

RECENT ADVANCES IN AVIATION MEDICINE

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In any attempt to review recent advances in Aviation Medicine, one must remember that almost all aviation medicine is recent. Although a not inconsiderable amount of experimental work was done during the first World War, nearly all of this work was discontinued shortly after 1918, and much of the material that had been studied and investigated was forgotten.

In marked contrast to the policy followed in Germany and Russia, where the subject of aviation medicine was continuously and actively investigated, this subject was completely neglected in the United States. Therefore our entry in the late war made it necessary for us to establish a multitude of laboratories and to carry on our investigations with almost feverish haste in order that we should not be hopelessly outdistanced by the Axis.

In a brief review such as this, it is obviously impossible to adequately cover the entire field. I have, therefore, somewhat arbitrarily selected three different sub fields in each of which outstanding results have been reported during the past year or two.

First, I want to take up the subject of angular acceleration or centrifugal force. If a fighter pilot is diving at the rate of 450 miles an hour and pulls out of this dive on the arc of a circle having a radius of about one-half mile, centrifugal force will press his body down onto the seat of the plane with a force which will be slightly more than five times the force of gravity. Thus if the flyer's weight is 180 lbs., the

force of his body against the seat of the plane would equal more than 900 lbs. His arms would be so heavy that he would be unable to lift them. The blood would have a weight five times its normal value. This would make it difficult for the heart to pump an adequate amount of blood to the brain. Blood would, therefore, tend to drain out of the upper part of the body and become pooled in the abdomen and the legs, and the aviator would suffer from cerebral ischemia. The first effect of this ischemia would be a blurring and graying of the vision as though a semi-transparent curtain were lowered before the eyes. This would be accompanied by a narrowing of the visual field. If the centrifugal force on the body is continued or increased, the aviator will "black out," that is, he will suffer a complete loss of vision and finally will lose consciousness. The extent of the physiological changes produced depends upon both the magnitude and duration of the centrifugal force applied to the body. Most young normal individuals can tolerate a centrifugal force as great as four to five G. for a period of as long as five to ten seconds.

One of the problems of aviation medicine has been the development of devices for protection of aviators against these effects of centrifugal force. So far the most successful device of this sort developed is the so-called arterial occlusion suit, of which you have probably read in the newspapers. This consists of a suit into which are incorporated three inflatable elastic bandages; one across the abdomen and one around each of the thighs. When the aviator performs any maneuver which involves increased centrifugal force, these bandages are automatically inflated in such a way that pressure is exerted on the wall of the abdomen and on the large arteries in the thighs. This pressure constricts or occludes these arteries, with the result that the blood is prevented from pooling in these parts. This means that more blood is retained in the upper part of the body and the aviator's tolerance of centrifugal force is increased. However, there is a definite limit to human tolerance of centrifugal force even with the best protective devices. This limit is on the average about 7 G.

The second development which I wish to discuss is the equipment for what is known as pressure breathing. It is well known that at altitudes of more than 15,000 feet men find it difficult or impossible to live and carry on ordinary activities because of oxygen lack. This lack of oxygen is not the result of any decreases in the percentage of this gas in the air, but rather of a lowering of the oxygen tension as a result of the decrease in total barometric pressure. It is quite obvious that protection against this anoxia of altitude can be obtained by breathing oxygen enriched air. Thus a person breathing 100 per cent oxygen at an altitude of 35,000 feet, where the barometric pressure is a little less than 100 mm. Hg. would be just as well off in regard to his oxygen supply as though he were breathing air at ground level. However, if he goes up to 40,000 feet, even breathing 100 per cent oxygen the alveolar oxygen tension would drop to about 55 mm. Hg. and his arterial oxygen saturation would be down to something under 80 per cent. He would, therefore, be in a precarious situation and on the verge of losing consciousness. It is generally stated that 42,000 feet is the absolute ceiling for the most resistant aviator, even when he is breathing 100 per cent oxygen.

During the latter part of the war, oxygen equipment was developed which delivered oxygen to the lungs of the aviator at a pressure of slightly greater than that of the ambient atmosphere. This results in an increased alveolar oxygen tension and makes it possible for the aviator to go a few hundred feet higher than he would otherwise be able to do. Two sorts of pressure breathing equipment have been developed: first, the constant pressure type in which the increased pressure is maintained continuously and second, the intermittent type in which the increased pressure is applied only during inhalation. Breathing is a little easier with this type of apparatus, but it is more complicated and therefore more likely to get out of order.

