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THE PREVENTION OF COMPRESSED-AIR ILLNESS.

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(*From the Lister Institute of Preventive Medicine.*)

[With 7 Figures and 3 Plates.]

CONTENTS.

INTRODUCTION.

MEN who have been working in compressed air, as in diving, preparing foundations of bridges, etc. under water, or making tunnels or shafts through water-bearing strata, are liable on their return to atmospheric pressure to a variety of symptoms generally known as "diver's palsy" or "caisson disease," but which may more conveniently be designated "compressed-air illness." It was shown experimentally by Paul Bert[1] that these symptoms are due to the fact that gas (chiefly nitrogen) which goes into solution in the blood and tissues during exposure to compressed air is liberated in the form of bubbles on too rapid decompression, and produces local or general blockage of the circulation or other injury. Subsequent investigations, for an account of which we must refer more particularly to the treatise on the subject by Heller, Mager and v. Schrötter[2] and to recent papers by Hill and McLeod[3] and Hill and Greenwood[4], have confirmed and extended Paul Bert's conclusions.

It was pointed out by Paul Bert that by means of very slow decompression the symptoms of caisson disease could be avoided, but his experiments were not sufficient to furnish data as to what rate of decompression would be safe. Nor has subsequent human experience in engineering undertakings solved this problem; and the risks attending work in compressed air at excess pressures of over 1½ to 2 atmospheres are notorious. Heller, Mager and v. Schrötter have endeavoured to formulate rules as to safe decompression; and they express the belief that perfectly uniform decompression at the rate of 20 minutes an atmosphere would always be safe. Following this rule, which is based on a calculation, Hill and Greenwood decompressed themselves, without any serious symptoms, after short exposures at excess pressures of as much as five and even six atmospheres.

Although the rules formulated by the above-mentioned observers constituted a distinct step in advance, it appeared to us that, for reasons which will be explained below, there were grave doubts as

[1] *La Pression Barometrique*, 1878.

[2] *Luftdruckerkrankungen*, 1900; also v. Schrötter, *Der Sauerstoff in der Prophylaxie und Therapie der Luftdruckerkrankungen*, 2nd edition, 1906. The former work contains a very full abstract of all previous investigations on the subject.

[3] This *Journal*, vol. III. (1903), p. 401 (and references there given): see also *Recent Advances in Physiology*, 1906, pp. 233—255.

[4] *Proceedings of the Royal Society*, vol. LXXVII. p. 442, 1906; vol. LXXIX. p. 21, 1907; also *British Medical Journal*, July 7th, 1906, Feb. 16th, 1907, June 22nd, 1907.

to the safety of their recommendations, and particularly as to whether uniform decompression is desirable. The need for framing definite rules as to safe decompression in the shortest possible time presented itself in a very definite form in connection with the work of the Admiralty Committee on Deep Diving[1], of which one of us was a member. Our investigation, which was planned with the more particular object of furnishing information required for securing the safety of divers ascending from deep water, was rendered possible by the gift to the Lister Institute by Dr Ludwig Mond, F.R.S. of a large experimental steel pressure chamber and by substantial financial and other help from the Admiralty, Messrs John Aird and Son, the late Mr Basil Ellis, Messrs S. Pearson and Son, Ltd., and Messrs Price and Reeves.

The formation of gas bubbles in the living body during or shortly after decompression evidently depends on the fact that the partial pressure of the gas or gases dissolved in the blood and tissues is in excess of the external pressure. But it is a well-known fact that liquids, and especially albuminous liquids such as blood, will hold gas for long periods in a state of supersaturation, provided the supersaturation does not exceed a certain limit. In order to decompress safely it is evidently necessary to prevent this limit being exceeded before the end of decompression. Whether or not the decompression is free from risk will depend on the degree of supersaturation which can be borne with safety, the extent to which the blood and tissues have had time or opportunity to become saturated, and the extent to which they have had time to become desaturated again during decompression. In carrying out our investigations we have kept these three factors constantly in view, and it is necessary to discuss them in some detail before proceeding further.

[1] The Report of this Committee, which has recently appeared as a blue-book, contains a full account of the experimental investigations on Diving, carried out under its auspices at Portsmouth, off the West Coast of Scotland, and elsewhere, during the last two years: also a short summary of the experiments detailed in the present paper, and many data as to the occurrence of compressed-air illness in connection with diving and other work in compressed air. The conclusions and recommendations of the Committee are summarised at the beginning of the Report.

Part I. Theoretical.

A. *The rate of saturation of the body with nitrogen during exposure to compressed air.*

When a man or animal is placed in compressed air, the blood passing through the lungs will undoubtedly take up in simple solution an amount of gas which will be increased above normal in proportion to the increase in partial pressure of each gas present in the alveolar air. The experiments of Haldane and Priestley[1], which have since been extended by Hill and Greenwood[2], show that the partial pressure of CO_2 in the alveolar air remains constant with a rise of atmospheric pressure: hence there can be no increase in the amount of CO_2 present in the blood during exposure to compressed air. As regards oxygen, the amount in simple solution in the arterial blood will certainly increase in proportion to the rise in alveolar oxygen pressure; but as soon as the blood reaches the tissues this extra dissolved oxygen, which (except with exposures to enormous pressures) is only a small part of the total available oxygen in the arterial blood, will be used up, so that in the tissues and venous blood there will be at most only a very slight increase in the partial pressure of oxygen. For practical purposes therefore we need only take into consideration the saturation of the body with nitrogen.

In view of what is known as to the ease and completeness with which the blood becomes aerated in its passage through the lungs, there seems no reason to doubt that in compressed air the blood reaching the lung capillaries must become instantly saturated with nitrogen at the partial pressure existing in the alveolar air (see p. 351). At the commencement of exposure to compressed air this blood, on being carried to the tissues, will by diffusion share with them its excess of nitrogen and then return to the lungs for a fresh charge. By the constant repetition of this process the tissues, and the venous blood leaving them, will gradually become more and more saturated with nitrogen at the partial pressure of the nitrogen in the alveolar air, which will be practically the same as in the inspired air. Since the rate of blood supply and the solubility of nitrogen per unit mass of tissue vary greatly in different parts of the body, the rate of saturation

[1] *Journal of Physiology*, vol. xxxii. (1905), p. 229.
[2] *Proc. Roy. Soc.*, B, vol. lxxvii. p. 442.

will vary correspondingly. We may however form some rough general idea of the average rate of saturation by assuming as a basis of calculation that the blood is evenly distributed throughout the body, and that the tissues are similarly constituted in all parts.

According to the figures adopted by Bohr[1], 100 c.c. of blood take up in simple solution at the body temperature 0·87 c.c. of nitrogen for each atmosphere of air pressure. This is only 8 % less than would be taken up by water under the same conditions. Blood contains nearly the same percentage of solids as the semi-liquid tissues (apart from fat) in most parts of the body, and we may assume that these tissues will take up nearly the same proportion of nitrogen as blood. The earthy constituents of bone (about 3 % of the body weight) probably take up no nitrogen. On the other hand the body fat, as was recently shown by Vernon[2], who made a number of determinations at the body temperature with special reference to our investigations, takes up about six times as much nitrogen as an equal weight of blood. The body of a well-nourished man probably contains fully 15 % of its weight as fat or fatty material. Hence it may be estimated that it will, when saturated at any given pressure, on an average take up, weight for weight, about 70 % more nitrogen in simple solution than the blood under the same conditions, and that the whole body of a man weighing 70 kilos will take up about one litre of nitrogen for each atmosphere of excess pressure.

Now the weight of the blood in man is about 4·9 % of the body weight[3]: hence the amount of nitrogen held in solution in the body, when it is completely saturated at any given pressure, will be about $\frac{170}{4{\cdot}9}$, or 35 times as great as the amount present in the blood alone. If therefore the blood distributed itself evenly and at the same rate throughout the body, the latter would have received, at the end of one complete round of the blood after sudden exposure to high pressure of air, one thirty-fifth of the excess of nitrogen corresponding to complete saturation. The second round of the circulation would add one thirty-fifth of the remaining deficit in saturation, *i.e.* 1/35 × 34/35 of the total excess: the third round would add 1/35 × (34/35 × 34/35) of the total excess, and so on. On following out this calculation, it will be found that half the total excess of nitrogen would have entered the body

[1] *Nagel's Handbuch der Physiologie*, vol. I., 1905, p. 63.

[2] *Proc. Roy. Soc.*, vol. LXXIX. B, 1907, p. 366.

[3] Haldane and Lorrain Smith, *Journal of Physiology*, vol. XXV., 1900, p. 340.

after 23 rounds of the circulation, three-fourths after 46 rounds, seven-eighths after 69 rounds, and so on. The progress of the saturation of the body with nitrogen is thus a logarithmic curve of the form shown in Figure 1[1].

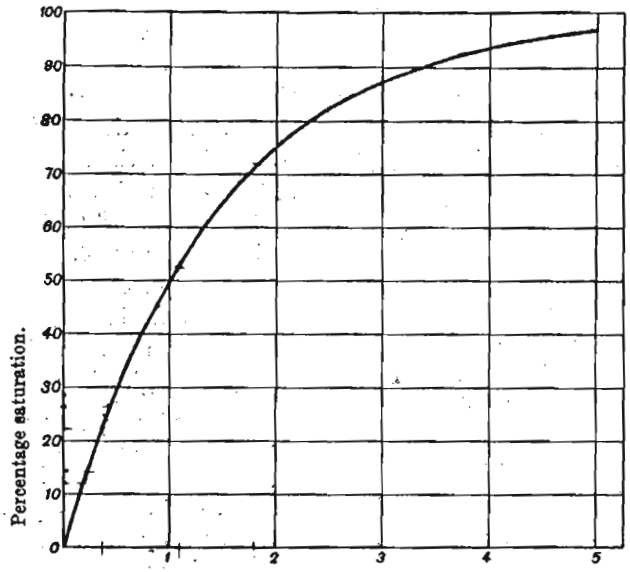


Fig. 1. Curve showing the progress of saturation of any part of the body with nitrogen after any given sudden rise of air pressure. The percentage saturation can be read off on the curve, provided the duration of exposure to the pressure, and the time required to produce half-saturation of the part in question, are both known. Thus a part which half-saturates in one hour would, as shown on the curve, be 30 % saturated in half-an-hour, or 94 % saturated in 4 hours.

Experiments on animals have shown that the venous blood entering the lungs contains about two-fifths less of oxygen than the arterial blood. If we assume that the same proportion holds good for a man at rest, and that very little oxygen is used up in the lungs themselves, the percentage of oxygen gained by the blood in the lungs must be about 8 %, or about double the percentage diminution in the expired

[1] This calculation is in principle similar to that made by Zuntz (*Fortschritte der Medizin*, 1897, No. 16), and worked out more fully by Heller, Mager and v. Schrötter (*loc. cit.*). On account, however, of the discovery that fat has a very high coefficient of absorption for nitrogen, and that the blood volume in man is considerably less than was formerly supposed, our calculation gives a much slower rate of saturation per round of the circulation.

air as compared with the inspired air. The volume of blood passing through the lungs is therefore about double the volume of air breathed. Since this volume of air (measured dry and at standard pressure and temperature) averages about seven litres per minute[1] for a man of 70 kilos during rest, the volume of blood passing through the lungs may be estimated at about 3·5 litres per minute[2]. The total blood volume is however also about 3·5 litres, so that a volume of blood equal to the total blood volume probably passes through the lungs about once a minute during rest. We may therefore substitute minutes for rounds of the circulation in the above calculation of the rate of saturation of the body with nitrogen, so that, if the assumptions made for the purposes of the calculation held good for a man exposed to compressed air, his body would be half saturated with the excess of nitrogen in 23 minutes, three-fourths saturated in 46 minutes, etc.

In reality, however, this calculation affords at best only a very rough general idea of the actual rate of saturation, since it is known that the distribution of blood per unit of body weight through various parts of the body varies greatly, and that the rate of circulation through any given part varies according as the part is at rest or in a state of activity. The proportion of fat and fatty material is also very different in different parts of the body, so that the capacities of different tissues for taking up nitrogen must vary accordingly. We should expect therefore that some parts of the body will saturate much more rapidly than the calculation shows, and other parts much more slowly. Direct experimental evidence of far more rapid saturation in some parts of the body has recently been furnished by Hill and Greenwood[3]. Their method was to determine the free nitrogen in samples of urine secreted shortly after exposure to high pressure, and shortly after return to normal pressure. A sufficiently copious secretion of urine was produced by previously administering large drinks of water to the subject of the experiment; and they found that, within about ten minutes of exposure to high pressure, samples of urine secreted were saturated at this pressure. Conversely, on lowering the pressure to normal, the excess of nitrogen disappeared within a few minutes.

[1] Haldane and Priestley, *loc. cit.*, p. 245.

[2] As a result of numerous experiments on man with the lung catheter Loewy and v. Schrötter (*Untersuchungen über die Blutcirculation beim Menschen*, 1905, p. 90) infer that the average rate of blood flow during rest is slightly faster. At present, however, there is some doubt as to the interpretation of results obtained by the lung catheter method.

[3] *Proc. Roy. Soc.* B, vol. LXXIX., p. 21, 1907.

These results seem to show conclusively that the kidney substance became saturated with nitrogen at a rate about ten times as great as would correspond to the above calculation. From the data given it appears, however, that urine was being secreted with great rapidity during the experiments. For instance, 135 c.c. were secreted in five minutes in one observation where the quantities and times are recorded. This is about thirty times the average rate of secretion, so that the circulation of blood through the actively working kidneys must have been greatly increased.

Equally clear evidence of the existence of a far slower rate of saturation is afforded by the experience of men working in compressed air, particularly in caissons and tunnels at moderate pressures. It is well known to those practically familiar with such work that the risk of symptoms occurring on decompression depends on the duration of the exposure. There is very little risk on rapid decompression after short exposures of less than an hour to an excess pressure of two atmospheres or even somewhat higher pressure; but as the duration of exposure increases hour by hour, so do the risks on decompression increase. We are assured by Mr E. W. Moir (of the firm of Messrs S. Pearson and Son, Ltd., Westminster), who has had an exceptionally large experience of tunnelling work in compressed air at excess pressures up to about $2\frac{1}{2}$ atmospheres, that the maximum of risk is not reached after even three hours, so that a limitation of working shifts to three hours markedly diminishes the frequency of compressed-air illness. Hence in some parts of the body saturation with nitrogen must still be incomplete after three hours. Another observation pointing in the same direction is that when the daily working period was $8\frac{1}{2}$ hours under pressure with two intervals of about $1\frac{1}{2}$ hours each for meals at ordinary atmospheric pressure, cases of caisson disease usually occurred after the last decompression in the evening and not when the men came out for meals[1].

Our own observations on animals afford fresh evidence bearing in the same direction. We found that in goats the risks on decompression increase with the length of exposure to pressure up to from two to three hours (see below, p. 396).

In different warm-blooded animals the rate of respiratory exchange varies, roughly speaking, according to the ratio of body surface to weight. The smaller the animals, therefore, the greater is the respiratory exchange per unit of body weight, and the more rapid must be the

[1] G. W. M. Boycott, *Trans. Inst. of Civil Engineers*, vol. CLXV., 1906.

circulation. In consequence small animals, when placed in compressed air, must saturate their tissues more rapidly in proportion to their more active respiratory exchange; and, conversely, they will free themselves more rapidly, during or after decompression, from the excess of nitrogen. Hence results obtained with small animals as to the time required for complete saturation, or for safe decompression, are not directly applicable to man. We selected goats for our experiments as they were the largest animals which could be conveniently used; but their weights averaged only about one-fourth to one-third of the weight of an adult man. As the surfaces of different mammals are roughly as the cube roots squared of their weights, we should expect that in goats of this size the respiratory exchange per kilo of body weight would be about two-thirds greater than in man. Direct determinations showed that this was the case (see p. 381). Hence if it required three hours exposure to a high pressure to effect practically complete saturation[1] of the more slowly saturating tissues of a goat with nitrogen, about five hours would be required for a man. An inspection of Fig. 1 (p. 347) will show that if these tissues became 50 % saturated in about 45 minutes in goats and 75 minutes in man, they would be 94 % saturated in three hours for goats, and in five hours for man. A higher degree of saturation than this would scarcely be appreciable, and we have concluded that for practical purposes any slower rate of saturation than this, and correspondingly slower rate of desaturation, need not be allowed for, unless the percentage of fat in the body is abnormally high. We must admit, however, that there is some evidence, both from our own experiments and from practical experience in work in compressed air, that in the parts of the body which are the seat of "bends" a still slower rate of saturation may exist.

B. *The rate of desaturation of the body with nitrogen during and after decompression.*

If the pressure is rapidly diminished to normal after exposure to saturation in compressed air, and no gas bubbles are liberated in the body, it is evident that for each part of the body the curve of desaturation will be similar to that of saturation, provided the physiological conditions are constant. The venous blood will give off practically the

[1] The only method apparently available to determine the time of complete saturation in normal animals is to subject them to a series of experiments in which the pressure and decompression are kept constant and the time of exposure varied, and to observe when the effects cease to become any worse. The method is open to obvious limitations.

whole of its excess of dissolved nitrogen during its passage through the lungs[1], and at each round of the circulation will bring back a fresh charge of nitrogen (at the partial pressure existing in the tissues) to be given off. The parts which become half desaturated by this process in a given time will be three-fourths desaturated in double the time, and so on. The slowest saturating tissues will thus, in accordance with our previous calculation, take one and a quarter hours to become half desaturated in man.

The normal combined gas pressure of nitrogen, oxygen and CO_2 in the tissues and venous blood may be estimated as about 90 % of an atmosphere, so that if the nitrogen pressure be more than an eighth above normal the total gas pressure will be above atmospheric pressure. Supposing therefore that before decompression the most slowly saturating parts of the body (*i.e.* those half saturating in one and a quarter hours) had been saturated to an excess pressure of two atmospheres of air, it would take about five hours at atmospheric pressure to reduce this excess pressure to a sixteenth (or an eighth of one atmosphere) and so bring down the total gas pressure in the parts in question to about atmospheric pressure. The slowness of desaturation must be as clearly borne in mind as the slowness of saturation, in connection with all the phenomena of compressed-air illness.

If gas bubbles are formed in consequence of too rapid decompression, they will naturally tend to increase in size by diffusion into them, in whatever part of the body they may be except the arteries, for some time after the end of decompression. They may thus easily cause blocking of small vessels, and even if they are carried to the right side of the heart or the pulmonary arteries, and lodge there, they will increase in bulk until the total gas pressure in the mixed venous blood falls to one atmosphere. The same remark applies to bubbles which

[1] In view of the enormous surface (probably more than 100 square metres) presented by the lung alveoli for diffusion it seems hardly possible to doubt that the blood during its passage through the lungs becomes saturated or desaturated to almost exactly the pressure of nitrogen in the alveolar air. According to the calculations of Loewy and Zuntz (*Die physiologischen Grundlagen der Sauerstoff-Therapie* in Michaelis' *Die Sauerstofftherapie*, Berlin, 1904), a difference in partial pressure of oxygen of less than 1 mm. of mercury would account for the diffusion of 250 c.c. of oxygen per minute through the alveolar walls. With a difference in partial pressure of nitrogen of two atmospheres, or 1520 mm. of mercury, between the blood and the alveolar air only about 70 c.c. of nitrogen would require to pass per minute in order to establish complete saturation, or desaturation, of the blood. The conditions are thus enormously more favourable for the taking up or giving off of this nitrogen than for the taking up of oxygen by diffusion during normal respiration.

lodge in the branches of the portal veins. If small bubbles are carried through the lung capillaries and pass, for instance, to a slowly desaturating part of the spinal cord, they will there increase in size and may produce serious blockage of the circulation or direct mechanical damage. Apart from this increase of size the air bubbles passing along the arteries are probably too small to cause any harm. Once formed they will under ordinary conditions take a long time to become reabsorbed, since even after the gas pressure in the blood and tissues has fallen to normal, the excess of nitrogen pressure in the bubbles over that in the blood and tissues will only be about a tenth of an atmosphere at most. In one case we found bubbles in the veins of an animal which died two days after suffering from severe decompression symptoms (see below p. 421).

