total upward reactive force, P, exerted by the floor on his feet. Thus we have the equation:-

$$mg - P = ma.$$

The reactive force is m(g - a), therefore, and although the force with which gravity acts on the man (i.e. his true weight) is still mg, he is now living under conditions corresponding to an acceleration due to gravity of only (g - a). He lifts weights more easily, stresses on his body are reduced, and all hydrostatic pressures are reduced. Physiologically, in fact, the effect is the same as if he had been transferred to a smaller planet exerting a smaller force of gravity of intensity (g - a), and the force m (g - a) will be called his 'effective weight' under these conditions.

It is therefore convenient to use the intensity of the gravitational field which would produce the 'effective weight' as a measure of the mechanical stresses, and to use g, the intensity of the gravitational field when the subject is at rest on the ground, as the unit of measurement. For example, in the case of the lift just considered, we say that the man is subjected to ng, where n is a fraction equal to (g - a)/g. In general the magnitude n can vary from zero upwards according to the conditions.

The direction of the effective weight with respect to the body is of physiological importance and by convention the g is said to be positive or negative respectively, according to whether it acts from head to foot or from foot to head. Thus a person standing or sitting on the ground would be subjected to + 1g, but if he were suspended head-downwards from a beam he

would be subjected to -1g.

The "effective" weight of an individual (or the number of g to which he is subjected) is the resultant of the acceleration of the seat or other support with which he is in contact, and the acceleration due to gravity. This applies whether the acceleration be linear or centripetal.

In flight both linear and centrifugal forces occur commonly, but with the occupants seated in the normal manner the former is of little importance. It is the centrifugal force which occurs when the aircraft is turned which causes the important physiological changes and this force most commonly acts from head to feet, when, as has been pointed out above, it is given a + sign and is referred to as positive g.

The first part of the study was devoted to obtaining basic information on the various effects of positive acceleration, and from it to devise a method of measuring human tolerance which would be safe, repeatable, accurate, and capable of being used by all other acceleration laboratories. The results of these experiments are contained in part I of the thesis.

Part II of this thesis is an investigation of the effects of various other stresses on tolerance to acceleration. There is an increasing number of cases of unconsciousness in flight occurring in all air forces for which no simple

explanation can be found. In the author's view one possible cause might be a summation of stresses each of which is sublimonal in itself. The experiments described in part II were designed to test this hypothesis.

CHAPTER II.

METHODS AND MATERIALS.

Only the general principles of centrifuge techniques and human instrumentation will be given in this chapter. The more detailed specific methods will be discussed in the individual sections.

Although it is possible to study the effects of positive acceleration in flight, reproduceable runs are impossible to achieve and full instrumentation is difficult. To overcome these limitations the types of acceleration encountered in flight can be reproduced at ground level by means of the human centrifuge. The first human centrifuge was built in Berlin at the beginning of the nineteenth century - (Plate I), and was used in the treatment of psychological disorders. Patients were spun so that they underwent negative g which must have been of the order of -4g. Most patients maintained they were much improved and required no further treatment. The rationale of this treatment would seem to be as dubious as our modern electroconvulsive therapy and the results as good!

The first human centrifuge to study aviation medicine was built at Templehof outside Berlin by von Diringshofen in 1932. This machine by modern standards was a miniature having a radius of only 9 feet and a peak g of 20g. Nevertheless it performed its purpose well and much valuable data was derived from it. At the beginning of the Second World War in 1939

Germany had a seven year lead in acceleration studies. After the advent of hostilities a number of centrifuges were constructed in the New World; the first was completed in Toronto in 1941 and was followed rapidly by others at Wright Field, Ohio, at the Mayo Clinic, and at Pensacola, Florida. The performances of these wartime machines are shown in table 1. Four more major centrifuges have been built in the western world since the end of the war; the U.S. Naval centrifuge at Johnsville, P.A., the Swedish Air Force centrifuge at the Karolinska Institute, Stockholm, the French Air Force centrifuge at Bretignie, and the R.A.F. centrifuge at the Institute of Aviation Medicine, Farnborough. These machines profit from experience gained on the earlier versions and represent a great advance in engineering skill; the most significant improvements are in the range of performance available and the accuracy with which the runs can be controlled. The performance figures for these centrifuges are also shown in table 1.

TABLE 1.

A COMPARISON OF THE PERFORMANCES OF VARIOUS HUMAN CENTRIFUGES.

Centrifuge.	Maximum g.	Maximum rate of application of acceleration g/sec.
Templehof	20	2.0
Japanese	25	1.7
Toronto	20	2.0
Mayo Clinic	18	2.0
University of Southern California	18	2.0
U.S.A.F. Wrightfield	20	6.0
U.S.N. Pensacola	15	2.0
U.S.N. Johnsville	40	10.0
Karolinska, Stockholm	30	3.5
R.A.F. Farnborough	30	4.0
F.A.A. Bretignie	15	10

In this study the human centrifuge at the R.A.F. Institute of Aviation Medicine was used (see frontispiece).

The centrifuge consists of a 60 ft. arm carrying at each end a gondola mounted on gimbals. On rotation the gondolas tilt so as to position themselves in the resultant of the centrifugal and gravitational g; consequently the subject experiences positive g. The arm is driven by a direct drive from a vertically mounted electric motor of 2800 H.P. which is controlled automatically by a cam. The cams are available in families. Each gondola has facilities for full instrumentation having 30 available channels for electrical recordings. The signals from the gondolas are led along the arms to the centre section where the leads ascend the central shaft in co-axial cables to the recording room two floors above. From the shaft the impulses are led out through silver-graphite slip rings and mercury troughs to a distribution board on which the amplifiers and recording apparatus are also represented thus enabling an experiment to be set up entirely on the single board. (Plate II).

The centrifuge is operated from the control room (Plate III) which overlooks the actual centrifuge chamber. Although manual control is available the method of choice is the use of families of programme cams. The variables in acceleration are peak g, rate of onset of g, and duration

of peak g. The first of these is determined by the total cam drop, that is, the depth of the step of the cam. The rate of onset is controlled by the rate of cam drop, i.e. the shape of the cam, and its rate of rotation. The latter is preset on an electrical timer. Thus a separate cam is essential for each peak g, and for varying rates of application one must either have different cams or a means of rotating a given cam at a large number of rates. In fact a combination is used. The motor driving the cam is capable of four speeds, one revolution lasting 9, 12, 18 or 30 seconds, thus giving four rates of application for each cam. Three families of cams are available, the first a constant time series, take 4.5 seconds to reach peak g from $\sqrt{2}$g when rotated at the 9 second rate. Thus the rate of application is continually changing and the rate of change varies with the peak g. The other two families are constant rate cams; one having a rate of 3.6 g/second when rotated at the 9 second rate, the other a rate of 1 g/second at the 9 second rate. A total range of constant rate cams is thus available of 3.6, 2.7, 1.8, 1.08, 1.0, 0.75, 0.5, 0.3 g/second. For completeness an auxillary motor has been constructed giving a rate of application of acceleration of 0.1 g/second. Sample g/time curves for these cams may be seen in figure 1.

Thus for any run the choice of an appropriate cam gives control of peak g and rate of application of g while the duration may also be preset. Runs are therefore exactly

repeatable. Normally deceleration is produced by reversing the cam drive thus obtaining a g/time pattern which is the mirror image of the acceleration.

For a more detailed description of the R.A.F. centrifuge see Latham (1955).

The gondolas are shown in figure 2. They consist of cabins 9' x 8' x 4' constructed of duralumin with perspex windows. In each gondola is mounted an adjustable seat similar in design to those fitted in aircraft. The seat is fitted with head and arm rests which are adjustable, as are the foot rests. A standard four point suspension safety harness is incorporated in each seat. The gondola is connected to the general intercommunication system and is so arranged that the microphone is "live". Thus whenever the subject speaks he can be heard throughout the centrifuge building. An automatic tape recorder allows permanent records of the conversation to be made. The gondolas can be fitted with most aircraft systems such as an oxygen supply, anti-g suit air supply and instrument presentations. Four blocks of Plessey plugs allow 30 channels of recordings to be made. A closed circuit television is also incorporated in one gondola as an additional safeguard.

Acceleration is measured by a 'lan-elec' variable inductance accelerometer mounted on the subject's seat at heart level. Since the subject is seated in a seat the back

of which is raked 20°, and since at low g levels the gondola does not each the horizontal, the applied g does not act in the long axis of the body. The resultant however can be resolved into two components, one of which is in the long axis of the body and the other of small magnitude acting transversely. The latter is of little physiological interest and can be neglected. The true positive g is measured by mounting the accelerometer in the axis of the subject's body. In the gondola there is a g gradient, since the head and feet of the subject represent different radii. By mounting the accelerometer at heart level, which is always in the axis of the car gimbals an approximate mean is obtained from which the g at other positions can be calculated. In addition the heart represents the "centre" of the circulation and information obtained at the heart level can be translated most easily. The output of the accelerometer is displayed on an Ediswan pen recorder for monitoring purposes.

The subject is provided with an event marker which in some experiments can be used to provide an emergency stop mechanism.

Routine human instrumentation involved the recording of electrocardiograms. The leads routinely used are the bipolar lead I and unipolar leads aVF and V_1 . On some occasions all 14 leads have been used, i.e. leads I, II, III, aVR, aVF and leads V_1-V_8. All electrodes are placed in

accordance with the recommendations of the American Heart Association and the British Cardiac Society (fig. 3). The indifferent electrode used was that described by Wilson et al (1934). To obviate muscle artefacts and allow free use of the hands the two wrist electrodes were replaced by disc electrodes on the shoulders over the acromion. The potentials measured at the shoulders are very little different from those found at the wrists (fig. 4). In both cases the potentials are being recorded essentially from the root of the upper limb and any difference in length of conducting medium is of little significance when compared with the skin resistance.

Small non-polarisable silver electrodes were attached to the skin by "Nobecutane" (Browne 1957). In some experiments it was found necessary to use a 15 c.p.s. filter to reduce muscle artefacts occurring during convulsions of the subject. This causes a slight reduction in the amplitude of the more steeply rising parts of the wave form. A time constant of 0.3 seconds in the A.C. coupled part of the amplifier was used throughout.

In some experiments recordings of arterial blood pressure were made. The measurement of rapid changes necessitated the use of a direct method. A No. 1 needle was inserted into the radial artery at the wrist. The needle was connected to a capacitance manometer by a short length of polythene tubing. The needle was kept patent by a saline

drip through a needle valve in the inlet to the manometer. For recording from the manometer it was found convenient to use frequency modulation, the carrier frequency being 2 megacycles. The blood pressure was monitored on an Ediswan ink-writing recorder and for more accurate time and amplitude measurements was recorded photographically using 150 c.p.s. galvanometers. It must be noted that the absolute measurement of the blood pressure under acceleration is extremely difficult due to the effect of g on the fluid columns involved and in addition the manometers are g-sensitive. Readings at any point are therefore only accurate to within about 10%. Since the hydrostatic pressures developed are of importance the blood pressure at eye level is measured by supporting the wrist so that the needle is at eye level. Under these conditions the pressure found may be assumed to be of the same order as that in the retinal artery. Similarly the pressure at heart level is determined by supporting the wrist at the level of the heart. That these conclusions are justified may be seen by comparing the results obtained with those expected on theoretical grounds. Thus blackout occurs when the radial artery pressure is 20 - 30 mm.Hg. at eye level. This corresponds to the intraocular tension which acting in opposition to the pressure in the retinal artery causes flow to cease when the two become equal. The flow ceases in the retinal artery when blackout occurs as has been shown by

Duane (1953), who examined the fundi during g. (see plate V).

Records of respiration were obtained during several sets of experiments. The effects of g preclude any device such as a mercury filled strain gauge and in any case the chest could not be used because of the battery of E.C.G. electrodes. Two systems were used. When the subject was breathing through a face mask a pressure tapping was made in the lateral wall of the mask and connected to a Statham pressure transducer from which pressure differentials due to inspiration and expiration were recorded as a low frequency wave form on the pen recorder. From these records respiratory rate and a rough measure of amplitude could be obtained. When a mask was not being worn respiration was recorded from a thermocouple placed in one nostril. Changes in thermal e.m.f. due to respiration were recorded as a low frequency wave form in a similar manner to the E.C.G. This recording more closely resembled a square wave. The steeply rising part of the wave form was used as a signal to trigger manually a C.R.O. when recording vectorcardiograms; the details of this technique are described in appendix I.

Some form of yardstick is necessary to compare the tolerance of different subjects to acceleration. The most common method is to measure the level of g at which vision is impaired or lost. The figure obtained is called the g threshold. In experiments in which other stresses are being used in addition to acceleration this method of threshold

determination is unsuitable since there is a danger of unconsciousness supervening. A new technique was developed for these experiments which it is hoped will become standard in other laboratories. The new method will be described in chapter IX.

CHAPTER III.

PHYSIOLOGY.

The changes occurring in the occupants of an aircraft during positive acceleration are of two kinds, (a) tissue distortion, (b) haemodynamic effects. Both are due to an increase in the effective weight of the organs concerned.

(a) Tissue Distortion. This, although dramatic to the onlooker (Plate VI), carries little danger to the pilot and is of little importance. Where internal organs are displaced the effects produced are concerned with circulation and will be considered under that heading.

(b) Haemodynamic. Since the changes induced by positive g are primarily circulatory, they take time to develop, and there is always a latent period during which the pilot is free of symptoms apart from the sensation of increased weight. If the force is prolonged, visual symptoms occur and later progress to unconsciousness. The symptoms (fig. 5) can be ascribed to a progressive decrease in the blood supply to the retina and brain under sustained positive acceleration, and the physiological reaction is an attempt to compensate for these changes. The importance of the time factor in the production of symptoms becomes apparent if the physiological effects of g are compared with the effects of alterations in cerebral blood flow produced by other methods. Sudden arrest of the cerebral

circulation by means of cervical occlusive cuffs will lead to unconsciousness in 6 - 6½ seconds (Rossen, Kabat and Anderson 1943). The oxygen content of the blood entrapped within the brain at any time will therefore support cerebral function for this period.

The mechanism of regulation of the blood supply to any given part of the body is a simple physical one, and in the brain, enclosed within the rigid skull, the intracranial fluids form a closed hydrodynamic system in which very little volume or flow change can occur without proportional pressure changes in the intracranial vessels or the cerebrospinal fluid. There is increasing evidence that with low blood pressure intracerebral vascular resistance may be modified significantly in an effort to preserve a constant flow through the brain. It is known that under normal conditions however, intrinsic factors play only a minor part in the control of cerebral blood flow (Schmidt 1950). Normal visual and cerebral functions therefore depend primarily on a pressure of blood in the head sufficient to overcome the resistance of the cranial contents. If, for the moment, one neglects the all important function of the carotid sinus in the regulation of cerebral arterial pressure and considers the purely haemodynamic factors, then the blood pressure at head level is determined by the pressure required to support a column of blood between the

heart and head, and also by the pressure at the heart. The former factors bear a direct relation to the magnitude of the force due to the acceleration and is not susceptible to physiological compensation. On the other hand the blood pressure at heart level is controlled by a complex series of reflex mechanisms, so that it is to be expected that factors influencing cardiac output such as blood pooling and venous return, peripheral resistance and heart rate will have a direct bearing on the problem.

The hydrostatic pressure exerted by the arterial column of blood between the heart and the brain is proportional to its length measured in the direction of the applied force, and is only under the pilot's control by virtue of crouching or the adoption of other body positions which reduce the component acting through the longitudinal axis of the body. The length of this column of blood, measured in this manner, will henceforth be termed the "hydrostatic heart-brain distance". If one assumes a heart-brain distance of 30 cm., then the arterial pressure required at heart level to elevate a column of blood to the base of the brain is about 25 mm. Hg. at 1 g. Under the influence of 5g, however, it has been shown that the heart-brain distance may increase by at least 6 cm. due to stretching of the great vessels in the neck and thorax, and descent of the diaphragm. (Rushmer 1944). Therefore, just to maintain a column of blood 36 cm. in

length under an acceleration of 5g would require a pressure at heart level of 150 mm. Hg. Elevating the diaphragm whilst under the influence of 5g to its normal mean level would produce the equivalent of at least 1g protection. X-rays of monkeys subjected to positive acceleration have demonstrated descent of the diaphragm associated with an apparent lengthening of the portion of the inferior vena cava above the diaphragm. (Ruff and Strughold 1939). This lengthening will increase the difference between the intra-abdominal pressure and the venous pressure at heart level which affects the venous return from the lower part of the body. If the abdominal contents are considered to behave as a column of fluid, and the increases in abdominal hydrostatic pressure are calculated and related to the magnitude of the centrifugal force, the increments in pressure with increasing g fail to reach the values expected by almost 40%. The discrepancy between the calculated rise and the actual rise confirms the gradual descent in the level of the diaphragm due to abdominal protrusion and compression of intestinal gas, with the consequent reduction in the height of the fluid column.

Lambert and Wood (1946) measured radial arterial pressures in human subjects with the wrists at head level. They concluded that the fall in blood pressure at head level was directly proportional to the acceleration and corresponded to a systolic fall of 32 mm. Hg./g and a diastolic fall of

20 mm.Hg./g. The mean pressure fall amounted to 25 mm.Hg./g which is the pressure required to support a column of blood between the heart and brain under 1g. Dimming of vision was associated with systolic blood pressures at head level of approximately 40 mm.Hg., although with an excess intra-ocular pressure of 26 mm.Hg. it is to be expected that the retinal circulation will not fail until the local systemic blood pressure approaches this latter figure. Complete black-out occurred when the pressure fell to 20 mm.Hg. and unconsciousness when it reached zero. At heart level they noted that the systolic pressure fell approximately 4 mm.Hg./g, whereas the diastolic pressure was maintained, which might reasonably imply a fall in mean pressure of 2 mm.Hg./g. As cerebral blood flow may be more closely related to the mean arterial pressure, it is desirable to examine the pressure changes in the arterial column in these terms. Assuming a normal blood pressure of 126/85 at heart level, then excluding effects due to descent of the diaphragm, one would expect blood flow to show significant impairment when accelerations of 4g were attained. This has been demonstrated in animals (Fischer 1937, Ranke 1937, Koenen and Ranke 1937, Greenfield 1938, and Britton, Pertzoff, French and Kline 1947), however average relaxed human thresholds appear higher than this. In addition, it has been shown that if the acceleration is sustained some

recovery may ensue after 6-12 seconds at the peak g. (Ranke 1938, Greenfield 1938, Mayerson 1942, Rose, Kerr and Kennedy 1942, Sturm, Wood and Lambert 1945, Britton et al 1947, Browne and Fitzsimons 1956). Although it is apparent that the cardiac output and hence blood pressure at heart level can be maintained at or above normal under these conditions, there is an additional factor concerned in the maintenance of an adequate cerebral blood flow at subthreshold levels of acceleration. Henry, Gauer, Kety and Kramer (1951) likened the cerebral arteriovenous circuit to an inverted 'U' tube system. In experiments on human subjects exposed to positive g they measured arterial pressures at eye level and internal jugular bulb pressures. The results appeared to indicate that the negative pressures developed within the intra-cranial veins compensated to some extent for the fall in cerebral arterial pressure. Thus the arteriovenous pressure differential across the brain was maintained and hence an adequate cerebral blood flow also.

This explanation depends upon the pressure in the jugular bulb being equal to that in the intra-cranial venous sinuses. This was assumed by the authors but has not been established experimentally.

The cerebral arteriovenous oxygen difference gave an indication of blood flow and it appeared that a marked decrease in cerebral venous oxygen saturation occurred only

when acceleration of 4.5g were reached. The authors pointed out that such a mechanism was ineffective in supporting cerebral circulation when the arterial pressures at the base of the brain dropped to zero. A hydrodynamical concept of the heart-brain circulation would theoretically permit blood flow at all values of acceleration if the blood vessels were considered as rigid tubes. This is not the case, and when the pressure on the arterial side drops to zero, collapse of unsupported vessels on the venous side, with the establishment of a fluid level, must presumably occur. On the other hand, under positive g the extensive internal vertebral venous plexus is prevented from collapse owing to the subatmospheric C.S.F. pressure which may therefore allow an uninterrupted blood flow and account for the maintenance of a high arteriovenous pressure differential in the brain. These theories were also subscribed to by Åkesson (1948) who reported them prior to J. P. Henry.

If the acceleration is applied rapidly to a peak above the level at which cerebral circulation ceases, it has been found that the time to produce unconsciousness is approximately the same regardless of the peak acceleration, up to a maximum of 15g (Beckman 1953). The mean time above values of 3g during accelerations leading to unconsciousness, appears to be 4.2 seconds which is shorter

than the conscious survival times when cerebral circulation is stopped by the application of a cervical cuff. Beckman attributed this difference to the fact that under high positive g loads the total quantity of blood entrapped within the brain after cessation of cerebral circulation was less. It must be noted however that high negative C.S.F. pressures can be assumed under these conditions and this fact may be more directly related to the discrepancy.

The above considerations have so far presupposed a constant blood pressure at heart level, but this is affected by many physiological variables which show marked changes under the influence of positive acceleration.

In the lower half of the body the essential change during positive acceleration is an increase in the hydrostatic pressure gradient along the blood vessels which results in a circulating blood loss due to pooling. The degree to which physiological mechanisms can compensate for this failure of venous return will determine the right heart filling pressure and hence cardiac output and mean blood pressure at heart level. In this case, as the stress and its effects have a mechanical basis, compensation can be augmented by mechanical devices or voluntary efforts such as breath holding and straining. Under such conditions subjects have been known to maintain a blood pressure at heart level of over 200 mm.Hg.

The onset of acceleration is accompanied by an immediate rise in pulse rate which may take 10-30 seconds to reach its maximum value (Ruff 1938) in the region of 195-220. There does not appear to be any relation between the pulse response and g tolerance or the severity of symptoms. During recovery there may be wide fluctuation in the rate but this is followed by a bradycardia. With the descent of the contents of the thoracic cage changes are seen in the electrocardiogram whose interpretation varies widely in the literature. The analysis of these changes will be discussed in chapter V.

It is the venous return, however, which will have the greatest influence on cardiac output. The extent of total blood pooling in the abdomen has not been accurately assessed, but the pooling in the lower limbs has been measured by Lambert (1946) during human centrifuge experiments, using leg plethysmographs. It would appear that under the influence of 5g for 15 seconds the total leg volume increased by only 60 ml. Chest x-rays during such centrifuge runs have demonstrated an appreciable reduction in heart size (Ruff and Strughold 1939, Rushmer 1944) - a result of the progressive failure of venous return. The rate of blood pooling in the lower limbs is governed by the peripheral vascular resistance as the venous valves prevent retrograde flow. The rise in venous pressure in the

lower limbs is slow and progressive depending on the inflow of blood from the arterial side. Thus the pooling requires a period of time to develop depending on the degree of peripheral vasoconstriction. In this connection Greenfield and Patterson (1954) have shown that there is a peripheral vasoconstriction which follows a local response to an increased transmural pressure on the arterial side of the circulation. They also indicate however that when the increase in transmural pressure exceeds 150-200 mm.Hg. there may be a large persisting vasodilatation which in consequence would lead to high arterial flow rates in the lower limbs under g with more rapid shunting of the blood into the venous side of the circulation. It is noteworthy that under 5g the systolic arterial pressure at ankle level may reach 350-400 mm.Hg. The rise in venous pressure in dependent parts of the body produces a fluid loss with a demonstrable haemoconcentration, but the magnitude of this phenomenon in relation to the time of exposure makes it of only academic significance. Petechial haemorrhages in dependent parts of the body during short exposure to high peak accelerations have rarely been described.

It is found that if g is maintained, the period of general deterioration of the subject lasts approximately 7-10 seconds, after which a limited degree of compensation takes place. It is difficult to see how the extent of the

abdominal pooling can be accurately evaluated, but any factor which assists venous return to the thorax has a profound effect on the cardiac efficiency. In this respect, intra-abdominal and intrathoracic pressures are of great importance. The production of an abdomino-thoracic pressure gradient, by respiratory efforts related to the Valsalva manoeuvre (i.e. forced expiration against a closed glottis) in order to facilitate venous return, have been studied by Rushmer (1944). These physiological implications are discussed in relation to methods of protection. Some fundamental work by E. H. Wood et al (1943) does throw some light on the significance of blood pooling, venous return and right auricular filling pressure. It can be shown in the human centrifuge that if the lower half of a subject is immersed in a water bath up to chest level, then during positive g hydrostatic counter-pressure is applied to the surface of the body which exactly opposes the pooling tendency. The experiments showed that the water level which gave maximum protection was critical. The average visual protection with water immersion to the xyphoid was 0.8g whereas with the water level at the 3rd costal cartilage rib the protection averaged 1.7g. On theoretical grounds, simple elevation of the diaphragm to its normal mean level should give a maximum of only 1g protection. The remaining 0.7g protection must be attributed to an increased cardiac output and raised systolic pressure at heart level.

This follows the reduction of circulating blood volume in the lower half of the body when counter-pressure is applied, not only during positive acceleration but also in the resting conditions.

On exposure to values of g which do not produce visual symptoms there is no demonstrable disturbance of C.N.S. function. If these exposures of g are repeated several times in one day they are followed by lassitude and exhaustion, the physiology of which is unknown. As a result of this, six centrifuge runs per day are the absolute maximum allowed for any subject. This tiredness may be the result of minimal cortical anoxia and has been compared with the fatigue following collapse on a tilting table.

The further sequence of events begins with a greying of vision manifested by an impairment of contrast discrimination, and the black-out threshold occurs when the sensation of light is completely lost. As a result of 215 centrifuge trials Lambert (1944) showed that when visual symptoms occurred they took place approximately 6 seconds after maximum g was reached. He further stated that if symptoms are going to appear on a given centrifuge run they happen before the tenth second. Hemianopia or scotomatous impairment of the visual fields has not been reported. Whilst consciousness is retained, oculo-motor functions are unaffected in common with other

co-ordinated movements, and although Stewart (1945) noted a left esophoria under g loadings, it could be overcome by voluntary effort and was ascribed to muscular imbalance. Nystagmus has never been observed.

Other sensory functions such as touch, pain, pressure and temperature are not markedly impaired, even when black-out lasts as long as 12 seconds, and they are only lost with the onset of unconsciousness. This final stage is heralded by a rapid loss of function of nervous tissue progressing to the total abolition of all motor and sensory functions. As is found in cases of anoxia, the subject often does not appreciate that consciousness has been lost unless there is some change in the environment. Thus, if consciousness is regained while the centrifuge is still rotating the subject will state that he feels fine and that nothing unusual has occurred. It is only if consciousness is regained when the centrifuge has come to a stop that the subject realises he has been unconscious, and often the presence of injuries is the final proof. The gross mental changes associated with high g just prior to, during and following unconsciousness may be manifested by confusion, dreams, amnesia, paraesthesia, or convulsive episodes. Local minor episodes are not uncommon, but clonic fits are not necessarily associated with an abnormal resting E.E.G. Recovery is usually rapid but complicated by disorientation,

confusion and dreams, which may last up to 20 seconds.

It will be seen that there are many ways in which the effects of g may be potentiated leading to a premature onset of black-out or unconsciousness.

CHAPTER IV.

HUMAN TOLERANCE TO ACCELERATION

Human tolerance to acceleration depends basically on the strength, duration, direction and rate of application of the applied force. From the discussion on physiology it can readily be understood that individual tolerances can be modified by various protective devices, such as crouching to diminish heart-brain distance or tensing the abdominal and leg musculature to maintain venous return. To obtain a base line for comparison of subjects, and to allow evaluation of methods and materials, it has been generally agreed that human tolerance to positive g should be measured with the subjects relaxed and in a normal sitting position. Since unconsciousness is not desirable in routine experiments the end point used for determining tolerance is grey-out or black-out. The g at which symptoms appear being called the threshold.

Grey-out and black-out, although commonly used by the aviator, are confusing terms and deserve some explanation. The sequence of visual changes which precede unconsciousness are as follows :-

(1) There is a general dimming of the visual fields associated with diminution of contrast discrimination and impaired colour vision.

This stage is called "greying" and lasts for a variable length of time. It immediately precedes :-

(2) Grey-out - this stage is characterised by loss of peripheral vision, central vision being unimpaired. It is associated with an arterial blood pressure at head level of 40-60 mm. of Hg. If the fundus is examined during this 'coning or tunnelling of vision' the vessels are seen to be pulsating, the arteries being empty during diastole and filling with each systole. If the g is increased this stage gives way to :-

(3) Black-out (amaurosis fugax). At this point vision is completely lost although consciousness is maintained. There are no changes in motor or sensory functions with the exception of vision which is completely abolished. Any increase in the applied force causes loss of consciousness.

The physiological processes underlying black-out are still controversial. The early German theories of deformation of the brain, compression of the optic chiasma as the brain slides over the tentorium, and intra-cellular migration of mitochondria were all unlikely and can now be disregarded. The modern view is that blackout is due to anoxia secondary to a failure of the blood supply. The controversial point is whether the primary failure is in the

retina or in the brain itself. The exponents of the latter view have received most support from the anatomical work of Young (1946). Briefly his theory is that with progressive acceleration there is a diminished blood supply to the occipital cortex from the vertebral arteries. At the same time there is an increased outflow from the dural sinuses via the jugular veins. Thus there is a decrease in the volume of the cranial contents offsetting the cushioning effect of the C.S.F. The brain is displaced in the direction of the applied force and compresses the vertebral vessels leading to a further reduction in blood flow to the visual cortex. In his view, this results in the visual cortex being deprived of its blood supply before the rest of the brain.

The other school of thought places the loss of function in the retina itself. Since the intra-ocular pressure is 20-30 mm.Hg., then flow might be expected to cease in the retinal vessels when the arterial pressure at eye level reaches this figure. This has been verified repeatedly and ophthalmoscopic examination of subjects reveals emptying of the blood vessels coincident with blackout (Bietti and Scano 1943, Duane 1953). It seems not unreasonable to suppose that the blood supply to the retina ceases when the blood pressure at head level is still adequate to maintain the cerebral circulation. Further weight is added to this argument from

studies on pressure blindness. When pressure is applied externally to the eyeball loss of vision follows the same time course as during acceleration (Lambert and Wood 1946, Howarth 1956). The pressures necessary to cause this blindness agree with those occurring during g (Andina 1937), while the last point of the visual field to be seen is parafoveal in pressure blindness (Howarth 1956) as it is with acceleration (Latham - unpublished observations).

The most recent work which bears on this problem is that of Lewis and Duane (1955) who have shown that the electro-retinogram persists during blackout as do the direct and consensual light reflexes. It is generally agreed that the electro-retinogram is derived from the receptor cells of the retina (i.e. the rods and cones) although part of the complex may arise from the other nervous elements involved. The retina would thus appear to respond to light although vision is lost. This is borne out by the presence of the light reflexes. Consideration of the pathways involved should therefore suggest the point where transmission is interrupted. Fig. 6 shows diagrammatically the visual pathway. The fibres in the optic nerve pass back through the chiasma to relay in the lateral geniculate body of the same side as their retinal origin. The next set of fibres (the optic radiation) travel via the retrolentiform part of the internal capsule to end in the visual cortex on the occipital pole of the cerebrum.

The path of the fibres subserving the light reflex is shown in fig. 7. The fibres pass medial to the geniculate body and via the superior colliculus to the pretectal nucleus where they synapse with fibres going to the Edinger-Westphal nucleus. At first sight it would appear that the block must be at the lateral geniculate body or in the higher centres, the latter probability being the more likely. However on five subjects I have shown that during the course of pressure blindness a light stimulus presented immediately after black-out will elicit a consensual light reflex, although further pressure abolishes this latter response also. In this case it is difficult to see by what mechanism pressure on the eyeball could influence the function of the lateral geniculate body or the occipital cortex. A possible explanation of the apparent contradiction in these results has been advanced by Lewis and Duane (1956). These authors have pointed out that vision has three cells and two synapses in the retina whereas the afferent pathway for the light reflex has but two cells and one synapse (fig. 8). Moreover the extra cell and synapse in the visual pathway are those of the ganglion cell layer which lies in the inner part of the retina supplied by the arteria retinae centralis; whereas the outer structures of rods, cones and bipolar cells are supplied by a plexus of vessels from the choroidal circulation. Thus when the blood flow of the central artery becomes zero

at 20-30 mm.Hg. arterial pressure the ganglion cell layer becomes anoxic although the outer layers have still an adequate blood supply. This theory explains the persistance of the electro-retinogram and light reflexes during black-out due to either g or external pressure. Grey-out is explicable in the same way since as the pressure in the retinal artery falls the flow becomes less and the peripheral part of the retina will have a reduced blood supply with a lower oxygen content.

In conclusion it may be said that although there is some indication of the loss of vision being central in origin the weight of evidence points to failure of conduction in the retina, the most probable site being the ganglion cell layer.

The purpose of the digression into the origin of black-out has been to establish its primarily haemodynamic nature in the normal subject during acceleration. It must also be borne in mind that changes in the vessels of the retina follow closely those of the cerebral vessels and changes in calibre may arise from many varied physiological conditions. In addition, in some special cases there may be upset of the higher centres although the retinal blood flow is adequate to support vision. This will be discussed in Part II.

The threshold depends on several physiological factors in addition to the mechanical ones already mentioned. Thus the variables involved are heart-brain distance, blood pressure at heart level, intra-ocular tension, peripheral resistance, and cardiac output. Other more tenuous variables are physical fitness, somatotype and temperament, a short stout subject has a better tolerance than a tall slim one, other things being equal, while the athlete has a higher threshold than the student. Age also plays a part, tolerance decreasing with age although this effect may be due to diminished physical fitness and lowered cardiac reserve rather than age per se. Depending on the foregoing, there is a large day to day variation in threshold which is also related to fatigue. With so many variables it is often difficult to decide which factors are operative at any one time and this has led to many misconceptions in the past. It is interesting to note that practice does not improve g tolerance but tends to decrease it.

These physiological variables tend to be static in any one individual and his tolerance is then determined by the strength, duration, direction and rate of application of the force.

1. Strength and duration.

In the early days of studies on acceleration, large numbers of threshold determinations were made in an attempt

to obtain a normal range. To obtain uniformity most centrifuges used a method whereby the subject was brought from 1.0g to peak g in 5 seconds regardless of the value of the peak g. Much valuable data was obtained in this way. From these results strength/duration curves could be constructed which would outline the limits of human tolerance. Fig. 9 shows the curve obtained at this laboratory compared with those of several American sources. It will be seen that the curves, although having similar characteristics, are widely displaced and tend falsely to suggest that certain areas produce people with a higher tolerance than others. This displacement is due largely to differences of technique between various laboratories and the use of different end points. More interesting is the fact that the curves all deviated from the general form of physiological strength/duration curves in having a secondary rise to the right of the graph, this occurs at about 6-10 seconds after peak g is reached. Thus a subject who greyed out after 5 seconds at 3.0 g might expect his vision to clear again at 7 seconds. This phenomenon only occurs at low values of g and would seem to be explicable by compensatory mechanisms.

To investigate this problem several centrifuge runs were carried out during which the blood pressure was measured at eye level. Pulse rate was obtained in a further 200 runs from the electro-cardiogram. A sample result can be

seen in fig. 10. It was found that as the acceleration began the blood pressure at eye level rapidly fell. If the S.B.P. reached 40 mm.Hg. then grey-out occurred and if it fell below 20 mm.Hg. then black-out rapidly followed. This agreed with the findings of Lambert and Wood (1946). From the electro-cardiograms it was found that the maximum pulse rates were reached 6-10 seconds after peak g, as had been noticed by Rose et al. (1942). The rise in pulse rate followed closely the fall in B.P. which commenced to rise again as the pulse rate reached levels of 120-130 per min. Following the rise in blood pressure the pulse slows again until the two stabilise, the final degree of tachycardia and hypotension depending on the g. Following the rise in blood pressure the visual symptoms disappear. One subject was run while the blood pressure was measured at heart level. The results obtained are shown in Fig. 11. It will be seen that the pressure at this level showed a slight initial fall followed by a rise. Recovery is rapid following cessation of g.

The secondary rise in the strength/duration curve would thus appear to be due to cardio-vascular reflex activity. The most probable mechanism is that the hydrostatic fall in blood pressure at head level stimulates the pressure sensitive areas of the carotid and aortic arteries leading to increased cardiac rate and output, which in turn raises the systemic blood pressure and restores the circulation to

the retina. This compensatory rise obviously depends on an adequate venous return to the right side of the heart and this in turn is limited by pooling. Thus this transient visual phenomenon is only found at lower values of g, since at higher levels venous return is inadequate to allow full compensation.

2. Rate of application.

The earlier work on threshold determinations did not take account of the rate of application of acceleration. In the common method of using a time constant of 5 seconds to reach peak g, each threshold level was reached by a different rate of rise of acceleration (this quantity $\frac{dg}{dt}$ is called 'jolt'). That there will be a difference in threshold under these circumstances can be readily appreciated by considering the physiological changes occurring under extreme conditions. If the jolt is very high, for example 200 g/sec. then failure of the body is mechanical since circulatory changes have no time to occur. At 5-6 g/sec. grey-out, black-out and unconsciousness succeed each other rapidly as compensatory reflexes do not have time to develop completely. At the other extreme, very low rates of application, for example 0.01 g/sec. also lead to a low threshold since although there is time for full reflex activity, the long time lapse also allows pooling to occur and so venous return is impaired and the heart cannot make

the appropriate response. Between these extremes lie the range of values which commonly occur in aircraft, varying from 0.1 g/sec. to 4.0 g/sec., and it is in this range that determinations of threshold are made in the human centrifuge. Variations in threshold within this range have previously been demonstrated (Browne and Fitzsimons 1956) using constant time cams. A further investigation was made into alterations in threshold with varying rates of application of g using cams giving a constant rate of rise above $\sqrt{2}g$.

Method and Material.

The rates of application of g were 3.6g/sec., 2.7g/sec., 1.8g/sec. and 1.08g/sec. The centrifuge was accelerated to a fixed g and maintained at a constant acceleration for 15 seconds and then decelerated to rest. Runs were repeated at 0.2g intervals until a grey-out threshold was found, a different rate of application was then substituted and further runs made until a new threshold was found. This was carried out for each of the four rates of application. In each set of runs the varying rates of application were presented in a random order and the subject had no knowledge of which run he was carrying out. Three minutes were allowed between runs to allow pulse rates to return to normal.

A few experiments were performed separately using rates

of application of 0.1g/sec.

All subjects were experienced on the centrifuge except two. These inexperienced subjects showed higher thresholds, as has been found before, but have been included here as the results represent relative differences rather than absolute values.

Grey-out was used as the end point and was measured by peripheral light loss. In all cases the initial grey-out was used although at the slower rates of application further grey-outs do sometimes occur (Browne and Fitzsimons 1956).

[illegible]	[illegible]	3.7	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	4.0	4.0	4.2	[illegible]
Means	[illegible].6	3.3	3.9	4.1

It will be seen that there is a trend towards higher thresholds as the rate of application decreases. This trend is statistically significant at the level of P = 0.[illegible] [illegible]

RESULTS.

TABLE 2.

GREY-OUT THRESHOLDS AT DIFFERENT RATES OF APPLICATION OF g.

Subject	Rate of application g/sec.			
	3.6	2.7	1.8	1.08
P.H.	3.3	3.4	3.4	3.6
K.B.	3.4	3.6	3.7	4.0
J.F.	3.6	4.0	4.0	4.2
F.L.	3.5	3.5	3.6	3.9
M.B.	3.2	3.4	3.4	3.8
A.C.	3.7	4.0	4.1	4.4
G.A.	3.6	3.7	3.8	4.0
J.B.	3.7	3.8	4.0	4.2
R.H.	4.3	4.5	4.6	4.8
B.M.	4.0	4.0	4.2	4.4
Means	3.6	3.8	3.9	4.1

It will be seen that there is a trend towards higher thresholds as the rate of application decreases. This trend is statistically significant at the level of P = 0.001. The difference between the highest and lowest rate of application was 0.5g, which corresponds to the figure found previously for the constant time cams (Browne and Fitzsimons 1956).

TABLE 3.

THRESHOLDS OF FIVE SUBJECTS AT A RATE OF 0.1g/sec.

Subject	Rate of application g/sec.				
	3.6	2.7	1.8	1.08	0.1
A3	3.3	3.4	3.4	3.6	4.2
A6	3.4	3.6	3.7	4.0	3.8
A4	3.6	4.0	4.0	4.2	3.8
A2	3.5	3.5	3.6	3.9	4.2
C71	4.0	4.0	4.2	4.4	3.8
Means	3.4	3.7	3.8	4.0	3.9

There is no significant increase in threshold when the rate of application is reduced from 1.08 to 0.1g/sec. on the data for five subjects.

DISCUSSION.

The increase in tolerance afforded by relatively slow rates of application of g is very real and of an order which approaches that of the ordinary g suit. With the rapid rate of onset the reflex time response from the pressure receptors is too long and cardiovascular compensation occurs too late to combat the hydrostatic pressure fall at eye level and so grey-out and black-out occur and persist. With

slower rates however the increase in reflex activity parallels the increasing g, thus maintaining pressure at eye level to a higher peak g. With intermediate rates of application of g, and at lower peak g levels with rapid application, the reflex response may lag sufficiently to allow a transitory visual disturbance to occur, but as the run proceeds there is complete recovery.

The interesting possibility of whether the protection afforded by the cardiovascular reflexes could be further increased by decreasing the application rate to low levels was studied to a limited extent (Tables 2 and 3) at this stage. From the encouraging results obtained a new method of threshold determination was evolved. This is described in detail in chapter IX.

Until recently the protective value of reflex responses have been of little practical application as the rate of application of acceleration in aircraft manoeuvres has been high, and in combat slow manoeuvres are precluded. However, at high altitudes navigational manoeuvres at high speed can be carried out at moderate g levels although of fairly long duration. Obviously in these cases where the rate of application of g is not so important and can be varied at will, this physiological factor should be considered in relation to such manoeuvres.

CHAPTER V.

THE ELECTROCARDIOGRAM DURING POSITIVE ACCELERATION.

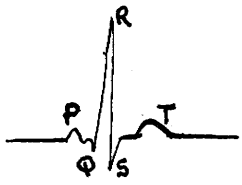
In the early stages of operating the human centrifuge objective methods of monitoring the subject's reactions to g were required, partly as a safety precaution and partly to provide basic data for future experimentation. Earlier investigators (Rose, Kerr and Kennedy 1942, Gauer 1950) had discussed pulse rate responses to g, while Zuidema, Cohen, Silverman and Riley (1956) described gross abnormalities of cardiac rhythm occurring under g associated with T wave changes. These they attribute to myocardial anoxia. On the basis of these observations it was decided that the electrocardiogram should be recorded routinely during acceleration.

The preliminary studies on the electrocardiographic changes with g were carried out in conjunction with J. T. Fitzsimons and the results of these pilot experiments have already been published (Browne and Fitzsimons 1959).

The normal electrocardiogram.

The electrocardiogram represents the electrical changes taking place in the heart muscle during the cardiac cycle. The representation varies with the position of the recording electrodes. The conventional letter by which the potentials are identified is shown in the accompanying

diagram. The first wave (P) represents the passage of the



electrical impulse through the auricles and its magnitude and direction vary with the position of the recording electrode. The second wave form (the QRS complex) represents the passage of the impulse through the bundle of His and the ventricles. The convention for lettering this complex is that any initial negative deflection is labelled Q, the first positive deflection R, and any subsequent negative deflection S. If there are two positive deflections they are designated R and R^1, similarly S and S^1 waves may occur. However Q may be absent but duplication is never observed. The T wave which follows the QRS complex represents the passing off of the impulse as the heart returns to its resting state. As with the other wave forms, its magnitude and direction depends on the position of the recording electrode.

The PR interval, the time from P to the first deflection of the QRS complex, be it Q or R, represents the time taken for the impulse to spread to the ventricles from the sino-auricular node and this is the measure of the auricular-ventricular conduction time.

Information as to the health of the ventricular musculature can also be obtained. Since the QRS complex represents the passage of an impulse through the ventricles

while the T wave (T_w) represents the passing off of this impulse, their algebraic sum should be zero and the ST junction should be isoelectric. Any variation from this condition represents an abnormality of conduction; this will be described in more detail in chapter VI. When using unipolar leads the labelling of the QRS complex can be elaborated by the use of capital letters for large deflections and small letters for small deflections. This is illustrated in fig. 12 which shows typical ventricular patterns obtained from various surfaces of the heart. Thus from any unipolar lead it is possible to deduce which surface of the heart is presenting and hence the position of the heart can be ascertained.

From the recordings of the E.C.G. we can obtain the pulse rate and much valuable information about the heart itself, its position, the state of the ventricles, the normality of conduction, and most important, its rhythm.

Methods and materials.

The method of electrode placement and fixation have already been described (chaper II). In the first stage of this investigation, 78 subjects have carried out 500 runs on the centrifuge at levels of 3.0, 3.5 and 4.0g. These subjects were drawn from two sources :-

(a) Experienced centrifuge subjects from the Institute of Aviation Medicine Staff.

(b) Inexperienced subjects drawn from the general duties branch of the R.A.F.

The centrifuge runs lasted for 15 seconds at peak g, the time taken to reach peak g and to return to rest being 9 seconds each. The rates of application of g varied from 1.0g/sec. to 3.6g/sec.

Results.

During positive acceleration the heart rate increases. The results are shown in Table 4, broken down into experienced and inexperienced subjects. The maximum pulse rates reached and the time to reach maximum pulse rate from the start of the run are shown for experienced and inexperienced subjects at different levels of g. The variance is given as ± 1.0 S.D.

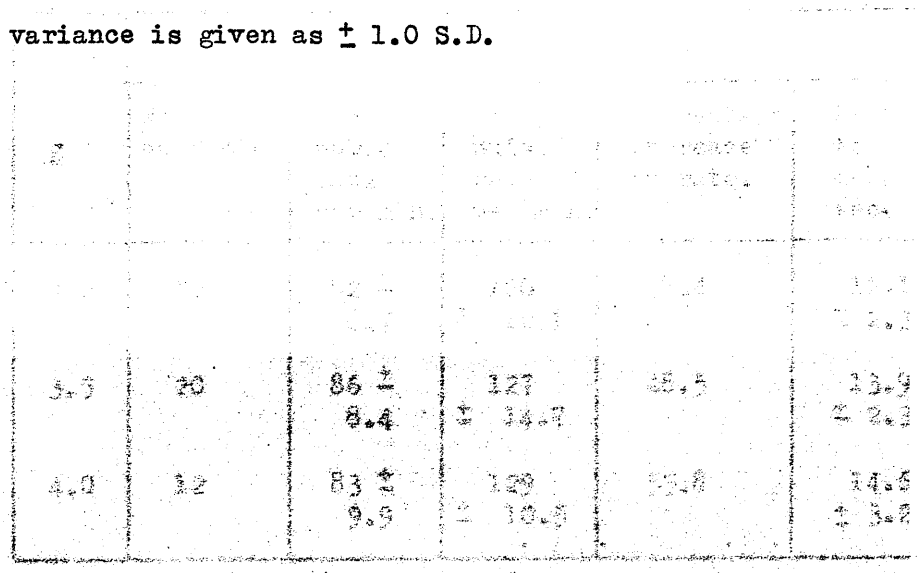


TABLE 4.

CHANGES IN PULSE RATE DURING POSITIVE g.

g	Experienced Subjects				
	No. of subjects.	Resting pulse rate per min.	Max. pulse rate per min.	Percentage increase in rate.	Time to max. sec.
3.0	12	76 ± 5.2	97 ± 7.1	25.5	16.6 ± 3.3
3.5	11	85 ± 10.7	121 ± 13.5	42.3	14.3 ± 2.3
4.0	3	77 ± 3.6	114 ± 1.6	48.0	16.1 ± 1.6

g	Inexperienced Subjects				
	No. of subjects.	Resting pulse rate per min.	Max. pulse rate per min.	Percentage increase in rate.	Time to max. sec.
3.0	20	82 ± 8.7	120 ± 12.1	47.4	12.1 ± 2.3
3.5	20	86 ± 8.4	127 ± 14.7	48.5	13.9 ± 2.3
4.0	12	83 ± 9.9	129 ± 10.5	55.8	14.6 ± 3.2

At 3.0g the difference in maximum pulse rate between experienced and inexperienced subjects is significant at the 1% level, but at 3.5g and 4.0g there is no significant difference between the two groups. Likewise the time to maximum pulse rate shows a significant difference at the 0.2% level at 3.0g between experienced and inexperienced subjects but no difference at higher g levels. The pulse rate does not begin to rise immediately the g commences but lags behind, often only starting to rise after peak g has been reached. It will be seen from the above figures that maximum pulse rates were always reached between 3 and 7 seconds after peak g. Following this peak the pulse rate invariably shows a decrease for the latter half of the run. Cessation of the g is followed by bradycardia, the rate being less than the resting value.

The PR interval was measured from some records as a check on auriculo-ventricular conduction during g. The results are shown in Table 5.

TABLE 5.

THE EFFECT OF g ON THE PR INTERVAL.

Each figure is the mean of five observations.

g	Static P.R. sec.	Stage of acceleration. (time from start of acceleration) 0 sec.	2.5 sec.	7.5 sec.	12.5 sec.	17.5 sec.	22.5 sec.
3.0	0.15	0.15	0.13	0.125	0.12	0.13	0.14
3.5	0.14	0.128	0.12	0.11	0.10	0.11	0.125
4.0	0.137	0.14	0.125	0.12	0.112	0.12	0.13
4.5	0.14	0.13	0.12	0.105	0.10	0.12	0.13
5.0	0.14	0.14	0.12	0.10	0.10	0.11	0.12

Analysis of variance shows that the sample was a representative one and that the change in P.R. interval during g is significant at the 0.1% level, as was also the progressive change in PR interval with higher g levels. It will be seen that the PR interval began to return to normal values before the end of the g run. This corresponds to the behaviour of the pulse rate which also seems to decline while the acceleration is still being applied.

Throughout the series all PR intervals fell within the normal range of 0.1 - 0.2 seconds, indicating that there was no abnormality of A-V conduction.

The cause of the increase in pulse rate is the fall in blood pressure at head level which occurs during acceleration.

On three volunteers blood pressure was measured in the radial artery at eye level and the results correlated with changes in heart rate. Fig. 10 shows a typical record. As the blood pressure falls there is cardiac acceleration. The blood pressure reaches a minimum and then rises, finally stabilising at a level intermediate between the resting value and the minimum which occurs at the beginning of the run. The pulse rate closely reflects these blood pressure changes, falling as the pressure rises and stabilising after 6-10 seconds at peak g.

In this series, which included a number of runs to 5.0g and several at lower g levels of up to 5 minutes duration, there were no abnormalities of cardiac rhythm apart from an occasional extrasystole which occurred randomly and unrelated to g. When sinus arrhythmia was present at rest it was frequently accentuated under g.

In view of the earlier observations on changes in the wave forms the records were carefully scrutinised for these characteristics. The T wave changes which were found by other workers were also found in this series of observations.

The changes across the unipolar chest leads varied from

subject to subject but the transition point of unchanged T waves was usually in the region of $V_2 - V_3$. There was no evidence of T_w inversion in any subject.

Associated with these changes there were changes in the QRS complex. These also showed a large subject variation but there was a certain basic pattern about all of them. There was the development of q waves in several leads, most commonly in aVF. In general there was an increase in amplitude of aVL and aVF, an RS pattern changing to an Rs. Increases in amplitude were also frequent in V_1 and V_2. Examples of these changes are shown in fig. 13. When viewing these traces a comparison springs to mind. The changes are very similar to those induced by respiration. Fig. 14 shows the changes due to respiration exaggerated by the subject breathing at maximum depth. It will be seen that both show the same characteristics. Since respiration becomes deeper and more rapid during acceleration (fig. 15) it is easily seen that many changes previously described as due to acceleration may be in fact respiratory. However, even allowing for errors due to respiration there are changes in the E.C.G. similar to those occurring in inspiration. The changes in inspiration are due to lowering of the diaphragm causing descent and rotation of the heart. It has been shown (Ruff and Strughold 1939, Rushmer 1944) that g also produces lowering of the diaphragm, accompanied by descent of the heart. This may amount to as much as 6 cm. at 5g (Rushmer 1944). It therefore seems

reasonable to presume that the changes in Q.R.S. complexes and T waves are positional in origin as in normal inspiration. Considering all leads together, there have been no changes in this series of runs which could be attributed to myocardial anoxia.

When measuring tolerance to acceleration by a visual threshold it is important to consider cardiac compensation. If the measurement is made when peak g is reached then recovery will occur at a variable time (6-10 seconds) thereafter due to compensatory cardiac reflexes (assuming rates of application of up to 4g/sec.). The more usual practice in this laboratory is therefore to measure threshold 10 seconds after peak g. Since higher thresholds are obtained with slower rates of application of g, presumably due to the more adequate time available for cardiac compensation, it was decided to investigate cardiac and blood pressure responses to a rate of change of acceleration of 0.1g/sec. Seven experienced centrifuge subjects were used for this investigation. Each subject carried out 5 runs per day for 5 days. On each run a lead I E.C.G. was recorded and analysed for pulse rate changes. The rate of change of acceleration was such that from $\sqrt{2}g$ to the threshold level the g/t curve was linear. Sections of the records were analysed for the relation between change in pulse rate to change in g.

Several records of blood pressure at eye level were obtained during these runs by the method described in chapter II.

TABLE 6.

CHANGES IN PULSE RATE DURING THE APPLICATION OF g AT 0.1g/sec.

Subject.	No. of runs.	Mean resting rate per min.	Mean final rate per min.	Increase in pulse rate per min.	'g'	Increase in pulse rate/g per min.
P.G.	23	91.1	107.0	15.8	3.02	5.2
G.H.B.	20	79.1	97.3	17.6	3.29	5.36
M.K.B.	18	75.2	88.7	13.6	3.13	4.36
N.L.A.	19	94.5	109.2	14.8	3.28	4.45
C.McE.	6	79.7	95.5	15.8	2.67	5.92
J.C.G.	13	84.2	99.7	15.5	2.71	5.67
A.C.	8	80.3	100.9	20.6	3.17	6.48

It will be seen that the increases in pulse rate are lower than those obtained using the conventional rates of application of 1.0 - 4.0g/sec. A typical graph obtained is shown in fig. 16. It will be seen that from 1.0 - 1.4g there is great variation in the pulse rate. This is the period during which the g/t relationship is not linear and there is a large tangential component in the acceleration. After 1.4g is reached there is a continuous rise in pulse rate with g. The abrupt

changes in rate seen with cams of 1-4g/sec. are not evident when using a rate of rise of g of 0.1g/sec. Thus there is a constantly increasing degree of cardiac compensation. The records were further examined to determine if the rate of increase of pulse rate with g was linear. The portion of the records below 1.4g were neglected as the accelerations at these low levels are complex and do not really concern us. Above 1.4g small increments of g were measured with the corresponding increase in pulse rate. A regression line was derived for these two variables, the results for all subjects were included. It was found that there is a significant linear relationship between the increase in pulse rate and the increase in acceleration. The formula of the regression line is :-

$$Y = 6.369 + 5.0875\ (x - 0.9618)$$

and the coefficient b = 5.0875 (S.E. $\pm$ 0.34). This line is shown in fig. 17.

As might be expected the records of blood pressure obtained showed that the blood pressure also fell in a linear fashion with this type of g/time pattern. These results are shown in figs. 18 and 19.

Discussion:

When subjected to positive acceleration all subjects show an increase in pulse rate. One cause of this increase is a fall in pressure in the pressure sensitive areas of the carotid sinus.

In fig. 10 it may be seen that the fall in blood pressure precedes the rise in pulse rate. Another possible cause however is an adrenal medullary response to stress. At 3.0g there is a significant difference in the maximum pulse rates reached by experienced and inexperienced subjects. If the fall in blood pressure in the carotid sinus was the only factor causing tachycardia there would be little difference between the groups. However, the fact that at 3.0g inexperienced subjects show an earlier and greater pulse rate response suggests that in these subjects other mechanisms play a greater part. Two factors suggest themselves, first an involuntary tightening of the abdominal and leg musculature which tends to increase venous return to the heart, and secondly, release of adrenal medullary hormones resulting from apprehension at being subjected to an unusual manoeuvre. There is some evidence for the former in that inexperienced subjects show more muscle artefact on the recordings, while in support of the latter, von Euler and Lundberg (1954) have shown an increased urinary output of catechol amines in inexperienced personnel when flying. At higher g levels where a maximum response is required on haemodynamic grounds, the difference in maximum pulse rate tends to disappear, though inexperienced subjects still tend to reach maximum pulse rates sooner, suggesting that apprehension is still having a significant effect. The times between peak g and maximum pulse rates reached are of the same order as those reported

by Rose, Kerr and Kennedy (1942). It should be noted that the duration of run up to peak g, 9 seconds, should be subtracted from the present results for comparison.

As has been pointed out in chapter IV, this type of blood pressure and pulse rate response leads to difficulty in estimating tolerance by visual means. Thus the question arises of where to measure visual loss. Should it be when the blood pressure reaches a minimum, or the pulse rate a maximum, or subsequently, when both are tending to reach equilibrium? The cause of the secondary rise in blood pressure is the full development of the carotid and aortic pressure reflexes; this in turn leads to a slowing of the pulse. Thus the 5 to 10 seconds taken for the maximum pulse rate to develop represents the time course of the reflex. If the reflex were fully operative then the abrupt fall in blood pressure would not occur and the problem of transitory visual symptoms would not arise. Since the reflex time can not be altered it seemed possible that by slowing the onset of the acceleration adequate time would be allowed for the full development of the reflex and the abrupt changes in blood pressure and pulse would be abolished. When a linear rate of application of g of 0.1g/sec. was used the results confirmed the above reasoning. The blood pressure fell smoothly and gradually showing no tendency to a secondary rise. Similarly the pulse rate rose linearly with the g and

continued to rise with increasing g. At any g level on the graphs the values of blood pressure are higher than those obtained with the more conventional rates, showing that a degree of protection is being afforded by the circulatory reflex activity. In fact using this rate of application subjects have maintained 8.0g for up to 60 seconds which was previously thought to be beyond unprotected human tolerance.

The PR interval varies inversely as the pulse rate (Goldberger 1953) and in this series behaved as predicted, indicating there was no abnormality of A-V conduction. This agrees with the results of Gauer and Wieckert 1944.

Since this experiment a further thousand centrifuge runs have been carried out with E.C.G. recordings, and of the total fifteen hundred runs only one case of cardiac arrhythmia has occurred, (see part II, chapter III), apart from sinus arrhythmia which is relatively common and is accentuated by g. This agrees with the findings of Pryor, Sieker and McWhorter (1952) who state that at levels of g insufficient to cause visual symptoms there were no cardiac abnormalities. It is difficult to reconcile these results with those of Zuidema, Cohen, Silverman and Riley (1956) who say, "four out of five subjects showed arrhythmias at high g levels.* This myocardial irritability may be attributed to a relative coronary insufficiency with maximum coronary flow proving to be inadequate for a massive work load."

* 4g to 5g for 60 seconds.

It might be that these subjects were older or in poor health, however, Bondurant (personal communication) repeated this work one year later with the same subjects and the same runs, and failed to confirm Zuidema's results. It is therefore fair to say that there is no evidence of cardiac irregularity at levels of g which occur in everyday flight.

There were changes in the form of the T wave as described by previous workers (Rose, Kerr and Kennedy 1942, Gauer 1950, Zuidema, Cohen, Silverman and Riley 1956). These changes in the T waves are analogous to those found in respiration and on assumption of the erect posture (fig. 20). They would therefore appear to be positional in origin, although Simonson, Nakagawa and Schmitt (1957) say that the E.C.G. changes during respiration are significant but can not be explained on positional changes alone. The effect of respiration appears to have been largely neglected in the past and yet respiratory and positional variation can explain the T wave changes entirely. There was no evidence in any record of myocardial anoxia or ventricular strain. It must be remembered that changes in the T waves alone are of little diagnostic significance, they must be considered in relation to the RS-T segment in multiple leads.

