A STUDY of "BLACKING OUT"

With Experiments Performed on Single-Seater Aeroplanes

by

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"BLACKING OUT"

INTRODUCTION

"Blacking Out" might be defined as the "Blind Spot of Aviators". It is a condition, brought on by a great increase in acceleration and characterised by sudden and complete blindness. The symptoms are experienced during a rapid change of direction in either the horizontal or the vertical plane. The blindness is apparently transient and lasts only for a few seconds. During these few seconds however, the aviator is at a very great disadvantage, being unable to see anything although he retains the full use of all his other senses.

The importance of this condition will be immediately recognised. During the last twenty years, aviation has developed at a very rapid pace and already is in a position to challenge seriously other methods of transit. Its importance in both civil and military spheres cannot be over-estimated but if aviation is to progress further every possible limitation must be investigated and every avenue explored to make it as safe as is humanly possible. For years this has been the aim of the engineers and designers, and in this they have been successful. Today, the aeroplane, as a machine, equals the ship, the railway train and the motor car.

But the future of aviation does not depend on the machine alone. Much will depend on the pilot controlling the mechanical structure. Is this very essential factor without blemish? Certainly at the present

time, every possible care is taken to ensure that these men and women are in perfect health. Constant watch is kept on their work and by medical examination at frequent intervals, their general health is closely observed. Even the presence of a common cold is sufficient to debar a pilot from flying until he is once again physically fit.

But this is not enough. Since the war, the speed of the aeroplane has been greatly increased and its construction so improved that now it is able to resist great stress and strain. But because of his physiological limitations the pilot is unable to make full use of this efficiency. In other words the efficiency of the machine is now greater than that of the pilot controlling it. As an example of this I need only mention the recent high altitude record attempts. In these attempts scientific appliances had to be used to enable the pilot to reach the heights attainable by the machine. Another example is the not too distant Schneider Trophy high speed races. During these races it was found that the planes were capable of turning at high speeds within a small circle. No benefit was derived from this, however, as the time thus gained was lost again by the pilot in recovering from the effects of the manoeuvre.

The aeroplane designers are capable, we are told, of increasing still further the machines' efficiency. But what practical benefit can be obtained if full use cannot be made of that increased efficiency. And it is of vital importance that the efficiency, not only be increased, but that it be used to its fullest extent. We are all now only too well aware that this country must depend, for its defence, on a large and

efficient Air Force. We may easily increase its size but we cannot increase its efficiency until we have eliminated the physiological limitations of the pilot.

In the realms of commerce the advantages of high speed flying may not, at present, be so obvious. But if aviation is to advance, and it is sure to advance, high speed flying will have to be taken into account. Already stratosphere flying is taking shape in the minds of inventors and in this speed must play its part. We must be prepared for that. We must increase the efficiency of the pilot and to do that we must, as soon as possible, ensure that he does not "Black Out".

My first experience of "Blacking Out" occurred about three years ago when, flying as a passenger, I blacked out as the machine was pulled out of a dive. The pilot afterwards explained to me that he too had blacked out at the same time but that it was nothing to worry about as the effect was only momentary. It was not until some time later that I really became aware of the real importance of the condition and decided to investigate the reason for it.

In the summer of 1936 I was watching several fighter machines practising attacks on another aircraft when I noticed that one of the machines all but collided with its neighbour. Only prompt action on the part of the second pilot averted an accident but both machines lost their chance of an attack. The pilot afterwards told me that he had blacked out during the turn. It was not a usual occurrence with him, he said, but on that day he had been flying for some time and was tired.

He had therefore turned more sharply than he had intended to do.

The incident however, impressed me with the importance of blacking out from a military point of view. If a pilot of a fast machine must keep his attention focussed on the rate of his turn in order to keep from blacking out, what would happen in actual warfare? Surely under the conditions of active service the pilots would be too engrossed in the actual fight to remember such details. Besides the pilot who could turn quickest and fly fastest would surely have the advantage whereas to black out would be fatal. To me the question seemed a serious one. I began my investigation that very evening.

In this I was fortunate in being the medical officer in charge of a station on which there were several fighter squadrons. At the same time, I was unfortunate, as will be seen later, in that the machines were all single-seaters. Almost immediately, however, I found my first difficulty. The pilots seemed extremely reticent on the subject and in almost every case denied that they had ever blacked out. On impressing them that I was not trying to limit their flying but was really trying to make some investigations on the subject they all endeavoured to help me. They seemed anxious, not only to discuss the subject but offered to perform any experiment I thought would be of value. All the pilots, irrespective of age, had blacked out and could do so at will. There were about fifty pilots, their ages ranging from 19 to 35 years of age. Each was interrogated and his story noted. All were medically examined in accordance with the standards for fitness for piloting duties and the results of these examinations compared.

Lastly practical experiments were carried out. Owing to the dangerous nature of the experiments and to the exigencies of the Service, the investigation has taken approximately eighteen months.

PHYSIOLOGY

Since the dominant feature of "Blacking Out" is loss of vision it will be convenient to consider, first of all, the eye itself.

Anatomy of the Eye.

The eye-ball is a sphere, the greater part of whose external surface is formed by a white membrane called the Sclera. In front, this white membrane is replaced by a transparent structure, the Cornea. Attached to the eye-ball behind and slightly to the inner side is the Optic Nerve. Lining the Sclera itself is a vascular pigmented coat which is termed the Choroid. Anteriorly, this has a circular aperture with which the Choroid becomes modified into several important structures, namely the Iris and the Ciliary Body. Spread out within the hollow cup formed by the Sclera and Choroid is the Retina. This is a soft delicate membrane of nervous tissue and is connected with the Optic Nerve. The spherical cavity thus formed is entirely filled by three transparent structures, the Lens, the Aqueous Humour and the Vitreous Humour. The Lens itself is a biconvex body of high refractive index, situated symmetrically behind the opening in the Iris. Held in place by suspensory ligaments it divides the cavity into two chambers. The anterior chamber is filled by the Aqueous Humour whilst the Vitreous Humour occupies the posterior cavity. Thus the eye is a solid organ having considerable rigidity. It is situated in the orbit, a rounded depression in the skull on either side of the nose, where it is held in place by several muscles. All but its anterior surface, therefore, is surrounded and protected by bone.

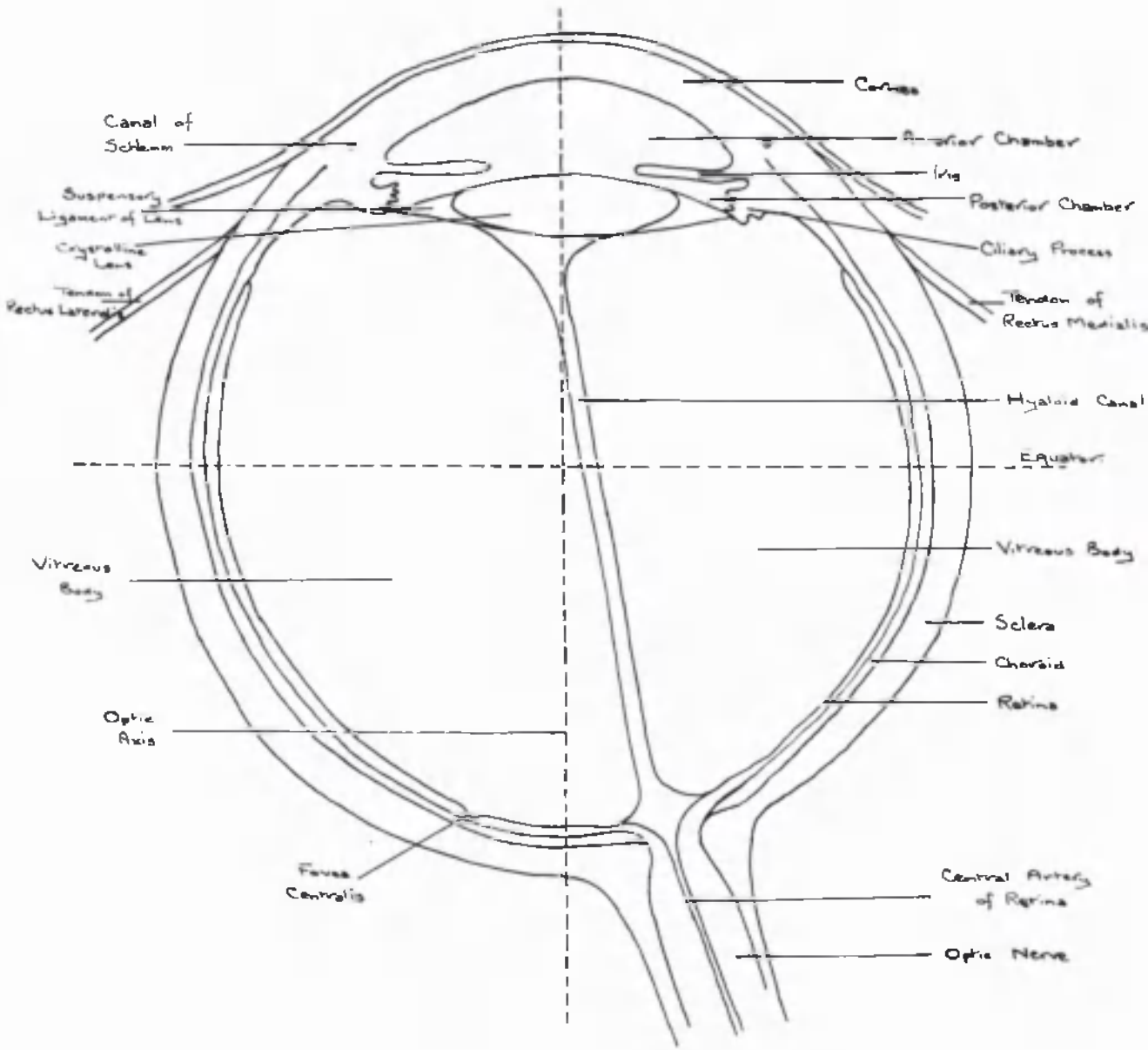


Fig.1 Diagram of Horizontal Section through the Left Eye and Optic Nerve.

