Aeroembolism : comparison with the bends of Caisson Disease

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AEROEMBOLISM
COMPARISON WITH THE BENDS OF CAISSON DISEASE

Donald F. Griess

Senior Thesis

The College of Medicine
University of Nebraska
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<table>
<thead>
<tr>
<th>Chapter</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Preface</td>
<td>1</td>
</tr>
<tr>
<td>II</td>
<td>History</td>
<td>6</td>
</tr>
<tr>
<td>III</td>
<td>Etiology</td>
<td>13</td>
</tr>
<tr>
<td>IV</td>
<td>Pathology</td>
<td>28</td>
</tr>
<tr>
<td>V</td>
<td>Symptomatology</td>
<td>37</td>
</tr>
<tr>
<td>VI</td>
<td>Prophylaxis</td>
<td>50</td>
</tr>
<tr>
<td>VII</td>
<td>Treatment</td>
<td>61</td>
</tr>
<tr>
<td>VIII</td>
<td>Summary</td>
<td>68</td>
</tr>
<tr>
<td>IX</td>
<td>Bibliography</td>
<td>73</td>
</tr>
</tbody>
</table>
PREFACE

Today we are faced with the problems of defense; tomorrow with the still greater problems of peace. In both of these eras aircraft and the peculiar problems in human physiology which man's flight through space has brought about will play an increasing part.

The field of aviation medicine is as old as flight itself; the very first ascent in a balloon in 1783, accomplished by the Montgolfiere brothers in Annonay, France, was an experiment in aviation medicine. The first three voyagers into the unknown being a cock, a duck and a sheep. Thus, we see that even these pioneers realized that there might be unknown quantities in flight which were incompatible with life as we know it on the surface of the globe. (39).

As man progressed from lighter to heavier than air craft, he found strange new problems confronting him. At first one of the major problems of both types was oxygen lack or anoxemia. Much has been written on this phase of aviation medicine, and much experimentation in the air and on the ground has been carried on. The problem still remains only partially solved by aviation engineers.

The last few years have brought forth an even
more interesting problem and one which promises to bring forth a great deal of experimentation in this phase of aviation medicine. I refer to what Captain Armstrong, of the United States Army Medical Corps, calls "aeroembolism". This phenomenon is a first cousin to that other condition known as "caisson disease", the "bends" or "divers' disease". For the sake of clarity it will henceforth be referred to as caisson disease in this paper. Caisson disease is caused by the too rapid ascent from a level below the surface of the earth in which there is a rapid decrease in atmospheric pressure. Aeroembolism is caused by a too rapid ascent from the surface of the earth to a high altitude such as is accomplished in modern interceptor planes.

We know from the work done on caisson disease, that if the ascent is made slowly, there will be no ill effects on the human mechanism. It is only when the ascent is so rapid that nitrogen bubbles are released that the ill effects are felt. (10).

It has only been in the last few years that airplanes were mechanically perfected so that they could climb several thousand feet per minute and maintain this rate of ascent to thirty thousand feet and above. In
PREFACE

testing these planes, pilots first began to notice strange joint pains and paralysis of a transitory type. At first this was thought to be due to oxygen lack, but pilots fully equipped with oxygen equipment still felt the peculiar effects of rapid ascent. If the rate of climb was slower, these symptoms were decidedly decreased or absent altogether. From this evidence and by comparing the symptoms with the older caisson disease, it was concluded that the symptoms were due to air emboli.

Several attacks have been made on the problem and these will be discussed later. Suffice it to say that none of the solutions are wholly satisfactory. If the ascent is made slowly, the chance of aeroemboli occurring is slight, so for bombardment planes rising to the offense the problem is of relatively little importance. The speed of the modern bomber, however, makes it imperative that the interceptor plane rise almost vertically at a rapid rate of ascent to meet it. In order to accomplish its mission, the interceptor must get on top in the shortest possible time, so aeroemboli really constitute a major defense problem to the nations of the world.

Up to the present time aeroemboli have had no
effect on our comparatively low altitude commercial air travel. The reason for this is that the ascents are slow and the altitudes have been below eighteen thousand feet. We have reason to believe that in the future, however, commercial aircraft will fly in the stratosphere or at least the troposphere or substratosphere. The advantages being, among other things, advantageous tail winds and lack of storms. In order that the full advantages of speed may be realized, these planes must rise at a rapid rate of climb and here is where the problem of aeroemboli will affect commercial aviation.

This paper will deal with both caisson disease and aeroembolism in order to contrast the two, and also to try to apply the lessons learned about caisson disease to aeroembolism.

The section on caisson disease will only cover the best literature in the field and only the best references will be quoted. On the other hand, the author has made an attempt to cover the entire field of aeroembolism as far as English literature is concerned and also some of the German material.

When "the bends" are referred to, it should be remembered that the bends are a symptom of decompres-
sion only, while caisson disease, in the strictest sense, refers both to the effects of compression and decompression.
HISTORY

In subaqueous engineering works it is necessary to keep out the water by using an artificially compressed atmosphere. This is highly essential for work in water bearing soils. A short review of the use of compressed air is, therefore, not out of place in this paper.

In the sixteenth century, the diving bell was invented by Sturmius. In this device works of simple nature could be carried out by men working in it for short times. However, the men could only work in this device for short periods, because no means of changing the air were incorporated in the machine. An attempt to remedy this situation was made by sending down bottles of fresh air which were broken in the bell. This, of course, was no real solution to the problem.

Halley, an English physician, used kegs which were passed down into the bell. These the laborers opened at will and released the fresh air. The foul air was allowed to escape by a valve at the top of the bell.

The early reports of physicians who investigated the symptoms of men descending in diving bells and working under pressure were confused by the effects of pressure and rapid decrease in pressure. All of these
HISTORY

early writers were greatly concerned with the pain in the ears experienced after the bell entered the water. These symptoms were found to be relieved by swallowing in order to keep the Eustachian tubes patent.

In 1820 Dr. Colladan descended in a diving bell near Dublin and reported that on ascending there was a sensation of the head getting larger; the bones of their heads felt as if they were going to separate.

We should state here that only the symptoms of the ascent will be discussed in this paper. The reason for this is that we are concerned here only with decreasing atmospheric pressure.

Three hundred years after the development of the diving bell the caisson or caisson was first employed. This caisson is a cylinder which is driven vertically into water containing soil. This acts to form a water free space in which men can work. The water is removed from the caisson by the action of pumps, or if they are unequal to the task, by the use of a diaphragm dividing the chamber into two parts. Air introduced into the lower chamber by a pump forces the water out. The diaphragm must have a lock or chamber with inner and outer air tight doors to allow men and materials to pass
HISTORY

through.

The French engineer, M. Triger, first used the caisson on work on wells and bridges. The suggestion for this device first came from Denis Papin in 1691, but it was not until 1830 that Lord Dundonald took out a patent on a compressed air device. This device was used near Chalonne, France, by M. Triger to mine coal through an extensive layer of quicksand.

Professor Trouessart, appointed by the Industrial Society of Angers to examine the practical results of Triger's apparatus in 1845, makes the following interesting statement: "It is this humidity which explains also perhaps the severe pain in the joints which some laborers have experienced a few hours after coming out".

M. Triger makes the following report on accidents of decompression: "I ought to declare that two workmen, after having spent seven hours in the compressed air, experienced severe pain in the joints half an hour after coming out. The first complained of extremely acute pain in the left arm, and the second of similar pain in the knees and left shoulder. Rubbing with spirits of wine caused the pain to disappear in both cases".

In the mines of Douchy the method employed by
HISTORY

Triger was first initiated. Blavien, an engineer on the project, made a report of a descent in which he describes the results of decompression three atmospheres' pressure. He states that he had a severe pain in his left side the day following the compression. He attributes this pain, which lasted for several days, to a cold or some other cause other than the compressed air. Several days later, after taking all precautions against catching a cold, he was again compressed and the next day he again had a severe pain in the right side, which incapacitated him for several days.