Summary.

There is a rise in pulse rate with g. The rise occurs

earlier and to a greater extent with inexperienced subjects probably due to apprehension. The changes in pulse rate can be related to the behaviour of the arterial blood pressure.

Using a linear rate of application of g of 0.1g/sec. the increase in pulse rate as a function of g becomes linear. This increase is smaller than that occurring with faster rates of application and is thought to be due to the full development of the cardiovascular reflexes. A significant degree of protection is therefore to be anticipated from this type of run. Similarly the arterial blood pressure at eye level shows a linear fall with increasing g, and the fall in pressure at any g level is smaller than with faster rates of application.

In 1,500 runs there has been only one gross cardiac arrhythmia and it is concluded that this is not a normal hazard to be encountered in flight. Sinus arrhythmia was not uncommon at rest and when present was accentuated by the g.

Changes in T waves were found, these are thought to be largely positional and respiratory, and not related to myocardial damage.

CHAPTER VI.

CHANGES IN THE ELECTRICAL AXIS AND THE VECTORCARDIOGRAM DURING ACCELERATION.

The interpretation of electrocardiographic changes has in the past been descriptive and subjective, using terms which are not capable of precise definition. The introduction of unipolar leads has overcome these difficulties to some extent and allowed interpretation to be based on established physiological principles. This improved understanding of the underlying physiology has led to attempts to analyse traces mathematically. The three most important methods which have gained acceptance are :-

1. The derivation of the electrical axis.
2. The calculation of ventricular gradient.
3. Vectorcardiography.

The electrical axis is a representation on one plane of the body of the instantaneous direction and magnitude of the resultant electrical impulse passing over the heart. It is therefore a vector quantity. Normally the axis measured is the major electrical axis, that is, the axis derived from the maximum potential, the R or S wave. However, a better method is the derivation of the mean instantaneous electrical axis in which the area of the whole QRS complex is used as a co-ordinate. By choosing leads which are at right angles to one another, e.g. lead I and aVF, and using the QRS potentials of each as co-ordinates, a vector can be drawn which represents

the electrical axis of the heart. The methods of derivation are standard procedures and will not be discussed here. There are certain conventions applying to the polarity of the co-ordinates, these are shown in fig. 21. By selection of suitable leads the electrical axis can be represented on any plane of the body, the one most commonly used is the projection on to the frontal plane. The two major limitations of this technique are :-

(a) The heart is three dimensional as is its electrical axis but the above representation is only a projection of it on to a two dimensional surface.

(b) The electrical axis is continually changing as the impulse spreads over the heart whereas it is only measued at one instant, the peak of R or S or, in the case of the mean axis, is averaged over a whole complex.

In spite of these limitations the analysis is useful and in common use. However, the normal range is from -90^{o} to $+150^{o}$, depending on the position of the heart in regard to its three axes and the method cannot therefore differentiate normal variations from ventricular hypertrophy. Its main use is in comparative work where changes in the position of the heart are being investigated.

The ventricular gradient represents the relation between the mean electrical axis of QRS and the mean electrical axis of the RS-T and T. Thus if a strip of muscle returns to the resting state in the same direction as it was stimulated these values are equal and opposite and the ventricular gradient is zero. In the heart the T_W normally points in the same direction as QRS showing that the heart returns to its resting state in a different direction to that in which it was stimulated. Thus the heart normally has a finite ventricular gradient. Any change in this gradient will reflect an alteration in the state of the muscle and is useful in interpreting T wave changes (Houssay 1955).

It has been pointed out above that the electrical axis represents only a single point in the changing electrical pattern of the heart. If successive points are taken in the QRS complexes of two leads then a series of electrical axes may be drawn which represent the changing direction of the impulse. If an infinite number of these are taken then a solid figure results whose outline is the change in electrical axis of the heart, this figure is the vectorcardiogram. It can be appreciated therefore that by deriving several electrical axes and joining their ends to form a loop a vectorcardiogram is obtained (called a derived vectorcardiogram). The larger the number of points used the closer will the resulting loop approximate to the

real condition. However by displaying the two E.C.Gs, on the axes of a cathode ray oscilloscope a continuous loop is obtained directly, this is called the direct vectorcardiogram (V.C.G.).

The V.C.G. suffers from the same defect as the electrical axis, namely that it is a two dimensional representation of a three dimensional pattern. This is obviated to some extent by obtaining V.C.Gs. in three planes at right angles to each other from which the spatial figure can be inferred. The three planes used in the body as the frontal, horizontal and sagittal. The polarities are arranged so that the sagittal plane is viewed from the left side and the horizontal plane is viewed from below. Rotation of the heart about its axes causes changes in the shape, area and direction of rotation of the vector loop.

These three mathematical analyses of the E.C.G. were used in this study in an attempt to determine the changes in the heart's position during g since there were no facilities for X-ray investigation.

TABLE 7.

MEAN Q.R.S. VOLTAGES AND DERIVED VECTORS DURING NORMAL RESPIRATION AND UNDER 3g.

Subject.	g	Phase of respiration	Amplitude			Direction (degrees)		
			I	aVF	V_1	Frontal	Sagittal	Horizontal
R.B.	1.0	Inspiration	438	588	750	+57	+137.5	-59
		Expiration	375	500	638	+57	+136	-159
	3.0	Inspiration	281	696	794	+70	+133	-70
		Expiration	316	509	691	+63	+140	-64.5
P.H.	1.0	Inspiration	325	234	1150	+50	+161	-73
		Expiration	334	334	193	+44.5	+160	-70.5
	3.0	Inspiration	389	287	1050	+52	+154	-70.5
		Expiration	343	390	967	+62	+145	-69.5
J.F.	1.0	Inspiration	245	475	1008	+65.5	+142	-64
		Expiration	250	392	1133	+60	+133	-66
	3.0	Inspiration	100	450	900	+79	+140	-77
		Expiration	179	383	1075	+68	+131	-71
N.K.	1.0	Inspiration	300	475	1900	+60.5	+150.5	-72.5
		Expiration	487	450	2050	+50	+153.5	-66.5
	3.0	Inspiration	287	1225	2000	+81	+125.5	-76.5
		Expiration	275	1075	2050	+77	+129	-74.5
P.G.	1.0	Inspiration	288	1000	1180	+75	+135	-67
		Expiration	214	800	1120	+76	+129.5	-69.5
	3.0	Inspiration	178	1050	1180	+80	+126	-83.5
		Expiration	232	800	1650	+77.5	+127.5	-79
F.Z.	1.0	Inspiration	883	768	1190	+46	+141	-53.5
		Expiration	824	616	845	+39	+144	-46.0
	3.0	Inspiration	650	821	813	+47.5	+131	-51.5
		Expiration	765	608	940	+42.5	+143.5	-50
M.S.	1.0	Inspiration	660	304	1243	+27	+164	-62
		Expiration	590	232	890	+24	+163	-56.5
	3.0	Inspiration	610	144	1180	+14	+172	-63.5
		Expiration	650	155	857	+17	+168	-53.5
M.K.B.	1.0	Inspiration	536	177	676	+22	+162	-51
		Expiration	547	132	605	+16	+165	-47
	3.0	Inspiration	547	158	470	+19	+165	-49
		Expiration	578	142	619	+12	+164	-38

Methods and Material.

The electrical axis was obtained from the E.C.G. The leads used were; frontal plane - leads I and aVF; sagittal plane - leads V_1 and aVF; horizontal plane - leads I and V_1. Since the leads were a mixture of bipolar and augmented unipolar leads a correction must be applied to the potentials measured. Thus the voltage of lead I must be multiplied by 1.15 to be compatible with aVF (Goldberger 1953). Lead V_1 lies in close proximity to the heart and since it therefore records much higher voltages than the peripheral leads, a correction must be applied. This is not capable of precise determination and on an arbitrary basis it has been divided by two as suggested by Goldberger (1953).

The vectorcardiograms were obtained by two methods. One, by the rectangular co-ordinate system of derivation (Goldberger 1953), the other by displaying the loop on a C.R.O. and photographing it. The first method is tedious and approximate, and although it closely resembles the direct trace (fig. 22), it was discontinued and all further V.C.Gs. were displayed on the C.R.O. Goldberger's electrode placements (lead 1, aVF and V_1) were used routinely. In some earlier runs Duchosal and Sulzer's placements were tried but abandoned because of excess muscle artefact.

In order that the phase of respiration be known when interpreting the results, it was arranged to record

respiration by means of a small thermocouple in the nose. Changes in thermal e.m.f. due to respiration were recorded as a low frequency wave form in a similar manner to the E.C.G.

Further refinements in the technique of vectorcardiography were subsequently made and a semi-automatic device built to select one QRS complex only at a fixed position in the respiratory cycle. This will be described in Part II in association with the experiment for which it was developed.

The centrifuge runs were made at various g levels using constant time cams allowing 4.5 seconds from $\sqrt{2}g$ to peak g. All runs were of 15 seconds duration unless otherwise stated.

Results.

1. The Electrical Axis.

Table 7 shows the magnitude of the QRS complex during g and at the peaks of normal inspiration and expiration. Also shown is the direction of the manifest instantaneous electrical axis in each plane calculated from these results. These are also shown graphically in fig. 23a-d. Each result is the mean of three observations for it was rarely possible to obtain more than three full cycles of respiration owing to the short duration of the runs. However the scatter was very narrow and figures for the same phase of respiration were usually identical.

Out of the eight subjects, five had vertical hearts, as judged from the electrical axis (i.e. over +45° in the front plane), two had horizontal hearts,and one subject (F.Z.) was border-line, 39-46°. In all subjects the range at 1g varied between +16° (M.K.B.) and 76° (P.G.) in expiration.

Respiration has a marked effect on the axis in most cases. In the frontal plane the heart becomes more vertical with inspiration. In the sagittal plane also the axis becomes more vertical although the movement is rather less. In two subjects, R.B. and M.S., the axis tended to become horizontal by a negligible amount. In the horizontal plane, viewed from below, the shifts are small. Two subjects show clockwise rotation and five show anti-clockwise rotation, and there is no change in one. There is no correlation between the direction of movement and the resting mean position of the heart.

Under 3g the respiratory changes are of the same order as at 1g. However, there is an overall shift in the mean axis.

In all the subjects with the exception of the two with horizontal hearts, (M.K.B. and M.S.), the axis becomes more vertical under g in the frontal plane, more vertical in the sagittal plane and shows anti-clockwise rotation in the horizontal plane when rotation occurs.

In the two horizontal cases the axis surprisingly becomes

more horizontal in the frontal and sagittal planes, and in the horizontal plane one rotates in a clockwise direction and the other shows no rotation. These changes in mean position are shown in Table 8.

TABLE 8.

CHANGE IN MEAN ELECTRICAL AXIS OF THE HEART AT 3g. (POSITIVE SIGNS SIGNIFY CLOCKWISE ROTATION).

Subject.	Frontal.	Sagittal.	Horizontal.
P.H.	+10	-11	-2.0
J.F.	+11	-1.0	+9.0
N.K.	+24	-25	+6.0
P.G.	+3.0	-6.0	+13.0
F.Z.	+2.5	-5.0	+2.0
R.B.	+9.5	0	+8.0
M.S.	-10	+6.5	-1.0
M.K.B.	-4	+1.0	-6.0

In order to try and relate these changes in electrical axis to alterations in the anatomical position of the heart, chest X-rays of three subjects were taken in full inspiration and expiration. The long axis of the heart derived from these radiographs is compared with the manifest electrical axis in Table 9.

TABLE 9.

Subject	Anatomical axis			Electrical axis		
	Inspiration	Expiration	Range	Inspiration	Expiration	Range
J.T.F.	+51	+33	+18	+84	+41.5	+42.5
P.G.	+58.5	+40	+18.5	+83	+78	+5.0
M.K.B.	+39.7	+36	+3.7	+67	+18	+49

In these three subjects the relationship between electrical and anatomical axis is, to say the least, doubtful. Thus we cannot deduce from changes in electrical axis under g what anatomical changes are occurring.

Four subjects carried out runs to a high g level using the rate of application of 0.1g/sec. which is associated with a smooth linear cardiac response. The results for these subjects are shown in fig. 24a and b. There is a change in electrical axis towards the vertical in both frontal and sagittal planes which is progressive with increasing g.

2. The Ventricular Gradient.

Since the ventricular gradient is said to be useful in interpreting T wave changes, some preliminary calculations were carried out. These showed there was in fact a change during acceleration. However in view of Wilson's remarks (quoted by Barker 1952), and more important, the fact that the gradient

will vary both with position and pulse rate, the method was abandoned.

3. The Vectorcardiogram.

There is little to comment on the V.C.Gs. obtained in this investigation. In fig. 22 direct and derived V.C.Gs. are compared for one subject at rest and at 3g. The resemblance of the QRS is obvious though the match is by no means perfect. Fulton's remarks (Fulton 1953) are therefore perhaps justified, that geometric construction is very likely to be erroneous. These V.C.Gs. are of one of the subjects (M.K.B.) with horizontal hearts who show rotation towards the horizontal. The reproducibility of the V.C.Gs. is good but positional changes are largely masked by respiration and the changes during acceleration provide no more information than is obtained from the electrical axis itself. It was hoped that direct observation of the loop might have been useful since in a vertical heart the direction of rotation of the loop is usually clockwise whereas in the horizontal heart it is usually counterclockwise. Observation showed no change of rotation in any of the subjects in this series. It is possible that this was due to the fact that the majority of the subjects had vertical hearts under static conditions.

Discussion.

The most outstanding feature of this series of observations

was the effect of respiration on the electrical axis of the heart. This is large enough to mask changes in axis due to postural changes and makes it imperative that in any investigation electrical axes should only be compared at identical points in the respiratory cycle. This effect of respiration is frequently neglected and leads to erroneous conclusions.

During acceleration there is a change in the electrical axis of the heart. At 3g this is quite small but increases progressively with g. Although there is no correlation between anatomical axis and electrical axis, it would seem not unreasonable to suppose that this change is related to the descent of the diaphragm which is known to occur under g. Support for this supposition is given by the fact that the change in position is similar and in the same direction as that occurring with inspiration which is again associated with diaphragmatic descent. Similar respiratory findings have recently been reported by Simonson, Nakagawa and Schmitt (1957). Since the application of +g is merely an accentuated form of the force which acts on tilting from the horizontal to the vertical, it is to be expected that similar findings would obtain on change of posture. This has been investigated by Mayerson and Davis (1942) who found identical changes in the electrical axis when a subject is swung from the horizontal to 75° head-up on a tilt table. In their

series they also found that if the upright posture was maintained there was a further clockwise swing of the axis which in some cases might be as great as the initial change. This obviously could not be positional or related to any anatomical change. It was related in time to the reflex vasoconstriction and cardiac acceleration which develops in response to the assumption of the upright posture. On the basis of these findings the authors concluded that the change in axis is a result of increased sympathetic activity, although it could equally well be due to decrease in vagal tone. If this is so then the alterations in axis found in the present study may also be partially due to increased sympathetic or decreased vagal tone as acceleration is accompanied by a tachycardia and an increase in peripheral resistance (see part II, chapter III). This would also explain the lack of correlation between electrical and anatomical axes.

The results of the vectorcardiographic analyses are open to the same criticism. Since they represent the changing electrical axis they also will reflect changes in autonomic activity and will therefore not form a reliable guide to positional changes. Nevertheless if this possibility is borne in mind and the phase of respiration is known then they may provide a crude guide to the changes in cardiac orientation.

Summary.

There is a progressive change in the electrical axis with increasing g. The majority of subjects show a clockwise rotation in the frontal plane, and counter-clockwise rotation in the sagittal plane, with little alteration in the horizontal plane. These rotations are similar in magnitude and direction to those occurring with inspiration. The causation is probably positional due to descent of the diaphragm, although there is no obvious correlation with the anatomical axis. The lack of correlation may be due in some part to similar changes occurring with increased sympathetic activity.

The ventricular gradient is of no value as it varies with position and rate of the heart, both of which vary during acceleration.

The information obtained from the V.C.G. adds nothing to that obtained from the electrical axis and is subject to the same errors. However it is displayed continuously and does not require calculation and is thus useful as a monitor of the change in position of the heart during comparative studies where the errors are liable to be constant.

CHAPTER VII.

THE ELECTRO-ENCEPHALOGRAM DURING POSITIVE ACCELERATION.

During acceleration there is a progressive decrease in the blood supply to the brain. This cerebral anaemia eventually leads to abolition of nervous function. The electro-encephalogram might therefore allow objective monitoring of the subject's condition. In addition it might provide some evidence of black-out or show premonitory signs of impending unconsciousness. Unconsciousness is to be avoided if possible and at high g levels the only clue to the subject's loss of consciousness may be the occurrence of convulsions.

Method and Materials.

The leads used were left and right bipolar occipital, and in some experiments left and right frontal were also recorded. During g there is a large amount of head and scalp movement and the normal E.E.G. electrodes are of no value. Those described by Pitman and Whiteside (1955) were tried but gave equally bad results. The method found to give the best records was the use of E.C.G. electrodes (see chapter II) stuck to the scalp with 'Nobecutane'. An initial source of interference was the amount of noise (10-50 μV) picked up from the centrifuge controls. This was eliminated by mounting the preamplifiers in the centre section of the centrifuge, thereby increasing the signal-noise ratio, since the noise originated between the centrifuge itself and the slip-rings two storeys above.

The E.E.Gs. were recorded on an Ediswan pen writing recorder.

Six experienced centrifuge subjects carried out four runs each to black-out level. The centrifuge was kept at peak g for twenty seconds during which the grey-out and black-out occurred.

Results.

1. Resting E.E.Gs.

Two subjects (P.G., J.C.G.) showed a well organised alpha rhythm (9-10 c.p.s. 30-70 μV), while two others (C.M.E.V., N.L.A.) showed occasional bursts of alpha rhythm interspersed with medium frequency-medium voltage waves. The last two (M.K.B., G.H.B.) showed no evidence of alpha activity. There were no recognisable wave forms obtained from the frontal electrodes. Each subject carried out two minutes voluntary over-breathing. This produced no abnormality in any of the E.E.Gs. although it was adequate to produce tingling of the extremities in all subjects. In two subjects (N.L.A., J.C.G.) it increased the amplitude of the alpha rhythm.

2. The E.E.G. during acceleration.

During acceleration there is no characteristic change in the E.E.G. In the earlier part of the runs there is a diminished alpha activity which returns to normal about the middle of the runs (fig. 25). In some subjects there is an inconstant change to faster frequencies (10-15 cps). These

changes are seen in the occipital leads, there is no change from the resting record in the frontal leads. Grey-out and black-out occur without any change in the E.E.G. although with black-out some change may be seen in the frontal leads due to eye movements. In one subject the run was continued until unconsciousness supervened. Unconsciousness was associated with the development of large slow waves (1.5 - 3 c.p.s. 50-120 μV) in the occipital leads. This delta activity did not appear to precede loss of consciousness but to be coincident with it. On recovery there was a return to a normal resting record.

Discussion.

Various workers have studied the electro-encephalogram during acceleration (in monkeys - Britton and Pertzoff 1943, monkeys and cats - Jasper 1942, Jasper, Cipriani and Lotspeick 1942a, b, 1943, man - Rose and Martin 1942, Rose, Kerr and Kennedy 1942, Kerr and Russell 1944, Franks 1956a and b, Powell 1956, Brent, Powell and Scott 1957).

In examining published reports it is surprising to find that there is a large gap between that carried out during the war and that reported in the last few years. It is worth while summarising the findings of the earlier workers in this field. In animals it was found that the onset of acceleration was accompanied by signs of excitation in the E.E.G. consisting of an increase in amplitude of the existing rhythm

with the appearance of large spikes similar to those occurring in epilepsy. This was followed by depression with the appearance of slow high voltage waves; continued g led to entire abolition of electrocortical activity. This extinction of the E.E.G. could not be produced below 3g, took 80-120 seconds at 3-4g and appeared sooner with increasing g. The absolute minimum time to extinction was 7-10 seconds which agreed with the time to extinction following clamping of the aorta. Recovery was associated with the above sequence of events occurring in the reverse order, final recovery occurring in 10 seconds to 3 minutes.

The findings in humans were essentially similar with the exception of extinction of activity. This is probably due to the fact that unconsciousness was never maintained for more than a few seconds. Specific wave forms were described by Rose, Kerr and Kennedy (1942) who found waves of 16-22 c.p.s. 30-50 μV which lasted throughout the run and waves of 5-14 c.p.s. 30-120 μV which occurred during rising acceleration but disappeared at peak g. The former activity the authors admit might well be muscle artefact although they suggest that the slower rhythm is probably caused by labyrinthine stimulation due to the changing angular acceleration during this period. When these experiments were carried out at Toronto the facilities for artefact-free recordings were not available (Scott - personal communication) and the suggestion of muscle activity is almost certainly true. In the author's

view the slow wave forms are explicable on the same basis. This is borne out by the fact that inexperienced subjects who are tense show activity of this sort whereas it is absent in experienced subjects. Experienced subjects by tensing their neck muscles can produce similar activity. It is agreed that these wave forms are probably due to labyrinthine stimulation but arise from the muscles of the head and neck and not from the cortex. In normal subjects spike and wave forms have never been described nor were they found in this investigation.

In humans the delta activity (waves 1.5-6 c.p.s. 50-150 μV) occurred associated with loss of consciousness. Occasionally consciousness was lost without their presence but they rarely occurred in the conscious subject and then only associated with 'deep black-out'.

More recent workers have only described E.E.G. changes during experimental procedures where stresses in addition to g have been applied. This will be discussed in Part II.

In various other experiments (Part II) where the E.E.G. was recorded, twenty-nine cases of unconsciousness in fourteen subjects have occurred. In fifteen of those the onset of unconsciousness was associated with delta rhythm. In no case did delta rhythm precede the onset of unconsciousness nor were any epileptiform spike and wave forms seen. In particular, delta activity was never associated with black-out. It is not

It is not clearly understood what Rose means when he describes them occasionally in 'deep black-out' as this term has no meaning to present day investigators.

With these slight modifications the gross findings are in accord with those of previous workers, whose more detailed descriptive changes were almost certainly due to artefact and muscle interference arising from their earlier equipment.

The conclusion is that the E.E.G. serves little purpose monitoring normal subjects as there are no characteristic changes with grey-out or black-out, and the only gross change in rhythm occurs after consciousness is lost.

CHAPTER VIII.

THE OCCURRENCE OF CONVULSIVE EPISODES ON THE CENTRIFUGE.

In the routine running of the human centrifuge convulsions are occasionally seen. These are only associated with loss of consciousness during acceleration. Apart from the thirty cases of unconsciousness mentioned in the preceeding chapter a further ten to twenty have occurred accidently during routine runs where no instrumentation had been employed. In the majority of cases convulsions occur.

These fits vary in intensity from minor spasmodic tremors to major convulsions. They are described by the observers sitting in the centre, thirty feet away, and are therefore not clinically detailed. The minor forms consist usually of tremors of the fingers followed by jerking movements of the arms; in this variety the lower limbs do not appear to participate although they are not easily seen by the observer. From this mild form all intervening stages can occur up to the major convulsion where the whole body convulses. The convulsions do not resemble the tonic and clonic epileptiform type so much as the march of events seen in a Jacksonian fit. They were described as convulsions by Rose and Martin (1942) but are usually referred to as jactitations in this laboratory as they do not closely resemble the types of fit more commonly encountered in clinical practice. As can well be appreciated these fits are potentially dangerous in the centrifuge gondola travelling at

60 m.p.h. with the subject seated amongst a mass of equipment with sharp angles and edges. They also preclude the use of intra-arterial needles in high g runs. Several superficial injuries have occurred and have led to stringent precautions being taken, including the use of safety harnesses and padding of all dangerous objects. These incidents were a stimulus to the development of a method of threshold determination which would be free of the risk of unconsciousness developing, since they depend not on the level of g but rather the individual's black-out threshold. Thus in some subjects they may occur at 4.0g and in others not until 6 or 7g.

These convulsions were first described by Rose and Martin (1942) who found them to occur in twentysix out of twentynine subjects who were run to unconscious levels. In their series the fits occurred after consciousness was lost and most commonly during recovery. It is often difficult to decide at which point they commence as the centrifuge is usually decelerated as soon as unconsciousness is seen. In some of the cases occurring in this laboratory the g level has continued to increase after the observation of the convulsion. The most likely cause of these convulsions is acute cerebral anoxia since they occur after the blood supply to the brain has failed. They would thus be analagous to the Kussmane-Tenner spasms which occur on occlusion of the carotids. It is interesting to note

that Schmitt (1944) says, "convulsions are not a prominent feature of positive g .... Acute cerebral anaemia does not characteristically cause stimulation before depression although stimulation may follow depression." It must be concluded that Schmitt had little experience of acceleration since convulsions are the rule rather than the exception. The observations on stimulation of the C.N.S. do not fit the picture found in acceleration as although most of Rose's cases occurred during recovery (i.e. post-depression), a certain number occurred at the onset while in the present series a large percentage occurred while the acceleration was still being applied. It may well be that acute cerebral anaemia is not the sole agent responsible.

The electro-encephalogram offers little useful information. The convulsions usually occur in the presence of slow large amplitude waves in the parietal and occipital regions. They produce no change in this rhythm apart from a certain amount of muscle noise if the scalp and neck muscles are involved in the convulsion. In some cases the head suddenly snaps forwards as extensor tone is lost since it is weighing 5-8 times its normal weight at this point. This strains the leads and frequently causes blocking of the amplifiers and the E.E.G. loses all evidence of wave forms. This may falsely suggest extinction of cortical activity as described by Jasper, Cipriani and Lotspeick (1942).

In the series described by Rose and Martin (1942) it was suggested that there was a correlation between the resting

E.E.G. and the occurrence of fits. Of the twentysix subjects who lost consciousness, eleven showed evidence of abnormal activity in the resting electro-encephalogram. Of these eleven, eight (about 75%) had major seizures while minor seizures were exhibited by two others. The authors suggested that these susceptible subjects may provide evidence for the epileptiform nature of the convulsion. In the series reported here none of the subjects showed any episodic activity in the resting E.E.G. ~~nor~~ did two minutes over-breathing produce any evidence of such activity. It must be pointed out that in no subject was there ever an aura, and control of the sphincters was not lost. Thus I have been unable to confirm the relationship between the E.E.G. and convulsions. Nevertheless there is no doubt that in a person with an ictal diathesis the strain of positive acceleration might well provoke a major or minor fit.

During unconsciousness the subject tends to dream, this is especially marked in a convulsion. On recovery there is a period of confusion where dream cannot be separated from reality, and in this period the subject, while conscious, does not respond to visual and auditory stimuli. Complete recovery is associated with a short period of retrograde amnesia, the subject not realising that anything abnormal has occurred.

Summary.

In the majority of cases loss of consciousness due to acceleration is accompanied by jactitations. These may be confined to the upper limb or involve the entire body. They

may occur as consciousness is lost or during recovery. Their variable time of onset raises doubt as to whether acute cerebral anaemia is the entire cause. They appear to be without any concomitant on the E.E.G. The associations with epilepsy and episodic activity in the resting E.E.G. has not been confirmed.

Recovery is associated with confusion and retrograde amnesia is common.

The dangers of convulsions in the centrifuge underline the need for a means of threshold determination which allows a large safety margin between it and the threshold of unconsciousness.

CHAPTER IX.

A METHOD FOR DETERMINING TOLERANCE IN THE HUMAN CENTRIFUGE BY MEANS OF THE VISUAL THRESHOLD.

The method of visual threshold determination in centrifuges varies from laboratory to laboratory throughout the world. Results from different workers are therefore difficult to correlate, especially since details of the variables involved are usually scanty if present at all. This chapter describes a method which has been developed by the author in which all the variables are capable of definition and which gives results in a range which is both safer and less fatiguing than that currently used.

In centrifuge work there is no doubt that black-out is the end point of choice. However, since black-out levels are so close to unconsciousness and the safety factor therefore low, the more elusive grey-out is more commonly used. The usual experimental method is based on the use of two dim lights situated peripherally in the visual field. The subject fixates on a central spot and the peripheral lights are switched on. The subject responds to this stimulus by switching the lights off and the sequence is repeated. At the threshold level when coning of vision occurs, there comes a point when the subject fails to respond to the peripheral lights and this is taken as the end point of grey-out. To reach threshold repeated g runs are carried out whose form is shown in fig. 26a, starting at about 3.2g and raising or lowering the g value as necessary

until grey-out occurs. There are many obvious disadvantages to this system. Firstly, the threshold measured depends on the intensity of the peripheral lights and on the angle they subtend at the eyes; these factors vary from laboratory to laboratory, while the latter varies with the position of the subject. Secondly, it may be necessary to carry out several runs to reach a threshold level and this is fatiguing to the subject quite apart from the discomfort caused by the "toppling" sensation experienced during the deceleration phase. Another point is that subconscious cheating may easily occur as a small eye movement to either side of the fixation point at threshold will once more bring the lights into view. This is probably a fairly common occurrence since, due to the general dimming of vision, there is a period of uncertainty as to whether the lights are on or not. These sources of error caused considerable doubts to be felt concerning the validity of the method. These were not allayed by the fact that it had been frequently noticed that the threshold varied with the rate of application of g (Maciolek 1955, Stole 1956, Edelberg, Henry, Maciolek, Salzman and Zuidema 1956, Browne and Fitzsimons 1956). This variation is thought to be due to the time response of cardiovascular reflexes, (Diringshofen 1942, Wood 1942) which only become operation some 6-12 seconds after the onset of acceleration (Wood 1944, Code, Wood, Sturm, Lambert and Baldes 1945, Browne and Fitzsimons 1956). In general with higher rates of rise of acceleration grey-out and black-out are indistinguishable and occur at a lower g value due to the lack of

reflex cardiovascular response. This also almost paradoxically occurs with very low rates of rise when the reflexes are fully operative due to the pure hydrostatic effect of blood pooling. However, in the middle ranges there is more distinction between the three phases and each occurs at a higher g value. These responses have a wide individual variation and also vary with time in any one individual (Staufer 1952).

It is obvious that there are many objections to the standard procedure and it was felt that it would be worthwhile to state the ideal conditions and then attempt to develop a method which would approach them as closely as possible.

The variables which should be defined in any method of threshold measurement have been described by Howard (1957) and are shown in fig. 27. It has also been emphasised that any method should be applicable to all the centrifuges of the Western world (Leverett and Zuidema 1957) and the conditions should be capable of precise specification to allow correlation of work in different laboratories. The first attempt to meet those requirements has been reported by Byford and Howard (1956). Briefly, it consisted of a central white light viewed through goggles which only transmitted the red end of the visual spectrum. By this means the subject viewed a monochromatic light against a homogeneous visual field. The intensity of the light was always adjusted by means of neutral

density filters to 0.4 log.units above absolute visual threshold. Using this method the above authors obtained black-out levels of 1.5 - 2.5g which were remarkably consistent in any one subject. Their measurement was made 10 seconds after peak g to allow time for cardiovascular compensation to take place. While this method gives excellent results, there are several limitations. The first is the use of goggles.. Goggles tend to mist despite the use of ventilation and anti-mist solution, and if a large range of subjects are to be used the fitting of the goggles, which must be light-tight, becomes a serious problem. The second limitation is the number of runs required to establish the threshold. The pattern of run is shown in fig. 26a. The centrifuge is accelerated to a level plateau and decelerated again, the run being repeated until the plateau is found at which black-out occurs. Thus several runs are necessary with the concomitant tumbling and fatigue. The present method was evolved to overcome these difficulties.

In any physiological study in acceleration, and also in the testing of equipment, the degree of cardiac compensation must be considered. After a trial of many rates of application of g it was found that a rate of rise of 0.1g/sec. allowed cardiac compensation to keep pace with the rising g stress (see chapter IV). This rate of change of acceleration also allows the g/time curve to be linear above $\sqrt{2}g$ and so allows a high degree of specificity. Since the g/sec. is low it was considered that black-out could

be signalled by the subject during the rise period, and assuming visual reaction time to be less than 0.5 seconds then the error should be less than 0.1g. The g/time curve is shown in fig. 26b.

Method.

When a small fixation light is viewed against a homogeneous background it undergoes apparent changes in brightness as g is increased. When white light is used its apparent brightness increases to a marked degree (on some rough preliminary measurements an apparent decrease of some 20% on pre-run voltage supply was observed at 3.0g) and low thresholds are difficult to obtain. In addition, changing the current supply to the lamp causes a change in colour temperature of the emitted light which introduces another variable. It was felt, therefore, that the light should be monochromatic and capable of being fixated centrally. A red light was chosen as it fulfilled these requirements and a filter was easily obtainable which allowed only a very narrow band of the visible spectrum to be transmitted (fig, 28). In addition, changes in colour temperature of the source were not reflected in the transmitted light.

Basically the apparatus is simple (fig. 29). The gondola of the centrifuge is divided one meter from the subject's eyes by a matt black partition which is carried round the sides of the car, thus giving a uniform field of view. The

centrifuge chamber is "blacked out" and in complete darkness. The safety precautions in the centrifuge require a Medical Officer to be seated in the centre section to observe the subject. Some provision had, therefore, to be made for observing the subject. This was done by installing a 25 watt 250 V. red lamp in the roof of the gondola which, projecting through a 2 inch square aperture, illuminated the subject's head without throwing any direct light onto his visual field and therefore without necessitating an increase in overall target brightness. In the centre of the visual field is an aperture 5 m.m. diameter behind which is mounted the light source. Between the two is an opal glass screen which diffuses the light, and an R.A.F. type 6 infra-red filter having the characteristics shown in fig. 28.

The subject is seated in the gondola and the chamber closed. Ten minutes are allowed for dark adaptation. While dark adaptation is not complete in this time, it was found by experience that a shorter time gave poor results by causing variations in the subsequent threshold measurements but that in normal subjects allowing longer for dark adaptation did not affect the results. Ten minutes were adopted as the minimum time which would give repeatable results and would allow the maximum number of runs per day. No attempt was made to determine the effects of vitamin A on the results. When the subject is dark adapted an 0.5 log.unit neutral density filter

is placed in front of the light source and the light intensity varied until the subject's visual threshold is reached. The neutral density filter is then removed and the subject sees a red disc whose intensity is 0.5 log.units above his threshold. The centrifuge is started and accelerates at 0.1g/sec. When the subject, who is relaxed, can no longer see the light he presses a push button which marked the record and brings the centrifuge to rest.

The value of the neutral density filter used was chosen as one which would give a threshold of between 2.0 and 3.0g. By using other values the threshold can be selected at will, with the upper limit being the absolute black-out threshold; i.e. where the pressure in the retinal artery equals intra-ocular pressure.

These is a continuous variation in the absolute visual threshold. This variation measured, following 45 minutes of dark adaptation, was found to have an amplitude of 0.05 to 0.2 log.density units and the consensus of opinion is that this is an entirely threshold effect and should not occur when the light is above that level as in the present method. A further possible criticism of the method described is that there is a fairly lengthy period (up to 30 seconds) of labyrinthine stimulation which might be expected to cause artefacts in the results and lead to undesirable side effects, e.g. nystagmus and motion sickness. The relation between the

resultant acceleration (a) experienced by the subject and angular acceleration of the centrifuge (w) is given by the equation :-

$$\alpha = \sqrt{1 + \left(\frac{rw^2}{g} \right)^2}$$

r = radius = 30 ft.
g = 32 ft./sec.^{2}

Whence it can be seen that with a rate of change of acceleration of 0.1g/sec. the angular acceleration is of the order of 2°8' ./sec.^{2} which is well above the threshold for labyrinthine stimulation given by Van Egmond, Groen and Jongkees (1949). This is further complicated by the fact that during this time the gondola is rotating about its longitudinal axis.

In the preliminary experiments on the method it was found that some subjects did in fact experience an oculogyral illusion, the spot rotating and rising to the right. To attempt to overcome this and to provide a visual clue to the position of the spot should it inadvertently be lost, two wedge-shaped horizontal white bars were placed round the spot with their broad ends inwards. Their brightness was higher than that of the spot. With this modification there was no further complaint about the oculogyral illusion. Subjectively there is no nystagmus nor are there any unpleasant sensations, all subjects have unanimously voted it the most comfortable "ride" of all the available cams.

RESULTS.

For the initial investigation of the method each subject

had his visual threshold determined as described above and this value was used throughout the series. Seven experienced centrifuge subjects were used. Five threshold determination were made on each subject every day for five days. Each subject carried out his runs at the same time each day and had a total of twentyfive determinations in the week. Repeated checks on pulse rates were made during the runs by means of a lead I E.C.G.

The results obtained are shown in Table 10. It will be seen that the threshold obtained lies between 2.0 and 3.5g. The standard deviations lie between $\pm$ 0.04 and $\pm$ 0.17 for any one subject in any one day. This is an acceptable variation since the tolerance limits for the human centrifuge from run to run are $\pm$ 0.10g up to peaks of 5g.

Within the group of seven subjects there is a daily variation for any one subject over the range 0.2 to 0.6g which is of the order found using the other methods of threshold determination. The subject to subject variation is up to 1.3g which again compares with the results obtained using peripheral lights and plateau runs (Browne and Fitzsimons 1956, Table 11). These are physiological variations and do not depend on the method used.

TABLE 10.

DAILY g THRESHOLDS OF SEVEN EXPERIENCED CENTRIFUGE SUBJECTS.

SUBJECT	DATE				
	30 Sept.57	1st Oct.57	2nd Oct.57	3rd Oct.57	4th Oct.57
J.G.	2.2	2.2	2.6	2.7	2.6
	2.6	2.3	2.8	2.7	2.9
	2.5	2.5	2.4	2.6	2.8
	2.5	2.5	2.6	2.8	2.7
	2.3	2.3	2.7	2.8	2.9
Mean & S.D.	2.4 ± 0.15	2.4 ± 0.12	2.6 ± 0.13	2.7 ± 0.08	2.8 ± 0.12
N.L.A.	3.1	3.4	3.3	3.3	3.2
	3.5	3.4	3.4	3.4	3.2
	3.5	3.3	3.2	3.4	3.1
	3.6	3.3	3.5	3.4	3.4
	3.5	3.3	3.4	3.5	3.3
Mean & S.D.	3.4 ± 0.17	3.3 ± 0.05	3.4 ± 0.10	3.4 ± 0.05	3.2 ± 0.10
C.McE.	2.6	2.5	2.5	2.7	2.7
	2.5	2.3	2.7	2.5	2.7
	2.6	2.5	2.5	2.8	2.8
	2.7	2.7	2.6	2.5	2.6
	2.6	2.5	2.7	2.5	2.8
Mean & S.D.	2.6 ± 0.06	2.5 ± 0.10	2.6 ± 0.09	2.6 ± 0.10	2.7 ± 0.07
G.H.B.	3.4	3.2	3.3	3.4	3.2
	3.4	3.3	3.4	3.1	3.1
	3.3	3.2	3.3	3.4	3.1
	3.3	3.2	3.5	3.1	3.1
	3.3	3.2	3.4	3.3	3.2
Mean & S.D.	3.3± 0.05	3.2 ± 0.04	3.4 ± 0.07	3.3 ± 0.14	3.1 ± 0.05
P.G.	2.8	3.3	2.9	3.0	3.0
	2.8	3.3	2.9	3.2	3.0
	2.6	3.3	3.0	3.2	2.9
	2.6	3.3	2.9	3.2	2.9
	2.6	3.2	3.0	3.0	2.7
Mean & S.D.	2.7 ± 0.10	3.3 ± 0.04	2.9 ± 0.05	3.1 ± 0.10	2.9 ± 0.11

TABLE 10 (contd.)

SUBJECT	DATE				
	30 Sept.57	1st Oct.57	2nd Oct.57	3rd Oct.57	4th Oct.57
M.K.B.	3.0	2.6	3.3	3.3	3.0
	3.1	2.5	3.3	3.0	3.2
	3.2	2.4	3.0	3.0	3.0
	2.7	2.9	3.2	2.9	3.3
	3.0	2.7	3.3	3.0	3.2
Mean & S.D.	3.0 ± 0.17	2.6 ± 0.17	3.2 ± 0.12	3.0 ± 0.14	3.1 ± 0.12
F.L.	2.3	2.3	2.7	2.7	2.7
	2.2	2.4	2.5	2.8	2.5
	2.2	2.8	2.5	2.7	2.6
	2.0	2.6	2.6	2.5	2.5
	1.9	2.6	2.4	2.3	2.9
Mean & S.D.	2.1 ± 0.15	2.5 ± 0.17	2.5 ± 0.10	2.6 ± 0.18	2.6 ± 0.15

In view of the satisfactory results obtained from th
trial the method is now used routinely in this laboratory
Two thousand runs have since been carried out of which one
third were on the normal threshold estimated to not be cor
rect more than experimental error. These runs have been
analysed for each subject and for all subjects together,
results are also divided into two six month periods. The

TABLE 11.

DAILY VARIATION IN THRESHOLD MEASURED BY PERIPHERAL LIGHT LOSS.
(3.6g/sec. RATE OF APPLICATION OF ACCELERATION)

DAY	SUBJECTS					
	J.T.F.	M.K.B.	F.L.	P.H.	N.L.A.	G.H.B.
1	3.6	3.4	3.5	3.3	3.7	3.0
2	3.5	3.5	3.6	3.4	3.8	2.5
3	3.8	3.6	3.7	3.2	3.6	2.8
4	3.7	3.4	3.5	3.6	4.0	3.0
5	4.0	3.6	3.8	3.4	4.0	3.0
6	3.6	3.5	3.6	3.6	3.8	3.0
7	3.6	3.6	3.6	3.4	3.6	3.0
Range	0.5	0.2	0.3	0.4	0.4	0.5

In view of the satisfactory results obtained from this trial the method is now used routinely in this laboratory. Two thousand runs have since been carried out of which one third represents normal thresholds estimated to act as controls for various experimental series. These runs have been analysed for each subject and for all subjects together. The results are also divided into two six month periods. The threshold results are shown in Table 12.

TABLE 12.

AVERAGE OF THRESHOLDS OF TEN EXPERIENCED CENTRIFUGE SUBJECTS OVER A TWELVE MONTH PERIOD. (THE NUMBER OF DETERMINATIONS SHOWN IN BRACKETS).

Subject	Average thresholds for 1st six month period (g)	Average thresholds for 2nd six month period (g)	Average threshold for twelve month period (g)	Standard deviation for all runs
C.McE.	2.6 (52)	2.6 (52)	2.6 (104)	± 0.23
N.L.A.	3.1 (25)	3.2 (26)	3.2 (51)	± 0.40
G.H.B.	3.0 (42)	3.1 (41)	3.0 (83)	± 0.28
M.K.B.	2.9 (64)	2.9 (63)	2.9 (127)	± 0.31
J.S.G.	2.5 (6)	3.3 (7)	2.9 (13)	± 0.43
A.C.	3.1 (11)	2.9 (12)	3.0 (23)	± 0.24
F.L.	2.6 (32)	2.6 (31)	2.6 (63)	± 0.28
M.H.O.	3.6 (4)	3.2 (4)	3.4 (8)	± 0.38
P.G.	2.9 (46)	2.8 (46)	2.8 (92)	± 0.43
J.C.G.	2.8 (55)	2.7 (55)	2.8 (110)	± 0.31
Means of all subjects	2.83 (337)	2.85 (337)	2.84 (674)	± 0.377

It can be seen that over a period of time the day to day variation cancels itself out and results are remarkably constant from one period to the next. The figures adequately display the low fatigue element and illustrate the lack of adaption to g. When all subjects are included there is a mean threshold which again is virtually constant over a long period of time. Thus the method could be used to compare results on any subject over a period of months without undue error.

DISCUSSION.

The results obtained in this series are higher than those described by Byford and Howard (1956). This can be accounted for by the fact that dark adaption is not complete in the present series and that there is a low level of ambient illumination. The thresholds could be made lower if desired by using neutral density filters of lower value. The present value of 0.5 log.units was chosen because the method was primarily developed to allow decrements in threshold to be measured and a very low initial value was not desirable.

The advantages of the method are :-

(a) specification of the variables is possible, thus allowing accurate duplication of the method in other laboratories (fig. 35);

(b) thresholds can be determined with a minimum of discomfort since only two runs are necessary at the most; and the jolt (0.1g/sec.) is so low

that unpleasant sensations are entirely absent;

(c) the threshold can be selected by the use of an appropriated neutral density filter. Black-out can be obtained at extremely low values of g thus allowing a large safety margin between black-out and unconsciousness;

(d) full cardiac compensation is continually present. This eliminates one of the physiological variables and further increases the safety margin;

(e) adaption and learning do not seem to occur, the mean thresholds remaining constant over many months;

(f) it is suitable for all centrifuges at present in existence.

No serious disadvantages have been found with this method. It is simple to use, is pleasant for the subjects and requires a minimum of equipment. It was described to the European Congress of Aviation Medicine in Stockholm in 1957 and proposed as the standard method of threshold determination in all centrifuges of the Western World. This method was again discussed at the Aero-Medical Association in Washington, 1958, and was issued by the centrifuge standardisation committee for test to the member countries. To date favourable results in agreement with those described here have been obtained from the major American acceleration units.

SUMMARY.

A method of threshold determination is described which utilises visual black-out as the end point. The method consists of viewing a pale red light against a homogeneous background with the dark adapted eye. The thresholds can be varied at will by the use of various filters and allow a large safety margin between black-out and unconsciousness. The method is capable of precise specification and is easily performed.

The use of a rate of application of 0.1g/sec. allows the acceleration time curve to be linear, while cardiac compensation has time to develop fully. The use of repeated plateau runs with their concomitant fatigue is avoided.

CHAPTER 1

INTRODUCTION

PART II

THE EFFECTS OF OTHER PHYSIOLOGICAL STRESSES ON MAN'S TOLERANCE TO INCREASED GRAVITATIONAL FORCE

CHAPTER I

INTRODUCTION

During the last few years the total number of aircraft accidents in the Royal Air Force has been steadily diminishing and the percentage attributable to pilot error has fallen proportionally. Despite these gratifying figures the fact remains that there is a number of fatal accidents in all the air forces of the world which appear to be due to failure of the human factor. In the majority of these cases observers on the ground tell a similar story; the aircraft is seen to be out of control and flies straight into the ground leaving no evidence save small pieces of wreckage strewn over the surrounding countryside. The most disturbing feature of these cases is that, although some degree of warning must always occur, the pilot makes no attempt to use his radio or his ejector seat. This can only mean that he has lost useful consciousness.

In the modern high speed aircraft the demand for a continued high degree of cerebral alertness is insistent, with any lapse of attention or concentration inevitably leading to disaster. This places a strong psychological stress on the pilot, whose mind is continually working at full capacity. This utter dependence on consciousness may seem not unreasonable since it is a state which is normally taken for granted. It must be remembered however that the physiological mechanisms maintaining consciousness are easily upset and that the brain, although the master control, has the least reserve. Thus the oxygen reserve of the brain allows only six seconds of useful consciousness, although the brain demands a blood flow ten times that of the muscles, weight for weight, for normal function. That consciousness may be lost quite simply can be shown in many ways. For example, passive tilting from the horizontal to the vertical causes syncope in many normal people (Brigden, Howarth and Sharpey - Schafer, 1950).

Since it seems unlikely that there is any gross ante mortem pathology in these fatal crashes, one must consider therefore the physiological causes of disorders of consciousness.

The final cause of loss of consciousness must be some change in the neurones themselves leading to explosive discharge or complete inhibition. Converging on this final stage, however, are many varied factors which may act singly or in combination. Some of these stresses which have particular application to aviation are discussed below.

There are a number of factors leading to loss of useful consciousness which produce little change at cell level. These arise in the highest centres of consciousness and are comprised of emotional changes and disorders of affect. In considering these causes there is no clear cut division between physiological processes and psychopathology; the only reason for considering them physiological in

this context is that they occur in normal people who, if they survive the incident, still appear normal by our methods of judgment.

Perhaps the commonest occurrences of this type are those arising from abnormal extremes of sensory inflow, In modern flying there is a large continual volume of sensory inflow, visual, auditory, tactile and proprioceptive. With adequate training the pilot can adapt himself to assimilate this variety of sensations, integrate them and make the appropriate responses. There are occasions, however, particularly in an emergency, when this inflow is suddenly increased and under the bombardment of stimuli requiring immediate attention function at higher level ceases and a trance-like state develops. Although not unconscious by the usual criteria, the subject behaves in a decerebrate way and can play no useful part in the control of his aircraft. Closely allied to this condition is the "panic state," when, under the same stimulation, the pilot becomes so agitated that he

finds himself unable to make the response required by the developing situation.

At the opposite extreme there are conditions of flight where the sensory inflow is reduced to a minimum. This is most commonly encountered at high altitudes. In the stratosphere weather conditions are perfect and the normal bumps and swings of the aircraft no longer occur. There are few visual stimuli; the sky is very dark, while below the earth is usually enshrouded with cloud like a white carpet. Apart from intermittent radio messages there is only the faint throbbing of the engines to keep out the invading silence. Under these conditions of sensory deprivation an interesting state develops called depersonalisation. This is a disorder of perception. The pilot's mind wanders from his undemanding task and roams freely. He begins to dream and, within a short time, dreams and reality become inseparably mixed. In this state purposeful action is beyond his ability and any flight emergency will invite disaster.

Emotional factors are also of major significance. The everyday emotions of fear, anger and anxiety can diminish the level of cerebral alertness and predispose to unconsciousness due to some other factor (Powell, 1956, 1957; Silverman and Cohen and Lazar, 1957) or produce it rarely on their own account (Rook, 1946).

Epilepsy is commonly invoked as an explanation of an unexplained loss of consciousness. At this point it is well to consider what is meant by epilepsy. To the clinician it is generally subdivided into grand mal and petit mal, both characterised by an interruption in the stream of consciousness. In the case met by the civilian medical practitioner these attacks occur spontaneously; however in many apparently normal people similar attacks can be provoked experimentally, the only difference being the intensity of the threshold stimulus. These latter cases are unlikely ever to have an attack under everyday conditions of life. The characteristic clonic seizures of epilepsy are also of doubtful significance: epilepsy is often associated with flaccidity,

while syncope may be associated with convulsive movements (Brown and McCormack, 1941; Medical Research Council Report, 1944). Similarly, in the Stokes-Adams syndrome convulsive movements are common: and, if cardiac ventricular standstill lasts for ten seconds or more, tonic and clonic convulsions usually supervene. Parkinson, Papp and Evans (1941) have also shown that incontinence can occur in Stokes-Adams attacks. Thus epilepsy is a condition in which patients are distinguished from the normal populace by their lowered threshold rather than by any inherent difference. It is fair to say that when an isolated episode of unconsciousness is attributed to epilepsy in an apparently healthy person with no previous convulsive history, the diagnosis is open to doubt. If by epilepsy is meant unconsciousness due to some unknown factor, then it is perhaps justified but an unfortunate choice of expression.

Nevertheless, in spite of the foregoing discussion, cases of epilepsy may occur in the

air due to some additional stimulus not normally occurring on the ground and it must be remembered that an epileptic may become a pilot by deliberately misleading the medical boards with regard to his past history. This was relatively common in wartime (Rook, 1946).

The post hoc diagnosis of unconsciousness in flight is fraught with difficulty as in many cases the pilot is dead and his body disintegrated. Apart from speculation there is little chance of firmly establishing the causation. Although the psycho-physiological factors mentioned above may be operative, it is always as well to look for more physical causes, as it seems probable that psychological strain is more likely to be a predisposing rather than a primary cause.

The physical factors which may be involved in disturbances of consciousness are changes in the cerebral circulation. Injury and noxious substances also play a part. However, in this discussion injury will not be considered as in

most cases the aircraft has not been in any accident when the incident occurs.

Although loss of consciousness in flight is commonly due to cerebral anaemia, it also occurs with cerebral congestion. The latter is due to arterial hypertension or increased venous back pressure. Arterial hypertension, while a cause of convulsions and unconsciousness, is not likely in the type of individual who is a pilot, since frequent medical examinations eliminate those who show any tendency to a high blood pressure. Venous congestion on the other hand is a distinct possibility. Cerebral congestion while flying classically occurs during manoeuvres involving negative g, that is, where the body is accelerated in a head to foot direction causing the blood to be forced into the vessels of the head and neck. Human tolerance limits are of the order of -3 to -3.5g for 30 sec. (Maher, 1948; Beckman, 1952; Sieker, 1952). The cause of unconsciousness appears to be cerebral anoxia arising from vascular stagnation. The latter

arises secondarily to the extreme bradycardia which develops in response to over-stimulation of the carotid sinus (Simons and Henry, 1950; Henry, 1950). However, negative g aerobatics have been banned in the R.A.F. and eye witness reports can usually establish whether this type of manoeuvre preceded an aircraft crash. A more mundane cause of cerebral venous congestion is a bout of coughing. Rook (1946) has described four cases of aircrew who lost consciousness during coughing while Whitley (1943) has described four similar ones and found a hundred other non-aircrew cases in the literature. The mechanism by which syncope is produced in these cases has been described by Sharpey-Schafer (1953). Thus coughing or choking may well be a cause of an incident in flight.

Cerebral anaemia is the classic cause of loss of consciousness. In the air the commonest cause of cerebral anaemia is positive acceleration. When g is applied two factors come into operation which cause or contribute to cerebral ischaemia.

Firstly, the abdominal contents are forced down into the pelvis with descent of the diaphragm (Ruff and Strughold, 1939; Rushmer, 1944). This effect is associated with descent and rotation of the heart - increasing the heart-brain distance. As the g increases so does the weight of the blood and the heart is called upon to do more work to maintain a supply of this heavy blood to the brain. This is aggravated by the increased heart-brain distance and so, with progressive g, the blood supply to the brain diminishes, the limiting factor being the pressure necessary to open the aortic valves. The second mechanism takes a longer time to develop and requires the g to be maintained for over 6 seconds. Blood pools in the abdomen and lower limbs so that venous return diminishes and as the systemic demand for a greater cardiac output arises the ventricles have less and less blood to fill them until eventually the ventricle may be completely empty during the latter stages of systole (Henry, 1950). In practice both of these mechanisms occur together and each contributes to the failure to maintain an adequate circulation under g.

Other factors predisposing to cerebral anaemia are general ones: loss of blood or extensive vasodilation elsewhere in the body, such as occurs with heat or after ingestion of alcohol. When there is extensive vasodilation then pooling has to some extent commenced and the reduced efficiency of the heart is manifest in its rapid action with a low or falling blood pressure.

Alterations in cerebral blood flow can also occur locally within the skull, by vasoconstriction or dilation. There is some evidence (Schmidt, 1950) that cerebro-vascular control is obtained by relaxation of a pre-existing high degree of tonus rather than by active vasoconstriction. These changes might be brought about by nervous control or by local changes in vessels due to the accumulation of metabolic products. The evidence for the former is scanty and has been discussed by Schmidt and Kety (1947) who conclude that nervous control is unlikely to be of any significance if it exists at all. On the other hand large variations in flow may be

produced by metabolic products. All the usual concomitants of metabolism cause dilation of cerebral blood vessels: thus decreasing the O_2 supply or increasing the pCO_2, hydrogen ion concentration, or deep body temperature causes an increased blood flow. In other words, cerebral flow is increased by the same factors as increased flow in other vital vascular territories. On the other hand the reverse condition is also produced by the opposite changes. This is especially true of the respiratory gases - a high O_2 tension and low CO_2 tension causing increased vascular resistance. In fact a decreased pCO_2 is the only physiological stimulus causing intense cerebral vasoconstriction and the response of the cerebral vessels is unique since no other vessels in the body are known to react to such a degree to hypocapnia. The changes due to blood gases are shown in Table 1.

TABLE 1

Effect of Changes in Arterial Gas Tensions on Cerebral Blood Flow in Man

(after Kety and Schmidt 1946, 1948)

Conditions	Mean Blood Pressure: mm.Hg.	Cerebral Blood Flow: ml/100 g./min.	Cerebral Vascular Resistance
Air Breathing Control	86	54	1.6
Hyperventilation (low CO_2)	98	36	2.7
High (5-7%) CO_2	93	93	1.0
Low (10%) O_2	78	73	1.06
High (85-100%) O_2	98	45	2.2

Changes in the blood gases produce the most marked effect; cold, acids and cholinesterases have little effect and it is doubtful if any vasoconstriction attributable to them occurs physiologically.

The effect of a high oxygen tension is interesting academically but until recently appeared to be of no practical importance. However, in modern high speed aircraft, pure oxygen is breathed from take-off, and in low level flight is being inspired at atmospheric pressure. Although under these conditions local cerebral blood flow is diminished the oxygen tension is sufficient to prevent impairment of consciousness. It must also be borne in mind that cerebral blood flow is largely determined by arterial blood pressure at head level.

To summarise, the main factors which may at various times cause a diminished cerebral circulation are a fall in blood pressure at head level, cerebral venous congestion, generalised vasodilation

and local changes in the calibre of the blood vessels due to a high partial pressure of oxygen or a low partial pressure of carbon dioxide.

The other factors on which consciousness depends are an adequate supply of oxygen and nutrients reaching the nerve cells in the absence of noxious substances.

Since the ill-fated balloon flight of Tissandier and his colleagues, anoxia has continued to be a constant hazard in aviation. When aircraft in the second world war started to climb higher, it became apparent that better oxygen supply systems were needed. Above 10,000 feet in daytime, oxygen rather than air must be breathed to maintain efficiency. At 34,000 feet, 100 per cent oxygen is required, while above 40,000 feet pressure breathing becomes necessary. To maintain adequate levels of blood pO_2 above this height, 100 per cent O_2 is forced into the aviator's lungs at a positive pressure which

would increase with altitude to a value of about 100 mm.Hg. at 70,000 feet. Thus above 10,000 feet the oxygen supply is maintained by a mechanical system in which there is always the possibility of failure. The effects of oxygen lack are notoriously insidious and have been compared to the early stages of drunkenness. At first there are errors of judgment associated with the inability to appreciate errors as they are made and this sense of over-confidence continues until consciousness is lost. It must be realised therefore that a pilot may be suffering from a mild degree of anoxia without being aware of it and, although he is conscious and quite confident of his ability, his alertness is diminished and he has moved down the scale towards the threshold of unconsciousness.

Other varieties of anoxia are encountered in flight. Anaemic anoxia is uncommon in aviators due to selection and the intensive medical attention they receive. However, stagnant anoxia

may arise in cerebral venous stasis as described above. Histotoxic anoxia may also occur when the oxygen is not utilizable at cell level. In flying this does occasionally occur when the causative factors are noxious fumes (from the engine) entering the cockpit. Carbon monoxide impairs the oxygen-carrying capacity of the blood; other vapours such as ethylene glycol act as cell poisons inhibiting the cellular enzyme systems responsible for oxygen transfer.

The most important nutrient for cerebral metabolism is glucose. A failure of the supply of this essential substance is rapidly followed by unconsciousness, while a sufficient decrease in its circulating level in the blood causes impairment of normal cerebration. There are wide individual variations in man's susceptibility to a low blood sugar but it is probably safe to say that a fall to 30 mg. per cent is usually associated with unconsciousness while levels below 70 mg. per cent will cause some diminution in cerebral function.

From these brief notes on the physiological factors leading to unconsciousness it can readily be appreciated that the continuum of consciousness is easily breached and may be interrupted by many and varied simple conditions. Since these rarely act singly and are closely interrelated, it seems reasonable to suppose that several stresses, each in itself of subliminal value, may summate to produce a profound effect which cannot be related to any single factor.

In flight many of these stresses are potentially present. Fear, anxiety and positive acceleration are constant companions of the modern aviator, while hyper-ventilation and anoxia frequently occur. In the rapid pace of squadron life, hypoglycaemia also can easily arise, either from missing meals or to rebound after a purely carbohydrate meal. It therefore seems reasonable in an investigation of the cause of unconscious episodes in flight to examine in the first place the physiological stresses which are known to occur and to determine whether their summation may give rise to an incident which is unexplainable by

any one factor.