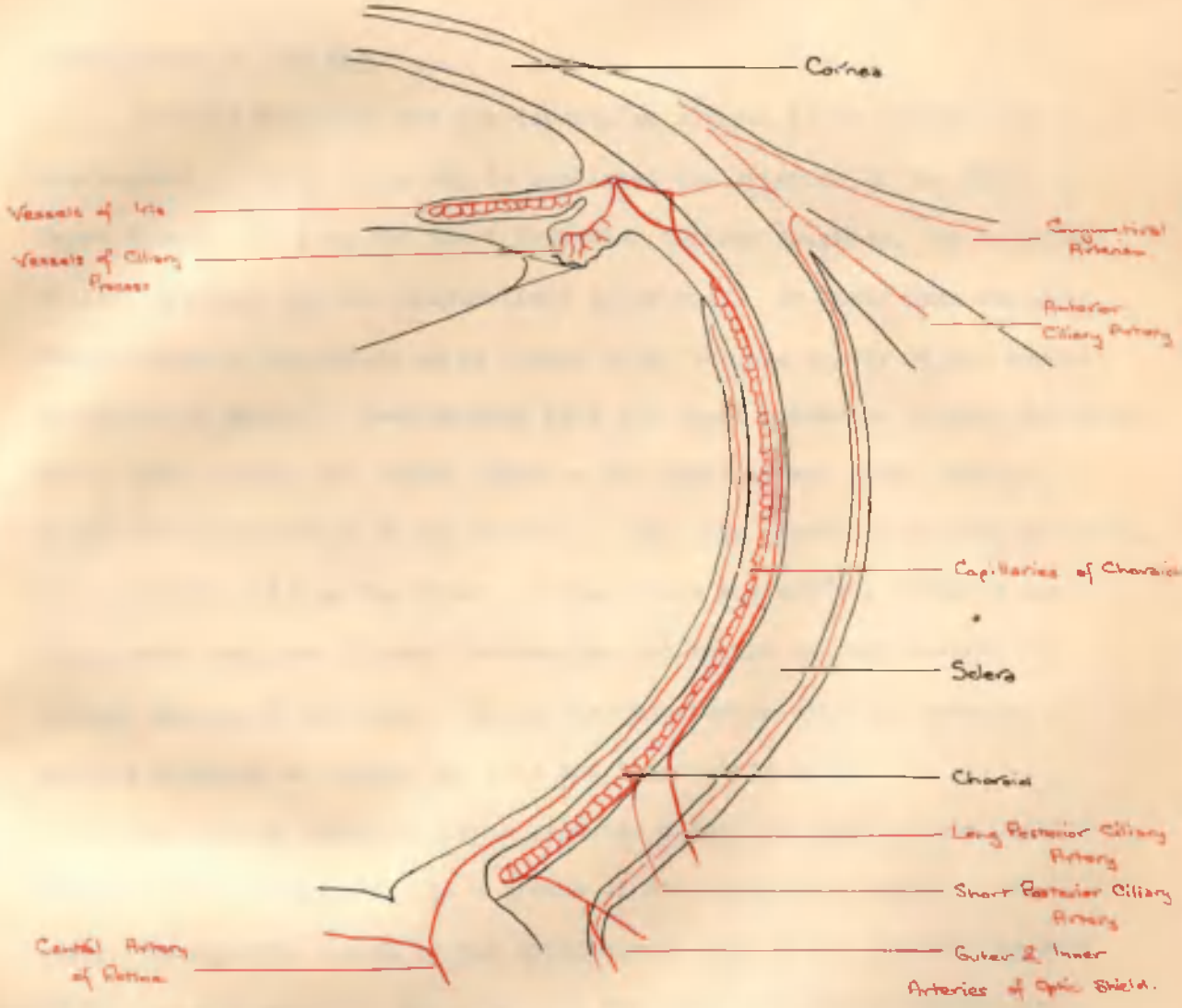


Fig.2 Rough Diagram of Arteries of the Eye.

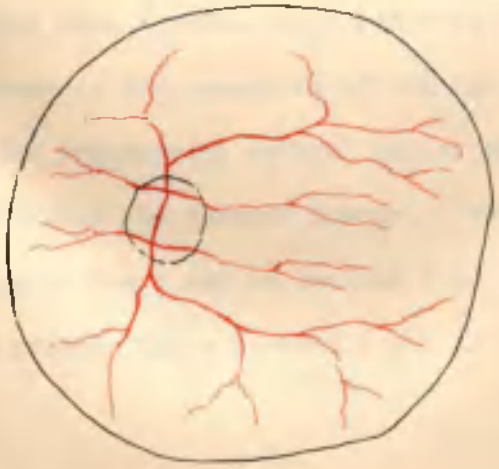


Fig.3 Diagram of Arteries of Retina

Blood Supply of the Eye

For its nutrition the eye depends on several blood vessels and anastomoses. Among those may be mentioned the Arteries of the Optic Nerve Sheath, the Long and Short Posterior Ciliary Arteries, the Anterior Ciliary Arteries and the Conjunctival Arteries. As their name suggests, the arteries of the optic nerve sheath enter the eye by way of the sheath of the optic nerve. Anastomosing with the short posterior ciliary arteries, which pass through the sclera close to the optic nerve, these vessels supply the capillaries of the choroid. The long posterior ciliary arteries, two in number, pierce the sclera on the medial and lateral sides of the optic nerve and pass forward between the sclera and choroid towards the ciliary margin of the iris. There they anastomose with the anterior ciliary arteries to supply the iris and the ciliary body.

The retina, however, has a separate supply by means of the Central Artery of the Retina which is a branch of the Ophthalmic Artery. It enters through the sheath of the optic nerve about 2 cms. behind the bulb of the eye and makes its appearance in the centre of the optic papilla. There it divides into an upper and a lower branch and each of these bifurcates into a nasal and a temporal branch. These four branches then ramify towards the periphery of the retina.

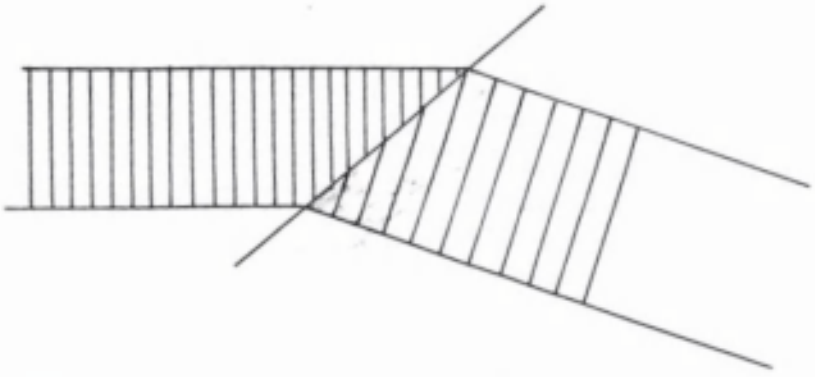
The transparent optical media, on the other hand, receives its nutrition from the aqueous humour. While it is certain that this fluid is a form of lymph and is derived from exudation of the vessels in the ciliary process there is some doubt as to how it leaves the eye.

There are three ways it may do this, namely, by way of the canal of Schlemm in to the ciliary veins, by the veins of the iris and by passing into the posterior chamber and thence into the lymphatics of the optic nerve sheath. Probably all three routes are employed.

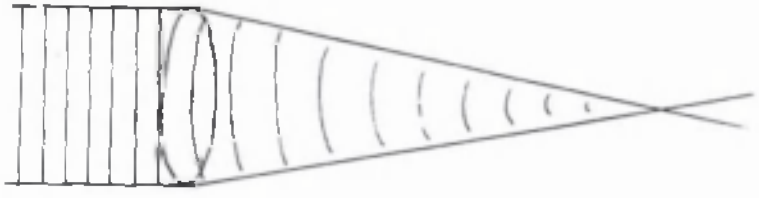
Function of the Eye

The function of the eye is to receive light impressions of external objects and to convey them to the brain. This is done by means of an intricate optical system, consisting of those structures which together focus the images on the retina where changes of a structural, physical and chemical nature take place. The impressions are then conveyed to the brain as impulses along the optic nerve. The optical system includes the cornea, the aqueous humour, the crystalline lens and the vitreous humour.

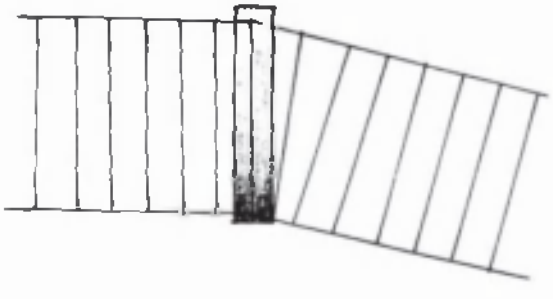
Since the waves, of which the beam of light is composed, travel more slowly in a dense medium than they do in one of less density, refraction occurs whenever light passes from one medium into another of different optical density. Figure 4 shows a diagrammatic representation of various types of refractive structures. In "E", the medium is lens-shaped, as in "B", and varies in density as in "D". The great refracting power of such a structure is well known. This is the arrangement in the lens of the eye in which, therefore, the power of refraction is very much greater than that of an ordinary lens of the same curvature and the same refractive index as the average density of its substance.