In 1847 the memoirs written by physicians on these accidents was given by M. M. Pol and Watelle. They reported on their own subjective feelings as well as on the pathological effect on workmen. The pressure was raised to four and one-half atmospheres in fifteen minutes, and decompression was accomplished in half an hour. This observation was made by the two medical men: "The danger is not in going into the compressed air. It is not a disadvantage to stop there a longer or shorter time. The decompression alone is to be feared. One only pays on coming out." (36).

The first important work on the physiology of
dancing was that of Boyle in 1670. He experimented on animals under different air pressures and demonstrated the presence of gases in the blood. His work will be referred to later in this thesis. Hoppe-Seyler in 1857 was the first to prove that bubbles of gas in the blood were the cause of death after the rapid decompression of animals. The work was carried further by Paul Bert, who placed on a firm foundation our present knowledge of the effects of variation in barometric pressure. (18).

We now turn to the comparatively recent history of aeroembolism, as caused by rapid ascent from sea level to high altitudes. Naturally the work done on this type of sickness is less than that done on caisson sickness, and the reference we have to the possibility of aeroembolism was made by Boycott and Haldane in 1908. They foresaw that caisson disease might develop if too rapid an ascent was made from seal level. They based their opinion on the fact that the blood and tissues of the body are always saturated with atmospheric gases at sea level, since the blood in the lungs is exposed to these gases and each is in solution according to its partial pressure. (21). Nitrogen which is physiologically inert, and hence not utilized, is taken up in physical
solution by the blood in the lungs in relatively large amounts because of its high partial pressure, (approximately 79% of an atmosphere), so that at sea level the nitrogen in the blood is in saturation equilibrium with the nitrogen in the air at a partial pressure of approximately 560 mm. (25).

When aircraft began to ascend to the higher altitudes and remain there for protracted lengths of time, the effects of air emboli began to be felt. These planes, however, rose slowly and consequently the symptoms were never acute. These symptoms consisted mostly of profound physical and mental fatigue. This, of course, was first ascribed to oxygen lack, but when the pilots were given oxygen they still suffered from this type of lassitude when going above thirty-five thousand feet. When they descended to twenty-five thousand feet, they immediately felt better.

In the late 1930's when the nations of the world were preparing madly for the latest debacle planes were climbing faster and faster; motors were supercharged and then double supercharged, so that, in effect, they carried two motors—one for high and one for low altitude—the change merely being made by switching from one
superchamber to the other. Pilots literally pointed their sleek nosed planes at the stars, gunned the motors and ascended almost vertically, traveling better than twelve thousand vertical per minute.

Now we began to see the real bends; the acute symptoms appeared. These consisted mainly of joint pain, especially in men who had had joint injuries at some previous time. As we will see in subsequent chapters, this condition was, in part, alleviated by giving the pilots oxygen before the ascent. The U. S. Navy Air Corps and various commercial air lines began to test the effects of altitude in pressure chambers after pilots had noticed the symptoms in rapid climbing fighters. It is mostly from these tests in pressure chambers that we have learned what are the effects of decreased atmospheric pressure on the human organism.
ETIOLOGY

Robert Boyle in 1670 made the first experiment dealing with the formation of bubbles under conditions of decompression. He has this to say of his experiment:

"Note, that the two foregoing Experiments were made with an Eye cast upon the inquiry, that I thought might be made; Whether, and how far the destructive operation of our Engin upon the included Animal, might be imputed to this, that upon the withdrawing of the Air, besides the removal of what the Airs presence contributes to life, the little Bubbles generated upon the absence of the Air in the Bloud, Juyces, and soft parts of the Body, may by their Vast number, and their conspiring distention, variously streighten in some places, and stretch in others, the Vessels, especially the smaller ones, that convey the Bloud and Nourishment; and so by choaking up some passages, and vitiating the figure of others, disturb or hinder the due circulation of the Bloud? Not to mention the pains that such distensions may cause in some Nerves, and membranous parts, which by irritating some of them into Convulsions may hasten the death of Animals, and destroy them sooner by occasion of that irritation, than they would be destroyed by the bare absence or loss of what the Air is necessary to supply
ETIOLOGY

them with, and to shew how this production of Bubbles reaches even to very minute parts of the Body, I shall add on this occasion (hoping that I have not prevented my self on any other,) what may seem somewhat strange, what I once observed in a Viper, furiously tortured in our Exhausted Receiver, namely that it had manifestly a conspicuous Bubble moving to and for in the waterish humour of one of its Eyes." (ll).

It would seem that "the bends" phase of caisson disease or aeroembolism of aviation could be explained quite simply by decreased pressure which is accomplished at too rapid a rate. This is, in the main, true. There are, however, some other factors which enter into the cause of this phenomenon.

Snell made the following observation: "Although I have no wish to underestimate the influence of decompression in producing the illness, I do not believe that it is the primary factor in its causation." While present investigators believe that the rapidity of decompression plays the major part in causing aeroembolism it is well to consider at this time the observations of Snell.

(a). The Fullness of Habit. "This has been
ETIOLOGY

bited by most observers. On this account, at the Blackwall Tunnel, I have as far as possible excluded men of very heavy build. On two occasions my advice to men of this type was disregarded, and their first sojourn in compressed air was followed in either case by an attack of the bends, which was effective in enforcing my advice. Because a man is stout, it does not follow that he necessarily suffers, it can only be stated that in many cases the liability to illness is increased to a remarkable degree."

Armstrong has this to say: "It has been noted that different individuals with similar body builds are not equally susceptible to attacks of aeroembolism under identical conditions of exposure. Fat individuals are generally much more susceptible to attack than are thin individuals because of the increased solubility of nitrogen in fat."

(b). Age. Below the age of twenty, men are remarkably free from compressed air illness. Between the ages of twenty-five and forty-five the liability to illness does not vary much, but the chances of illness are about twice as much as between the ages of fifteen and twenty years. Above the age of forty-five the lia-
ETIOLOGY

bility to illness is greatly increased.

As far as aviation is concerned, age has ruled out the older men before they would get to such planes as interceptors, so we cannot say what effect age has on this type of aeroembolism.

(c). Alcoholic effects. Dr. Snell states that men who have been drinking heavily are much more subject to the bends than are temperate men. Dr. Lehwess, in the Petersburg Med. Woch, September 1, 1877, flatly states that alcohol has no effect whatsoever on the bends. For obvious reasons aviation will shed little or no light on this subject.

As far as caisson disease is concerned, we may be sure that the length of time for which an individual remains in the compressed air has a decided bearing on the illness following decompression. Men who enter and leave the locks many times, such as lock tenders, do not suffer from the illness. While men remain in the compressed air for several without leaving and who are engaged in actual physical labor are much more subject to the illness. There is one case on record in which a foreman on leaving the compressed air was so severely affected with pains in the joints that he immediately
ETIOLOGY

returned to the compressed air. Being relieved, he remained in for several hours. When he finally emerged, he died within an hour of his exit. (36).

When we consider caisson disease we must remember that the decompression is a return to normal. So we can draw our conclusions as to the effects of a longer or shorter sojourn in the abnormal. On the other hand, we must remember that decompression in high altitude flight is a change from the normal to the abnormal. Thus, we have been compressed ever since birth, and all our heredity has been in a state of compression since the beginning of time. Therefore, we cannot draw conclusions as to the length of compression as we did in caisson disease.

We now know that the greatest factor in the etiology of caisson disease and aeroembolism is the release of nitrogen bubbles into the tissues and the blood stream. It is from this source that we get the group of symptoms which we call the bends. Other factors, such as age, body shape and physical condition of the individual are only contributory factors.