In order to avoid the risk of bubbles being formed on decompression, it has hitherto been recommended that decompression should be slow and at as nearly a uniform rate throughout as possible. We must therefore carefully consider the process of desaturation of the body during slow and uniform decompression. For convenience in calculation we may imagine the process as occurring in a series of time-intervals, the first half of each of which is spent at the pressure existing at the beginning of the interval, and the second half at the pressure existing at the end. Let us suppose, for instance, that the body has been completely saturated with nitrogen at an excess pressure of five atmospheres of air, and that decompression occurs at a rate of one atmosphere in 20 minutes. The process may be divided into five periods of 20 minutes, during each of which the pressure falls one atmosphere. We can then easily calculate how far desaturation will have gone at the end of each period, and from these data construct a desaturation curve.

Let us first consider the mean desaturation rate of the whole body, assuming that, when the pressure is suddenly raised or diminished to a certain level, the tissues will on an average saturate or desaturate themselves by 50 % in 23 minutes, which was shown above to be a probable average rate. A reference to the curve (Fig. 1) shows that ten minutes' exposure to the reduced pressure of four atmospheres in excess will reduce the saturation by 28 % of the difference between five and four atmospheres, *i.e.* by 0·28 of an atmosphere. Hence at the end of 20 minutes the tissues will on an average be saturated to 4·72 atmospheres. Ten more minutes at four atmospheres will reduce the saturation to 4·5 atmospheres, and ten minutes at three atmospheres

will further reduce it by 28% of 4·5 − 3, *i.e.* by 0·42 atmosphere. Hence at the end of the second twenty minutes the saturation of the tissues will be 4·08 atmospheres. Continuing this calculation we get the desaturation curve shown in Fig. 2, from which it will be seen that when atmospheric pressure is reached the tissues are still saturated to an excess pressure corresponding to 1·4 atmospheres of air.

Fig. 2 also shows a similar curve for the parts which saturate and desaturate most slowly, and which, according to our previous calculations, take one and a quarter hours to become half saturated. At the end of decompression these slowly desaturating parts, as shown on the curve, are still saturated to 3·15 atmospheres. This of course represents a most formidable excess; and, as will be shown below (p. 401), uniform decompression at this rate is dangerous even to goats, and would certainly be extremely dangerous to men, who desaturate a good deal more slowly than goats.

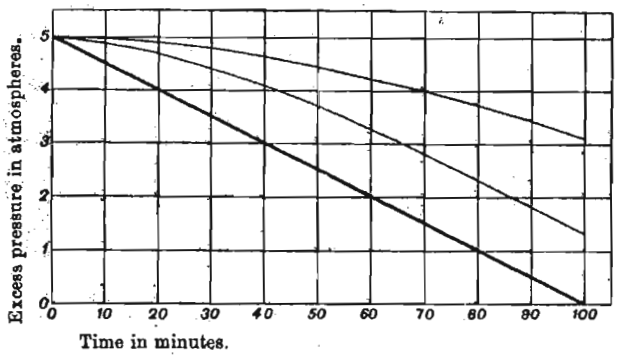


Fig. 2. Desaturation during uniform decompression after complete saturation at 5 atmospheres excess pressure. The thick line represents the air pressure: the upper and lower thin lines represent respectively the progress of desaturation in parts of the body which half saturate in 75 and 23 minutes.

Inspection of Fig. 2 shows that with uniform decompression the nitrogen pressure in the body lags behind that of the air, and that (in the case of the slowly desaturating parts) the amount of the lag increases during the whole time of a decompression lasting 100 minutes. No other result seems possible, and actual experiments point strongly in the same direction, as will be shown presently. We must emphatically dissent from the conclusion drawn by Heller, Mager and v. Schrötter that decompression at the uniform rate of 20 minutes an atmosphere prevents any dangerous retention of gas in the body. To

prevent a maximum lag of more than one atmosphere, it would be necessary to decompress at a rate of over one and a half hours an atmosphere if the decompression were uniform and from an excess pressure of five atmospheres[1].

The examples given will be sufficient to illustrate the extreme slowness with which desaturation must occur with a uniform rate of decompression. This slowness has never hitherto been recognised, but must evidently be reckoned with in devising measures for the prevention of caisson disease.

It is clear that the rate of desaturation might be hastened by either (1) increasing the difference in nitrogen pressure between the venous blood and the air in the lungs, or (2) increasing the rate of blood circulation. In either case the blood would give off through the lungs an increased amount of the excess of nitrogen in a given time.

In order to increase the difference in nitrogen pressure between the venous blood and the alveolar air it has been proposed to give a diver oxygen to breathe during, or before decompression. As long, however, as the pressure was above about one atmosphere in excess, or 15 lbs., it would be impossible to do this safely, since, as will be explained more fully below, the effects might be rapidly fatal owing to oxygen poisoning. The possible applications of oxygen are thus somewhat limited, while the complications involved would be very considerable. The same end can, however, be attained in another way, as will be shown in the following section.

The rate of blood circulation can be increased considerably by muscular exertion. Quite moderate exertion is sufficient to increase the respiratory exchange to three or four times the normal; and the rate of blood flow through the lungs must be increased to something approaching to a corresponding extent. Unfortunately, the increased blood flow is chiefly through the muscles which are working, but probably many parts of the body participate to a greater or less extent in the extra blood supply. Muscular work must correspondingly increase the rate of saturation of the body with nitrogen. For this reason it seems desirable that where work has been done in compressed air, so that the muscles and associated tissues have probably become rapidly saturated with nitrogen, there should also be muscular exertion

[1] It is evidently a mistake to assume that a given rate of uniform decompression, such as 20 minutes per atmosphere, is either necessary for safety in all cases, or would be actually safe except from some limit of pressure. From a pressure below this limit the rate will be unnecessarily slow, and from above it dangerously fast.

during decompression. The rate of desaturation will thus be increased so as to compensate for the increased rate of saturation. In the case of short exposures to compressed air, as in diving work, this is specially important. Even, however, when there has been no special muscular work in the compressed air movements of joints and massage of the skin etc. will probably hasten desaturation. This has been clearly pointed out by Hill and Greenwood[1].

Another method which can be employed for increasing the circulation in the case of divers is to restrict the air supply, so that the partial pressure of CO_2 in the air of the helmet may rise sufficiently to stimulate the respiration and circulation. Both methods are now used in the Royal Navy during the ascent of divers.

C. *The limits of safety in decompression.*

It is a fact well known to those practically acquainted with work in compressed air that even with very rapid decompression there is no risk of caisson disease unless the pressure has exceeded a certain amount. It seems perfectly clear that no symptoms occur with less than one atmosphere[2] of excess pressure, however long the exposure may be. Whether any distinct symptoms ever occur with less than about 1·25 atmospheres (18½ lbs. per square inch or 41 feet of sea water) seems very doubtful: at any rate they are very exceptional. At pressures a little above 1·25 atmospheres occasional slight cases begin to be observed, and their frequency and gravity rapidly increase with higher pressures unless the time of exposure is limited or slow decompression is resorted to. The lowest pressure at which we have been able to find any record of a death occurring from caisson disease is 23 lbs. or 1·6 atmospheres[3]. As will be seen below, we were able to obtain slight symptoms on rapid decompression in 1 out of 22 goats after long

[1] *Proc. Roy. Soc.* B, vol. LXXVII., p. 449, 1906.

[2] One atmosphere or 760 mm. of mercury = 14·7 lbs. per square inch, about 1 kilogram per square centimetre, 34 feet of fresh water, 33 feet of sea water. In this paper where pressures are defined in pounds or atmospheres without qualification, reference is intended to the excess over atmospheric pressure as shown on gauges, not to the absolute total pressure.

[3] Babington and Cuthbert, *Dublin Quarterly Journal of Medical Sciences*, vol. XXXVI., 1863, p. 312. In the list of fatal cases given by Heller, Mager and v. Schrötter (*Luftdruckerkrankungen*, p. 1072), are entered two deaths at a pressure of 1·4 atmospheres. A perusal of Paul Bert's original account (*La Pression Barometrique*, p. 401) shows that both the pressure and the cause of death are quite uncertain.

exposure (four hours) to 1·36 atmospheres or 20 lbs. With 25 lbs. (1·7 atmospheres) two cases of slight illness occurred out of 23 animals.

If the risks of rapid decompression depended simply on the extent to which the blood and tissues are supersaturated with nitrogen on decompression, we should expect to find that even a short exposure to such an excess pressure as two atmospheres would be risky with rapid decompression: for there can be no doubt that within, say, half an hour or forty minutes the tissues, and the blood returning from them, must be for all practical purposes fully saturated in many parts of the body, and particularly in parts of great physiological importance which are richly supplied with blood. Nevertheless it seems to be well established that a man may stay without serious risk for forty minutes at a pressure which would involve great danger on rapid decompression if he remained in it for several hours.

Parts of the body with a rapid circulation will become very completely saturated in a comparatively short time, but the highly supersaturated blood which first returns from them on rapid decompression can remain but a very short time supersaturated during each round of the circulation, and on reaching the large veins will mix with less highly saturated blood from other parts of the body. It would seem that the state of high supersaturation in any portion of blood lasts for too short a time to enable bubbles to form.

If this interpretation of the facts is correct, we should expect to find with small animals, which rapidly saturate and desaturate, that a higher pressure would be required to produce symptoms on rapid decompression after a long exposure than in the case of larger animals. The general experience of previous observers is in accord with this, and our own experiments (see below p. 402) showed that we could produce no obvious effects in mice, and very few in rabbits, rats, and guinea-pigs, by sudden decompression after exposures at pressures which were invariably or frequently fatal to goats.

Since supersaturation to the extent of about 1·25 atmospheres above normal atmospheric pressure can be borne with impunity, though a greater degree of supersaturation is risky, it seems clear that, in decompressing after prolonged exposure to high pressures, the rate of decompression should be sufficiently slow to prevent any greater excess of saturation than this in any part of the body at the end of decompression. On the other hand decompression should evidently be as rapid as is possible, consistently with safety. A pressure of 1 to 1·25 atmospheres above normal corresponds to from 2 to 2·25 times the

normal atmospheric pressure; but the *volume* (not the *mass*) of gas (measured at the existing pressure) which would be liberated if the whole excess of gas present in supersaturation were given off is the same whether the absolute pressure is reduced from two to one atmospheres, or from four to two, or from eight to four. Hence it seemed probable that, if it is safe to decompress suddenly from two atmospheres of absolute pressure to one, it would be equally safe to decompress from four atmospheres absolute to two, from six atmospheres absolute to three, etc. Our experiments, which are detailed below (p. 398), have shown that this is the case[1]. The process of desaturation can therefore be hastened very greatly by rapidly reducing the absolute pressure to half, and so arranging the rest of the decompression that the saturation in no part of the body shall ever be allowed to correspond to more than about double the air pressure. The main advantage of this plan is that the discharge of nitrogen from the tissues is from the outset of decompression increased to the greatest rate which is safe. The rate of discharge evidently depends on the difference in partial pressure of nitrogen between the venous blood and the alveolar air; and by keeping this difference at the maximum consistent with safety a great saving of time is effected. Detailed investigations have completely justified the adoption of this principle: they are described below, and comprise, besides a series of observations on animals, a number of experiments in which Lieut. Damant and Mr Catto were exposed to excess pressures up to 80 pounds, or 6·4 atmospheres of absolute pressure, in the experimental chamber and to 93½ pounds, or 7·4 atmospheres, in actual diving. The method greatly simplifies the problem of safe decompression, and gets rid of many practical difficulties, particularly in connection with deep diving. It may be conveniently referred to as the method of "stage decompression," and is so described in the sequel, though its essential peculiarity does not lie in the decompression being done in stages but in its being rapid till the absolute pressure is halved and slow afterwards.

[1] Whether the law holds good for pressures much exceeding six atmospheres is still doubtful, as no experimental data exist.

D. *Practical measures for avoiding Compressed-air Illness.*

From the foregoing discussion the general nature of the measures needed to prevent compressed-air illness will be evident enough. The risks may best be avoided by properly calculated stage decompression, or by cutting down the period of exposure to a safe limit, or by both methods combined. In the case of work in compressed air in caissons, tunnels, etc., it is for economic reasons very undesirable to greatly reduce the period of exposure. In diving work, on the other hand, the periods of exposure are generally short in any case, and they can, without great inconvenience, be confined within limits which largely reduce the risks of compressed-air illness. Long periods of decompression are also very undesirable in diving, since changes of weather or tide or other causes may render a return to the surface necessary without any long delay in coming up, and since very prolonged stays under water are exhausting, and the diver's hands may become benumbed by cold.

As our investigations were in the first instance made with the object of securing safety from compressed-air illness in diving work, we may first consider the precautions desirable in connection with diving.

(1) *Diving work.*

The ordinary diving dress (Plate IV) consists of a copper helmet screwed to a corselet, the latter being in its turn connected water-tight to a stout water-proof dress covering every part of the body except the hands, which project through elastic cuffs. Air is supplied through a non-return valve on the helmet from a flexible pipe connected with an air-pump on a boat or ship. The air escapes through an adjustable spring valve at the side of the helmet. The arrangement is thus such that the pressure of the helmet air breathed by the diver is always at least equal to, and usually slightly greater than, the pressure of the water at the valve outlet. At a depth of 33 feet or 10 metres the diver is therefore breathing air at an excess pressure of one atmosphere, or at an absolute pressure of two atmospheres; and every additional 33 feet will add another atmosphere to the pressure. To enable the diver to sink, the dress and boots are suitably weighted. He is usually in connection with surface by a life-line containing a telephone wire, as well as by the air-pipe.

In descending or ascending a diver usually makes use of a rope attached to a heavy sinker at the bottom. He can thus easily regulate the rate of his ascent or descent, and take care that this rate is not so rapid as to cause any discomfort or pain in the ears owing to incomplete opening of the Eustachian tubes. A too rapid descent or ascent might cause mechanical injury followed by middle ear inflammation.

As explained above, there appears to be practically no risk of symptoms occurring from liberation of gas bubbles on rapid decompression if the pressure has not exceeded 1·25 atmospheres, corresponding to a depth of about seven fathoms or 42 feet of sea water. Up to this depth therefore no special precautions against caisson disease need be taken[1]. At greater depths precautions depending on the duration of exposure are evidently needed. The precautions which we have calculated to be desirable are embodied in the table given below (Appendix IV.); and the principles and experimental results on which this table is based must now be discussed.

It will be convenient to consider first the case of diving to a very great depth, and we shall take as an extreme example the case of exposure at a depth of 35½ fathoms (213 feet) of sea water, corresponding to an excess pressure of nearly 6·5 atmospheres, or an absolute pressure of 7·5 atmospheres.

Let us first suppose that the body of a diver is completely saturated with the nitrogen of air at this pressure, and that it is required to conduct his ascent to surface as rapidly as possible but without any risk of symptoms due to bubble formation, *i.e.* in such a way that, in accordance with the principles already laid down, the nitrogen pressure in no part of the body shall ever be more than double that of the air breathed at the same time.

The first step would obviously be to reduce the absolute pressure to about half, *i.e.* from 7·5 atmospheres absolute to 3·75 or from 6·5 atmospheres in excess to 2·75. This would be *ex hypothesi* the greatest initial drop in pressure which would be perfectly safe. The remainder of the decompression would evidently need to be conducted in such a way that the maximum partial pressure of nitrogen in any part of the body should diminish at double the rate of the fall in absolute pressure of the air. The ascent of a diver can be conveniently regulated from

[1] Heller, Mager and v. Schrötter recommend that at all depths decompression should be at a rate of at least 20 minutes per atmosphere. This would imply a delay of 25 minutes in coming up from 42 feet. Both common practical experience and our own experiments show that this excess of caution is quite unnecessary at small depths.

the surface by signalling to him to stop or come on at every ten feet as indicated on the pressure gauge attached to the pump. We may therefore divide the ascent into stages of ten feet, and the short periods occupied in the actual ascents may be neglected.

Since the depth was 213 feet, corresponding to 246 feet of water in absolute pressure, it would be safe to come up at once to a depth corresponding to 123 feet of absolute water pressure, *i.e.* to 90 feet of actual depth. Consequently the first stage would be a rapid ascent of 123 feet, and it would be necessary to wait here before the next ascent of 10 feet until the maximum partial pressure of nitrogen in the body had fallen to that of the nitrogen in air at $2 \times (80 + 33) = 226$ feet of absolute water pressure. The difference between 246 and 226 is 20, and this is 16 % of $213 - 90 = 123$, the difference between the original and the reduced pressure. The most slowly desaturating parts of the body will, according to our previous calculations, take 75 minutes to give off half of any excess of nitrogen which they may contain at any given air pressure; by inspection of the curve (Fig. 1) it will be seen that they will take about 19 minutes to lose 16 % of the excess. Hence a delay of 19 minutes would be necessary at 90 feet before coming up to 80 feet. At 80 feet the partial pressure in the body would require to fall an amount corresponding to 20 feet, which is about $17\frac{1}{2}$ % of $193 - 80 = 113$, the new difference in relative pressure between the nitrogen in the body and in the air. This would necessitate a delay of 21 minutes before ascending to 70 feet. The further delays needed would be 23 minutes at 70 feet, 26 minutes at 60 feet, 30 minutes at 50 feet, 35 minutes at 40 feet, 42 minutes at 30 feet, 51 minutes at 20 feet, and 62 minutes at 10 feet. It would thus take 309 minutes, or more than five hours, to reach surface.

This calculation is represented graphically in Fig. 3. It will be noticed from the figure that the time required for safe decompression does not increase proportionally to the increase in depth. For instance, an increase in depth of 15 feet from 50 to 63 feet necessitates an increase of 45 minutes in the time required for safe decompression; but the same increase in depth from 198 to 213 feet only requires an increase of 15 minutes in the time of decompression.

A somewhat more rapid rate of stage decompression could probably be adopted without appreciable risk to life, but the occurrence under water of even one of the less serious decompression symptoms might be extremely unpleasant or indirectly dangerous, so that a factor which we believe to be thoroughly safe in this respect has been used in the

calculation. The possible occurrence of slight symptoms after surface had been reached would not, however, be a serious matter: for this reason half of the last stop at 10 feet from surface might be dispensed with, which would save half an hour. The most slowly desaturating tissues would, according to the calculation, still be only saturated to an excess pressure of 1·3 atmospheres—a safe enough limit perhaps, but leaving no great margin to spare.