A. By inclined Plane

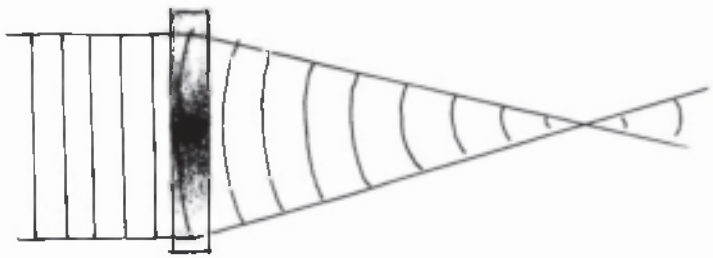


B. By Lens.

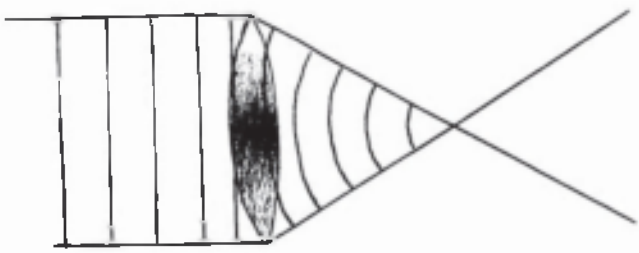


C. By Plate of greater density at its lower end than at its upper.

Fig.4 Various types of Refracting Substances.



D. By Plate of greater density at its centre than at its edges.



E. By Lens of greater density at its centre than at its edges.

Fig.4 (Continued)

The method of varying the focus is called accommodation. According to the theory of Helmholtz, which is the one most generally accepted, the lens, when in the eye, is flattened by the traction of the suspensory ligaments and is therefore focussed for distant vision. By contraction of the ciliary muscle, the tension is released and allows the lens, by its elasticity, to regain a more spherical form and hence focus for near vision.

Intra-ocular Tension

In order that the eye may carry out its function properly it must receive plenty of nutrition. Hence the blood must be able to flow freely into the eye. For that, it is necessary that the blood pressure of the vessels be greater than the tension within the eye-ball. Normally this is so. When measured by the Schiotz tonometer, the eye is found to have a tension of from 20-30 mm. of mercury. Experiments on blood pressures by Magitot and Bailliart (1) have shown that in a normal individual the retinal arterial pressure is 70 mm. systolic and 35 mm. of mercury diastolic. These pressures have been confirmed by Duke Elder. In the disease GLAUCOMA, in which the intra-ocular pressure is raised, we find there is definite impairment of the nutrition of the eye. It is important to note that, in this disease, two of the important symptoms are impairment of vision and pain in the eye-ball.

Since the eye is a sphere, however, its tension will vary as the intra-ocular contents increase or decrease. Those portions of the

contents of the eye, the amounts of which are readily variable are the aqueous, the vitreous and the blood circulating in the inner tunics. According to Fuch[3](2), the last is the most important and every increase or decrease in blood pressure in these vessels must result in a rise or a fall of intra-ocular tension. Because of the small size of these vessels, however, the pulsatory variations of blood pressure are slight and are compensated for by the elasticity of the fibrous coats of the eye. But even great and lasting changes in blood pressure only affect the eye momentarily and not permanently (3). There is a protective mechanism checking the rise or the fall of intra-ocular tension. The real workings of that mechanism is doubtfull. Adler (4) on the one hand, believes that the intra-ocular pressure, whilst tending to vary with arterial pressure, is held in check by local vaso-constriction due to the action of the cervical sympathetics. Starling and Henderson (5) on the other hand, state that the rate of secretion from the vessels of the ciliary processes depends on the difference between arterial pressure and intra-ocular tension. The secretion of the ciliary processes is really an osmotic process and the specific gravity of the secretion varies directly with the blood pressure and inversely with the ocular tension.

Whatever then, the mechanism involved may be, it seems evident that the intra-ocular tension, though varying to some extent with the general arterial blood pressure will almost at once regain its normal pressure.

Let us now consider the general arterial blood pressure of which we have spoken.

General Arterial Blood Pressure

Measurement of the general arterial blood pressure as estimated by the sphygmomanometer is, at the best, only approximate. The maximal pressure existing whilst the heart is expelling blood into the aorta is called the Systolic Pressure whilst the minimal pressure existing at the end of the hearts resting period is the Diastolic Pressure. These pressures are registered as being equal to the pressure exerted by a column of mercury measured in millimetres.

The arterial blood pressure is not constant but is subject to variations depending on several factors. For example an increase in the cardiac output causes a rise in the systolic pressure and to a lesser degree in the diastolic value. Respiration also causes small variations whilst lower oscillations may sometimes be present due to vasomotor changes. Perhaps the factor influencing it most, however, is Emotion. This is especially so at medical examinations. This factor affects mainly the systolic pressure and thus pressures of 160 mm. of mercury may be recorded with a relatively low diastolic pressure. Provided the diastolic pressure is between 60 mm. and 80 mm. of mercury however, one can usually feel certain that emotion is the cause. A poor cardio-vascular efficiency however, will also cause a rise mainly in the systolic pressure. Thus a patient with a sitting pulse of 60 beats per minute may show a blood pressure of 128 mm. systolic pressure and 78 mm. diastolic pressure whilst another

with a sitting pulse of 112 beats per minute might have a blood pressure of 141 mm. systolic and 84 mm. diastolic. Since hyper-thyroidism is usually associated with cardio-vascular inefficiency there is usually a rise of blood pressure present in this type of case.

A rise mainly in the diastolic pressure, on the other hand, is caused by increased peripheral resistance. Thus patients with a past history of Scarlet Fever occasionally show a diastolic pressure of 90 mm. or over, probably due to renal involvement. It has long been recognised that vessels of the splanchnic area and skin constitute the major part of the peripheral resistance. Vaso-dilation or vaso-constriction of the vessels of the splanchnic area alone may cause a variation of as much as 100 mm. of mercury (6). The systolic pressure is of course subsequently affected.

Age is another factor influencing blood pressure. Over 40 years of age there is usually a gradual and steady rise as the age increases. This is due to arterial disease and in the healthy subject it is absent. Between the ages of 18 and 40 there is little change.

There is apparently an increase of blood pressure with increase of weight. Thus among candidates for the Royal Air Force it is found that the pressure rises from 118 mm. systolic, 75 mm. diastolic in the lightest under-weight subjects, to 132 mm. systolic, 82 mm. diastolic in the heaviest over-weight subjects. All of these are usually found to be physically fit. Systolic pressure even below 100 mm. can be found in

perfect health. The subjects are usually found to possess a cardio-vascular efficiency higher than the average and a tendency to under-weight. The emotional factor may mask the presence of a low systolic pressure at the examination, but a diastolic pressure of 70 mm. or under should suggest its presence. Candidates of this type however, are apt to be subject to attacks of syncope.

On the other hand a raised blood pressure may exist in a fit subject. Six per cent of the fit pilots of the Royal Air Force have systolic pressures of 140 mm. and over while two per cent have blood pressures of 140 mm. systolic and 90 mm. and over diastolic. (7). In these subjects there is usually a tendency to over-weight and the cardio-vascular efficiency is slightly less than normal.

The height of an individual has no effect on blood pressure provided the body-build is normal.

Since such variations of blood pressure may exist in physically fit subjects we cannot speak of any value as being the normal blood pressure. We must allocate by experience certain limits within which a normal pressure may fluctuate.

There are other factors, however, which affect individual blood pressures. Thus posture causes a change in the systolic pressure which seems to vary with the individual. In rising from the recumbent position to the standing position however, it would seem that the diastolic pressure is always increased and that there is a constant decrease in the pulse

pressure. The cause of these changes is probably the result of increased cardiac output or of increased vasomotor tone, or as the result of both factors.

Effects of Acceleration

While velocity of itself has no effect on the blood pressure, the rate of change of velocity or, as it is called, acceleration does have an effect on these pressures. Ruff (8) has described an ingenious method of recording the systolic and diastolic pressures even during high acceleration. In a series of experiments on a centrifuge, using this method, he has shown that, in the human being, there is a lowering of the systolic pressure during acceleration. This lowering of the systolic pressure, he says, depends on the amount of acceleration applied. Under 3 g. the pressure does not vary considerably though the pulse rate rises quickly from the beginning.

Fischer (9) has also performed recently some experiments with a centrifuge. By means of an X-Ray apparatus fixed to the centrifuge he took photographs of a series of ten monkeys whilst they were rotating. In a series of pictures he shows that during acceleration there is a decrease in the filling of the heart and at the same time the heart is turned and pushed downwards. This decrease in filling increases in extent with increase of acceleration. With the emptying of the heart the diameter of the aorta and the vena cava also decreases. His experiments also show that a great deal depends on the direction in which the acceleration is applied. If it takes place in the head to feet direction the above changes take

place but if it is applied in the direction of front to back the filling of the heart is satisfactory up to 10 g.

By applying a low centrifugal force to six dogs which had been previously anaesthetised Koenen and Ranke (10) have shown that the blood pressure falls immediately but rises again after 18 to 20 seconds. This, they say, is in accordance with the reflex time of the carotid sinus. With forces above 4 g. however the carotid pressure is always below zero. In the dog however the pulse rate apparently falls from the beginning owing to some irritation of the vagus which is left unexplained.

It is evident then, from these observations, that acceleration of sufficient amplitude will cause a fall in the systolic pressure. This is due to a decrease in the filling of the heart and takes place apparently only when the acceleration is applied in the direction of head to feet.

Effects of Altitude.

It is generally agreed that altitude tends to lower the blood pressure. According to Norris, Bazett and McMillan (11) this is due mainly to oxygen deficiency. The effect is more marked in those with a low blood pressure but it is impossible to forecast from a person's general physique what the result will be. Amongst aviators, who are subjected suddenly to the atmospheric changes dependent on altitude, two reactions are observed. Either the pilot faints and does not regain consciousness until he has lost a great deal of height or else he is aware of an increasing lack of attention and concentration, when every movement is irksome. According to Schneider and Truesdell (12) the type of reaction depends on whether the circulatory centre or that controlling motor action and co-ordination is the first to fail. Not only is the reaction an individual one but the height at which it takes

effect also seems to depend on the particular person concerned. There are however very few pilots who can exceed 20,000 feet without the use of oxygen. Fortunately this is not now necessary as all aeroplanes which are expected to ascend higher than 15,000 feet are fitted with an oxygen apparatus and the pilot begins to use it at 5,000 feet. Slight elevations however produce little change. In experiments performed by Clough (13) the readings at 5,000 feet were practically identical with those at sea level whilst miners ascending or descending 17,000 feet showed variations hardly exceeding 5 mm. of mercury.