Since acceleration is ever present in high speed flight, it has been used as the basic stress to which others might be added. Loss of vision also provides a useful end point short of unconsciousness by which the effects of stress summation can be studied.

This part of the thesis presents the results of investigations into the effect of in-flight stresses on tolerance to positive acceleration.

There is little historical background to this problem which is essentially one of the "space age." Over the last few years, however, various pointers have emerged from the air forces of the western world. These will now be summarised briefly.

It is felt that loss of consciousness of the pilot is probably not uncommon in modern high performance aircraft. Its incidence is difficult to determine as in most cases the episode will lead

to a fatal accident. In the U.S. Naval Centre at Pensacola, a questionnaire was circulated to pilots on the final stages of flying training asking for details of episodes in the air. The results have been described by Stauffer (1952) who found that of a hundred and nineteen pilots, twenty-seven (twenty-three per cent) had been unconscious one or more times and often when flying solo. Stauffer concluded that these were due to positive acceleration and a lack of knowledge amongst the pilots of how to protect themselves. In the U.S.A.F. training command anxiety was also being felt about the number of aircraft accidents in which failure of the human factor appeared to be the cause. A study of the aircrew's routine revealed that many pilots missed meals, or "made do" with snacks of coffee and doughnuts. Regulations insisting on regular adequate meals resulted in a dramatic fall in the accident rate. This finding led Lawton (1953) to deduce that hypoglycaemia was a factor in the causation of aircraft accidents.

In France investigations of accidents due to pilot error have been reported by Senegas and Cantoni (1957) and Rémond (1957). The former, on the basis of ten fatal cases, ascribed the accidents to disorientation or misreading of instruments due to inexperience. A feature of all their cases was the failure of any pilots to eject, although adequate warning occurred. (Several pilots were ordered to eject by their flight commanders but did not do so, nor did they acknowledge radio signals). The description of the cases does not allow any clear cut conclusions to be drawn in spite of their ingenious analysis by the authors. Each case might well have been due to loss of consciousness of the pilot.

Rémond analyses loss of consciousness of obscure origin. His analysis involves psychological mechanisms and he lists five probable causes. These are:

1. Abnormal sensory sensitivity (e.g. reflex epilepsies), when various stimuli are repeated at certain frequencies in hyperexcitable subjects.

2. Psychomotor epilepsies occurring as a result of stress causing activation of a cerebral area whose resistance is lowered by previous stress.

3. Syncopes in subjects with vagal hyper-excitability in conditions such as heat, acceleration, emotional upset.

4. Paroxysmal sleep.

5. Paroxysmal coma associated with visceral diseases.

There is no doubt these are causes of unconsciousness and may occur in flight. However, it is dubious if these causes alone would explain many aircraft accidents. The importance of this paper is that Rémond points out that there are predisposing causes and that summation of stresses may occur to produce loss of consciousness.

In Holland there has been little information obtained. Palthe (1959) describes a condition of "lipothymie" which is similar to Rémond's

syncope and is a general term to cover unexplained loss of consciousness in a normal person. He finds certain people exhibit this condition, e.g. fainting at the sight of blood, vasovagal attack in the heat or orthostatic collapse. He maintains that these lipothymic subjects are not more prone to aircraft accidents than those who have not this diathesis. He also describes a condition occurring with violent emotional stimulation called "Totstellreflexe" which is akinesia with mutism, or to panic with loss of purposeful action. He is unable to relate these conditions to known accidents but points out that they may be involved.

Previously Palthe (1953) had pointed out that the effect of hyperventilation was potentiated by a concurrent hypoglycaemia and that a number of people were susceptible to this combination which he called the Hv.Hg. complex. These people would lose consciousness by "taking one hundred breaths in three minutes" if their blood sugar was low. This could be aborted by induced hyperglycaemia which also normalised the E.E.G. The condition does not appear if the subject hyperventilates half an hour after the ingestion of fifty grams

of glucose. On the basis of these findings, Palthe advocates that pilots should not fly on an empty stomach and, when physical or mental stress is anticipated, the taking of sugar is advisable. Thus the elements of summation of effect of hyperventilation and hypoglycaemia were appreciated by Palthe and in his description of "lipothymie" he acknowledges several of the other factors which may cause loss of consciousness.

The Norwegian Air Force were amongst those who recognised that heat might influence the pilot's tolerance to acceleration. No research has been carried out but, on questioning, aircrew stated that they could not "pull tight turns" after they had been flying in immersion suits which made them hot. A similar finding was reported by Martin and Henry (1951). These latter authors carried out an experiment in the centrifuge at Wright Patterson Air Force Base which consisted of determining a man's g threshold using the peripheral light system and then heating or cooling his environment and carrying out duplicate

measurements. There was no attempt to control the experiment and the subject was either normal, cold and shivering or hot and sweating. It is therefore difficult to know just what heat load was applied. However, when the subject was hot his threshold fell by about 0.2g, from which the authors conclude that as long as heat regulation is successful the subject will not show significant impairment of g tolerance.

The same anxiety over loss of consciousness is being felt by the Indian Air Force. In many of their fatal aircraft accidents a similar history can be obtained. An aircraft takes off in the early morning to avoid the excessive heat load which occurs later in the day. The pilot climbs to about ten thousand feet and carries out some aerobatics, the aircraft is then seen to fly into the ground. Subsequent analysis frequently shows that the pilot has risen late and missed his breakfast. Although no experimental data has been obtained, these observations have led Indian medical officers to postulate that hypoglycaemia and heat may lower the threshold of consciousness and predispose to collapse in the air.

The most pertinent data has been provided by the researches of the Canadian team working at the Aviation Medical Laboratory (D.R.M.L.), Toronto. Powell (1956) collected a series of nine cases of pilots who had survived an episode of unconsciousness in the air. No pathological lesion was found on examination but, in each case, there were several factors operating which may have contributed to the incident. The episodes were labelled "physiological unconsciousness" and the possible operating factors in their production were:

(a) Previous or concomitant positive acceleration;

(b) Hypoglycaemia;

(c) Hyperventilation;

(d) Fear or anxiety;

(e) Susceptible subjects who had early slow E.E.G. activity with hyperventilation.

Further work in D.R.M.L. established this concept more firmly and has been reported by Brent, Caney, Powell, Scott, Taylor and Franks (1957), who

found fasting, hyperventilation and g summated to produce earlier showing of the E.E.G. than one alone or any combination of two. Of their fourteen cases, three lost consciousness at levels of 3.0g - 3.5g. In the last year Scott (personal communication), working at D.R.M.L., has continued this work and confirmed the findings, although he believes that delta activity in the E.E.G. preceeds the loss of consciousness and is of diagnostic importance. This has not been the finding in the present study.

CHAPTER II

THE EFFECT OF HEAT ON TOLERANCE TO ACCELERATION

A synergism between heat and other flight stresses is suggested by the accident rates in the eastern air forces. In the Middle and Far East Air Forces the fatal accident rates where pilot error is thought to be the cause are higher in the hot season, while in the Indian Air Force the accident rate shows a sharp increase in February and is maintained high throughout the summer months (Nath, personal communication).

Although the development of high body temperatures in normal people is associated with the tropics, in a modern air force it may occur in aircrew regardless of the operational theatre and is one of the immediate problems of high speed flight.

The origin of the heat may conveniently be considered under two headings, static and kinetic.

(a) Static.

To-day aircraft on "standby" require to be standing on the runway with their crews in place ready for immediate take-off. It is in this situation that high static heat loads are mainly encountered. In Fig. 1 the temperature inside the cockpit of a Canberra aircraft is compared with the ambient air temperature on a sunny day. The difference between the two represents the effect of solar radiation due to the "glasshouse effect." It can well be understood that at hign altitudes the effect of solar radiation will be increased as the filtering effect of the atmosphere diminishes. Blockly, McCutchan and Taylor (1954) showed a 20 per cent increase in heat load due to solar radiation at 100,000 ft. as compared to 5,000 ft. Thus even the static heat load alone is sufficient to limit human performance or in some cases to cause collapse.

(b) Kinetic heating (sometimes called aerodynamic heating) is derived from two sources, "ram air effect," which is compression of air by the impact

of the aircraft structure and skin friction due to the viscous motion of the air over the aircraft surfaces. Both of these depend on the velocity of the aircraft and will become increasingly important as aircraft progress even further beyond the speed of sound.

Fig. 2 shows the aircraft surface temperature which occurs at Mach 1.0 at various altitudes. For comparison, curves are also shown for Mach 1.5 through 4.0 which will be being attained in the near future (Mach number is the ratio of true air speed to the local speed of sound at any altitude). From this graph it will be seen that above Mach 1.0 at altitudes below 35,000 ft. the temperatures may be beyond the limits of the human body. When one adds the additional heat arising in the cockpit due to the "glasshouse effect" of solar radiation, the heat load becomes extremely high and even with present cooling systems the aviator may be unable to maintain heat balance.

With these points in mind, it is of interest and of importance to study the effect of heat on

human tolerance to acceleration. This chapter presents the results of such a study.

METHOD

Tolerance to acceleration was measured by means of a visual threshold determination. The end point used was loss of central vision with the dark adapted eye as described in Part I, Chapter IX.

The temperature of the gondolas of the human centrifuge cannot be accurately controlled. It was decided therefore to heat the man, rather than his environment. The method was to supply an adequate amount of heat to the subject and as far as possible to eliminate heat loss from the body. At high temperatures loss of heat is by evaporation of sweat. The prime object therefore was to exclude this means of temperature regulation. To this end the subject, clad in shorts and suitably instrumented, (Plate I) donned a polythene overall which was tight fitting at the neck, wrists and ankles. On his feet he wore polythene boots which overlapped the suit and rubber gloves were worn

overlapping the sleeves. This assembly is shown in Plate II. When sweating commenced, the atmosphere within this clothing assembly rapidly became saturated after which no further evaporation of sweat took place. On top of the insulating polythene layer was worn an R.A.F. electrically heated suit. This suit operated from a 24v. D.C. supply and had an output of 300 watts. Further insulation against heat loss was provided by sheepskin flying boots and lined flying gauntlets. This final assembly is shown in Plate III. As an additional safeguard against heat loss by radiation and convection, the ambient temperature of the gondola was raised by means of a 1500-watt convector heater below the subject's seat and a 500-watt infra-red lamp directed at the subject's head. The effect of these car heaters on ambient air temperature is shown in Fig. 3. By the above means it was found possible to raise the mouth temperature of the subject to 101°F. in 45-60 min.

Seven experienced centrifuge subjects were used for the investigation, none of whom had taken

part in any other heat experiments in the previous year. Each subject carried out two runs to act as controls for threshold and mouth temperature. Both the subject's and the gondola heating were then switched on and further thresholds estimated at varying periods of time until a mouth temperature of 101°F. was reached, or until the subject asked for the experiment to be terminated.

Routine recordings were made of mouth temperature and pulse rate. The latter was obtained from the electrocardiogram - the leads used for the E.C.G.'s were standard lead I, aVF, V_1, V_3, V_4. In order to minimise 50 cycle mains frequency interference induced in the E.C.G. leads from the elements of the electrically heated suit, the D.C. supply was earthed, thereby surrounding the subject with an effective screen. In some subjects records of respiration and skin temperature were obtained. Mouth temperatures were measured by means of a thermocouple embedded in dental wax held between teeth and cheek; this was frequently checked against a clinical thermometer. Respiration was recorded from a thermocouple in

the nostril. Plate I shows the instrumented subject before donning the clothing assembly.

RESULTS

Two distinct types of response to the effect of heat on g tolerance were obtained. The first group of three subjects (M.K.B., N.L.A., P.G.) showed a large fall in threshold when their deep body temperature was raised (Fig. 4). The total changes in the three parameters measured are shown in Table 2.

T A B L E 2

Changes in Mouth Temperature, g Threshold and Pulse Rate in the Subjects of Group I during Heating

Subjects	M.K.B.	N.L.A.	P.G.
Duration of heating (min.)	50	49	40
Initial temperature (°F.)	99.2	98.8	98.7
Final temperature (°F.)	101.0	101.5	101.2
Rise in temperature (°F.)	1.8	2.7	2.5
Initial threshold (g)	2.6	3.4	2.7
Final threshold (g)	1.2	2.1	1.8
Fall in threshold (g)	1.4	1.3	0.9
Initial pulse rate/min.	96	93	115
% increase in pulse rate	34.4	44.0	27.8

From Fig. 4 it will be seen that the mouth temperature rose slowly at first during the stage when the gondola was cool and the subject's sweat was evaporating within the suit. After about 20 min. the mouth temperature began to rise rapidly and in a linear fashion. It is reasonable to assume that this point of inflection of the curve represents the time at which the heat capacity of the shell was exceeded and heat loss from the head could not compensate for the steady heat input and is a measure of the efficiency of the polythene clothing.

During the first 20-30 min. there is an inconstant change in the threshold; M.K.B. shows little change, N.L.A. a slight fall and P.G. a rise. Thereafter there is a progressive fall in threshold as the temperature rises. It is interesting to examine the changes occurring in this latter phase of the experiment. These are shown in Table 3.

TABLE 3

The changes in the mouth temperature and g threshold of the subjects in Group I are shown for the latter part of the experiment (from the inflection of the threshold curve).

Subjects	M.K.B.	N.L.A.	P.G.
Threshold at point of inflection (g)	2.6	3.2	3.2
Final threshold (g)	1.2	2.1	1.8
Change in threshold (g)	-1.4	-1.1	-1.4
Mouth temperature at point of inflection (°F.)	99.4	100.4	99.6
Final temperature (°F.)	101.0	101.5	101.2
Change in temperature (°F.)	+1.6	+1.1	+1.6
Rate of change of threshold with respect to temperature (g/°F.)	0.88	1.00	0.88

From Table 3 it will be seen that the rate of change of threshold with temperature is very similar in the three subjects and, as can be seen in Fig. 4, is almost linear.

The changes in pulse rate with heat are as expected. The added g produces the same change

in pulse rate whether the temperature is normal or high. The effect of g on the pulse rate is represented by the shaded areas in the graphs.

In the second group of subjects the changes in body temperature were of the same order as in Group I. The change in threshold, however, was very much less (Fig. 5). There was no significant difference between the two groups of subjects as regards age, height or weight. The results for this group of subjects are shown in Table 4.

TABLE 4

Changes in Mouth Temperature, g Threshold and Pulse Rates in the Subjects of Group II during Heating.

Subjects	J.C.G.	G.H.B.	C.McE.	F.L.
Duration of heating (min.)	55	72	65	60
Initial temperature (°F.)	99.1	97.7	98.0	99.0
Final temperature (°F.)	102	100.9	100.7	100.9
Rise in temperature (°F.)	2.9	3.2	2.7	1.9
Initial threshold (g)	3.4	2.9	2.4	2.6
Final threshold (g)	2.7	2.4	2.4	2.1
Fall in threshold (g)	0.7	0.5	0.0	0.5
Initial pulse rate (per min.)	100	72	70	-
% increase in pulse rate	40	62.5	-	-

In this group of subjects the increase in body temperature was greater than in the previous group due to lower initial temperatures. The fall in threshold, however, was much less, one subject (C.McE.) showing no change at all. The change in pulse rate was larger than those of group I; this can be explained by the slightly larger temperature increases to which they were subjected. One subject (C.McE.) became unconscious during the last centrifuge run, just at the moment he signalled black-out.

The subjective sensations recorded by the two groups of subjects varied. The subjects of Group I noticed no change in the quality of the end-point and did not realize their threshold was falling. The subjects of Group II all noticed that the end-point was associated with unpleasant constitutional upset and was a true black-out rather than simple central light loss. Fig. 6 shows the changes in threshold for all subjects, the results are shown in Table 5.

TABLE 5

Shows the Changes in Threshold of all Subjects expressed as a Percentage of the Normal Value. The Relation between this Fall and the Change in Temperature is also shown.

Group	Subject	% Change in Threshold	Change in Mouth Temperature	% Fall in Temperature per $^{o}F.$
I	M.K.B.	-53.5	+1.8	29.8
	N.L.A.	-36.8	+2.7	13.6
	P.G.	-39.0	+2.5	15.6
II	J.C.G.	-20.0	+2.9	6.9
	G.H.B.	-15.5	+3.2	4.9
	C.McE.	0.0	+2.7	0.0
	F.L.	-18.5	+1.9	9.75

From Table 5 it can be seen there is a large difference in response to heat between the two groups, Group II showing a smaller percentage fall in threshold in spite of a greater increase in temperature.

DISCUSSION

"When man stands erect without moving, he hovers on the verge of circulatory collapse" (Asmussen, Christiansen and Nielson, 1940). This statement expresses the problem of gravity shock and orthostatic hypotension to a nicety. The adoption of the erect posture by the primates has placed a strain on the circulatory system. Despite compensatory reflexes such as tachycardia and vasoconstriction arising from the pressure sensitive areas of the carotid sinus and aorta, and increased production of adrenaline and noradrenaline (Munro, 1958), there is still a fall in blood pressure when man rises from the reclining to the erect posture. This fall may be transient if the compensatory mechanisms are efficient, but there is a certain number of individuals in whom the hypotension persists and often leads to syncope.

This fall in blood pressure is aggravated in all subjects if the change in position is passive or if, when erect, the subject stands without moving. The hypotension is due to the pooling of blood in the dependent parts of the body under the action of gravity. The pooling in the abdomen on standing is small compared to the loss into the legs (McLean, Allen and Magath, 1944) and many orthostatic circulatory failures can be prevented by tourniquets round the legs. These circulatory changes are analogous to the circulatory changes under acceleration. In the latter case we are merely aggravating the effect of gravity; thus the subject standing erect without moving can tolerate little acceleration even with full use of his compensatory reflexes. The situation arising in the human centrifuge is very similar to a subject being tilted from the horizontal to the vertical only in the centrifuge the subject stays seated at rest and a g field is created around him.

Heat produces similar changes on the circulation. As the body temperature rises, there is a progressive vasodilation, large areas

of the cutaneous vascular bed opening to allow heat exchange. These changes are accompanied by changes in the blood pressure, cardiac output and blood volume (Scott, Bazett and Mackie, 1940). There is, at first, a fall in blood pressure which, however, is offset by an increased cardiac output which may amount to 30 per cent of the basal value (Herrington, 1940), and for the subject reclining at rest, this allows the mean blood pressure to remain at normal levels (there is a rise in systolic blood pressure and a fall in diastolic blood pressure). The vasodilation accompanied by an increased cardiac output is associated with an increase in plasma volume (Bazett, Sunderman, Doupe and Scott, 1940). This increase, however, takes at least several hours to develop (Newburgh, 1949) and is not applicable to short exposures. In a short exposure there is a loss of effective circulating blood volume and a tachycardia and increased cardiac output. Thus the subject at rest in the heat is already utilizing his compensatory mechanisms to maintain his circulation. If any additional stress is added, e.g. gravity, then he will have little reserve to

combat it and collapse may ensue. This is borne out by experimental data. Eichna (1947) found 23 per cent of 150 men had orthostatic hypotension with a reduced cardiac output and tachycardia when standing in a hot environment. Thus heat can push man over the brink into orthostatic circulatory collapse. The position has been summed up by Hellebrandt and Franseen (1943) who say: "Gravity shock is aggravated by high environmental temperatures which augment the disparity between the volume capacity of the vascular bed and the volume blood flow."

During accelerations, which are equivalent to an augmented postural change in their effect on the circulation (vide supra), it is to be anticipated that heat will have a similar adverse effect on tolerance. This change was shown by the subjects of Group I, whose threshold fell progressively with rise in body temperature. Taken as a whole there was a large subject to subject variation in the fall in threshold (Table 5). However, if the experiment was considered in two parts - (a) the initial half when the heat capacity of the shell was falling and the mouth

temperature rising slowly and (b) the second half when the compensation was inadequate and the temperature rising rapidly - there is more correlation between subjects. In the first half (a), there is a small inconstant change in the threshold. In (b), however, all subjects show an almost linear fall in threshold; the value for the three subjects of this group being 0.9, 1.0 and 0.9 g/°F.

In the second group there was a fall in threshold in three of the four subjects but this was small in comparison to the change in Group I. A clue to the probable reason for this is given by the fourth subject (C.McE.) who showed no change in threshold measured by central light loss, but lost consciousness at threshold on the last run. The level of g at which this episode took place was 2.4 g, which is well below the unconscious threshold of the relaxed subject (4-5 g). There was therefore a fall in tolerance to acceleration of about 40-50 per cent, although this was not reflected in the determination of central light loss. This subject said that he felt panic-

stricken and could feel his heart pounding, which frightened him even more. The central light loss under the present conditions of determination occurs about 1-2 g below the level of unconsciousness and 0.5 - 1 g below absolute blackout levels. If the subject tenses himself, however, he can retain his vision up to the absolute blackout level, since by tensing he improves the venous return to the heart and elevates his diaphragm and so the blood pressure at head level is maintained. If fear and muscle tension are present, then the difference in level between greyout, blackout and unconsciousness becomes negligible and collapse occurs with little visual warning. This is almost certainly what happened to C. McE. He admits to being frightened and being unable to relax and it seems likely that he maintained the level of C.L.L. by tensing until, in the last run, his absolute blackout level had come down to 2.4 g and at this point, knowing it was the final run, he relaxed somewhat and immediately lost consciousness. Thus the three other subjects of Group II may also have been tense and so have shown only a small fall

in threshold as measured by C.L.L., although in fact their absolute blackout and unconscious thresholds were falling markedly. This hypothesis is borne out to some extent by their subjective statements that the end point was more like an absolute blackout than mere central light loss; and by the fact that two of them (J.C.G. and G.H.B.) were noticeably restless and were continually shuffling their feet. This latter change is reminiscent of the compulsive restlessness which occurs in resting men subjected to high heat loads. Thus, although the subjects of Group II showed a smaller fall in threshold as measured under the conditions of the experiment, it is probable that their threshold to classical blackout or unconsciousness would be similar to that of Group I.

CONCLUSIONS.

Raising the body temperature to levels which occur in modern aircraft under tropical conditions and during high speed flight diminishes man's ability to withstand other circulatory stress. In hot conditions, orthostatic hypotension is more

common and any increase of gravitational force may be associated with collapse. Similarly heat lowers man's tolerance to positive acceleration and may precipitate unconsciousness at very low g levels. Thus summation of the effects of heat and g do occur.

SUMMARY

The effect of heat on human tolerance to acceleration has been studied in the human centrifuge. Seven subjects were heated by means of electrically heated suits worn over polythene overalls until their body temperature was 100 - 102°F. Using the technique of central light loss with the dark-adapted eye, their thresholds were measured before and during the application of heat stress. Changes in pulse rates were also measured during the course of the experiments.

In all cases raising the body temperature resulted in a fall of the g level at which central light loss occurred. Two types of response were noted. Heat caused a fall of 15-30 per cent in threshold in one group and of 5-10 per cent in the

other. It is postulated that the small fall in the latter group is artefact and that in fact they also experienced a large fall in threshold. One subject of the latter group lost consciousness at 2.4 g with a mouth temperature of 100.7°F.

It has been shown that these two stresses (g and heat) summate in their effect and may produce unconsciousness at a low level of g.

CHAPTER III

The Effect of Breathing Pure Oxygen at Atmospheric Pressure on Tolerance to Acceleration.

It has already been pointed out (Part I, Chapter III; Part II, Chapter I) that the effects of positive acceleration are primarily due to a diminished cerebral blood flow (C.B.F.). It therefore seems not unlikely that substances lowering the cerebral blood flow will have an adverse effect on tolerance to acceleration. One such substance of prime importance in aviation is pure oxygen.

The respiratory response to pure oxygen has been described by Shephard (1955) and the cardiovascular effects by Whitehorn, Edelman and Hitchcock (1946) and more recently by Alveryd and Brody (1948). The action of excess of oxygen on the systemic blood vessels is twofold, a direct vasoconstrictive effect and a reflex tendency to vasodilation mediated by the chemo-receptors. This latter action is minimal in

the cerebral circulation where the effect of 100 per cent oxygen is vasoconstriction (Schmidt, 1950). It has recently been suggested (Lambertson, 1958) that this effect is not due to the oxygen itself but is the result of local tissue hypocapnia which arises due to abolition of the Bohr effect by oxygen. Nevertheless, the nett effect of breathing pure oxygen, whatever the mechanism, is a reduction in C.B.F. of some 10-13 per cent (Kety and Schmidt, 1948). This vasoconstriction occurs also in the retinal vessels (Taylor and Marburger, 1943; Bean, 1945), where a reduction in calibre of 10-38 per cent has been reported by Cusick, Benson and Boothby (1940). Thus even short of unconsciousness, blackout thresholds might be lowered when breathing pure oxygen. It has also recently been shown by Miles (1957) that the use of 100 per cent oxygen lowers the syncope threshold of normal individuals during passive tilting.

It is obvious that any mutual interaction of these two factors (g and 100 per cent O_2) leading to a lowering of the threshold of consciousness

would be of serious import in a high speed aircraft, especially at low level.

This chapter presents the results of an investigation into the effect of breathing pure oxygen at atmospheric pressure on the g threshold.

The experiments were carried out in two stages. The first stage consisted of estimating g tolerance by means of the dark adaptation method described in Part I, Chapter IX. The second stage arose from the results of the first and consisted of measuring tolerance by the levels of g at which classical blackout and unconsciousness developed. These two parts will be discussed separately in the order in which they presented.

STAGE I

Methods and Materials

The threshold was measured using central light loss by the dark adapted eye as the end-point (Part I, Chapter IX).

The 100 per cent oxygen was breathed through

a demand system using a Mk.17 demand regulator and a "P" mask with toggle harness. This mask is fitted with a harness which presses it firmly against the face and prevents slip under g. It also serves to ensure a fit so that the slight negative pressure developed in the mask at the beginning of inspiration does not allow air leak at the sides. The inspiratory valve of the mask was removed and a simple expiratory valve fitted to reduce the back pressure which tends to produce hyperventilation. The oxygen was supplied from a high pressure cylinder. Two demand regulators were fitted side by side, one connected to the oxygen supply, the other to the atmosphere. From the regulators the hoses ran to a three-way tap from whence a connection ran to the mask (Fig. 7). Thus, by selection, air or oxygen could be supplied to the subject, while the use of two regulators allowed an approximately equal resistance to breathing regardless of which gas was being used.

Six experienced centrifuge subjects were used for the experiment. All had considerable experience of the method of threshold determination and

repeated thresholds could be obtained with a very small scatter (Max. S.D. ± 0.15 g). The subjects were told they might breathe air, 10 per cent oxygen or 100 per cent oxygen, but were not told which mixtures were in use. The subject seated himself in the car, the mask was fitted comfortably to his face and the appropriate gas selected. In all cases air was used first followed by 100 per cent oxygen. Two control threshold values were determined at 10 min. and 15 min. while breathing air. Following the latter run, the tap was turned to 100 per cent oxygen and further threshold runs were made after 5, 10, 15 and 20 min. on pure oxygen.

As the subject's microphone is always alive and there is a loud click from the regulator at the beginning and end of inspiration, the breathing pattern could be easily heard. In addition records of respiration were obtained from a pressure tapping in the mask, differences in pressure across the mask being recorded as a low frequency wave form via a Statham pressure transducer (Fig. 7). Pulses were obtained from a

lead I electrocardiogram.

Results

The changes in blackout threshold while breathing pure oxygen are shown graphically in Fig. 8. It will be seen that there is an increase in threshold for the first ten minutes followed by a fall to normal values or, in one subject (N.L.A.), to a subnormal value (See Table 6).

TABLE 6

Blackout Thresholds breathing Air and breathing 100 per cent Oxygen at Atmospheric Pressure.

Subject	Control Thresholds-g		Thresholds while breathing 100% O_2 -g			
	(a)	(b)	5 min.	10 min.	15 min.	20 min.
F.L.	2.6	2.5	2.7	3.0	2.7	2.7
N.L.A.	3.3	3.3	3.3	3.4	3.1	2.7
A.C.	2.9	2.9	3.3	3.5	3.4	3.2
C.McE.	2.4	2.4	2.9	3.0	2.9	2.8
M.K.B.	2.3	2.3	2.5	2.7	2.7	2.5
P.G.	3.1	3.1	3.8	4.0	3.5	3.1
Means	2.8	2.75	3.1	3.3	3.05	2.8

Analysis of variance shows the difference between the control levels and the value at 10 min. to be significant ($P = .001$); and the difference between the values at 10 and 20 min. to be significant ($P = .001$). There is no significant difference between the control threshold and the threshold measured after 20 min. breathing oxygen.

From Table 7 it can be seen that there is no significant change in the pulse rate while breathing oxygen. The response of the heart rate to g similarly shows no significant difference when breathing oxygen as compared to breathing air.

With the apparatus used in these experiments the inhalation of oxygen at atmospheric pressure produces no significant difference in the respiratory rate (Table 8). There is no tendency of the subjects to hyperventilate as judged from the records and from listening to their breathing. The change in respiratory rate due to acceleration was measured. The results are shown in Table 9. There is a significant increase in respiratory rate under g. This increase was from 24-28 per cent of the resting value.

TABLE 7. PULSE RATE CHANGES DURING 100% OXYGEN ADMINISTRATION. THE PULSE RATES AT THE BEGINNING OF EACH THRESHOLD RUN ARE COMPARED WITH THE PULSE RATES AT PEAK g.

GAS MIXTURE	AIR				100% OXYGEN							
TIME (min)	10		15		5		10		15		20	
Subjects	P.R. Static /min.	P.R. at Peak g /min.	P.R. Static /min.	P.R. at Peak g / min.	P.R. Static /min.	P.R. at Peak g /min.	P.R. Static /min.	P.R. at Peak g /min.	P.R. Static /min.	P.R. at Peak g /min.	P.R. Static /min.	P.R. at Peak g /min.
N.L.A.	86	100	85	96	79	92	77	92	83	-	-	-
A.C.	80	96	86	105	81	110	82	110	80	102	82	104
C.McE.	80	89	80	88	65	88	68	84	82	87	72	80
J.C.G.	86	110	80	112	75	107	80	105	70	104	78	104
M.K.B.	82	92	81	89	82	87	72	91	80	89	77	91

TABLE 8

Effect of Pure Oxygen at Atmospheric Pressure on Respiratory Rate. The Values represent the Mean Rate measured over 30 sec. prior to the Threshold Runs

Subject	BREATHING RESPIRATORY RATE: Breaths/min.				
	Breathing Air	Breathing 100% Oxygen for			
		5 min.	10 min.	15 min.	20 min.
P.G.	16	19	17	18	21
N.L.A.	12	11	11	11	11
M.K.B.	16	16	17	15	14
J.C.G.	19	18	14	15	15

TABLE 9

Effect of Acceleration on Respiratory Rate. The Increase in Rate at Peak g is expressed as a Percentage of the Resting Static Value. Values are given for air and oxygen.

Subject	% INCREASE IN RESPIRATORY RATE			
	Breathing Air	Breathing O_2 for		
		10 min.	15 min.	20 min.
P.G.	25	22	26	20
N.L.A.	25	24	28	24
M.K.B.	25	23	24	20
J.C.G.	36	29	32	32

Since it seemed possible that false results might be obtained if dark adaptation were affected by 100 per cent O_2, two subjects breathed pure O_2 for 30 min., their absolute visual threshold being estimated every 5 min. The results showed no significant change in absolute visual threshold under the conditions of the experiment.

DISCUSSION

Although breathing pure oxygen at atmospheric pressure causes a reduction of C.B.F., it caused no fall in threshold under the conditions of the present experiment. A possible explanation is that, although C.B.F. is diminished the higher oxygen content of the blood allows a normal cerebral oxygen utilisation. If this be the case, then it might be anticipated that no change in threshold would occur. However, there was a significant rise in threshold for the first ten minutes of the experiment followed by a fall to pre-oxygen levels at twenty minutes. There is a large variation in tissue sensitivity to anoxia, the retina being one of the most sensitive tissues. Night vision is especially susceptible to hypoxia and a diminution can be shown at heights of 400 feet

when the barometric pressure has fallen by 100 mm.Hg. Since the present method depends on the ability to see a central spot with the dark adapted eye, the oxygen content of the arterial blood may well be critical. Let us assume that central light loss occurs at 3.0 g. At this level the arterial pressure is in the range of 60-90 mm.Hg. systolic and 30-50 mm.Hg. diastolic (Fig. 9) which allows an adequate retinal blood flow to support day vision. However, the sensitivity of the retina to anoxia is such that the reduced quantity of oxygen reaching the eye cannot support dark vision and so the central light disappears. When the subject breathes pure oxygen then, although the blood flow is the same (assuming no change in calibre of the vessels), the quantity of oxygen reaching the eye is increased and so vision is retained. At higher levels, when the blood flow is reduced still further, then vision is again lost when the quantity of oxygen reaching the eye is diminished to the previous level. If this concept is true, it provides an adequate explanation of the rise in threshold to a peak at 10 min., since equilibrium between the inspired oxygen and the arterial

oxygen takes 7-10 min. to be reached. The fall in threshold following the peak can be explained by the same principle. As vasoconstriction of the retinal vessels due to oxygen increases, then the blood flow is further reduced and blackout occurs at lower g levels until equilibrium is reached. On the basis of the above theory, there would be no further fall in threshold once vasoconstriction is complete. This was borne out in two subjects (P.G., F.L.) who continued breathing 100 per cent O_2 for a further 10 min. During this period there was no further change in threshold. There is no experimental proof for the above theory, apart from the fact that equilibrium between inspired 100 per cent O_2 and arterial blood is not attained for 7-10 min., and therefore maximal vasoconstriction only occurs after the tenth minute. If this theory is correct, then the blackout obtained at values from 1.5 g upwards using the various methods depending on dark adaptation is a result of the sensitivity of the retina to anoxia and central theories do not require to be invoked. Although this investigation has shed some light on the mechanism of the low thresholds measured in the

dark, it has not provided the desired information on tolerance. This was investigated further using unconsciousness as the end point (Stage II).

There was no significant change in the resting respiratory rate, although three of the four subjects showed a slight fall. This agrees with the finding of Poulton (1953) who found no change in mean respiratory rate when breathing pure oxygen at atmospheric pressure over a period of 8 min., although Dripps and Comroe (1947) found a slight fall in rate followed by a rise. There is, however, a change in respiratory rate under g. The increase of about 25 per cent in rate at 3-3.5 g agrees well with the findings of Lombard, Roth and Drury (1948), who noted an increase of 4 respirations per minute at 3 g and Von Diringshofen (1934), who noticed a slight increase during g. It does not agree with the large increases in rate reported by Gemelli (1936) and Gauer (1938). It should be noted that the rates of 40/min. and 30/min. quoted by these authors were obtained at 4.0 g and 4.7 g respectively and the rates of application were undoubtedly much faster than that in use in the present experiment.

Dripps and Comroe (1947) described a bradycardia during oxygen administration, the fall in pulse rate amounting to 3-4 per cent of the resting value. In this experiment no significant change in pulse rate was found, This was probably due to the fact that apprehension in the present series of subjects produced a slight tachycardia which obscured any slowing of the pulse due to the oxygen administration.

S T A G E II

METHODS AND MATERIALS

The two end points used for threshold determination in this stage were (a) classical blackout and (b) unconsciousness. The type of centrifuge run was the same as that used in the dark adaptation method. The centrifuge was accelerated linearly at 0.1 g/sec. to a peak of 8.0 g. If the subject had not lost consciousness when peak g was reached the run was continued at 8.0 g until the end point occurred. A bright central red light was fitted in the gondola on which the subject fixated. He was given a push button and

instructed to press it firmly when the central light disappeared and on no account was he to release the button until the termination of the run. In actual fact the run was continued until it was noted on the record that his finger had released the button, this was assumed to be the point where consciousness was lost. This end point was verified in each case by the medical observer in the centrifuge centre section who could see when the subject became unconscious.

The gas supply system was the same as that described in Stage I. An additional measure of respiration was obtained by fitting a differential capacitance manometer in the gas supply tube. This utilises the principle that gas flowing past a wire mesh produces a change in pressure from which the flow can be measured. From these two respiratory recordings the efforts of the subject to breathe could be compared with the actual flow of gas induced.

The other parameters recorded were three channels of E.C.G.s (standard lead I, aVF and V_1), two to four channels of E.E.G.s (left and right frontal, left and right occipital) and eye

movements. These latter measurements were made in the horizontal and vertical planes from electrodes placed as shown in Fig. 10. The results confirmed the subjects signal of black-out since, when vision is lost, fixation disappears and the eyes commence to move in a random fashion. A sample record is shown in Fig. 11.

The subjects used in this stage of the investigation were seven naval divers who had no experience of acceleration. On the morning of the experiment they were given two indoctrination runs to 3.5 and 4.5 g. In the afternoon they were instrumented, installed in the gondola and then carried out two runs, one on air and one on oxygen. The run on oxygen was carried out ten minutes after they started breathing the gas to allow time for equilibrium. They were allowed a period of ten minutes between the runs or longer if they felt unfit. They were not told which gas mixtures were in use.

Of these subjects several volunteered to allow arterial cannulation to be performed. In two subjects the radial artery was cannulated

and the arm supported so that the needle was at eye level. Only one record was obtained as one subject had a vasovagal attack and became extremely nauseated due to arterial pain, necessitating removal of the cannula.

In the other subjects the brachial artery was cannulated at heart level and the blood pressure recorded. In these subjects the degree of peripheral resistance was measured by the "run-off curve" method of Hayter and Sharpey-Schafer (1958).

RESULTS

The threshold results are shown diagrammatically in Figs. 12 and 13.

(a) From Fig. 12 it will be seen that breathing pure oxygen was associated with a fall in blackout threshold in four subjects, a slight rise in two and no change in subject number six. Subjects 1, 3, 4 and 7 breathed air for the first run, the other subjects started with oxygen. Thus the order in which the gases were breathed

does not affect the results. Statistical analysis of the results for classical blackout showed the difference in threshold breathing air and oxygen was not significant ($P = 0.2$).

(b) The results using unconsciousness as the end point (Fig. 13) show the same pattern. Four subjects show a fall in threshold while breathing oxygen, two show a rise and in one there is no difference. The subject distribution of the results is not the same as in the blackout runs. Statistical analysis of the figures shows that there is no significant difference ($P = 0.1$) between breathing air and breathing oxygen.

In the one subject with the radial artery cannulated, the blood pressure reached zero six seconds before unconsciousness occurred. In the subjects with the brachial artery cannulated, the blood pressure rose during the accelerative phase. This was because the artery lay slightly lower than the mean heart level. There was no significant difference between the blood pressure response breathing air and breathing oxygen.

Representative graphs are shown in Fig. 14.

Fig. 15 shows the mean pulse rate for all subjects and all runs compared with the pulse rate regression line obtained for this type of run as described in Part I, Chapter V. There is a marked difference in pulse rate response. In this series the subjects were inexperienced, apprehensive, and were straining continually as they knew they would be going to high g levels, whereas in all other runs carried out the subjects were experienced, relaxed and knew that the peak g would be less than 3.5 g. Thus the pulse rate response in this experiment is not entirely a reaction to g but a combination of anxiety and physical effort in addition. Comparison of the individual pulse rates shows that there is no significant difference breathing air or oxygeh.

The only gross cardiac arrhythmia which has occurred during g in this laboratory was noted during this experiment. This is shown in Fig. 16. Just before losing consciousness an isolated ventricular extrasystole was noted. This was rapidly followed by pulsus bigeminis which continued

for two minutes after the centrifuge had been stopped and consciousness regained. During this phase the normal beat was characterised by an absent P wave in all leads and inversion of the T_W in lead V_1. Artefact obscured any changes which may have been present in the ST segment. The extrasystoles were ventricular in origin. Cessation of the pulsus bigeminis was followed by a bradycardia of 48/min. The Q.R.S. and T complexes had reverted to normal but P waves were still absent. This ventricular rhythm continued for fifteen minutes before reverting to sinus rhythm of 76/min. The subject complained of palpitations and retrosternal ache and said he "felt ill and funny all over" but was unable to localise any other specific symptoms. He rested for 30 min. by which time he felt better. No further runs were attempted in this subject.

Analysis of the records for changes in peripheral resistance was carried out by Dr. P. Howard. Complete records were obtained in only

two subjects. The results that he obtained are shown graphically in Fig. 17. It will be seen that the onset of acceleration is accompanied by a sharp rise in peripheral resistance which continues to increase throughout the acceleration. Deceleration causes an immediate return to normal which is followed by a mild degree of vasodilation. There was no difference in response breathing air or breathing oxygen.

At high levels of g (above 6 g) the respiratory flow diminished in spite of a normal pressure differential being maintained across the mask. As the subjects approached the limits of tolerance, there was a period of apnoea. The flow record showed that no gas was reaching the mask and the mask pressure differential showed only slight oscillations about the base line. On deceleration the apnoea was followed by large gasping respirations characterised by high flows and large pressure differentials in the mask. Subjectively, there is difficulty in breathing above 5.0 g, which becomes worse with increasing g. This is a

mechanical effect and the level at which breathing becomes impossible depends on the muscular development of the subject. In this series of experiments apnoea developed at an average of 5-10 sec. before unconsciousness occurred.

DISCUSSION

Statistically there is no significant difference in the unconscious threshold when the subjects breathe oxygen as compared to air. The same finding applies when blackout is used as the end point. The level of significance is such ($P = 0.1, 0.2$) that a large series of subjects might have established the relationship of the effects of the two gases more clearly. Examination of the results (Figs. 12 and 13) shows an interesting point. In both series, the subjects whose tolerance was lower on oxygen showed a much larger fall in threshold than those subjects whose thresholds were lower on air. Although an overall statistical analysis swamps this difference in response, the possibility of individual susceptibility must be borne in mind.

The subjects who showed a large fall in threshold when breathing oxygen may be more sensitive to the toxic effects of this gas than their fellows to whom the gas breathed made little difference to the threshold. This view is supported by Miles (personal communication) who has found in tilt table experiments that a person who faints when breathing air always faints when breathing oxygen. A few individuals, however, faint when breathing oxygen but not when breathing air. This also points to individual susceptibility as a factor.

The cause of oxygen poisoning is still obscure. Thus there is no valid hypothesis which can be advanced to account for this possible variation in sensitivity to the toxic effects of oxygen. It has been well established that breathing pure oxygen at atmospheric pressure causes an increase in cerebral vascular resistance with a diminished blood flow (vide supra) but none of the authors have described any difference in subjective response. This fall in C.B.F. might

be expected to cause a fall in threshold in all subjects. That this did not occur must be attributed to the increased oxygen content of the blood ($\sim$ 2 vols. %) allowing a normal tissue oxygenation. It has been shown by Lambertson (1953) that the high arterial pO_2 results in a reduction of the buffering power of the blood so that the jugular venous pCO_2 rises and there is a fall in the cerebral arterio-venous pH difference. This difference is enhanced by a fall in arterial pCO_2. He suggests that these changes are protective and permit better gaseous interchange in the brain. It may be that in some subjects these adaptive responses do not occur and the toxic effects of oxygen are quickly apparent.

In conclusion it may be said that the results of this experiment do not firmly establish pure oxygen at atmospheric pressure as a factor in lowering tolerance to acceleration. However, there is the possibility that some individuals may be more susceptible to the toxic effects of oxygen than others, although the mechanism of this

can not be deduced from our present knowledge of the influence of high pressure oxygen on the tissues.

The finding that there was no difference in the pulse rate and blood pressure breathing air or oxygen agrees with results of Lamberton (1953). The response to g similarly showed no difference. Thus changes in threshold are not due to any general cardiovascular change. The cardiac arrhythmia which occurred in one subject was the only one which has been found during g in this laboratory. The tracing obtained resembled that occurring in a vasovagal syncope. It occurred when the blood pressure at head level was zero and could not be correlated with any blood pressure response. Vagal stimulation is extremely unlikely in an experiment of this sort as g is associated with increased sympathetic activity and an increased output of adrenaline by the adrenal medulla. Similarly the alternative explanation of myocardial ischaemia is difficult to accept as the changes in the E.C.G. consisted merely of an inverted T wave in lead V_1 which

could easily have been due to the sympathetic activity (Myerson and Davis, 1942) or to change in electrical axis. However, there is the possibility of an area of increased myocardial irritability acting as an ectopic focus, the irritability being due to a combination of anoxia and strong sympathetic stimulation. Further evidence of the latter is provided by the vasoconstriction which was found during g. This is shown in Fig. 17 expressed as peripheral resistance, as described by Hayter and Sharpey-Schafer (1958). These changes in peripheral resistance index were identical whether breathing air or oxygen. This is the first time peripheral vasoconstriction has been demonstrated during acceleration and has been described in detail elsewhere (Howard, 1958).

CHAPTER IV

The Effect of Hyperglycaemia on Tolerance to Acceleration

It has been noticed by most workers on acceleration that tolerance is higher following a meal. Blackout thresholds measured after lunch by Clark and Jorgensen (1945) showed an increase of 0-1.1 g. The mechanism of this protection has not been established. Two possibilities exist. Firstly the increase in blood sugar may increase tolerance by elevating the supply to the brain. Secondly distension of the stomach may increase tolerance by increasing intra-abdominal pressure and so maintaining a better venous return, or alternatively by splinting the diaphragm it may prevent descent of the heart. All these factors may play a part in the added protection afforded by a full stomach. This phase of the investigation is an attempt to examine the first of these hypotheses.

A further stimulus to this investigation was the fact that some cases of unconsciousness during positive acceleration appear to be related to factors other than cerebral anoxia. One of the suggested factors is an altered glucose content of the blood (Stewart, 1945). If the low cerebral blood flows

obtaining at unconscious levels do not allow an adequate supply of glucose for cerebral metabolism then unconsciousness might occur in the presence of a reasonable oxygen supply. If this hypothesis is true then raising the glucose content of the blood might be expected to raise the subject's tolerance.

Method

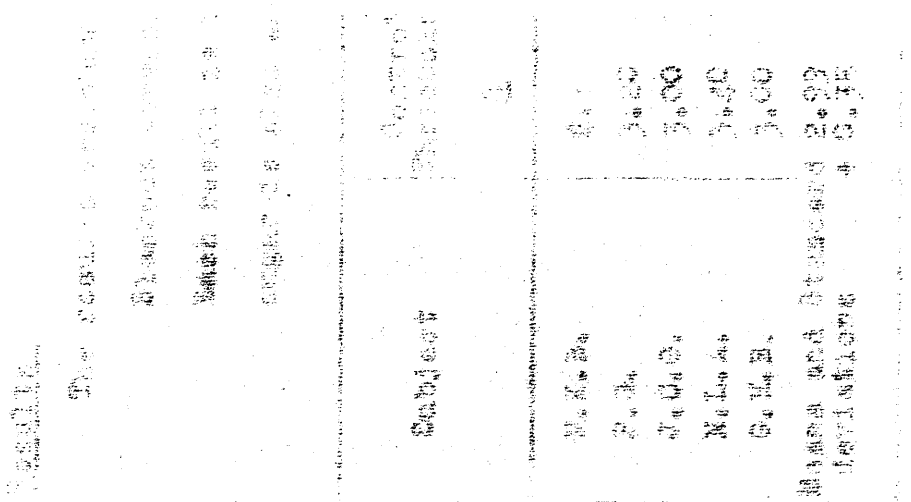
Five experienced centrifuge subjects took part, all of whom were used to the technique of threshold determination described in part I chapter and whose thresholds for repeated runs showed very little scatter (max. S.D.= ± 0.15g).

Each subject had his threshold determined twice, the mean of which was used as the control value. He then had a sample of blood taken for the estimation of blood sugar. Immediately after the control runs he was given 100g. of glucose in 250 ml. of water. The blackout threshold was again measured at 20 min. and 25 min. after ingestion of the sugar solution. Further samples of blood were collected at the beginning and end of the latter runs and the blood sugar estimated by the following method:-

1) 0.1 ml. blood was added to 3.5 ml. distilled water and deproteinised by the addition of 0.2 ml.

5% $ZnSO_4.7H_2O$ which were neutralised exactly to phenol-phthalein. The mixture was shaken vigorously and filtered.

2) 2.0 ml. of the filtrate, blank and standard (100 mg. glucose in 100 ml. H_2O: diluted 1:20 before use) were taken and to each was added 2.0 ml. of copper reagent (Somogyi 1952). These were heated in a boiling water bath for 10 mins. After cooling 2.0 mls. Nelson's reagent (Nelson 1944) was added to each. After ensuring that all the cuprous oxide had dissolved 4.0 mls. of water was added to each and readings taken at 500-520 mμ in a spectrophotometer.



Results

The results are shown in Table 10.

Blackout thresholds before and after the ingestion of 100g. glucose. Each result is the mean of two observations. The rise in blood sugar is also shown.

Subject	Control Threshold g	Control Blood Sugar mg%	Threshold after ingestion of glucose g	Change in Threshold g	% increase in blood sugar
M.K.B.	2.35	116	2.45	+ 0.10	41.4
P.G.	3.20	94	3.85	+ 0.65	24.5
J.C.G.	3.00	90	3.15	+ 0.15	98.0
N.L.A.	3.40	126	3.35	- 0.05	19.1
G.H.B.	3.00	91	3.20	+ 0.20	64.8
Means and Standard deviations	2.99 ± 0.35	103	3.20 ± 0.45	+ 0.21	49.6

From the above results it will be seen that there is a slight degree of protection afforded by a raised blood sugar. This mean increase of + 0.21g is not statistically significant (P=0.2).

Discussion

There is a large subject variation in the response to ingestion of sugar (Table 10). This does not bear any relation to the percentage rise of blood sugar or to the control blood sugar level. The mean increase in blackout threshold found (0.21g) is less than that found by Clark and Jorgenson (1945a) who noted a mean increase of 0.3 to 0.4g after a heavy meal or ingestion of 2 litres of milk. This may be due to the fact that these authors did not attempt to differentiate between a pure hyperglycaemic effect and one due to mechanical distension of the stomach. In a further paper Clark et al. (1945b) induced hyperglycaemia by feeding subjects glucose (2g/Kg. body weight) and found an increase in threshold of 0.20g which agrees closely with the figure found in this investigation. On the basis of this finding these authors suggest that there is a possibility that extracellular sugar may, by acting as a substrate in anaerobic glycolysis,

alleviate somewhat the brief brain anaemia induced by the hydrostatic fall in the arterial blood pressure at eye level. It should also be noted that their results lie within the experimental error of their method. From their results and those results obtained in the present study, there does not appear to be sufficient evidence to warrant such a broad interpretation, especially since the results are not statistically significant.

In conclusion one is only justified in saying that hyperglycaemia may slightly increase tolerance to acceleration.

SUMMARY

The influence of a raised blood sugar on g tolerance has been studied. Hyperglycaemia was produced by the ingestion of 100 g. glucose in 250 ml. water, which raised the blood sugar level by about 50 per cent.

There is a large subject variation in response (from -0.05 g to +0.65 g), the mean being an increase of 0.2 g in blackout threshold, which is not statistically significant. It is therefore unlikely that the known g protection afforded by a meal is due to an increase in blood sugar level.

CHAPTER V

The Effect of Insulin Hypoglycaemia on Tolerance to Acceleration.

In certain cases a reduced glucose content of the blood may lead to unconsciousness during positive accelerations (Stewart, 1945), possibly by lowering the substrate supply to the brain. During insulin hypoglycaemia, there is little change in the cerebral blood flow (no change: Kety, Woodford, Freyhan Appel and Schmidt, 1948; slight increase: Ferris, Rosenbaum, Aring, Ryder and Hawkins, 1941; slight decrease: Himwich, Bowman, Daly and Fazekas, 1941), although there is a fall in cerebral oxygen utilisation and an even larger fall in the sugar utilisation (Kety et al, 1948). Thus at rest hypoglycaemia appears to be related to cerebral anoxia in spite of a minimal change in the cerebral blood flow. Since unconsciousness occurring during positive acceleration is usually finally due to cerebral anoxia, then on these grounds also hypoglycaemia might be expected to precipitate collapse.

It is common knowledge that a feeling of faintness is commonly associated with hunger.

This lowering of the "syncope threshold" has been placed on a firmer basis by Miles (1957) who found that naval divers were more prone to collapse when fasting. In an analysis of the operative factors in 19 cases of unconsciousness in divers, Miles (1957) found hypoglycaemia to be present in 21%. This high incidence is unlikely to be due to chance alone. In aviators loss of consciousness is more difficult to study since it usually leads to a fatal outcome. However, Powell and his co-workers (1956-57) have studied survivors of unconscious episodes in flight in the R.C.A.F. and analysed the operative factors (these are summarised in Table 11). It will be seen that hypoglycaemia is once more a fairly common occurrence (present in 55.5%). In the R.A.F. there is no official method for reporting such occurrences and the incidence is therefore difficult to assess. Some indication may, however, be obtained from fatal crashes A typical example of a fatal incident is described below.

Case History.

M.J.L. a naval pilot aged 25 with a total of 224 flying hours was flying solo in a fighter aircraft on which he had 97 hours experience. Twenty

TABLE 11. ANALYSIS OF FACTORS OPERATING IN CASES OF LOSS OF CONSCIOUSNESS IN FLIGHT (RCAF)+

CASE	DUAL	SOLO	AIRCRAFT JET	AIRCRAFT PISTON	AGE	HYPERVENTILATION	HYPOGLYCAEMIA	HEAT	ANOXIA	g FORCES	PSYCHOLOGICAL STRESS
1		-	-	-	19	-	-	-	+	-	-
2		-		-	20	-	-	-	-	-	-
3	-			-	22	?	-	-	-	+	-
4	-			-	27	+	+	-	-	-	-
		-		-	27	+	+	-	-	-	-
5	-	-		-	21	+	-	-	-	-	-
6	-		-		18	-	?	-	-	+	+
7	-		-		20	?	+	-	-	+	+
8		-		-	25	?	?	-	-	+	+
9		-		-	30	-	-	-	+	-	-
10		-		-	33	+	+	+	-	-	+
11		-	-		20	-	+	-	-	-	+
12	-			-	29	+	+	-	-	+	+
13	-			-	23	?	+	-	-	+	+
14	-			-	22	±	+	-	-	-	-
15		-		-	20	?	-	-	-	-	+
16		-		-	20	-	-	-	-	-	-
17	-			-	35	+	+	-	-	-	+
-	-			-	35	+	+	-	-	-	+

+ Data derived from Powell et al (1957); Powell (1956).

minutes after take off, while flying number 3 in a tail chase at 10-12,000 ft., he entered a loop. Halfway through this manoeovre his aircraft fell out of control and dived towards the ground. He made no attempt to regain control of the aircraft nor did he make any radio call. Just before the aircraft hit the ground the canopy was jettisoned. The aircraft disintegrated on striking the ground and the pilot was killed. Examination of the wreckage revealed that no attempt had been made to use the ejector seat. From the description of the incident it would appear that the pilot lost consciousness halfway round the loop. From the fact that the canepy was jettisoned just before striking the ground it can be assumed that he regained consciousness just before the crash. In the loop he probably was pulling .3 to 4g which is well below the threshold of unconsciousness for a fit man, and his threshold must have been normal or he would have been removed from flying training. The operating height of 10,000 - 12,000 ft rules out anoxia although in a tail chase it is quite possible that he was overbreathing to some extent. The only other factor which is known is that he had missed breakfast and had been without

food for some 14 hours. Thus the only reasons contributing to this assumed loss of consciousness appear to be a moderate degree of g and a fasting blood sugar with the added possibility of hyperventilation. This type of case is by no means uncommon in many air forces and although the causation can not be established with certainty hypoglycaemia occurs too frequently to be neglected.

The investigation of any synergism which might exist between hypoglycaemia and positive acceleration forms the basis of this Chapter.

Methods and Material

Six experienced centrifuge subjects were used for this investigation, all of whom had considerable experience in the method of threshold determination (Part I Chapter IX) which was carried out in the dark.

Two centrifuge runs were carried out to establish a base line threshold in each subject and a sample of capillary blood was collected for the estimation of blood sugar. At this stage 2.0 ml. of soluble insulin in normal saline was given intravenously in a dose of 0.15 units/kg. body weight.

Twenty minutes later the subject recommenced dark adaptation and two runs were made between 25 and 35 minutes following administration of the insulin. Samples of capillary blood were collected at the beginning and end of these two runs and their sugar content estimated. Subjective sensations were noted, especially any visual signs which occurred. Following the third blood sample 100 grammes of glucose in 500ml. of orange juice was given orally. Fifteen minutes after this two further runs were made and a final sample of blood collected. The glucose content of the blood samples was estimated by the method described in Chapter III.

It was anticipated that secretion of adrenalin due to a fall in blood sugar might produce artefacts. An attempt was made to estimate any increased production of this substance during the experimental period. It has been suggested that the oxidation product of this hormone is excreted in the urine as 3 - methoxy-4-hydroxy-mandelic acid (Shaw et al.1956, Armstrong & McMillan 1957). Urine was collected for 1½ hours before the experiment as a control, and half an hour after completion of the runs the experimental sample was collected which

represented an equivalent period of time. The urines were extracted with ethyl acetate at pH 3 and hydrolysed by boiling for 30 min. with enough conc. HCl to give a 15 per cent solution. The hydrolysate was then examined by two dimensional chromatography using the method of Armstrong et al.(1956).

Pulse rates were obtained from a lead I electrocardiogram, the wrist electrodes being replaced by disc electrodes over the acromion processes to obviate muscle artefact (Browne and Fitzsimons, 1959).

RESULTS

The effect of insulin administration on the threshold and on the blood sugar is shown graphically in Fig. 18. The injection of insulin causes a fall in blood sugar (Table 12) which reached 50 per cent of the control threshold after 30 min. 50 min. after the injection and 15 min. after the ingestion of 100 g. glucose, the blood sugar had returned to control levels. From Fig. 18 it will be seen that insulin causes a fall in threshold 30 - 35 min. after injection.

Table 12

The effect of insulin on the blood sugar

No. of Subjects	Control blood Sugar mg/100ml. range.	Mean blood sugar 30 min. after insulin. as percentage of control level ± S.E.	Mean blood sugar 50 min. after insulin as % of control level ± S.E.
6	71-131	49.2 ± 5.4	99.9 ± 11.0

This is followed by a rise in threshold to above the control level at 50 min. i.e., 15 min after the oral administration of 100 g. of glucose. The individual values are shown in Tables 13.

TABLE 13

The Changes in Blackout Threshold occurring following Administration of Insulin intravenously (0.15 units/Kg. body weight)

Subject Member	Control Threshold g	Threshold 30 min. after Insulin g	Threshold 50 min. after Insulin g
G.H.B.	3.1	2.9	3.6
N.L.A.	2.9	2.1	3.7
J.C.G.	3.0	2.6	3.5
C.McE.	2.7	2.3	2.9
M.K.B.	2.5	2.1	3.3
P.G.	2.9	1.7	3.3
Means and Standard Deviation	2.85 ± 0.21	2.28 ± 0.42	3.38 ± 0.35

Analysis of variance shows both the fall and the rise in threshold to be significantly different from the control value ($P = 0.001$).

Since the administration of 100g. of glucose has an insignificant effect on the g tolerance (Browne, 1958), it seemed unlikely that the rise in threshold was entirely due to the sugar, unless the hypoglycaemia due to insulin potentiates the effect of the oral glucose. This latter hypothesis is unlikely, since the final blood sugar was slightly lower than the control value. It seemed more likely that this secondary rise was due to a late effect of the insulin. To test this possibility one subject carried out a further series of runs omitting the ingestion of sugar. The results of these runs are shown in Fig. 2, which shows the threshold 30 min. after insulin fell by 0.4 g, while at 50 min. it rose 0.8 g above the control level.

Intravenous injection of insulin produces a series of symptoms known as the reaction. The symptoms which occur are as follows in decreasing order of ffequency: sweating, warmth, flushing, drowsiness, finger tremor, light-headedness, palpitations, weakness and hunger (French and Kilpatrick, 1955). The incidence of these symptoms in the present series of subjects is shown in Table 14.