We shall now take a brief review of certain physical laws and their bearing on the etiology of this
ETIOLOGY

subject. We must remember that when we are at sea level we are always under a pressure of 760 mms. of mercury, and that each square inch of skin is subject to the pressure of about fifteen pounds, or to be exact fourteen and seven tenths pounds. Of course, the inside of the body has exactly the same pressure as the outside in just the same manner as the inside of a sponge submerged in water has the same pressure as the outside of the sponge. If a man had a vacuum on the inside of his body the total pressure on the outside would be enormous, corresponding roughly to that which would be exerted on a man if he were made to lie on the bottom of a large box which was then filled with iron crowbars standing vertically, the butt of each crowbar resting on the man's skin. In the horrible accident of squeeze which occurs when the air pressure inside of a diver's helmet is less than that of the water outside, this pressure effect is actually exerted with fatal results. Ordinarily the air pressure inside is a little greater than that of the surrounding water and the diver hardly notices any sense of pressure no matter how great the depth. Man can adapt himself readily to great changes in outside pressure; for example, one man states he had the opportunity
ETIOLOGY

of being a passenger in an altitude flight to the height of 23,200 feet, where the barometer read 325 mms. of mercury, or 42% of an atmosphere. The next day in a diving suit he was under the pressure of 189 feet of water, 511.4.8 mms. of mercury or 6.73 atmospheres absolute. Thus the greatest pressure was more than fifteen times the lowest pressure, but there was no discomfort at either extreme.

In all calculations we must remember the difference between absolute pressure and gauge pressure. The ordinary pressure gauge registers zero in the open air, when the absolute pressure is 760 mms. of mercury or 14.7 pounds to the square inch. When one additional atmosphere of pressure is added, the gauge will read one atmosphere, or roughly fifteen pounds, but the absolute pressure will be thirty pounds. In all physical and physiological calculations we must use the absolute pressure. If we multiply the absolute pressure in millimeters of mercury by the percentage of each gas present in the air, we obtain the true partial pressure of each gas. We must think in terms of partial pressure. The physiological action of gases are strictly conditioned by their partial pressures. Thus, 10% of oxygen
ETIOLOGY

at thirty pounds pressure, absolute, (fifteen pounds gauge pressure), has the same physiological effect as 20% oxygen at fifteen pounds pressure, absolute, (zero pounds gauge).

It is for this reason that we have no difficulty in supplying the diver with oxygen. At a dept of one hundred thirty-two feet of seawater, an absolute pressure of five atmospheres, the man could live comfortably with a mixture of 4% oxygen. Oxygen, the good gas, is concentrated at great depths, but unfortunately nitrogen, the inert gas, and carbon dioxide, the harmful gas, are concentrated also. Thus, a man can breathe a mixture containing 1% carbon dioxide at sea level without any noticeable effects, but if this percentage were present in his helmet at a depth of five atmospheres, absolute, he would almost suffocate. Nitrogen, the gas which causes the bends, goes into solution in the blood and tissues according to its partial pressure. If a man remains a sufficient length of time at a pressure of five atmospheres, absolute, his blood and tissues will contain five times as much nitrogen as at sea level. If he returns to sea level too suddenly, the tissues are caught with more nitrogen than they can hold, and,
ETIOLOGY

therefore, bubbles of nitrogen are liberated at the most inconvenient places.

When the air surrounding a man is compressed, let us say to four atmospheres, the whole body tends to come into a new gaseous equilibrium. The gases are soluble in water and in the body fluids in proportion to their partial pressure according to Dalton's law. It is assumed, perhaps incorrectly, that the blood in the pulmonary capillaries comes into immediate equilibrium with the air in the alveoli. At four atmospheres the plasma leaving the lungs would, therefore, hold four times as much nitrogen and oxygen as at one atmosphere pressure. There would be no change in the carbon dioxide because the partial pressure of carbon dioxide in the alveolar air is always maintained about 5.6% of one atmosphere at all pressures. It requires a considerable length of time for the fresh blood leaving the lungs with its increased oxygen and nitrogen to bring the whole body up to the new level of saturation. The increase of oxygen is of some physiological importance because it makes tissue respiration a little easier, but it is the nitrogen that should engage our chief attention. Nitrogen at 37° C. has a coefficient of absorption of 0.97 in
water, and according to Hill, the coefficient is a little less for blood. In round figures, the blood at sea level contains 1% nitrogen gas in solution and most of the tissues are supposed to be in equilibrium at this level. This is not true for all tissues because oxygen and nitrogen are about five times as soluble in fat as in water. Vernon found that at 37° C. oxygen was 4.5 times as soluble in fat as in water, nitrogen 5.3 as soluble. (38).

It takes several hours before the blood can transport from the lungs enough nitrogen to saturate the whole body at the new pressure. Those parts of the body where the circulation of blood is good, such as the muscles, glands and brain, saturate more rapidly than the ligaments, bones and white matter of the spinal cord. The fat depots with their high affinity for nitrogen and very scant vascular supply will not be fully saturated for a long time. We must remember also that the rate of saturation for any given tissue is not uniform, but is relatively rapid in the first few minutes after compression when the difference between blood and tissue is great and then slows up in a logarithmic curve as the pressure difference decreases. Conversely, when
ETIOLOGY

the pressure is reduced the rate of desaturation is also irregular and those parts of the body which acquire their nitrogen slowly give it up reluctantly. It seems probable that the fat acts as a reservoir during decompression, and that the nitrogen tardily removed from it in solution may be liberated in the form of bubbles in parts of the body where they can do great damage. (18).

The effects on the various body tissues which are caused by the release of nitrogen will be further discussed in the chapters on Symptomatology and Pathology of the bends and aeroembolism.

Armstrong has this to say about the etiology of aeroembolism in aviation medicine. Aeroembolism is a condition caused by the same general process that causes compressed air illness, with the exception that the former occurs from compression to two or more atmospheres followed by decompression, while the latter occurs from decompression from one atmosphere pressure or less. In aeroembolism the body tissues and fluids are saturated with the atmospheric gases at sea level pressure since the blood in the lungs is exposed to these gases and dissolves them each according to its partial pressure. It has been determined that at sea
ETIOLOGY

level pressure, 100 cc. of blood dissolves about 1.5 cc. nitrogen, .36 cc. of oxygen and 2.7 cc. carbon dioxide, while much greater amounts of the latter two are carried in loose chemical conformation. While most of the dissolved oxygen in the blood is consumed by the body tissues, the nitrogen is inert physiologically and is not utilized but goes into simple solution in the body tissue in an amount dependent on the partial pressure of the nitrogen in the lungs. Thus, at sea level pressure the tissues of the body are always completely saturated with atmospheric oxygen. (3).

During ascent in aircraft or in any other situation in which the atmospheric pressure is decreased, the internal partial pressure of the body nitrogen is above that of the nitrogen in the lungs and the tissues are therefore, supersaturated. As a consequence, the nitrogen dissolved in the blood begins to be given off in the lungs, the nitrogen in the tissues begins to enter the blood stream, and by this dual process the body tends to rid itself of excess nitrogen. If the ascent is slow enough so that the nitrogen in the body can be eliminated and not reach approximately double its normal saturation value at the prevailing altitude,
ETIOLOGY

nothing unusual will occur. If, on the other hand, the concentration of nitrogen in the body becomes more than double its normal saturation value at any altitude pressure, the nitrogen gas will come out of solution and form bubbles to which is added some oxygen, carbon dioxide and water vapor from the surround blood and tissue. The bubbles are formed, not only in the blood, but also in other tissues of the body. The most likely site for this formation is those tissues of the body which have the highest fat content and the poorest blood supply; the reason for this being as follows:

In aeroembolism as distinguished from compressed air illness, all of the tissues of the body are always completely saturated with nitrogen initially. With reference to the influence of blood supply, it has but to be recalled that since the elimination of nitrogen from the body is entirely through the blood stream, those parts of the body or those tissues which have the poorest blood supply will be the least able to lose their excess nitrogen. From this it would seem that the blood would lose its excess nitrogen first, the body tissues, (other than fatty tissues), second, and the fatty tissues last; the delay in the latter being due to both its higher
ETIOLOGY

nitrogen content and its poorer blood supply. That this is essentially correct is indicated by the fact that with rapidly decreased atmospheric pressure, gas bubbles are found in the spinal fluid and about the spinal cord at eighteen thousand feet altitude, while bubbles in the blood and body tissues generally have not been found below thirty thousand feet altitude. This can be readily accounted for by the fact that the spinal cord contains a high percentage of fat, and hence a high nitrogen gas content and that the spinal fluid has no direct connection with circulating blood. (2).