The Carotid Sinus.

Since so many external influences may affect the blood pressure there must be some form of protective mechanism. We find this in the carotid sinus. Situated at the bifurcation of the common carotid artery the carotid sinus is very sensitive to changes of internal pressure. Normally the sinus nerves exert a tonic inhibitory influence on the vaso-motor centre and by perfusion experiments it can be shown that a rise of internal pressure will cause a vaso-dilation mainly in the splanchnic area. The sinus is remarkably sensitive and even a deviation of 10 mm. will cause a reflex response. Koch, in experiments on the isolated innervated sinus has shown that, up to a point, the greater the pressure the greater the reflex fall and that the normal blood pressure of the species is the region in which the most sensitive sinus reflexes are obtained.

SYMPTOMS.

In a preceding section I mentioned that there were approximately 50 pilots on the station, all of whom admitted that they, at some time or other, had "blacked out". To compile a list of symptoms, therefore, seemed, at first, an easy task. Surely interrogation of all the pilots would reveal the common sensations and a few tests would be sufficient to make an accurate list. This, however, was not to be. Interrogation of all the pilots only revealed that none of them could say more than that, during certain evolutions, everything went black. It was apparent then that only by inviting them to black out purposely could anything further be learned. Thus I made it known that I would like volunteers to black out, by any method they liked to adopt, and record to me afterwards the sensations experienced. In order that I might have an idea of the condition myself and be able to ask intelligent questions I also participated in these tests. Thus on several occasions, whilst flying as a passenger, I had the doubtful pleasure of being "blacked out". It is not a pleasant experience and, on the first occasion at least, is a little frightening. There was no scarcity of volunteers and it was evident that the pilots were only too willing to do all they could to reduce this limitation.

For several months these pilots performed test after test, whenever their other duties permitted. None of the pilots failed to "black out" though reports on the ease with which it was accomplished varied tremendously. This gave rise to the thought that it was easier to accomplish on some days than on others and that some pilots "blacked out" more readily than others. As these tests were not standardised in any way, no accurate record of the flights was kept but the sensations experienced in each case were carefully noted. These

were repeated again and again and gradually a list of symptoms was compiled. The common sensations are as follows:

1. The sensation of being pressed into the seat and that the contents of the abdomen are increasing.
2. Gradual dimness of vision.
3. Sudden blindness.
4. Blindness passes off.

The sensations occurred during any manoeuvre which involved a rapid change of direction such as pulling out of a dive into a loop or turning quickly to one or other side with a steep bank. First of all the pilots had the sensation of being pressed, steadily but strongly into their seats. At the same time they experienced a peculiar feeling of fullness of the abdomen. There was no tendency to sickness nor nausea but merely the feeling that the stomach was being displaced downwards. A dimness of vision then appeared affecting the whole field at once. At least it was not possible to say that this blurring of vision commenced in any one part of the field before another. One was only aware that vision as a whole was blurred and that only bright objects were seen clearly. But this did not last for long. With alarming suddenness complete darkness occurred and the pilots were unable to see anything at all. This sensation was so alarming that, for the moment one could concentrate on nothing else. Gradually, however, the pilot was aware that none of his other senses seemed to be affected and that he was quite conscious. This blindness, however, was not of long duration. Within approximately three seconds, and with the same suddenness with which it appeared, the blindness passed off and once more the pilot could see the relative position of his plane to the ground. Vision returned throughout the whole field at the same time

and the bright objects were the first to be recognised. The duration of the complete sequence of symptoms was approximately 5-6 seconds.

During these experiments, several of the pilots complained that, after a flight during which they had blacked themselves out repeatedly, they found difficulty in mental concentration. It was difficult to assess the percentage of pilots thus affected as not all of them are required for duties other than flying. But all of those whose duties entailed office work, found that, on landing and applying themselves to these routine duties, they did not seem able to think clearly nor systematically and that they felt tired and sleepy. I myself experienced this, but with it I had the peculiar sensation, not reported by any of the others, that, on alighting from the aircraft, my legs were moving automatically and that I did not have complete control over them. It must however be borne in mind that I am not yet an experienced flyer nor am I accustomed to "blacking out". Also, I have experienced the same sensation on former occasions after nervous tension.

One pilot reported that invariably during the "blacking out" he perspired freely. This was not experienced by any of the others though special note of the symptom was made during repeated tests. This same pilot also complained of another sensation not experienced by any of the others, namely that just before blacking out he felt that the blood was draining away from his head. As he is a very able man and usually accurate in his observations, I have no doubt that he actually did experience these symptoms.

On one occasion another pilot reported that he had fainted. His report stated that he had been endeavouring to black out by maintaining a rapid turn with very steep bank. After about two turns, during which he experienced the usual manifestations he became unconscious. This apparently differed from an

ordinary faint for on recovery he found that he was sitting upright and that his plane was not markedly out of control. Though he made several attempts he could not repeat the performance. This is not surprising as, on that occasion, he had no method of estimating his rate of turn. But the possibility of the pilots becoming unconscious has been confirmed. One of our medical officers, himself a pilot, reports that, while flying as a passenger, he was blacked out and that on that occasion he became unconscious. He says that, on regaining consciousness he found that he had not gone flaccid as might have been expected and that he was still holding the speaking tube.

It is interesting to note that on no occasion did the pilots complain of any pain in the eyes nor was any sensation felt in the lower limbs.

It was not practicable to examine the pilots before each flight but this was done, as soon as possible, after they had landed. The result of this examination was checked with the record of the pilots last examination. These examinations proved negative and, with one exception, no variation worthy of note was detected. Invariably the pilots blood pressure was lower than normal. After landing, the pressure rose gradually and, in most cases, reached its normal level within two hours. In a few pilots, however, the time taken to return to normal was greatly increased.

The age of the pilot did not matter at all. None of the experienced pilots found any difficulty in blacking out. This was not so with the less experienced ones and on several occasions these pilots reported that they had succeeded only with difficulty. This, I am confident, was mainly due to their inexperience and lack of confidence. At the same time it was noted for consideration at a later date.

Since the commencement of these tests in 1936, reports have been received continually but so far no further symptoms have been noted. On no occasion during that period have I heard of any disability among the pilots which was likely to have been caused or aggravated by the condition. Apparently it does not injure their general health in any way nor affect their general physical fitness.

INVESTIGATION

For the investigation it was not possible to use all the pilots and I had to be content with a selected few. Thus, from among the keenest of the pilots I chose ten to perform the proposed tests. Because of the nature of these tests the pilots were selected for their powers of observation irrespective of their age or physical efficiency. With the exception of one pilot whose age was 35 years there was a difference of only two years between the age of the youngest and that of the oldest while, for practical purposes, the general physical efficiency of all the pilots was equal. In blood pressure the highest reading was separated from the lowest by 10 mm. of mercury.

So that the experiments might be standardised and as safe as possible, certain regulations were laid down. Firstly, all the experiments were to be performed at a height of 5,000 feet. This height was chosen for the following reasons. In aviation the higher a pilot flies the more safe he is should anything unforseen happen to the machine. Although every pilot must wear a parachute, it is not safe to use this at a height of less than 2,000 feet. As a pilot, flying "blind", might lose many hundreds of feet without realising it, it was essential that the chosen height be greater than this. But physiological conditions had also to be considered. The higher a pilot climbs the more is his blood pressure affected by atmospheric conditions. As some limit had to be made this was set at 5,000 feet. At this height it was thought that the standards of safety were observed and yet the pilots were not high enough to be affected by atmospheric conditions. The tests were all carried out in ordinary Service single-seater aeroplanes of the same type and the pilots were fitted as for ordinary flying with flying suit,