TABLE 14

Incidence of Symptoms following the I.V. Injection of Insulin (0.15 units/Kg. body weight) in Six Normal Subjects

Symptoms	SUBJECTS						Number of Subjects with Symptoms
	C.McE.	J.C.G.	G.H.B.	N.L.A.	P.G.	M.K.B.	
Sweating	+	+	+	++	++	++	6
Warmth	-	-	+	++	-	-	2
Drowsiness	-	+	±	+	-	-	3
Tremor	++	-	-	-	++	++	3
Lightheaded-ness	+	+	+	+	-	+	5
Palpitations	-	±	-	-	-	-	1
Weakness	-	-	+	+	+	-	3
Hunger	+	-	++	-	++	++	4
Cold	++	+	-		+	++	4

A symptom which is not generally reported but which was particularly noticeable in this series was the loquaciousness of the subjects. Considerable difficulty was experienced in persuading normally taciturn subjects to remain silent long enough to carry out the runs.

Some degree of reaction occurred in all subjects, the time of onset varying from 25-40 min., after injection of insulin. The duration of the reaction is difficult to assess as the symptoms gradually disappear. However, in two of the subjects there was some degree of upset still present after the completion of the experiment;

N.L.A. a competant electronic engineer, returned to his workshop and soldered together two live main leads. This incident occurred 60-80 minutes after injection of insulin.

M.K.B. still had some finger tremor and dysphasia ten minutes after completion of the experiment.

Pulse rates were obtained throughout the entire experiment in only two subjects. These results are shown in fig. 20. It will be seen that a tachycardia develops between 30 and 35 min. after

the injection of insulin which returns to normal after 45 min.

Analysis of the urines for homovanillic acid and 3-methoxy-4-hydroxymandelic acid showed no significant difference between control and experimental samples on visual examination of the chromatograms.

DISCUSSION

The use of a small dose of insulin to produce hypoglycaemia in this experiment is not entirely an artificial procedure. The occurrence of hypoglycaemia in everyday life is probably not as uncommon as was once thought. It may occur as a slightly lowered value in the fasting state but can also come about due to a variety of causes which are loosely labelled as "endogenous hypoglycaemia." This condition has been ably reviewed by Garland (1958), who divides the aetiological factors into organic, post-gastrectomy and psychosomatic. The middle variety is of no concern in the present context. Organic hypoglycaemia covers a wide variety of conditions varying from a frank islet cell tumour of the pancreas to a diffuse hypertrophy of the whole pancreas.

Often, however, there is no clear-cut pathology and we have the condition of functional hyperinsulinism. In many otherwise normal people the taking of a high carbohydrate meal produces symptoms of hypoglycaemia several hours later. This may again be due to hyperinsulinism giving rise to a rebound hypoglycaemia, and shows a fallacy of the usual three-hour glucose tolerance curve which may be within normal limits in these cases, although, as has been pointed out by Garland (1958), there is often no close relation between the blood sugar and the occurrence of symptoms. Indeed classical symptoms of hypoglycaemia may occur with tremor, sweating, confusion and a tendency to faint, when the venous blood sugar is over 100 milligrams per cent, and the attack be relieved by the administration of glucose (Franks, 1956). It is known that the rate of absorption of sugar is a factor influencing insulin production, as is the quantity of carbohydrate in the diet. Thus in a pure carbohydrate meal both factors are present and there is an increased risk of hyperinsulinism. Similarly it seems possible that an effect of positive acceleration would be to increase the rate of emptying of the stomach and so promote rapid absorption

of sugar, and an increased output of insulin.

The other variety of hypoglycaemia which may occur is the psychosomatic type. Due to mental upset or to a frank psychoneurosis there is an instability of the homeostasis of blood sugar and bouts of hypoglycaemia may occur. This is particularly likely to occur in the stress and strain of modern high speed flight and must be considered as a causative factor in the production of inflight hypoglycaemia.

The tragedy of these endogenous hypoglycaemias is that they occur in normal people who are quite capable of passing medical boards with the highest medical category. It is reasonable to suppose that a large number of pilots will belong to one or other group and who have so far escaped incidents by the lack of suitable circumstances. The only test which may give any evidence of this upset is the six hour glucose tolerance test, which is not a routine investigation unless the condition is suspected, and in pilots this may well be too late. However, apart from diagnosis the treatment is remarkably effective and consists of regular well balanced meals which have a reasonable proportion

of fat and protein to carbohydrate. If diet is adequate then few of these hypoglycaemic attacks will occur. It seems clear that if aircrew are suitably indoctrinated with regard to their eating habits, major incidents due to hypoglycaemia will be avoided.

However, there is still the possibility that small alterations in blood sugar while producing no symptoms on their own may influence tolerance to other stresses. It has been shown that mild hypoglycaemia increases neuromuscular tremor and lowers work output (Tuttle, Wilson & Daume 1949) so it might be anticipated that tolerance to increased gravitational force would be diminished. Such small changes in blood sugar could well occur due to psychologic upset. Although the hypoglycaemia in all these endogenous varieties might be due to anti-insulinases, pituitary hormones or glucagon they are more likely to be due to hyperinsulinism.

The general effects of insulin hypoglycaemia are well known and have frequently been described Briefly in the initial stages there are no signs of symptoms although blood samples show that the blood sugar is falling. Then at a variable time

(usually 30-40 min.) after the injection there is a dramatic onset of signs and symptoms which is called the reaction. The presenting symptoms vary but commonly include feeling of heat or cold, palpitations, hunger and drowsiness. Objectively there is tachycardia, increased irritability, often shown as muscle tremors, and increased pulse pressure. Sweating invariably occurs at some point and is often profuse. Following the onset of the reaction the blood sugar commences its return towards normal levels.

Thus, the effect of insulin hypoglycaemia on g threshold occur in two distinct phases; the initial stage when the blood sugar is falling and reaction has not occurred, and secondly during the reaction stage when the blood sugar, pulse pressure and pulse rate are rising. Since only six centrifuge runs can be carried out with a subject in any one day this experiment was planned on a fixed time basis, i.e., two control values were established, then two runs were carried out at about 30 min. and a further two runs about 40 min. after injection of the insulin. The times were arranged on the assumption that the minimal blood sugar level would occur at about 30 min. while the reaction would have occurred at 50 min.

In fact the reaction had already started in one subject (G.H.B) before the 30 min. runs were commenced while another subject (M.K.B.) noticed symptoms during the run at 35 min. after insulin. Thus a slight error may have been introduced in these results. In the initial stage of insulin hypoglycaemia (i.e. before the onset of the reaction) there is a fall in the g threshold of about 0.6 g. During this phase there is a fall in the mean blood sugar to about 50% of its control value. Three possible mechanisms for this fall in threshold suggest themselves. There may be a diminished cerebral blood flow due either to a fall in arterial blood pressure at head level or to an increased cerebrovascular resistance; or as has been mentioned before the supply of glucose may become insufficient to allow normal cerebral metabolism. There may also be a degree of cerebral anoxia due to an upset cerebral metabolism.

The cerebral blood flow changes but little during hypoglycaemia. In 1941 Himwich et al. found a slight decrease (7%) in cerebral blood flow during insulin coma while Ferris et al.

found no change or a slight increase. More recently Kety et al. (1948) using the nitrous oxide technique could find no significant change in cerebral blood flow during insulin hypoglycaemia. The blood sugar does not effect blood vessels directly and any change in cerebral blood flow would be expected to be passive following changes in arterial blood pressure at head level. There is much evidence to suggest that there is no change in blood pressure during the early stages of hypoglycaemia before the reaction occurs (French and Kilpatrick 1955, Allwood 1957). Thus a change in cerebral blood flow does not seem a likely cause of the fall in threshold.

During insulin hypoglycaemia there is a fall in cerebral arterio-venous oxygen difference. Since there is no increase in cerebral blood flow (vide supra) and since the blood sugar does not effect the oxygen carrying capacity of the blood it would appear that the oxygen uptake of the brain diminishes in hypoglycaemia with an associated fall in cerebral metabolism. Thus cerebral hypoxia might ba a possible explanation of the fall in threshold.

Associated with the diminished oxygen

uptake of the brain is an even greater fall in the glucose uptake. This agrees with the concept of a diminished cerebral metabolism. Thus during hypoglycaemia there is a steadily decreasing oxygen and glucose utilisation. The retina shares the changes in the brain and also presumably has a normal blood flow but a lowered metabolism. Visual changes have been described during hypoglycaemia but those due to alteration in the refraction of the eye (Duke-Elder 1925) and to structural changes (Vere and Verel 1955) are long term effects and have no hearing upon the present results. One subject (G.H.B) had some visual hallucinations (purple lights and flashes of red and orange) at the onset of the reaction which may have caused artefact in his 30 min. threshold.

In the present method of threshold determination the loss of central vision in the dark adapted eye is almost certainly due to the retinal anoxia which occurs with the reduction in blood flow. During hypoglycaemia there is probably no change in retinal blood flow at rest. Since however, there is a fall in metabolism, central vision will be lost with a smaller decrement of flow

than in the normal. Whether the prime factor in this fall is the low sugar level or the hypoxia it induces cannot be determined from the present experiment.

The second set of experimental values was obtained during the period of reaction. The symptoms which occurred and their frequency is shown in Table 14. The frequency of the symptoms is different to that found by French and Kilpatrick (1955). It is interesting that two-thirds of the subjects complained mainly of cold and hunger. It is felt that the cold was probably due to the low ambient temperature of the gondola, associated with the subjects sweating. A symptom which is not generally described is the increased talkativeness of the subjects. This finding overshadowed all the other signs and symptoms and was commented upon by various non-medical personnal in the vicinity, in one instance it was reminiscent of the speeches made by manic-depressives in hypomania. The second set of experimental observations were made about 50 min. after the injection of insulin and 10-25 min. after the onset of the reaction. They showed a mean increase

of 1.1g over pre-reaction thresholds and 0.5g over control values. A complication was introduced by giving the subject 100g. of glucose immediately after the pre-reaction thresholds were measured. This administration of sugar would tend to restore the threshold to control levels but would be unlikely to raise it above the control value.

There is evidence that induced hypoglycaemia causes a release of adrenaline from the adrenal medulla in animals and in man (Burn, Hutcheon, and Parker, 1950), Cannon, McIver and Bliss,1924, French and Kilpatrick, 1955, Holzbauer and Vogt 1954). Using biological assay, von Euler and Luft (1952) have demonstrated a tenfold increase in the excretion of adrenaline after insulin but no change in the excretion of noradrenaline. Since the adrenal medulla produces a variable quantity of noradrenaline on stimulation von Euler and Luft's results suggest there is also an inhibition of the adrenergic part of the sympathetic nervous system. By the techniques used here no change was found in the output of homovanilic acid or 3-methoxy-4-hydroxy-mandelic acid. This may have been due to the short collection preiod (1½ hrs) or to faulty technique

but can certainly be disregarded as the weight of evidence is in favour of adrenaline production.

This release of adrenaline is a homeostatic mechanism. It halts the fall in blood sugar and by causing glycogenolysis starts the blood sugar rising again towards normal levels. Adrenaline causes no constant effect on cerebral vascular resistance, but C.B.F. increases passively with the blood pressure. There is a rise in the systolic blood pressure with a fall in diastolic pressure which gives an increased pulse pressure with little change in the mean blood pressure. There is also a tachycardia which corresponds in its time relations to the change in blood pressure although French and Kilpatrick (1955) do not attribute it to adrenaline since it occurred in their sympathectomised patients in whom there was no increased production of adrenaline in resonse to hypoglycaemia. The circulatory effects of adrenaline production can explain the rise in threshold during the reaction period.

Since the level at which the central red light is lost depends on the retinal blood flow (which largely follows arterial blood pressure at head level), it is of interest to examine the blood pressure changes during the reaction phase. In the

course of this experiment attempts were made to cannulate the radial artery for blood pressure measurements. Unfortunately the system always blocked before the reaction phase was reached and blood pressures were obtained in only one subject (M.K.B). In this subject the control blood pressure was 115 mm.Hg. systolic and 83 mm.Hg. diastolic. The value reached during the reaction (45 min) was 130 mm.Hg. systolic and 74 mm.Hg. diastolic. These results agree well with those of French and Kilpatrick (1955) who in 12 normal subjects found the mean change in blood pressure (± S.E.) during reaction to be +16±3.8 mm.Hg. systolic and - 14±1.4 mm. Hg. diastolic. With the type of acceleration time pattern used in these experiments the blood pressure response to g is almost linear (Part I Chapter IX) and these blood pressure changes can be related to acceleration. Thus an increase of 16 mm.Hg. in systolic blood pressure represents an increase of threshold of 0.6g. This calculated figure agrees well with the results of the experimental series. Thus the rise above control threshold during the reaction phase of insulin hypoglycaemia is probably due to adrenaline production. The return to control values is due to

an increase in the blood sugar which was accelerated in these experiments by the administration of 100 g. of glucose.

This would correspond to the immediate effect of glucose in restoring to normality a patient suffering from the nervous and circulatory manifestations of endogenous hypoglycaemia.

Conclusions.

From the evidence it is clear that hypoglycaemia induced by insulin lowers the individual's tolerance to acceleration. In this experiment the threshold in the pre-reaction phase of insulin hypoglycaemia fell to 80% of its normal value. If this figure is translated to the g limits found in aircraft, then the pilot who would lose consciousness normally between 6g and 7g may well succumb between 4.5g and 5.5g if he has a low blood sugar. Hypoglycaemia may arise in flight due to fasting, or to hyperinsulinism. The latter may be reactive following a light meal composed largely of carbohydrate or may be induced by psychological stimulation. It is therefore important that pilots should not miss any meals and should avoid 'making do' with snacks of coffee and buns. Similarly any aircrew member who complains of weakness, dizziness or sweating while flying in the late morning or afternoon should be strenuously investigated for endogenous hypoglycaemia.

During the reaction phase of insulin hypoglycaemia there is a rise in threshold. This is due to the endogenous liberation of adrenaline

which probably produces this rise in tolerance by mobilising the glycogen reserves and more important by its effect on the cardio-vascular system.

Summary.

1. The influence of insulin hypoglycaemia on tolerance to acceleration has been studied in six subjects. The end point was central light loss with the dark adapted eye.
2. Hypoglycaemia in the pre-reaction phase is associated with a mean fall in threshold of 0.6g.
3. During the reaction the threshold rises above the control value. The mean rise is 0.5g above the control and 1.1g above the prereaction threshold.
4. The possible mechanism of these changes is discussed in relation to spontaneously occurring hypoglycaemia.

CHAPTER VI

The effect of the degree of filling of the stomach on tolerance to acceleration

It has been noticed by most workers in acceleration that tolerance is higher following a meal. Blackout thresholds measured after lunch by Clark and Jorgenson (1945) showed an increase in threshold of 0-1.1 g. Two possibilities exist. The first, that the increase may be due to raised blood sugar, is untenable (Chapter IV) since there is only a slight tendency to increase in threshold with hyperglycaemia, the change not being statistically significant. The second possibility is that the rise is due to the mechanical effects of distension of the stomach. This may increase tolerance by increasing intra-abdominal pressure, so facilitating venous return, or alternatively by 'splinting' the diaphragm it may prevent descent of the heart. This chapter presents the results of an investigation to test these hypotheses.

A further stimulus to this investigation was the fact that in many fatal aircraft accidents one of the findings is that the pilot has missed a meal. In the Indian Air Force many fatal accidents occur in the morning and a missed breakfast is a relatively

common finding (Ajit Nath - personal communication). Similarly Lawton (1957) found that improvement of the eating habits of the students in the Flying Training Air Force (U.S.A.) coincided with an improvement in the accident rate. Whilst all these cases might well be due to hypoglycaemia or to hyperinsulinism, as described in chapter IV, the additional mechanical factor of an empty stomach must also be considered.

Methods and Materials

The experiments were carried out in the centrifuge at this laboratory. Tolerance to acceleration was measured by the threshold at which central light loss occurred with the dark adapted eye (Part I Chapter IX).

Six experienced centrifuge subjects were used, all of whom were used to the technique of threshold measurement and whose thresholds for repeated runs showed very little scatter (max S.D. = $\pm$ 0.15 g).

Each subject was tested between 11 a.m. and 12.30p.m. having had a light breakfast of toast and tea at 8a.m. Subjects were assumed to have empty stomachs at the start of the experiment. Each had his threshold determined twice, the mean of the two

readings being used as the control value. He was then given a container holding 2.0 l. of tap water at a temperature of 20°C. and told to drink as much as he possibly could in 5 minutes. When he had finished he carried out two further threshold runs, the mean of which was used as the experimental value. The use of tap water to produce gastric distention was influenced by several factors. The rate of gastric emptying depends on the volume of the meal, the degree of distention of the stomach preceeding the meal, the ratio of the volume in the stomach to that which has flowed into the intestine, and the composition of the meal, (Hunt and McDonald 1954). The volume in this experiment was variable and in all cases the stomach was empty preceeding the meal so that the main variable was the composition of the meal. Many substances delay gastric emptying, for example, marked slowing occurs with sorbitol, sucrose and fat. However, since only the mechanical effects were to be determined, substances which alter blood sugar level were unacceptable. Similarly, high concentrations of sodium chloride or potassium chloride slow stomach emptying (Hunt 1956) but are unpalatable in large quantities. Therefore plain water seemed to be the most acceptable form of producing distention.. (No

subject was willing to swallow a balloon.) From the literature it would seem that in the first 15 min. 25%-30% of the amount ingested leaves the stomach (Smirk 1933 a&b, Hunt and Spurrell 1951), and in this experiment the first threshold determination was carried out within 30 sec. of the completion of drinking and the second run within 4 min. The degree of stomach emptying in this time could be neglected. Five of the subjects carried out the experiment a second time after a period of one week to check the validity of the results (the sixth subject was unavailable for this second series of runs).

Only three subjects were available for the recording of intra-abdominal pressures. Twelve records were obtained. These were recorded from a balloon attached to a thick walled rubber tube. The balloon was inserted rectally to a distance 6"-9" and the rubber tube connected to a strain gauge (Langham-Thompson). The system was primed by the injection of 10 ml. of air, and changes in pressure were recorded on an Ediswan ink-writing recorder after suitable amplification. Tests for artifact revealed only a rise of 2-3 mm.Hg. by compression of the rubber tube, and this was obviated by leading the

tube between the legs and over the front of the thigh.

X-ray facilities are not available in the human centrifuge and to examine changes in the position of the heart recourse was made to vectorcardiography. Vectorcardiography is largely empirical and has many theoretical limitations (Fulton 1951) it does give an immediate picture of the electrical position of the heart. There is no close correlation between the electrical axis of the heart and the anatomical axis but anatomical changes can be deduced from the vectorcardiographic changes by analogy. There are well marked changes in the vectorcardiogram with respiration. With inspiration there is a decreased amplitude of Q.R.S. in lead I and an increase in amplitude of Q.R.S. in leads aVF and V_1. Both frontal and sagittal V.C.G.s show a swing towards the vertical. There may also be changes in the shape of the loop and direction of rotation but these do not affect the overall change. Since inspiration is associated with descent of the diaphragm and a descent and elongation of the heart, a rough anatomical correlation can be drawn. Similarly in expiration the V.C.G. becomes more horizontal and often shows a move into the +X-Y quadrant due to the development of a q wave in lead aVF. We can deduce that raising

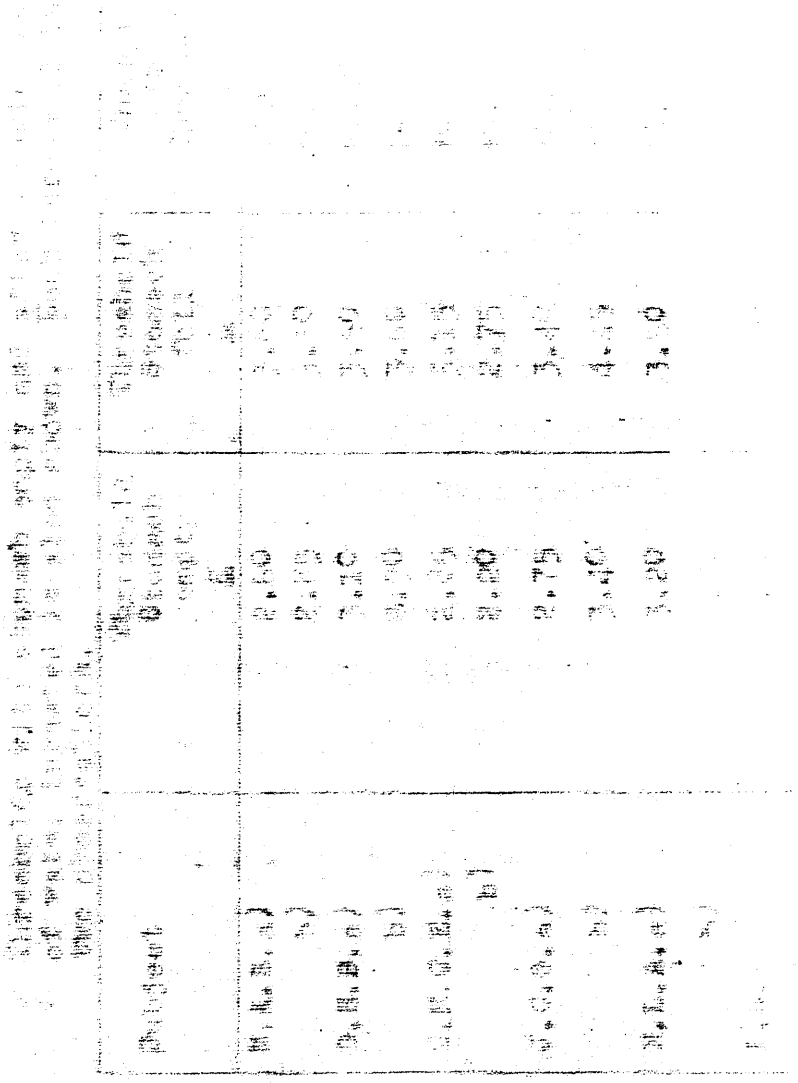
the diaphragm may play a part in these changes in the V.C.G., bearing in mind that changing autonomic balance may also be responsible (Simonson et al. 1946).

In this investigation it was decided to record the frontal and sagittal planes simultaneously. A Cossar split beam C.R.O. was used with aVF as the common lead, the connections are shown in Fig.20.

Since respiration produces changes in the V.C.G. it was important that the V.C.G. should always be recorded at the same phase of the respiratory cycle. The best time is mid-inspiration or mid-expiration which gives a mean position. In this series of experiments recordings were made at mid-expiration. A respiratory record was obtained from a thermocouple in the nostril. The most sharply defined part of the respiratory record is the peak of inspiration and this was used as the signal to record the V.C.G. When each peak appeared on the record a button was pushed which caused a complex to be photographed on 35 mm film approximately 0.5 sec. later. The details of the circuits involved are given in the appendix.

Several normal resting V.C.G.s are recorded with the stomach empty, and a record of full

inspiration and expiration was obtained for comparison. When the stomach was distended duplicate records were made. Traces were also photographed at peak g both with and without distention of the stomach.



Results.

a) Thresholds

TABLE 15.

Thresholds with stomach empty and after drinking water. The quantity of water ingested is also shown. Each threshold is the mean of two observations.

Subject	Threshold Stomach empty g	Threshold Stomach full g	Change in Threshold g	Quantity of water Ingested. ml.
M.K.B. a)	2.60	3.60	+1.0	1500
b)	2.85	3.10	+0.25	800
G.H.B. a)	3.10	3.50	+0.40	1250
b)	2.90	3.60	+0.70	1650
C.M.C.E. a)	2.85	3.15	+0.30	500
b)	2.20	2.75	+0.55	1400
J.C.G. a)	2.75	3.40	+0.65	1400
b)	3.40	4.45	+1.05	1350
N.L.A. a)	3.20	3.50	+0.65	1100
b)	3.80	4.45	+1.30	1500
P.G.	3.10	3.80	+0.70	1750

The results for subject P.G. are included in Table 15 but are excluded from the statistical analysis as he did not carry out the duplicate runs. It will be seen from Table 15 that after drinking water, all subjects showed a raised threshold. Analysis of variance shows this rise is significant at the 1% level, it also shows that occasion, i.e. whether (a) or (b), was not significant.

The amount of water a subject could consume varied with the occasion. An attempt was therefore made to detect any correlation between the quantity of water ingested and the rise in threshold produced. There is a significant linear relationship between these two variables, the formula of the regression line being

$$Y = -0.04 + 0.485x$$

where Y = the increase in g tolerance and x the quantity of water ingested in ml., the coefficient b = 0.085 (P=0.01). This line is shown in fig. 21. Stomach distension causes a rise in threshold of some 18.5%.

b) Intra-abdominal pressures.

Table 16. shows the changes in intra-abdominal pressure with g before and after drinking water. The initial pressure has no absolute significance

TABLE 16. CHANGES IN INTRA-ABDOMINAL PRESSURE WITH g BEFORE AND AFTER DRINKING WATER

	STOMACH EMPTY					STOMACH DISTENDED					
Subject	Initial Pressure mm.Hg.	Final Pressure mm.Hg.	Increase in Pressure mm.Hg.	Peak g	Rate of Increase of Pressure mm.Hg/g	Quantity of Water Ingested ml.	Initial Pressure mm.Hg.	Final Pressure mm.Hg.	Increase in Pressure mm.Hg.	Peak g	Rate of Increase of Pressure mm.Hg/g
M.K.B.	40.9 40.9 -	72.2 66.1 -	31.3 26.0 -	2.85 2.43 -	16.9 18.2 -	1500	49.2 49.6 50.0	93.1 103.5 113.1	44.0 53.5 63.1	3.48 3.66 4.00	17.8 20.1 21.0
J.C.G. (a)	47.9 47.9	99.2 84.8	57.3 36.9	3.95 3.48	17.4 14.9	1400	47.9 49.2	111.4 114.6	63.5 65.4	4.38 4.42	18.8 19.1
J.C.G. (b)	18.4 18.4	68.0 69.2	50.0 50.8	3.40 3.40	20.8 21.0	1350	23.2 23.2	84.0 79.2	60.8 56.0	3.4 3.4	25.3 23.3

and depends on the capacity of the system and the quantity of air used to prime it. The measurements are all relative to this arbitrary starting point. With the stomach empty there is a mean increase of 18.2 mm.Hg./g in intra-abdominal pressure. After drinking water there is an increase in the resting pressure varying from 8.7 mm.Hg. (M.K.B.) to 0.7 mm. Hg. (J.C.G.a). The rate of increase in pressure with g also shows an increase to 20.8 mm.Hg./g. This rate of rise of intra-abdominal pressure is fairly constant for any subject in any day but varies from day to day, presumably due to a difference in posture, and perhaps in the tone of the abdominal musculature.

c) The E.C.G. and vectorcardiogram.

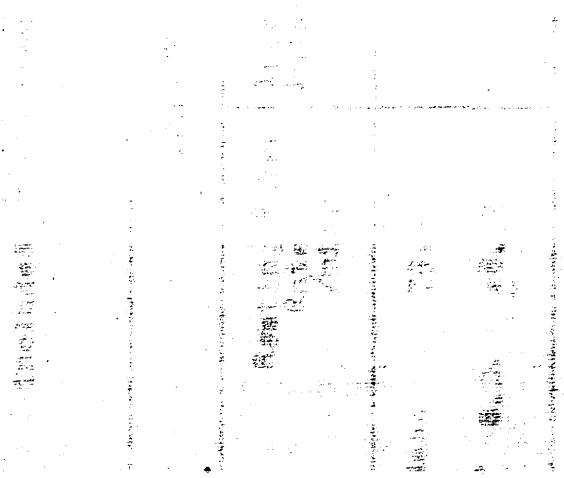


TABLE 17.

Changes in resting pulse rate and in rate of increase of pulse rate with g before and after drinking water. All subjects included. —(The results are the means of twenty observations)

	STOMACH EMPTY		STOMACH FULL	
	Resting Pulse Rate /min.	Increase in Pulse rate/g /min.	Resting Pulse Rate /min.	Increase in Pulse rate/g /min.
Mean	79.6	5.85	70.9	4.75
± S.D.	±8.23	±1.98	±5.96	±1.61

The effect of stomach distension on the pulse rate can be seen from Table 17. When the stomach is full there is a fall in the resting pulse rate and a decrease in the rate of increase of pulse rate with g. These changes are not statistically significant.

The effect of respiration on the E.C.G. is shown in fig. 22. Expiration, which is associated with elevation of the diaphragm, produces an increase in amplitude of Q.R.S. and T in lead I, a fall in amplitude of Q.R.S. and T in lead aVF, and an increased amplitude of Q.R.S. with a diminished T in lead V_1. On comparison with fig. 23 it will be seen that the effect of distending the stomach is to produce a change similar to that of deep expiration. This can be clearly seen from the vectorcardiograms. Fig. 24 shows sample records of full inspiration and expiration for two of the subjects. It may be seen that inspiration is association with a vertical type of V.C.G. with open loops while with full expiration there is a swing to the horizontal with narrowing of the loops and a tendency to crossing in the frontal plane V.C.G. The P. and T loops are not considered here. The changes in the V.C.G. taken at mid-expiration with the stomach empty and distended are

shown in fig. 25. It will be seen that in all cases there is a swing of the loop towards the horizontal with narrowing. Thus the effect of ingestion of water is to cause a change in the position of the heart, the change being similar to that produced by full expiration. This change may be due to elevation of the diaphragm.

This change due to distention of the stomach is maintained during g, when the loops show a move towards the inspiratory position with the stomach empty but are maintained at the expiratory position when the stomach is full.

Discussion.

Ruff (1938) found that tolerance to acceleration increased following a meal, and German investigators in general stated that fasting lowered g tolerance. However Ruff compared tolerance before and one and a half hours after a heavy meal and made no attempt to determine the cause of the change. Using unconsciousness as an end point he found a protection of 0.4 - 1.5g with his subjects tense and straining. His explanation depended on the fact that engorgement of the splanchnic area was accompanied by a generalised vasoconstriction, both of which would resist the pooling of blood under g. This explanation had no

experimental backing, and somatic vasoconstriction is not a normal consequence of digestion.

A more careful investigation was carried out by Clark and Jorgenson (1945) who use 1½-2 litres of water or milk to produce stomach distention and measured the threshold before and 10-20 min. after drinking. They found an average protection of 0.3-0.4g the range being 0-1.1g. This is somewhat lower than the results of the present experiment which showed a mean increase of 0.7g. with a range of 0.25-1.30g. This difference is surprising in view of the fact that similar quantities of water were ingested in both cases. A possible explanation may lie in the fact that their experimental runs were carried out 10-20 min. after beginning to drink, whereas in the present experiment the corresponding runs were carried out after 5½ min. In this experiment there may have been more stomach distension at the time of the runs than in the American experiment.

The changes in intra-abdominal pressure with g in the subjects with empty stomachs (18.2 mm.Hg./g.) agrees well with the findings of Clark and Jorgenson who obtained a mean increase of 18.5 mm.Hg./g., and Rushmer who found 25 mm.Hg./g. The increase in pressure

due to fluid ingestion is slightly greater (20.8 mm.Hg./g.) and increases linearly with g as in the empty stomach runs. At 3g there is an increase in pressure of 12-16 mm.Hg. due to ingestion of water, this is much higher than the change found by Clark and Jorgenson (7-9 mm.Hg.) which again may be attributable to the difference in time of measurement. These authors had found that by carrying out a straining manoeuvre which maintained the intra-abdominal pressure 40 mm.Hg. above normal levels a protection of 1.0g could be obtained. Using this figure as a basis, the increases of pressure found by them should have given a protection of 0.2g whereas, in fact, their mean protection was 0.3-0.4g. The difference between the experimental and calculated values was accepted as experimental error, and they deduced that the increased threshold was due to an increased intra-abdominal pressure, which, by producing a steeper abdomino-thoracic pressure gradient, maintained venous return to the right side of the heart. Using this figure of 40 mm.Hg. for 1.0g protection the increased intra-abdominal pressure found in the present study should give 0.4g protection. However, the actual protection was 0.7g. This discrepency is

of the same order as that of Clark, but under the conditions of the experiment is greater than can be attributed to experimental error, it is clear that the increase in intra-abdominal pressure cannot explain entirely the increased protection arising from stomach distension. A further reason for doubting this simple explanation is that trials of methods for pressurising the abdomen externally gave little protection (Stewart 1941). On theoretical grounds also there is reason to doubt this hypothesis since, although increased intra-abdominal pressure increases the abdomino-thoracic pressure gradient, it also impedes venous return from the lower extremities and increases pooling in the periphery. It would appear that the increase in threshold over the predicted value is probably due to some other mechanism.

Descent of the diaphragm and heart under g has been shown by Ruff et al. 1939 in monkeys. It has been confirmed by Rushmer (1944) for humans in whom the diaphragmatic descent may amount to 5-6 cm. at 5.0g. This is obviously a large factor in lowering g tolerance, since a difference in heart brain distance of 5-6 cm. at 5.0g necessitates an increased blood pressure of 25 mm.Hg. at heart level to maintain a blood supply

to the base of the brain. At 1.0g. a pressure of 25 mm.Hg. at heart level would be adequate to support a column of blood 30 cm. high which is the mean heart-brain distance. If descent of the heart could be prevented under g a protection of about 1.0g would theoretically result. If distension of the stomach acts as a cushion and supports the diaphragm preventing its descent, this would provide the explanation of the protection afforded by a full stomach. The position of the diaphragm can be inferred from the vectorcardiogram. As has been shown in figs. 23 & 25 , ingestion of water causes a change in the E.C.G. and V.C.G. towards the position seen in full expiration, which may be due to elevation of the diaphragm,. This does not agree with the findings of Simonson et al. (1946) who investigated changes in the E.C.G. following the ingestion of a meal of 200-269 g.weight. These authors appreciated the effect of respiration and recorded their E.C.G.s with respiration arrested. They reported no change in the Q.R.S. axis. However, they did not examine the E.C.G. till 30 min., after the meal and provide no information concerning the volume ingested. In addition, their recordings were restricted to the standard leads. It may well be that the volume of the stomach 30 min. after their test meal was inadequate to produce any changes, while the standard

bipolar leads do not give a good indication of the electrical axis. During g there is a swing of the frontal and sagittal vectorcardiograms towards the vertical. This implies an antero-posterior rotation to the right with a counter rotation to the left about the longitudinal axis, and is an exaggeration of the change seen on tilting a horizontal subject to the vertical (Mayerson & Davies,1942). This change which may be due to descent of the diaphragm, is similar to the changes seen in inspiration. When the stomach is distended this swing also shows a tendency to occur but the E.C.G. and vectorcardiogram remain in the position of full expiration. This suggests that the diaphragm remains high and its descent is being resisted. It seems likely therefore that distension of the stomach prevents the increase of heart-brain distance which occurs during positive acceleration. In this case a significant degree of protection would be anticipated.

Conclusions

There is definite advantage to be gained by flying with a full stomach. By missing a meal the aviator lowers his tolerance to accelerative forces and may show a fall of 20% in his blackout threshold.

The increased g protection afforded by a full

stomach may be due in part to the increased intra-abdominal pressure it produces. However, both experimentally and theoretically this is inadequate to explain the findings. In the author's view added protection is gained by the prevention of an increase in heart-brain distance. This is presumably due to the distended stomach resisting descent of the diaphragm during the application of positive acceleration.

Summary

1. Distension of the stomach by ingestion of water produces an increase in g tolerance of 0.7g, the range being 0.3-1.3g.

2. There is a significant linear relationship between the quantity of water ingested and the rise in blackout threshold.

3. Intra-abdominal pressure rises linearly with g, and ingestion of water raises this pressure and rate of rise. This increase is inadequate to explain the rise in threshold produced.

4. It is suggested that the rise in threshold is produced largely by the distended stomach supporting the diaphragm and preventing descent of the heart under g.

CHAPTER VII

The Effects of Ingestion of Ethyl Alcohol on Tolerance to Acceleration.

Aircrew should not consume alcohol in any form prior to flying. The general effects of alcohol intoxication are too well known to need description here: suffice it to say that the false confidence and errors of judgment which it provokes are not conducive to the safe handling of an aircraft. Since aircrew do partake of alcohol at varying periods of time before flying, it was felt that an objective examination of their ability to withstand flight stresses thereafter was required.

This experiment was planned to obtain information which would correspond to the lunchtime drinker who would fly in the course of the afternoon, or the pilot who had a few drinks in the evening prior to night flying. A dose of alcohol was given orally and the tolerance to acceleration measured at varying time intervals thereafter.

Methods and Materials

The method of administering alcohol should be as realistic as possible, since other factors such as aldehydes and higher esters might also influence the reaction of the subject. The most realistic and acceptable drinks were beer and whisky. Beer, however, introduces side effects by producing stomach distension and so whisky was used. The brand was the same throughout and was "Black and White", blended by Jas. Buchanan & Co., Glasgow. The quantity ingested was 114 ml. which has an ethyl alcohol content of 40 ml. A comparison of alcoholic beverages and their alcoholic content is given in table 18.

Table 18

The alcoholic content of several common drinks and the maximum levels of blood alcohol which they could produce (after Malmejac 1957).

Drink	Quantity ingested ml.	Pure alcohol content ml.	Before or After Meal.	Max. level of blood Alcohol mg/ml.
Gin	126	55	a.c.	1.01
Gin	126	55	p.c.	0.41
Whisky	122	55	a.c.	0.89
Whisky	122	55	p.c.	0.35
Beer	1.222	55	a.c.	0.44
Beer	1.222	55	p.c.	0.22

Absorption of alcohol from the empty stomach is rapid and blood levels reach a maximum in 30-60 min. The presence of food slows and prolongs the absorption. The subjects in this experiment partook of a light breakfast 2 hours before the experiment in order that the stomach should be empty and absorption rapid and predictable, since there were no facilities for the estimation of blood alcohol.

Six experienced subjects took part in this investigation.

The subjects' tolerance to acceleration was measured by means of the threshold technique utilizing central light loss with the dark adapted eye as the end point. Two hours after breakfast the subjects carried out two threshold runs, the mean of which was used as a control. Following the second run they were given 114 ml. whisky to drink. The whisky was drunk neat within a period of 15 min.

The threshold measurement was then repeated after one hour and after two hours. Each measurement was on two runs, the mean of which was used as the experimental value.

In addition a series of runs were carried out in which subjective impressions of visual changes were recorded. With bright ambient illumination the subjects were given a series of runs until a level was found at which visual changes were beginning to occur. These runs were of the plateau type, i.e., the centrifuge was brought to peak g at a rate of 1.0 g/sec., maintained at peak g for 15 sec , and then decelerated to rest at 1.0 g/sec. Two and a quarter hours after the ingestion of whisky a further run was made at this predetermined level and the symptoms compared.

Results

Part I.

The changes in threshold measured by the dark adaption technique are shown in Table 19.

Table 19

Changes in visual threshold during acceleration at various time intervals after the ingestion of 114 ml. of whisky.

Subject	Control Threshold g.	Threshold 1 hour after Whisky g.	Threshold 2 hours after Whisky g
P.G.	3.3	2.9	2.5
N.L.A.	3.2	3.4	3.6
M.K.B.	2.7	1.9	2.5
G.H.B.	3.0	2.9	2.8
J.C.G.	2.4	2.3	2.0
F.L.	2.4	2.3	2.7
Means	2.8	2.6	2.7

From table 19 it will be seen that there is no significant difference between the means. However, there is a large variation in control thresholds and since the effects of alcohol have a large individual variation it is more informative

to examine each case separately then in a group. Thus 5 out of 6 subjects show a fall in threshold as measured by this technique although these individual thresholds are of doubtful significance as the maximum standard deviation for any subject in a series of runs is ± 1.5 g. One subject (N.L.A.) shows a rise in both the experimental sets of runs. The other subjects show a varying time course, three (P.G., G.H.B.,S.C.G.,) show a fall at both one and two hours whereas M.KB. and F.L. show a fall at one hour followed by a rise after two hours. The maximum percentage fall in threshold measured is shown in Table 20.

TABLE 20

The maximum percentage fall in threshold measured within two hours of the ingestion of 114 ml, of whisky.

P.G.	24.3%
M.K.B.	29.6%
G.H.B.	6.7%
J.C.G.	16.7%
F.L.	4.2%

It must be remembered that the observations were made at hourly intervals and that the maximum threshold change may have occurred between runs. The absorption time of the alcohol would vary from subject to subject and the rate was not known as it was not possible to estimate blood alcohol.

None of the subjects showed signs of severe intoxication. It was noticed that most subjects showed less of control of fine movements and there was slight dysphasia in all cases. The most interesting observation arising from this first series of runs concerned the occurrence of disorientation. In the human centrifuge deceleration is accompanied by feeling of falling forwards flat on one's face. This is due to conflicting sensory information derived from the semicircular canals and is called "toppling". Although it is very noticeable and upsetting when it is first encountered, this sensation is soon suppressed and experienced subjects seldom notice it.

In this series of experiments all six subjects complained of the "toppling" and emphasised that it was the worst they had ever experienced. In two very experienced subjects (M.K.B., P.G.) who had not noticed this sensation in the previous two years it produced marked nausea and necessitated a period of rest before further runs could be carried out.

The results obtained from the other method of examination are shown in Table 21. When very experienced subjects are used, their visual observations have been found to be just as reliable as those obtained by more objective methods.

TABLE 21

Subjective Visual Impressions during 15 sec. Runs at a Level of g where Transient Greyout normally occurs. The Rate of Application of g is 1.0 g/sec. Results obtained before and after drinking Whisky are compared.

Subject	Peak g	Control g Alone	g two hrs. after drinking 114 ml. Whisky.
P.G.	3.6	n.v.s.	Severe GO
N.L.A.	3.6	veiling	BO
M.K.B.	3.6	n.v.s.	Severe GO
G.H.B.	3.4	mild GO	BO
J.C.G.	3.4	veiling	Severe GO
F.L.	3.6	veiling	BO

KEY: n.v.s. = no visual symptoms.
veiling= a general misting of visual fields.
GO = greyout - (a) mild - loss of peripheral vision only
(b) severe - only central vision retained.
BO = blackout.

By this method of determination all subjects showed a lowered tolerance to acceleration. This is manifested by a distinct worsening of the visual sequelae of a fixed amount of g.

Discussion

The variation in results using the dark adaptation method of threshold determination may be due to several factors. Firstly the absorption of alcohol may have been erratic and given rise to the varying time response of the subjects. A more likely cause however is the excitement produced by alcohol. The first effect of alcohol is a depression of the higher centres which by removing inhibitions causes restlessness and excitement. The altered cerebration in this series was manifest in the dysphasia of all the subjects and minor alterations in their behaviour. The method of threshold determination is extremely sensitive and requires relaxation on the part of the subject, and it seems likely that after consuming 114 ml. of whisky this condition did not obtain. It was for this reason that the experiment was repeated using a different method.

By determining a change in the visual symptoms at a fixed level of g. it was found that they became much worse after the consumption of whisky.

In three cases who had experienced mild visual symptoms on the control run, an identical run two and a quarter hours after drinking whisky led to blackout. The mean increment of g necessary to change greyout to blackout was found to be 0.6 g in a series of 1000 subjects by Cochran Gard and Norsworthy (1954) which corresponds to the figure found in this laboratory. It would seem therefore that a considerable fall in tolerance occurs following the absorption of ethyl alcohol.

The harmful effects of alcoholic beverages on the aviator has recently been reviewed by Malmejac (1957) who concludes that "alcohol' decreases the resistance to anoxia. It modifies 'the psychic balance and deteriorates psychosensory 'as well as psychomotor reactions". He has not, however, considered the effect on tolerance to acceleration. The influence of alcohol on the various systems of the body is described by Goodman and Gilman (1952).

Briefly it causes a depression of nervous function which begins at the highest centres and progresses downwards. Contrary to popular belief it does not at any time produce stimulation, but in the early stages of intoxication it removes the inhibition of the higher centres and so causes restlessness, excitement and looses the emotions from their normal control. There is an unappreciated loss of judgement, a sense of euphoria and a loss of the sense of fatigue. The effect on the cardio-vascular system varies, the most constant changes being peripheral vasodilation and a slight tachycardia.

The cause of the toxic effects of alcohol have not yet been elucidated. There are several theories. Smith (1930) suggested a duel action, the first phase being surface effect due to the higher concentration of alcohol outside the cell, the second phase was when equilibrium was reached and was possibly due to an interference with cellular exidation-reduction systems.

The fact that the action is to cause anoxia at cell level is universally agreed and Peters and Van Slyke (1931) classes alcohol as a cause of histotoxic anoxia. . This is further illustrated by the fact that anoxic anoxia due to high altitudes cause a similar set of signs and symptoms. Alcohol is not excreted by the body to any extent but is metabolised to acetic acid, with the intermediate step being the formation of acetaldehyde. It has been suggested that the acetaldehyde is the active agent, since intravenous injection of acetaldehyde gives rise to the same symptomatology as alcohol intoxication (Assmussen, Hald and Larsun 1948) This hypothesis is further strengthened by the fact that the ingestion of tetraethylthiuramd-isulphide (Antabuse) with alcohol increases the formation of acetaldehyde (Hald and Jacobson 1958), and so produces its profound effect. Whatever the biochemical site of action it seems clear that there is a degree of cerebral anoxia due to alcohol.

This depression of nervous function would probably account for the deterioration in threshold following alcohol. However, it is interesting to examine the haemodynamic changes also. It has already been pointed out (vide supra) that alcohol causes cutaneous vasodilation and thus pooling has to some extent commenced before g is applied. There is no change in the cerebral blood flow or cerebral vascular resistance following the ingestion of alcohol (Loman and Myerson 1942). In dogs Machne (1950) has shown that the carotid sinus response may be augmented by small doses of alcohol but abolished by larger doses - this has also been noticed by Liljestrand who suggested that alcohol possibly blocks the action of cholinesterase. It would appear that on a purely mechanical bases there might be a fall in threshold. Although alcohol per se does not diminish the cerebral circulation, the vasodilation it produces will allow pooling to occur which will prevent adequate cardiac compensation when g is applied.

In addition if the pressor reflex from the carotid sinus in man is inhibited there will be no stimulus to cardiac compensation during the application of acceleration.

Although Battey et al.(1953) found no change in cerebral metabolism following alcohol and Loman and Myerson (1942) found no change in glucose or oxygen uptake with levels of alcohol sufficient to cause facial flushing and mild mental changes, there is no doubt that alcohol causes cerebral anoxia. Since the retina is extremely sensitive to anoxia it seems reasonable to suppose that depression of the retinal cells might be additional factor although the fall may also be wholly or partly due to haemodynamic causes.

The other interesting finding in this experiment was the gross degree of disorientation which occurred at the end of the runs. It would seem as if an effect of alcohol is to eliminate the adaptation which has occurred to the combined angular acceleration.

This might be due to depression of the higher centres removing their inhibiting influence from vestibular sensation. Alternately it may be a result of head movement during the acceleration phase adding a further angular acceleration. This sensation was so disagreeable and nauseating that if it occurred in flight the pilot would have extreme difficulty in controlling his aircraft.

Conclusion

The ingestion of alcohol prior to flying is likely to diminish the pilots ability to control his aircraft. Quite apart from the false confidence and errors of judgement which it induces, there is a diminished tolerance to positive acceleration and to disorientation. It is not possible to say how long these effects last, They were present at the termination of the present experiments. i.e., two and a quarter hours after the ingestion of alcohol.

Summary

Six subjects drank 114 ml. of whisky and had their thresholds measured at varying time intervals thereafter.

Thresholds measured at hourly intervals by the dark adaptation method were extremely variable and showed no significant change. This was possibly due to the method being too sensitive for the state of the subjects.

Using plateau runs at a peak g of 3.4 - 3.6 g the ingestion of whisky was associated with a worsening of the visual sequelae after two and a quarter hours. This represented a fall in tolerance which may amount to 0.6 g.

Disorientation was aggrevated by the ingestion of alcohol.

These effects were found during two and a quarter hours after drinking whisky.

CHAPTER VIII

The effect of hyperventilation on tolerance to acceleration.

Apart from positive acceleration the commonest in-flight stress which plagues the aviator today is hyperventilation. In the normal subject overbreathing occurs with emotional stress, excitement and anxiety. All of these are present to a greater or lesser extent in every flight, their degree being related to the performance of the aircraft.

During the war hyperventilation was described by Goldie (1943) in bomber pilots on the outward flight to the target, the breathing patterns returning to normal on the flight home. This potential hazard was also appreciated by American aviation workers. Rushmer, Boothby and Hinshaw (1941) pointed out the dangers in aviation and emphasized the slowing of co-ordination and cerebration which occurred with even mild overbreathing. In 1943 Hinshaw, Rushner and Boothby pointed out that mild hyperventilation over a long period of time produced effects which were just as profound as in an acute severe bout. In spite of the recognition of the risks little positive action was taken in bringing it to the notice of the pilots themselves until 1955 when Brown described several incidents

in a magazine circulated to aircrews. The most detailed investigation was that of Balke and his co-workers (1957) who obtained alveolar samples from pilots of jet aircraft during routine training flights. They found evidence of hyperventilation in 78% of samples and that hyperventilation became more frequent with increased high performance capabilities of the aircraft flown. In the F.86 aircraft (jet fighter trainer) 63% of samples showed hyperventilation, the alveolar pCO_2 levels being 25-30 mm.Hg. (41%), 20-25 mm.Hg. (14%) and below 20 mm.Hg. (8%). Similar results were reported by Ellis et al. (1957) to the American Aeromedical Conference. It is clear from these results that hyperventilation is common in flight,.

Hyperventilation produces a respiratory alkalosis of which the most dramatic sign is the development of tetany. More important in the present context however is the effect on the cerebral blood flow, which may be reduced by as much as 50% when the arterial pCO_2 falls by 6 mm.Hg. It seems reasonable that hyperventilation will accentuate the effects of any other stress lowering cerebral blood flow. Hyperventilation also has the ability to provoke an epileptic seizure in susceptible patients,

this effect is potentiated by a low blood sugar and forms the basis of a diagnostic test, this will be discussed further in chapter IX.

This chapter presents the results of an investigation to examine the effect of hyperventilation on tolerance to positive acceleration.

Method and Materials

The method of threshold determination which uses central light loss with the dark adapted eye was initially used on this experiment. However it was soon found that it was too delicate and was abandoned. The method relies upon complete relaxation of the subject for accurate results since minor fluctuations in blood pressure and muscular effort raise the threshold. During voluntary overbreathing there is strenuous muscular activity and change in position which affects the results.

The method was to determine a level of acceleration at which the subject just began to have dimming of vision with bright ambient illumination. When this level had been found and the subject had been rested he was asked to overbreathe and to continue until the centrifuge had come to rest. The degree of hyperventilation was entirely in the subjects' control as it was felt that this was more natural than setting the rate by means of signals. The subject was allowed to overbreathe for one and half minutes at which time the centrifuge was started. The run consisted of accelerating at 1.0g/sec. to the previously determined peak g, staying at peak for twenty seconds and then decelerating to rest. The subject continued overbreathing throughout the run. On completion of the run the visual symptoms were reported by the subject and compared with the control run.

Six experienced centrifuge subjects were used for this experiment. All of them had carried out over five hundred runs and experience had shown that their subjective impressions of degree of visual loss were as reliable as any system of peripheral lights.

The instrumentation on these runs consisted of

bipolar occipital electrodes from which the E.E.G. was recorded. It has previously been determined in these subjects (Part 1 chapter VII) that two minutes hyperventilation at rest produced no abnormality in the E.E.G.

Results.

The results are shown in table 22. The degree of greyout is judged from the amount of narrowing of the visual field.

Table 22.

Subjective visual impressions in bright light during 20 sec. runs at g levels where visual symptoms are just beginning to appear. The rate of application of g was 1.0g/sec.

Subject	Peak g	visual symptoms	
		g alone	g + 2 min overbreathing.
P.G.	3.6	clear	mild grey-out
N.L.A.	3.6	veiling	severe grey-out
M.K.B.	3.6	clear	mild grey-out
G.H.B.	3.4	grey-out	unconscious
J.C.G.	3.4	veiling	mild grey-out
F.L.	3.6	veiling	severe grey-out

It can be seen that in all subjects the visual symptoms occurring under g were aggravated by hyperventilation. One subject (G.H.B.) lost consciousness at the low level of 3.4 g when he had a major convulsion. His E.E.G. is shown in fig. 27. There are no changes in the E.E.G. until loss of consciousness occurs when there is emergance of large slow waves.

No samples of alveolar air were obtained. Subjectively the subjects all complained of tingling of the limbs, dizziness and two (P.G., F.L.) of headache.

Discussion

Active hyperventilation causes a reduction in cerebral blood flow of 33-35% (Kety & Schmidt 1946) which is due to cerebral vasoconstriction (Kety & Schmidt 1946,1947, Schmidt 1950). This reduction in cerebral blood flow is almost certainly the cause of the diminished cerebration. When overbreathing the blood flow through the brain and retina is already reduced and any given degree of acceleration will produce a more profound effect. Similarly, if the decrease in flow to cause symptoms is known, hyperventilation will contribute to this diminished flow and the visual symptoms will occur at a lower g level. The experimental findings agree with this

theory. The one subject who lost consciousness may have been more susceptible to overbreathing than the others or alternatively he may have been more vigorous in his efforts and reduced his arterial pCO_2 to a lower level. Unfortunately the lack of blood or alveolar air samples precludes any definite conclusion on his lowered threshold, although it is interesting to note that Hinshaw et al. (1943) say that the severity of symptoms is not directly related to the degree of hypocapnia.

The occurrence of delta waves in the E.E.G. of the subject who lost consciousness is in accord with the findings of the other workers. Hyperventilation is accompanied by slowing of the electroencephalogram (Engel, Romano, Ferris, Webb & Stevens 1944, Palthe 1953, Brent, Powell & Scott 1957) This slowing is directly correlated with the lowering of the level of consciousness (Engel, Ferris, Stevens, Logan & Webb 1946) and is due to cerebral anoxia. The cerebral anoxia is not only due to the diminished cerebral blood flow but also to the fact that cerebral metabolism is increased during active hyperventilation (Kety & Schmidt 1946). Delta activity (1-3 c.p.s., 50-200 μV) is the end result of this anoxaemia and represents maximal slowing of

the E.E.G. It is not characteristic of hyperventilation but occurs in severe anoxia of any cause and is normally associated with loss of consciousness. Its occurrence during positive acceleration has already been described (Part I chapter VII).

It has been shown that hyperventilation is extremely dangerous in flight both on its own account and because it lowers tolerance to positive acceleration. In the modern high speed aircraft oxygen is breathed as a routine regardless of the operating altitude, and it would appear that the dangers of hyperventilation could be averted by the addition of small quantities of carbon dioxide to the oxygen breathed. The benefits of this procedure however would require to be weighed against its disadvantages, the main one of which would be the lowering of the altitude at which pressure breathing becomes necessary.

Conclusions

Overbreathing is a common occurence in flight and is potentially dangerous. When it occurs in conjunction with positive acceleration there is summation of effect and tolerance to acceleration is lowered. This is due to the fact that both stresses diminish the cerebral blood flow. It may be that small quantities of carbon dioxide added to the inspired oxygen should be beneficial.

Summary.

In six subjects hyperventilation caused a worsening of the visual symptoms caused by a given acceleration. This indicates a lowering of tolerance to acceleration. One subject lost consciousness following two minutes voluntary overbreathing culminating in a centrifuge run of 3.4 g.

CHAPTER IX

Multiple stress summation

In the preceeding chapters it has been shown that the various flight stresses used experimentally have all lowered tolerance to acceleration. In this final set of experiments the aim was to examine the effects of these stresses in combination and determine if further summation of effect would occur. So far the basic stress used has been positive acceleration. In this experiment another standard stress was used in addition. The choice of the other basic stress was determined by its frequency of occurrence in the air. Hyperventilation was the obvious choice.

Both hyperventilation and positive acceleration lower the blood supply to the brain and their effects summate (Chapter VIII). It has also been shown that the effect of hyperventilation alone is potentiated by a low blood sugar (Engel et al. 1944, 1946, Palthe 1953). Similarly it has been shown that hypoglycaemia aggravates the symptoms due to positive acceleration (Chapter IV).

It appears likely that these three stresses might provide a potent combination. The possibility of a combination of these three factors being the underlying cause of many aircraft accidents has been pointed out by Powell (1956,1957), who suggested a diagnosis of " physiological unconsciousness" for these cases of medically fit aircrew who lost consciousness while flying. From an analysis of operative factors (Table II), he suggested the following five factors were incriminated and that combinations of them might be the cause of accidents due to loss of consciousness.

a) Previous or concommitant positive acceleration.

b) Hypoglycaemia

c) Hyperventilation

d) Anger or anxiety.

e) Early slow E.E.G. activity with hyperventilation.

However he was unable to produce the condition experimentally under laboratory conditions.

More recent work from Canada (Brent et al.1957) has established this concept more clearly. These authors studied twenty one healthy aircrew subjects and found fourteen showed early slow E.E.G. activity while fasting, two showed similar changes while under 3g and eleven of the above fourteen showed greater slowing with shorter periods of hyperventilation while under 3g. Of these eleven, three lost consciousness, of whom two manifested major convulsions. The body of evidence suggests that these three factors are particularly potent. The first part of the present experiment was an attempt to establish the validity of the above theory and work.

In the author's view there was no reason why the other factors already examined should not also act synergistically. The effect of heat, as already pointed out, is to cause a loss of effective circulating blood volume and would therefore be expected to diminish further the tolerance to g when in combination with hyperventilation,

Similarly although the mode of action of alcohol is not clearly understood it also lowers the tolerance to g and might well be more potent when associated with hyperventilation. An attempt to study these factors was also made and formed the second part of this study.

Methods & Materials

The same subjects were used as in all the other experiments of this series with the exception of G.H.B. The latter subject was excluded as it had been demonstrated (Chapter VIII) that he lost consciousness with the combination of g and hyperventilation alone. Five subjects participated in the present series.

The form of centrifuge run was the same as used for the study of hyperventilation alone. Having previously determined the level of g at which the subject had minimal visual signs, all future runs were made at that level. The centrifuge was accelerated to the determined peak g maintained there for fifteen sec. and decelerated to rest.

The subject commenced voluntary overbreathing ; one and a half minutes later the centrifuge run was started. Hyperventilation was continued throughout the run if the subject was able.

(a) Hypoglycaemia

The hypoglycaemia was again induced by soluble insulin. It was decided that a very slight fall in blood sugar was desirable. This was partly because the condition should be similar to the fasting state, but largely because it was felt that it was the fall in blood sugar which was important rather than the absolute value. The dose used was 2 units of soluble insulin given intravenously. The runs were carried out twenty minutes after the injection to minimise any chance that a reaction might occur during the run.

(b) Heat

When the subject attained a mouth temperature of 101° F. a combined hyperventilation run was carried out.

Unfortunately only one result was obtained since the other subjects had reached the limits of tolerance with g alone.

(c) Alcohol.

Two and a quarter hours after having consumed 114 ml. of whisky the subjects carried out a g run with hyperventilation as described above.

In all cases a control run was carried out before the experimental procedure. The E.E.G. was recorded from bipolar occipital electrodes throughout the runs.

Results

In each case the effect of g alone is compared with g plus two minutes hyperventilation and then with the results of the three factors together.

(a) Hypoglycaemia

Table 23

The effect of 2 units of intravenous soluable insulin on the visual symptoms produced by g and 2 min hyperventilation. G.O. = greyout B.O. = blackout N.V.S. = no visual symptoms.

Subject	g alone	g + 2 min H.V.	g + 2 mins H.V. + 2 units insulin intravenously
P.G.	N.V.S.	mild GO	unconscious
N.L.A.	veiling	severe GO	unconscious
M.K.B.	N.V.S.	mild GO	unconscious
J.C.G.	veiling	mild GO	unconscious
F.L.	veiling	severe GO	unconscious

As will be seen from table 23 the effect of g and hyperventilation when hypoglycaemia has been produced by insulin is unconsciousness in all subjects Three of the subjects exhibited major convulsions (P.G., M.K.B., F.L.) and one other (N.L.A.) twitching of the arms. The E.E.G. showed the development of slow large amplitude waves (1-3 c.p.s.