Fig. 3 also shows the maximum excess of saturation with uniform decompression in the same time and in 10 hours. It will be seen that uniform decompression in about five hours would leave at the end of decompression an excess saturation within the body of 2·1 atmospheres; and even if uniform decompression were extended to ten hours the excess saturation would still exceed one atmosphere. It is also perfectly clear that uniform decompression is an unsuitable way of bringing a man out of compressed air. Where a sufficiently safe rate of uniform decompression is employed (as, for instance, with 10 hours in the case under consideration), it is only at the very end (when the nitrogen pressure inside the body becomes more than double that of the air) that there is any risk of symptoms occurring; and for the sake of safety at the end the whole process is made quite unnecessarily long. Increased safety at the end is only secured in combination with useless delay at the beginning[1].

As will be seen in Part II, the results of our experiments, allowance being made for the difference between goats and men, fully confirm the foregoing mode of calculation. Not only has stage decompression in the calculated time proved safe where uniform decompression in the same total time was unsafe, but shorter periods of stage decompression than those calculated have been proved to involve risk of symptoms, increasing in gravity and frequency with the shortening of the time, though always less than the risk from uniform decompression in the same time.

If the whole body of a diver were allowed to become saturated at any great depth, it is evident that the time needed for safe decompression would be impracticably long. To reduce the time of de-

[1] The regulations of the Dutch Government make the following method of decompression obligatory for work in caissons, &c. The pressure is to be lowered at the rate of not more than $\frac{1}{10}$th of an atmosphere in 3 minutes till 3 atmospheres of excess pressure is reached: then at not more than $\frac{1}{10}$th of an atmosphere in 2 minutes till $1\frac{1}{2}$ atmospheres excess pressure is reached; and finally at not more than $\frac{1}{10}$th of an atmosphere in $1\frac{1}{2}$ minutes till normal pressure is reached. This method is still more unsuitable than uniform decompression, and would be very unsafe with high pressures.

compression to within limits practicable for divers, it is evidently necessary to greatly reduce the period of exposure to high pressure[1]. At great depths limitation of the exposure is also necessary in order to avoid toxic effects from the high pressure of oxygen (see p. 371). Calculation of the mode and period of decompression required after a limited exposure to pressure is a somewhat complicated matter, but the principles already laid down render it quite possible.

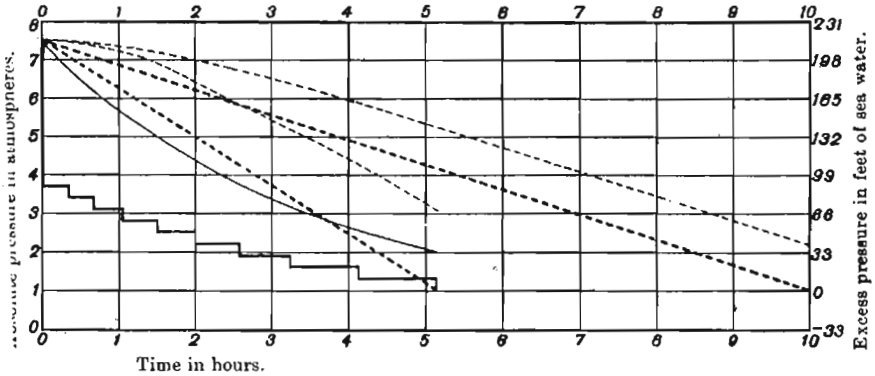


Fig. 3. Theoretical ascents of a diver after a prolonged stay at 213 feet of sea water. Stage decompression in 309 minutes compared with uniform decompressions in 309 minutes and in 10 hours. Continuous lines = stage decompression: interrupted lines = uniform decompression. Thick lines = air pressure: thin lines = saturation with atmospheric nitrogen in parts of the body which half saturate in 75 minutes.

When a diver goes down for a very short time, we have to take into consideration not only the time which he spends at the maximum pressure on the bottom but also the time occupied in the descent and the ascent. During the descent he is all the time saturating himself with nitrogen, and during most of the ascent he may be doing so also. Calculation will show that, if he descends and ascends at a uniform rate, the time spent in this process will be nearly equivalent, as regards the saturation of the body with nitrogen, to half the same time spent at the maximum depth. It is therefore clear that in deep diving the diver should descend as rapidly as is practicable, and should also ascend at once, on completion of his work, as far as he safely can. The rate of descent may be limited either by pain in the ears or by an air supply insufficient to keep the upper part of the dress full of air.

[1] This was fully realised by Catsaras who recommended a stay on the bottom of only 1 minute at 30 fathoms.

Both these causes are avoidable, and an experienced diver, with his Eustachian tubes well opened and a proper supply of air, can get to an excess pressure of six atmospheres (198 feet) in two minutes. This time was found sufficient in experimental dives up to 210 feet made by Lieut. Damant and Mr Catto (Appendix II). The recommendation commonly made that the rate of both ascent and descent should be slow is evidently quite unsound. A man who spent half an hour in descending to 30 fathoms, and an equal time in ascending at a uniform rate, would run a considerable risk of perishing on his return to the surface.

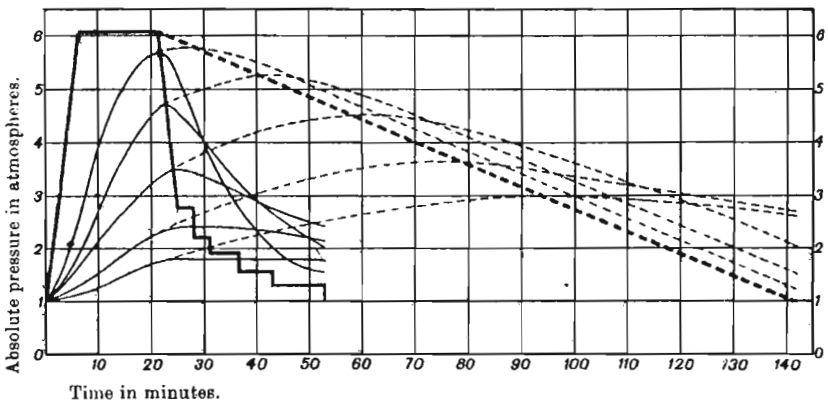


Fig. 4. Desaturation during stage decompression in 32 minutes and uniform decompression in 2 hours, after exposure for 15 minutes at 75 lbs. pressure with compression in 6 minutes. Thick lines = air pressure: continuous lines = stage decompression: dotted lines = uniform decompression. The curves from above downwards represent respectively the variations in saturation with nitrogen of parts of the body which half saturate in 5, 10, 20, 40, and 75 minutes.

In order to illustrate the method by which we have calculated safe modes of ascent in the minimum period of time we may take as an example the case of exposure for 15 minutes to a pressure of 75 pounds (6·1 atmospheres absolute or 28 fathoms = 168 feet). Many of our experiments on goats were made with this pressure and exposure. It took about six minutes to raise the pressure in the experimental chamber to 75 pounds, so that the total virtual exposure till decompression began was about 18 minutes. Fig. 4 shows graphically the variations of pressure during this period: also the calculated partial pressure of nitrogen in different parts of the body, as compared with the nitrogen pressure in the air. The first stage was from 6·1 to 2·8

atmospheres absolute (corresponding to an ascent in sea water from 168 to 60 feet) and occupied four minutes. The subsequent stoppages were:—

2	minutes	at 2·8	atmospheres	(60	feet of sea	water),
3	„	2·2	„	(40	„	),
5	„	1·9	„	(30	„	),
7	„	1·6	„	(20	„	),
10	„	1·3	„	(10	„	).

It will be seen from the figure that this rate of decompression was slightly faster than what was calculated above to be desirable. At the end of decompression the nitrogen pressure in those parts of the body which became half saturated in about 20 minutes under pressure would be equivalent to that of air at about 1·4 atmospheres, or 20·6 pounds per square inch. If the circulation in one of these parts were less vigorous during decompression than during exposure to the high pressure, it might well be that the nitrogen pressure in this part at the end of decompression would be higher than corresponded to the calculation. As a matter of fact minor symptoms ("bends") were observed five times in 34 decompressions of 18 goats, although no serious effects occurred. We concluded that the period of virtual exposure (18 minutes) was slightly longer than is desirable with stage decompression in 31 minutes: in the table below (p. 442) the limit has been set down at 15 minutes.

Fig. 5 shows the calculated nitrogen pressure in different parts of the body during uniform decompression in 31 minutes after the same exposure at 75 pounds. It will be noticed that at the end of decompression there is a dangerous excess of saturation in all parts of the body except those which half saturate in less than about seven or eight minutes, and that this supersaturation corresponds to an excess pressure of as much as 2·1 atmospheres of air. The goats used for the stage decompression experiments were on alternate occasions subjected to uniform decompression in the same time and with the same exposure. The result was that, in 36 decompressions, one died, two were paralysed, one had indefinite general symptoms of a severe character, and in 11 other cases "bends" occurred, besides two doubtful cases. This was entirely in accord with what the calculation would lead us to expect; and uniform decompression in 31 minutes is evidently dangerous under the conditions given.

It might be supposed that safety would be secured by extending to

a moderate degree the length of uniform decompression. It must be remembered however that the more the duration of uniform decompression is extended, the longer is the period during which the body is exposed to high pressure. Fig. 4 shows the calculated effects of uniform decompression extended to two hours. Although the quickly saturating parts of the body are desaturating during the greater part of the decompression, the slowly saturating parts are, on the other hand, becoming more and more saturated, so that at the end of decompression the parts which half saturate in from 40 to 75 minutes are saturated to an excess pressure of about 1·7 atmospheres, although at the beginning of decompression they were only saturated to from 0·7 to 1·3 atmospheres and could consequently have given no trouble.

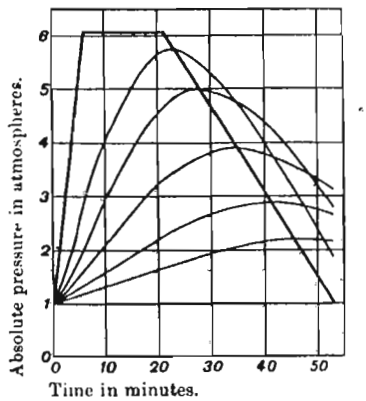


Fig. 5. Desaturation during uniform decompression in 32 minutes after exposure for 15 minutes at 75 lbs. pressure with compression in 6 minutes. Thick line = air pressure. The curves from above downwards represent respectively the variations in saturation with nitrogen of parts of the body which half saturate in 5, 10, 20, 40 and 75 minutes.

Very prolonged uniform decompressions are extremely tedious, and it seemed scarcely worth while to make any extensive series of such experiments. We found however that out of 12 goats uniformly decompressed in 90 minutes after 18 minutes virtual exposure at 75 pounds (6·1 atmospheres of absolute pressure) three developed symptoms of bends after decompression. The proportion of illnesses was thus greater than with stage decompression in a third of the time. With men the results would certainly be much worse, and we calculate that for a man, after the same exposure, several hours would be needed for uniform decompression in order to escape all risk of

symptoms occurring. The time would in fact require to be nearly as long as if the body had been completely saturated at the maximum pressure.

With very short exposures to high pressure, rapid decompression is probably safer than uniform decompression at a moderate rate. There is a considerable human experience on this point: divers working at great depths would seem to consider it fairly safe to go rapidly to the bottom at a depth of 160 or 180 feet and return equally rapidly, provided the time spent on the bottom does not exceed six or eight minutes and provided also that the dives are not repeated at short intervals. It is reported of the skilled Greek divers of the Mediterranean that, in case their gear becomes entangled on the bottom, they will cut their air-pipe and line and blow themselves up to the surface in less than a minute from a depth of 30 fathoms or the like rather than stop more than about ten minutes on the bottom. Our experiments on goats are in accordance with this practice. We found that no symptoms were produced by sudden decompression in less than a minute after virtual exposures at 75 pounds up to four minutes, and even in some trials up to six minutes (see below, p. 394).

With exposures exceeding a very few minutes, or such brief exposures frequently repeated, so that during the intervals the body has not time to become desaturated, we have little doubt that slow and uniform decompression—the slower the better—is at any rate preferable to sudden decompression. Uniform decompression must however be extremely slow to make it entirely free from risk of death or very serious symptoms, and the time required is so great that this method seems to us quite impracticable in connection with diving work. There appears to be very little human experience of slow uniform decompression. Divers usually come up in a few minutes at most, and even half an hour spent in the ascent would appear to be quite exceptional. Almost the only definite observations are those of Hill and Greenwood, who recently experimented on themselves at very high pressures. Fig. 6 shows the variations of pressure and the calculated saturations of different parts of the body during the experiment in which Greenwood went to a pressure of 91 pounds (7·1 atmospheres absolute). This experiment appears to have been a very risky one. After decompression he had bends in both arms, and Hill also had symptoms pointing towards blockage of vessels in the subcutaneous fat after similar experiences at 75 pounds pressure.

In Appendix IV two tables are given for the safe decompression of

divers after exposure for varying periods of time at different depths. These tables are the same as are now in use for divers in the Royal Navy, on the recommendation of the Committee on Deep Diving. In Table I the period of virtual exposure is so limited that the diver can return to surface by stages in half an hour or less. It will be noted that the maximum periods of exposure are from the time of leaving surface, so that there should be no chance of increased danger from undue delay in descending. The stoppages during the ascent are so calculated that, until surface is nearly reached, the excess of nitrogen pressure in any part of the body should never be more than double the nitrogen pressure of the air breathed, and not more

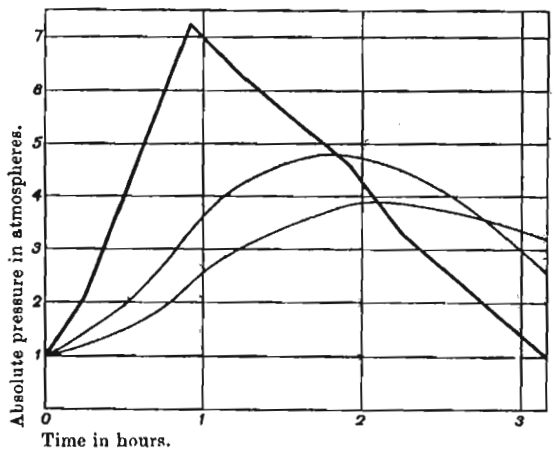


Fig. 6. Showing calculated variations in saturation with nitrogen during Dr Greenwood's experiment on himself. Thick line = air pressure: the two curves from above downwards represent respectively the variations in saturation with nitrogen of parts of the body which half-saturate in 40 and 75 minutes.

than two and a quarter times this pressure when surface is reached. The only case in which these limits are allowed to be slightly exceeded is with short exposures in comparatively shallow water. This slight excess is, however, only in parts of the body which saturate and desaturate very rapidly, and, as already explained, give rise to no danger. As an additional safeguard the diver is directed to keep his arms and legs constantly moving during each stoppage, so as to increase the rate of circulation and guard against the chance of the rate of desaturation during his ascent being proportionally less than the rate of saturation during his stay on the bottom while he was doing work.

The second table provides for the case of exceptionally long stays under water. A diver may be delayed by his air-pipe or life-line being fouled, or by other exceptional circumstances, against which it is necessary to provide. Where the fouling has been complicated by the action of tide the delay on the bottom has occasionally amounted to several hours, until the tide has slackened or turned. If the diver is at a great depth the calculated time required for safe decompression after so prolonged a stay is very long. On the other hand the dangers from cold and exhaustion have to be considered, and the difficulties caused by a strong tide during the diver's ascent. In view of these difficulties the time allowed for decompression after very prolonged exposures is somewhat curtailed, but not so much as to permit of risk of more serious symptoms than "bends," in so far as experiments on animals, and human experience, render it possible to calculate. In the case of men of exceptionally heavy build, and inclined to obesity, the time allowed after very prolonged exposures ought to be increased by about a third, although such men, particularly if over about 45 years of age, ought not to expose themselves to the risk of a prolonged stay in very deep water.

It might appear as if the rate of stage decompression recommended after prolonged exposures was slower than is actually required. A very unfortunate accident which occurred recently has shown only too clearly that this is not the case. In connection with the work of raising a torpedo boat which had sunk in 25 fathoms (150 feet or 46 metres) several divers were employed. They were working in 20 minute spells, and returning to surface by stages in 32 minutes, in accordance with the first table, which was the only one then in use. No symptoms of any kind were observed after the divers' return to surface under these conditions, nor have any symptoms ever been observed hitherto among divers working according to the table. One of the divers, however, became fouled in a very exceptional manner. His life-line was fixed in one direction over a spar or rope belonging to the sunk vessel, and his air-pipe was fixed in the other direction. He was thus prevented from going to free either his air-pipe or life-line. A second diver at once went down, but was unable to free him owing to the drag caused by the tide; and it was only after two and a half hours, when the tide had slackened, that he got free. He was then brought up by stages under the direction of Staff Surgeon Rees and Lieut. Damant. For the decompression two and a half hours were allowed, which we then believed would be a sufficient time in case of a diver being badly fouled

at 25 fathoms. The diver was, however, a man of heavy build with much fat in his body, and aged 49. Owing to his exhausted condition he did not come up on the ordinary rope, but had to be pulled up, hanging motionless on the life-line during the long stoppages. On reaching surface he was very exhausted and could hardly have been safely kept longer in the water. He had no paralysis or other definite symptoms of caisson disease. A bed was arranged for him on deck, and hot bottles &c. applied. After a time he complained of some pain in the legs, but this soon subsided; and as he seemed much better in the morning he was removed to hospital, since there was no suitable accommodation for him on the gun-boat where he was. The moving made him worse, and he gradually became restless and delirious in spite of administration of oxygen at intervals, showed signs of cardiac failure, and died somewhat suddenly about 24 hours after he had been brought up. At the post-mortem examination 12 hours later a moderate number of bubbles were found in the right side of the heart, the veins of the liver and intestines, while scattered bubbles were present in vessels elsewhere, including the coronary vessels, though none were seen in the vessels of the brain. The mesenteric fat, which was very abundant, was in places distended with small bubbles. There was about an inch of subcutaneous fat over the trunk, but no bubbles were seen in this layer. There seemed no reason to doubt that death was largely due to the bubbles, although the more usual symptoms of caisson disease were absent. There were no signs of pneumonia.

This is the only known case of prolonged exposure of a man to such a high excess pressure as four and a half atmospheres; and although his age, heavy build, and exhausted condition combined to make the circumstances very unfavourable, the fact of his death shows that the long decompression periods recommended in the second table after prolonged exposures are none too long, even for a man of ordinary build. Every precaution should be taken to guard against such long exposures at high pressures.