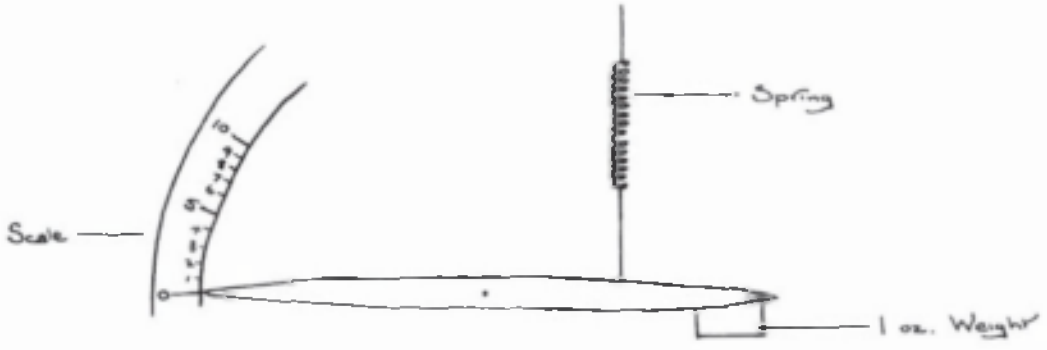


Fig. 5

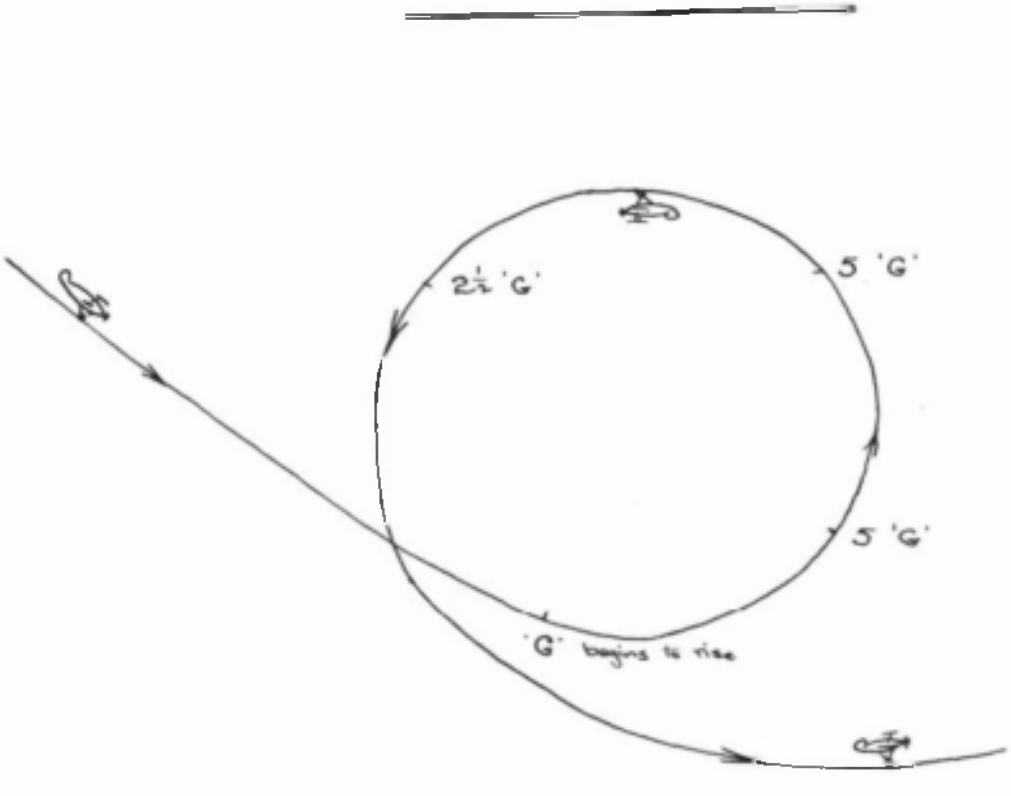


Fig. 6

parachute and oxygen apparatus. At this height, of course, the latter was not used.

The preliminary tests had shown that, since "blacking out" was caused by rapid changes of direction at high speed, it must be due in some way to acceleration. Provided the speed and rate of turn is known, the acceleration acting on the machine can be calculated from the formula $G = \frac{V^2}{r}$. But at a height of 5,000 feet it is impossible to calculate accurately the rate of turn. Hence some other method of acquiring the acceleration value had to be devised. For the purpose an accelerometer was made in the following manner. A small lever was fixed at its central point. To one end was attached a 1 oz. weight while the other end moved freely along a scale. The lever was held in a horizontal position by a spring which exactly counterbalanced the weight (diagram 5). The point thus denoted on the scale was marked as zero. By adding another 1 oz. weight, the pointer was made to move a certain distance along the scale and this point was marked "1.G". This process was repeated again and again until the scale was marked off in 1.G intervals up to 10.G. The instrument was then fixed firmly in the aeroplane so that the lever moved in a vertical direction. Periodically the instrument was tested by adding the weights and checking the points on the scale. Though I cannot vouch for the accuracy, the instrument did set a standard and was the same for all the pilots.

Test No. 1.

This test was designed to find at what G value the pilots blacked out. The pilots were asked to fly at 5,000 feet and from there to perform loops, increasing the G value each time until they blacked out. Each pilot carried a stop watch and with this he endeavoured to measure the duration of the

black out.

During the first attempts low values of G only were recorded and none of the pilots blacked out at all. It was observed during these attempts that immediately the pilot pulled the stick back to enter the loop the G value began to rise. It rose rapidly until a certain value was reached and then remained stationary for a time. The speed of the aeroplane remaining constant, the rate of increase in G value varied directly, of course, with the amount of pressure applied to the stick. Nearing the top of the loop, however, the G value began to drop again as soon as the speed of the aeroplane began to decrease. This is illustrated in diagram 6.

Each pilot performed many loops noting the G value recorded and as far as possible the position of the aeroplane in the loop. On blacking out, he noted the G value recorded, the position of the aeroplane in the loop and also endeavoured by means of the stop watch to record the duration of the blacking out. When one remembers that he had also to control the aeroplane one cannot place too much reliance on the accuracy of the figures obtained. The test was repeated many times, however, by each pilot and an average of each man's figures was taken.

All the pilots did not black out at the same G value nor did each individual pilot black out at the same value each time. Likewise the duration of blacking out varied considerably with each attempt. It was noted however that the higher the G value recorded the shorter was the duration of blacking out. This is to be expected for the high G value means that either the speed was increased or the radius of the loop was decreased. Thus in either case the time taken to reach the top of the loop was decreased. The highest G value recorded before blacking out took place was 7 G whilst the lowest value

TABLE OF "G" VALUES IN TEST NO.1.

Manoeuvre	"G" Value	Time taken for manoeuvre	Time "Blacked out"
Loop	4	11 secs	nil
"	5	11 secs	nil
"	3	12 secs	nil
Pulling out of a dive	6	15 secs	5 secs
"	5	60 secs	5-6 secs
"	5	60 secs	3 secs
"	6	13 secs	4 secs
"	7	11 secs	2 secs

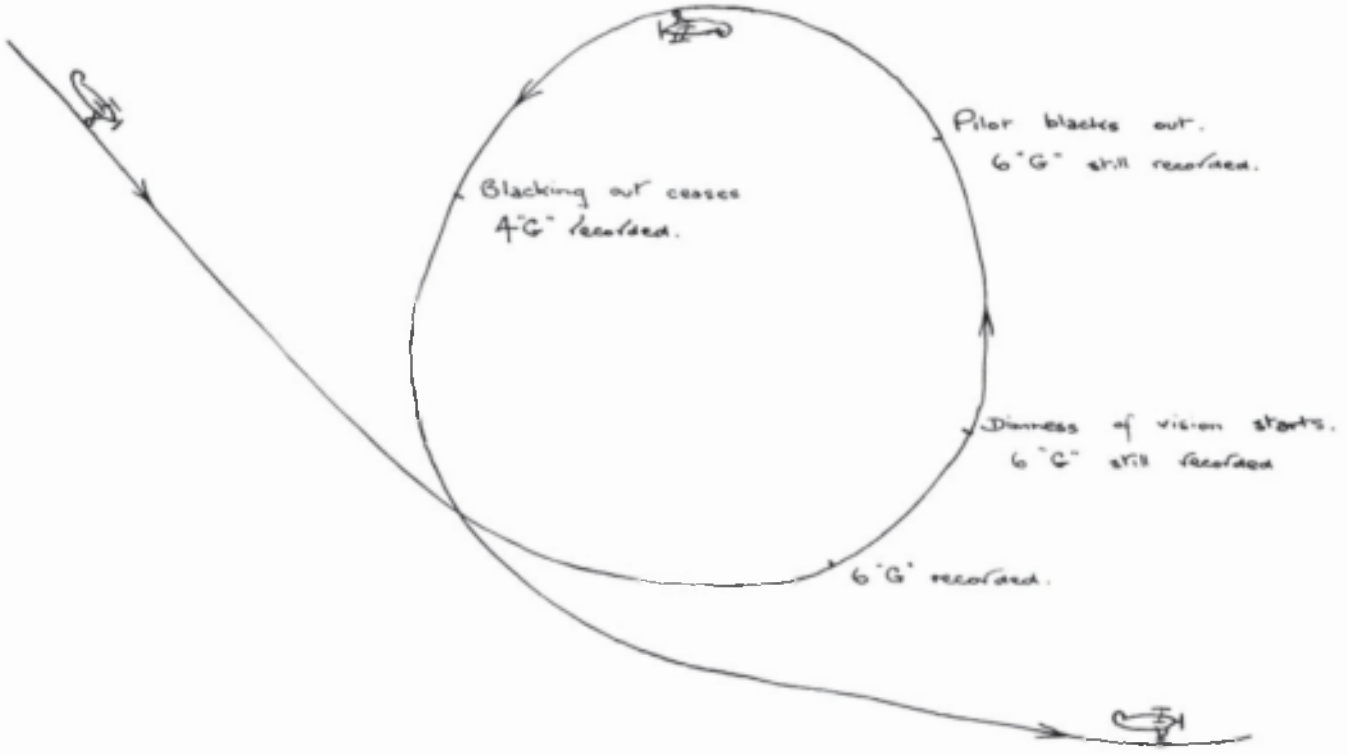

Pilot blacks out.
6 "G" still recorded.
Blacking out ceases
4 "G" recorded.
Dimness of vision starts.
6 "G" still recorded
6 "G" recorded.

Fig. 7

at which it occurred was 5 G. The duration of blacking out varied from two seconds to five seconds. It was observed that some of the pilots persistently recorded low G values whilst the others always returned higher G values. Thus of the ten pilots four had an average of approximately 5 G whilst the other six had an average of between 6 and 7 G.

The most important finding from these tests however, was common to all the pilots. None of the pilots blacked out immediately the highest G reading for the manoeuvre was recorded but only after that value had been maintained for some appreciable time. This may be illustrated as shown in diagram 7.

These tests show that pilots black out only when subjected to a sufficiently high G value maintained for a certain period of time. It would seem that the amount of G necessary varies with the individual.

Whilst it has been shown that, with the lowest G value necessary to produce blacking out, it is necessary for this value to be maintained it does not follow that higher G values will not cause blacking out immediately they are applied. To show that this is not so the following tests were performed.

Test No.2.

The pilots were asked to fly as before at 5,000 feet and then, by means of pulling out of a dive, to record as high a G as possible in as short a time as possible but not to maintain that G value. This test, of course, was not without danger. There is naturally a limit to the amount of G one can apply to an aeroplane and if one exceeds that the aeroplane will break into pieces. I made enquiries, however, and was informed by a test pilot that the modern aeroplane is stressed to withstand 9 G. During their trials this figure is exceeded however and I am told that, in practice, the aeroplanes will resist from 12 to 15 G.