70-130μV) when consciousness was lost in three subjects; the E.E.G.s of the other two subjects were valueless as the amplifiers blocked due to the convulsions. The best record is shown in fig.27.

(b) Heat.

Only one subject (F.L.) felt able to attempt a combined hyperventilation and g run when his mouth temperature was 101° F. He lost consciousness as soon as peak g(3.1g) was reached and had a major convulsion during which the E.E.G. electrodes became detached and no record was obtained.

(c) Alcohol.

Table 24

The effect of combined g and hyperventilation in subjects who had ingested 114. ml. of whisky 2½ hours before the run.

Subject	g alone	g + 2 min.H.V.	g + whisky + 2 min H.V.
P.G.	N.V.S.	mild GO	unconscious
N.L.A.	veiling	severe GO	unconscious
M.K.B.	N.V.S.	mild GO	unconscious
J.C.G.	veiling	mild GO	unconscious
F.L.	veiling	severe GO	unconscious

It may be seen from table 24 that whereas hyperventilation alone caused mild aggravation of visual symptoms, the same procedure two and a half hours after the ingestion of 114. ml. of whisky resulted in loss of consciousness in all subjects.

The E.E.Gs in this series also showed delta activity which was associated with loss of consciousness. (Fig. 28).

Major convulsions occurred in two subjects (P.G., M.K.B.) and minor twitching in another two (N.L.A., F.L.). The fifth subject (J.C.G.) showed no evidence of increased muscular activity. J.C.G. has never had jactitations under g and on the occasions when he lost consciousness he became limp and completely relaxed. There is no difference between his resting E.E.G. and those of the other subjects, nor is there any difference in response to overbreathing. There is therefore no obvious reason why this subject should react differently to the others.

Discussion

The results obtained by combining hyperventilation, hypoglycaemia and g in the present experiment confirm the theories of Powell (1956) and Brent, Powell and Taylor (1957). However, Powell was unable to reproduce the effects in normal people under laboratory conditions, while Brent obtained unconsciousness in three out of eleven subjects who showed abnormal slowing of the E.E.G. with hyperventilation alone.

In this series none of the subjects showed abnormal slowing of the E.E.G. with hyperventilation alone but all five subjects lost consciousness under the additional stimulus of g. Brent produced hypoglycaemia by fasting his dubjects and also by carrying out runs during the period of rebound hypoglycaemia produced by giving a fasting subject a glucose feed. It seems likely that the difference in response was due to the use of insulin to produce hypoglycaemia in the present experiment. Although the dose was extremely small it produces a fall in blood sugar as compared to the steady state which occurs with fasting. One would have expected the results to be more comparable with Brent's subjects who were tested during the rebound hypoglycaemia period after a glucose feed.

The result of the one subject who carried out the combined hyperventilation, heat and g run was as expected. The subjects were all at their limits of tolerance and any additional stimulus such as hyperventilation was likely to cause collapse.

This occurred at 3.1 g in the subject who carried out the experiment. His normal threshold of unconsciousness is between 5-5.5 g.

From the third set of results it would appear that the combination of alcohol, hyperventilation and g is just as potent a combination as that suggested by Powell. In this experiment the mode of action of alcohol is not clear. It may be that the whisky reduces the sensitivity of the carotid sinus and so the compensatory mechanisms fail to develop. On the other hand it may be that the change is at the cellular level and is an interference with the normal metabolism of the cell.

The only significant changes in the E.E.G. were the appearance of large slow waves when consciousness was lost. These are not specific, and as have been pointed out before they occur with loss of consciousness in many people regardless of the cause.

Conclusions.

The combination of positive acceleration, hyperventilation and either hypoglycaemia, heat or ethyl alcohol caused unconsciousness in all subjects tested at very low levels of g (3.4 - 3.6). This represents a fall in tolerance of about 2g. This represents a greater fall than caused by any two of the factors alone. It would appear therefore that there is a further summation of effect when an additional stress is added.

Summary

When centrifuge runs were carried out at 3.4 - 3.6 g after one and a half minutes of hyperventilation unconsciousness developed on the addition of the following factors:

1. Insulin hypoglycaemia
2. Heating the subject until his mouth temperature was 101°F.
4. The ingestion of 114. ml of whisky two and a quarter hr.. before the run.

When unconsciousness occurred the E.E.G. showed delta rhythm, and several subjects had major or minor convulsions.

CHAPTER X.

General discussion and conclusions.

In the second part of this thesis an attempt has been made to test the validity of the concept of physiological unconsciousness, and to examine a larger number of physiological factors which, in theory, might be expected to summate with the effects of positive acceleration and produce changes in consciousness. In most cases the presence of an additional stress has diminished tolerance to g.

It has been shown that heat, hypogylcaemia, hyperventilation, alcohol administration and an empty stomach all lower tolerance to increased gravitational force. The results of breathing oxygen at atmospheric pressure are not statistically significant but there is a possibility that some subjects may be susceptible to the toxic effect of pure oxygen. In the few experiments when some of these individual stresses were combined, summation occurred.

The combination of hyperventilation and hypoglycaemia resulted in a loss of consciousness of all subjects at the low level of 3.4 g - 3.6 g. This confirms the findings of Palthe (1953) and supports those of the investigations at D.R.M.L. The latter workers found difficulty in reproducing "Physiological unconsciousness" in the laboratory. Only three subjects out of twenty one succumbed at 3 g, and these subjects showed slow activity in the E.E.G, while fasting, and during hyperventilation under g. In the present experiments none of the subjects showed slowing of the E.E.G. with hypoglycaemia, or hyperventilation under g, but all lost consciousness when these were combined, This difference in response might be due to three factors, viz:-

(a) The runs in this study were 0.5 g higher.

(b) The use of insulin would produce a falling blood sugar.

(c) The pattern of centrifuge run may have been different.

The last factor cannot be compared, as the Canadian reports do not state the specifications of the runs and as has been shown in Part I Ch.IX. variations in the acceleration-time pattern can produce changes in tolerance. The dose of insulin used to produce hypoglycaemia was small (2 units of soluble insulin intravenously) and the resulting blood sugar level would probably be little different to the fasting levels (unquoted) of the Canadian workers. However as has been pointed out before, the falling blood sugar may be more important then the absolute level reached, in which case the use of insulin would produce a more profound effect than fasting. The runs carried out in this experiment were 0.5g higher than those at D.R.M.L. which would also give a diminished tolerance, although Scott (personal communication) reports that their results are essentially unchanged using runs of 3.4 g. Thus the results of applying these stresses differ only in degree from those of the Canadian team, and establish the concept of a "physiological unconsciousness".

In only one subject was heat and hyperventilation combined. This resulted in loss of consciousness at 3.4g. The remaining subjects were at the limits of their endurance with heat alone and hyperventilation was not attempted. Although this is a solitary result it seems reasonable to postulate excessive heat as a further factor which may summate to produce "physiological unconsciousness".

When hyperventilation was carried out after the ingestion of whisky, unconsciousness again occurred at levels of 3.4g-3.6g in all subjects. Thus the effects of alcohol can also be included in those physiological stresses which may summate to produce loss of consciousness in flight.

It is well known that tolerance to acceleration is higher following a meal, and in this thesis an attempt has been made to examine the factors causing this change. Two factors have emerged. Firstly that hypoglycaemia lowers tolerance to acceleration although hyperglycaemia has little effect.

Secondly, the purely mechanical effect of stomach distension increases tolerance by increasing intra-abdominal pressure and in the author's view, by preventing descent of the heart during g. The effect of missing a meal is therefore of importance as both the above factors will be operative. The advice given by Lawton (1953) and Palthe (1953) that pilots should not miss meals and fly on an empty stomach, although based on observations on hypoglycaemia alone, is especially true in view of the effect of g, and absence of protection afforded by a full stomach. It was not possible to combine these stresses with hyperventilation in this series of experiments but on theoretical grounds it is to be expected that summation of effect will occur.

This series of experiments has shown that tolerances to positive acceleration can be diminished by a variety of physiological concitions which may occur in flight. These factors may summate and result in loss of consciousness at low levels of g.

It is considered probable that many cases of episodic unconsciousness in flight can be explained on this basis without recourse to obscure pathological and psychological causations. The concept of "physiological unconsciousness" suggested by Powell (1956) has been confirmed and amplified by these experiments. As originally described, the aetiological factors were g, hypoglycaemia, hyperventilation, and fear or anxiety occuring in an individual who showed early slow activity in the E.E.G., with overbreathing. From the results described above it is believed by the author that these criteria are too circumscribed, and that the concept should be based on a broader physiological foundation. I would suggest that "physiological unconsciousness" is a loss of consciousness occuring in flight due to the summation of a number of physiological stresses each of which is, in itself, inadequate to produce such an effect. These stresses may be any physiological conditions which reduces circulating blood volume, arterial blood pressure at head level or cerebral blood flow, or reduces the substrate supply to the brain itself.

CHAPTER XI

Summary

The first part of this thesis consists of a general consideration of the physiological effects of acceleration. The means of determining human tolerance to this force is by means of threshold determinations. These are discussed in relation to the mechanical variables involved: strength, duration and rate of application of g. The changes in pulse rate, blood pressure, the electrocardiogram vectorcardiogram and electroencephalogram, have all been analysed and compared with the results of previous investigators. It was found that the pulse rate varied directly with g while the blood pressure bears a reciprocal relationship to g. The changes in pulse rate closely followed changes in blood pressure, and both depended not only on the peak g but also on the rate with which it is applied. There were no characteristic changes in the electrocardiogram, the electrical axis shows a clockwise rotation which is partly positional and may also

be related to increased autonomic activity. There was only one arrhythmia in a series of two thousand runs, this occurred at 8.0 g. and reverted spontaneously to sinus rhythm on returning to 1.0 g. No evidence of myocardial ischaemia was obtained from any of the records. The vectorcardiogram shows instantaneously any changes in the electrical axis of the heart but otherwise gives no additional information to that obtained from the E.C.G.

There are no characteristic changes in the electroencephalogram during positive acceleration. During the early part of the runs there is a fall in the amplitude of the alpha rhythm which reverts to normal after five to ten seconds. Greyout and blackout occur without any corresponding change in electrical activity. The onset of unconsciousness however is accompanied by the appearance of slow, large amplitude waves. Most subjects exhibit convulsions of a major or minor type when consciousness is lost. These similarly are not accompanied by any E.E.G. changes apart from the delta activity found with unconsciousness.

The optimal conditions for threshold determination are outlined and a method is described which fulfils these conditions. The method depends on loss of central vision by the dark adapted eye and is accurately repeatable. The value of g at which the end point occurs can be varied at will, the upper limit being the level of absolute blackout. The variables are capable of precise specification. The type of g run used is based on the results obtained in the earlier part of this study and allows cardiac compensation to develop as the g increases.

The second half of the thesis is an investigation into stress summation in flight. It was thought that many other factors might operate to produce a diminished tolerance to acceleration, and even to lead to loss of consciousness. The results of these experiments are summarised below:-

1) The application of heat lowers the subjects' tolerance to g. There is a large subject variation which is probably allied to their emotional state.

2) Breathing pure oxygen at atmospheric pressure produces a variable effect. There is some evidence that there may be an individual susceptability to the effects of oxygen: susceptable subjects showing a fall in threshold.

3) Hyperglycaemia does not significantly alter tolerance to g.

4) Insulin hypoglycaemia lowers tolerance to g. although during the 'reaction phase' the tolerance rises again probably due to endogenous production of adrenaline.

5) An empty stomach is associated with a low g tolerance and distending the stomach results in a rise in tolerance. This is only partly due to raised intra-abdominal pressure, and it

is postulated that the distended stomach by supporting the diaphragm prevents descent of the heart during g and so affords some protection.

6) Tolerance to g is diminished after the ingestion of whisky. The mechanism of this fall has not been established.

7) Hyperventilation lowers g tolerance and resulted in unconsciousness in one subject.

8) The combination of g and hyperventilation in subjects who were hypoglycaemic resulted in loss of consciousness at levels of 3.4 g - 3.6 g. In one subject who was hot (mouth temperature 101° F), g. plus hyperventilation resulted in unconsciousness at 3.4 g - 3.6 g. After the ingestion of 114. ml. of whisky all subjects lost consciousness at 3.4 g. - 3.6 g. while hyperventilating.

The relation of these results to the concept of "physiological unconsciousness", or episodic unconsciousness in flight, is discussed.

APPENDIX

Method of triggering used to obtain vectorcardiograms in mid-expiration

The general lay-out of the citcuit used is shown in fig. 29. The potentials arising in the various electrodes are led into the A.C. preamplifiers, from which the amplified signals are split, one set going to the C.R.O, the other set going to the final pen amplifiers and being displayed on the ink-writing recorder. The potential arising from V1 is duplicated in the A.C. preamplifiers and is padded through the final pen amplifiers to the triggering circuit.

The triggering citcuit is an arrangement of relays by means of which the brightness of the C.R.O spot can be adjusted. Normally the spot is blanked out, at the set time the spot is switched on for a period sufficiently long to allow one V.C.G. to be photographed.

The timing of the circuits can be seen from fig. 30. The signal ffom lead V.1 is lead into this circuit but is blocked at the " delay " trigger stage. This block is controlled by a push button. When the button is pushed it completes the circuit and allows

operation of the trigger. Following the pushing of the button, the next R wave stimulates the delay trigger to produce a square wave, whose duration (x) can be controlled. This wave operates the bright-up trigger which in turn produces a pulse of duration which can also be varied. The relations of these three pulses can be seen in fig. 30(II). This pulse produced by the bright-up trigger switches on the spot on the C.R.O. and allows it to be photographed. The result is shown in fig. 30(III). When the push button is pressed the second PQRST is displayed on the C.R.O. and photographed. The variable times (X & Y) of the two triggers allow compensations to be made for changes in the pulse rate. Thus adjustments of X determines which of the succeeding complexes is displayed and also allows compensation for rate. The adjustment of Y allows only one complex of part of one complex to be selected.

The vectorcardiograms must always be obtained at the same phase of respiration. In this particular experiment it was decided that the recording should be made in a position mid way between full inspiration and full experation in order that the V.C.G. obtained would represent the heart's position when

the diaphragm was in its mean position. The V.C.G. was therefore triggered from the respiratory trace. This was done manually by means of the push button described above. A respiratory record was obtained from a thermocouple on the nostril. The best delineated point in the record was the peak of inspiration which was an upward pointing spike. When this spike occurred, the recorder pressed the push button and the next but one PQRST was photographed. This was found to correspond to mid-expiration in the majority of cases.

much to endure.

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GRAVITY AND MAN

VOLUME II

ILLUSTRATIONS

M. KENNEDY BROWNE

THESIS PRESENTED FOR THE DEGREE OF DOCTOR OF MEDICINE OF THE UNIVERSITY OF GLASGOW.

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PLATE 1 Subject instrumented for heat run.

II Subject in polythene overall for heat run

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4 Changes in threshold and body temperature during heating (GROUP I)

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12 Histogram of blackout thresholds breathing air and breathing oxygen.

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27 The E.E.G. during various experimental conditions.
28 The E.E.G. during various experimental conditions.
29 Bipolar occipital E.E. Gs. during various experimental conditions.
30 Circuit diagram of the apparatus for vectorcardiography.
31 Time relations of the Triggering circuit for vectorcardiogram display.

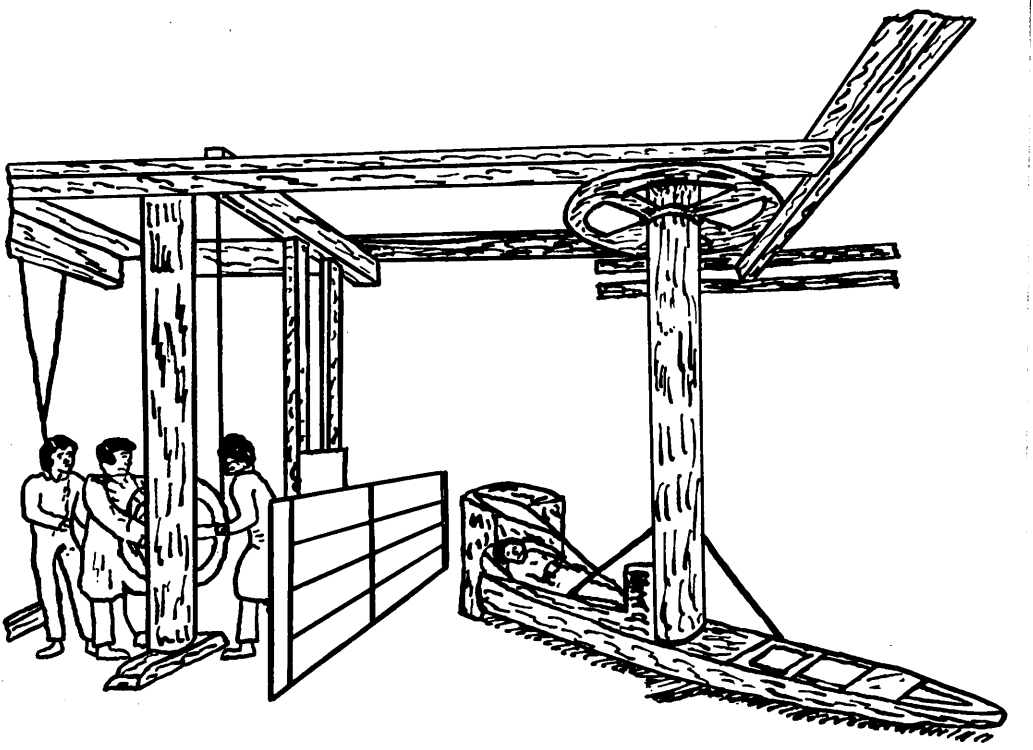


PLATE I. Drawing of the human centrifuge installed in the Charité Hospital, Berlin, at the beginning of the nineteenth century (after Horn 1818). It will be seen that the patient sustained negative g in this machine.

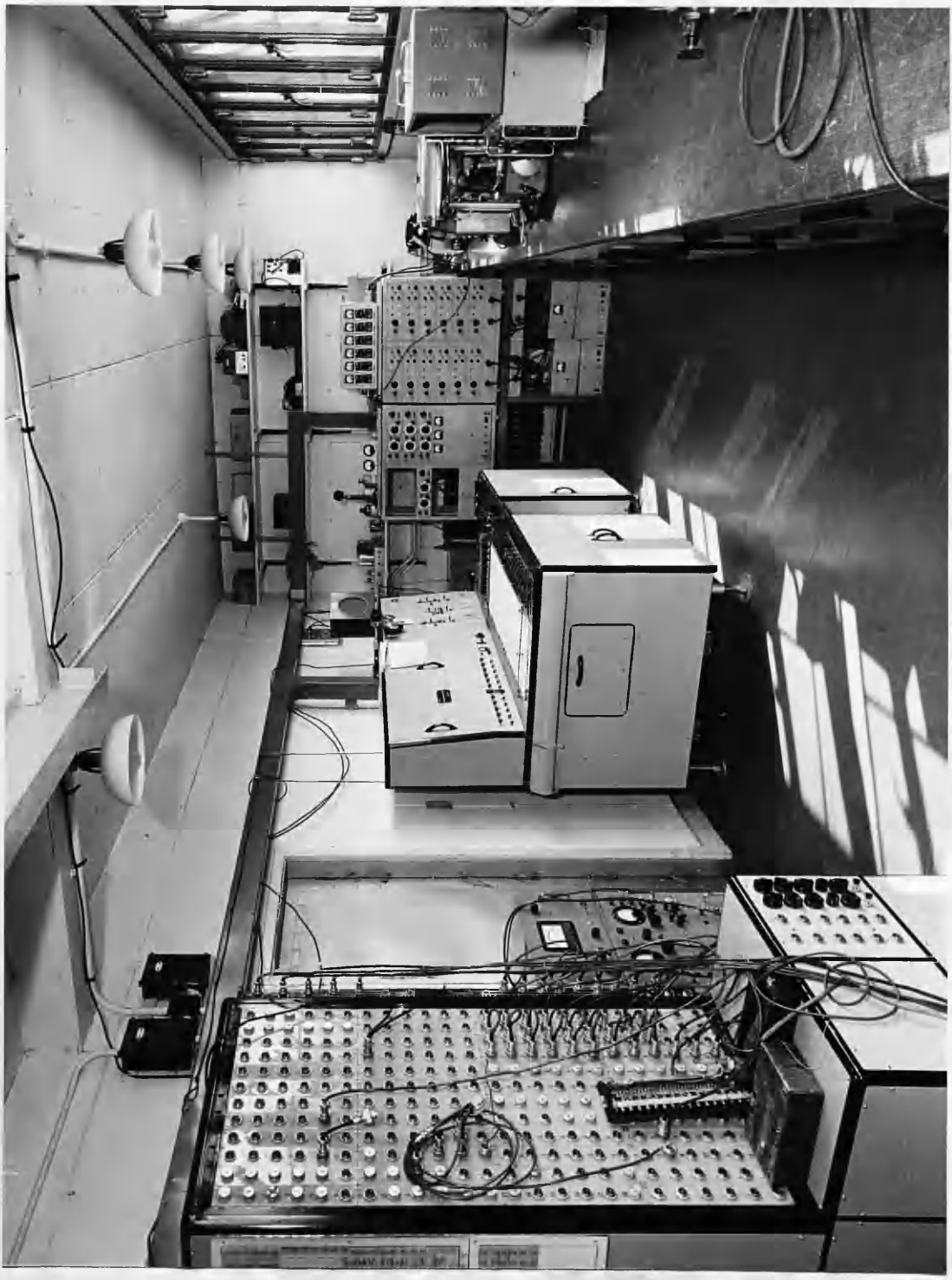


PLATE II. The recording room attached to the human centrifuge at Farnborough. The twelve channel Ediswan recorder can be seen in the left background and the distribution board in the left foreground.

PLATE III. The Control room of the human centrifuge. This room overlooks the centrifuge chamber. The centrifuge can be seen in the centre background.

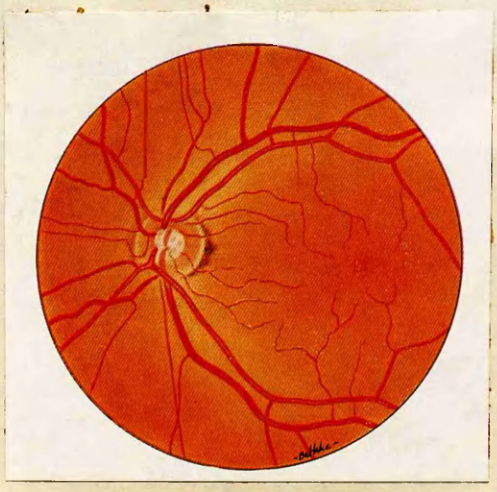
PLATE IV. The automatic control unit for the human centrifuge. A 15g cam is in position. The automatic timer can be seen in the left foreground.

(a)

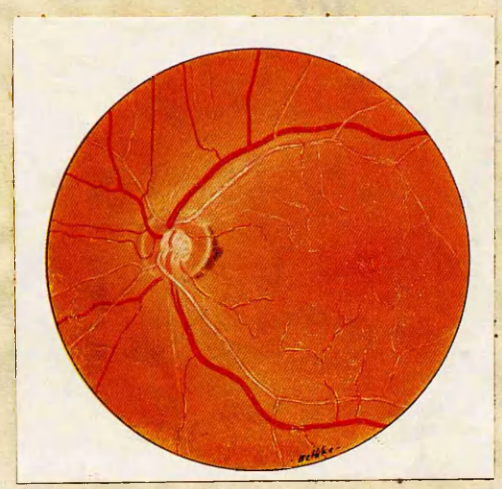
PLATE V (a & b) Facial distortion produced by g.
(a) Subject at rest
(b) Subject at 4.0 g

(b)

PLATE V (b)



(a)



(b)

PLATE VI Retinal changes with g

(a) Normal fundus

(b) Appearance of fundus at blackout. Note the empty arteries.

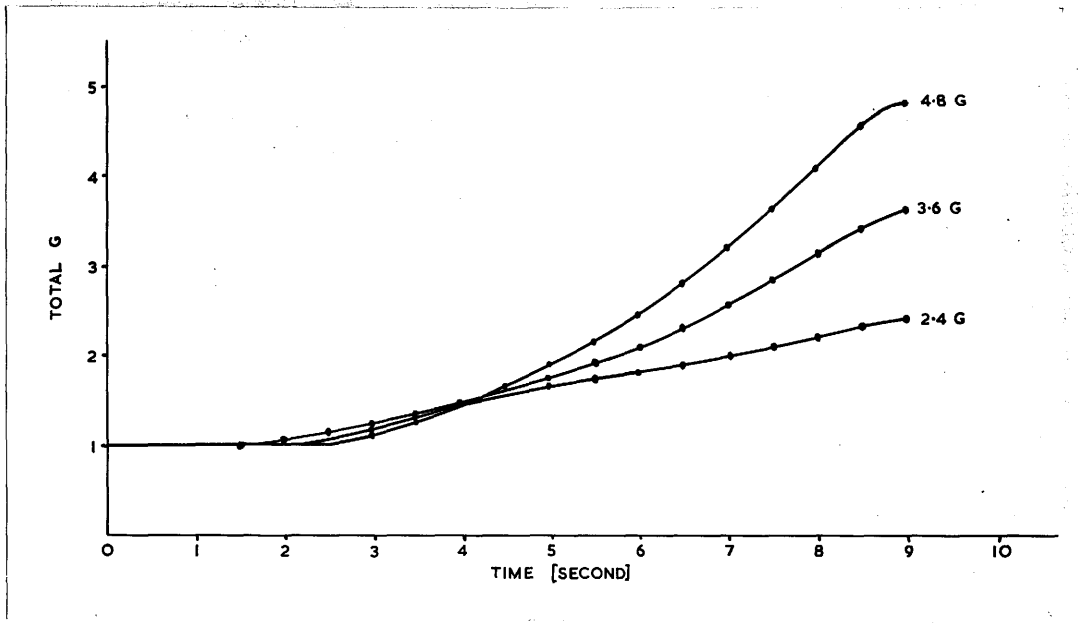


Fig. 1(a). Sample graphs of acceleration v. time for the constant time family of cams.

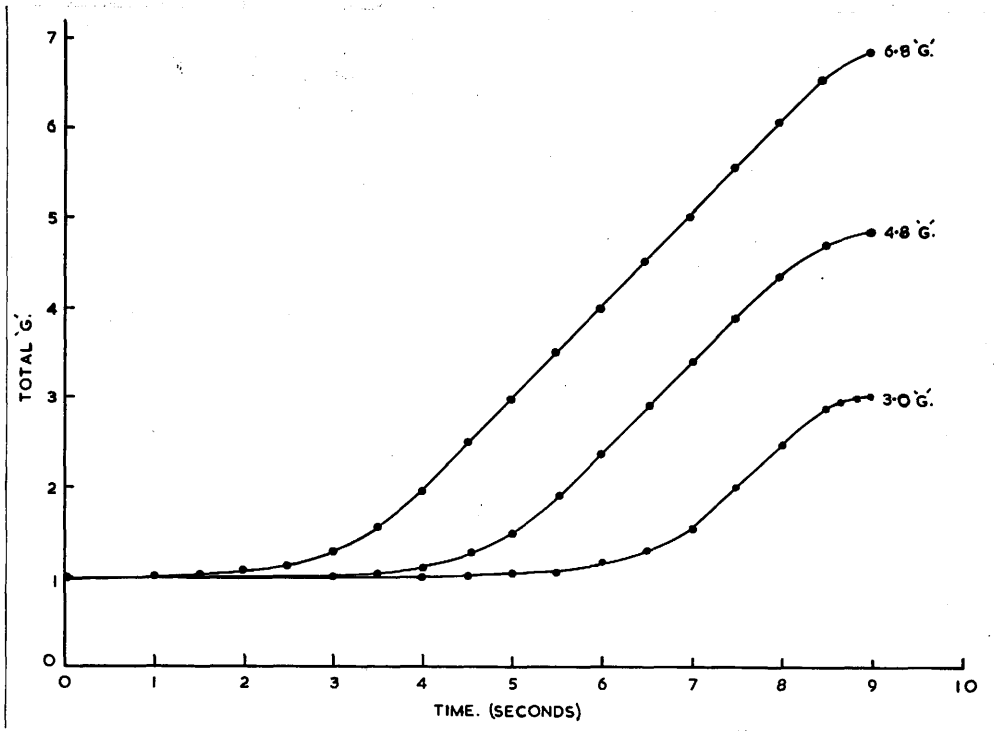


Fig. 1(b). Sample graphs of acceleration v. time for the constant rate family of cams.

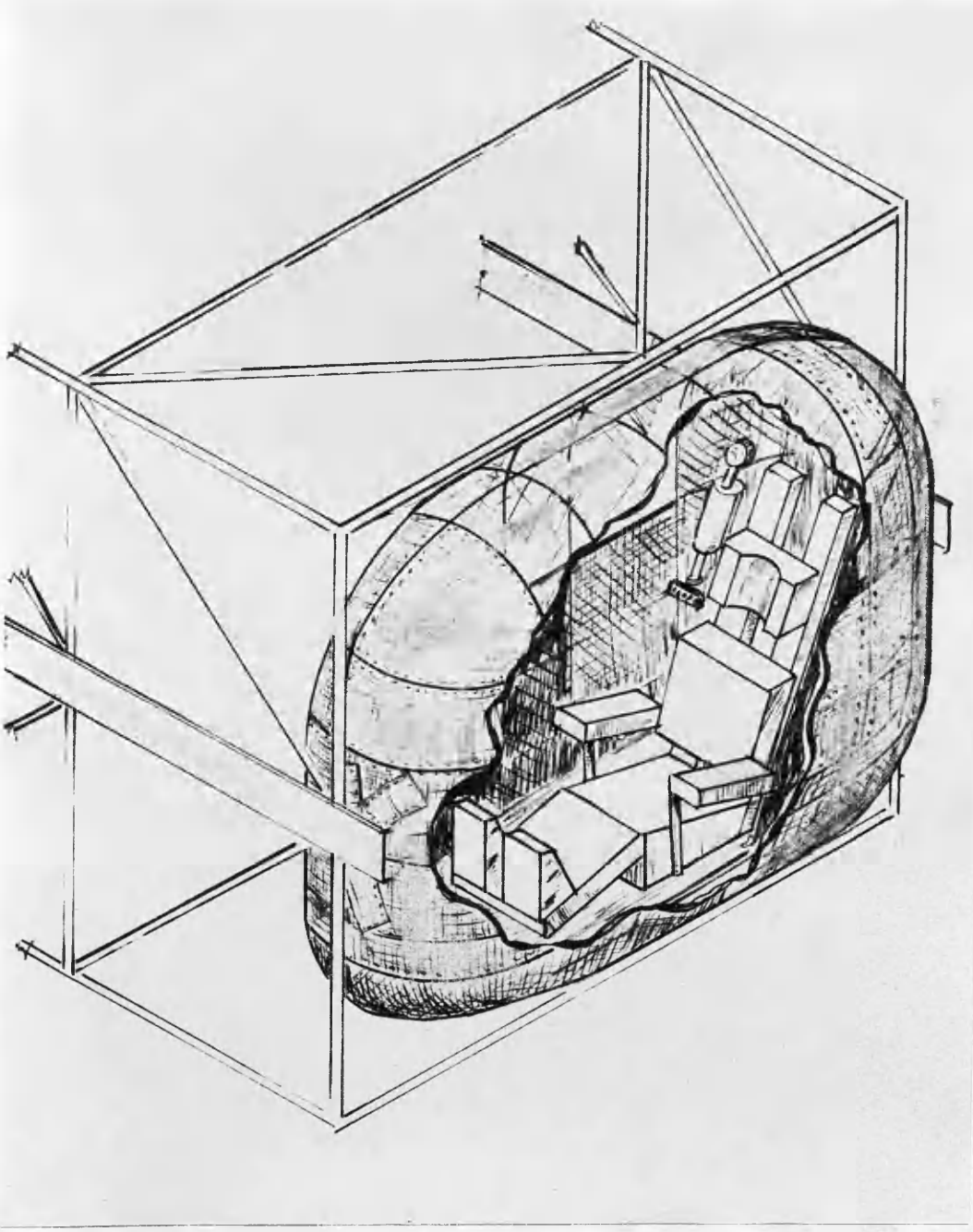


Fig. 2. Schematic cut-away drawing of a gondola showing the position of the seat which is raked 20° to the vertical.

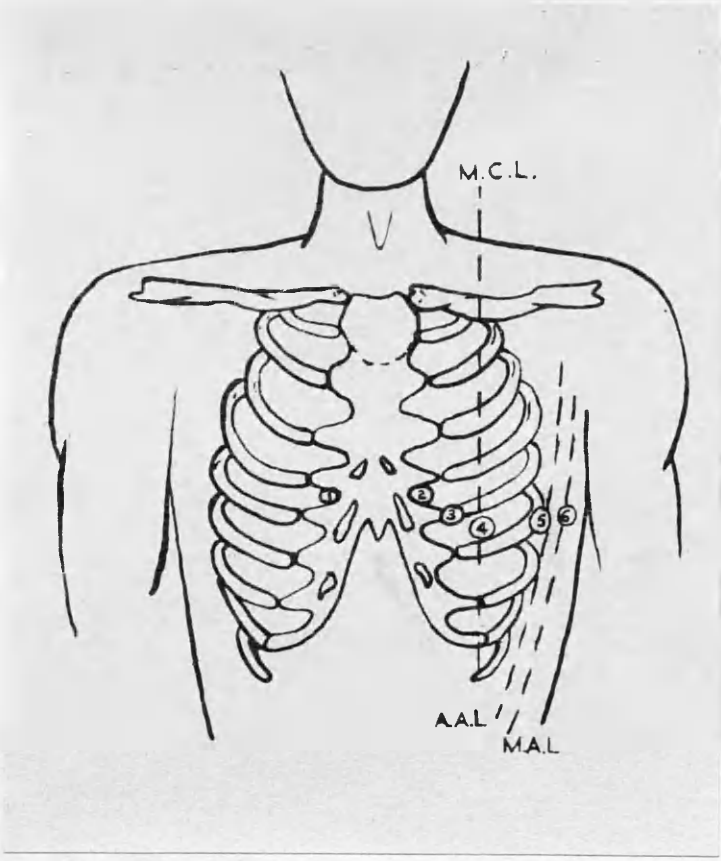


Fig. 3(a) The positions of the unipolar chest electrodes.

M.C.L. - mid-clavicular line

A.A.L. - anterior axillary line

M.A.L. - mid-axillary line

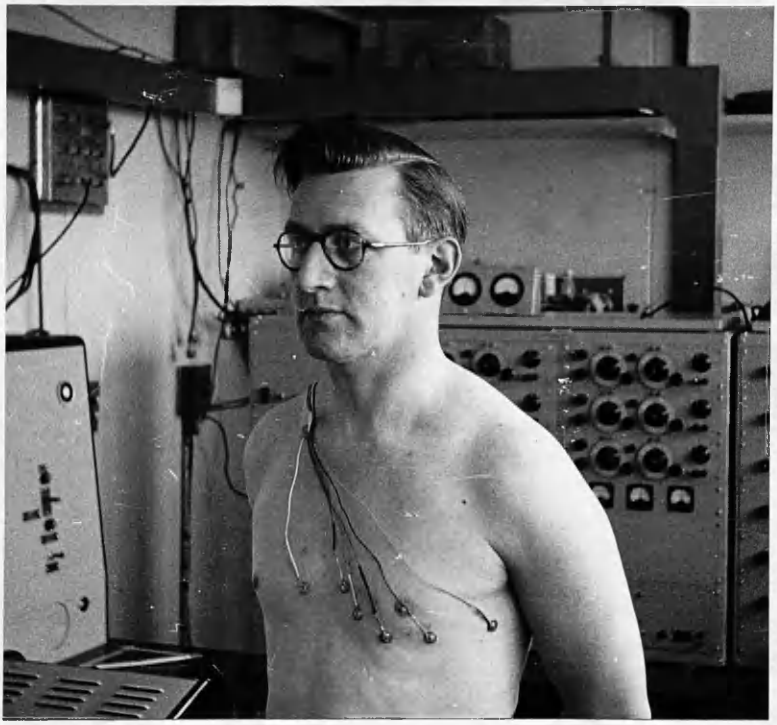


Fig. 3(b) Subject with electrodes in position prior to a run.

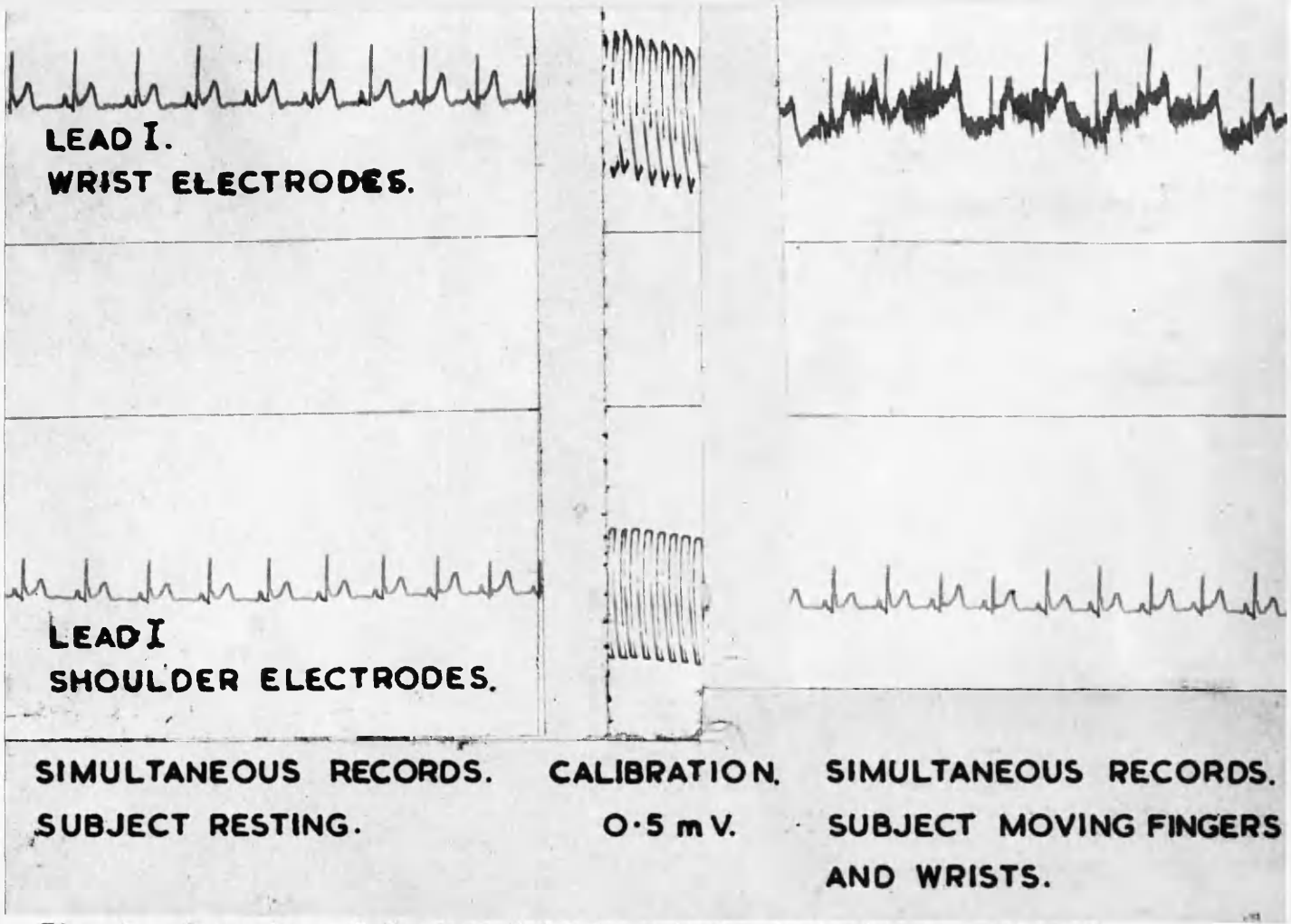


Fig. 4. Comparison of the lead I E.C.G. with the electrodes on wrists and acromion processes. It will be seen that the records from the shoulders are free from muscle artefact and there is little change in the wave form from that obtained from the wrists.

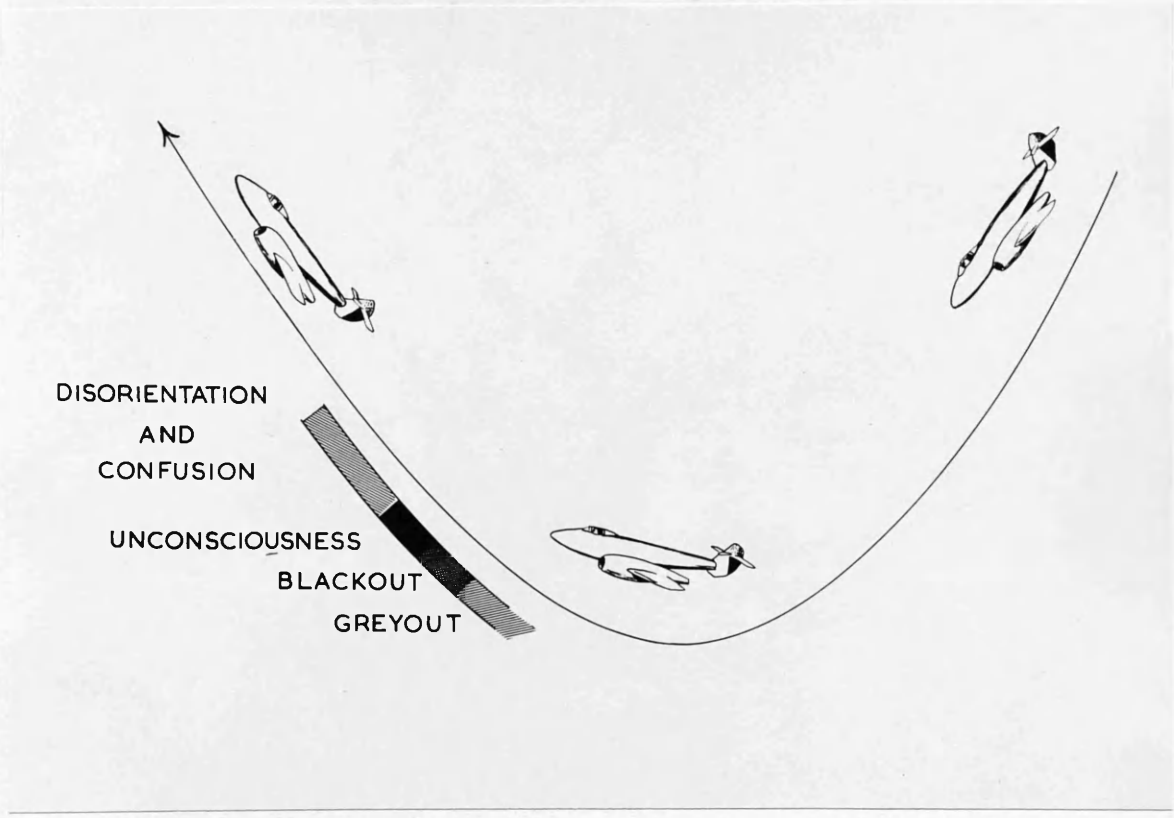


Fig. 5. The visual changes due to positive acceleration are shown in relation to the normal manoevre which causes them.

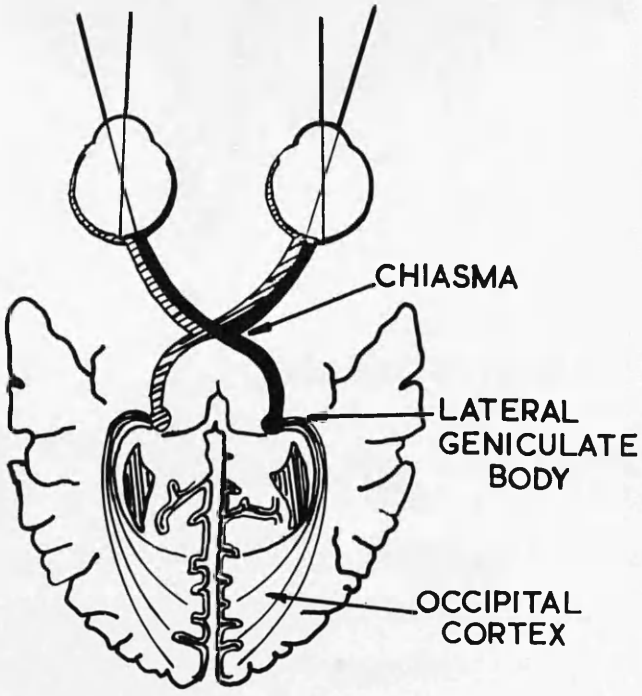


Fig. 6. Diagrammatic representation of the visual pathway. Fibres from medial half of the retina decussate in the optic chiasma to join with those from the lateral side of the opposite eye. The tracts run back to the lateral geniculate body of the same side where they relay, the second stage neurones travelling via the retrolentiform part of the internal capsule to reach the occipital cortex.

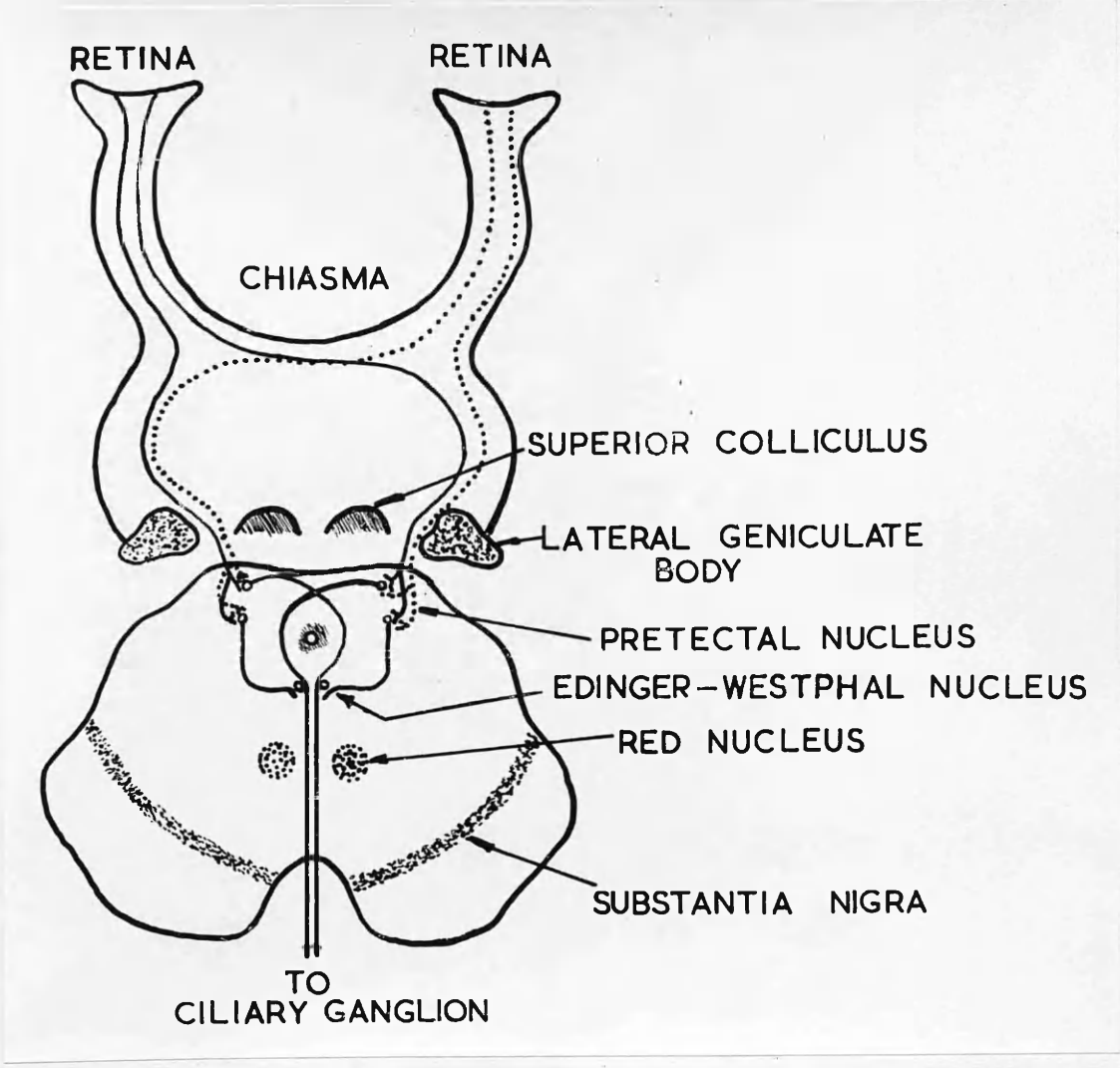


Fig. 7. Diagrammatic representation of the path of the fibres subserving the light reflex. The fibres pass back in the optic tract, passing medial to the lateral geniculate body without synapsing until they reach the pretectal nucleus from which the 2nd stage neurones pass to the Edinger-Westphal nucleus.

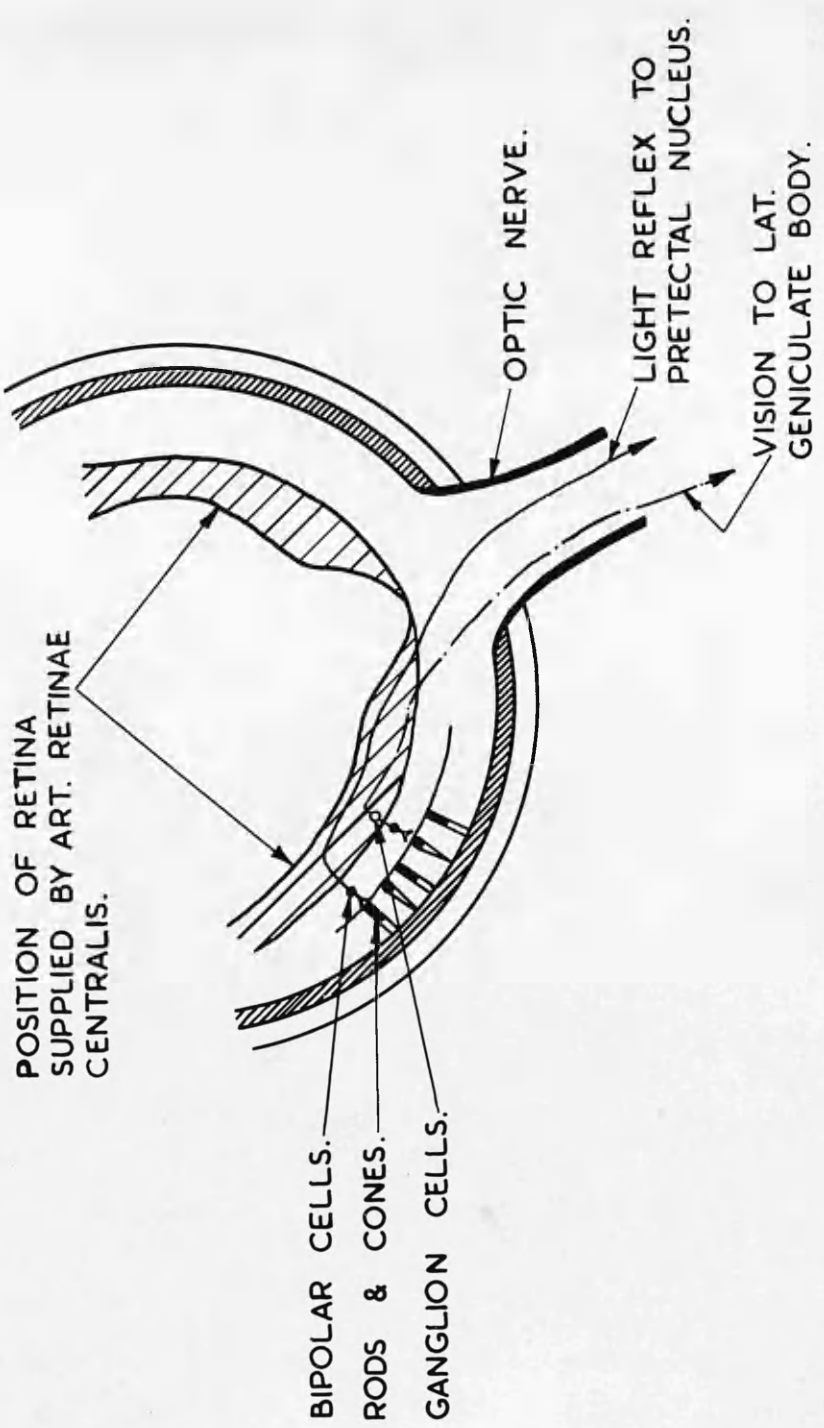


Fig. 8. Diagram showing the nervous connections in the retina of the nerves serving vision and the light reflex (after Lewis and Duane 1956).

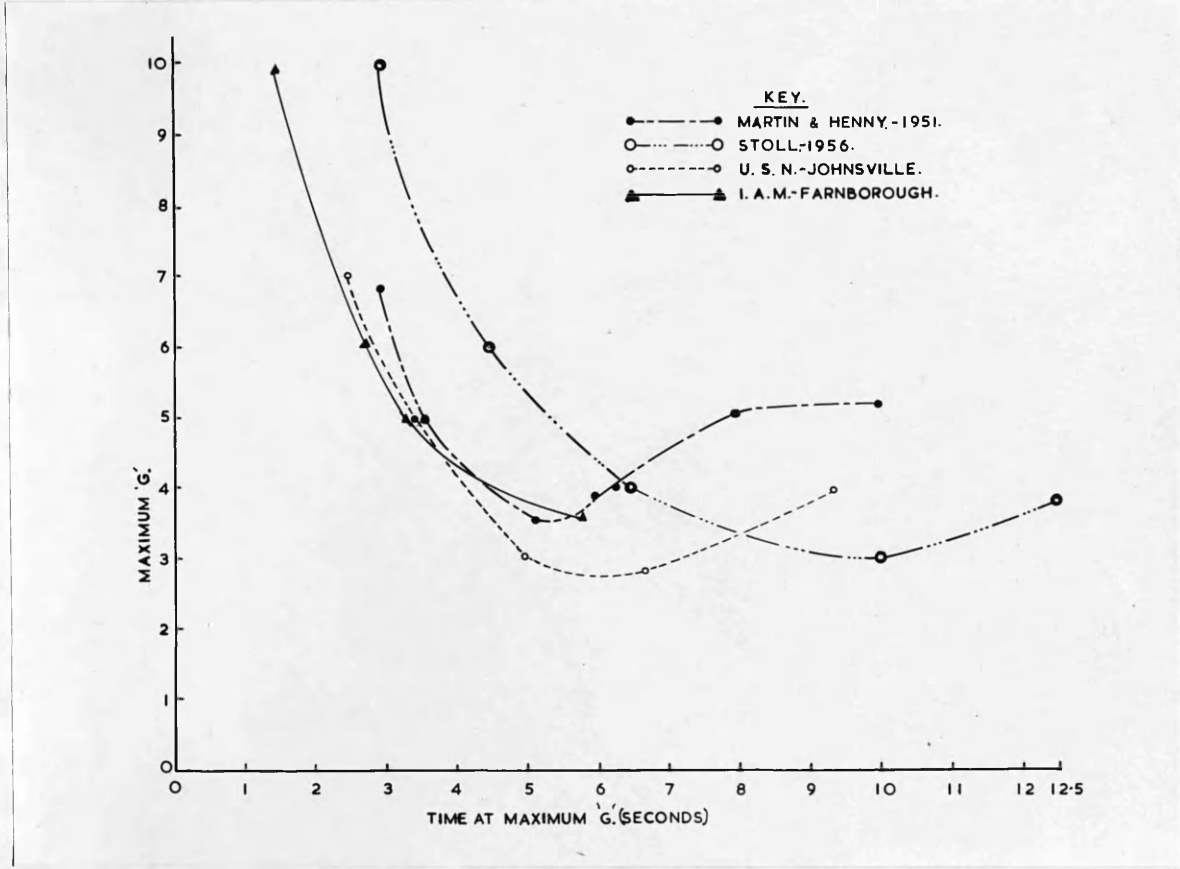


Fig. 9. Strength duration curves for tolerance to positive acceleration. The key shows the sources of the data used in the construction of the curves.

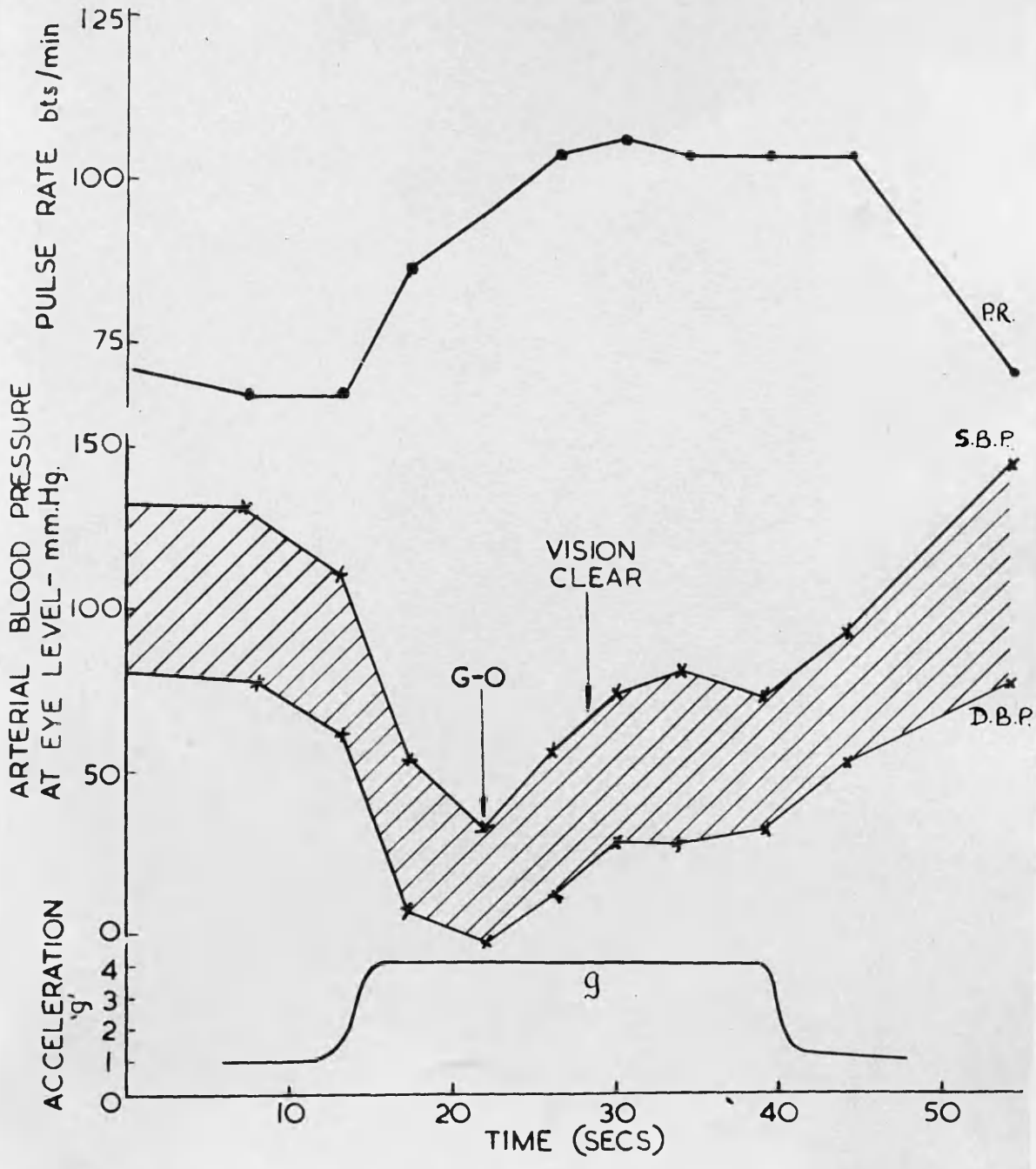


Fig. 10. Graph showing the change in blood pressure at eye level and pulse rate during a centrifuge run to 4.4 g. 'G-O' represents the point at which grey-out occurred.