From the foregoing discussion on etiology we can see that aeroembolism and compressed air illness have essentially the same etiology; that is, the release of nitrogen into the tissues of the body, especially the fatty tissues, as a gas, when released too rapidly by too speedy an ascent either from the surface of the earth or to the surface of the earth. As was stated before, there are other factors, such as age, to be considered, but after weighing all the evidence the only one which I believe to be of any consequence is the one dealing with the adipose tissue. As was pointed out, fatty tissue gives up its nitrogen much more slowly than
ETIOLOGY

other body tissues. Consequently, a fat individual will suffer more than his more slender brother.
PATHOLOGY

Pol and Watelle were the first to attempt to explain compressed air illness; they were the first to determine positively its time of onset: "Payment is only made on coming out", say they. With the exception of cases of muscular pain where there is no evidence of the nervous centers being affected, these cases were probably produced, in their opinion, by the circulation of hyperoxygenated blood through the capillaries of the nervous system. Excepting, also, the gastric pains, which appear often to be of a sympathetic character, the illnesses would appear to suggest a congested state of the brain and lungs. In order to explain why the symptoms only supervene after the decompression, they state that the compression itself exercises a corrective influence over the possibly bad results of the congestion it produces.

Guerard points out that there must be an increase of oxygen and nitrogen dissolved in the blood, and consequently, he says, there must be an increase of the amount of interstitial combustion, from which emaciation will result. He considers that the pains in the extremities are rheumatic in nature, and due to the rapid cooling of the air lock during the locking-out process.
PATHOLOGY

Hoppe found bubbles of free air in animals killed suddenly by rarefied atmosphere. He reasons that the same thing happens in sudden decompression to normal air pressure.

Dr. James Hunter, after his observations made at the Forth Bridge, has this to say: "Were decompression to be effected sufficiently slowly, and with proper precautions, probably little would be heard of caisson symptoms. But by existing methods it takes place by considerable rapidity; there is a great giving-out of heat, producing an extremely devitalizing effect on the workers. There is a loss in the balance between oxygenation and tissue waste, which held during the increased vitality of compression, and there is a sudden liberation of the gases, which had been absorbed, and which now tend to interrupt the circulation, causing severe pain in the neighbourhood of the vascular fringes of joint surfaces.

But to me it seems also that there is a nerve element in the production of this severe pain. Sir Joseph Fayrer has pointed out that in cases of sciatica he has obtained relief by incising the nerve sheath, releasing thereby a varying quantity of fluid, which has
PATHOLOGY

accumulated within the sheath, and, by its painful pressure on the nerve substance proper, has given rise to a condition whose name indicates the locality of that special neuralgia.

"It is quite a possibility, then, that there occurs in certain areas richly supplied with nerve filaments, an absorption of nitrogen during compression by the infra-perineurial fluid, and that the excessive pain referred to these areas is produced by attempts at release of bubbles of gas when decompression has begun to take place."

Haldane in his Admirality Report of 1907 discusses the mathematics of the rates of saturation and desaturation. The equation takes into account the volume of blood in the body, the time required for this blood to make one complete circulation and the relative amount of fat in the body. There is a great deal of uncertainty in regard to all of the factors, and it is doubtful if we shall ever be able to approximate a measurement of the blood flow in certain parts of the body, such as the spinal cord, the bone marrow and fat deposits—the very places that hold the key to the puzzle. For this reason we will not reproduce these calculations
It is well known that there are great differences in the response of different individuals and this has made all calculations difficult. Haldane has pointed out, however, that we do know from practical experience that saturation is not complete until a man has been exposed to a given pressure for about three hours. We also know that divers or caisson workers who have been exposed to pressure of two and one-fourth atmospheres, absolute, never get any symptoms due to the formation of bubbles, even if the pressure is suddenly reduced to normal (one atmosphere). The blood and tissues can hold twice as much nitrogen as we would expect from the saturation point. This is probably due, in part, to the colloidal nature of the blood. Even water that has been exposed to a high pressure will hold much more than the calculated amount for saturation, if it is not shaken, when the pressure is removed. Hill and Twort have shown this in experiments and they refer to the fact that supersaturated solutions will not froth unless "points" are given for the bubbles to form on. Small bubbles may act as "points" for larger bubbles to form on. If small bubbles are formed in the tissues they may disappear on recompression, but large bubbles, especially if they
block up the blood vessels in places where the circulation is slow, may take a long time to disappear, and this probably one reason why it is necessary to keep patients a long time in the recompression chamber when treating caisson sickness.

Young men, with their higher metabolism, saturate faster than older men, thin men much faster than fat men. They also desaturate much faster. The man who works hard at the bottom increases his saturation much more rapidly than the man who stays quiet.

Caisson workers are not exposed to very high pressure, and in modern engineering works are carefully supervised so as to prevent many severe cases. Divers go to much greater pressures, and on account of circumstances which cannot always be controlled they may have to come to the surface before an adequate period of decompression. They frequently suffer from prickly sensations in the skin or deep pain caused by bubbles of gas in the periosteum, bone or tendon. These may appear shortly after coming to the surface, but often they do not occur for several hours, since it may take a long time before the bubbles form.

After sudden decompression from high pressure
death may follow rapidly due to liberation of gas in the blood vessels and chambers of the heart. Most of the serious cases are due to the formation of bubbles in the white matter of the spinal cord in the dorsal region. This part of the nervous system seems to be particularly susceptible, perhaps on account of poor blood supply. If the bubbles are of considerable size they can cause great damage by pressure and by interference with the nutrition to the part. Paralysis follows as a result of destruction of nerve tracts. These paralyses are usually permanent; they affect chiefly the body and legs, but may affect any part. Patients often succumb to bed sores. (12).

We now come to the discussion of the pathological effects of high altitude ascent at a rapid rate as caused by release of nitrogen in the form of a gas. Needless to say, the symptoms and pathology greatly resemble those found in the bends of caisson sickness. Probably one of the first discussions of aeroembolism was made by Corning regarding Glaisher's balloon ascent from Wolverhampton in 1862. Glaisher suffered from loss of use of the hands and limbs, and unconsciousness—as precisely analogous to the symptoms of compressed air
illness; he attributes both, without proof, to anaemia of the spinal cord, and adds: "At one time it was believed that, on leaving the condensed atmosphere, there was a development of gas from the blood which caused a rupture of the neighbouring tissue. What added plausibility to this theory was the fact that Bert and Hoppe-Seyler had been able to produce analogous phenomena experimentally. It has been urged, however, in rebuttal, that the limitation to the dorsal portion of the cord and the infrequency or total absence of vascular hemorrhages, are totally opposed to this theory."

When ascending to a high altitude the nitrogen in the body fluids and tissues finally reaches a stage of supersaturation which results in the formation of nitrogen bubbles. The nitrogen in the blood is given off as the blood transverses the capillaries of the lungs. The nitrogen in the tissues, however, is not disposed of so easily. The tissues contain about twenty-six times the amount of nitrogen that the blood does. This excess nitrogen passes rapidly into the capillaries and produces a supersaturation which may result in bubble formation in the venous circulation before the lungs are reached. The excess nitrogen may not be carried away
rapidly enough by the blood and the result is nitrogen bubble formation in the tissues themselves. As has been mentioned before, this is particularly true in fatty tissue.

Bubbles in the circulation are practically always due to an embolus formation, usually in an end artery, resulting in an infarct. How emboli form in the arterial blood is not exactly understood. We believe that the gas bubbles are always found in the venous blood, but how they pass through the capillary beds of the lungs is still a mystery. It is conceivable, of course, that microscopic nitrogen bubbles might pass through the lungs and by coalescing form large bubbles on the arterial side of the circulation. In any event, the most common finding at autopsy is gas bubbles in the veins. Bubbles are not only found in the veins, but also in the end arteries with local anemia beyond the point of lodgement.