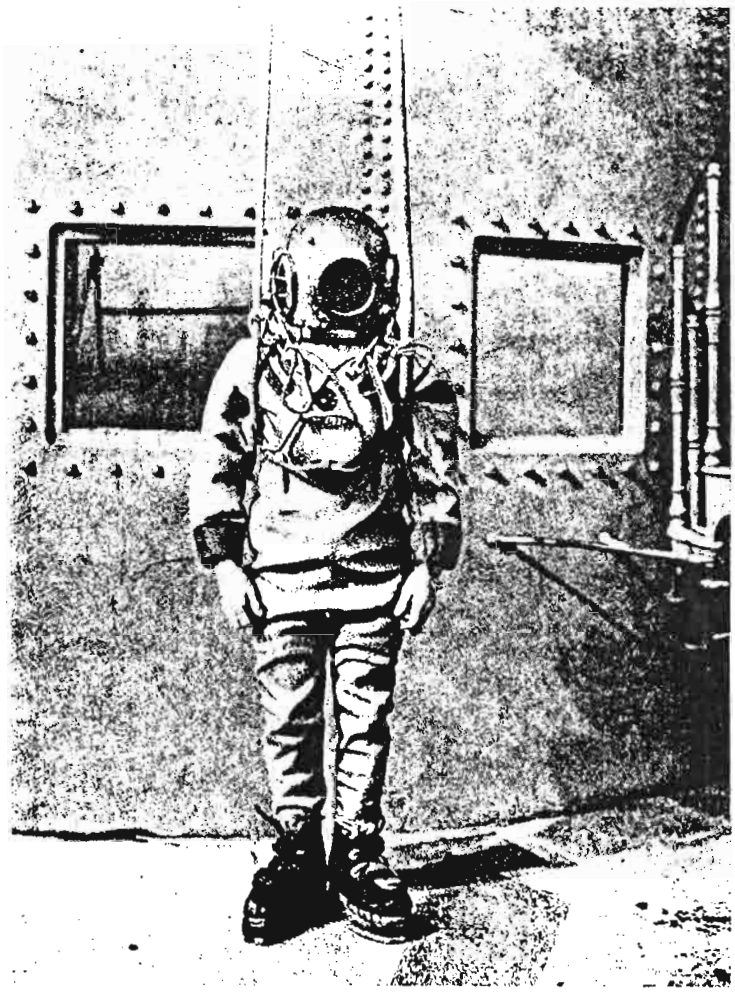
A diver has often to descend twice or oftener at short intervals. At the beginning of the second descent the more slowly desaturating parts of the body will not have had time to lose their excess of nitrogen, and consequently they will be more highly saturated at the end of the second descent than would otherwise have been the case. This will be clear from a study of Figs. 2 or 3. To meet the increased risk in decompression it is desirable, in calculating the proper stoppages, to add together the two periods of exposure, and adopt the corresponding rate

of decompression shown in the tables. For the first half of the stoppages this is not necessary, but for the second half, including the longer stoppages needed to meet the case of the more slowly desaturating parts, the rule should be carried out. The increasing danger after successive short dives by pearl divers, &c. without any precautions in decompression is notorious. This danger does not mount up to the same extent with stage decompression, but nevertheless exists. As the interval between successive dives increases the added danger on decompression diminishes. With an hour's interval the extra precautions might be halved, and with two or three hours' interval they might be omitted.

It may be remarked that the precautions recommended in the tables are greatly in excess of those which have hitherto been commonly employed in either diving work, or work in caissons, tunnels, &c. We have endeavoured to leave a clear margin beyond everything which either human experience or experiments on animals, or calculation, has shown to be risky. In connection with diving, the practice hitherto recommended in the British and other navies has been that the diver should both descend and ascend at a uniform slow rate. By abolishing the slow descent and ascent, and substituting stage decompression, it has been possible to combine greater safety with a clear saving of time under water for a given working period on the bottom. Where the air supply to the diver is managed in accordance with the recommendations of the Diving Committee there is also very greatly increased working efficiency in deep water. For a discussion of the air supply to divers, and many other practical points relating to diving, we must refer to the Committee's Report; and to the "Diving Manual," which has just been re-written and issued to the Royal Navy.

A possible complication to which we have not hitherto referred in connection with compressed-air illness arises from the fact that at very high pressures of air the partial pressure of oxygen begins to be so high as to be capable of producing serious effects. Paul Bert discovered that oxygen at a partial pressure exceeding about three atmospheres (corresponding to 14·3 atmospheres of air) causes animals to go into convulsions and die, even a short exposure being often fatal. More recently, Lorrain Smith, who experimented on mice, and whose results have been confirmed and extended by Hill and Macleod, showed that oxygen at high pressure acts on the lungs, producing pneumonia[1]. He

[1] Lorrain Smith, *Journ. of Physiology*, vol. xxiv., p. 19, 1899; Hill and Macleod, *Journ. of Hygiene*, vol. iii., p. 401, 1903.



Diving dress, front view, with air-pipe and life-line, which are connected with the helmet behind.

found that fatal pneumonia may be produced after four days' exposure to an oxygen pressure of as little as 75 % of an atmosphere, corresponding to air at an absolute pressure of 3·6 atmospheres (88 feet of sea water). At a pressure of about 1·25 atmospheres of oxygen (6 atmospheres of air, or 165 feet of water) death from pneumonia was produced in about 48 hours. At about 1·8 atmospheres of oxygen (eight and a half atmospheres of air, or 250 feet of water), marked symptoms usually occurred in about 12 hours, and death in 20 hours, though in one case death followed in seven hours. At about 2·8 atmospheres of oxygen (13·3 atmospheres of air, or 406 feet of water) marked symptoms were observed in about three hours, and death in nine hours.

The steel chamber at the Lister Institute was not made to withstand such high pressures as would produce within a short time symptoms of oxygen poisoning if air alone was pumped into the chamber. We have, however, made a few observations in the chamber when the oxygen pressure of the air breathed was raised by other means. In one experiment seven goats were placed in the chamber, and the oxygen pressure raised by opening three large cylinders of oxygen, and at the same time pumping in air to 81 pounds pressure. The total oxygen pressure was thus raised to 2·3 atmospheres, corresponding to a depth of 55 fathoms, or 330 feet, or 100 metres. After three hours one animal had died of pneumonia in the chamber, and most of the others seemed more or less affected, though they rapidly recovered on decompression[1]. We also tried on ourselves the effects of breathing nearly pure oxygen from a bag while we were in the chamber at an absolute pressure of two atmospheres; but we could not detect any effects after a few minutes with an oxygen pressure of 1·7 atmospheres, corresponding to about 40 fathoms (240 feet or 73 metres). In a number of goats which were exposed to 75 pounds' pressure (168 feet or 51 metres of water) for three hours, no symptoms indicative of oxygen poisoning were observed.

To judge from these data there is no immediate risk to a diver from oxygen poisoning at depths up to 40, or perhaps 50 fathoms (73 to 90 metres) if ordinary air is breathed, provided the stay is not long. With stage decompression the diver could rapidly return to a perfectly safe oxygen pressure; but, as already remarked, we do not yet know with

[1] One animal showed bends after decompression which was effected in 133 minutes by stages. After exposure at +75 lbs. for 3 hours in air this decompression gave 2 bends in 14 goats. There is therefore no evidence that the exposure to high pressure oxygen increased the susceptibility to caisson disease.

certainty whether it is perfectly safe to rapidly reduce the pressure to half after exposure to such very high air pressures.

(2) *Work in caissons, tunnels, and diving bells.*

In connection with various kinds of engineering work under water, or in soft water-bearing strata, compressed air is commonly used for keeping water out of the working place and preventing collapses. The men have thus to work continuously in compressed air.

In tunnels or 'tubes' through soft water-bearing strata, where a steel lining has to be erected to keep water out and resist pressure, the working face, or blind end of the tunnel under construction, is kept free of water by the air pressure with the help of a circular shield with a cutting edge which is advanced as each section of steel lining is erected into position. The soil is excavated by hand labour, and passed out on trucks through an air-lock.

In constructing foundations for the piers of bridges over rivers, caissons are employed. A caisson is a steel tube, which ultimately forms the lining of the pier, and is shaped accordingly. Near the lower end there is a steel diaphragm, forming a working chamber. An inner steel tube passes through this diaphragm, and serves for ingress and egress, and for passing up the material excavated. At the top of this inner tube there are air-locks for allowing the passage of men and material without escape of the compressed air contained in the working chamber. The latter is kept free from water by the air pressure, and the excess of air escapes beneath the cutting edge of the caisson. When a secure foundation for the pier has been reached this chamber is filled up with concrete. In constructing mine shafts through soft water-bearing strata the same principle may be employed. For work of a simpler kind on river or harbour bottoms diving bells are often used, the bell being simply lowered to the bottom at any required place, so that the men can work on the area covered by it and are kept dry by the air pressure.

The circumstances connected with work in compressed air in caissons, tunnels, &c., differ in certain respects from those associated with diving work.

In the first place the duration of exposure is far longer. A caisson or tunnel worker is usually in compressed air for six or eight hours daily, or even longer. The conditions of the work render any great limitation of the periods of exposure very difficult and expensive.

Usually, however, the workman comes out for meals at intervals of about three hours.

A second difference is that the very high pressures to which a diver may have to go are not needed in caisson or tunnel work. An excess pressure of about 3¼ atmospheres, or 48 lbs., is, we believe, the extreme limit hitherto employed; and usually the excess pressure does not exceed about two atmospheres or 30 lbs. Decompression seems to be usually effected in 10 to 20 minutes, or even, with the lower pressures, in three to five minutes.

With properly arranged air-locks for men and material there should be no need for hurry in coming out; and undue hurry is specially undesirable if the workman leaves the works at once, since he would be liable to develop symptoms when he was so far away that he could not be readily recompressed. To obviate this risk as far as possible, it is customary to endeavour to keep men for half to one hour on the works after they come out; and with the usual rates of uniform decompression this precaution is very necessary. Evidently, however, it is greatly preferable to prevent all practical risks of serious symptoms.

In order to attain this end stage decompression as recommended for divers in the tables in Appendix IV may be employed. An accurate and easily read pressure gauge, visible from both inside and outside the air-lock, is of course essential; and a reliable man should be in charge of the tap. As a further control it would be desirable to have an automatic graphic record of the variations of pressure each time the lock for men is used. As any very sudden drop in pressure might cause mechanical injury, the outlet tap should be so arranged as to prevent decompression at a maximum initial rate of more than about one pound in five seconds[1]. With this arrangement and an ordinary tap, the rate of decompression would diminish considerably as the pressure fell, and the proper point for interrupting the decompression could be accurately reached.

The tables in Appendix IV have been calculated with special regard to the comparatively short periods of exposure to pressure in diving work;

[1] The delivery of the inlet tap should also be restricted, and the man in charge should have strict directions to take care that the rate of admission or discharge of air does not cause pain in the ears, &c. of any of the men in the lock. To avoid pain a very slow rate of air admission may sometimes be needed, but with practice a rise of pressure of one atmosphere per minute is often not too much, so that any definite rule, limiting the rate to much less than this, seems scarcely desirable.

and the stoppages recommended during the divers' ascent after exceptionally long periods of exposure are somewhat shorter than would be desirable apart from the risks entailed by the long stay under water. In the case of caisson and tunnel workers, on the other hand, it is only in exceptional cases that the exposure to pressure lasts less than three hours; and usually the exposure during the day lasts at least six hours.

With such long exposures and only moderate pressures the calculated theoretical rate of safe decompression after the first rapid stage is nearly uniform; and the rules for decompression may be greatly simplified by adopting uniform slow decompression or uniform stages[1].

The following table shows the rate of uniform slow decompression calculated to be safe after the initial diminution of absolute pressure in the proportion of 2 : 1. Suppose, for instance, that men were working at a pressure of 24 pounds in 3-hour spells, with an hour's interval between for a meal. In coming out they would be rapidly decompressed to an absolute pressure of $\frac{24+15}{2} = 19{\cdot}5$ pounds or 4·5 pounds of excess pressure. After the first 3-hour spell of work the slow decompression would be at the rate of one pound in three minutes, or $3 \times 4{\cdot}5 = 13\frac{1}{2}$ minutes in all. After the second spell the rate would be one pound in five minutes, corresponding to $22\frac{1}{2}$ minutes in all. If they stayed for the whole period in the compressed air the rate of slow decompression would be one pound in seven minutes corresponding to $31\frac{1}{2}$ minutes in all. To take another example, if the work were at 40 pounds excess pressure the men could be rapidly decompressed to $\frac{40+15}{2} = 27\frac{1}{2}$ pounds of absolute pressure, or $12\frac{1}{2}$ pounds excess pressure. After a first 3-hour spell of work the period of slow decompression would therefore be $12\frac{1}{2} \times 7 = 87$ minutes: after a second spell (with an interval of 30 or 45 minutes outside the lock) $12\frac{1}{2} \times 8 = 100$ minutes; and after a continuous exposure of six or seven hours, $12\frac{1}{2} \times 9 = 112$ minutes[2].

[1] With the lock air-tight, and no ventilation, uniform decompression at any required rate could be easily secured by means of a reducing valve on an outlet, with a graduated tap beyond it, the arrangement being similar to the reducing valve and tap usually connected to a cylinder of compressed oxygen or gas used for limelight. If the delay in the lock is so long that ventilation is required, or if ventilation is needed in order to compensate for accidental leakage, it would be best to have an adjustable safety valve on the outlet, and adjust this by one pound at a time at the proper intervals.

[2] We have some doubt as to whether the increased slowness of decompression after very long exposures would be altogether sufficient to meet the increased tendency to slight symptoms ("bends"). These are, however, of minor importance if all serious symptoms

TABLE I.

Table showing rate of decompression in caisson and tunnel work.

Working pressure in pounds per square inch	Number of minutes for each pound of decompression after the first rapid stage: After first three hours' exposure	After second or third three hours' exposure, following an interval for a meal	After six hours or more of continuous exposure
18—20 pounds	2	3	5
21—24 ,,	3	5	7
25—29 ,,	5	7	8
30—34 ,,	6	7	9
35—39 ,,	7	8	9
40—45 ,,	7	8	9

It will be evident from the last example that in order to avoid waste of time in the lock it would be preferable with pressures exceeding about 25 pounds to keep the men under pressure continuously during each shift. Thus with two 3-hour spells of work separated by a decompression, the time spent in the lock would be $87 + 100 = 187$ minutes; whereas if the meal were taken in the compressed air, the two 3-hour spells would only imply 112 minutes in the lock.

With working pressures exceeding about 25 pounds the air-lock should be roomy and comfortably arranged, and large enough to take the whole of a shift of men. It should be provided with an electric heater, telephone, and if possible some sort of lavatory accommodation.

With pressures up to 45 pounds, or four atmospheres of absolute pressure, there appears to be no substantial objection to keeping men for six hours, or even more, continuously under pressure, provided that the mode of decompression is thoroughly safe. With pressures exceeding about 40 pounds, the practice has hitherto been to limit the exposure to about one hour, and employ rates of decompression which are dangerously rapid. This plan implies greatly increased risk and expense, since for the accomplishment of the work the number of decompressions is six times as great, and the men are idle most of the day. The actual increase in risk must be very great.

In tunnel work, or any other kind of work where plenty of space is available, there would be great advantage in providing a large air-lock, or section of tunnel, in which the pressure was constantly maintained at a little less than half the absolute pressure in the working section.

are prevented. We also think that with long shifts, exceeding a total of about 3 hours, still slower decompression would be needed for any men inclined to obesity. Such men should, therefore, be excluded in the medical examination which all men working in air at high pressures ought previously to undergo.

The men could then pass rapidly (in two or three minutes) from the working section into this intermediate lock or section, where they could take their meals, wash, and change their clothes. After a sufficient delay (dependent on the working pressure) they could then pass out rapidly. If, for instance, the working section was at a pressure of 30 pounds, the intermediate or "purgatory" lock could be kept at an absolute pressure of about $\frac{30+15}{2{\cdot}2} = 20{\cdot}5$ lbs., or $5\frac{1}{2}$ pounds of excess pressure[1]. At the end of the day's work there would be a delay of about 50 minutes in this large lock, during which the men could wash and change, or take a meal. With this plan all delays during actual decompression would be obviated, so that ingress and egress would be free at all times, and the men could use the locks employed for material. For persons going in for only short periods the delay in the "purgatory" lock could be curtailed in accordance with the tables in Appendix IV. The movement of the men while employed in washing, changing clothes, &c. would hasten the process of desaturation, and this would be a further advantage.

In any case where it was specially desirable to reduce the period of delay in the air-lock to a minimum, recourse could of course be had to breathing oxygen during the period of slow decompression. This would about double the rate of desaturation, and therefore halve the delay. The oxygen could be breathed from a bag, and the CO_2 absorbed by a purifier, so that very little oxygen would be needed. By so arranging the mouthpiece that part of the expired CO_2 was rebreathed, and the respiration and circulation thus stimulated, a still better result would be attained.

The results of some of our experiments seem to indicate that even the very slow rate of stage decompression which has been recommended above would be insufficient to completely obviate the risk of "bends" occurring after prolonged exposure. The rate of saturation and desaturation of some of the tissues which are the seat of "bends" is possibly slower than we have provisionally assumed. What we have aimed at is to completely obviate the risk of any serious symptom, while at the same time reducing the chances of "bends" to a minimum.

[1] A comparatively rapid fall in absolute pressure in the proportion of 2·2 to 1 is within practically safe limits, particularly if the previous period of continued exposure has not exceeded three or four hours.

Part II. Experimental.

1. *Apparatus.*

We owe the large pressure chamber (Plate V) in which both human and animal experiments were conducted to the generosity of Dr Ludwig Mond, F.R.S. It is a short segment of a boiler of $\frac{5}{8}$ inch plate resting on its side; the ends are slightly dished steel plates $\frac{1}{2}$ inch thick. Inside it measures $7\frac{1}{3}$ feet long by 7 feet wide and high, and has a capacity of 9500 litres (336 cubic feet). It is thus large enough to hold 3 or 4 persons comfortable and can be used for animal experiments lasting several hours without the necessity of ventilating. There are two doors: one, an oval manhole (24 × 15 inches), is easily removed and is in common use; at the other end is a large rectangular plate (28 × 24 inches) which can be unbolted for the admission of bulky articles. There are a number of spring and simple valves; the largest is in the floor of the chamber and serves also as a drain; when fully opened it reduces the pressure from 100 lbs. to atmospheric pressure in rather less than a minute. Besides this there are four spring and three simple valves so arranged that the pressure can be completely controlled either from inside or outside. The front is also furnished with an air-lock, by means of which small articles can be passed in or out of the chamber during an experiment. Three windows are provided of stout glass; as a precaution for safety these are fitted with an arrangement whereby the breaking of the glass releases a solid metal rubber-faced plug which falls into the hole. Wiring for lights, a telephone, electric heaters and a motor to drive a fan, kymograph, &c., is introduced through fibre plugs.

The pressure is raised or reduced by a simple compressor driven by a gas engine. While this has proved quite satisfactory for negative pressure experiments, the rate at which the pressure can be raised by its means is only about 2 lbs. per minute. This was a serious obstacle to the examination of the effects of exposure to high pressures of short duration. Accordingly after the preliminary experiments, a multitubular compressed air reservoir was placed at our disposal by the Admiralty. This reservoir has a capacity of about 22 cubic feet, and by charging it to about 70 atmospheres with a two-stage liquid-air compressor and also another steel bottle to 180 atmospheres we were enabled to suddenly blow the contents into the chamber and so reach a pressure of 60 lbs. in

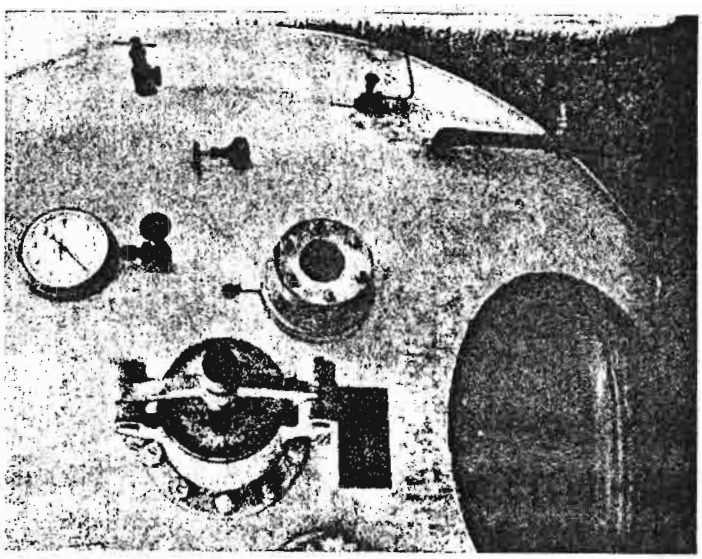
4 minutes, and 75 lbs. in 5½—6½ minutes according to the temperature. The pressure is indicated by two Schaffer spring gauges (one of which is visible from within the chamber) for positive pressures, and one spring gauge outside and a mercurial barometer inside for negative pressures. The spring gauges show a lag of nearly 2 lbs. up to about half an atmosphere, but above one atmosphere they are concordant and, as far as could be ascertained, correct.