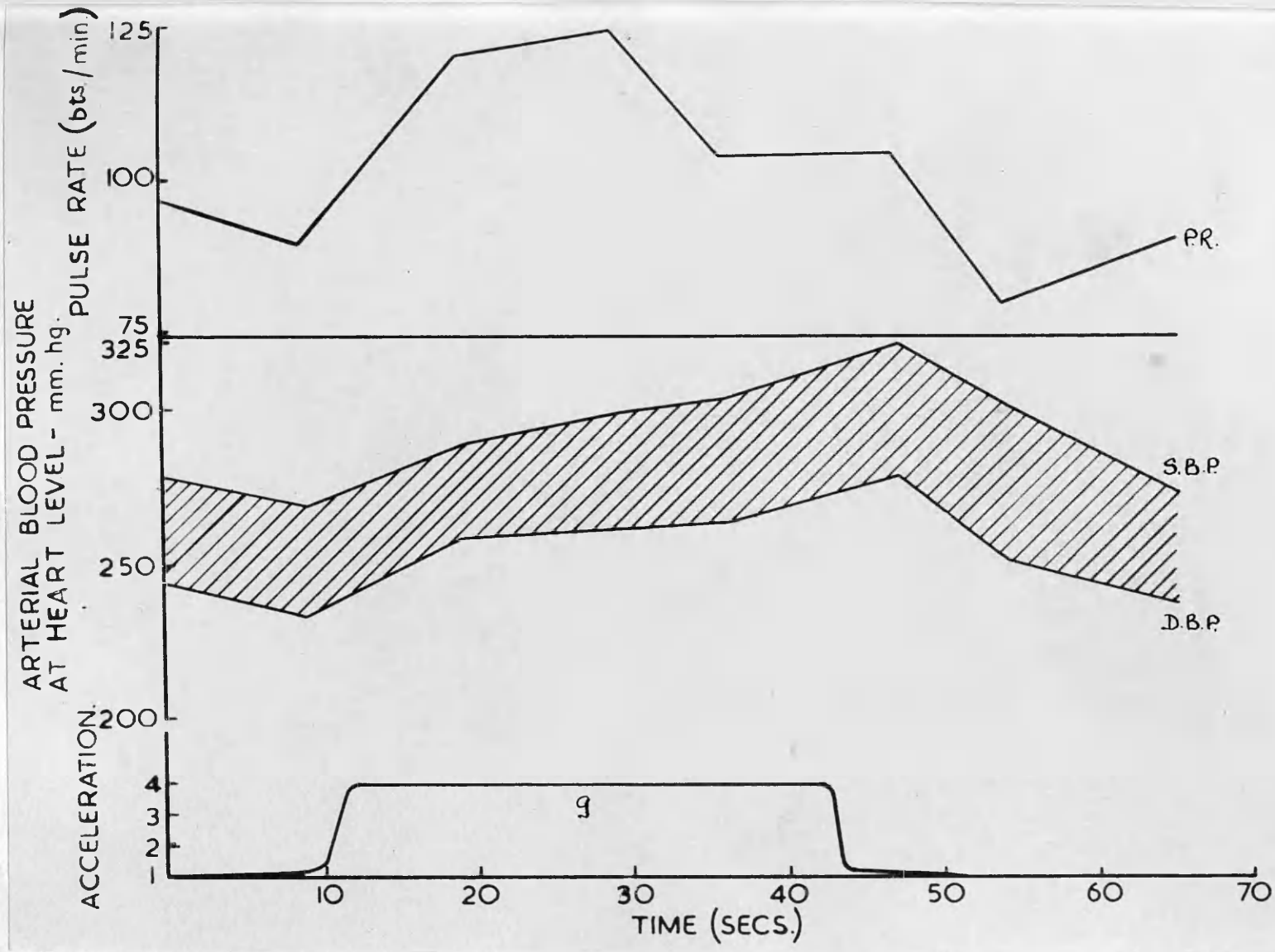


Fig. 11. Changes in pulse rate and in blood pressure at heart level during a centrifuge run to 4g. The calibration of the blood pressure is too high due to acceleration artefact on the fluid columns of the recording system. However the general pattern of response can be seen.

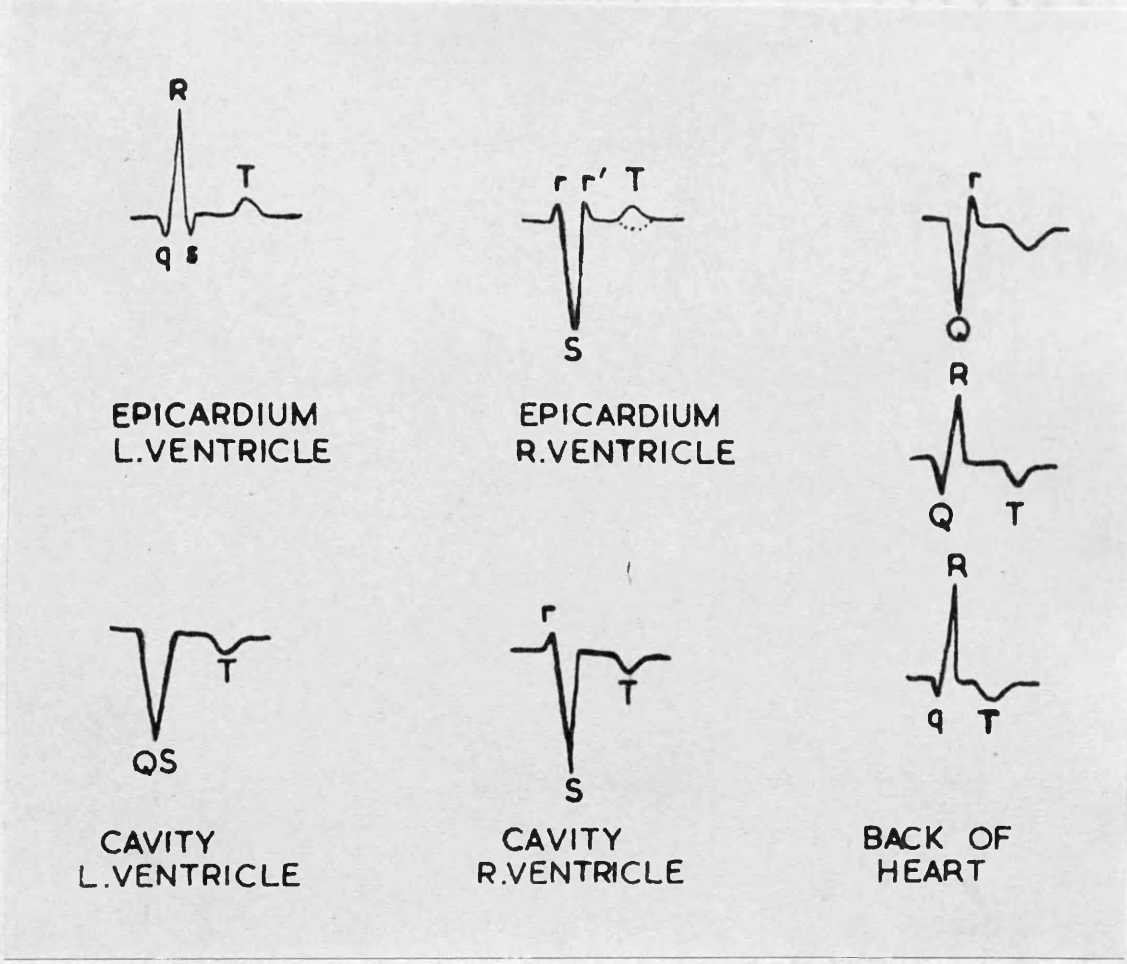


Fig. 12. The patterns obtained from various surfaces of the heart using unipolar leads. This diagram shows the use of small and capital letters in labelling the complexes. (After Goldberger 1953).

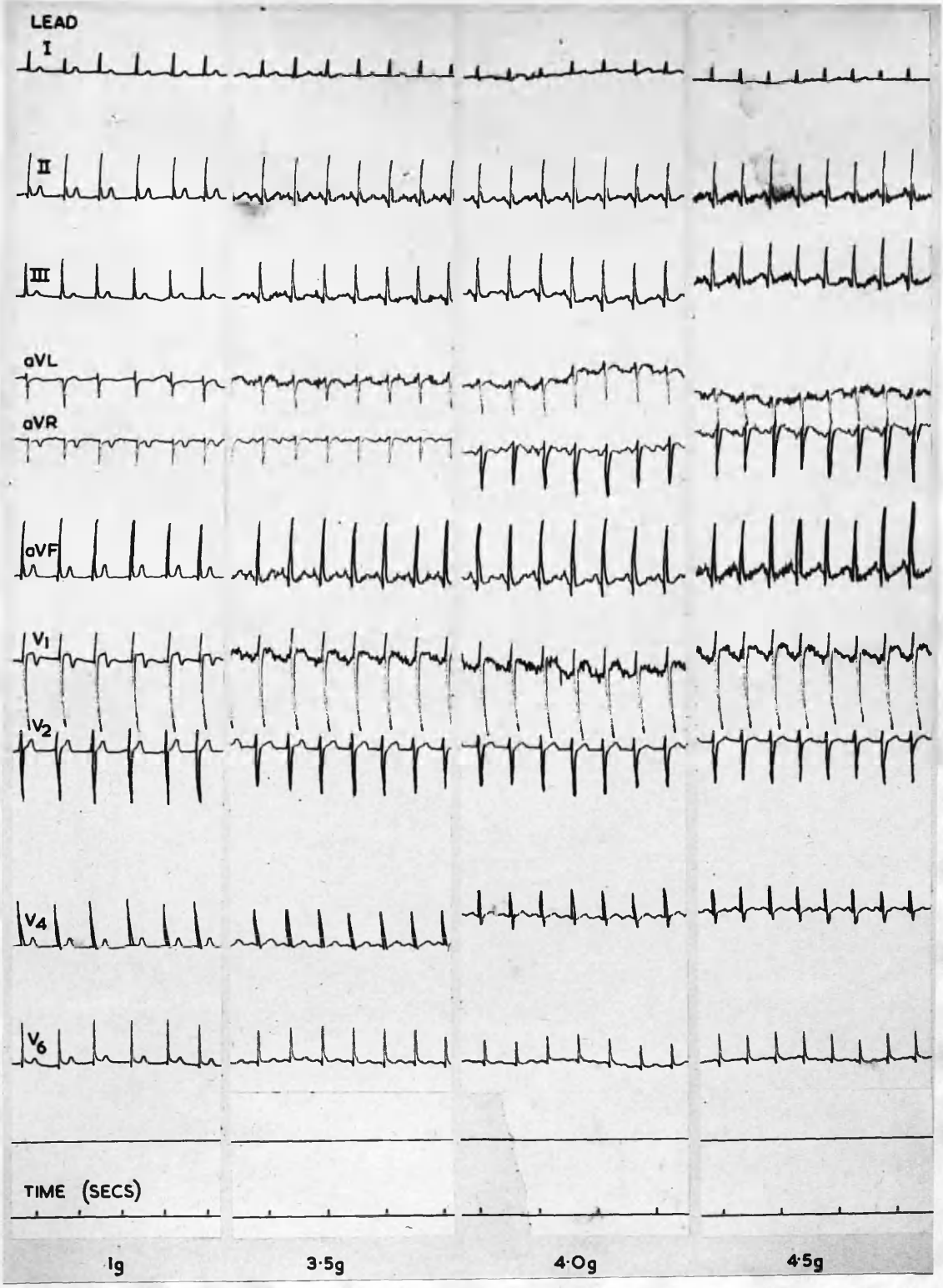


Fig. 13. Changes in the electrocardiogram under g. Note especially the changes in the amplitude of QRS in leads I and AVF, the large Q waves in leads II, III and AVF, and the changes in the T wave in the chest leads.

Fig. 14. Changes in the electrocardiogram during deep respiration.

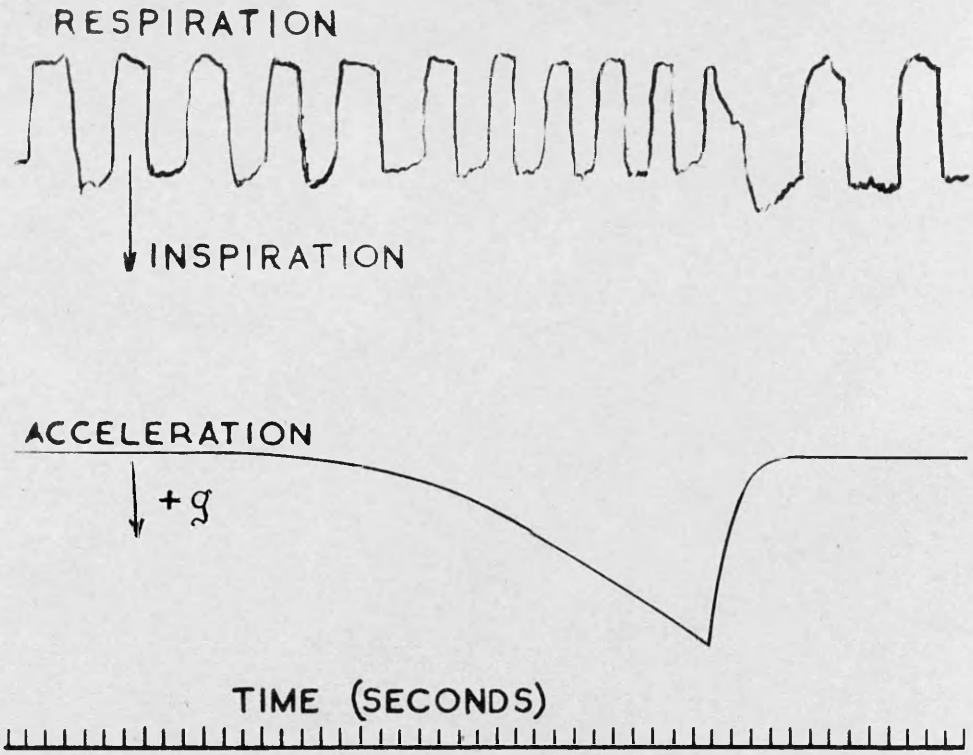


Fig. 15. A tracing of a record of respiration during g measured from a pressure tapping in an oxygen mask.

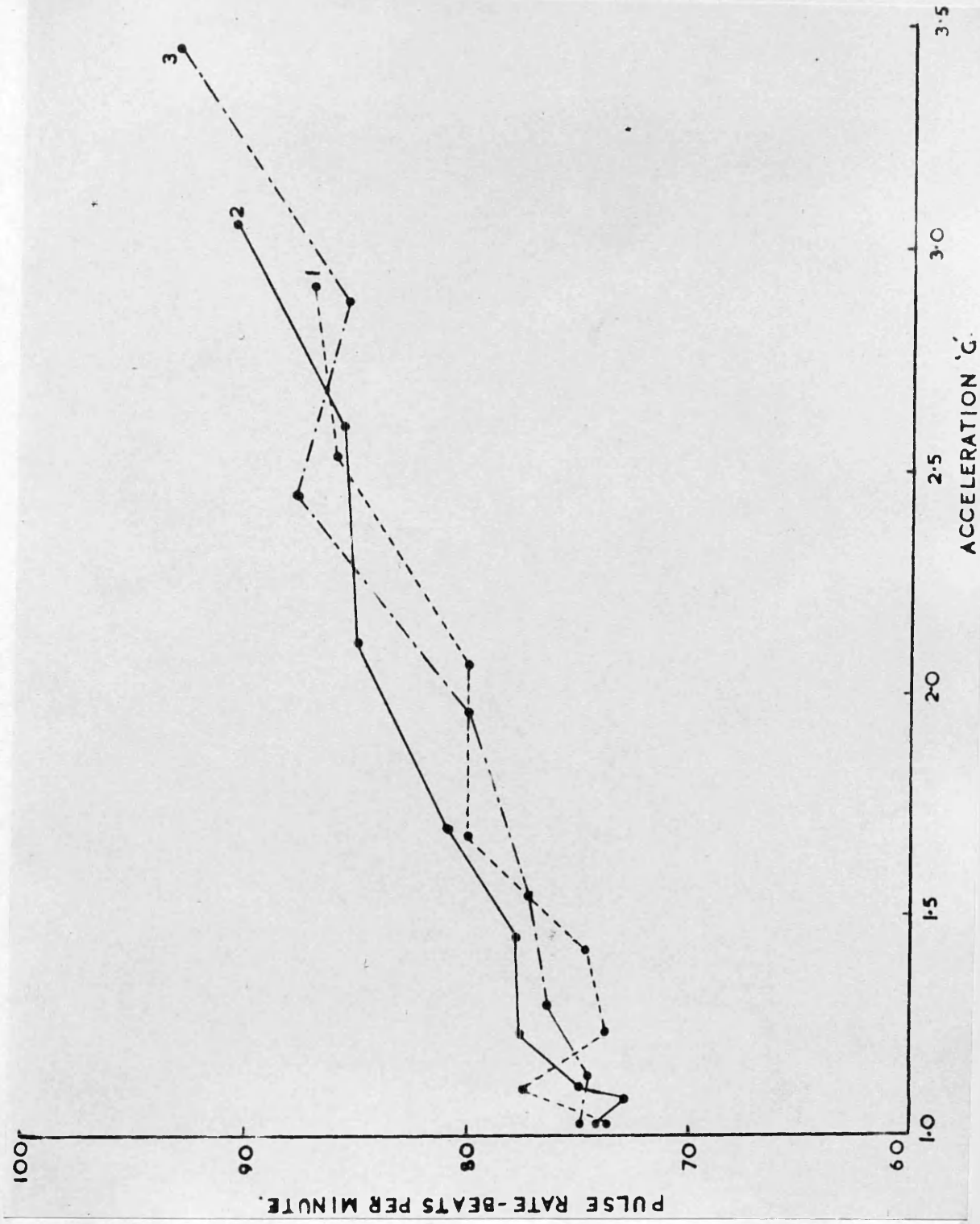


Fig. 16. Graph showing change in pulse rate when g is applied at 0.1 g/sec.

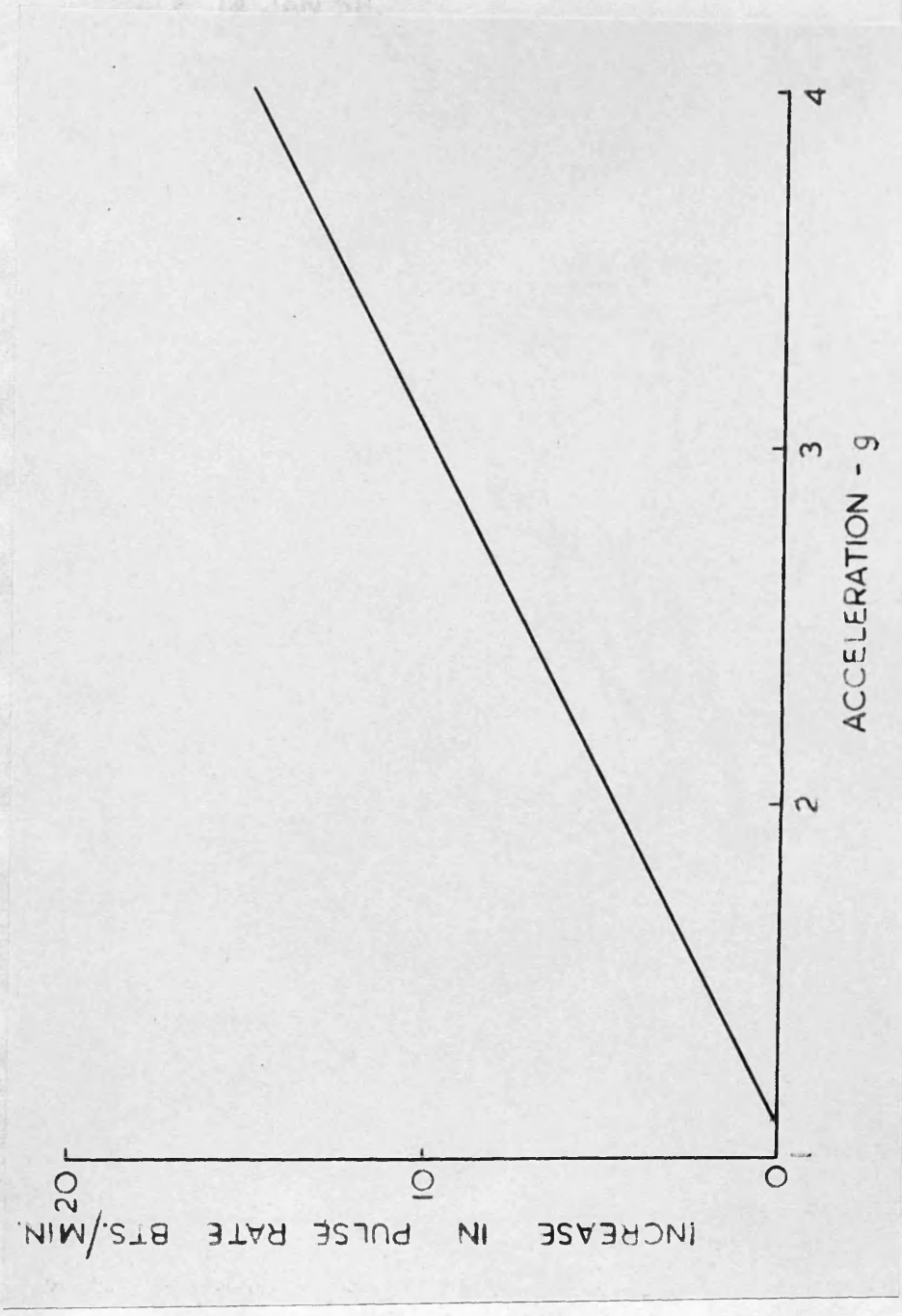


Fig. 17. Graph of increase in pulse rate with g. Derived from the results of 125 runs. The equation is Y = 6.369 + 5.0875 (x - 0.9618) and the coefficient b = 5.0875 (SE = ± 0.34)

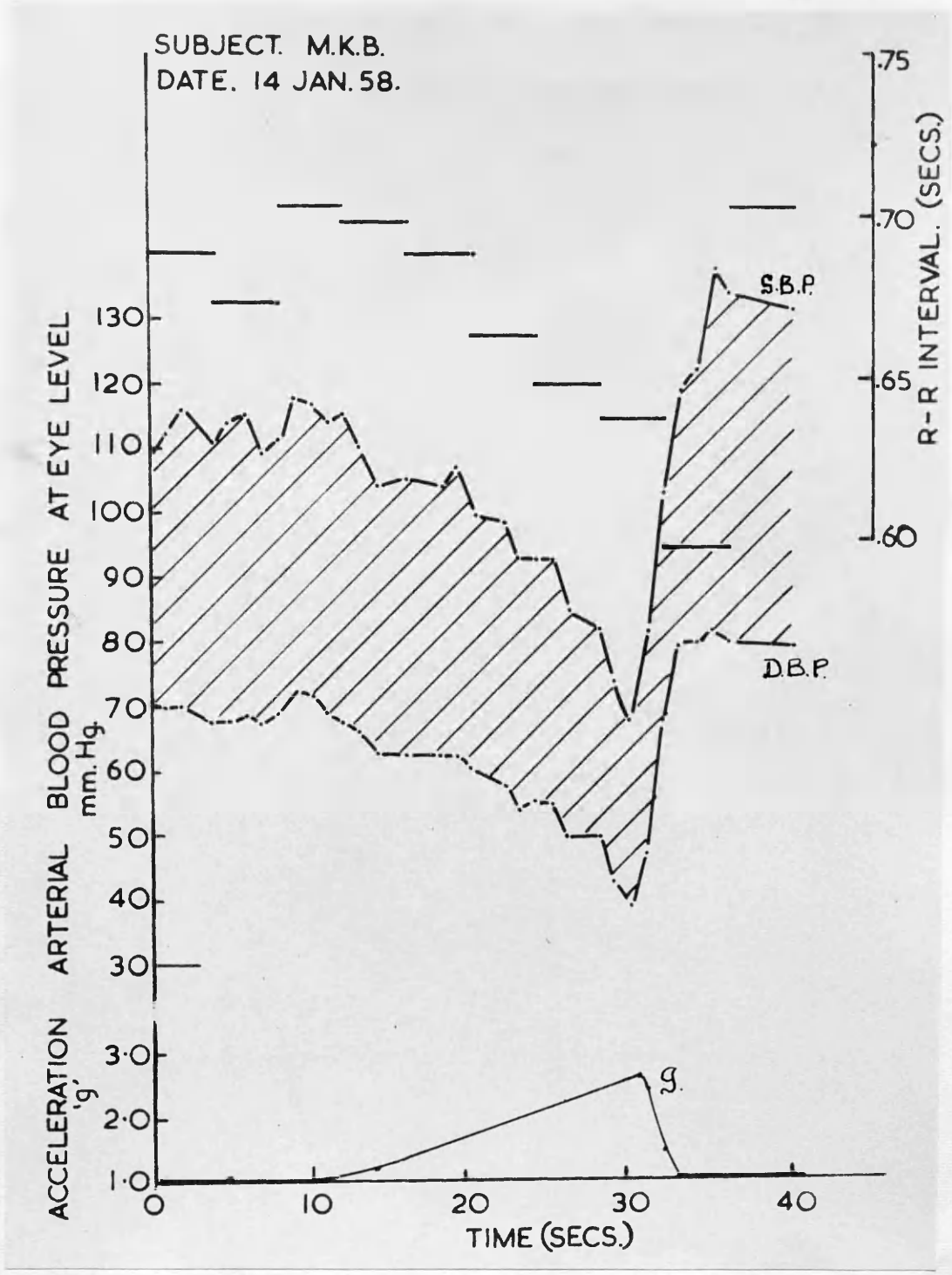


Fig. 18. Graph showing changes in blood pressure at eye level using a rate of application of acceleration of 0.1 g/sec. The change in pulse rate can be seen from theplot of the R-R interval obtained from the E.C.G.

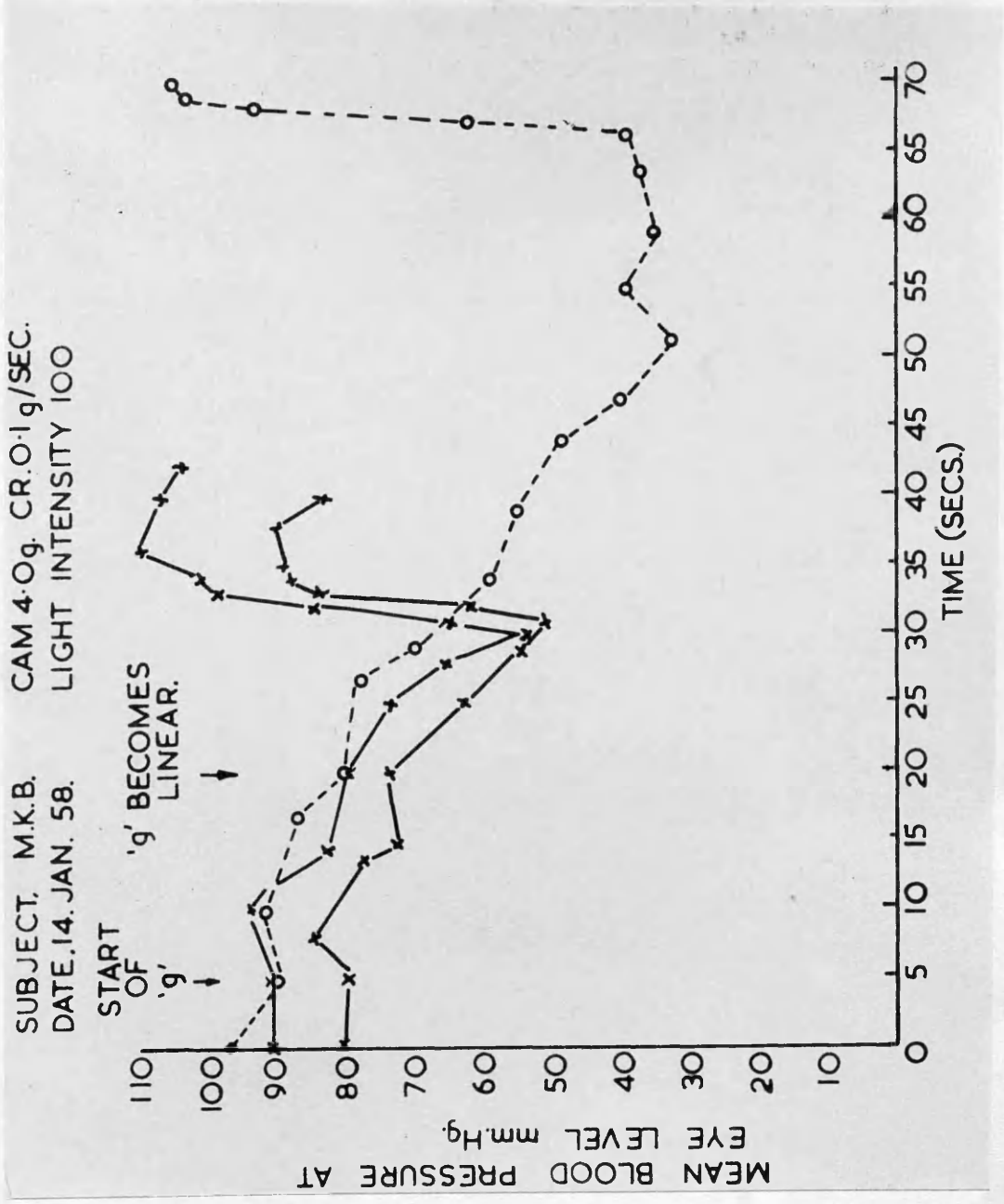


Fig. 19. Changes in the mean blood pressure at eye level during positive g applied at a rate of 0.1 g/sec. The B.P. is plotted against time of maintenance of increasing g.

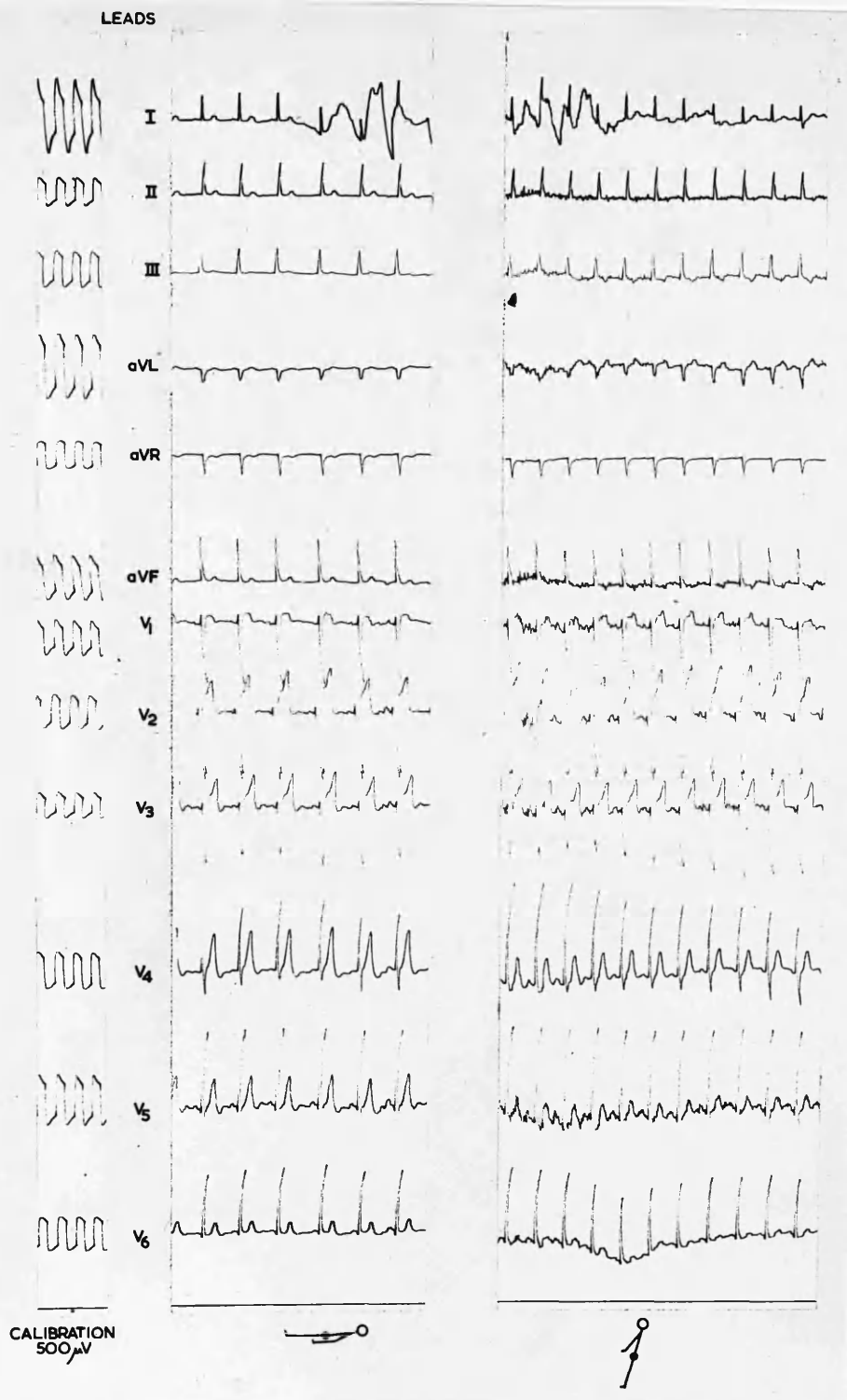


Fig. 20. Changes in the electrocardiogram occurring when a subject is tilted from the horizontal to 75°. Compare with fig. 13.

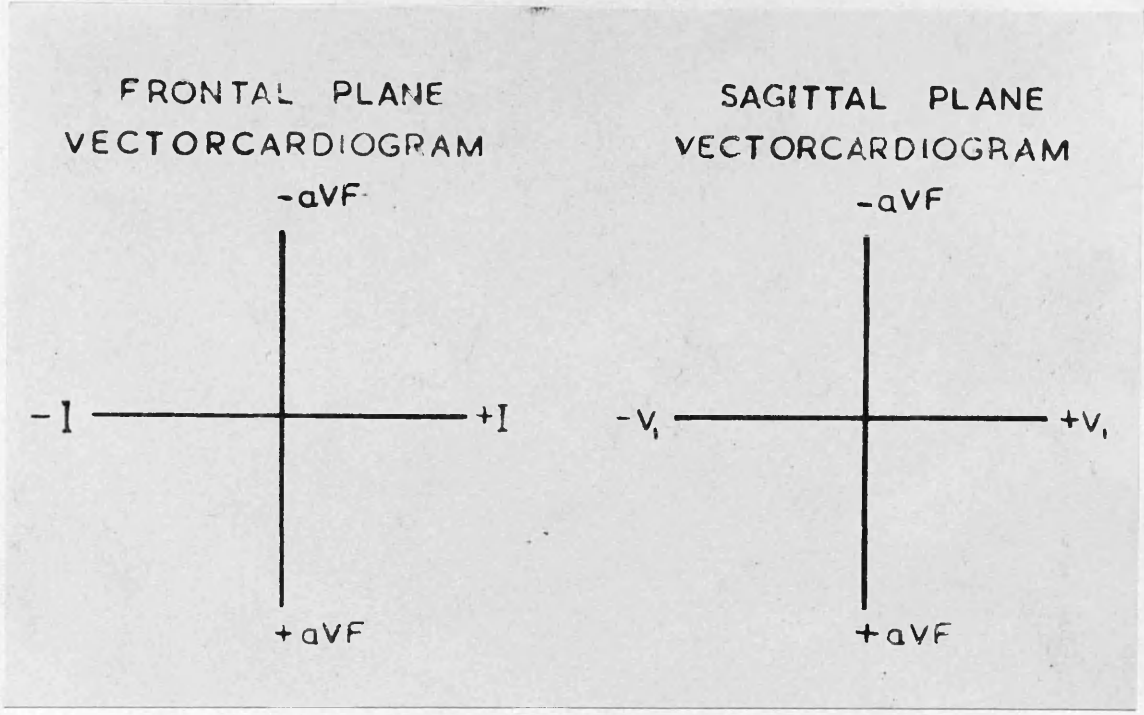


Fig. 21. The polarities which are conventionally ascribed to the leads in vectorcardiography and in the derivation of the electrical axis.

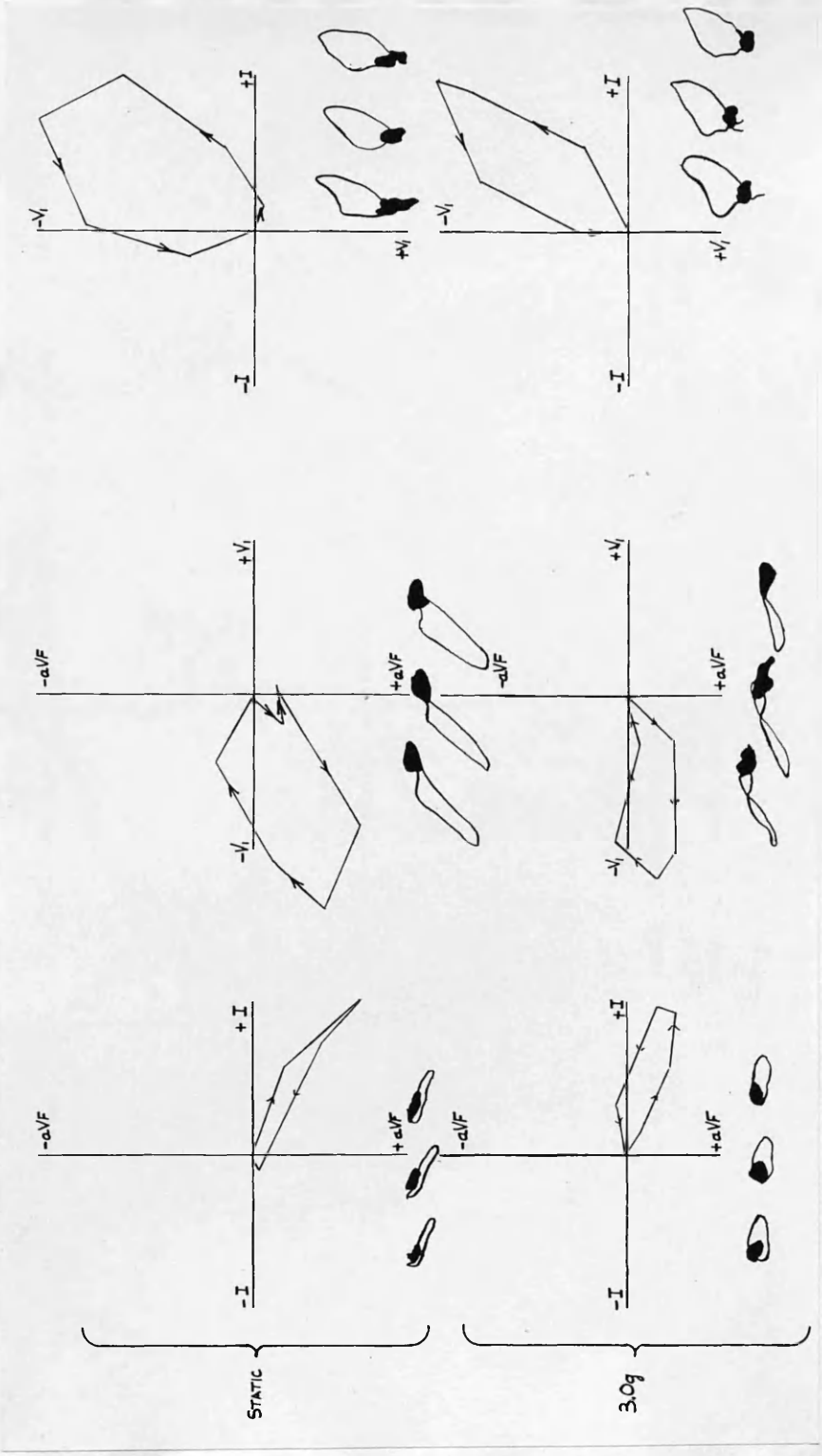


Fig. 22. Comparison of derived and direct vectorcardiograms before and during 3.0 g.

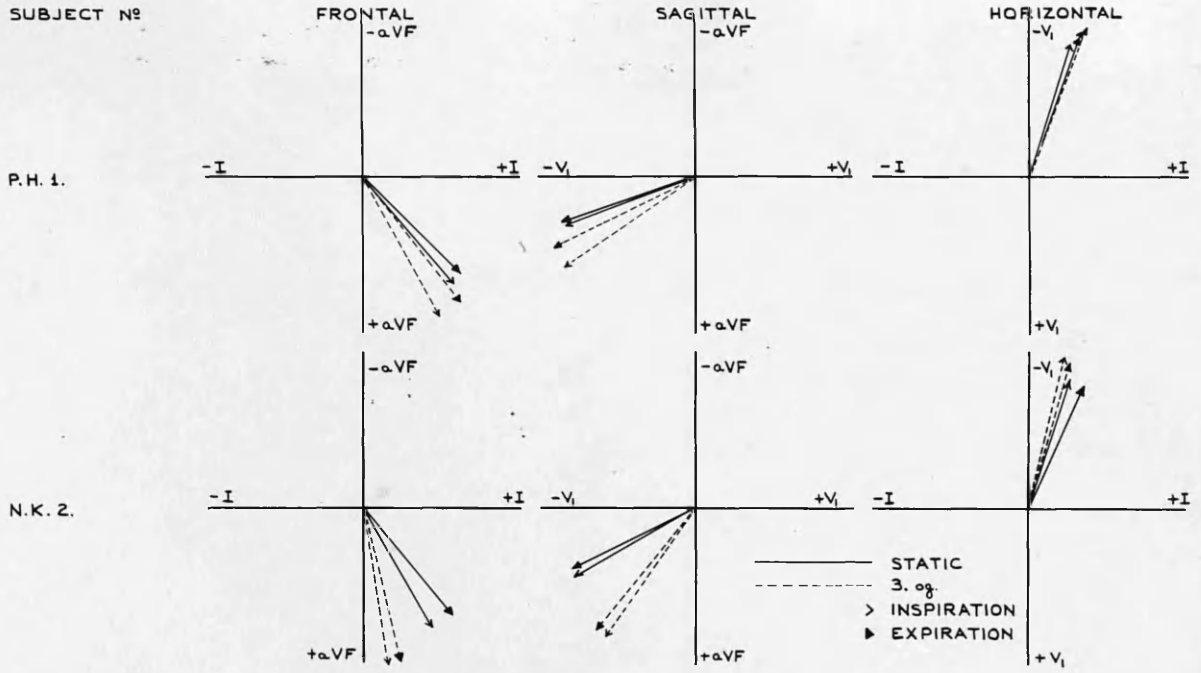


Fig. 23(a) to Fig. 23(d). Mean instantaneous electrical axis of the heart at rest and at 3.0 g. The respiratory change is also shown at rest and at 3.0 g.

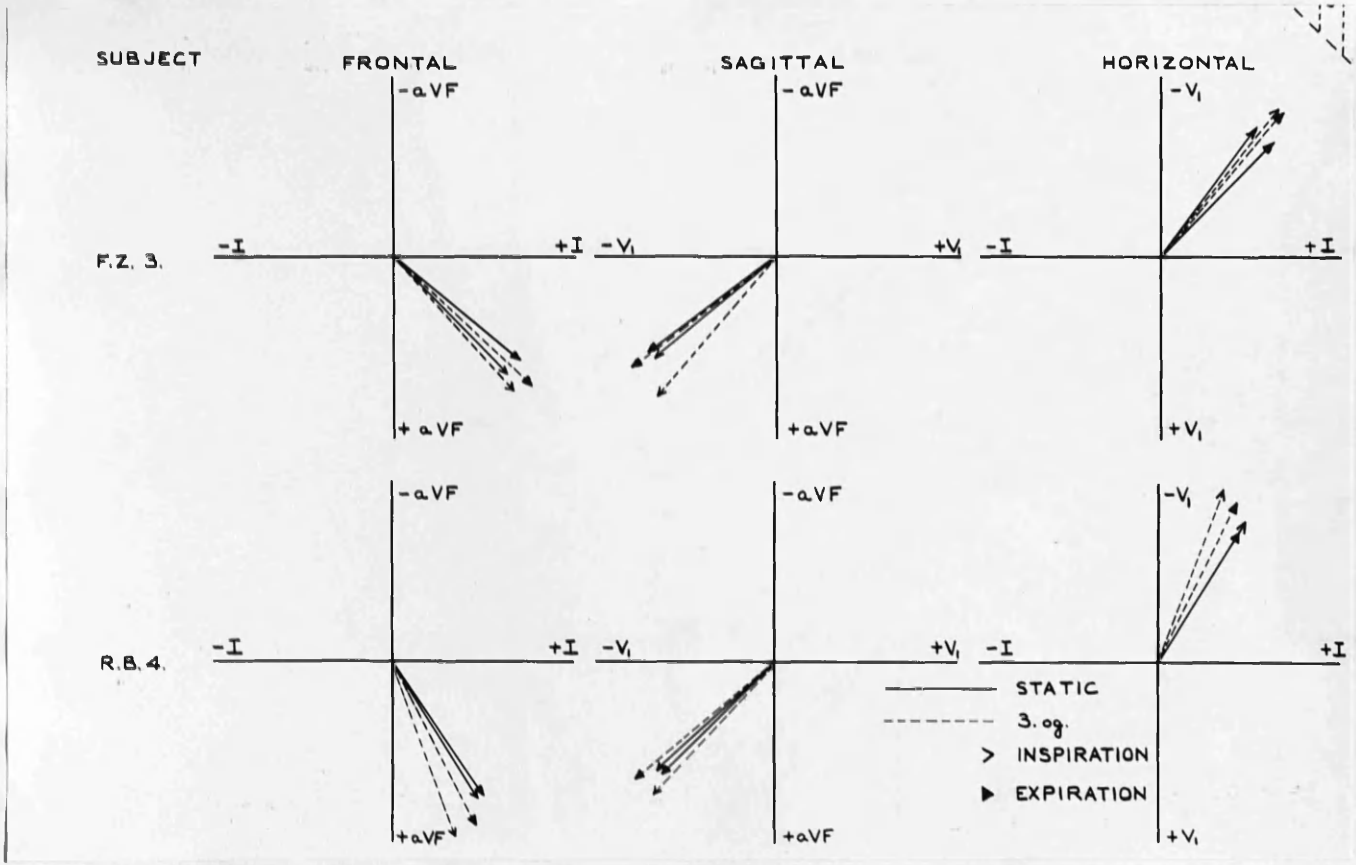

SUBJECT
FRONTAL
SAGITTAL
HORIZONTAL
F.Z. 3.
R.B. 4.
-aVF
+aVF
-I
+I
-V̇
+V̇
STATIC
3. og.
INSPIRATION
EXPIRATION

Fig. 23(b).

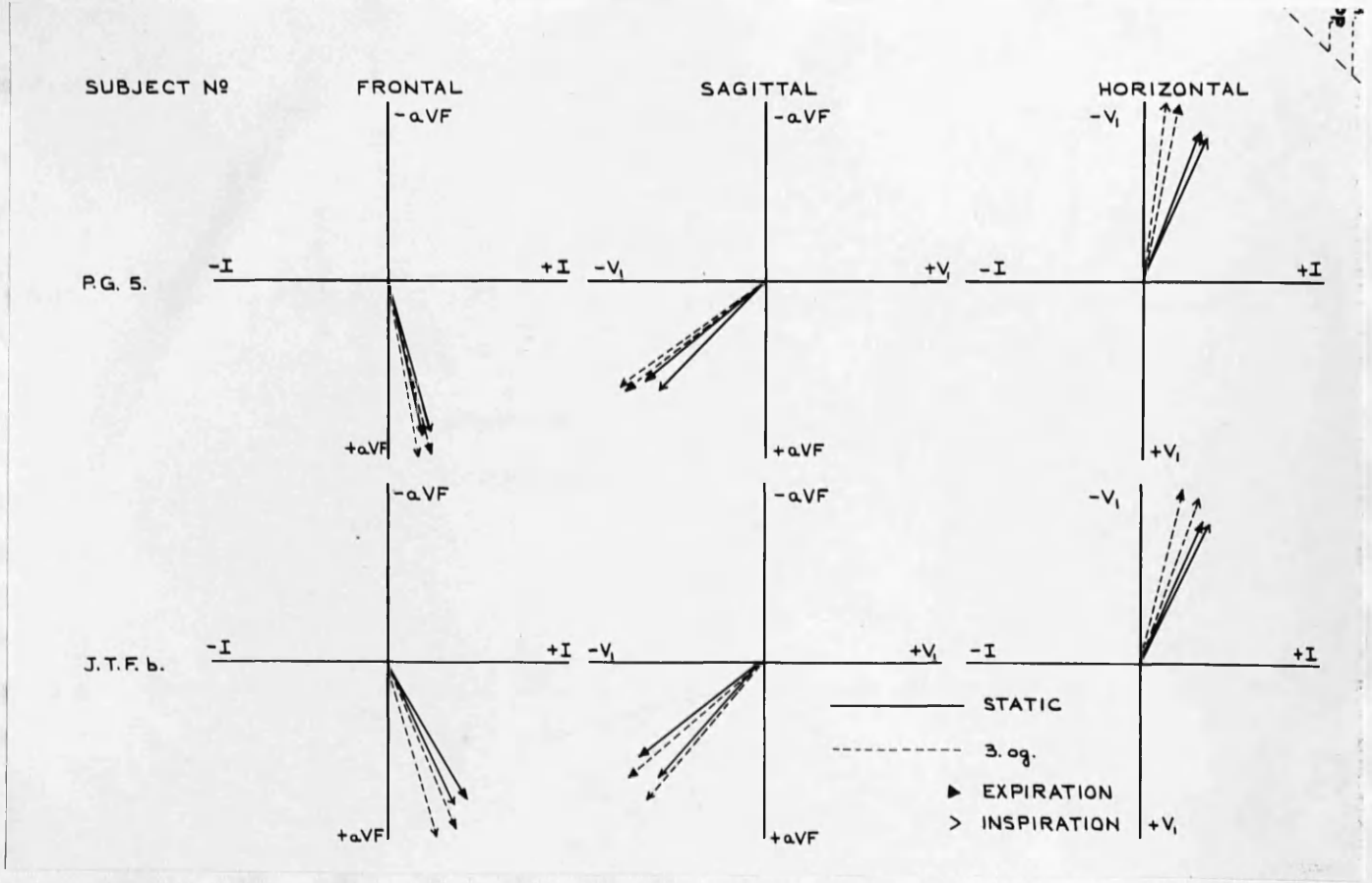

SUBJECT Nº
FRONTAL
SAGITTAL
HORIZONTAL
P.G. 5.
J.T.F. b.
-aVF
+aVF
-I
+I
-V1
+V1
STATIC
3. og.
EXPIRATION
INSPIRATION

Fig. 23(c).

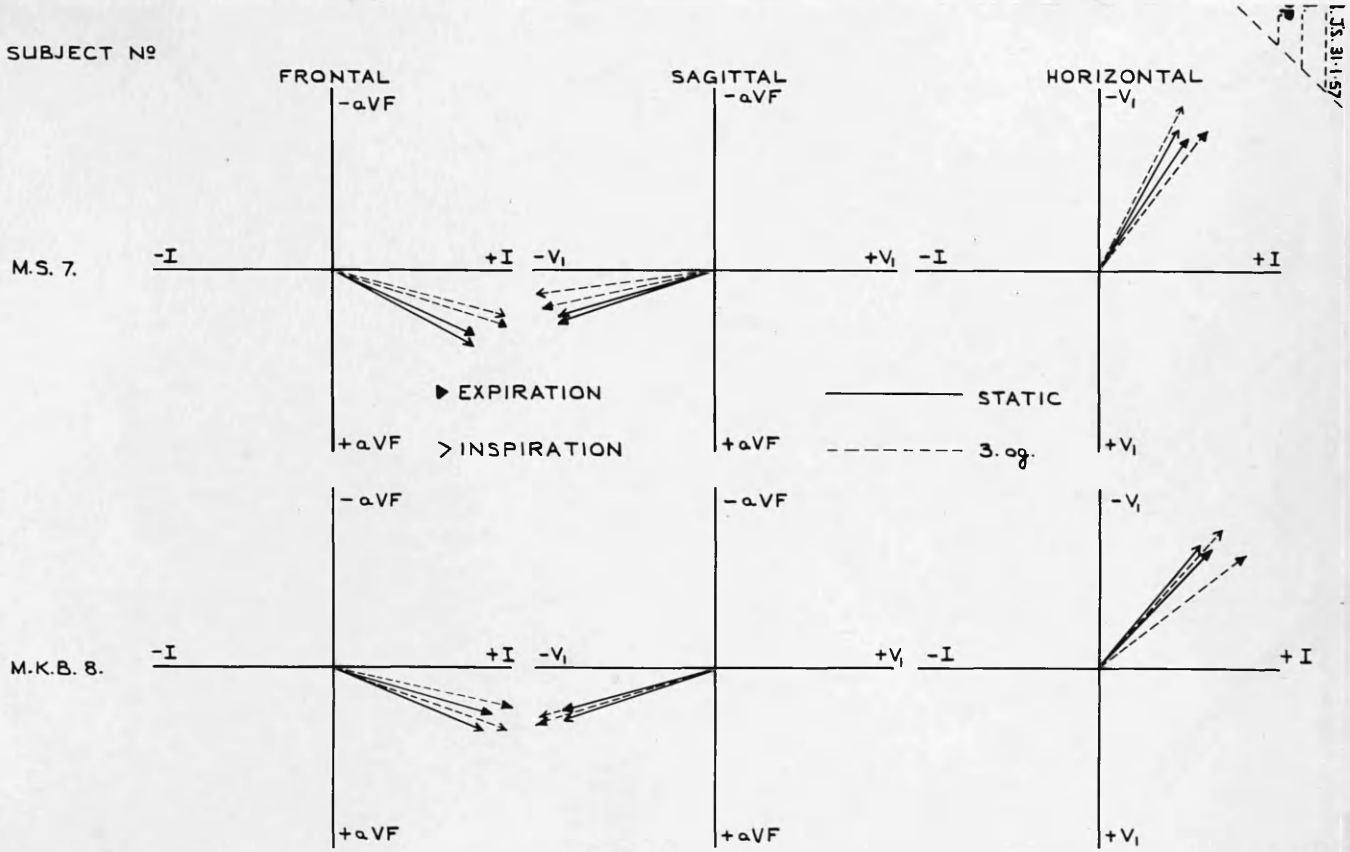

SUBJECT Nº
FRONTAL
SAGITTAL
HORIZONTAL
-aVF
+aVF
-V1
+V1
-I
+I
M.S. 7.
M.K.B. 8.
EXPIRATION
INSPIRATION
STATIC
3.09.

Fig. 23(d).

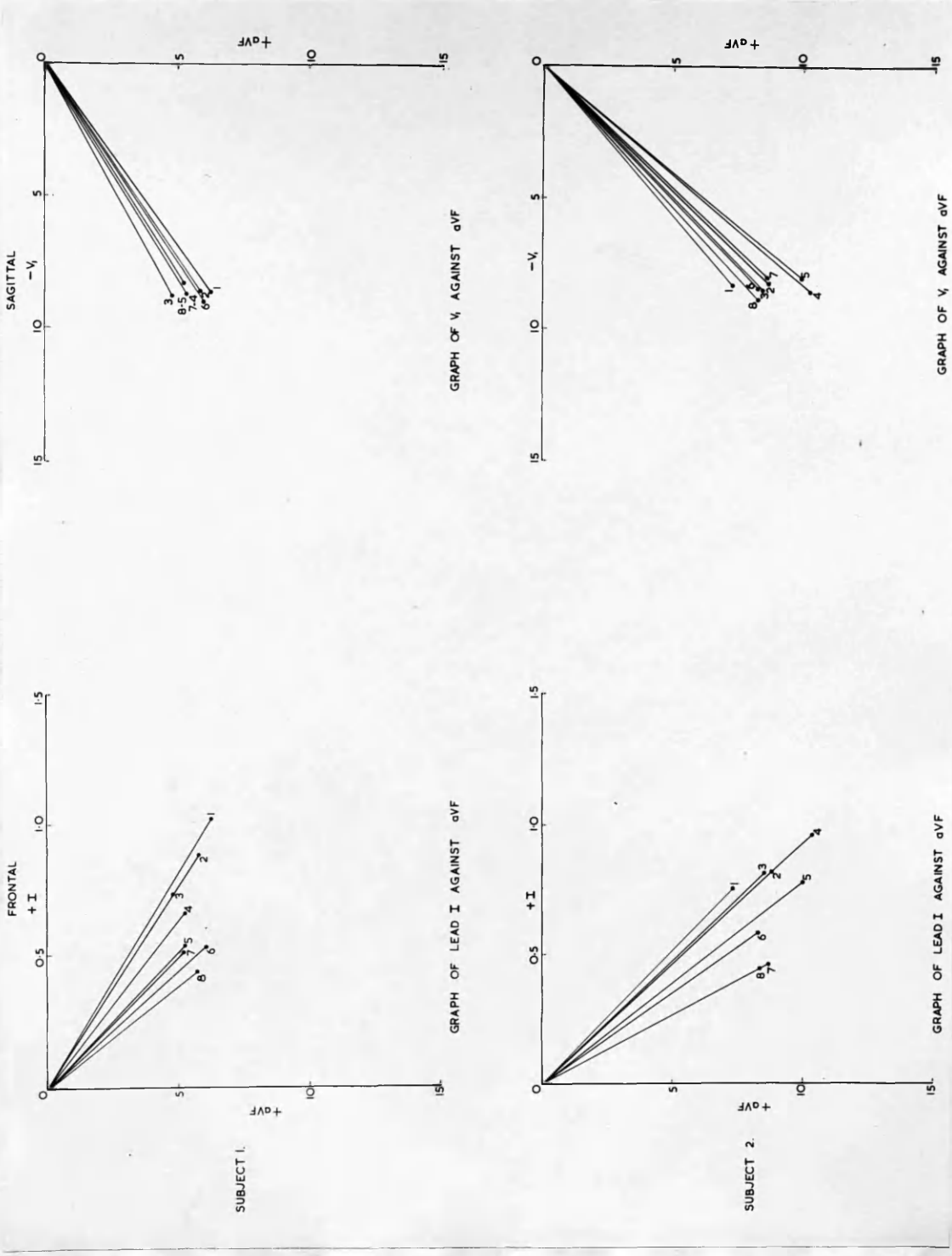
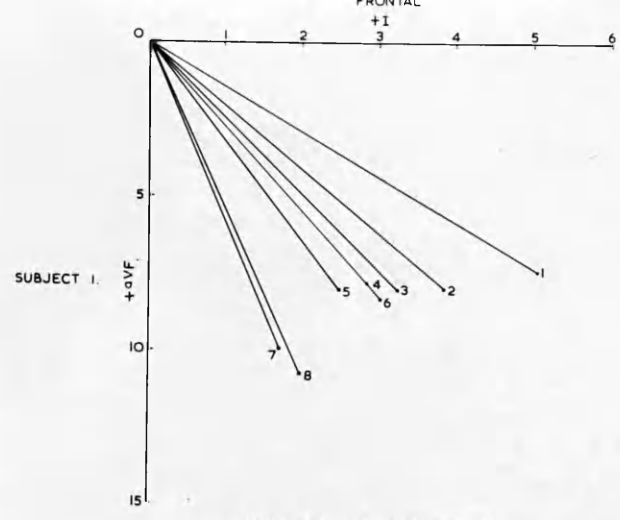
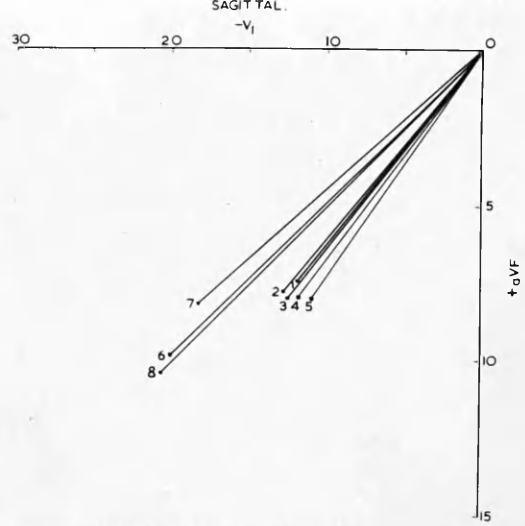


Fig. 24(a) to 24(b). Changes in the manifest instantaneous electrical axis of the heart with increasing g.