Outside the blood stream itself nitrogen bubbles are found in the myelin sheath of the nerves, the spinal fluid, body fat, the white matter of the cord and all the other tissues of the body.

Armstrong states, "The real pathological lesions are those produced by the blocking of the circulation or
PATHOLOGY

the mechanical pressure exerted by the bubbles in the tissues. The former may lead to heart failure or failure of the general circulation, pulmonary embolism with congestion and edema, or local disturbances of the circulation in any of the organs of the body or in any part of the central nervous system. The bubbles in the tissues act principally to produce pain in the structural system or lesions in the central nervous system, (stretching and tearing), causing altered sensory or motor function. (2).
SYMPTOMATOLOGY

It has been constantly reiterated in the literature that true compressed air illness occurs during or after decompression. This sickness may come on a longer or shorter time after decompression has been fully affected. It may occur as long as fourteen hours after the worker has left the compressed air. Usually the interval is from a few minutes to one hour. In cases where the interval is longer, the man has gone home and fallen asleep only to be awakened some hours later by severe pain in one or more limbs.

The pain usually affects the legs and principally the parts about the knees. It also may affect the upper extremities, particularly the shoulders and the elbows. The pain varies in severity; it may be only slight and cause only slight inconvenience or it may be very excruciating and cause the victim to writhe about in agony. Some patients describe the pain as dull, heavy aching pain, others describe it as sharp and knife-like in character. Some people appear to feel the pain deep down in the bones, others localize it to the more fleshy parts. Still others locate the pain in the joints. It should be born in mind that the pain does not follow in anatomical distribution, such as that of
the large nerves. Usually there is tenderness over the
effected part, which is elicited by both superficial and
deep palpation. Most cases present no visible evidence
of any pathology; there is usually no swelling, discolor­
oration or heat. There have been a few cases in which
objective findings were observed. These consisted of
ecchymotic patches and some swelling over the painful
areas. Because of the scarcity of cases in which these
findings were found, they should probably be discredited.
Some writers have described a cold perspiration as being
characteristic of this phenomenon. The pulse shows no
great change.

The duration of the pain is also variable.
If the pain is slight, it may pass off in a few hours;
in the worst cases it may persist for one or two weeks
and in some cases even more. It tends to disappear by
itself, but recovery may be hastened by recompression.

There is some dispute about the epigastric
pain which is sometimes present. Jaminet described it
as being present in 78% of his cases. Other investiga­
tors have only found it present in 10% of the cases. The
epigastric pain may occur by itself or may be accompanied
by pain in the limbs. There may be vomiting or only
SYMPTOMATOLOGY

nausea with the epigastric pain. The pain is not girdle-like in character, but is generally localized at the epigastrium.

One of the most dreadful after-effects of decompression is paralysis. Its time of onset is any time immediately after exit from the compressed air. There is generally involvement of the bladder and rectum, and in fatal cases cystitis is often the cause of death. It should be remembered that many cases of so called paralysis have been reported by lay observers. Because of the severe pain associated with movements of the effected joint it may appear to the uninitiated to be paralyzed. (36).

In cases of paralysis the symptoms come on rapidly. The paralysis is usually in the legs and it varies from a slight paralysis to a complete loss of motion and sensation. In the extremes, the attacks resemble apoplexy; the patient rapidly becomes comatose and death occurs in a few hours. The paraplegia may be permanent, but in slight cases it gradually disappears and recovery is complete. Where an air embolism may lodge we shall find a symptom of the effected part which may mean any part of the human body. (9, 14).
SYMPTOMATOLOGY

The cause of the symptoms of either "the bends" of diving or aeroembolism of aviation is free nitrogen bubbles in the tissues of the body. From this it may be seen that there may be a great diversity of symptoms and these symptoms may appear any place in the body, depending on the relative importance of the part of the body where the bubbles appear, such as the spinal cord, on the size of the bubbles and the rapidity with which they are absorbed, and also the number of bubbles released in the body. The large amount of gas released from the spinal fluid seems to have no great effect directly, but acts principally by increasing the intracranial pressure. The gas released in the tissues appears to act principally by causing pain the unyielding tissues, such as bone, tendons, fascia, periosteum and nerve sheaths. The blood stream seems to be the most dangerous point of involvement, although bubbles in the central nervous system can also produce very serious results. (3).

We shall consider the symptoms according to the various body systems as Armstrong has done. This is the most logical way to "break down" the symptoms.

1. Cerebrospinal System. In animals it has been demonstrated that bubbles form in the spinal fluid
SYMPTOMATOLOGY

at a pressure such as would be found at eighteen thousand feet or above. This has also been recently demonstrated to be the case with human beings. However, no definite symptoms have yet been attributed to the bubbles. There is some evidence that the profound physical and mental depression observed after high altitude flights, even in the presence of sufficient oxygen, may be caused by aeroemboli. If there is no irritation of the meninges there is usually no headache even in the presence of increased pressure.

Other manifestations which may be due to this condition include certain mental aberrations which have been noticed at thirty thousand feet and above. These include sudden loss of memory; the individual not being able to remember where he is. There are also cases recorded of vivid dreams in which the subject appears to be wide awake. Still another test case became very violent and boisterous; in fact, he developed a typical manic reaction.

One subject exposed in the altitude chamber to an altitude of 37,500 feet for about fifteen minutes became unconscious and developed convulsions. The convulsions were of the clonic type; they affected all the
SYMPTOMATOLOGY

limbs and were gross in nature. When the subject was returned to twenty-five thousand feet, the attack was entirely relieved. This attack was thought to be caused by an embolus in the motor area of the brain or in the motor tracts. In another instance, a subject at thirty-five thousand feet in the altitude chamber developed a paralysis from the waist up; the patient could only breathe through movement of the diaphragm due to paralysis of the other respiratory muscles. After being returned to sea level pressure, the patient quickly recovered.

There may be localized pain in the larger nerves, and in addition, there may be involvement along the whole course or distribution of a peripheral nerve trunk. This may take the form of a descending neuritis which is manifested by severe pain and marked tenderness along the course of the whole nerve, or it may be peripheral, affecting only the nerve endings. In the former instance the condition is immediately relieved by descent and leaves no after effects. In the latter there is usually a neurodermatitis which will be described under the dermal system.

2. Cardiovascular System. Many difficulties
SYMPTOMATOLOGY

may arise in this system because the bubbles may be carried to all parts of the body by the blood stream. One of the great dangers in this system is that a bubble may be carried to an end artery in the cerebrospinal system and lead to infarction of that area, resulting in a catastrophe such as those mentioned under the cerebrospinal system. It is conceivable that emboli could lodge in the coronary vessels and cause cardiac embarrassment or failure. Emboli could also cause pulmonary edema when lodged in the lungs. Although emboli in the viscera have never been subjectively recorded on high altitude flight, they have been found in abundance during postmortem examination of experimental animals.

3. Respiratory System. As in the cases of all emboli which come from the arterial side, one of the first lodging places is in the lungs. The capillaries of the lungs act as a giant sieve which strains the blood and sifts out any of the bubbles which appear in the general circulation. As a result more and more of the vessels in the lungs become obstructed and occasionally a large one is suddenly deprived of its circulation when one of the larger vessels becomes obstructed.

If a number of small vessels are obstructed
SYMPTOMATOLOGY

there is a burning sensation in the lungs. If, on the other hand, there is a sudden obstruction of a larger vessel, there is a sharp stabbing pain. In both cases edema of the lungs rapidly develops, followed by unproductive coughing spells. When a descent to a lower altitude is made the edema is relieved and the accumulated fluid in the alveoli is coughed up and removed. Due to the rarefied air at high altitudes the cough is unproductive until descent is made.

4. Structural System, (including osseous, periosteal, connective, muscular and fatty tissue). The most frequent symptom of aeroembolism is pain and it may occur alone or in conjunction with other symptoms. The joints are often the sites of intense pain. This pain may be from the unyielding tissue around the joint or it may arise from the distention of the joint cavity itself.