And now we come to the pressurized cabin and the problem of explosive decom-

pression. Even before the war the pressurized cabin was beginning to come into use as a means of protecting passengers in aircraft against the unpleasant effects of decreased barometric pressure. This mechanical development was carried to a much higher state of perfection during the war and as everyone knows, was a not inconsiderable factor in the success of our long distance, high altitude bombers. The pressurized cabin, of course, is the ideal solution to many of the problems of high altitude flying. But as is so often the case, the solution of one problem pushes to the forefront other as yet unsolved problems. Thus with the pressurized cabin in common use, the problem of explosive decompression became one of paramount importance. If a bomber is flying at an altitude of 35,000 feet where the barometric pressure is 180 mm. Hg., with the pressure inside the cabin at 560 mm. Hg., the crew would be subject to the hazard of an extremely rapid drop in barometric pressure in case of a large caliber shell piercing the cabin. Under these circumstances, the barometric pressure in the cabin might drop as much as 380 mm. Hg. (one-half atmosphere) in a fraction of a second. This is a pressure change of $7\frac{1}{2}$ lbs. per square inch. At first thought one would be inclined to feel that human beings could not tolerate such rapid and extensive changes in barometric pressure. In the early days of the war, explosive decompression was considered an extremely serious hazard. It became necessary that we should know what the tolerance of human beings was to such rapid changes in pressure and also what harmful effects might be expected to result. Here in our Ohio State laboratory, we began investigating these problems in January, 1942. We developed a technique for producing explosive decompressions at a rate which was equivalent to going from ground level to 50,000 feet in .02 of a second. Various species of animals were subjected to such explosive decompressions and to our surprise there were no harmful effects. A few minor hemorrhages of the lungs and occasional small hemorrhages in the ventricle of the brain were the most serious effects produced. None of these was of such a nature as to seriously incapacitate the animal. In fact, experimental animals showed complete recovery from such minor lesions in a period of two or three days. The only serious hazard seemed to be the possibility of the extreme expansion of the gas trapped in the stomach and intestine. At 40,000 feet, a gas would occupy a volume five times as great as that occupied at ground level. This means, of course, that when the gas is trapped in a confined space in the body, considerable pressure might be developed. We found that herbivorous animals, such as guinea pigs and rabbits, that are known to have considerable amounts of gas in the gastrointestinal tract, occasionally died following explosive decompression as a result of ruptured stomachs. In no case, however, did any of the dogs or cats which we used suffer serious injuries from explosive decompression.

We investigated in some detail the physiological effects of such rapid changes in barometric pressure. As would be expected, we found that there was a momentary but rather marked increase in the intrathoracic pressure. This is, of course, caused by the fact that the expanding air in the lungs is unable to escape through the trachea at a rate sufficient to keep pace with the fall in the ambient pressure. In other words, the lungs decompress at a slower rate than does the experimental chamber or the cabin of the aircraft. This rise in intrathoracic pressure is, we believe, the fundamental cause of all other physiological changes that take place following explosive decompression. Among these effects is a rise in the cerebrospinal fluid pressure. While our experiments on this point cannot as yet be considered final, such data as we have seem to indicate that the rise in cerebrospinal fluid pressure parallels, both in extent and duration, the rise in intrathoracic pressure. We also found a marked slowing of the heart and a drop in blood pressure. The slowing of the heart occurs immediately following the explosion. The drop in blood pressure begins after a delay of perhaps a second or so. This drop in blood pressure is probably due, at least in part, to interference with the venous

return to the heart by the increased intrathoracic pressure. There are also undoubtedly reflexes involved. Cardiac slowing seems to be solely of reflex origin since it is abolished by bilateral vagotomy. Our work on animals was followed by many experiments with human subjects. In general these experiments verified the results which we had previously attained with animals, although of course there were many things that we had done with animals that it was impossible to do with men. Furthermore we have never subjected our human subjects to the extremely rapid decompressions used with animals. We have shown, however, that explosive decompression is not a serious hazard to aviators.

In this short paper I have not attempted to give credit either to individuals or to laboratories for developments which I have mentioned. They were all cooperative undertakings in which a number of laboratories participated. It must also be borne in mind that there have been many other developments which were no less important than those I have discussed, but time does not permit further elaboration of the subject.