Owing to the dangerous nature of the test the experiment was limited to two pilots both of whom blacked out at approximately 7 G. They were instructed not to exceed 9 G thus leaving what seemed to be a reasonable margin of safety. They were to apply the G value as suddenly as possible but were not to maintain it. In each case the pilot easily recorded the required figure and on one occasion one of the pilots exceeded it.

In no case did the pilot black out nor did he experience any of the preliminary symptoms.

Blacking out is not caused therefore by a suddenly applied acceleration of values up to at least 9 G. It would appear then, that, for the blacking out, the G value not only be of sufficient magnitude but also that it be maintained. This is most important from the point of view of physiology and will be discussed later.

In test No.1, I have shown that whilst performing a loop the pilot of an aeroplane will black out if he maintains a certain G value for a definite period of time. If this were the only manoeuvre in which blacking out occurs it would seem that the position of the pilot is also an exciting factor.
In performing the loop the pilot is always upside-down during the blacking out stage and therefore liable to stasis in the cerebral vessels. But a pilot may black out whilst performing other manoeuvres.

Test No.3.

For the purpose of this test the pilots were again asked to fly at 5,000 feet and after attaining sufficient speed to turn quickly with steep bank and hold the machine in the turn for as long as possible. During this manoeuvre the pilot may be inclined at an angle of anything under 90° from the vertical. At no time however, is his head situated lower than his heart or

other portion of his body. High G values may be recorded in such a turn and may be maintained for a longer time than is possible in a loop. But even here there is a limit for sooner or later the speed of the machine decreases.

During this experiment the pilots found that they blacked out as before at from 5 to 6 G but that they could prolong the period of blacking out much longer. Most of the pilots could prolong the blacking out for from 5 to 6 seconds and in one case the pilot succeeded in maintaining it for from 10 to 12 seconds.

It was also found during this test that by maintaining a low G value of from 4 to 5 G, it was possible to experience the dimness of vision without actually blacking out. In no case however did the pilot actually become unconscious.

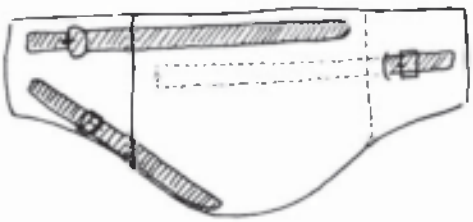
Thus we see that in maintaining various degrees of G different symptoms are experienced. By maintaining a low G value no symptoms at all are felt. If we increase the G however and maintain it we may experience dimness of vision whilst if we increase it still further blacking out takes place. It may be that the stage of unconsciousness is only obtained by maintaining a still higher value of G. Some of the pilots were anxious to try and demonstrate this but as the risk is enormous I was not prepared to accept the responsibility.

From these tests it is seen that the position of the pilot, relative to the ground, is not an important factor. As regards the position one factor only is common to both manoeuvres. That is that in both cases the head of the pilot is directed towards the centre of rotation and thus the acceleration is acting in a head to feet direction. In performing an out-side loop, a manoeuvre which is now forbidden, the pilots, I am told, did not experience blacking out but suffered from what was called "redding out". In that every-

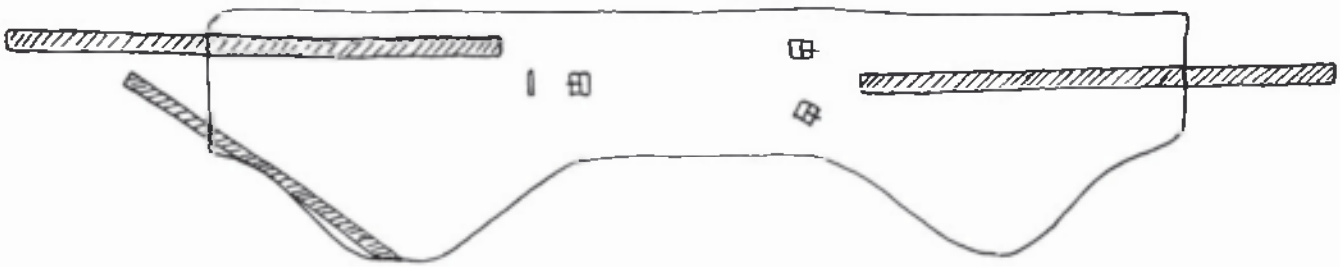
TABLE OF "G" VALUES IN TEST NO.3.

Manoeuvre	"G" Value	Time taken for manoeuvre	Time "Blacked out"
Steep turn	5	9 secs	dimness of vision only
"	5-6	60 secs	10-12 secs
"	5	14 secs	2 secs
"	4-5	15 secs	nil
"	7	9 secs	dimness of vision only

Back View



Front View



Belt stretched out -

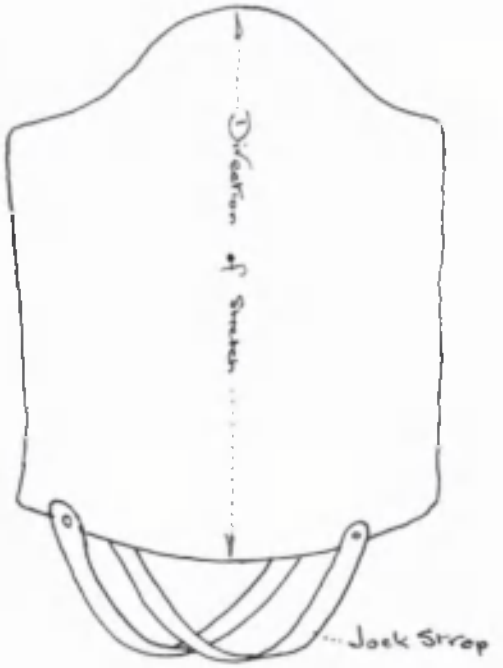
Fig. 8

thing became red. During this manoeuvre the pilot's feet were directed towards the centre of rotation and the acceleration was acting in a feet to head direction.

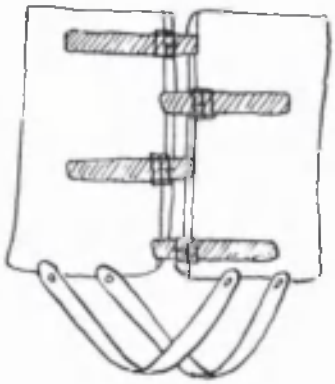
That "blacking out" was due to the effects of acceleration on the circulation now seemed clear and it was decided to try the effect of wearing an abdominal belt. It was thought that such an appliance would relieve any drag on the mesenteries caused by the acceleration acting on the abdominal organs and that it would also prevent the blood pooling in the abdominal vessels. It was realised that the abdominal vessels did not constitute the whole of the peripheral circulation and also that, during the acceleration, the legs are also placed hydrostatically downwards. But it was felt that the abdomen certainly contained the greatest part of the circulation and that the pooling in the other parts of the body was probably negligible.

Two different types of abdominal belts were tried and the results were as follows. The first was more in the form of an abdominal binder and was intended merely to assist the abdominal muscles in maintaining their tone. It was made of stiff buckram and lined inside with silk. The front, which was of double thickness, was shaped to fit down over the pubis and to lie along the line of the groin. Above, it reached to the level of the 10th rib. (See diagram 8).

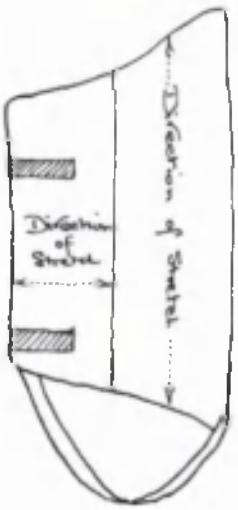
The belt was made to fit one pilot and could not be worn by any of the others. Several tests were made but with no success. The belt did not fit closely to the abdomen, was uncomfortable to wear and chaffed the skin. When the pilot sat down the front of the belt bent forwards and formed creases. No matter how firmly the lower straps were fixed this could not be eliminated. It had no effect whatsoever on the point at which the pilot blacked out and merely



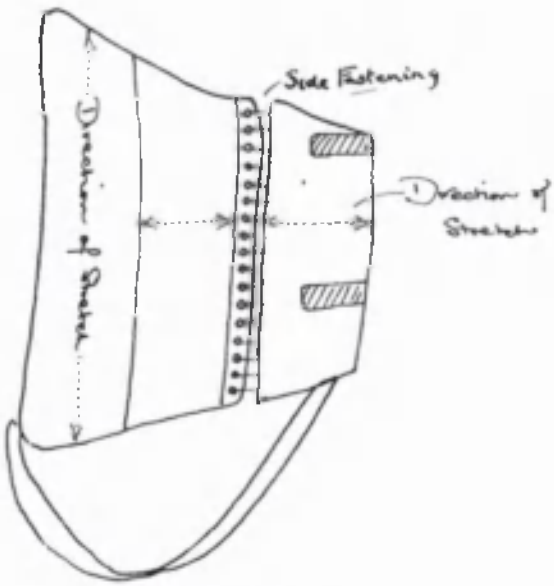
FRONT.



BACK.



RIGHT SIDE.



LEFT SIDE.

Fig. 9

made it impossible for him to remain in the air.

A second belt was therefore designed and constructed, this time in the form of an abdominal corset. It was intended to fit more closely to the abdomen and to act as support to the organs as well as to increase the muscular tone. This belt was made of semi-elastic tricot, a material which stretches slightly in one direction only. The front of the belt, where the elasticity was in a vertical direction, was made in one piece and reached from the lower end of the sternum to the pubis. The top was shaped to fit just over the ribs whilst the lower end just covered the crest of the ileum. A jock-strap was fitted to keep the lower end in position when the pilot sat down. At the back and sides the belt was designed to stretch slightly in a horizontal direction. Four straps were fitted at the back so that the tension on the front could be varied at any point. The belt was fastened down the left hand side, and was worn next to the skin. (See diagram 9).