In cases where joint pain has been observed the subjective sensations are not located in the joint cavities, but rather just above or below it; this would indicate that the difficulty is centered either in the bone, the periosteum, or the tendinous connections about the joint. There is no pain noted along the shaft of the bone so we can assume that the periosteum and the
bone itself are not involved, but rather the tendons are the seat of the trouble. The joint usually acts as a barometer to indicate the onset of an attack of aeroembolism or caisson disease. The pain is usually mild at first, being no more than an ache, but it rapidly grows worse and soon becomes boring or gnawing in character and is so severe as to be almost intolerable. The onset of pain about a joint is seldom followed by pain in a second joint, but is frequently followed by symptoms in other systems of the body.

5. Dermal System. Dermatitis is usually manifested by the appearance of red, macular rash, but sometimes it appears as a giant urticaria with a smooth white elevated surface. One of these giant urticaria was observed to cover the whole surface of the subject's abdomen—it was circular in outline and was twelve inches in diameter. The subject had never before been bothered by urticaria or any other allergic conditions, and from this it would appear that the mechanical irritation of gas bubbles in the posterior horn cells of the spinal cord may offer some clue to some of those neuro-dermal conditions whose etiology is as yet more or less obscure.
SYMPTOMATOLOGY

Pruritis and thermal sensations of hot and cold without any objective signs have also been noted. Another very interesting and frequently noted symptom is a type of formication which does not stay localized, but feels exactly like a small compact colony of ants rushing madly over the surface of the body. (2).

"With ascents at rates as high as 12,000 feet per minute up to 30,000 feet, symptoms are seldom noted even after prolonged stays at that altitude. At 32,500 feet susceptible individuals may develop symptoms after a few minutes or after two or three hours if the rate of climb is 200 feet a minute or above. At 35,000 feet a rate of climb of 500 to 1,000 feet per minute may or may not produce symptoms but where they do appear they are apt to come after only a short period of delay and to be rather severe. At 37,500 feet and above any reasonable rate of climb is very likely to produce symptoms in even the most resistant individual.

The relative frequency with which the different symptoms appear in the body seems to differ somewhat among individuals but in general the order of frequency is as follows:
SYMPTOMATOLOGY

1. Structural system:
   a. Knee joint pain
   b. Finger joint pain
   c. Shoulder joint pain
   d. Ankle joint pain
   e. Hip joint pain
   f. Others.

2. Dermal system:
   a. Thermal hyperesthesia
   b. Formication
   c. Pruritis
   d. Neurodermatitis
   e. Urticaria

3. Cerebrospinal system:
   a. Neuritis
   b. Paralysis
   c. Convulsions
   d. Coma

4. Respiratory system:
   a. Pulmonary pain
      (1) Burning
      (2) Stabbing
   b. Pulmonary edema
SYMPTOMATOLOGY

c. Cough
d. Expectoration." (2).

Lovelace has this to say about the symptoms of aeroembolism: "Some of the symptoms of air emboli are slight headache, smarting or stinging of the conjunctiva, formication and fleeting joint pains. Aching pain in the extremities and joints, stabbing chest pains and inces­ sant nonproductive cough are believed to be quite patho­ gonomic, especially if they begin suddenly and become progressively more severe at the higher altitudes, only to subside at the lower ones." (25).

We have seen that the symptoms of aeroembolism are very much like those which are observed in the bends of caisson disease; the striking difference is that they are not so severe nor do they last so long in aeroembol­ ism. There are no deaths which are recorded as caused by aeroembolism primarily. Perhaps planes have crashed to earth because of pilots temporarily paralyzed or rendered unconscious by aeroembolism. In modern planes with their great weight a free fall out of control for any great distance is apt to result in disintegration of the plane and death to the occupants. If, however, the plane should fall in a shallow dive to, let us say,
SYMPTOMATOLOGY

twenty-five thousand or twenty thousand feet the automatic recompression will bring the crew back to normal physiological function. Perhaps, therapeutic falls like this have actually occurred; the literature does not record any, but even if they have occurred, they were probably attributed to anoxia and not to aeroembolism.
PROPHYLAXIS

We now come to the most interesting chapter on aeroembolism; namely, prophylaxis. All other parts of this thesis merely point the way, so that our knowledge of etiology and pathology may help to prevent the phenomenon itself, just as a thorough knowledge of this entity will help towards a solution of the problem, so will a knowledge of how the problem was solved in the related phenomenon of caisson disease aid in the solution of this problem.

Deep sea diving has been made comparatively safe by the adoption of the decompression tables formulated by Boycott, Damant and Haldane, and the hazards of work in compressed air have been minimized as a result of the practise of decompression evolved from extensive tunnelling projects in New York state. Empirical rather than quantitative data, however, formed the basis for the formation of decompression tables. Thus, two of the basic assumptions underlying the diving tables are that the absolute pressure can be safely halved during the first stage of decompression, and that about five hours are necessary for the complete abortion or elimination of nitrogen from the body. (7).

Campbell and Hill in 1931 measured the nitrogen
eliminated by man when oxygen was breathed for short intervals at normal barometric pressure. In 1933 they worked on the quantitative data with reference to nitrogen absorption and solubility in the brain, liver and bone marrow of the goat. (13).

As a result of 1,360,000 decompressions up to 1822 and over 3,000,000 at the present time attended by a negligible number of accidents, (six cases per hundred thousand decompressions), the decompression schedule of New York state serves as an excellent criteria for the evaluation of conclusions drawn from laboratory results. This schedule differs from that of the standard diving table in that the gauge and not the absolute pressure is halved rapidly during the first stage of decompression and then more slowly at a uniform rate during the remainder of decompression. The duration of exposure, moreover, to excess pressure from twenty-two to fifty pounds is so reduced that when the gauge pressure is halved a fairly constant difference not exceeding thirteen pounds exists between the nitrogen tension in the body and the nitrogen tension in the lungs.

In the formulation of a decompression schedule from the laboratory data, the body is regarded, not
as five tissues with varying nitrogen tensions, but essentially as a unit composed of fat and water in which the process of diffusion from water into fat and vice versa tends to equalize the nitrogen during saturation and desaturation.

Decompression can be correctly divided into two stages. During the first stage the pressure is rapidly lowered, (fifteen pounds per minute), until a designated difference is created between the average nitrogen tension in the body and the nitrogen tension in the lungs. During the second stage the gauge pressure is lowered at a uniform rate so that this pressure difference is maintained at a constant value during the remainder of decompression.

The safe difference in nitrogen tension that can be maintained depends upon the degree that nitrogen can be held in supersaturation by the blood and other tissues of the body. For the present this value must be estimated from empirical data with the provision that it may be modified as a result of diving and other tests. If rapid decompression from nineteen to zero pounds is safe, then a difference in nitrogen tension between the body and lungs of $0.79 \times 19$, (15 pounds), will certainly
be safe at any pressure level. From the analysis of the New York state regulations a difference in nitrogen pressure of ten pounds, (air pressure 12.5 pounds), may be regarded as the probable minimum for maintenance during decompression. The probable value, therefore, which will not unduly delay decompression or produce bubble formation lies between ten and fifteen pounds, (twelve to nineteen pounds air pressure). (7).

Since the object of this thesis is not to go into the details of decompression up to one atmosphere, we will not delve any further into the realm of decompression and its various mathematical aspects. Suffice to say that very elaborate decompression rate tables have been made and are used by the several navies of the world and also by engineering companies engaged in underwater work where caissons are used.

It should be remember that the obese type of individual saturates with nitrogen much slower than the thin, and conversely he desaturates much slower.

The individual who is exercising vigorously, such as a diver working on the ocean floor or "sandhog" in a caisson, saturates much more rapidly than an individual who is not exercising but who is exposed to the
PROPHYLAXIS

same pressure. On the other hand, the individual who exercises during decompression desaturates much more rapidly than the passive one. Therefore, it has been recommended that in order to shorten the period of decompression the individual should exercise as violently and as long as feasible at each rest during his decompression. (18).

Helium mixed with oxygen has been used to eliminate nitrogen from the body in place of pure oxygen. Since the use of helium has also been taken advantage of by aviation we shall discuss it more in detail under aeroembolism.