The chamber and accessory apparatus have now been frequently used during eighteen months for experiments at pressures varying from 100 lbs. above to 8 lbs. below atmospheric pressure, and have been found very satisfactory and convenient.

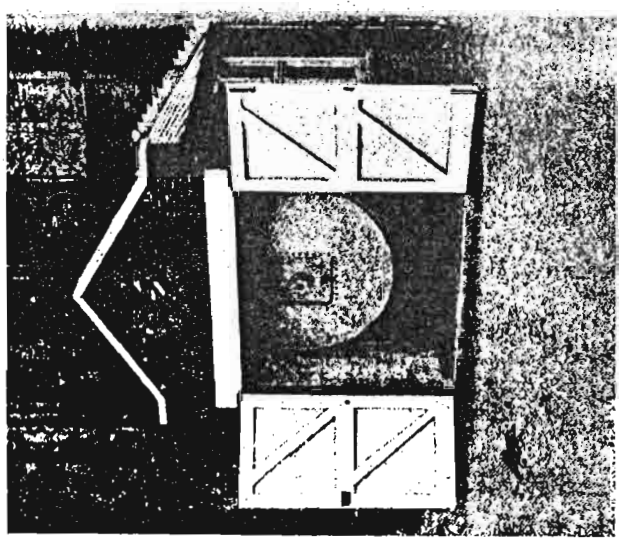
2. *Choice of experimental animals.*

A few experiments were made with rabbits, guinea-pigs, rats, and mice, but for regular use goats were selected chiefly because they were the largest animals which could be conveniently dealt with and which could be obtained in considerable numbers. The questions under consideration depend in a very fundamental way upon the rate of circulation in the animal under investigation. Among the ordinary mammals this must vary with the rate of the respiratory exchange per unit of body weight and is therefore proportional to the ratio between body surface and body weight. The susceptibility of any animal to caisson disease after sufficiently long exposure to compressed air must depend in the main upon the rate at which its respiration and circulation removes the excess of dissolved nitrogen on decompression.

Not only is this excess removed more rapidly in small animals, so that the time during which bubbles might be formed is correspondingly less, but, as already pointed out, there is every reason to believe that the time during which the venous blood remains in a supersaturated state during each round of the circulation largely determines the formation of bubbles. This time is so short in small animals that no bubbles at all are formed, in spite of the temporary existence of very great supersaturation in the blood and tissues. The susceptibility of any species of animal then varies enormously with the size. Thus a mouse, weighing 20 grammes and with a CO_2 production of about 8 grammes per kilo per hour, is much less susceptible than a goat, weighing 20,000 grammes and producing about 0·8 gramme CO_2. We have indeed failed to produce any symptoms at all in mice on decompression in less than a minute after one hour's exposure at 75 lbs., an experience



The steel chamber at the Lister Institute. Front end, showing the manhole for entering, the small air-lock for passing food, &c. into the chamber, an inspection window, a pressure gauge, and several valves, &c.



The steel chamber at the Lister Institute. View from outside, showing the back end of the chamber, with the large door and one inspection window.

invariably fatal to goats. In the same way dogs, with a respiratory exchange of some 1·3 gms. CO_2 per kilo per hour, are much less susceptible than men with an exchange of about 0·5 gms. Thus Heller, Mager and v. Schrötter observed no symptoms in dogs[1] on sudden decompression from any pressure less than about 60 lbs., while abundant illnesses are caused in man, and for the matter of that in goats also, by inappropriate release from 30 lbs. pressure. It therefore appears clear that it is necessary to use large animals for experiments which are designed to illustrate the incidence of caisson disease in man. Indeed the quantitative factor by which the results obtained on quite small animals might be translated into human experience is so large as to become qualitative in character.

Since pressures of some 100 lbs. or more are required to produce symptoms in a reasonable proportion of small animals, the use of animals such as goats is also very desirable in order to keep as far away as possible from the point at which the partial pressure of oxygen is high enough to cause toxic effects. We found that an exposure of three hours at 81 lbs. to an atmosphere containing 36 % oxygen (the oxygen pressure being thus equal to that of 150 lbs. excess air pressure) killed one goat out of seven with "pneumonia." Our experience shows that it is not necessary to exceed an air pressure of half this (75 lbs.) to produce symptoms which are sufficiently varied and severe to satisfy experimental requirements.

Experience also showed that goats were very suitable animals in that slight symptoms were presented to our notice in a definite objective form. The lesser symptoms of caisson disease cannot be neglected, and there are reasons for supposing that their occurrence is not exactly conditioned by those experimental circumstances which in a more severe form produce serious and fatal results. They cannot be properly detected in mice or guinea-pigs or even in rabbits. Goats, while they are not perhaps such delicate indicators as monkeys or dogs, and though they are somewhat stupid and definitely insensitive to pain, are capable of entering into emotional relationships with their surroundings, animate and inanimate, of a kind sufficiently nice to enable those who are familiar with them to detect slight abnormalities with a fair degree of certainty.

The animals, 85 in number, used in the present experiments were a mixed collection of ordinary English goats of no particular breed. They

[1] The weights are only given in a few instances; from these it may be surmised that the dogs were small (5 to 12 kilos).

were about equally distributed between the sexes, and varied in weight from 10 to 30 kilos, the average being rather less than 20 kilogrammes. All were apparently adult, judging from the fact that none showed any increase in weight while in our possession. One or two (XIII A, XXXII A) seemed to be quite aged, but the rest were fully active.

On the whole the herd remained healthy. Two died of apical pneumonia and two of diarrhoea, which was at one time epidemic in a severe form. The cause could not be determined, but the trouble became much less marked after the animals were placed on a more meagre diet and corn withheld. Three animals were under some suspicion of being infected with *M. melitensis*[1]; two of them seemed rather depressed (though not more so than appears to be natural in some goats), while the third showed no signs of ill-health. Various items of pathological interest were found in those which came to post-mortem: in the lungs various nematodes were found several times, *Linguatula* once, and a surgical needle once; a *Streptothrix* abscess in the stomach wall followed puncture with a needle to relieve distension; a bony tumour was found in an adrenal gland; in one old goat (XXXII A) the aorta was extensively atheromatous; flukes occurred in the liver once, while hydatids in the peritoneum were very common and intestinal worms abundant. None of these conditions (except possibly the arterial disease) can however be considered to have rendered the animals definitely abnormal as far as caisson disease was concerned, and none of them could be attributed to exposure to compressed air.

3. *Respiratory exchange of goats.*

The difficulties of measuring directly the circulatory activity of normal animals are almost insuperable. This must be however in general proportionate to the rate of respiratory exchange, and a number of determinations of the CO_2 production of our goats were made in order to get a line of comparison with other animals (and especially man) in respect of the rate at which air would be taken up by and discharged from the body.

[1] Of 22 animals whose blood was examined, 16 gave no reaction with *M. melitensis* at a dilution of 1:20, 3 gave some reaction at 1:20, while 3 animals gave complete agglutination up to 1:200 (XVIII A, XXVI A, XXIII A). Cultures from the blood during life were negative, and when they eventually came to autopsy cultures of blood, spleen, liver, inguinal, axillary, mesenteric and mammary glands were negative as regards *M. melitensis*. The exact history of these animals could not be obtained, but there is practically no doubt that they had never been out of England.

The observations were made by using the pressure-box as a respiration chamber. The animals were enclosed and hourly samples removed (after thorough mixing with an electric fan) and analysed in a delicate form of Haldane's gas analysis apparatus. The results were entirely satisfactory, the successive analyses showing a regular increase in the CO_2. The goats led a regular life, and all the observations were made at approximately the same time of day so that they are fairly comparable with one another in respect of the influence of food. The animals remained fairly quiet, though they seldom lay down.

The results of 27 experiments are given in the next table. The analyses have been calculated in grammes of CO_2 per hour per kilo of

TABLE II.

Number of experiment	Temp. °C.	Bar. mm.	Duration hours	Pressure lbs. positive	Goats: Number	Goats: Males	Goats: Females	Goats: Total weight kilos.	Goats: Average wt. kilos.	CO_2 gms. per hour: Per kilo body wt.	CO_2 gms. per hour: Per 1000 sq. cms. surface	Remarks
I	19	764	3	0	4	—	4	99·0	24·7	1·006	2·625	
II	13	763	2½	0	4	2	2	62·8	15·7	1·123	2·070	
III	15·5	765	1½	0	9	4	5	159·7	17·7	0·908	1·823	
IV	15	752	1½	0	9	3	6	171·1	19·0	0·727	1·749	
V	17	762	3	0	6	6	—	111·8	18·6	1·104	2·554	R. Q. 1·03.
VI	11	769	6	0	6	—	6	138·8	23·1	0·670	1·771	R. Q. 0·90.
VII	13	765	5	0	6	6	—	121·3	20·2	0·975	2·389	R. Q. 1·06.
VIII	13	754	4	45	7	7	—	142·7	20·4	0·887	2·187	
IX	13	740	4	45	8	—	8	193·7	24·2	0·627	1·682	
X	15	754	7	0	7	7	—	142·1	20·3	0·664	1·630	R. Q. 0·91.
XI	16	780	6	0	6	6	—	126·3	21·0	0·615	1·533	Fasting 20 hrs.: R. Q. 0·82.
XII	15	778	4	21	7	7	—	140·9	20·1	0·763	1·770	
XIII	15	774	7	0	8	—	8	193·7	24·2	0·548	1·469	R. Q. 0·82.
XIV	14	760	4	45	13	5	8	295·7	22·7	0·667	1·738	R. Q. 1·08.
XV	16	758	3½	25	6	6	—	127·9	21·3	0·959	2·367	
XVI	17	764	3¾	20	7	3	4	148·7	21·2	0·635	1·572	
XVII	15	762	4	0	6	6	—	127·9	21·3	0·669	1·652	
XVIII	17	762	1¾	45	6	—	6	122·1	20·3	1·020	2·504	
XIX	14	761	4	45	6	6	—	127·9	21·3	0·697	1·722	
XX	13	760	3	0	5	—	5	100·1	20·0	0·921	2·258	
XXI	16	760	4	45	5	—	5	100·1	20·0	1·104	2·701	
XXII	15	762	4	45	6	6	—	127·9	21·3	0·967	2·390	
XXIII	16	768	4	45	5	—	5	100·1	20·0	0·852	2·083	
XXIV	12	776	5	0	4	2	2	95·3	23·8	0·717	1·853	
XXV	14	775	5	0	4	2	2	95·3	23·8	0·751	1·941	
XXVI	14	775	5	0	4	1	3	76·9	19·2	0·624	1·501	
XXVII	13	766	5	0	4	1	3	76·9	19·2	0·704	1·693	

body weight and also per 1000 square centimetres of surface according to the usual formula $S \times 100 = \sqrt[3]{W^2} \times 11{\cdot}2$, where S = surface in square centimetres and W the body weight in kilogrammes.

The goats used belonged to Series II (Exps. 1—4), III and IV.

The results of these experiments are very variable; the averages are shown in the next table:

TABLE III.

	No. of experiments	CO_2 in grms. per hour: Per kilo body-weight	CO_2 in grms. per hour: Per 1000 sq. cms. surface
At atmospheric pressure	16	0·795	1·907
At 45 lbs. positive	8	0·853	2·126
At 20, 21 and 25 lbs. positive	3	0·786	1·903
All pressure experiments	11	0·834	2·065
Males only	10	0·830	2·019
Females only	8	0·843	2·137
Mixed experiments	9	0·762	1·771
All experiments	27	0·811	1·971
		(410 c.c.)	(997 c.c.)

One may conclude that goats produce about 0·8 grm. CO_2 per kilo per hour under conditions of incomplete rest, and that no great departure from this figure is occasioned by the animals being under pressure up to 45 lbs. or by sex. It is shown elsewhere[1] that something more than 10 % of the total CO_2 produced by goats comes from the fermentation of the contents of the alimentary canal, and figures detailed below (p. 409) indicate that one-fifth of the body weight is contributed by these contents. In comparing the CO_2 production of goats with that of man, we may regard these two corrections as roughly balancing one another and may neglect them.

It appears that man produces under conditions of bodily activity comparable to that of our experimental animals, about 0·45 to 0·5 grm. CO_2 per kilo per hour. Goats therefore show a respiratory activity approximately 1·7 times that of man. This figure corresponds fairly well with that calculated from the size. If the respiratory exchange per unit of surface is the same, a goat of 20 kilos will produce 1·5 times as much CO_2 per unit of weight as a man of 70 kilos.

4. *Method of conducting the experiments.*

No animals were subjected to experiment when obviously ill. As a rule five to eight animals were put in at one time. The pressure

[1] *Journal of Physiology*, vol. xxxvi. (1907), p. 283.

having been raised to the desired point, the chamber was entirely closed and no ventilation given until decompression began. The average CO_2 production of goats is about 435 c.c. per kilo per hour at ordinary temperatures. The chamber usually contained 100 to 150 kilos of goat so that the CO_2 rose about 0·45 to 0·55 % (measured at atmospheric pressure) per hour. In this way it never attained a harmful partial pressure in experiments lasting from a few minutes to four hours. In the few observations made with an exposure of eight hours, the CO_2 was allowed to accumulate for four hours and afterwards the chamber was ventilated so that the CO_2 did not exceed a partial pressure of 2 % of an atmosphere. No experiments have been made to directly examine the possible influence of CO_2 upon the incidence of caisson disease. It appears to the authors that the effect must (1) in any case be very slight with partial pressures of less than 2 or 3 %, and the result, if any, of the increased respiratory and circulatory activity must be in the direction of diminishing the ill-effects of decompression after any but quite short exposures[1].

After the preliminary experiments (Series I), the animals were never used more than once on the same day, and, with rare exceptions, not on succeeding days. In many cases indeed individual goats rested for a week or more between the experiments.

During decompression the animals could be watched fairly satisfactorily through the windows of the chamber, though fog of course completely blocked the view during the actual moments of rapid decompression. At the end they were allowed to escape from the chamber and run about free in the yard. They were kept under continuous observation for half an hour or longer, and were frequently seen throughout the day. We found that practically all the symptoms which were going to appear declared themselves within thirty minutes, though a few slight signs were probably missed. We also found that slight signs were much more obvious when the animals were not distracted or excited by food or other causes. During the breeding season it is advisable to keep the males and females separate, and, by removing any sources of interest, to allow the animals to fall into a state of meditative boredom. Under these circumstances, trivial symptoms are easily detected which are not made the subject of objective demonstration by animals engaged with their appetites.

[1] Greenwood (*British Medical Journal*, June 22nd, 1907, Supplement, p. 409) has recently found that high percentages of CO_2 do not increase the liability to decompression symptoms.

No observations were made of the temperature within the chamber during an experiment. Very hot and very cold weather did not seem to influence the results. The air in the chamber was always warmed by compression and sometimes also artificially, while decompression was of course accompanied by sudden, often very severe, spells of cold. No account has been taken of variations in atmospheric pressure. The extreme readings of the barometer on record are 806 and 689 mm. at sea level, and in this country 790 and 695 mm.[1], giving ranges of 117 mm. and 95 mm. or about $2\frac{1}{4}$ and $1\frac{4}{5}$ pounds. Even this variation, though it occurs at an important part of the absolute pressure scale, cannot be of great significance.

Times of exposure of one hour or less are, unless the contrary is directly specified, to be taken as indicating actual exposure to the given pressure, the time of compression (six minutes) being neglected. For longer exposures it was sometimes convenient to raise the pressure more slowly: in these cases therefore the times specified may indicate either the actual exposure *plus* four to six minutes compression or a virtual exposure calculated by adding the actual exposure to half the time of compression which is in minutes roughly one quarter of the pressure in pounds positive (see above, p. 362).

As will be gathered from the details given below, the general scheme of the experiments involved the examination of three variable factors—degree of pressure, duration of exposure and duration and mode of decompression. For the most part the degree of pressure was kept constant while the other two factors were varied. It soon appeared from the preliminary experiments that the individual variability of the animals was very large—larger indeed than the difference between many of the modes of decompression which it was desired to examine. It also appeared that the relative susceptibility of the different individual animals remained fairly constant so that after a time one could pick out goats which were known to be either susceptible above the average or definitely resistant to caisson disease. It was therefore clear that either an enormous number of animals had to be employed or the experiments had to be so framed as not to produce fatal results and so reduce the proportion of susceptible individuals in the herd. It appears probable that any 20 or 30 goats would give much the same results, but if many are lost it is necessary to discard the remainder and procure a fresh batch to be subjected to the comparative experience. Obvious reasons prevented this procedure. It was therefore necessary to be at

[1] *Nature*, vol. LXXV., 1907, p. 330.

some pains to secure that the deaths should be as few as possible so that the same individuals might pass through a number of different combinations of pressure, exposure and decompression. The animals were therefore first put through the experiments which we surmised would give least symptoms, and were subsequently exposed to circumstances of progressively increasing severity. Even so, each batch of animals became selected to a more or less considerable degree. Be it noted however that one may in this way obtain strong evidence of an *a fortiori* kind. For, if the selected resistant members of the herd show many symptoms in the severest experiments, so much the more would the whole original lot of average animals have been affected. This individual variability of the animals renders many of our experiments incomplete, and should be constantly borne in mind in considering the results obtained.

5. *The symptoms observed in goats.*

The symptoms observed in goats in sequence to decompression are protean in character. The majority may however be grouped under a few definite heads.

1. *Bends.* The commonest symptom which we have observed consists of the exhibition of signs indicating that the animal feels uneasy in one or more of its legs. The limb, most commonly a fore-leg, is held up prominently in the air and the animal is evidently loth to bear weight upon it (see Plate VI[1]). In mild cases such a limb is used normally in walking or running, but in other instances the animal limps more or less considerably when it is forced to use the affected member, and is often very anxious to lie down. No tenderness can be detected on pressure or manipulation of the leg and it is not altogether clear that the animal suffers definite pain. We have however noted that a goat may break its leg and immediately use it for progression without evincing any signs of pain. We may conclude from this that the response to stimuli which in many animals would be distinctly painful is largely suppressed in the goat to the level of the exhibition of a consciousness that the limb is somewhat abnormal and not well suited for active use. But it must be understood that this objective demonstration is a very conspicuous and definite symptom. There is little doubt that these symptoms observed in the legs of goats are the equivalent of the "bends" or "screws" which are the commonest

[1] We are indebted to Dr H. W. Armit for this photograph.

"Bends" of fore-leg in a goat.

symptoms in caisson workers; in human experience they are of course accompanied by definite pain, often of a severe character.

The following table shows the distribution of "bends" in the last 110 cases observed:

TABLE IV.