At first the belt was tried with all four straps drawn tightly. This made it slightly uncomfortable as it restricted breathing and caused a desire to micturate. The pilots still blacked out but felt that they took longer to recover. This was thought to be due to the constriction of the diaphragm for, if the blood was pooling in the vessels of the abdomen this constriction would prevent its return. Accordingly the belt was tried again but this time the upper straps were loosened sufficiently to make respiration easy and comfortable. This time it was found that whilst the pilots still blacked out as before there was no feeling of weight in the abdomen. Also in most cases the dimness of vision preceding the blacking out was not so marked and on landing there was no feeling of fatigue.

It was therefore decided to apply more pressure to the middle of the

abdomen and so a further attempt was made. On this occasion a pneumatic pad containing a very small quantity of air was inserted between the belt and the abdomen at the level of the umbilicus. After many trials, during which both loops and steep turns were performed it was found that the belt eliminated the effects of maintaining G values up to and including 6 G as recorded on my accelerometer. Blacking out still occurred at 7 G but it came on without any preliminary signs. The maintenance of lower G values did not produce dimness of vision as before. This was checked by repeated tests with and without the belt.

One other test was carried out with the belt. A pilot from another station, who blacked out easily and had often become unconscious, consented to try the effects of the belt. Whilst flying as a passenger in a two-seater aeroplane, this pilot was first of all blacked out during a steep turn. The manoeuvre was then repeated with the belt in position. No pneumatic pad was used and no pressure other than that of the tightly drawn straps was applied. In his report afterwards he stated that, when he was not using the belt, he had blacked out and remained in that condition until the end of the manoeuvre which lasted for approximately 30 to 40 seconds. On the second occasion the same G value was recorded as before and the manoeuvre lasted for approximately the same length of time. His report for this manoeuvre stated that, whilst he had blacked out, he had not remained in that condition. The blindness had alternated several times with periods of dimness of vision and had never been so complete as on the previous test. It is interesting that this man is a hypotensive having a blood pressure normally of 92 mm. systolic and 55 mm. diastolic. On landing after the first test his blood pressure was found to be 77 mm. systolic and 55 mm. diastolic. After the second test his blood

pressure, on landing, was 115 mm. systolic and 80 mm. diastolic whilst after one hour it was 105 mm. systolic and 64 mm. diastolic. By the next day it had returned to its normal value.

These experiments with the abdominal belt show several things. First that by applying pressure to the abdominal wall it is possible to alleviate to some extent the effects of acceleration and that the more pressure one applies the greater is the benefit obtained. It also shows, however, that, in the normal individual at least, the pressure required to prevent blacking out is quite considerable. This is due to the fact that one can apply pressure to only one wall of the abdomen. The diaphragm, which is another wall, must be left free as restriction of this will merely prolong the condition. These factors certainly suggest that there is a pooling of blood in the abdomen but, of course, do not prove that this does not occur elsewhere as well. They do suggest, however, that the abdominal pooling is by far the most important and that its complete prevention, providing it can be done without discomfort, will prevent blacking out during any acceleration likely to be encountered in present day aeroplanes. The last test shows that, in the hypotensive at least, the belt does increase the abdominal tone and so increases the blood pressure.

As there is some resemblance in the symptoms of blacking out to those of oxygen lack it was decided to try the effect of increased oxygenation of the blood. Thus several tests were made during which the pilots, whilst flying at 5,000 feet, used the requisite amount of oxygen for firstly 10,000 feet and later for 15,000 feet. No benefit whatsoever was obtained and in fact there was no difference in the symptoms.

This concluded the investigation. During the experiments the

following factors were demonstrated.

1. That "blacking out" only occurs after a certain acceleration is applied.

2. That the requisite amount of acceleration is different in different individuals.

3. That the acceleration must be maintained before the symptoms occur. They are not produced by the sudden application of even high values of acceleration so long as it is not maintained.

4. That increasing amounts of acceleration produce increasing degrees of the condition.

5. That the condition can be alleviated by pressure on the abdomen and that the greater the pressure the greater is the relief obtained.

6. The more easily an individual blacks out the greater is the benefit obtained by pressure on the abdomen.

7. Restriction of the diaphragm prolongs the effects of the condition.

8. No benefit is obtained by increased oxygenation of the blood.

These points are all of value in helping us to find the cause of the condition and also in finding some practical method of prevention.

GENERAL DISCUSSION.

As we have seen the symptoms of "blacking out" are impressive. This is especially so in the case of the blindness during which none of the other senses are affected. It is natural therefore, that, in seeking a cause for the condition, our thoughts should turn to this symptom. Why should vision alone be impaired whilst other parts of the brain continue their function? It is true that unconsciousness does occur but only when higher accelerations are employed. That the condition is due to the effects of acceleration there can be no doubt. The earlier experiments showed that it only occurs when an acceleration of sufficient magnitude is being maintained and its degree depends on the amount of that acceleration. But why should acceleration affect an an individual in that particular way? Let us try to find the reason, working backwards from these symptoms and using all the knowledge at our disposal.

Cause of Loss of Vision.

Sudden loss of vision may be produced in two ways, namely by injury to any part of the optical system or by anaemia of any part of that system. The first of these may be dismissed summarily. Any injury, sufficiently severe to cause blindness, would not be so evanescent and cause such a temporary defect as is found in "blacking out". Such an injury would require to be due to movement of the brain under the acceleration and so the greater the force of the acceleration the greater would be the injury. But this is not so with "blacking out". We have found on investigation that one essential point is that the acceleration be maintained and that great accelerations can be applied suddenly without effect. The fact that a latent period is present is very important. It shows immediately that the effects of the acceleration are somehow connected with the circulation - in other words that the effects are

due to anaemia. But which part of the optical system does this anaemia affect? Is it a part inside the cranium or is it the eye itself? The brain we know, is semi-fluid and capable of movements under acceleration in which case pressure might occur at various points and cause anaemia. For example pressure might occur on the calcarine fissure with resultant blindness of conscious perception or the brain might be pressed into the foramen magnum with occlusion of the vascular supply. But none of these suppositions will explain why only part of the brain is affected and why, except in extreme cases, only the sense of vision is impaired. Certainly it is possible for one part of the cerebrum alone to suffer from anaemia for, because of the falx cerebri and falx cerebelli pressure is not communicated throughout the cranial cavity. But during acceleration the whole brain will move, if at all. And it is possible for even small movements of the brain to cause unconsciousness. Some hold that mere vibration of the brain will interfere with its function, putting it crudely, by a dislocation of the neurones. Duret holds that cerebral anaemia may be caused by a wave of cerebral fluid stimulating the restiform bodies. But that, according to McDowall, is rather conjectural. Certainly we must look further than pressure on the brain itself for our explanation of "blacking out". We are now left with the explanation that it is due to anaemia of the eye itself. Can this hypothesis explain all the symptoms? I, myself, think that it does.

We have already seen that the retina of the eye has a blood supply of its own by way of the Central Artery of the Retina. If then this artery is occluded the retina will suffer from anaemia and blindness will occur. The artery enters the eye via the optic nerve which it pierces 2 cms. behind the bulb of the eye. A wave of cerebral fluid, therefore produced by the

acceleration and passing along the sheath of the nerve could compress this vessel. This probability, however, can be rejected on the grounds that once again the effects would be evanescent and sudden. But there is another way in which the central artery of the retina might be occluded.

We have seen that blood enters the eye by virtue of the fact that the blood pressure existing in the central artery of the retina is greater than the intra-ocular tension. If then this balance of pressure is upset the retina will suffer from anaemia. It is obvious that this balance may be upset in two ways. Firstly the intra-ocular pressure may be increased until it is greater than the pressure in the central artery of the retina. This happens in the disease Glaucoma in which one of the principal symptoms is pain in the eye. But in "blacking out" there is no pain in the eyes. The increase in pressure could occur during acceleration, and probably does, by the eyeball being pressed against the floor of the orbit. But I do not think that this pressure is great enough, of itself, to cause the blindness. It may, however, increase the intra-ocular tension to a slight degree and thus may have an adjuvant effect. Since the eye has no support anteriorly it is more likely to cause an antero-posterior stretching affecting the continuity of attachment of the retina. This, however, does not explain the symptoms experienced in the more severe degrees of "blacking out".

The other obvious way in which the balance may be upset is by a decrease in the blood pressure existing in the central artery of the retina. That this is the actual cause of "blacking out" is suggested by Andina (16) and with him I entirely agree. Ruff (8) has shown that, with accelerations of over 4 G, there is a lowering of the systolic pressure and an increase of pulse rate. The blood pressure in the central artery of the retina will naturally be

affected. It must be remembered that the pressure in that vessel is not the same as the general arterial blood pressure but there is a definite relationship between the two. According to Magitot and Bailliart (10) and Duke Elder (15) the ratio is generally 0.5 to 1. Thus the pressure of the central retinal artery is approximately 60 to 70 mm. systolic and 35 mm. diastolic. The intra-ocular tension varies from 20 mm. to 30 mm. of mercury.

It has been shown by Andina (16) that, by external pressure on the eyeball, symptoms can be produced which are identical to those experienced in "blacking out." By means of a dynamometer he has recorded the pressures necessary to cause these symptoms. Correlating them with the pressure in the central retinal artery he states that before blacking out occurs that pressure must fall to 21 mm. of mercury. He states definitely that he is presupposing that it is possible to make such measurements with the dynamometer and that the intra-ocular pressure remains constant when the blood pressure falls suddenly. I do not agree, however, that it is necessary to presuppose the latter. There is, as we know, a protective mechanism regulating the intra-ocular pressure and though this must be affected by changes in blood pressure, we are assured by Scarlet (3) that the effects are only momentarily and not permanent. The initial fall in pressure must be slight since it is mainly due to fall in pressure in the vessels of the outer tunics of the eye. This can easily be compensated by the elasticity of the fibrous tunics and by the increase in tension due to pressure of the eyeball on the floor of the orbit. Again I do not believe that the blood pressure drops suddenly as Andina suggests. It is a characteristic of the condition that some time must elapse, after the application of the acceleration before the symptoms appear and I feel sure that this is sufficient for the intra-ocular tension to readjust itself. The theory

of a fall in pressure in the central artery being the immediate cause of the visual symptoms seems to me to be more than a possibility. It not only explains the visual symptoms but when carried further will explain also the other symptoms. That it does not cause permanent blindness is probably due to the fact that it is never sustained for a sufficient length of time.