Let us now proceed to the problem of prophylaxis as we find it in aviation medicine. This is, indeed, a serious problem especially at the present time. It is not too much to say that in a large part the defense of our big cities and warplants from aerial bombardment hinges on the successful solution of this problem.

One of the simplest ways to prevent aeroembolism in high altitude flying is by removing as much of the nitrogen which will form bubbles as possible. This is done by having the pilot and crew breathe pure oxygen while exercising. When Lockheed wished to test their
new twin motored interceptor plane, known as the P-38 by the U. S. Army or the Lightning by the British, they wanted to determine its maximum rate of climb. The engineers knew that the plane would climb in excess of 12,000 feet per minute for at least 28,000 feet and would still rise rapidly to 35,000 feet and above. The medical men connected with Lockheed realized that the test pilot would have to be protected against the hazards of aeroembolism, so they set about to do this by having him breathe pure oxygen. The pilot entered a small room in which there was a stationary bicycle and there he donned an oxygen mask connected with a tank of pure oxygen. Mounting the bicycle he peddled it for one-half hour, at the rate of two and one-half miles per hour, while inhaling the oxygen and thus ridding his body of the dangerous nitrogen. After one-half hour, the pilot still wearing his mask, hooks up to a portable oxygen tank and goes to his plane, where he again changes to a tank located in the plane. He must not take one breath of "fresh air" or all the half hour's work will be lost and his tissues will again contain nitrogen. (24, 29).

An even simpler way to eliminate the nitrogen
PROPHYLAXIS

from the body would be by ascending slowly after an altitude of 32,500 feet has been reached. By slowly we mean less than 200 feet of altitude gained per minute up to 37,000 feet. Above 37,000 feet the rise would have to be in tens of feet per minute rather than hundreds.

Dr. Behnke reports that when the body has been completely denitrogenated, subjects who previously suffered from the bends after several hours at 40,000 feet now felt perfectly alright, even after long exposure. However, pure oxygen must be breathed for several hours, (at least five), before 95% of the nitrogen is eliminated. Therefore, pre-oxygenation is not feasible in fighter pilots who must constantly be ready to ascend.

If the fighter pilot breathes pure oxygen for an hour before he enters the plane, the oxygen will decrease his susceptibility to the bends, but will not prevent them ultimately if the pilot remains at a high altitude for several hours.

In a susceptible subject studied by Commander Behnke severe bends developed between 25,000 and 28,000 feet without preoxygenation. With forty-five minutes preoxygenation bends did not develop until 30,000 feet;
PROPHYLAXIS

with ninety minutes preoxygenation the ceiling was raised to 34,000 feet; with three hours to 37,000 feet; and after five hours preoxygenation the bends susceptible subject withstood 40,000 feet for two hours without experiencing symptoms. (8).

One other possible method of eliminating nitrogen from the body is through the use of a mixture of oxygen, (21%), and helium, (79%), as was suggested by Sayers, Yant and Hildebrand, (1925). This has found great favor in diving as it replaces the body nitrogen without the use of dangerous high partial pressures of oxygen. However, since the latter condition is not a problem at pressures of less than one atmosphere, this method appears to offer no advantage over pure oxygen inhalation for use in aviation. Even assuming a slight for the oxygen-helium mixture, the use of this mixture would involve the necessity of procuring extra equipment and supplies in addition to those already available for oxygen. (2, 31).

Dr. J. F. Fulton of Yale University has this to say about the use of helium: "Oxygen for prolonged administration is hazardous because of its toxicity. Nitrogen can be removed as effectively from the body
PROPHYLAXIS

of a helium-oxygen mixture as with pure oxygen. Since helium is only one-third as soluble in fat as nitrogen, the quantity of gas available for bubble formation, especially in the bone marrow, which comprises as much as 90% fat, is greatly reduced. If a human being were saturated with helium, instead of nitrogen, it would require only ninety minutes of oxygen inhalation to eliminate dissolved helium in contrast to the five hour period for nitrogen elimination. Commander Behnke believes it would be practical to have bomber pilots in a ready room filled with an atmosphere of oxygen and helium prior to flight. So far, however, this proposal has not had service trial. (20).

A third method would be to pressurize the cabins of planes which rise to high altitudes. This at first glance would seem to be the best solution to the problem of aeroemboli. And, indeed, it would were it not for certain practical considerations. One of these is weight; a plane which has a pressurized cabin must be internally braced so that it will not explode when a high altitude is reached. If the same pressure is maintained in the cabin as we find at sea level, we will have a pressure of roughly fifteen pounds to the
square inch pressing out on the cabin walls, and thus transmitting this thrust to the bulkheads and stringers of the fuselage itself, while on the outside the pressure may be half this pressure or even less. Thus, we have all the elements necessary for a violent explosion if the fuselage is not well braced, and bracing means extra weight. Weight in the commercial plane means the loss of dollars, but in the military plane it may mean the loss of life itself. Weight which is used in strengthening the fuselage is just that much lost to the use of heavier armament and armor, of extra ammunition or gasoline, of a more powerful motor. Even if the plane is supercharged and the cabin successfully hermetically sealed, only one bullet from a thirty caliber machine gun is needed to undo all the work of sealing the cabin. From what has been said in this chapter, the reader will probably assume that there has been no satisfactory method for prophylaxis devised to prevent aeroembolism, that is no method that is practical.

The most practical method is probably nitrogen desaturation, and this is highly impractical for interceptor pilots. For this reason, we should strive to pick men for our high altitude combat planes who are the
least susceptible to aeroembolism.

In choosing pilots we must remember several different factors which enter into the susceptibility of individuals to the bends. Young men are less susceptible to the bends than older men. Pilots in the age group eighteen to twenty-four years can often stand pressures as low as found at 40,000 feet for prolonged periods without any symptoms.

Secondly, men who have had a joint injury are more susceptible to the bends in the effected joint.

Thirdly, men of the same age group are not all affected by high altitudes in the same manner. It is believed, that at least 50% of the young adult population can stand high altitude work. It is possible to weed out the susceptible pilots by placing them in decompression chambers for tests.

In the future, we may discover that certain drugs may also diminish the susceptibility of individuals to aeroembolism, but so far no such drug has been found.
The best treatment in any phase of medicine is, of course, prophylaxis, but this is even more true in the bends in either caisson disease or aeroembolism. There is no good treatment once they occur, except immediate recompression followed by slow decompression, so we are really using prophylaxis as treatment. It is interesting to review some of the methods that have been tried as treatment in the past.

Very early in the history of the bends it was realized by medical men that recompression was the best method of treatment. This was sometimes accomplished in rather crude ways. The first method was to immediately lower the patient with bends back to the depth from which he has just ascended, or if the symptoms were mild he would only be lowered a fraction of the depth. It is easy to understand why it would be difficult to lower a diver suffering from the bends back into the depths. Especially, when we consider that he has to be placed back into his diving suit. He is lowered to the required depth and then kept there for the required length of time and slowly raised. Needless to say, he is without the benefit of medical assistance, and merely hangs suspended like a drag anchor to the boat. The early workers
TREATMENT

with caisson who had an attack of the bends when they emerged, were placed back in the caisson which they had just left, and were then slowly removed to areas of decreased pressure.

Some of the men did not show symptoms of the bends until after they had left the scene of their work. In cases like this, decompression was out of the question so other treatments were tried. It is interesting to note these treatments in passing:

Morphine was used and still is used in order to relieve the pain, but of course, in the strictest sense is not a treatment.

Ergot and Ergotinine have both been employed, but the results were not good. Digitalis also had no effect. Ingestion of atropine was not as satisfactory as was morphine in alleviating the pain.

A Faradic battery was much more useful in relieving the attendant's symptoms than those of the patient suffering from the bends. (36).

Paul Bert tried using oxygen inhalations in animals whose heart sounds indicated the presence of free gases. He considered that while the oxygen prolonged their lives, death was never averted. (6).
A moderately tight bandage applied to the painful limb seems to afford some relief to the patient, as do stimulating linaments such as those containing aconite and belladonna. (36).