One hind leg	28
One fore leg	70
Both hind legs	1
Both fore legs	1
One fore and one hind leg	10
Total one leg	98
Total two legs	12
Total right	50
Total left	48

"Bends" may be seen immediately at, or indeed (but very rarely) shortly before the end of a long decompression. Most commonly however they come on after an interval of about 15 minutes; on the other hand they may be delayed still more. As might be expected, the period of delay varies with the duration of decompression: thus the average delay in a number of cases after rapid decompression (1 to 10 minutes) was 16 minutes, which was reduced by long decompression to six minutes. Their duration appears to be brief; all evidence of their presence has usually disappeared in one or two hours and it has been very exceptional for any trace of them to be present next day (16 to 20 hours).

"Bends" in parts of the body other than the limbs are very difficult to identify in animals; we have however occasionally noted symptoms which might well be bends in the trunk, though we are not prepared to definitely identify them as such.

2. *Temporary paralyses* may be of two kinds. In the first a general weakness is present accompanied with dyspnoea and there is dragging of the hind legs with foot-drop. These are clearly symptoms due to a general deficiency of oxygen from pulmonary embolism and are comparable to the paralyses seen in, *e.g.*, carbon monoxide poisoning in animals and men. In our records and the tables such cases are not classified as "paralysis" but as "dyspnoea." In the second group fall a series of cases which are obviously of nervous origin. The animal, while showing no signs of general illness, or in other instances having already had bends, exhibits foot-drop or a more extensive palsy in one or more hind- or fore-limbs. The paralysis does not usually come on till about 15 minutes after decompression, rapidly becomes more marked

for a few minutes after the first signs are noted, and then soon begins to mend, so that there is marked improvement in about half an hour, and by next day the animal is found quite well. This form of paralysis chiefly involves the hind legs (16 out of 19 cases).

3. *Pain.* In some cases the animals have shown signs of acute pain by urgent bleating and continual restlessness. Bleating in goats after decompression is usually a sign of distress such as is produced by cardiac and respiratory embarrassment and is often present in fatal cases. In other instances animals showing only severe bends bleat in a most distressing manner and are evidently in acute pain: at the same time they may gnaw at some part of their body (such as the testicles) as if localising the origin of the pain. In animals which have recovered, we have not had any instance where these signs persisted for more than 10 or 15 minutes.

4. *Permanent paralyses.* The onset is usually immediately after decompression, the condition is complete from the first and for at least several days there are no signs of improvement. In a few cases the first paralysis has passed off (to all appearances completely) in two or three hours and the animal has been found next morning to be again paralysed. This second paralysis is permanent. A similar history has often been noted in human cases. In 15 cases out of 16 the condition has been a paraplegia, and in one all four legs were affected more or less. In some there has been retention of urine, and one animal had to be killed on account of acute distension of the stomach which came on some 20 hours after the onset of the paraplegia. In the most severe cases the animals have been killed; others have however soon begun to mend and have lived for some months with a slight spastic paralysis of the hind legs.

5. A fair number of cases have occurred where the animal has been obviously ill, but in which it has been impossible to identify any definite local symptoms or any definite dyspnoea. The goat may lie down, refuse to move or to be tempted with corn (of which goats are inordinately fond), sometimes lying extended on the side, sometimes hurriedly rising, walking a few steps and then lying down again. On two occasions the most probable interpretation of the symptoms was that the animal was blind. The goat may run wildly about instead of becoming very apathetic and depressed. These and other such symptoms are on the whole somewhat persistent and the animal is often dull and poorly the next day. In one case (XXV A) the goat showed little but a marked apathy and distaste for food, but died 16 hours later.

6. *Dyspnoea* is usually the precursor of (7) *death* and only a minority of goats survived after showing clear dyspnoea. In these cases the condition has rapidly improved; more commonly however it progressively increases till the animal is moribund, when it is replaced by irregular, faint, gasping respiration. The mucous membranes become livid and pale and the animal lies for a short time unconscious before respiration stops. The heart continues to beat regularly throughout and the rate is not apparently much altered. Only on one occasion have we been able to hear gurgling in the heart on auscultation: it was then audible at some distance. Death ensues at varying periods after decompression; with very severe experiments (*e.g.* 100 lbs.: 1 hour: 1 minute)[1] it may follow in five or ten minutes: with more moderate conditions it is delayed for 20 or 30 minutes, or rarely for two or three hours: on three occasions it has followed still later, up to 40 hours. The delay in the onset of the first symptoms is often most striking; the animal may appear quite normal for as long as 10 or 15 minutes, dyspnoea then appears, the goat falls down helpless and in another 15 minutes is dead.

8. *Mechanical symptoms* are not important. We have not been able to satisfy ourselves that goats ever suffer materially during compression from the ear troubles which are so common in men. Abdominal distension is occasionally extreme, but the animal soon empties its distended stomach and seems to be little inconvenienced[2].

Our index throughout has been the presence of symptoms, not the presence of bubbles. Anticipating here a later section (p. 410) we may say we are in entire agreement with the view which attributes most of the severe symptoms of caisson disease to local or general blocking of the circulation by bubbles of gas. One might suppose in consequence that the incidence of severe symptoms, especially of paralyses, would be of a haphazard kind, since they would be to a large extent dependent on the chance distribution of bubbles by the blood stream. Some support for this view is perhaps to be found in the records of caisson workers given by von Schrötter; as far as can be ascertained from the details given, the cases of paralysis and dyspnoea were distributed through the whole range of pressure experienced by the men in about the same proportion to the total number of illnesses of all kinds, which latter increased greatly as the pressure became higher. It should however be noted that the range of pressure was small (up to 2·4 atmospheres positive), and

[1] *i.e.* pressure 100 lbs. positive; exposure for 1 hour; decompression in 1 minute.

[2] Post-mortem experience shows that the stomach alone is distended, not the bowels.

the general experience of caisson works as well as our own experiments with animals are distinctly at variance with these results. We have noted only two instances of what may be called "chance incidence" of paralysis: (*a*) goat XV (Series II) had a paraplegia after 15 minutes' exposure at 75 lbs. and decompression in 30 minutes uniformly, and afterwards lived for some time, passing through much more severe experiments without symptoms, and eventually being killed with some difficulty by 75 lbs: 2 hours: 1 min.: (*b*) goat XII A (Series III) after 45 lbs.: 1 hour: 10 min. uniform, had apparently very severe bends; it did not however recover in the usual way and became partially paraplegic, subsequently passing through many comparatively severe experiments without symptoms. The tables of our results seem to show quite clearly that as the conditions of experiment become more searching, not only does the frequency of symptoms increase but the proportion of severe to total symptoms becomes much greater.

It is necessary for comparative purposes to form some idea of the relative importance of these different symptoms, and to consider how far they may be classed as relatively dangerous or comparatively negligible. "Bends" are clearly a slight symptom; there is abundant evidence both in goats and men that their occurrence is no indication of urgent danger to life. At the other end of the scale we have death. Dyspnoea is not far removed in significance from death, and lasting paralyses are somewhat less serious than dyspnoea. Next in order come pain and those indeterminate conditions which we have grouped as "indefinite and various general": these may be followed by death and are much more indicative of danger than bends. Temporary paralyses are not so important and we are inclined to the view that they are not much more dangerous than bad bends. This classification is based for the most part on our experience as to the kind of experiment with which each group of symptoms is commonly associated, and the way in which the different groups are associated together in the same experiment. The individual variability of the animals introduces many difficulties, but it is certain that the more severe the conditions of pressure, exposure and decompression, the more likely it is that the animals will suffer from symptoms which we have classed as severe.

Immunity to symptoms. There is not the slighest ground, either theoretical or experimental, for supposing that animals or men, as the result of repeated exposure to compressed air, acquire any immunity to the formation of bubbles within their persons. It must be remembered in this connection that the susceptible individuals become eliminated,

so that those who have been through many decompressions necessarily show more than average resistance. The matter is not so clear with regard to the exhibition of symptoms resulting from such bubbling. We have a certain amount of evidence, too vague to be detailed, that some goats show slight bends rather more easily in their first few experiences, and it is not difficult to imagine that they might grow to neglect altogether those bubbles which evidently cause them no very great inconvenience at any time. With severe symptoms it is of course different: no one can suppose that a goat acquires immunity to extensive pulmonary air emboli or to infarction of the spinal cord.

TABLE V.

Series I. June—July 1906. Pressure 75 lbs. positive (=6 atmospheres absolute). Compression in 39—41 minutes. The details of the stage decompressions are shown in Table IX.

Decompression minutes		40—50 uniform			40–50 stages		
Actual exposure minutes		12	15	30	12	15	30
No. of goat	Sex						
I	M	0					
Pa	M	0	bends	0		0	0
XIII	F	bends					
XVIII	F	bends + indefinite[1]	0	0			0
XXI	F	0+0[1]	bends				
XXII	F	0	bends				
X	F		bends	bends			0
XVI			bends	bends		0	
XX	F		bends	bends	bends		
XXIV	M		0	bends		bends	0
XXVII	F		0			0	0
XXIX	F		0	bends, dyspnoea		0	
XXX			bends			bends	
XXXII			bends				
III	M			0			0
IV	M			0			bends
VI	M			0			0
XXVI	M			bends			bends + bends[1]
XXVIII	F			bad bends			
VIII	M				0		
XIV					bends		
XIX					bends		
II	M						bends
XXV	M						bad bends

[1] Each of these experiments was repeated upon the same animal: both results are shown. XVIII was generally uneasy, lay down, nothing definite.

TABLE VI.

Series II. December 1906 to January 1907. Pressure 75 lbs. positive (6 atmospheres absolute); compression 6 minutes.
The details of the decompressions are shown in Table IX.

Actual exposure mins.			1	3	15				30			60		120[17]			240[17]	
Decompression mins.			1	10 uniform	10 uniform	31 stages	31 stages	31 uniform	31 uniform	31 stages	31 stages	31 uniform	31 stages	31 stages	31 stages	70 stages	70 uniform	31 stages	31 uniform
No. of goat	Sex	Weight kilos																	
1	M	18·3		0		0	bad bends	0	0	bends	0		bends	0	bad bends	0	bends		
2	M	16·2	0			0	bends	sl. bends	paraplegia[5]	temp. paral.			0			bends	paraplegia[6]	bad bends, pain	
X	M	16·4	0		bad bends[1]	0	0	0	0	bends	0		0	0	bad bends[1]	bends	0	bends	temp. paral.
XI	F	17·1			bends	0	0	0	0	0	0	bad bends	bends	0	bad bends	0	bends		died[11]
XII	M	19·8			died[7]														
XIII	M	17·8			0	0	0	0	0	0	0		bends	0	bends	0	0	0	
XIV	F	18·6		0		0	[14]	bends	bends										
XV[12]	M	16·8	0		0	sl. bends[18]	0	paraplegia[4]	0	bends			0			bends	bends	0	bad bends
XVI	M	10·7	0		bends	0[18]	0	bends	0	bends	temp. paral.	bends	0	0	bends	0	bends	temp. paral.	
XVII	F	24·8				0[18]	0	0	0	bends			bends, dyspnoea			0	temp. paral.		
XVIII	F	26·3	0			0	0	bends	0	0	bends	bends	0	obscure[3]	bad bends	0	0	bends	
XIX	F	15·0				0	0	sl. bends	0	bends	0	bends	0	0	bends	0	bends		
XX	F	15·0				0	0	0	? bends	0	0	0	0	temp. paral.	bends	0	slight bends	died[10]	
XXI[15]	F	29·7				0	bends	obscure[2]	bends	bad bends						bends, dyspnoea			
XXII	F	19·0				sl. bends	0	0	? bends	0					died[9]				
XXIII	F	21·4				0		bends	died[8]										
XXIV[16]	M	35 about																	
XXV[13]	M	21·5	0		bends	0	0	bends	sl. bends	0		temp. paral.	0			bends	bends	bends	bad bends
XXVI	F	17·5				0	0	0	0				bad bends						
XXVII	F	12·0				0	0	bends	0	temp. paral.			0			0	0		

[1] Animal also lay down and seemed generally ill: no dyspnoea. [2] Lay down, grunted, seemed quite ill: no definite bends or dyspnoea. [3] Lay down and refused to move: no definite local symptoms. [4] Could walk with feet dragging in 6 days; nearly well when killed 9 weeks later. [5] Well in 9 days. [6] Almost well in 48 hours. [7] Died in 15 minutes. [8] Died in 30 minutes. [9] Died in 76 minutes: bad bends, dyspnoea, no paralysis. [10] Pain, paralysis, dyspnoea; died 43 minutes. [11] Dyspnoea; died in 26 minutes. [12] 72 lbs., 4 hours, 40 seconds; convulsions, dyspnoea, died 20 minutes. [13] 75 lbs., 2 hours, 45 seconds; bends, convulsions, dyspnoea, died 17 minutes. [14] Injured shoulder: killed immediately after decompression: see p. 412. [15] 75 lbs., 1 hour, 1¾ minutes; died in 10 minutes without dyspnoea or any symptoms except collapse. [16] 80 lbs., 2 hours, 10 minutes uniform; bends, paralysis, dyspnoea; died in 12 minutes. [17] In some of these experiments slow compression in 39 minutes was used, in which case the actual exposure was reduced by half the time of compression. [18] A somewhat different decompression was used in this experiment, viz. 75 lbs. to 17 lbs. in 5 minutes, wait 5 minutes, then 5 minutes at 13 lbs., 10 minutes at 9 lbs., and 10 minutes at 4 lbs.

25—2

TABLE VII.

Series III. February to June 1907. Pressure 45 lbs. positive (4 atmospheres absolute). The details of the stage decompressions are shown in Table IX.

Exposure minutes			15	30	45		60			90	120				240			480
Decompression minutes ..			2	2	30 stages	30 stages	1[1]	10 uniform	30 stages	52 stages	30 stages	1	10 uniform	30 stages	57 stages	10 uniform	30 stages	62 stages	10 uniform
No. of goat	Sex	Weight kilos																	
3	M	24·3	0	0	0	0	0	bad bends	bends	0		0	0		0	0		bends	0
4	M	19·6	0	0	0	0		0	0	0		paraplegia[4]	0	0	0	0	0	0	0
X A	M	17·0	0	0	0	0	bends	0	sl. bends	0		temp. paral.	temp. paral.	0	0	bends	0	0	temp. paral.
XI A	M	20·0	0	bends	0	0	0	0	0	0		0	bends	0	0	0	0	0	0
XII A	F	19·2	0	0				paraplegia[2]			0	0	0	0	0	0	0	bends	temp. paral.
XIII A[12]	F	28·2	0	0				bends	bends	0	bad bends		bends dyspnoea paraplegia[7]	0	0		0	0	
XIV A[8]	M	16·4	0	0	0	0		bends	0	sl. bends					0			bends	
XV A	F	15·5	0	0			0	0	0	0	obscure[3]	paraplegia[5]	bends	bends	0	bends	bends	bends	bends
XVI A	M	21·1	0	bends	0	0		bends	0	0		paraplegia[6]	0	0	0	temp. paral.	0	0	0
XVII A[9]	F	16·8	0	0					0	0	0			0	0		0	0	
XVIII A[11]	F	32·0	0	0			0	0	0	0	0	0	0	0	0	bends	0	0	bends
XIX A	M	22·4	bends	0	0	0	0	0	bends	sl. bends		bends	bends	0	bends	0	0	0	0
XX A[10]	F	19·8	0	0					0	0	0			0	0		0	bends	
XXI A	F	16·0	0	bends			0	bends	0	bends	bends	bad bends	bends	0	bends	bends	bends	bends	bends
XXIII A	F	26·3	0	0				0			0		0	0	0	0	0	0	0

[1] Six other animals had this experience also, of which 7, XXVII A, and XXIX A had bends, and XXIV A, XXX A and 9 showed no symptoms. [2] Never so completely paralysed that it could not walk, but no improvement occurred during 21 weeks. [3] Bleating, seemed lost and walked into objects as if blind: well in 2 hours. [4] Acute distension of stomach developed next day; goat killed. [5] Killed next day. [6] Hind legs and, to a less degree, fore legs also: could walk in 3 days; killed 15 days when much improved. [7] Killed 3 days. [8] Broke leg, killed. [9] Died of pneumonia: no reason to connect this with decompression. [10] Died, cause unknown. [11] 75 lbs., 3 hours, 58 seconds: died 12 minutes without showing dyspnoea or any symptoms except collapse. [12] Apparently elderly.

[12] Apparently elderly.

TABLE VIII.

Series IV. January to June 1907.

Pressure lbs. positive.....			75											75	51	45	39	30	25	20
Exposure minutes			3	6	10	15	½	15	30		60	120		180		180	120	120	60	240	240
Decompression minutes ...			1	1	¾	1	60 uniform	90 uniform	68 stages	68 uniform	86 stages	92 stages	100 uniform	134 stages	134 uniform	75 to 24 in 1¼ mins., wait 1 hr., total 118 or 131 (1)[1]	4	to – 6 lbs. in 6 mins.[7]	to – 6 lbs. in 6¼ mins.[7]	10 uniform	2	2
No. of goat	Sex	Weight kilos[2]																				
3	M	19·2		0	0	bends			0	0		0	0	0	bends				0	0	0
4	M	20·0						0				0	0						0	0	0
X A	M	17·8		0	paraplegia[11]			0	0	bends		0	temp. paral.	0					0	0	0
XI A	M	23·0		0	0	0		0	0	0		0	0	0	sl. bends				0	0	0
XII A	F	19·2		0	0	0						0	0				bends		bends	0	0
XIII A	F	28·2						0				temp. paral.	paral., dyspnoea						0	0	0
XIV A	M	16·4						0													
XV A	F	15·5						bends	0	bends		0	0	0					bends	0	0
XVI A	M	22·6						0				0	bad bends				died[6]		0	0	0
XVII A	F	26·8						0												0	0
XVIII A	F	32·0							0	0		0	0	bends	bends				0	0	0
XIX A	M	25·3						0	0	bends		bends	bends	0	? bends				bends	0	0
XX A	F	19·8						bends				0	0						0	0	0
XXI A	F	16·0		0	0	bends		bends	0	bends		0	0	0	0				bends	0	0
XXIII A	F	26·3						0				0	0				para-plegia[12]		0	0	0
XXIV A	F	14·4	0		0	temp. paral.	0		0	bends	0	bends	temp. paral.	0		? bends	bends, dyspnoea		0	0	0	0
7	M[14]	19·0	0				0		0	0				bends	bends	+ bends	bends		dyspnoea	1	bends	0
XXV A	M	14·6										0	died[4]									0
XXVI A	M	14·4	0								bends	0	0			0	0			0		
XXVII A	F	27·6	0	0	0	died[3]	0		0	bends	0	0	bad bends	0		0	bad bends[13]			0	bends	0
XXVIII A	F	21·1									0	bends	pain[9], temp. paral.	0	0	bad bends	died[14]				0	
XXIX A	M	18·5					0		0	bends	0	0	pain, dyspnoea	0	0	0	indefinite[10]		bends, dyspnoea	0	0	0
XXX A	M	20·6					0		0	0						+ 0	0		bends, dyspnoea	0	0	0
9	F	26·6	obscure[8]				0		0	0				0	0	+ bends	bends, dyspnoea			0	0	bends
XXXII A[15]	F	22·0					bends									+ 0	died[5]			0	0	0
XXVII	F	12·0					0		0	0				0	0	+ 0	bends					

[1] None of the animals in these two experiments showed any symptoms while watched for 1 hour at 24 lbs. and for ½ hour at 17 lbs. The results in the Table are those observed after final decompression. [2] Some of these weights differ from those in Series III, Table VII: several of the animals became somewhat thinner in the later part of the experiments. [3] Bleated, some convulsions, fore and hind legs paralysed, a little dyspnoea; found dead next morning; no bubbles. [4] Cried out, lay down, refused to move, evidently very ill but no local symptoms or dyspnoea; died between 17½ and 18 hours later; bubbles in heart. [5] Bleating, dyspnoea, died 27 minutes. [6] Paraplegia, dyspnoea, died 190 minutes after decompression and 130 minutes after returning to atmospheric pressure. [7] In these experiments the pressure fell to atmospheric pressure in 30—35 seconds, the thermal effect then produced a delay of about 2 minutes when the pressure began to fall further, reaching – 6 lbs. in about 4 minutes more: decompression was therefore in rough stages. The animals were maintained at – 6 lbs. for 1 hour. [8] No symptoms for 18 minutes, then cried out, was moderately convulsed and right fore leg appeared paralysed, no dyspnoea, seemed very ill for about 10 minutes but then recovered and showed no symptoms later. [9] Constant nystagmus also noted lasting about ¼ hour. [10] Lay down, evidently ill, no definite symptoms. [11] No improvement, killed 4 days. [12] No improvement, killed 6 days. [13] On a second trial again showed severe bends. [14] Castrated, of male habit. [14] Paralysis, nystagmus, no apparent dyspnoea, died 24 minutes. [15] An aged animal.