Cause of the Cerebral Symptoms.

During accelerations of over 4 G there is a lowering of the blood pressure (10) and all the cerebral vessels are affected. Since the intra-ocular tension must be overcome before the retina can be supplied with blood, there is a stage at which the blood cannot flow into the eye and yet is able to supply the brain. This is the stage, existing during the lesser accelerations, in which there is blindness but no unconsciousness. As the accelerations are increased, however, the fall in blood pressure becomes so great that cerebral anaemia occurs and unconsciousness is experienced.

Cause of Lowering of the Blood Pressure.

The symptoms of "Blacking out" can therefore be accounted for by the theory that during acceleration there is a fall in blood pressure. But how does this fall occur? Is it that, since the acceleration is in the head to feet direction the blood is shot down the carotids or is it due to some other cause? Certainly at the beginning the first of these is bound to occur but it cannot be the cause of "blacking out". If it were the symptoms would occur immediately high accelerations were applied. They would disappear while the acceleration was still acting and would not occur at all with a gradually applied acceleration. Immediately the blood is thrown down the carotids it will be pumped back again by the heart and there will never be more than a temporary anaemia. According to Koch, the carotid sinus is

extremely sensitive and as the pressure decreases the heart will be stimulated to increase its out-put. There will, of course, be a slight delay in accordance with the reflex time of the carotid sinus. This actually does occur. Koenen and Ranke (10) found that on applying accelerations to anaethetised dogs by rotating them on a centrifuge the blood pressure at first drops but rises again after 18 to 20 seconds. During accelerations of more than 4 G, however, the carotid pressure is always below zero. Fischer (9) has gone further and in an excellent paper describes several experiments performed on monkeys. By means of mounting an X Ray apparatus on a centrifuge he has X rayed these monkeys under different degrees of acceleration applied in different directions. His photographs show that whilst the acceleration is acting in the head to feet direction there is a decreased filling of the heart with increasing acceleration and at the same time the heart is turned and pushed downwards. The diameter of the aorta and vena cava decreases with the emptying of the heart. The effects he assures us are more sudden with greater accelerations but the duration of the acceleration is also of importance.

Thus the blood must be held up in the systemic circulation, in fact, in those parts which are placed hydrostatically downwards such as the abdomen and the legs. The heart is working at an increased rate but cannot raise the pressure as it is not receiving sufficient blood. The abdomen can accommodate vast quantities of blood and it is likely that most of the blood is held in these vessels. During the experiments with the abdominal belt it was noted that when this was used there was a certain degree of improvement. By its pressure on the abdomen the belt was doing two things. It was increasing the amount of tone in the abdominal wall and it was supporting the

abdominal organs and preventing the drag on the mesenteries. Both of these help to reduce the amount of dilatation of the abdominal vessels. That the belt failed to relieve the symptoms even more may be due to several factors. The belt was an experimental one and the design was not perfect from the point of view of relief. For example it restricted the action of the diaphragm which in itself prevents the return of blood to the heart. Moreover the abdominal wall is by no means the only boundary of the abdomen but there is no way of immobilising the diaphragm nor the pelvic floor. Thirdly pooling probably takes place in other parts of the body such as the legs. As regards the latter however I feel that if the pooling of the abdomen could be entirely eliminated this would not matter. The amount of blood that could be accommodated in the legs must be slight in comparison to that in the other. During the tests one other point was noted. When the diaphragm was restricted the symptoms were prolonged. This showed that the blood was being held up somewhere below the diaphragm. The feeling of fullness of the abdomen during "blacking out" is probably due mostly to the increased gravity of the abdominal organs. The drag on the mesenteries caused by this increased gravity of the organs cannot of itself be the cause of the pooling for this can be relieved by means of a belt. And yet when the belt was applied tightly, the diaphragm being left free, the "blacking out" still occurred. Thus it must be due to something else. This other factor I believe to be increased viscosity of the blood due to the increased acceleration. Not only will acceleration cause an increase in the viscosity but as the acceleration increases so does the viscosity. This, of course, explains why acceleration has an effect only when it is maintained. It also explains why the effects are experienced with a gradual increase of G and why they continue so long as the acceleration is

applied.

Since it has been necessary to discuss the cause of this condition in the reverse direction from what actually occurs let us now recapitulate putting the happenings in their proper order. During the maintained application of acceleration of more than 4 G the viscosity of the blood is so increased as to lower the circulation and cause a pooling of the blood in the parts placed hydrostatically downwards. This causes a decreased filling of the heart which in turn lowers the general arterial blood pressure. The pressure in the cerebral vessels is naturally affected and as the acceleration increases the pressure in the retinal artery falls until it is unable to overcome the intra-ocular tension. Dimness of vision then blindness occur and as the pressure falls further, cerebral anaemia with unconsciousness takes place. These symptoms remain until the acceleration is withdrawn when they disappear, the consciousness returning first then the dimness of vision and lastly clear vision.

Prevention of the Condition

Whereas the cause of the condition is interesting from an academic point of view, the most important problem is to find a method of prevention. Experiments with the abdominal belt have shown that some degree of improvement is to be experienced by its use and I am convinced that much value would accrue from a belt, properly designed to fit each pilot. This would give immunity to all pilots for the lower degrees of acceleration and its benefits would be great in those who are most susceptible. This was seen when the belt was worn by the hypotensive who was very easily affected by the effects of acceleration. But there are other factors to be considered and these are important from the practical side. At the present time, pilots, and most of

all service pilots, are burdened with too many encumbrances already and to enforce the use of an abdominal belt would merely add to their troubles. Since it would mean undressing before and after each flight, for to be of use the belt must be worn next to the skin, it is doubtful if they would wear it. Altogether it seems doubtful if the benefits to be derived would outweigh the disadvantages.

But is it not better to try to remove the cause rather than try to eliminate its effects? This, of course, would be the ideal method of prevention. One cannot remove the acceleration but it can be forced to act in such a way as to have no effect. Fischer has shown that acceleration has little effect unless applied in the head to feet direction. If, therefore, a pilot could fly an aeroplane whilst lying in the prone position he would be immune to the effects of vast degrees of acceleration. This, of course, is impossible because of the limitation of the field of vision but there are many other positions between these two extremes. Ruff and Ranke suggest that the recumbent position need be used only during the higher accelerations and suggest the use of a folding chair. Whatever the method employed it is evident that an alteration in the present-day design of the aeroplane is essential.

One other method remains. That is to use only pilots who do not black out. But does such a person exist? I do not think so. That there is a difference in the susceptibility of pilots is true and some pilots seem to be able to withstand a maintained acceleration of much greater magnitude than others. But what causes that difference cannot be demonstrated by medical examination. It seems most likely that the explanation is to be found in general body tone. Unfortunately there is no practical method of estimating

this deficiency with any degree of accuracy and thus only practical experiment can reveal the sufferers. Its effects may be reduced by physical training but one cannot be certain that an improvement will be maintained. Until that is possible the only solution seems to lie in alteration of the present design of the aeroplane. This can be done and when it is completed the aeroplane designers may endeavour then to increase further the efficiency of the machine.

SUMMARY AND CONCLUSIONS.

The design of the aeroplane is constantly changing but new designs are drawn with regard to increasing the efficiency of the machine. Already we have reached a stage at which, on account of "blacking out", the efficiency of the machine, under certain conditions at least, is greater than that of the pilot controlling it. This deficiency on the part of the pilot is due to the force of the acceleration acting on him during these manoeuvres. One cannot remove these forces but their effect can be rendered void by changing their direction of application. This, unfortunately involves revolution in the design of the aeroplane but it can be done. If, then, the future of the aeroplane is to advance, future designs of the aeroplane must be drawn with regard to the efficiency of the pilot as well as to that of the machine itself.

BIBLIOGRAPHY

(1) Magitot and Bailliart. Blood Pressure in Vessels of the Eye. Am. Journ. Ophth. 1922,5,824.

(2) Fuchs. Text Book of Ophth. 7th Edit. p.81.

(3) Scarlet. Blood Pressures in Ophth. Blood Pressure by Norris, Bazett and McMillan, 1928,p.363.

(4) Adler. Arch. Ophth. 1924,53,239.

(5) Henderson, T. Glaucoma, E. Arnold, 1910.

(6) Samson Wright. Applied Physiology, p.337.

(7) Medical Exam. for Fitness for Flying. A.P. 130,p.107.

(8) Ruff, S. Luftfahrtmedizin, 1938,2,3/4,259.

(9) Fischer, V. Luftfahrtmedizin, 1937,2,1,1.

(10) Koenen, R, and Ranke, O.F. Luftfahrtmedizin, 1937,1,1,14.

(11) Norris, Bazett & McMillan. Blood Pressure, 1928,p.136.

(12) Schneider & Truesdale. Am. Journ. Physiol. 1921,p.223.

(13) Clough, F.E. Arch. Int. Med. 1913,11,590.

(14) McDowall. Clinical Physiology, Chap.11.

(15) Duke Elder. Recent Advances in Ophth. 3rd Edit,p.5.

(16) Andina, F. Schweizerische medizinische, Wochenschrift, 1937,18,753.

(17) Ranke, O.F. Luftfahrtmedizin, 1938,2,3/4,243.