As was stated at the first of this chapter, the best method of treating the bends is by recompression, and the best way to recompress is by using a medical chamber. This is a small room in which any depth can be simulated by merely raising the pressure by the use of air pumps. The patient is placed in the chamber and the atmosphere raised to the desired pressure. The medical attendant observes the patient through glass partitions and if he needs attention it is immediately available. It should be stressed here that all ships such as naval salvage vessels should be equipped with at least one recompression room. This is also true of gangs working in caissons on bridges and other depth work. These men should also be under the constant supervision of a medical expert on caisson disease.

In cases of emergency, divers have been brought to the surface immediately and placed in a recompression chamber, where the pressure is raised to the equivalent of the first stop in the water and the diver is given
TREATMENT

the decompression he would have received in the water. This is referred to as "water stage elimination decompression" or surface decompression.

Surface decompression is advantageous because it gets the diver out of the cold water and the tide into a warm chamber where he can be easily cared for. Also it releases diving apparatus for use by other divers. (23).

The length of time that the diver remains in the chamber depends on how long he has been submerged and to what depth. This paper will not reproduce the charts and complicated formulas by which this time is determined, instead a typical case will be dealt with. This case illustrates the symptoms as well as the treatment of the bends in their more severe form.

In the following case the torpedo room of the submarine served as a recompression chamber: "The patient, after diving to a depth of 120 feet on the morning of January 11, 1923, removed his helmet when he had slowly ascended to a depth of 60 feet and immediately came to the surface. Shortly after noon he developed symptoms of caisson disease, and having become unconscious at 2:00 P.M., he was placed in a diving suite and lowered
TREATMENT

gradually to a depth of 60 feet. When the writer arrived on the scene the patient was at a depth of 20 feet from which he was gradually brought to the surface. He was still unconscious and quite cyanotic. Pulse was over 150 and respirations were rapid. As the patient did not improve at a depth of 20 feet, he was placed in the torpedo room of a submarine at a pressure of 16 pounds, at which pressure he remained from 6:10 P.M. to 6:45 P.M. During this time he had violent convulsions, but gradually improved. Regaining consciousness, he complained of general body pain. From 6:45 to 7:15 P.M. the pressure was slowly reduced to 10 pounds, at 7:30 P.M. to 8 pounds, and at 7:45 P.M. to 6 pounds. At this time his pulse was 140 and respirations were 30. Pressure was reduced to 4 pounds at 8:10 P.M., and at 8:20 P.M. to 2 pounds. He complained of diminished vision. By 8:45 P.M. all excess pressure had been removed. The patient was apparently in fair condition, but at 9:15 P.M. he had a severe convolution and became unconscious and cyanotic. He was placed one more in the pressure chamber and the pressure gradually increased to 16 pounds, all the pressure available. At 10:15 P.M. he had a second convolution, pulse became too rapid to count,
TREATMENT

respiration was over 50, but cyanosis had disappeared. At midnight patient was still unconscious, respiration 56, again cyanotic and pulse too fast to count. At 1:00 A.M. Cheyne-Stokes breathing occurred, shortly after this the patient died." (1).

The treatment of aeroembolism, as far as aviation medicine is concerned, can be summed up in one word, descent. The pilot, if he or any member of his crew is suffering from the bends, shall immediately descend. Usually after he has descended to 25,000 feet, the symptoms will be relieved and the flight can be continued at this level. If, however, the symptoms are severe, a landing should be affected as soon as possible. After landing, the nearer the airport is to sea level the better, the patient should be placed on 100% oxygen inhalation until his symptoms entirely disappear. (2).

The effected individual should not attempt flight again until all the excess nitrogen has been "washed" out of his system. If the individual is particularly susceptible to altitude he should be either grounded or transferred to some branch of aviation, such as attack aviation, where the required altitudes are low enough not to affect him adversely.
TREATMENT

Fundamentally we see that the treatment for both aeroembolism and caisson disease is the same. Namely, recompression at the earliest possible moment.
SUMMARY

This writer has endeavored in this thesis to compare that older entity, caisson disease, with its younger prototype, aeroembolism. As can be seen, there is really no marked difference between the two, except perhaps, that the symptoms of caisson disease are more severe.

In the next several paragraphs an attempt will be made to reiterate what this writer considers to be the salient points of this paper.

Etiology. Caisson disease as well as aeroembolism is caused by too rapid decompression from either one atmosphere or to one atmosphere, depending whether we are considering diving or high altitude flight.

Pathology. The rapid decrease in pressure releases nitrogen in the blood and in the tissues in the form of bubbles. These bubbles are released in all parts of the body, but are found in the greatest abundance in adipose tissue. The symptoms are usually caused by the release of nitrogen in the tissues and not by the nitrogen released in the blood where it is rapidly carried to the lungs and eliminated. Of course, if the bubbles are too large, they will cause trouble by acting as emboli and obstructing the blood flow.
SUMMARY

Symptomatology. The chief symptom of both caisson disease and aeroembolism is the bends or joint pains. There may also be symptoms of mental and physical lassitude, especially in aeroembolism. In extreme cases, we sometimes find paralysis and even death due to caisson disease. There are no actual recorded cases of death from aeroembolism, although pilots may have died from this cause and the pathological evidence obliterated in the resultant crash.

Prophylaxis. The best treatment for the bends is, of course, prophylaxis. In the case of divers or caisson workers, slow decompression should be used by raising the diver to the surface slowly or raising him rapidly and placing him immediately in a decompression chamber. As far as aviation is concerned, the ascent should be made slowly and the type of men who fly at high altitudes should be carefully selected. The other alternative is to have the pilot wash the nitrogen out of his tissues by breathing pure oxygen from one-half hour to five hours while exercising before the ascent. Of course, sealed pressure cabins are the ideal solution when they are feasible.

Treatment. Treatment of caisson disease is
SUMMARY

best affected by placing the sufferer in a decompression chamber and rapidly raising the pressure, and then slowly decompressing him. Morphine should be used to ease the pain. In aeroembolism immediate descent, which is a form of decompression, is the treatment of choice.

According to Dr. J. F. Fulton of Yale University, the bends have been seriously overemphasized in aviation medicine and have made the military pilot unduly apprehensive about this disease. He states that Lieutenant-Commander A. R. Behnke believes that the bends are no longer of great importance in military aviation due to the developments in the past year. (20).

Without having read Dr. Behnke's article, which is not yet available, and with all due respect to both Dr. Fulton and Commander Behnke, I would like to take issue with them on this statement.

First. What improvements have taken place? Surely human physiology has not undergone any radical changes in the last year. If technical improvements have been made in the planes, which they undoubtedly have, this is all to the good. But the danger of bends still lurks, if we grow careless, just as smallpox does when we grow complacent about vaccination.
Second. We must never forget that aviation is always fluid, even if we have frozen our military types of planes temporarily. We must remember that there is good evidence that the Germans and perhaps the English have used rocket power in aiding their heavy bombing planes to get off the ground. What is to prevent the use of rockets as motive power to completely supplement air screws as steam did sail? The answer is not, as many think, so much a suitable fuel, as it is a metal which can be used in the rocket nozzle which will not melt under the intense heat. We must remember that while air screws become larger and larger and less and less efficient as we approach the near vacuum of the stratosphere, rockets operate as well in a vacuum as in the atmosphere close to the earth. Now, if rocket propulsion is used, and we have reason to believe that it may be in the near future, the ascents will be vertical and the speed tremendous, so unless we have completely sealed pressurized cabins, aeroembolism will become even more important. Does this sound fantastic? So, in 1927, did a bomber that could cross the Atlantic in four hundred minutes.

Third. We must remember that in the days of
Summary

Peace to follow, all types of people will take to the stratosphere at a rapid rate of ascent. These will not be picked, lean young men, but fat people, aged people, alcoholics and near alcoholics; in fact, any type of individual who will use the commercial airlines. So, although we may hold aeroembolism in check by aircraft improvements, we will still have it with us in case of structural failure.
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