6. *Results of goat experiments.*

The detailed results of the experiments on goats are set out in the accompanying tables. With the exception of those in Series I, these tables contain nearly all the experiments which were made. Read vertically the columns give the results of the different combinations of pressure, exposure and decompression: the records of individual goats can at the same time be read on the horizontal columns. The tables give however no indication of the chronological sequence of events. In Series I only a few experiments are given; the series actually comprised 164 experiments on 34 animals, but the procedures adopted were, through ignorance, so ill-devised that no very definite results were obtained, though we gained information which enabled us to devise more satisfactory experiments subsequently. We have therefore extracted from Series I only a few results which illustrate the difference between stage and uniform decompression. The four series roughly represent four batches of goats, except that the animals of Series IV are the remnant of Series III with the addition of a further small herd. When reference is made to individual goats of Series I, the series is noted; otherwise the goats of Series III and IV are distinguished by "A": this does not apply to hornless animals which are specified by Arabic instead of Roman numerals, no two goats having the same number.

TABLE IX. *Showing the decompressions of goats from 75 lbs. and 45 lbs. Times given in minutes, one minute being occupied in each drop after the first.*

	Decompressions from 75 lbs. +								Decompressions from 45 lbs. +			
Series	I	II	II	II	IV	IV	IV	IV	III	III	III	III
Columns..	4, 5, 6	4, 5, 8, 9, 11, 12, 13, 16	4 (part)	14	12	7	10	9	3, 4, 7, 9 12, 15	8	13	16
First drop in	4	4	5	2	3	3	3	4	1	2	2	2
Wait at 32							3					
,, 27	5	2		7	15	4	9	2				
,, 22½	4			5	19	4	14	4				
,, 18	4	2	5	5	19	9	14	16		4	9	14
,, 14	4	4	4	10	24	9	14	16	3	14	14	14
,, 9	4 or 9	4	9	15	24	14	14	19	9	14	14	14
,, 4½	10 or 14	10	9	20	24	19	14	19	14	14	14	14
Total time	41 or 50	31	36	70	134	68	92	86	30	52	57	62

In the next table (Table X) the results are condensed and grouped in a simpler way, and one or two more experiments are given from Series I.

TABLE X.

Pressure lbs. positive	Compression minutes	Actual exposure minutes	Decompression minutes	No. of goats	No symptoms		Bends				Temporary paralysis	Various indefinite	Paraplegia	Dyspnoea	Total severe symptoms	Death
					Number	Percent.	?	Slight	Bends	Bad	Total						
75	6	½	60 un.	8	7	87			1		1					0	0
		1	1	6	6	100					0					0	0
		3	1	5	4	80					0		1			1	0
		3	10 un.	2	2	100					0					0	0
		6	1	6	6	100					0					0	0
		10	1	7	6	86					0			1		1	0
		15	1	6	2	33	1		1		2	1				1	1
		15	10 un.	7	2	29			3		3		1			1	1
		15	31 st.	34	29	85		2	2	1	5					0	0
		15	31 un.	36	19	53	2	3	8		13		1	2		3	1
		15	90 un.	12	9	75			3		3						
		30	31 st.	23	12	52			7	1	8	3				3	0
		30	31 un.	6	1	17			3	1	4	1				1	0
		30	68 st.	14	14	100					0					0	0
		30	68 un.	14	7	50			7		7					0	0
		60	31 st.	22	15	68			3	1	4	1	1		1	3	0
		120	31 st.	9	0	0			4	3	7		1			1	1
		120	70 st.	14	9	64			4		4				1	1	0
		120	70 un.	13	4	31		1	6		7	1		1		2	0
		120	92 st.	19	15	79			3		3	1				1	0
		120	100 un.	19	10	53			1	2	3	2	1		2	5	1
		180	134 st.	14	12	86			2		2					0	0
		180	134 un.	10	5	50	1	1	3		5					0	0
		240	31 st.	8	2	25			3	1	4	1				1	1
		240	31 un.	4	0	0				2	2	1				1	1
51	6	180	4	10	2	20			2	1	3		1		2	3	2
45	6	15	2	15	14	93			1		1					0	0
		30	2	15	12	80			3		3					0	0
		45	30 st.	14	14	100					0					0	0
		60	1	13	10	77			4		4					0	0
		60	10 un.	13	7	54			4	1	5			1		1	0
		60	30 st.	13	9	69		1	3		4					0	0
		60	52 st.	13	10	77		2	1		3					0	0
		90	30 st.	8	5	62			1	1	2		1			1	0
		120	1	10	4	40			1	1	2	1		3		4	0
		120	10 un.	12	6	50			4		4	1			1	2	0
		120	30 st.	13	12	92			1		1					0	0
		120	57 st.	15	13	87			2		2					0	0
		240	10 un.	11	6	55			4		4	1				1	0
		240	30 st.	13	11	85			2		2					0	0
		240	62 st.	15	9	60			6		6					0	0
		480	10 un.	11	6	55			3		3	2				2	0
30	6	60	10 un.	19	15	79			4		4					0	0
25		240	2	23	21	91			2		2					0	0
20		240	2	22	21	95			1		1					0	0
75	39	1	7 un.	8	7	87					0			1		1	0
		10	10 un.	4	3	75					0					0	1
		12	45 st.	4	1	25			3		3					0	0
		12	45 un.	6	4	67			2		2					0	0
		15	45 st.	6	4	67			2		2					0	0
		15	45 un.	12	4	33			8		8					0	0
		30	45 st.	11	7	64			3	1	4					0	0
		30	45 un.	12	5	42			5	1	6				1	1	0
		30	10 un.	4	0	0					0					0	4
		30	10 un.[1]	4	4	100					0					0	0
		30	10 un.[2]	4	0	0			2		2			1		1	1
		60	75 un.	4	2	50			1		1					0	1

[1] Recompressed at once to 15 lbs. for 32 minutes.

[2] Recompressed to 15 lbs. for 37 minutes 18 minutes after decompression.

un. = uniform decompression : st. = decompression by stages.

The following tables give in the simplest form the experimental evidence on certain points which are of especial importance.

(I) *Experiments showing that a certain minimum pressure is required to give symptoms in goats, and that the results vary with the pressure.*

TABLE XI.

Pressure in lbs. positive	Exposure in minutes	Decompression in minutes	No. of goats	No symptoms	Bends	Severe symptoms	Death
20	240	2	22	21	1	0	0
25	240	2	23	21	2	0	0
30	60	10 uniform	19	15	4	0	0
45	60	10 ,,	11	7	3	1	0
60[1]	45	15 ,,	4	1	3	0	0
75	15	31 ,,	36	19	13	3	1
75[2]	50	10 ,,	4	0	0	0	4

[1] Experiment in Series I: compression 30 minutes, exposure 30 minutes.
[2] Series I: compression 40 minutes, exposure 30 minutes.

These experiments show that the effects become more severe as the pressure increases although the duration of exposure was at the same time diminished and the duration of decompression increased. It was necessary to arrange the experiments in this way to prevent an inconvenient mortality among the animals.

(II) *Experiments showing that the duration of exposure to high pressures is of great importance.*

TABLE XII.

Pressure 75 lbs. positive, reached in 6 minutes.

Exposure in minutes	Decompression in minutes	No. of goats	No symptoms	Bends	Severe symptoms	Death
1	1	6	6	0	0	0
3	1	5	4	0	1	0
6	1	6	6	0	0	0
10	1	7	6	0	1	0
15	10 uniform	7	2	3	1	1
15	31 stages	34	29	5	0	0
30	31 ,,	23	12	8	3	0
60	31 ,,	22	15	4	3	0
120	31 ,,	9	0	7	1	1
240	31 ,,	8	2	4	1	1

These experiments show that goats have taken up enough air in 15 minutes to give severe symptoms on decompression in 10 minutes, while, if the exposure is less than 10 minutes, nearly all the animals escape, even with sudden decompression. Note too that with short

exposures and rapid decompressions such symptoms as appear are more frequently severe, and that bends are proportionately less common than with longer exposures and slower decompressions. Beyond 15 minutes exposure the results are somewhat irregular, but on the whole there is a progressive increase of bad symptoms up to two hours exposure. The results after four hours exposure are about the same, but the animals used (see Table VI, Series II) were to a large extent selected by previous experiments, so that it would appear that goats are practically saturated in about three hours[1].

TABLE XIII.

Pressure 45 lbs. positive.

Exposure in minutes	Decompression in minutes	No. of goats	No symptoms	Bends	Severe symptoms	Death
15	1	15	14	1	0	0
30	1	15	12	3	0	0
60	1	14	10	4	0	0
120	1	10	4	2	4	0
60	10 uniform	11	7	3	1	0
120	10 ,,	11	6	4	1	0
240	10 ,,	11	6	4	1	0
460	10 ,,	11	6	3	2	0

These figures show that with a duration of exposure up to about three quarters of an hour, no severe symptoms follow even sudden decompression. The series with sudden decompression shows that the results after two hours are much worse than after one hour. This is not clear from the series with 10 minutes decompression, which, however, show that the results do not become distinctly worse even after

[1] The following figures have been compiled from the records of Helier, Mager and von Schrötter as illustrating the saturation time for dogs of about 10 (?) kilos. The corresponding data for other animals do not seem to have been determined. Pressure 62—69 lbs., compression in 5—16 minutes, decompression ½ to 1 minute.

Exposure minutes	Number of experiments	No symptoms	Mild paralysis and bends	Lasting paralysis	Paralysis and asphyxia	Asphyxia
Less than 10	1	1	0	0	0	0
10—29	6	5	1	0	0	0
30—59	12	0	6	2	3	1 (lived)
60—120	19	2	2	2	3	10

Four of the group "paralysis and asphyxia" died, and the other two would probably have died if they had not been killed. All but one in the "asphyxia" group died, but in none of the rest was the decompression immediately fatal. These results seem to show pretty clearly that dogs require more than an hour to become saturated. It is strange that the authors conclude (*Luftdruckerkrankungen*, p. 806) that saturation is so far complete in about 38 minutes *in man* that no further intake of nitrogen is of any practical importance.

eight hours exposure. Note that of the nine severe symptoms, five were temporary and four permanent paralyses: only one case of dyspnoea was seen in the whole of the experiments at 45 lbs. (Series III) and the one case of severe illness of obscure nature was suggestive of temporary local cerebral anaemia. At 75 lbs., out of 26 severe cases, four had dyspnoea, four permanent and 12 temporary paralysis, and six indefinite: seven died.

(III) *Experiments to show that the duration of decompression is of great importance.*

TABLE XIV.

Pressure 75 lbs. positive, reached in 6 minutes.

Exposure	Decompression	No. of goats	No symptoms	Bends	Severe symptoms	Death
15	10 uniform	7	2	3	1	1
15	31 ,,	36	19	13	3	1
15	90 ,,	12	9	3	0	0
30	31 stages	23	12	8	3	0
30	68 ,,	14	14	0	0	0
120	31 ,,	9	0	7	1	1
120	92 ,,	19	15	3	1	0

(IV) *Experiments to show that the absolute range of pressure through which decompression occurs may be of less importance than the relative range of absolute pressure.*

TABLE XV.

Pressure in lbs.+	Exposure in minutes	Decompression to lbs.	Fall of pressure in lbs.	Relative reduction of absolute pressure	Duration of decompression in minutes	No. of goats	No symptoms	Bends	Severe symptoms	Death
75	180	+24	51	2·3 : 1	1½	10	10	0[1]	0	0
51	180	0	51	4·4 : 1	4	10	2	3	3	2
45	120	−6	51	6·7 : 1	6	3	0	1	1	1
39	120	−6	45	6·0 : 1	6	4	1	0	3	0
45	120	0	45	4·0 : 1	1	10	4	2	4	0

[1] There were three cases of bends at the ultimate end of a two hours' decompression.

A sudden drop of about 50 lbs. from 75 lbs. positive to 27 or 24 lbs. positive has been made about 200 times altogether in the course of these experiments without producing any symptoms, and about two-thirds of the animals showed no symptoms at the end of the stage decompression. The animals were however only left a short time at 27 lbs. before proceeding with the further decompression. In the

present series the animals were left for one hour at 24 lbs. and watched very carefully, and afterwards suddenly decompressed to 17 lbs. and again observed for half an hour. The same goats were subsequently dropped suddenly from + 51 lbs. to atmospheric pressure with very disastrous results, and a drop of 51 lbs. from + 45 lbs. to − 6 lbs. was even worse. The details of these experiments are given in Appendix III. Owing to the cooling effect of rapid decompression, the falls from + 45 and + 39 to − 6 lbs. were interrupted by a delay of about two minutes at atmospheric pressure so that they were in a rough way stage decompressions.

(V) *Experiments showing the importance of the mode and spacing of decompression.*

The next table shows in brief the results of seven groups of experiments undertaken with the purpose of directly testing the results of stage decompression in comparison with those of uniform decompression in the same total time. The only exceptions to the parallelism of the experimental conditions are (1) in group ζ the time of uniform decompression was extended from 92 to 100 minutes in order that it might correspond to the supposed safe rate of 20 minutes an atmosphere[1]; and (2) in group β stage decompression, three animals were decompressed by stages in an abnormal way (see Table VI and note, Series II); since these stages were certainly not more favourable to the animals than those used for the rest of the group, we have included the results.

In considering these results it must be clearly understood that the stage decompressions used were not in most cases intended to be safe for the particular exposure to which they were attached. The only two groups which were intended to be safe (δ and η) gave fairly satisfactory results; with 30 minutes exposure (+ 6 minutes compression) at 75 lbs. and 68 minutes stage decompression, we obtained no illnesses in 14 goats, and with three hours exposure and two and a quarter hours stage decompression only two cases of bends in the same number of animals. For comparative purposes it was desirable that the stage decompressions should produce symptoms of some kind, and they were intentionally designed so to do in so far as our knowledge allowed[2].

[1] The details of the experiments in this group are given in Appendix III.

[2] The stage decompressions from 45 lbs. pressure are likewise all shorter than what we calculate to be safe. The stoppages are also imperfectly spaced. The proper spacing and duration of stoppages could not be calculated till the results of the experiments were known, and we realised the extreme slowness of saturation and desaturation.

TABLE XVI. *Showing the comparison between the results of stage and uniform decompression. Pressure 75 lbs. positive.*

A. *All experiments.*

Group	Exposure minutes[1]	Decompression, minutes	Decompression, method	Number of animals	Number of decompressions	No symptoms	Per cent.	Bends: Doubtful	Bends: Slight	Bends: Bends	Bends: Bad	Bends: Total	Temporary paralysis	Various general	Paraplegia	Dyspnoea	Total severe	Death
α	12–30	45	stages	18	22	12	55			9	1	10					0	
β	15	31	,,	18	34	29	85		2	2	1	5					0	
γ	30	31	,,	15	23	12	52			7	1	8	3				3	
δ	30	68	,,	14	14	14	100					0					0	
ε	120	70	,,	14	14	9	64			4		4				1	1	
ζ	120	92	,,	19	19	15	79			3		3	1				1	
η	180	134	,,	14	14	12	86			2		2					0	
Total					140	103	74	0	2	27	3	32	4	0	0	1	5	0
α	12–30	45	uniform	19	32	14	44			15	1	16		1		1	2	
β	15	31	,,	18	36	19	53	2	3	8		13		1	2		3	1
γ	30	31	,,	6	6	1	17			3	1	4	1				1	
δ	30	68	,,	14	14	7	50			7		7					0	
ε	120	70	,,	13	13	4	31		1	6		7	1		1		2	
ζ	120	100	,,	19	19	10	53			1	2	3	2	1		2	5	1
η	180	134	,,	10	10	5	50	1	1	3		5					0	
Total					130	60	46	3	5	43	4	55	4	3	3	3	13	2

B. *Experiments on identical animals.*

β	15	31	stages	18	34	29	85		2	2	1	5					0	
γ	30	31	,,	6	6	4	67			2		2					0	
δ	30	68	,,	14	14	14	100					0					0	
ε	120	70	,,	13	13	9	69			4		4					0	
ζ	120	92	,,	19	19	15	79			3		3	1				1	
η	180	134	,,	10	10	8	80			2		2					0	
Total					96	79	82	0	2	13	1	16	1	0	0	0	1	0
β	15	31	uniform	18	36	19	53	2	3	8		13		1	2		3	1
γ	30	31	,,	6	6	1	17			3	1	4	1				1	
δ	30	68	,,	14	14	7	50			7		7					0	
ε	120	70	,,	13	13	4	31		1	6		7	1		1		2	
ζ	120	100	,,	19	19	10	53			1	2	3	2	1		2	5	1
η	180	134	,,	10	10	5	50	1	1	3		5					0	
Total					94	46	49	3	5	28	3	39	4	2	3	2	11	2

[1] Group α compressed in 39 minutes; the rest in 6 minutes or, with long exposures, in 39 minutes and half the time of compression deducted from the actual time of exposure.

The results are given in two forms: (*A*) shows the fate of all the animals tested to obtain the direct comparison between stage and uniform decompression, while in (*B*) the figures are confined to the effects (in the same experiments) on animals which were exposed to both stage and uniform decompression in each group. This emendation removes the only very severe symptom and three out of four of the temporary paralyses caused by stage decompression. Goat XXI (Series II) was advancing in pregnancy and, after having nearly died as the result of quick stage decompression, was excluded from the experimental troupe; the effect of the corresponding uniform decompression on this animal can therefore be only surmised. The effects of 31 minutes stage decompression after 30 minutes exposure were so bad that, not wishing at this stage to risk losing any animals, the parallel experiment with uniform decompression was limited to the more resistant animals. In group β (*B*) two of the animals were only decompressed once by stages. One had died from uniform decompression, and the other had broken a leg and had to be killed.

The figures show that the ratio of animals showing no symptoms with stage decompression to those escaping after uniform decompression in the same total time is about eight to five. Be it noted too that the difference between the two methods is in the same sense, *i.e.* in favour of stage decompression, in each of the seven groups, including group α (Series I) where the stages were less well arranged than afterwards. The difference between the two methods appears still more strikingly in the quality than in the quantity of the symptoms produced. For while but one animal had symptoms which can be called distinctly severe after stage decompression, as many as eleven were materially ill after the corresponding uniform decompressions, and one died.