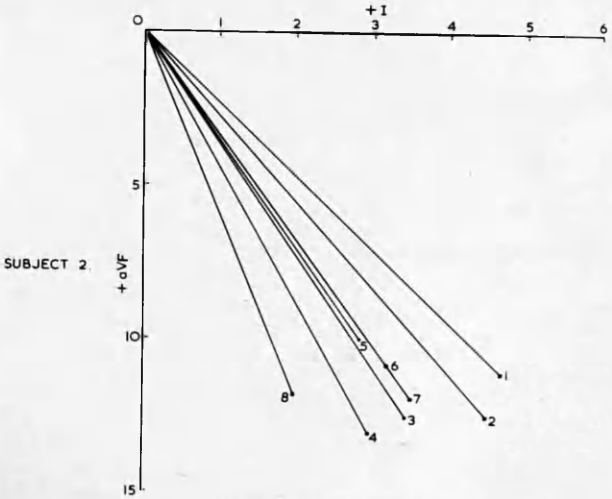
Fig. 24(b).



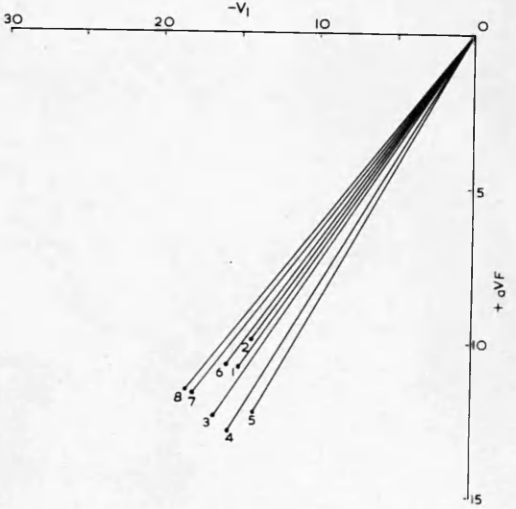
GRAPH OF LEAD I AGAINST aVF



GRAPH OF LEAD V_1 AGAINST aVF



GRAPH OF LEAD I AGAINST aVF



GRAPH OF LEAD V_1 AGAINST aVF

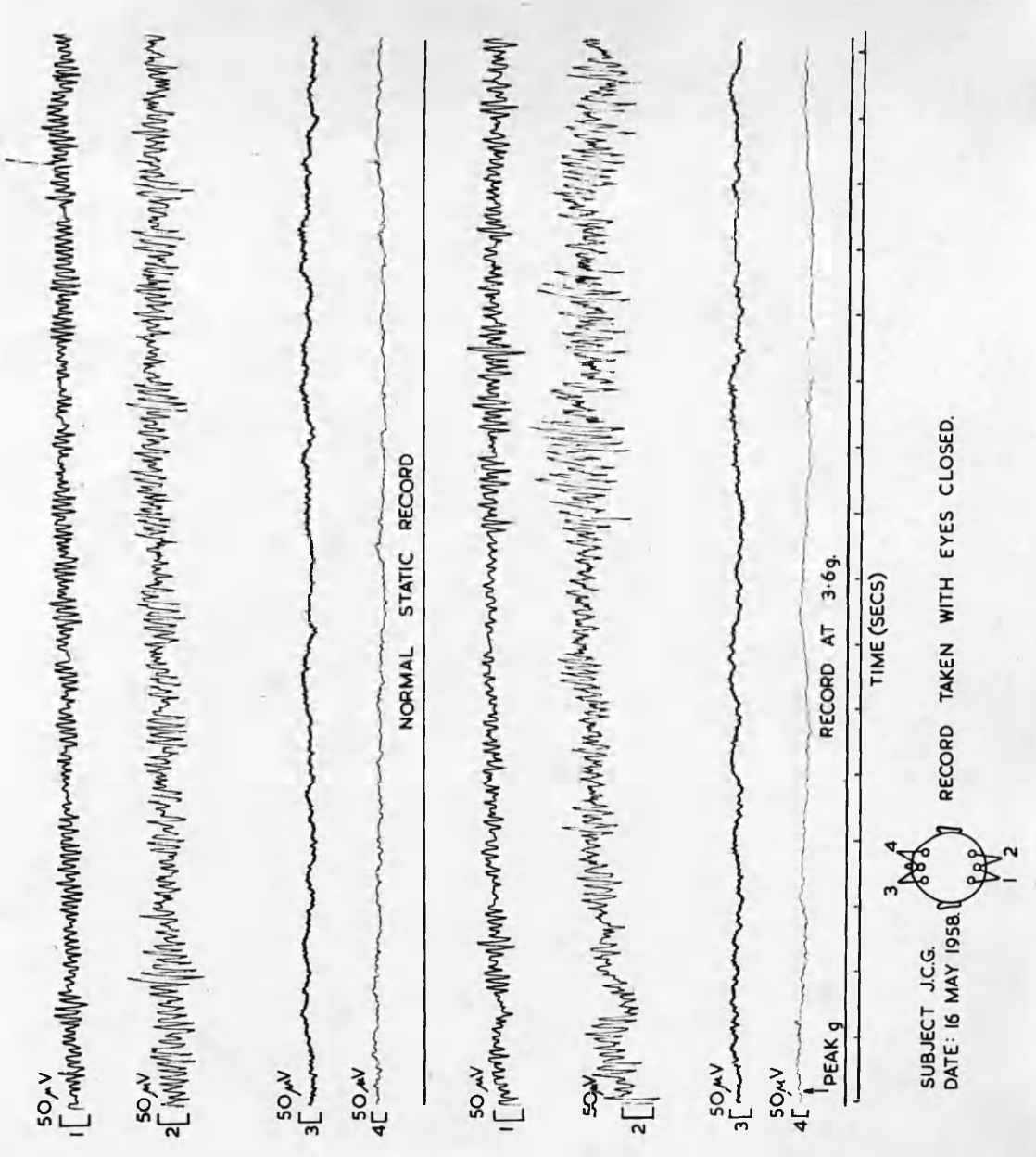


Fig. 25. The electro encephalogram during positive acceleration. The upper trace is taken with the centrifuge at rest. The lower trace is at 3.6 g. The diminished alpha-rhythm which occurs at peak g can be easily seen.

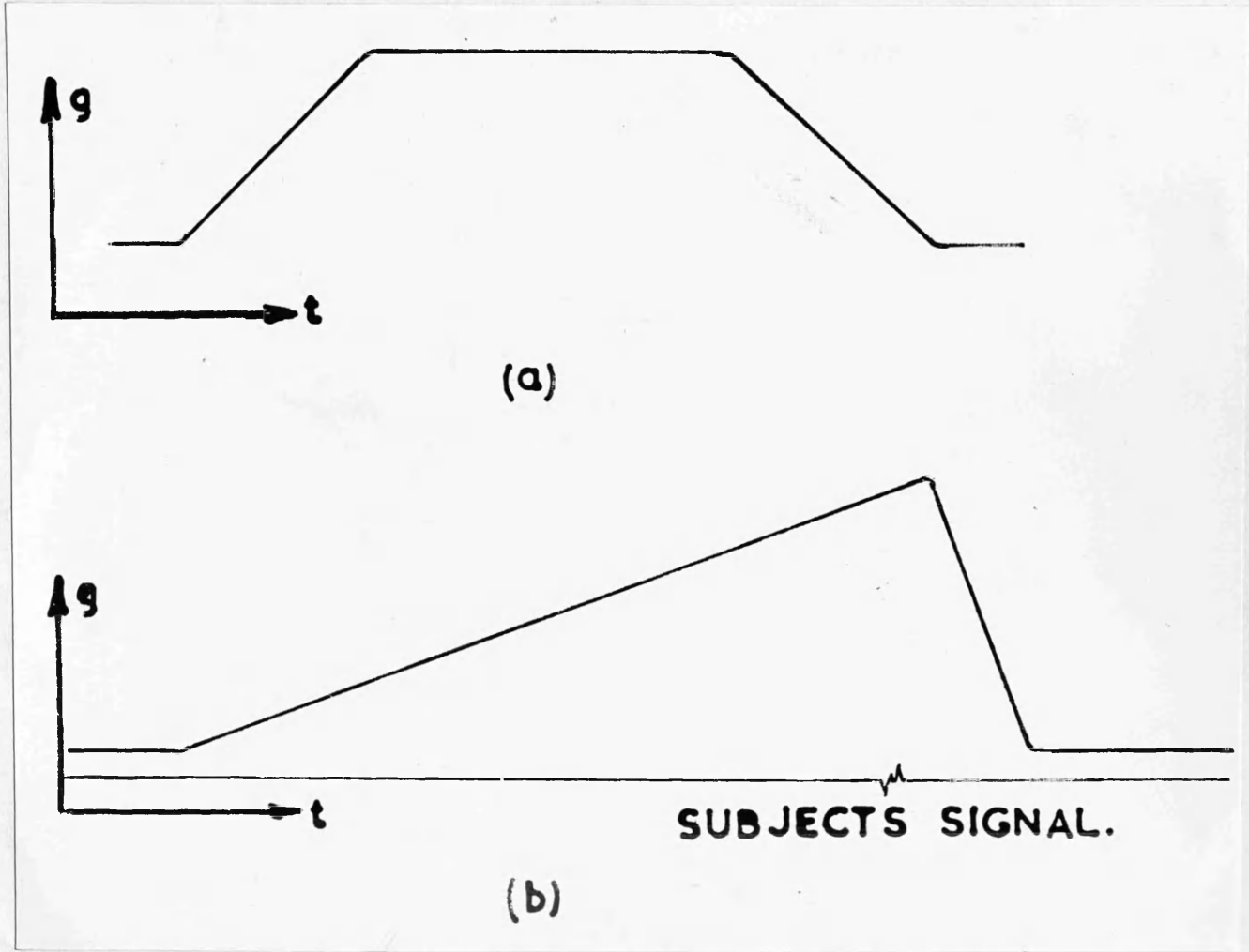


Fig. 26. Acceleration - time relationship of threshold runs.

END-POINT.	LOSS OF PERIPHERAL VISION."GREY-OUT"	
	LOSS(OR IMPAIRMENT) OF CENTRAL VISION."BLACK-OUT."	
	DURATION OF LOSS AND TIME OF OCCURRENCE.	
SIGNAL-LIGHTS.	ARRANGEMENT.	- VISUAL ANGLE SUBTENDED.
		- HEIGHT RELATIVE TO EYE LEVEL.
	LAMPS.	- SIZE.
		- COLOUR.
		- INTENSITY.
	BACKGROUND,	- UNIFORMITY.
		- ILLUMINATION.
AMBIENT ILLUMINATION.	UNIFORMITY.	
	BRIGHTNESS.	
	COLOUR.	
	LIGHTS IN GONDOLA.	
	LIGHTS IN ROOM.	
TYPE OF RUN.	RATE OF APPLICATION OF "g".	
	MAXIMUM TIME AT PEAK.	
	INTERVAL BETWEEN SUCCESSIVE RUNS.	
SUBJECT.	POSITION OF LIMBS AND BODY.	
	DEGREE OF ACTIVITY.	- VERBAL REPORTING.
		- MANUAL SIGNALS.

Fig. 27. The variables which should be controlled in any acceleration experiment, and should be described in any communication on acceleration research. (Howard 1957).

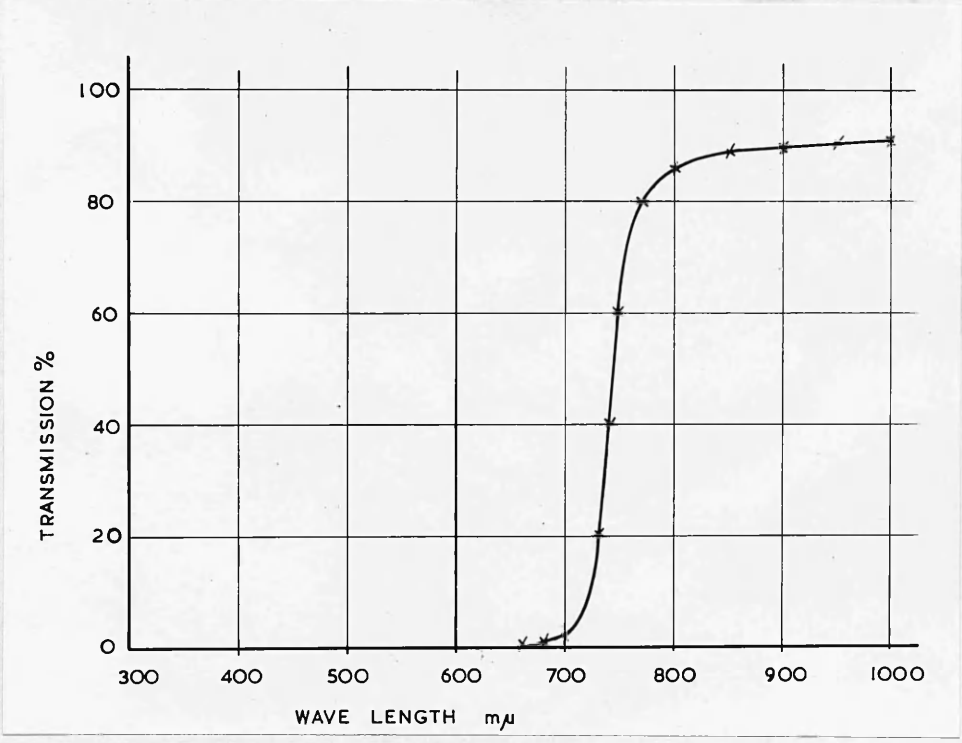


Fig. 28. Characteristics of the type 6 R.A.F. infrared filter used in the technique of threshold measurement.

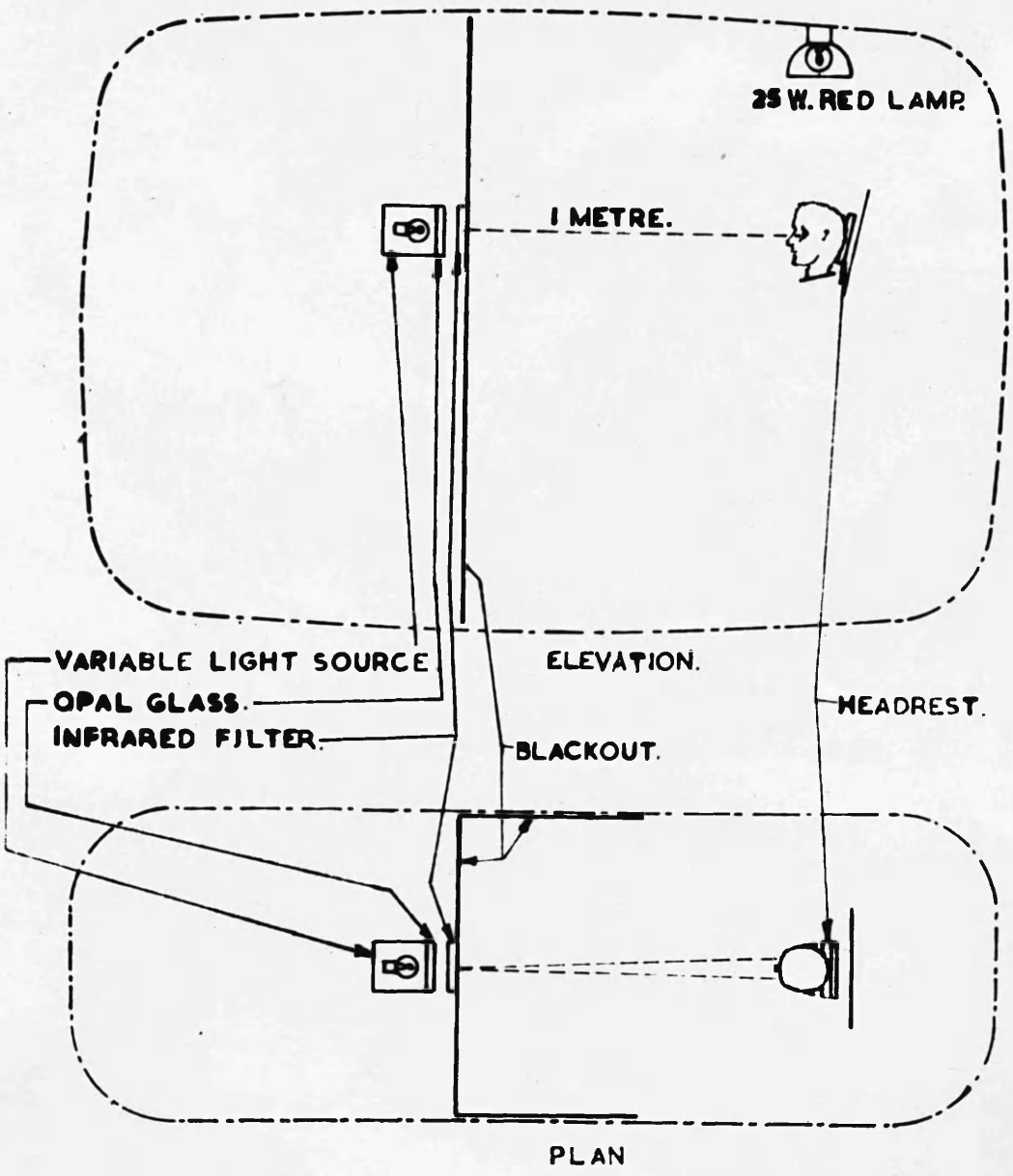


Fig. 29. Diagrammatic representation of the apparatus in the gondola when used for threshold determination.

END POINT. BLACK OUT.
WHEN FIRST OCCURS.

SIGNAL LIGHTS. CENTRAL SUBTENDING ~ 1·5 MINS. OF ARC.
AT EYE LEVEL.
LAMPS — 3/16" DIA
— RED — 780mμ
— 0·5 LOG UNITS ABOVE ABSOLUTE
VISUAL THRESHOLD.
BACKGROUND - EMPTY - BLACK.

AMBIENT ILLUMINATION. CENTRIFUGE CHAMBER IN DARKNESS.
CAR LIT BY 25 W. 250V. RED LIGHT THROUGH
APERTURES 2" SQUARE PROJECTED ON TO
SUBJECTS HEAD.

TYPE OF RUN. 0·1 g/SEC· LINEAR FROM $\sqrt{2g}$ WITH NO PLATEAU.
3 MINS. BETWEEN RUNS TO ALLOW PULSE TO
FALL TO NORMAL VALUE.

SUBJECT. SEATED IN CHAIR RAKED 20°, KNEES BENT,
HANDS ON LAP.

SUBJECT RELAXED, SILENT, ONLY SIGNAL BEING
PUSH BUTTON TO SIGNAL END OF RUN.

Fig. 30. A statement of the variables in the method of threshold determination, laid out in the scheme suggested by Howard (1957).

PART II

THE EFFECTS OF OTHER PHYSIOLOGICAL STRESSES ON MAN'S TOLERANCE TO INCREASED GRAVITATIONAL FORCE+

Fig. 1. Graph showing the rise in cabin temperature of a Canberra aircraft standing on the tarmac on a sunny day at Khartoum.

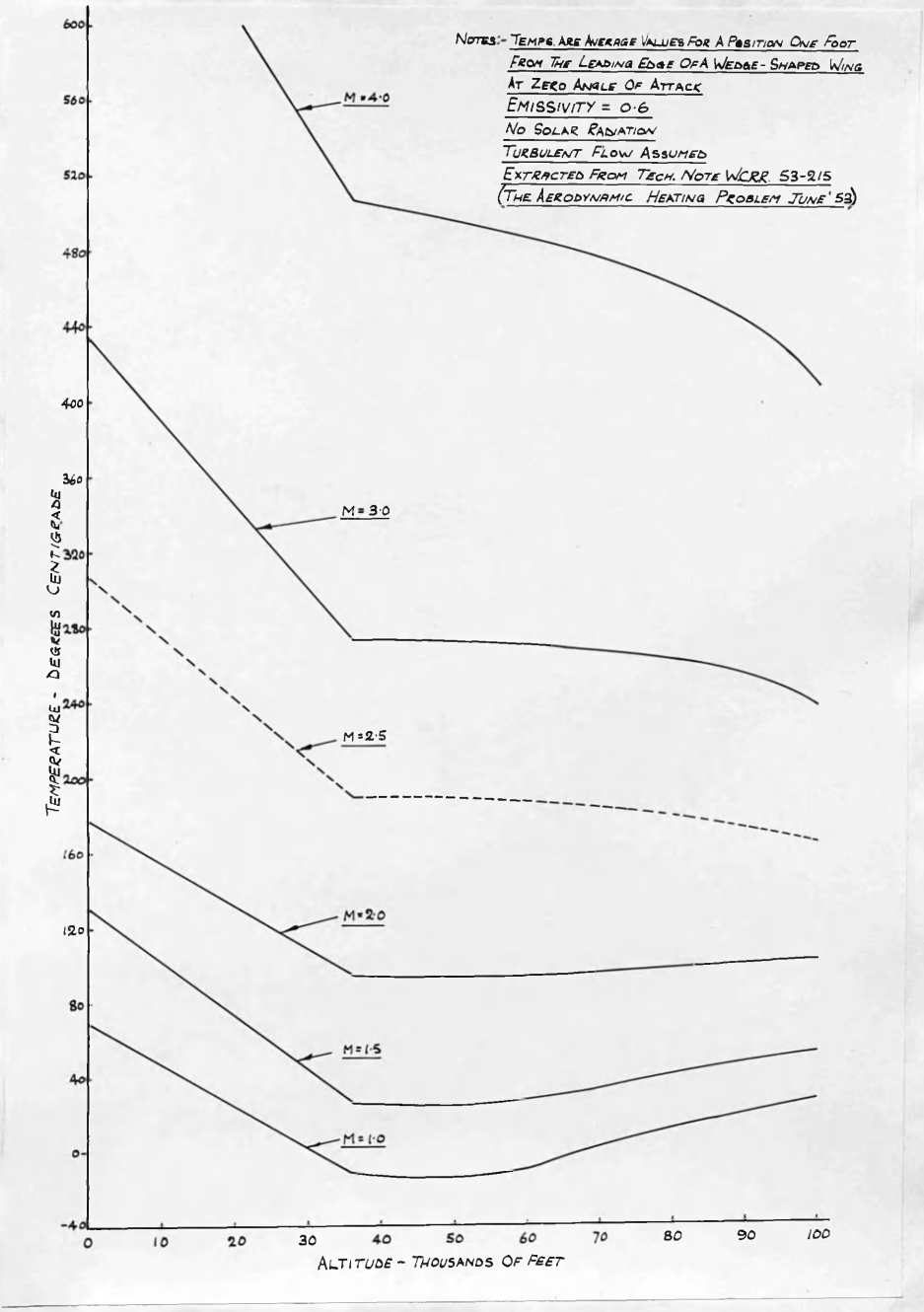


Fig. 2. Graph showing the average equilibrium skin temperate of an aircraft flying at various speeds and altitudes.

Plate I. Subject instrumented for heat run.

Plate II. Subject for heat run clad in polythene overall and boots, with rubber gloves. This assembly which is tight fitting at the neck effectively prevents evaporation of sweat from the majority of the body surface.

Plate III. Subject for heat run. The final stage of the clothing assembly is a type H electrically heated suit, flying gauntlets and sheepskin lined flying boots.

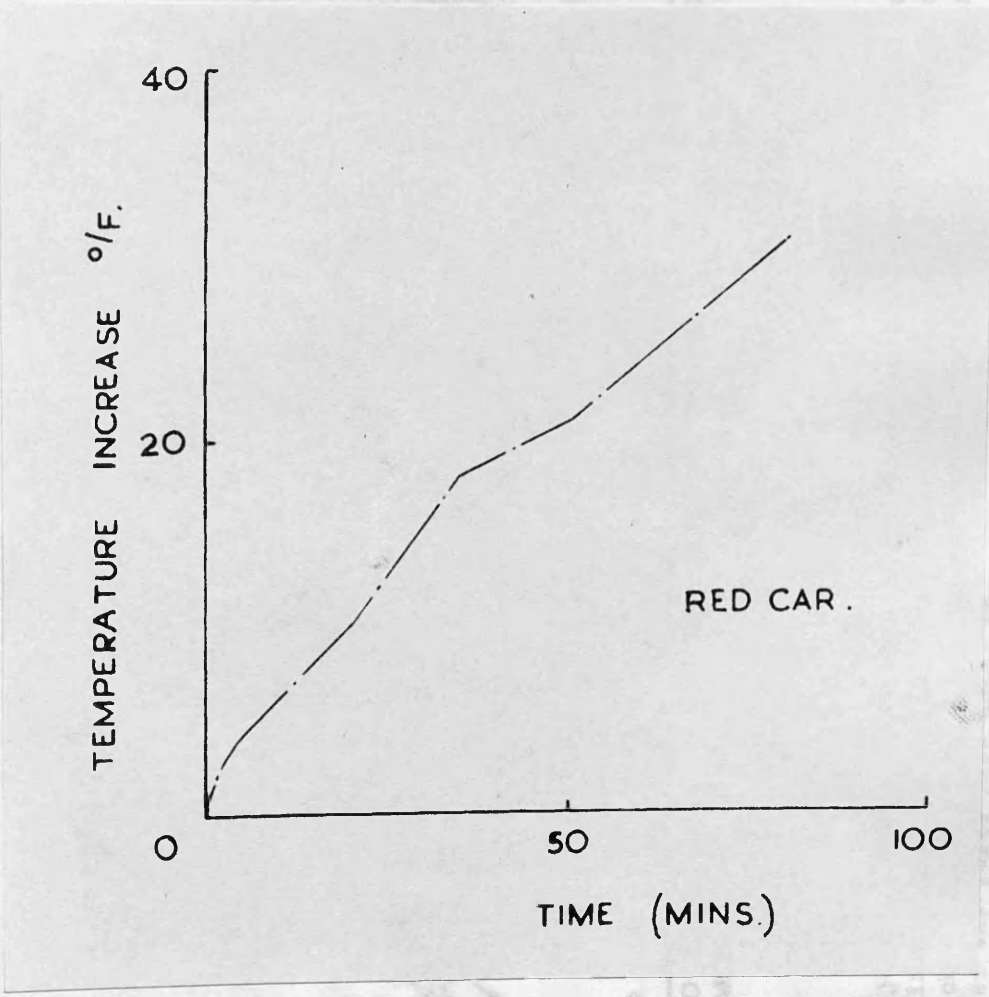


Fig. 3. Graph showing the rise in ambient temperature of the gondola with the convector and infrared heaters in operation.

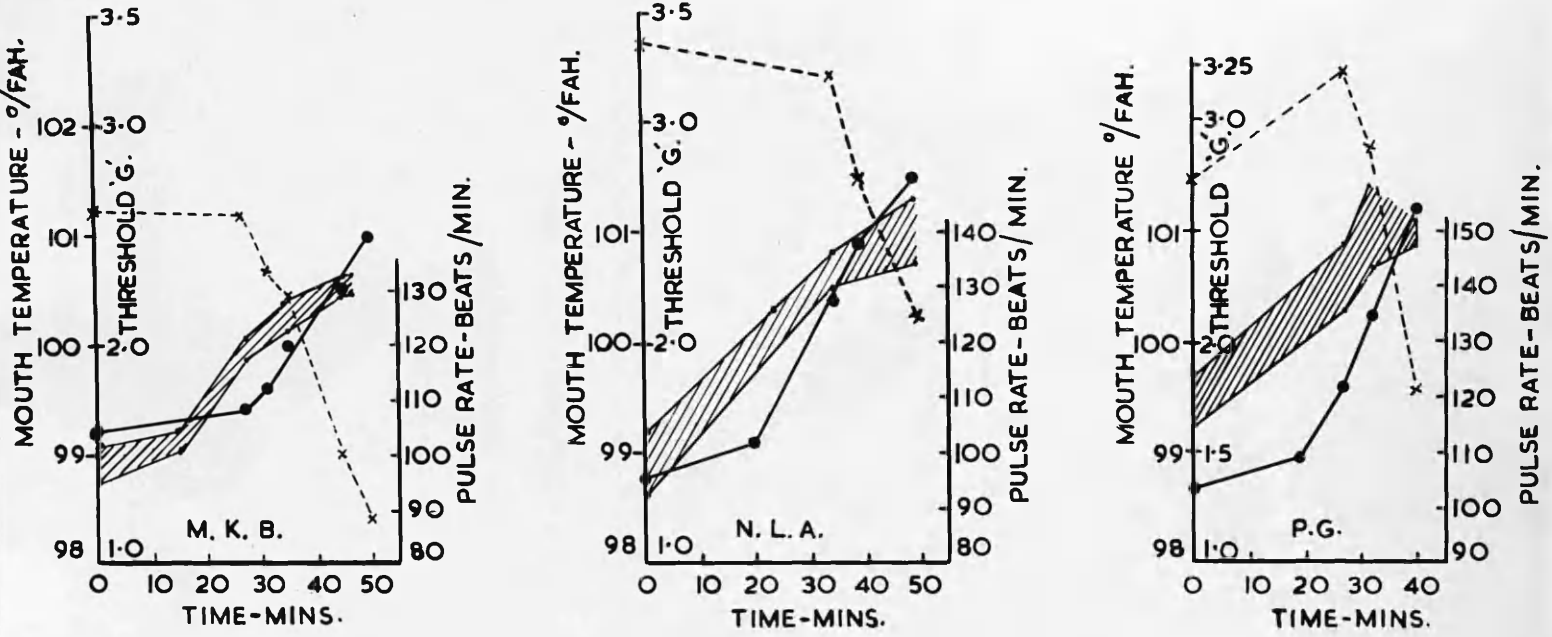


Fig. 4. Subjects of Group I. Graphs of mouth temperature, threshold, and pulse rate against time. Solid line temperature, interrupted line, threshold cross hatched area - pulse rate, the lower line represents resting pulse rate, the upper the pulse rate under g.

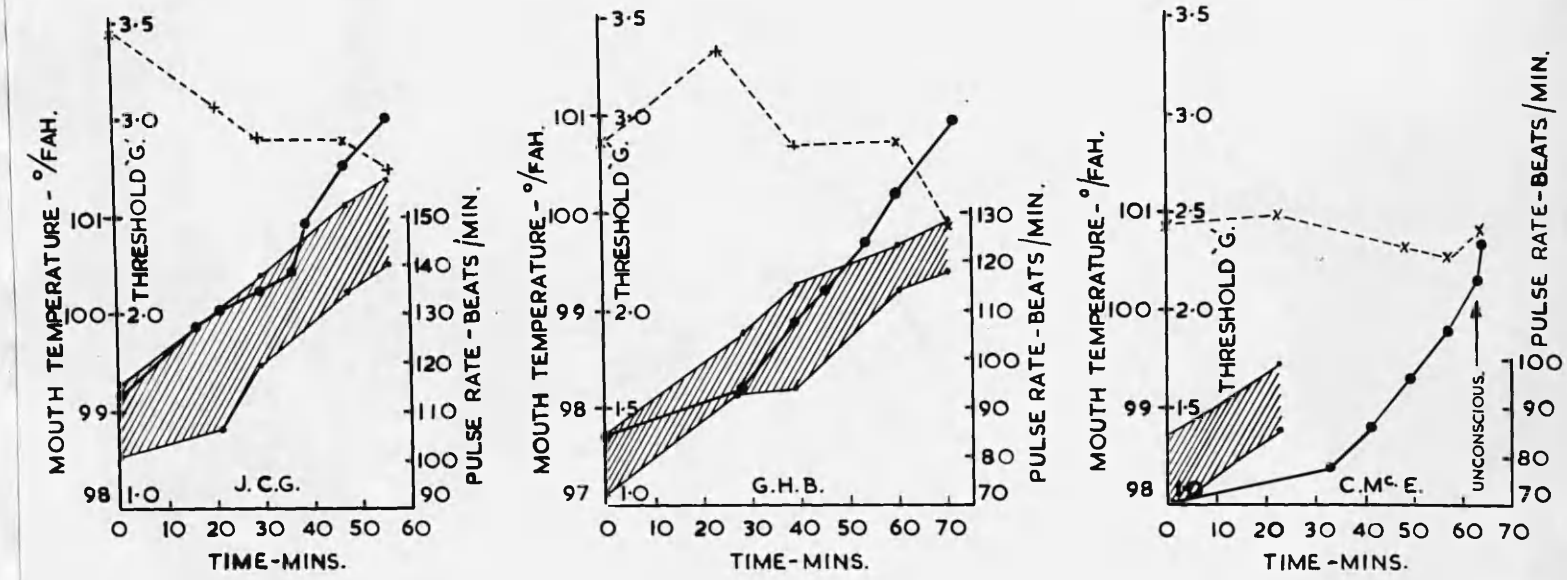


Fig. 5. Subjects of Group II. Graphs of mouth temperature (solid line), threshold (interrupted line) and pulse rate (crosshatched area) against time. The lower line of the pulse rate area represents resting pulse rate, the upper line the pulse rate under *g*.

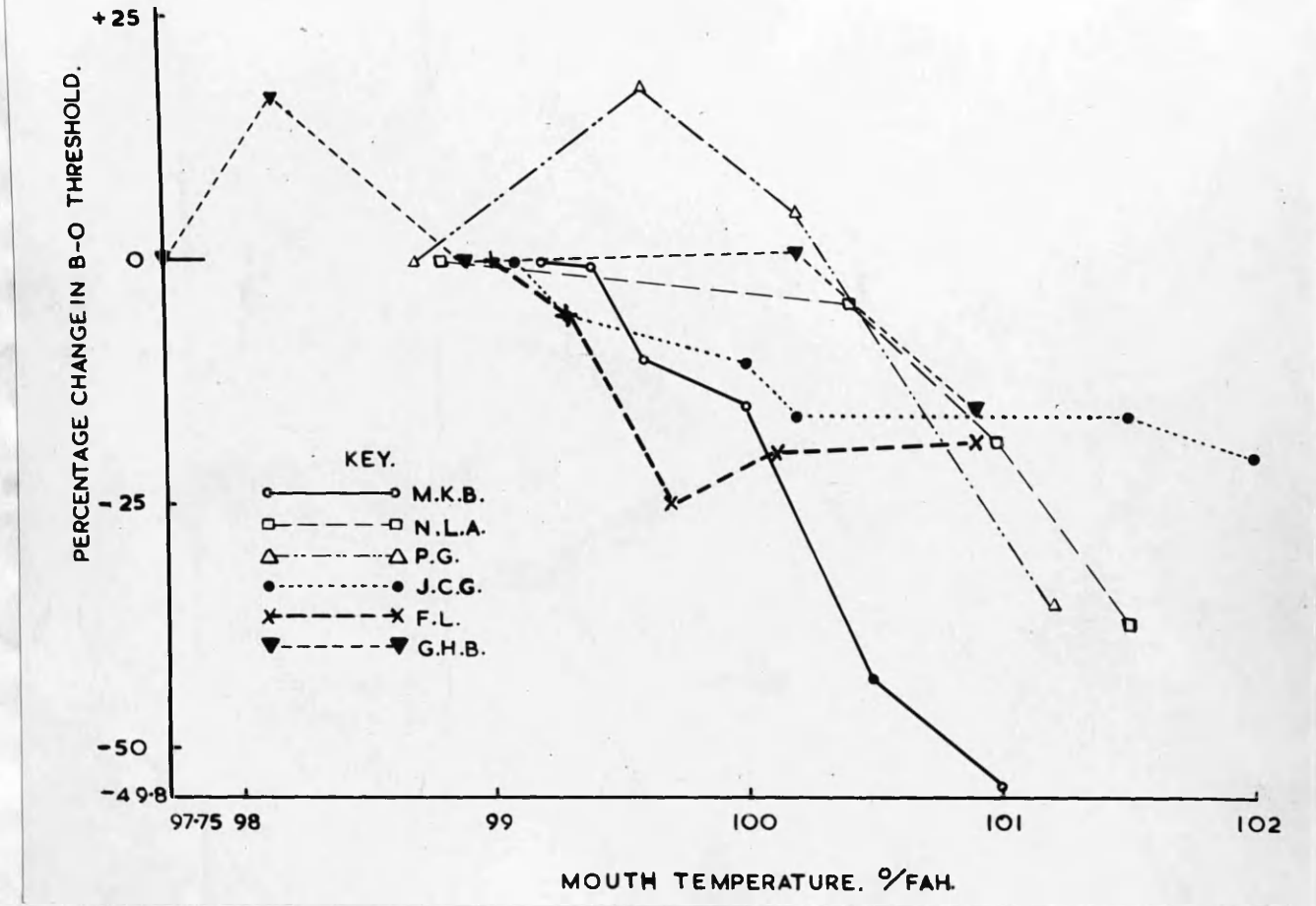


Fig. 6. Graph showing the percentage change in the level of central light loss with increasing temperature.

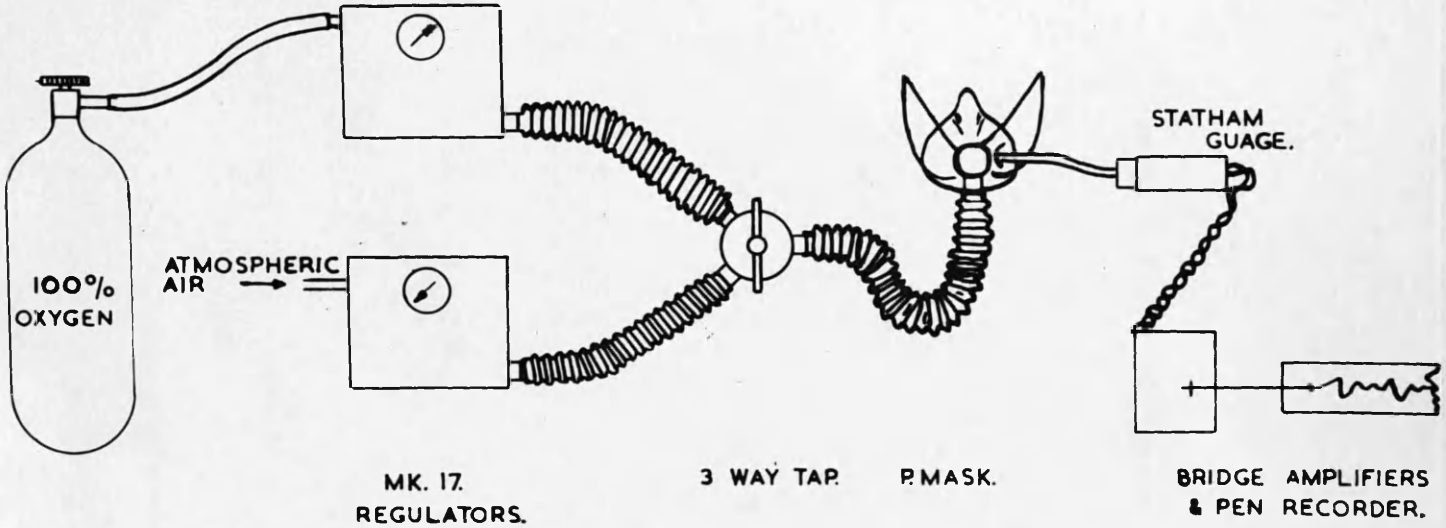


Fig. 7. Diagrammatic representation of the arrangement of the apparatus used to study the effects of pure oxygen on tolerance to g.

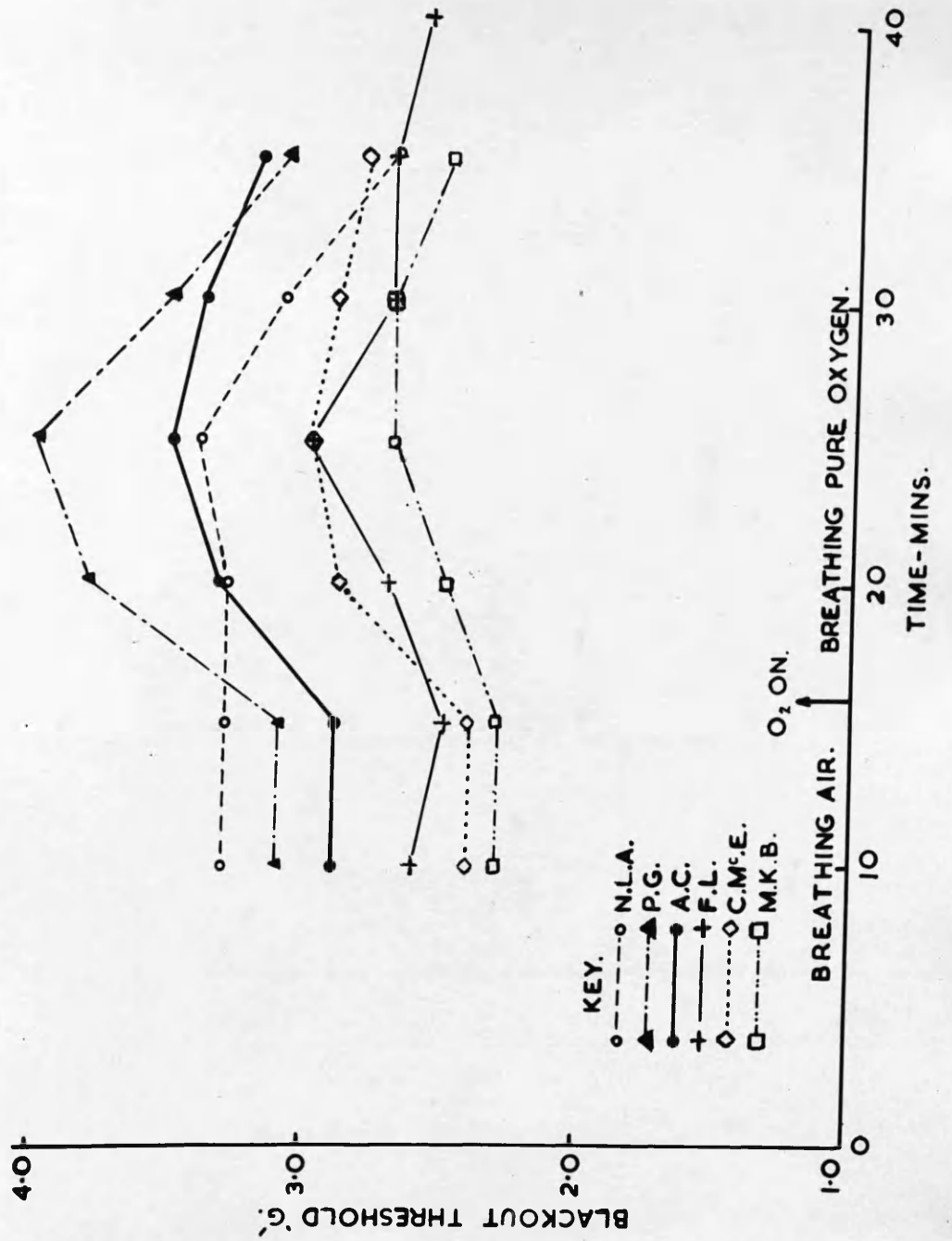


Fig. 8. Graph showing changes in blackout threshold when the subjects change from breathing air to breathing pure oxygen.

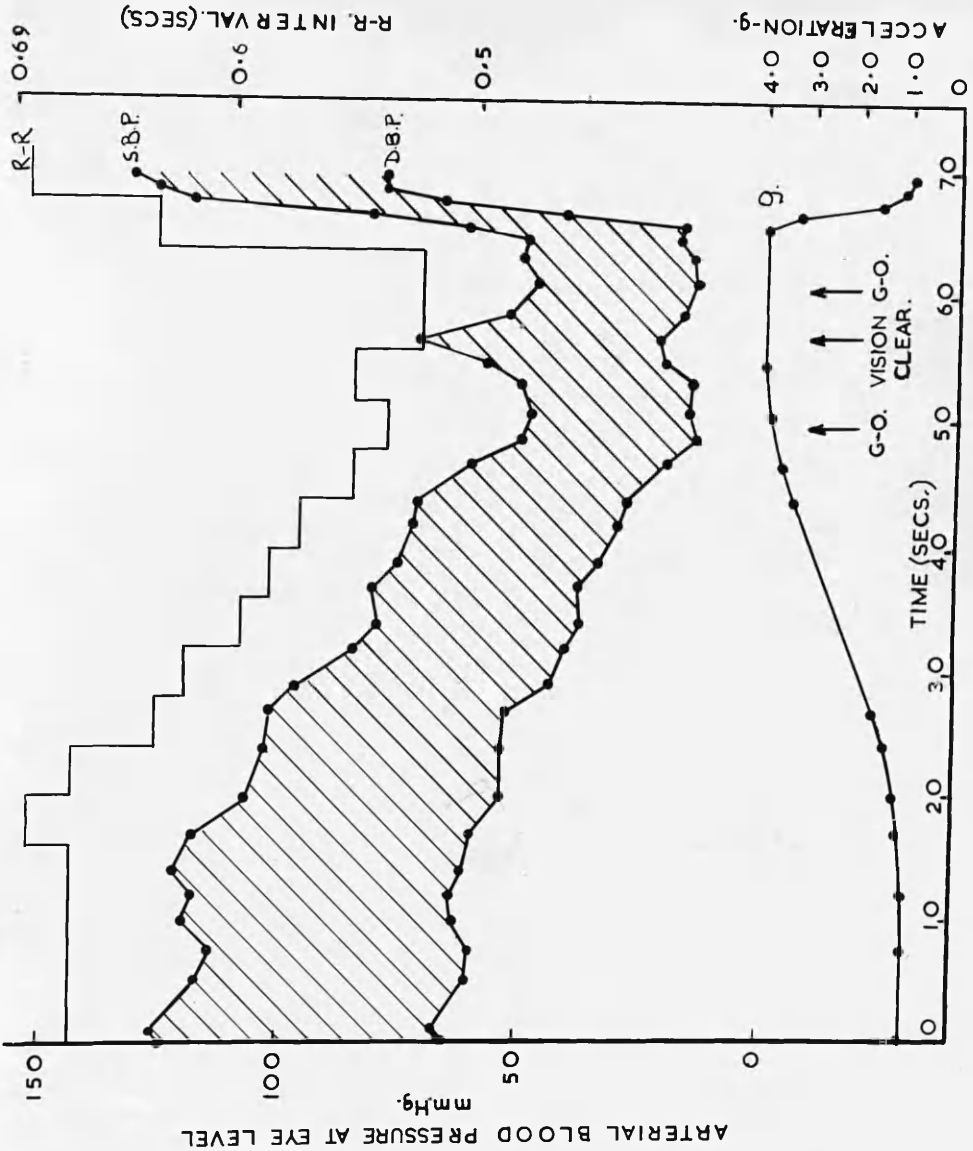


Fig. 9. Graph showing changes in arterial blood pressure at eye level with increasing positive acceleration. Also shown is the change in the R-R interval measured from the E.C.G.

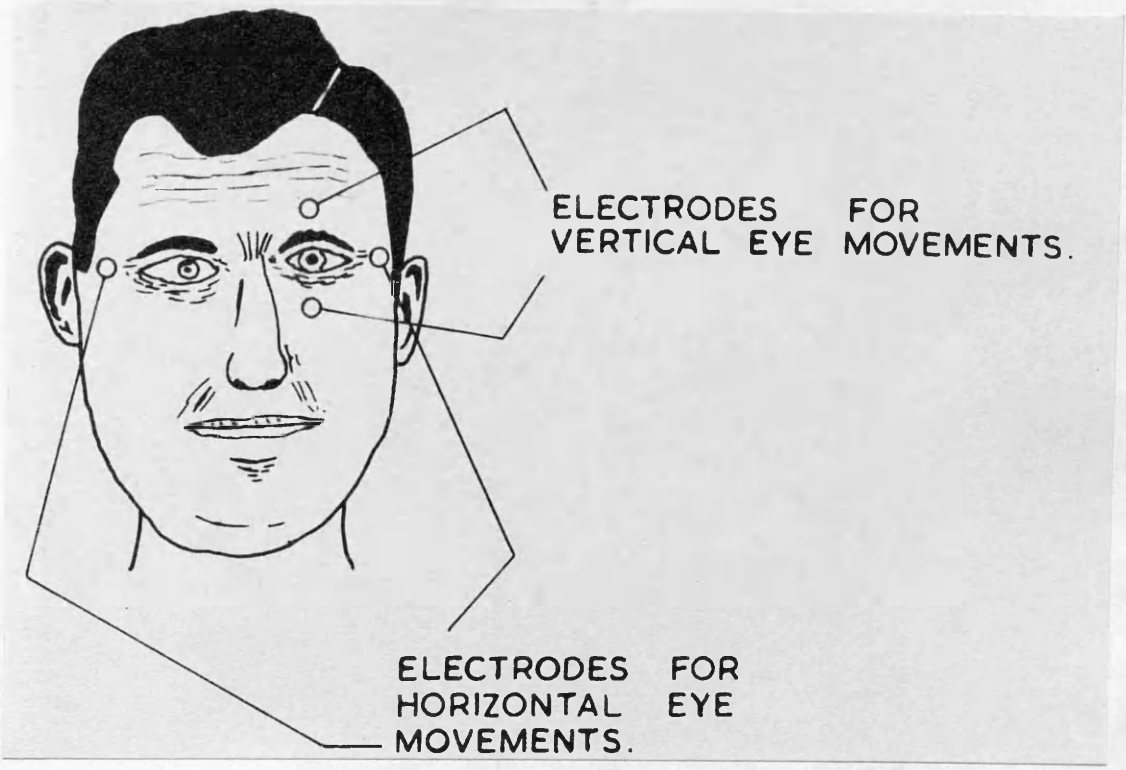


Fig. 10. Positions of electrodes for recording eye movements.

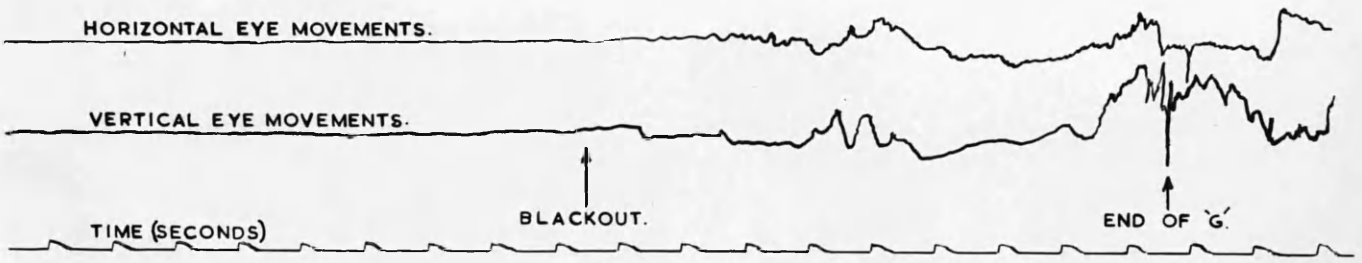


Fig. 11. Sample record of eye movements obtained during a g run to blackout level. It can be seen that loss of fixation occurs following blackout.

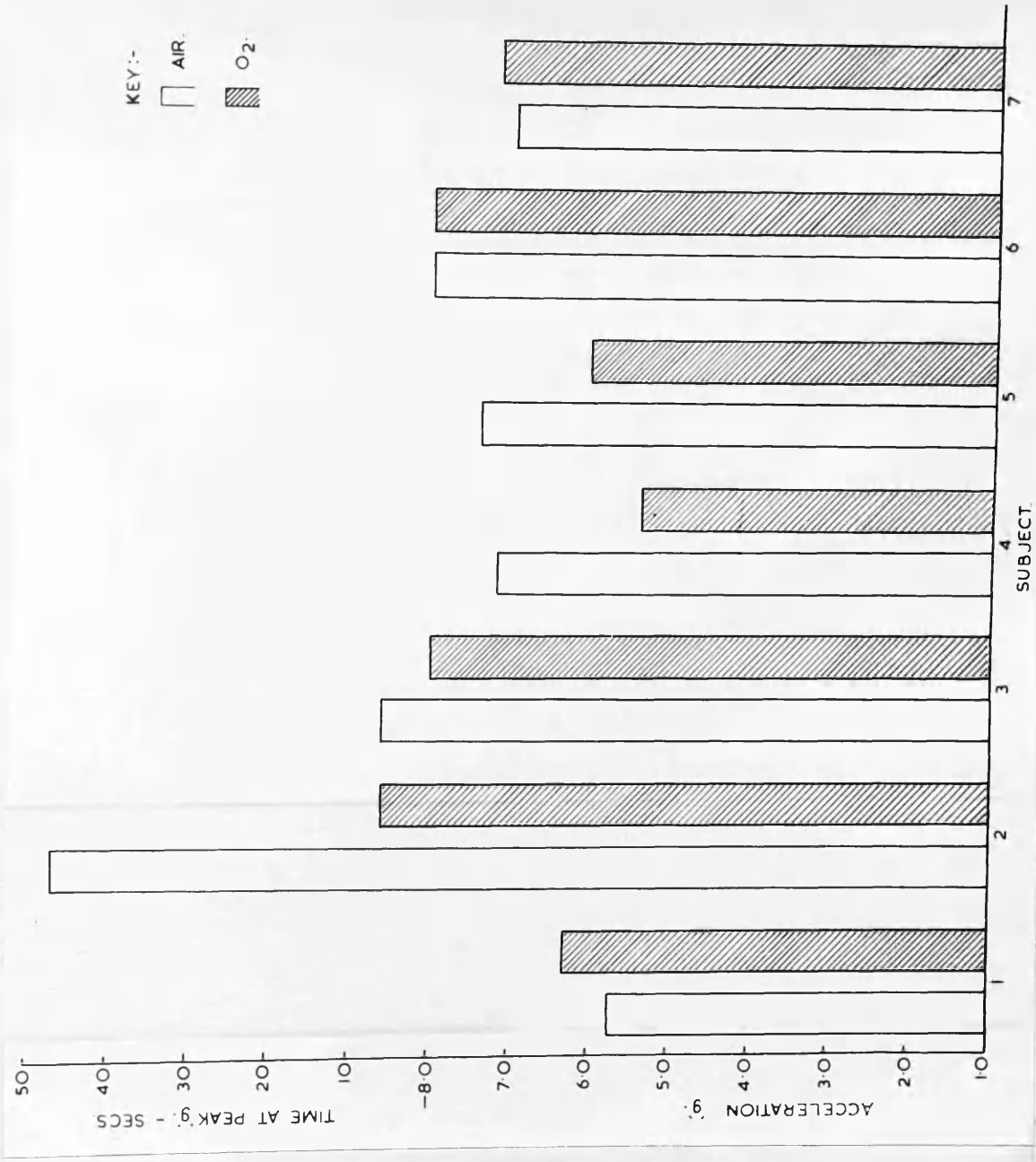


Fig. 12. Histogram showing the blackout thresholds of seven subjects when breathing air and oxygen under the same conditions.

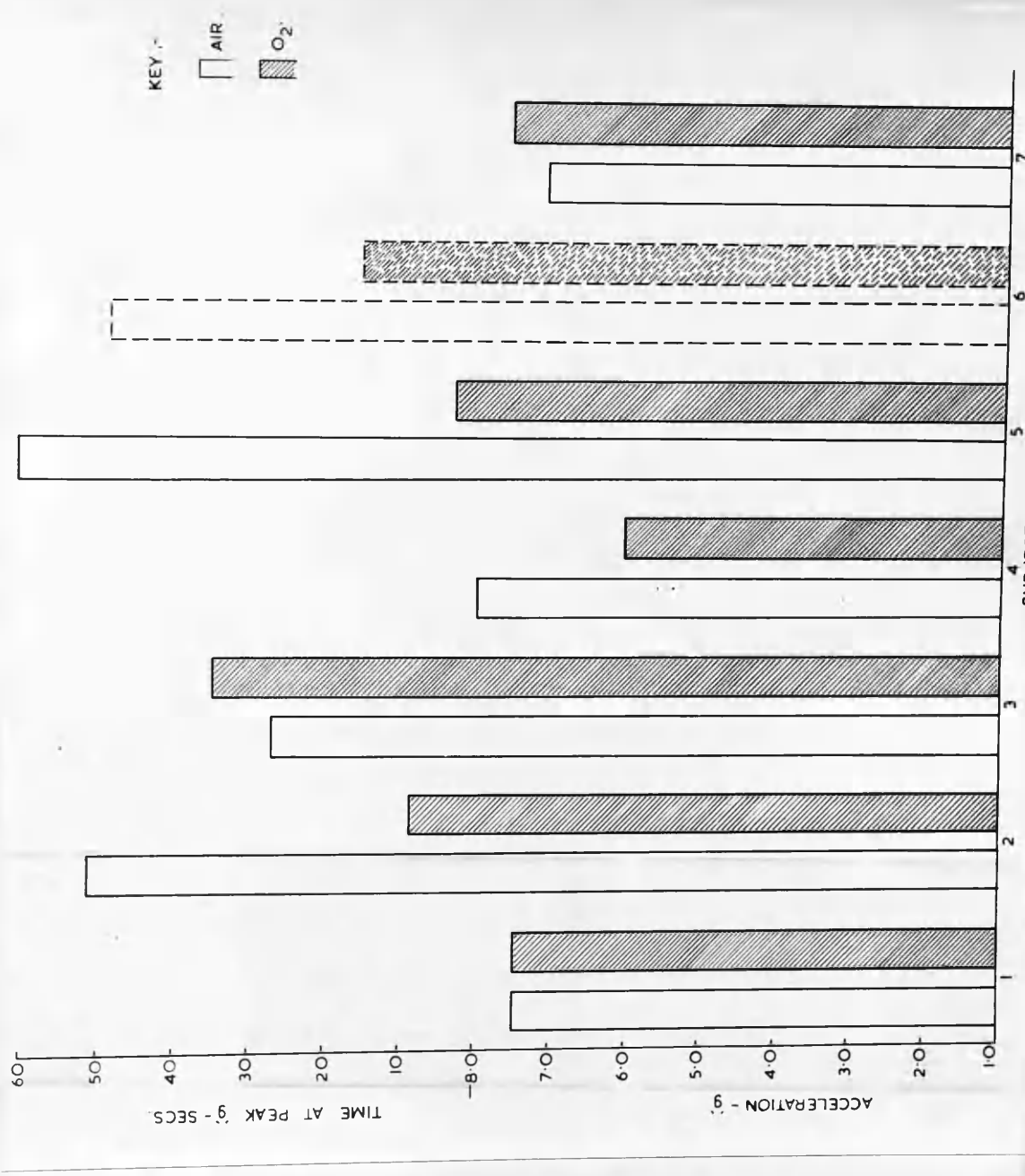


Fig. 13. Histogram showing the unconscious thresholds of seven subjects when breathing air and oxygen under the same conditions.

Fig. 14. Sample curves of arterial blood pressure recorded from the brachial artery at heart level during acceleration.

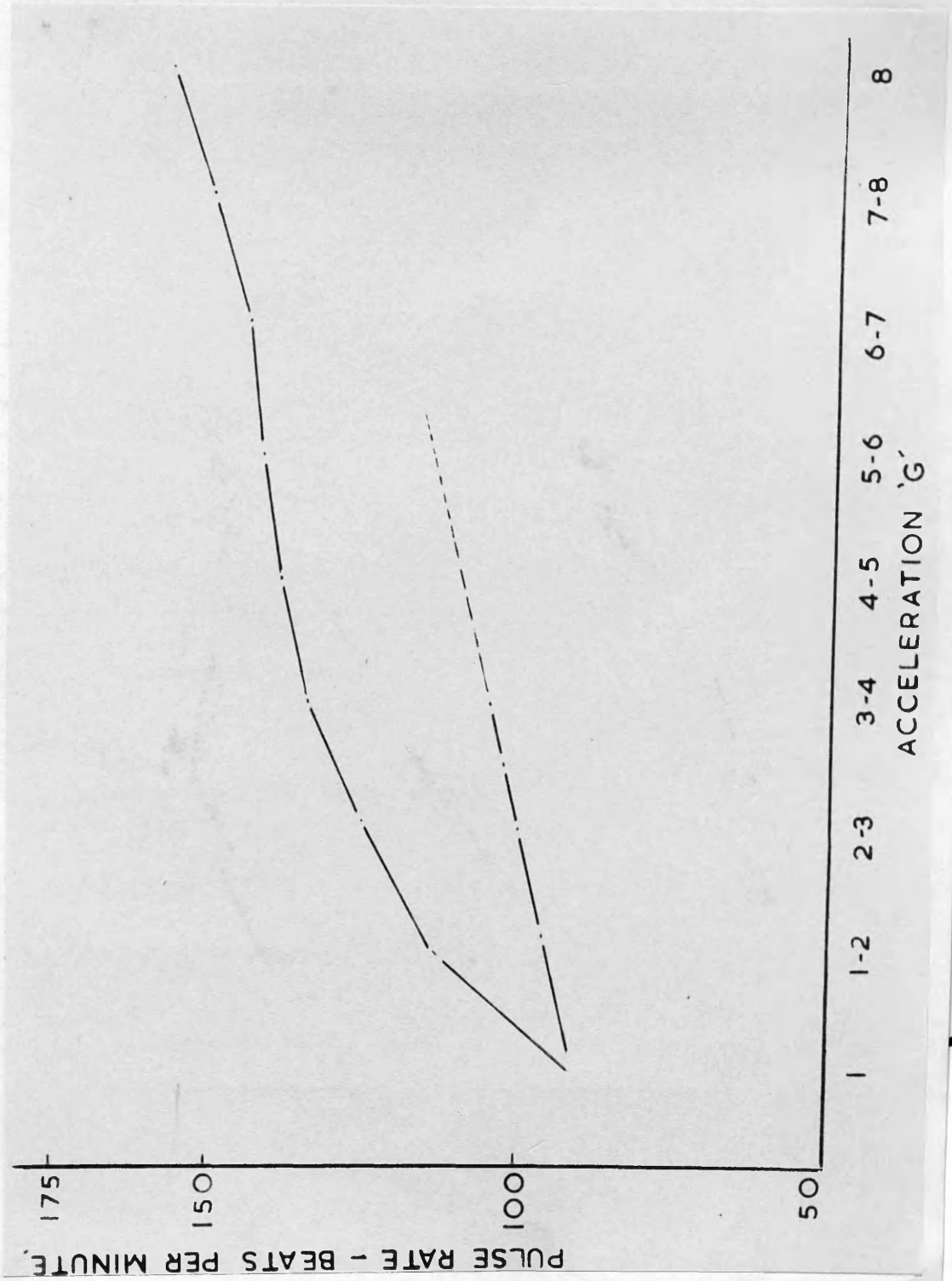


Fig. 15. Mean pulse rate changes during g.
(a) as calculated in part I. (b) in naval divers breathing oxygen.

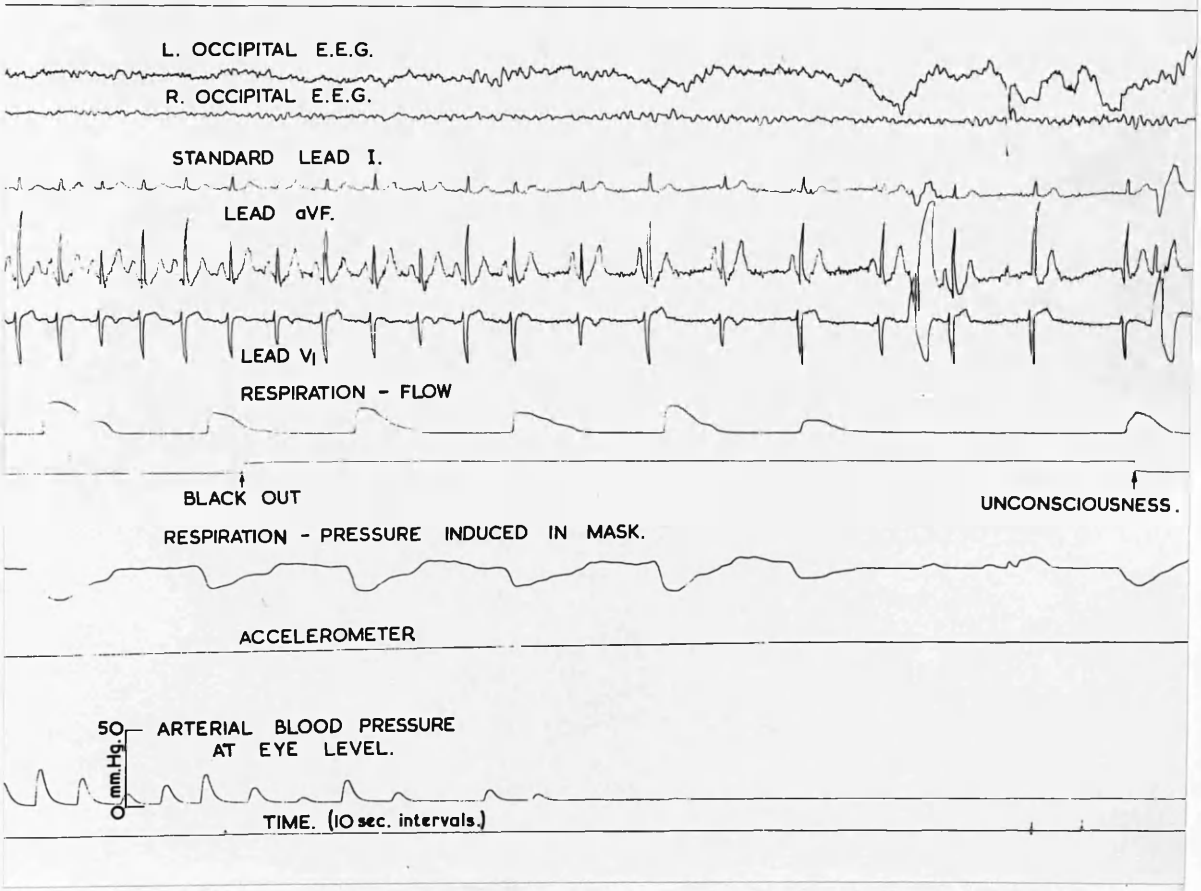


Fig. 16 (a). Record obtained from a subject breathing air during a run to 8.0g. This shows the only cardiac arrhythmia which occurred in this series.

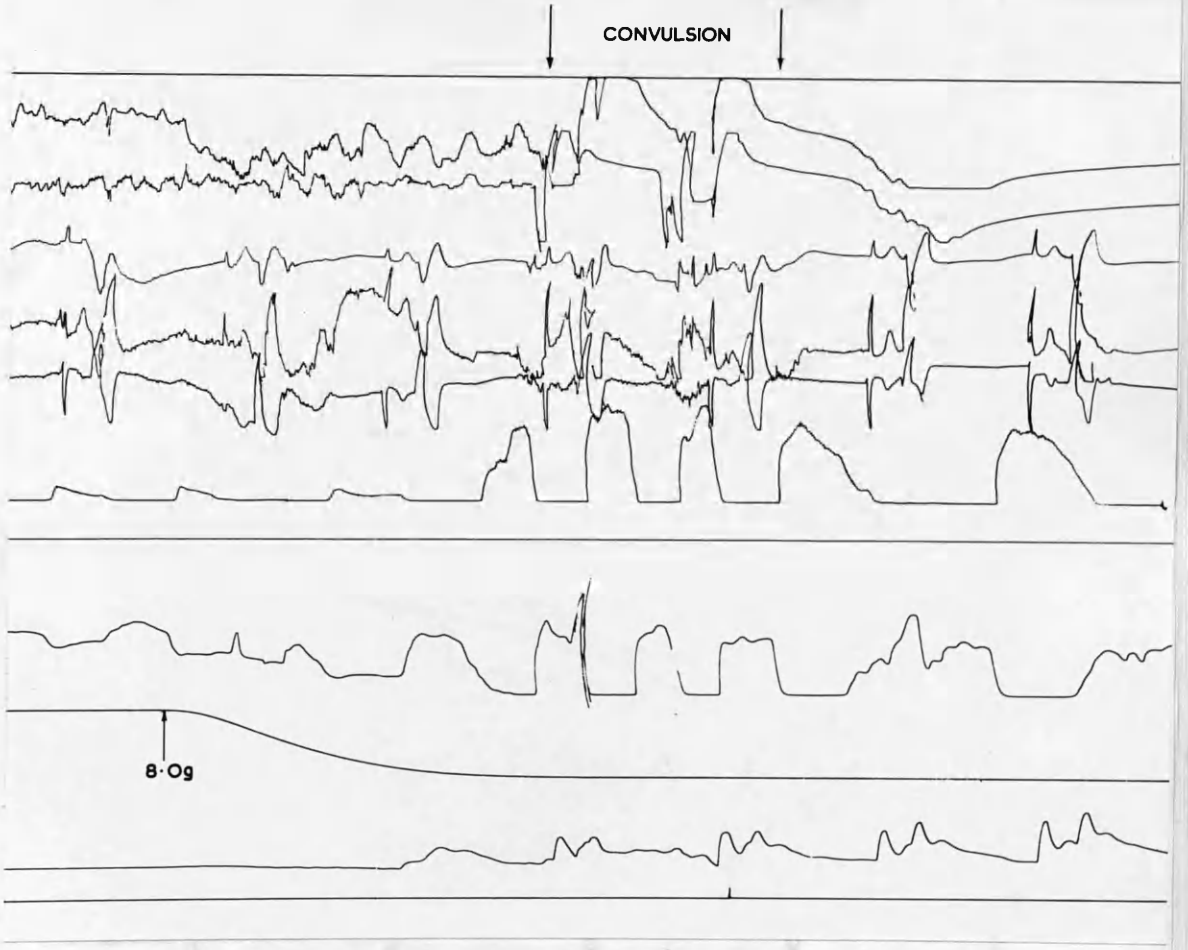


Fig. 16 (b).

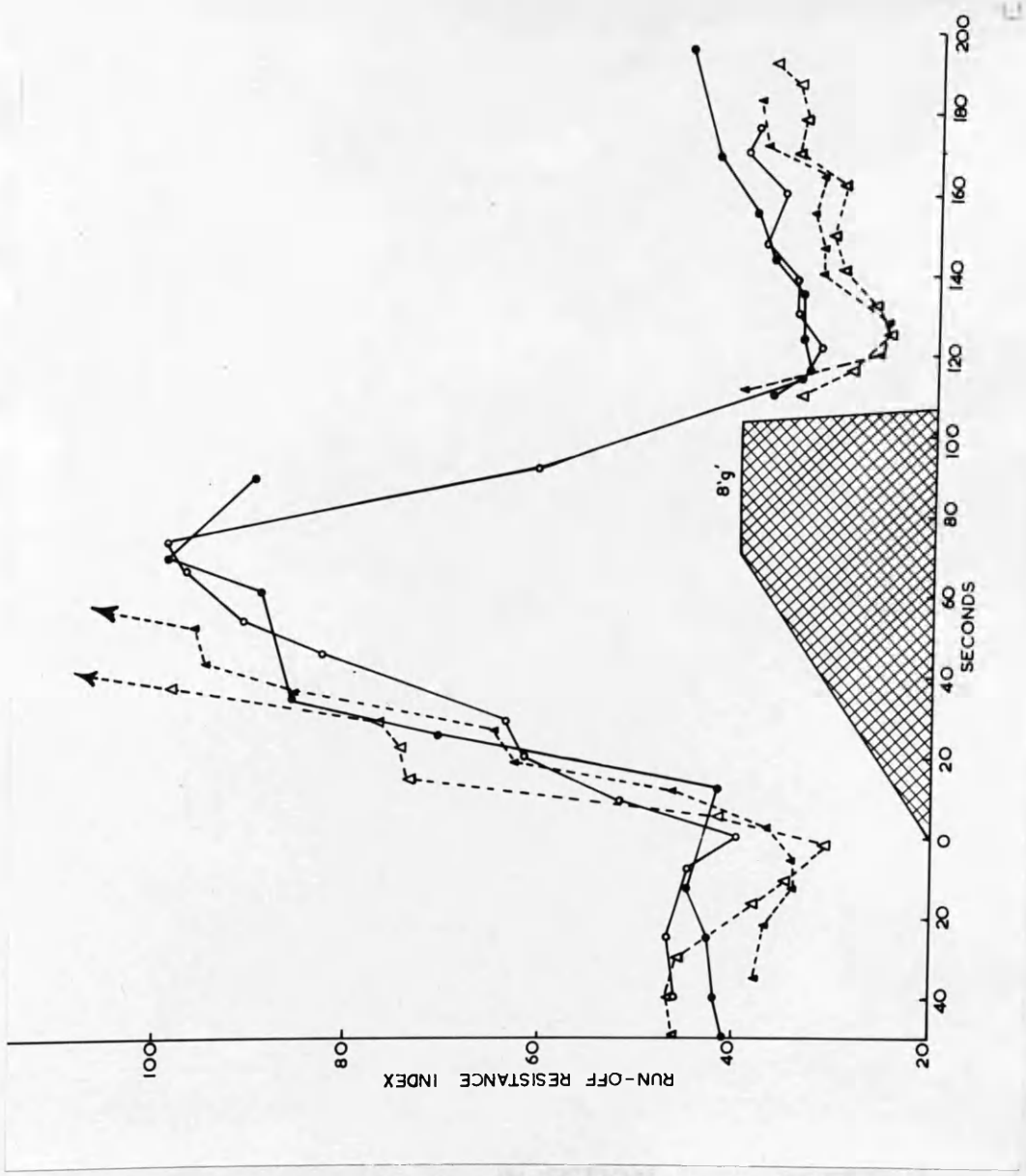


Fig. 17. Changes in peripheral vascular resistance during g. The resistance index is an arbitary figure derived by the method of Hayter and Sharpey - Schafer (1958).

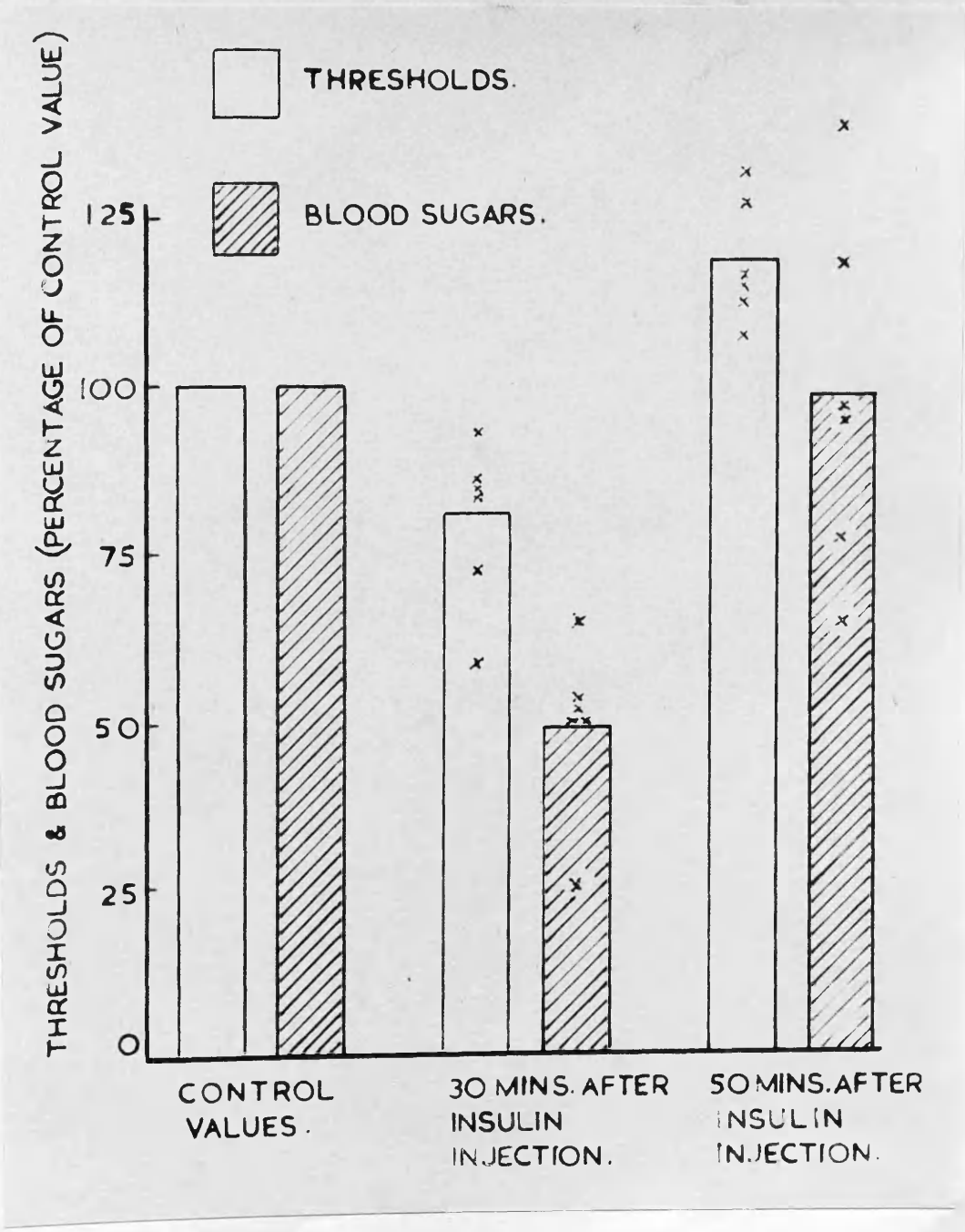


Fig. 18. The effect of the intravenous injection of insulin (0.15 units/Kg.body wt.) on the threshold and blood sugar. The individual values are plotted as crosses.

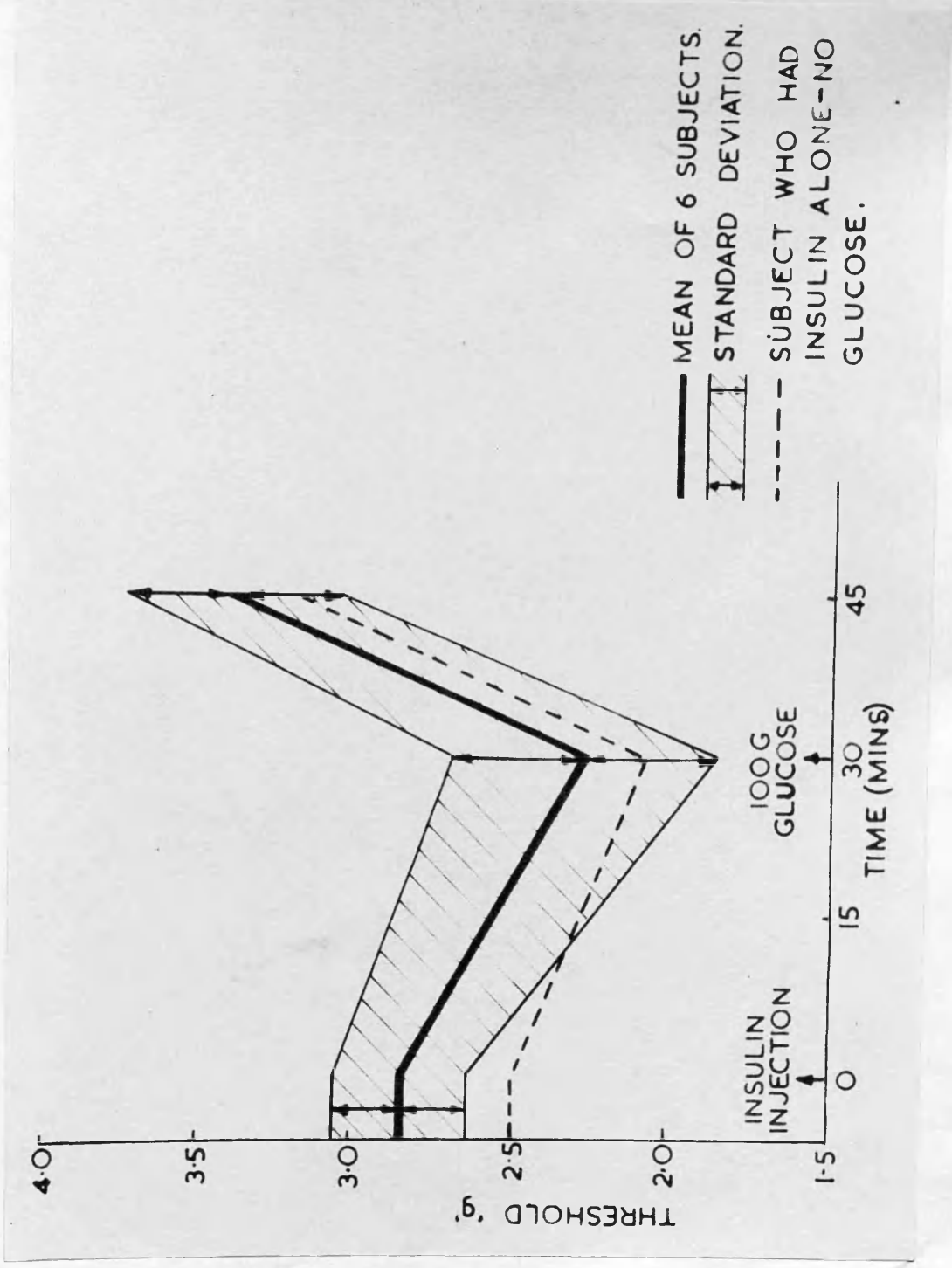


Fig. 19. Changes in g threshold following the intravenous injection of insulin (0.15 units/Kg.body wt.). The results of six subjects who received 100g. glucose after 30 minutes are compared with a subject who received insulin alone.

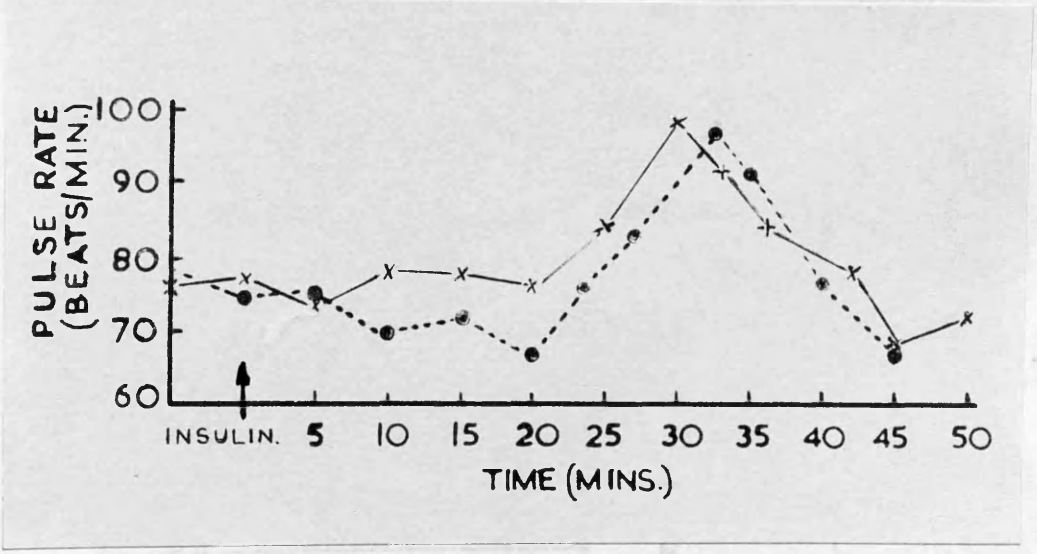


Fig. 20. Shows the pulse rate response of two subjects following the injection of insulin (0.15 units/Kg. body wt. intravenously).

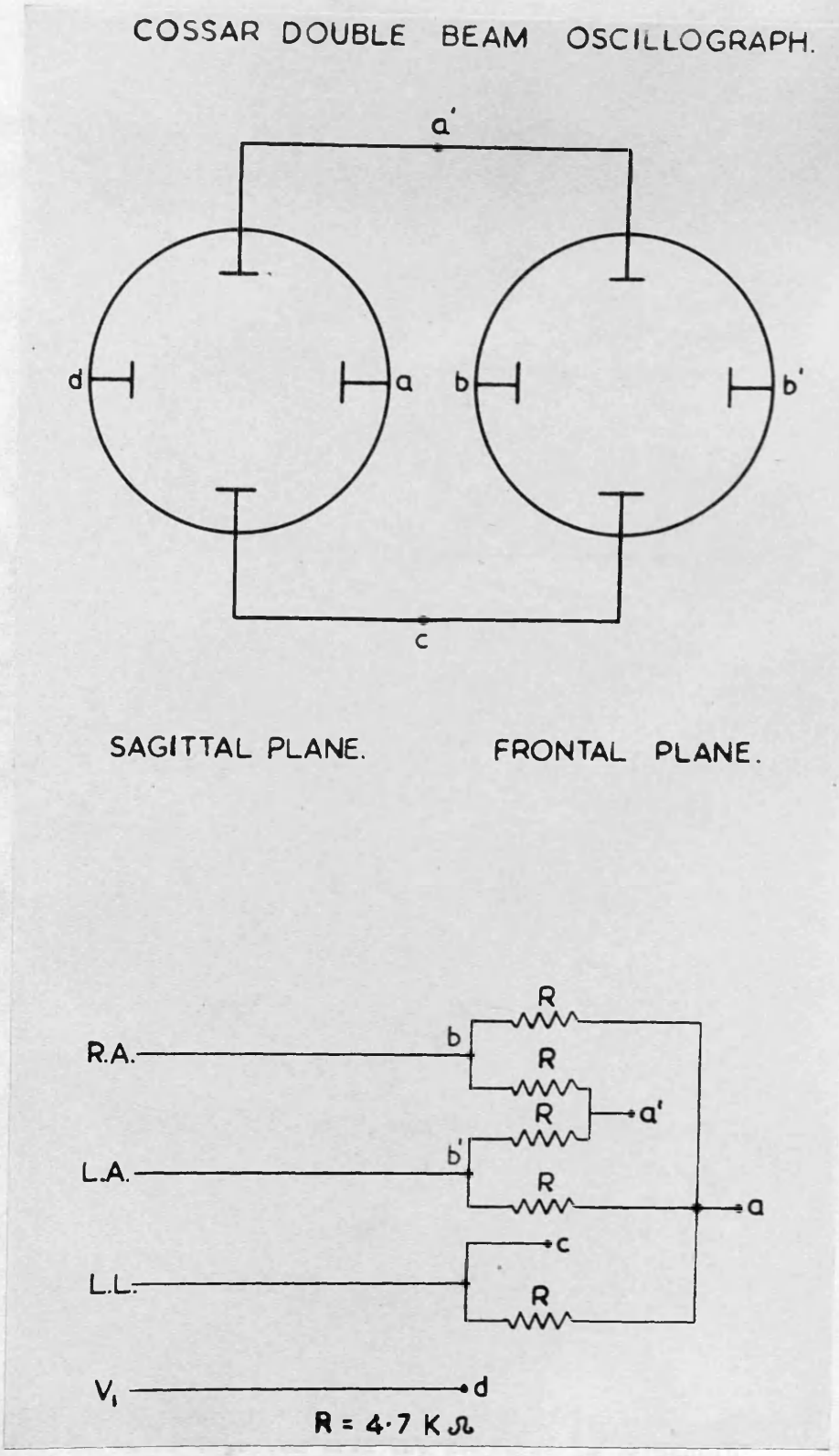


Fig. 21. Diagram showing the connections from subject to the oscilliscope. R.A. - right arm. L.A. - left arm. L.L. - left leg. V_1 = unipolar chest lead.

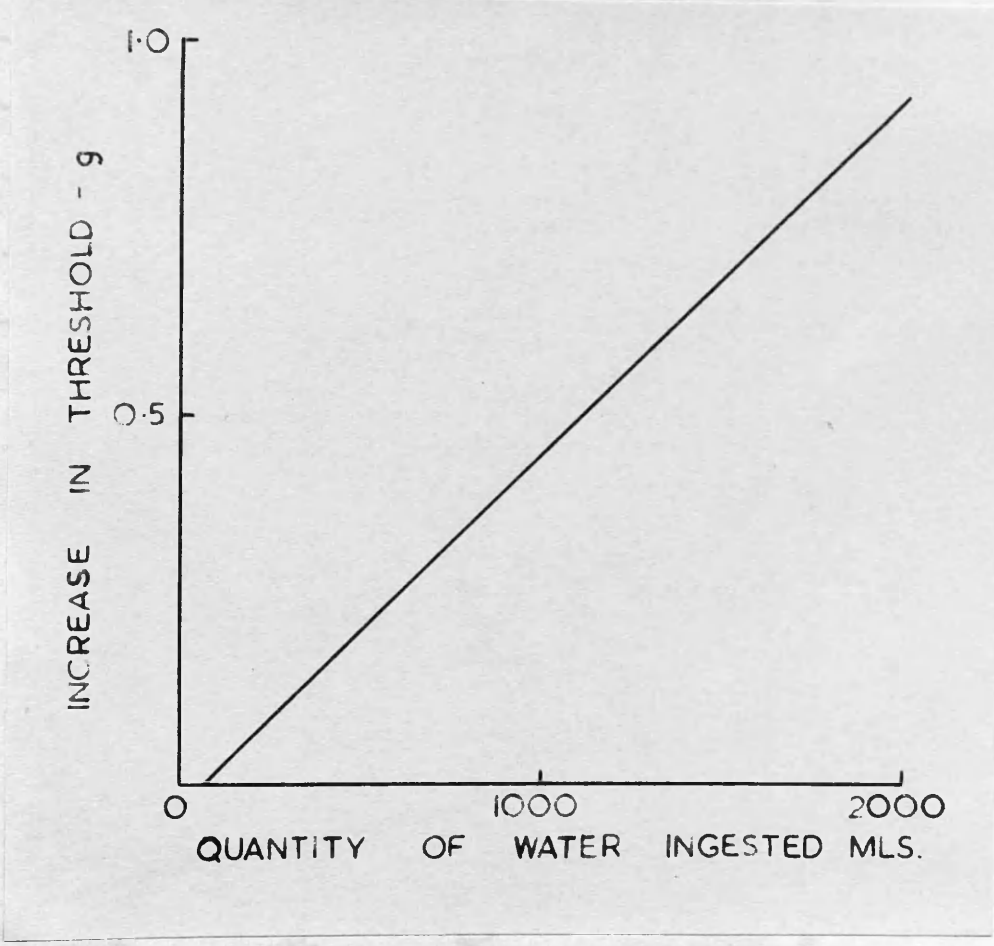


Fig. 22. Regression line obtained by correlating the quantity of water ingested with the increase in threshold produced. $Y = -0.04 + 0.485x$. $b = 0.085$ ($P = 0.01$)

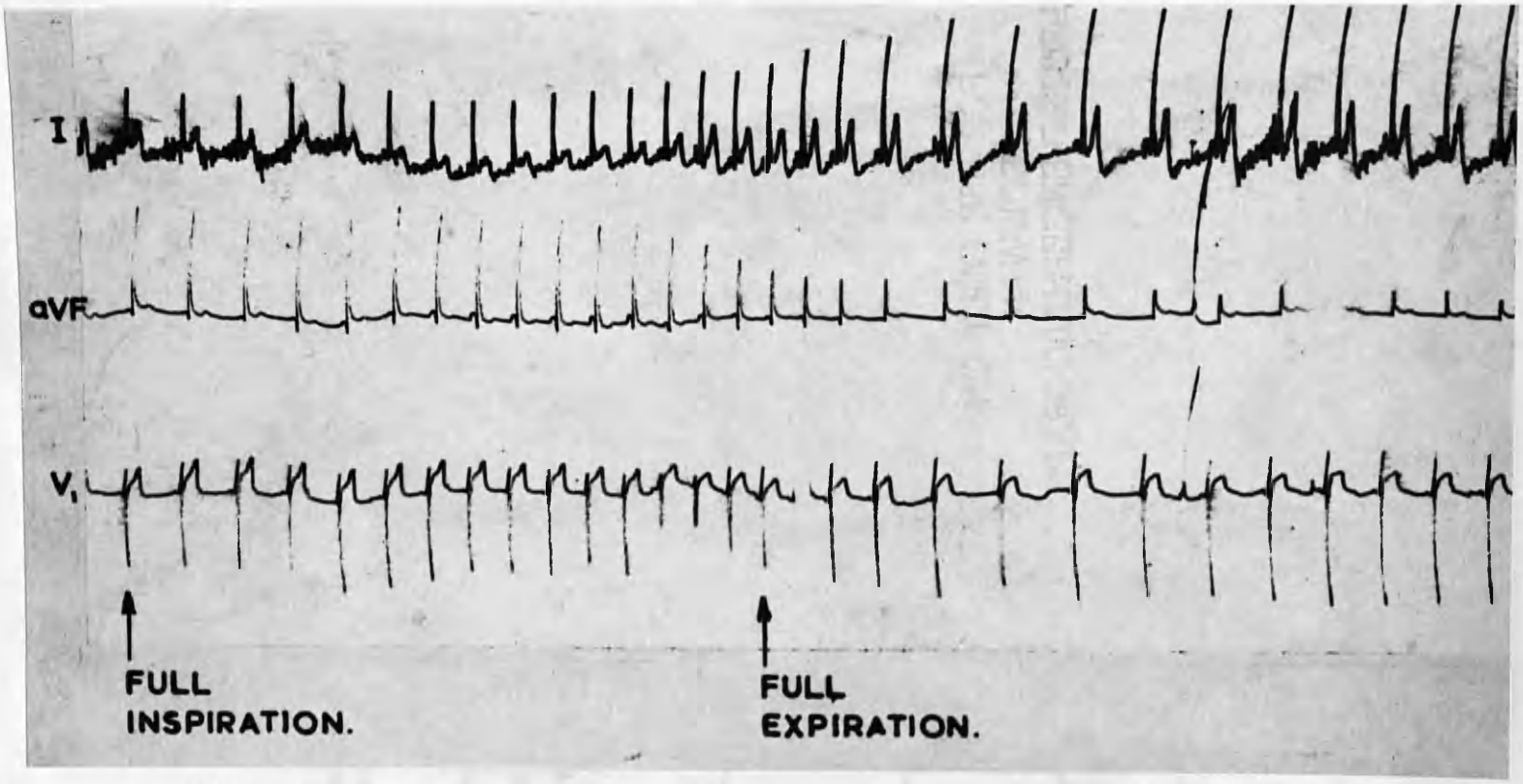


Fig. 23. Record showing the changes in amplitude of the E.C.G. with respiration.

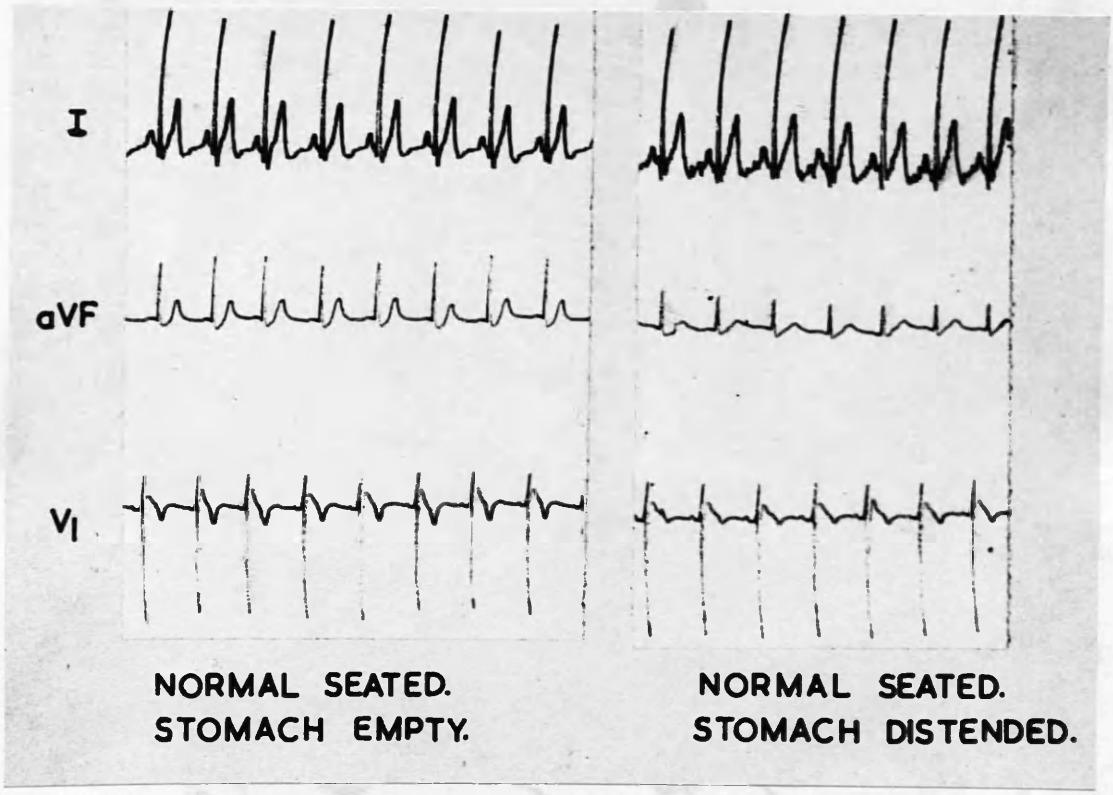


Fig. 24. The effect of stomach distension on the amplitude of the E.C.G. It will be seen that the changes are similar to those occurring with respiration (Fig. 23).

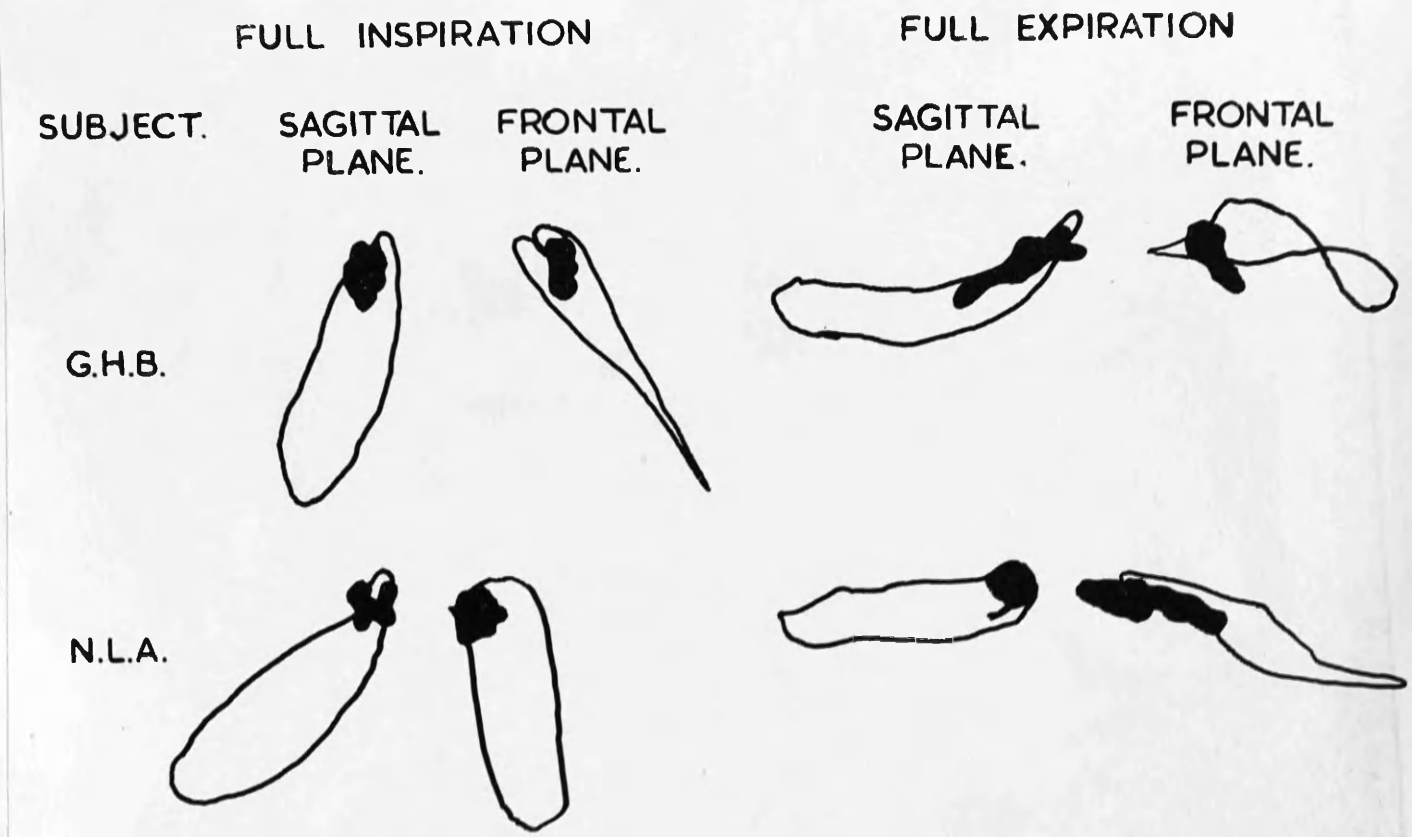


Fig. 25. The effect of respiration on the vectorcardiogram. It will be seen that counter clockwise rotation occurs in the frontal plane and clockwise rotation in the sagittal plane.

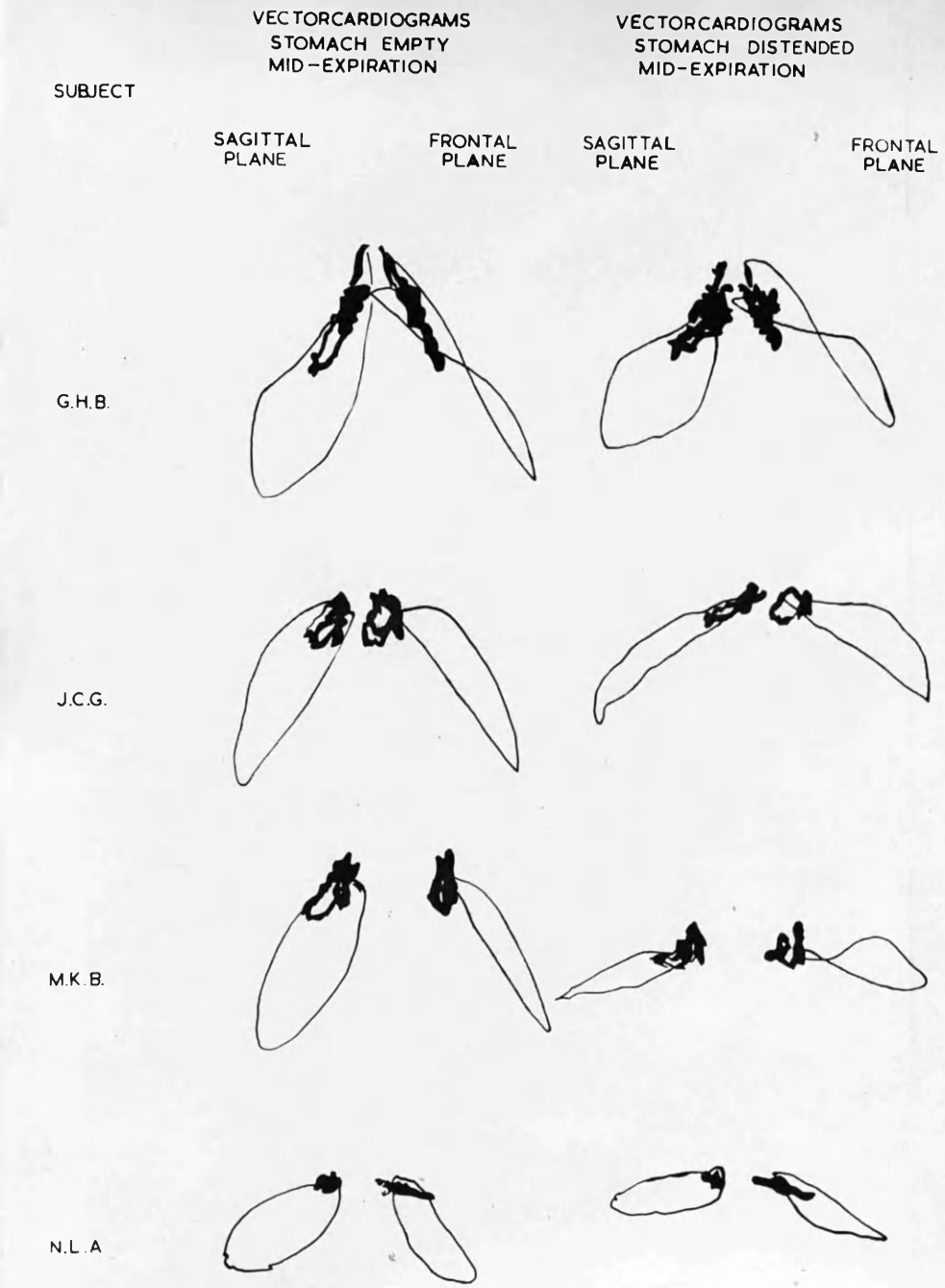


Fig. 26. Vectorcardiograms taken with the stomach empty and with the stomach distended. It will be seen that the change due to stomach distension is very similar to that due to full expiration (Fig. 25).

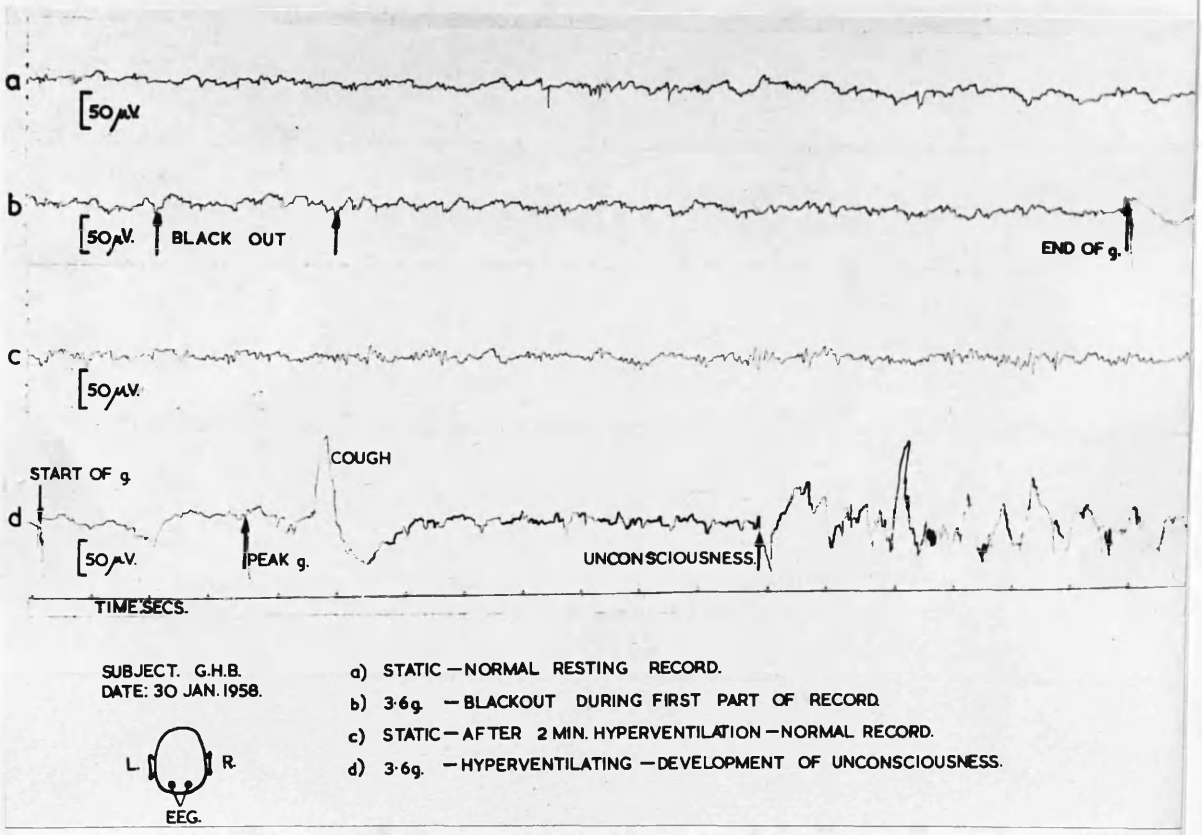


Fig. 27. The electro-encephalogram obtained in one subject under various conditions. It will be seen that hyperventilation or g alone produced little change, whereas in combination unconsciousness resulted associated with the appearance of slow high voltage waves.

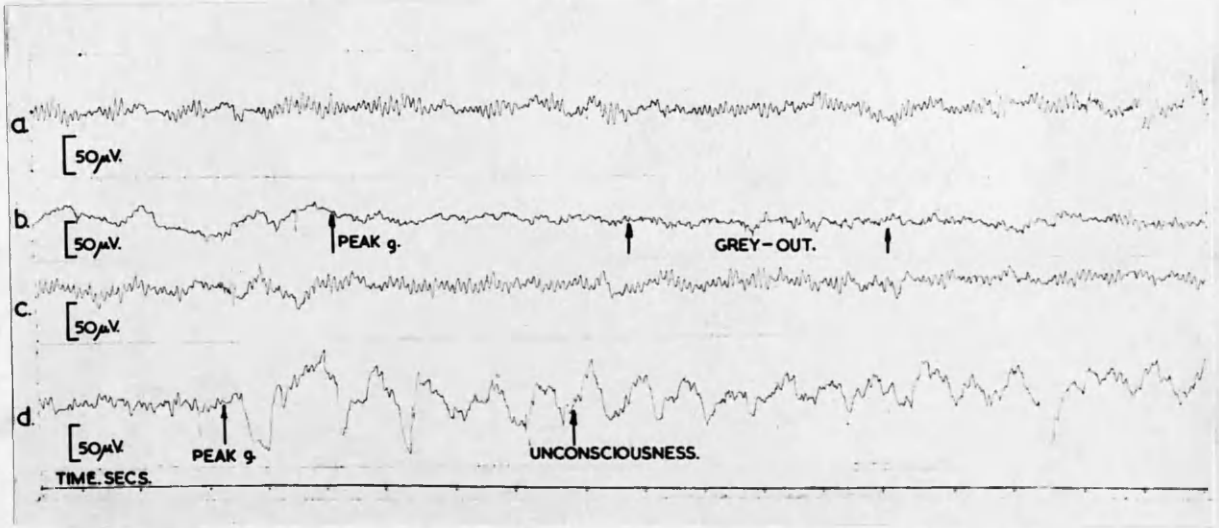


Fig. 28. Record showing changes in the electro-encephalogram under various experimental conditions.

Subject P.G.
29-1-58.

a) Static - normal resting record.

b) 3.6g - brief transitory grey-out.

c) Static - after two minutes hyperventilation.

d) 3.6g - 2 mins. hyperventilation 30 mins. after the injection of 2 units soluble insulin intravenously.

Fig. 29. Bipolar occipital electro-encephalograms obtained during various experimental conditions. It will be seen that g or hyperventilation alone caused little change but the combination of the two caused unconsciousness with the development of slow high amplitude waves.

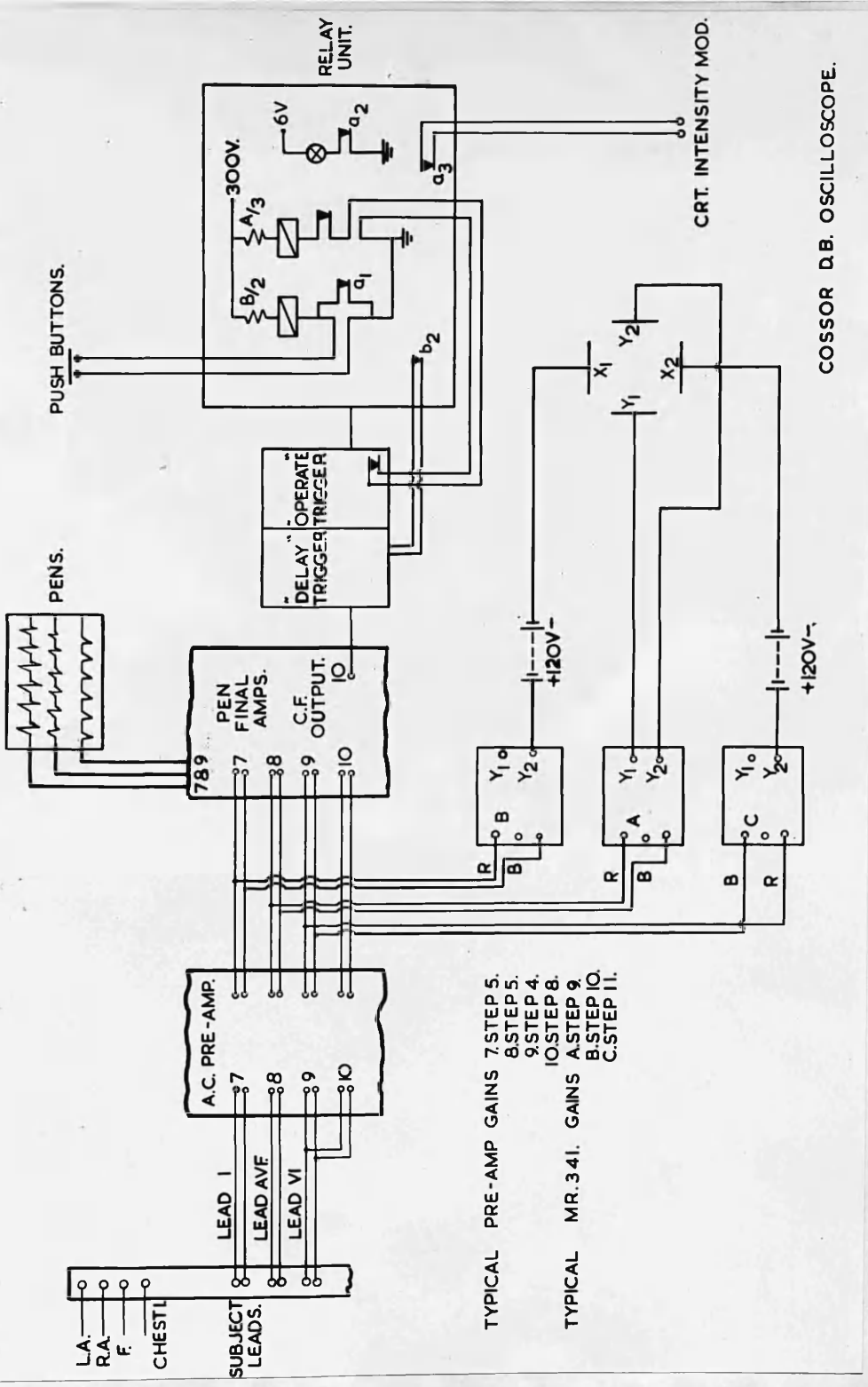


Fig. 30. Diagram of the general layout of the circuit used for recording vectorcardiograms.

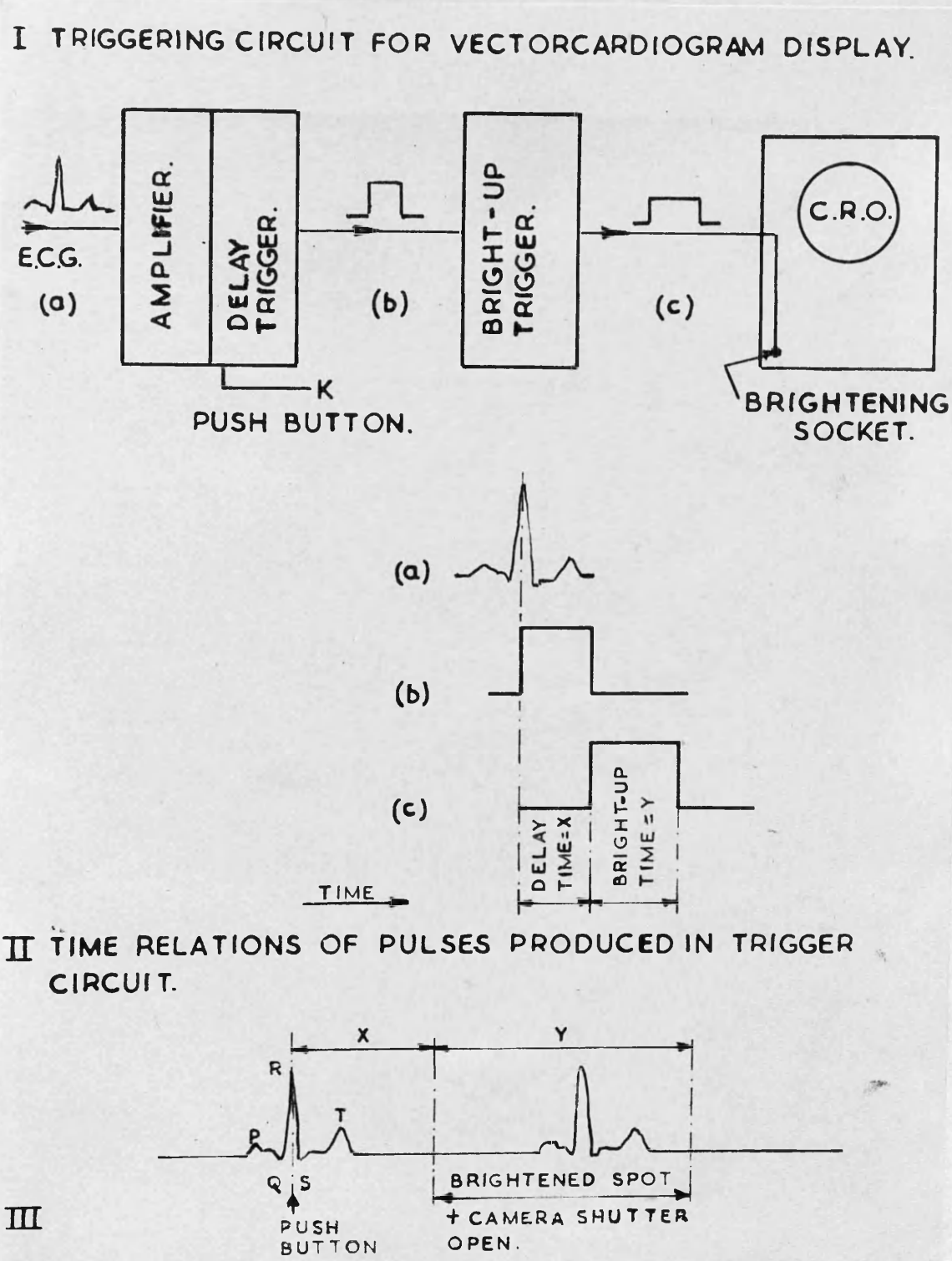


Fig. 31. Diagram showing the time relations of the pulses in the triggering circuit for vectorcardiogram display. (See text).