This difference may perhaps obtain more definite expression if we assign numerical values to the different symptoms. Making bends = 1, temporary paralysis = 2, and so on up to death = 6, we obtain the following results, showing a ratio of nearly five to one (*B* grouping) in favour of stage decompression:—

Group	Stages	Uniform
β	5	30
γ	2	6
δ	0	7
ϵ	4	13
ζ	5	26
η	2	5
Total	18	87

This method is of course very rough. "Death" is worth more than six times "bends," and bends should have different values according to the sort of experiment. Bends arising from short exposures and relatively rapid decompressions (*e.g.* group β) indicate that the exposure has been long enough to allow material saturation and are very significant, while if bends show merely the extreme slowness with which the tissues in which they arise get rid of the excess gas (*e.g.* group η), they are of much less moment.

If we exclude bends, and count only the more serious symptoms, or death, the comparison becomes still more striking, the ratio being then two for stage decompression, as compared with 50 for uniform decompression.

(VI) *Experiments illustrating the difference between different kinds of animals.*

(1) Five goats (XXIV A, XXVI A, XXVII A, 7 and 9), 10 small guinea-pigs (175 to 275 gms., average 230 gms.), 9 mice (average 20 gms.), 12 small rats (average 35 gms.), 9 medium rats (average[1] 85 gms.), 8 large rats (average[1] 200 gms.), and 4 rabbits (1285, 1450, 1850, 2850 gms.) were compressed to 72 lbs. in 7 minutes and left at that pressure for 3 more minutes and decompressed in 50 seconds. Goat 9 had a curious short seizure and rolled over on the ground 10 minutes after decompression; it seemed alright immediately afterwards and showed no after effects. One small rat became paraplegic at once, and two other small rats were found dead next morning; one of these had bubbles in the heart. The rest of the animals showed no symptoms. The incidence of illness on the young rather than on the old rats is curious in view of the demonstration of the general immunity of young animals by Hill and Greenwood[2]: it was perhaps correlated with the shortness of the exposure.

(2) Twelve small rats, 13 medium rats, 8 large rats, 59 mice, 7 rabbits, 10 guinea-pigs, and 1 old hen were raised to 72 lbs. in 10 minutes, left for 1 hour and then decompressed in 50 seconds. No goats were put in since it was well established that this experience would have killed all of them. The hen and the largest rabbit (weight 2800 gms.) died in 5 minutes, and 1 guinea-pig became paraplegic in 10 minutes and died in 20 minutes. All three were extensively

[1] The details were eaten by a goat. All the animals were about the same size.

[2] *British Medical Journal*, June 22nd, 1907, Supplement, p. 408.

bubbled; it is interesting to note that there were no bubbles in the avascular eggs of the hen. None of the other animals showed any symptoms.

(3) Five rabbits, 10 guinea-pigs, 23 mice, 10 small, 9 medium and 6 large rats were compressed to 51 lbs. in 7 minutes, left there for 2 hours 56 minutes and decompressed in 45 seconds. In similar experiments, out of 10 goats 2 died and only 2 escaped without symptoms (see above, p. 398). The largest rabbit, a very fat animal weighing 2·9 kilos, died 9 minutes after decompression: the rest showed no symptoms.

(4) Six goats (3, XI A, XII A, XXI A, XXIV A, XXVII A), 7 guinea-pigs and six rabbits were raised to 75 lbs. in 5½ minutes, left for 15 minutes and decompressed in 42 seconds. Goat XXVII A had dyspnoea and paraplegia and was found dead next morning: XXIV A had temporary paralysis of both hind legs without dyspnoea and was quite recovered in an hour: 3 and XXI A had bends, while XI A and XII A showed no symptoms. None of the small animals were affected.

(5) Seven goats (3, X A, XI A, XII A, XXI A, XXIV A, XXVII A), 7 guinea-pigs, 5 rabbits, 7 medium and 12 large rats and 37 mice were compressed to 75 lbs. in 6 minutes, left 10 minutes and decompressed in 48 seconds. Goat X A had paraplegia. The other goats and the small animals showed no symptoms.

(6) Guinea-pigs, mice and rats were compressed with ourselves to 30 lbs. in 15 minutes, and 1 guinea-pig, 1 mouse, 1 medium and 1 large rat were killed with chloroform after 33 minutes. After decompression in 26 minutes by stages, many bubbles were found in the heart and vessels of the guinea-pig, a few in the mouse's heart, a few in the great vessels of the large rat, but none in the medium sized rat.

7. *Individual variation among the experimental animals in their susceptibility to decompression symptoms.*

The variation in the individual susceptibility of different goats is very marked. The same variation has been noted constantly among both divers and caisson workers, and is apparent in most of the published animal experiments. As an example, the following figures have been extracted from the tables of experiments at 75 lbs. All four animals were males and very similar to one another in all obvious respects: two were resistant and two susceptible.

TABLE XVII.

Exposure	Decompression	XIII (17·8 kg.)	X (16·4 kg.)	2 (16·2 kg.)	XV (16·8 kg.)
15 mins.	31 mins. uniform	0	0	slight bends	paraplegia
15 ,,	31 ,, ,,	0	0	paraplegia	0
15 ,,	31 mins. stages	0	0	0	slight bends
15 ,,	31 ,, ,,	0	0	bends	0
30 ,,	31 ,, ,,	0	bends	pain, temporary paralysis	bends
60 ,,	31 ,, ,,	bends	0	0	0
120 ,,	70 mins. uniform	0	bends	paraplegia	bends
120 ,,	70 mins. stages	0	0	bends	bends

In all, therefore, goats X and XIII showed mild symptoms three times in 16 decompressions, while in the same experiments goats 2 and XV showed symptoms 11 times, and on 4 occasions these were of a severe character.

It might be supposed that this variation was only in the exhibition of symptoms, depending on individual susceptibility to pain, &c., and did not represent a variation in the amount and distribution of bubbles within the body. But post-mortem experience shows that the amount of bubbling present in two animals killed in the same experiment may be very different; and in living animals it is clear that on the whole susceptibility to bends involves susceptibility also to the more severe symptoms, which cannot be much altered by the temperament of the animal.

The complete explanation of this individual variation in susceptibility probably requires a knowledge of the details of caisson disease far beyond that which we at present possess. Data exist, however, on which the influence of several factors may be discussed.

(A) *Influence of sex.* The following table shows the sum of the results of the experiments grouped according to the sex of the animals. The groups defined as "selected" include only those experiments in which the animals examined were approximately representative: in Series II for example the figures given are the totals of those experiments in which 10 or more animals were examined, while in Series III and IV are summed only those observations which included both sexes about equally (Series III, expts. 1, 2, 5—8, 10—17, Series IV, expts. 18—20).

It would appear from this that there is no clear difference between the sexes in liability to decompression symptoms in general. The experiments suggested however that under certain circumstances there might be a marked difference in the susceptibility to death. In

Series I, of 5 deaths, 3 were in females, a distribution of fatalities corresponding to the numbers of the sexes (males 12, females 16) used, while in Series II are shown 1 death in 7 males and 4 deaths in 11 females. All these last four animals were to some degree advanced in pregnancy, and their mortality is very probably to be associated with this condition, which, in the goat, is accompanied by a marked increase in the subcutaneous and intra-abdominal fat. That the deaths in Series I did not fall more heavily upon the females is perhaps to be correlated with the fact that these experiments were made in the summer and none of the goats were found pregnant, while the autopsies of Series II showed that in the winter practically every female is pregnant.

TABLE XVIII.

	Males				Females			
	Number	Decompressions	Illnesses	Per cent.	Number	Decompressions	Illnesses	Per cent.
Series I:								
Total	12	78	25	32	16	71	35	49
Series II:								
Total	7	84	42	50	11	91	38	42
Selected	7	64	26	41	11	79	27	34
15 mins. exposures	7	28	9	32	11	42	13	31
1 and 2 hrs. exposures	7	25	11	44	8	24	10	42
Series III and IV:								
Selected	7	108	26	24	8	113	29	26
Total	26	270	93	34	35	275	102	37

Influence of size. In the same way the influence of size on susceptibility may be examined. In the next table the animals are grouped as above and below the average weight for each sex.

TABLE XIX.

	Above average weight			Below average weight		
	Decompressions	Illnesses	Per cent.	Decompressions	Illnesses	Per cent.
Series II:						
Selected	55	22	40	80	26	32·5
15 mins. exposure	27	9	33	39	9	23
1 and 2 hrs. exposure	20	9	45	26	12	46
Series III and IV:						
Selected	102	19	19	119	36	30
Total	157	41	26	199	62	31

The results are contradictory unless (which appears hardly possible) there is an essential difference between exposures to high (75 lbs.) and to low (45 lbs.) pressures. The sums of the whole show no material difference between large and small goats. Theoretically, with decompressions of moderate length such as were used in the experiments under consideration, small goats should be somewhat more susceptible than large goats with short exposures since they should saturate more quickly in proportion to their relatively greater gaseous exchange[1]. This is not borne out by the experiments, in which however the rate of decompression may not have been quick enough to bring out the difference. It is, on the other hand, obvious that the larger goats should be more susceptible after long exposures with any except very short or very long decompressions: this is confirmed by the experiments at 75 lbs. (Series II), but those at 45 lbs. (Series III) show a greater difference in the opposite sense.

In comparing the incidence according to sex with those arranged according to weight, it will be noted that in Series II the males are somewhat more susceptible though they are rather smaller, while the same experiments, arranged by weights, show that the heavier animals suffer more frequently. In Series III, in which the male group is again composed of smaller animals, the susceptibility of the sexes is equal, while the lighter animals are more susceptible if weight be taken as the criterion. The only conclusion to be drawn is that these figures do not indicate that either sex or weight was a determining factor in the incidence of decompression symptoms.

Influence of the activity of gaseous exchange. General considerations suggest rather strongly that the susceptibility would be found to vary with the activity of gaseous exchange, directly as regards short exposures and inversely as regards long exposures. In most of our experiments, especially those of Series III, the incidence of symptoms has been conditioned rather by the mode of decompression than by the duration of exposure. As a whole, then, the goats with the most active exchange should prove to be the least susceptible.

The respiration results already given have been analysed in reference to this point: the results are variable and inconclusive and need not be detailed. This is perhaps not very remarkable when we consider that the animals were not grouped for the respiration experiments according

[1] The respiratory activity per unit of body weight, being proportional to the ratio of surface to mass, would of course vary but little in the goats, and would only be about a fourth greater in a goat of 15 kilos than in one of 30 kilos.

to their susceptibility but by the bands into which they had been marshalled for the pressure experiments. A factor of considerable importance, which is to a large extent beyond control, is the activity of the goats at the moment. Some goats are naturally vivacious while others are almost constantly lethargic. These individual idiosyncrasies are no doubt of some moment in relation to susceptibility, but the customary habits of a group of animals may be altogether upset by an incompatible companionship in the chamber during an experiment.

One group of measurements gave for example the results shown in the following table. The CO_2 production of each group was determined on four separate occasions under conditions similar to those obtaining in the pressure experiments.

TABLE XX.

Females.						Males.					
No. of goat	Weight kilos	Expts.	Illnesses	Pressure lbs.	CO_2 gms. per kilo per hour	No. of goat	Weight kilos.	Expts.	Illnesses	Pressure lbs.	CO_2 gms. per kilo per hour
XII A	14·2	14	4	0	0·921	3	19·2	15	3		
XV A	15·5	17	8	45	1·104	4	20·0	16	1	0	0·669
XVIII A	31·4	17	2	45	1·020	X A	17·8	17	6	45	0·697
XXI A	17·0	17	11	45	0·852	XI A	23·0	17	2	25	0·959
XXIII A	22·0	13	0			XVI A	22·6	16	4	45	0·967
						XIX A	25·3	17	7		
Average	20·0	78	25 (32 %)	—	0·974		21·3	98	23 (23 %)	—	0·823

The average size in each group is about the same, and the sex incidence for all goats of Series III is the same. The results therefore appear to show that the males are 32 % less susceptible and produce 17 % less CO_2—a result which cannot be correlated with theory.

The only experiments made with the animals grouped according to their susceptibilities gave much more rational results. Great care was taken in this series to make the conditions as nearly identical as possible in all four observations; the animals were kept in the dark and remained quite quiet throughout. The results show a CO_2 production by the susceptible animals one-sixth less than that of the non-susceptible.

Influence of blood volume. The volume of the blood was determined in 8 goats, in 7 of which the susceptibility to caisson symptoms had been ascertained. The method used was the simple one of Welcker, in which, after taking a standard sample of arterial blood, the animal is bled to death and then thoroughly washed out with salt solution. The

TABLE XXI.

Non-susceptible goats.

Animals							Respiration					
			Total		Selected						CO_2 gms. per hour	
No. of goat	Sex	Weight	Decomp.[1]	Ill	Decomp.	Ill	No. of expt.	Temp.	Bar. m.m.	Duration in hours	per kilo	per 1000 sq. cms.
4	M	18·6	15	0	10	0						
XI A	M	20·8	16	1	11	1	I	12°	776	5	0·717	1·853
XVIII A	F	30·7	14	0	10	0	III	14°	775	5	0·751	1·941
XXIII A	F	25·2	12	0	8	0						
Average		23·8	57	1 (2 %)	39	(2½ %)					0·734	1·897
Susceptible goats.												
XV A	F	15·0	15	6	10	4						
XXI A	F	15·4	15	8	10	6	II	14°	775	5	0·624	1·501
XIX A	M	21·3	15	7	10	5	IV	13°	766	5	0·704	1·693
XIII A	F	25·2	16	5	11	2						
Average		19·2	61	26 (43 %)	41	17 (41 %)					0·664	1·597

[1] These figures are given up to the date at which the respiration experiments were made. Some time elapsed before the final susceptibilities were ascertained: these were 8 % for the non-susceptible group and 45 % for the susceptible animals.

tissues were not afterwards extracted with water: the red colouring matter so obtained is so small in amount that it can have little influence on the final result, and Douglas[1] has shown that additional difficulties are thereby introduced. For purposes of calculation the specific gravity of the blood has been taken as 1050. The results and the decompression records of the goats are given in the two following tables. The figures should be read in relation to the "clean" weight, *i.e.* the crude weight less the weight of the contents of the alimentary canal, which in these animals is very considerable.

The results seem to indicate that there are two types of blood volume in goats: one about 7½ % of the clean body weight and the other about 6½ %, the first type being also associated with a higher percentage of haemoglobin. No relation between blood volume and susceptibility is apparent; thus goats 2 and XIII, both males, have identical blood volumes and differ about as widely in their susceptibility as any two goats which have come under our notice.

Conclusions. Of the four factors considered in detail, it appears therefore that age, sex and blood volume were without appreciable influence. Pregnancy and a low rate of respiratory exchange seem to favour the occurrence of symptoms.

[1] *Journal of Physiology*, vol. XXXIII. (1906), p. 499.

TABLE XXII.

No. of goat	Sex	Whole weight kilos	Weight of contents of stomach and intestines: Total kg.	Weight of contents of stomach and intestines: Per cent. of whole weight	Volume of blood c.c.	Mass of blood per cent. of: Whole weight	Mass of blood per cent. of: Clean weight	Haemoglobin p.c. of human standard [1]
1	M	19·9	3·8	19·1	1006	5·31	6·56	74
XIII	M	18·9	4·3	22·7	883	4·91	6·36	—
2	M	18·0	3·7	20·5	874	5·10	6·42	64
X	M	17·0	3·0	17·6	833	5·14	6·25	72
XVI	M	11·3	2·3	20·3	592	5·50	6·91	64
A	F	31·2	5·2	16·7	1874	6·31	7·57	78
XVIII	F	27·1	4·6	17·0	1395	5·41	6·47	75
XVII	F	24·4	4·0	16·4	1520	6·54	7·83	84
Average		21·0	3·9	18·8	1122	5·53	6·80	73
Average of males		17·0	3·4	20·0	838	5·19	6·50	68·5
Average of females		27·6	4·6	16·7	1596	6·09	7·29	79

[1] The red blood corpuscles of goats are very small, about 4μ in diameter (Jolly, *C. R. Soc. de Biol.*, vol. LXIII (1907), p. 210).

TABLE XXIII.

Pressure 75 lbs.

Exposure minutes	Decomp. minutes	Goat 1	2	X	XIII	XVI	XVII	XVIII
15	30 un.	0	slight bends	0	0	bends	0	slight bends
15	30 un.	0	para-plegia	0	0	0	0	0
15	30 st.	0	0	0	0	0	0	0
15	30 st.	bad bends	bends	0	0	0	0	0
30	30 st.	bends	temp. paral.	bends	0	bends	bends	0
60	30 st.	bends	0	0	bends	0	dyspnoea	0
120	70 un.	bends	para-plegia	0	0	bends	temp. paral.	0
120	70 st.	0	bends	bends	0	0	0	0
15	1	—	—	—	—	bends	—	—
15	10 un.	—	—	bad bends	0	bends	—	—
30	30 un.	—	—	—	—	bends	—	bends
30	30 st.	0	—	0	0	temp. paral.	—	bends
60	30 st.	0	—	0	0	0	—	obscure
120	30 st.	bad bends	—	bad bends	bends	bends	—	bad bends
240	30 st.	—	bad bends	bends	0	temp. paral.	—	bends
240	30 un.	—	—	temp. paral.	—	—	—	—

But there are doubtless other particulars which, alone or in combination, are of fundamental importance. Such other factors we have not yet been able to examine in detail. Fatness for example can be gauged only in the dead, and, though we have the distinct impression that the goats which die easily (*i.e.* under circumstances of pressure, exposure and decompression which cause very few severe symptoms and deaths) are fatter than those which are killed with difficulty, we have had no means of extending our observations on this head to the great majority of our animals[1]. Fatness also involves a low rate of respiratory exchange per unit body weight. There are grounds in human experience for holding that age may have an important share in the production of symptoms and Hill and Greenwood have recently shown[2] clearly that young animals (rats, rabbits and cats) are far less susceptible than adults of the same species. All the goats used by us appeared to be adult; in the two cases (XIII A, XXXII A) in which old age had obviously set in, the susceptibility seemed to be somewhat above the average, but the ages of the animals as a whole were unknown. In any case such a factor as old age must be reduced to simpler components before it can be correlated with the theory of decompression.

8. *The pathology of caisson disease in goats.*

We have hitherto dealt exclusively with the symptoms exhibited by the experimental animals rather than with the actual or possible presence of bubbles within them. We have however made a number of observations on the post-mortem appearances of goats after decompression, which may be shortly dealt with here. Most of the animals had died from caisson disease but in other instances they were killed at varying periods after decompression.

The presence of bubbles *in vivo* must be inferred from their discovery post-mortem with considerable caution. The supersaturation of the body may be such that the separation of the gas as bubbles may take place after death. There are reasons for supposing that the living body presents nothing in the way of points or surfaces on which bubbles might arise in the blood and tissues as they do upon the glass and dust in soda-water, and a remote analogy may perhaps be drawn

[1] We have since examined this point by direct analysis of rats and guinea-pigs divided into susceptible and non-susceptible groups by decompression experiments. The results, which will be published in detail later, show that fatness is a very important factor in individual susceptibility to death.

[2] Meeting of Physiological Society, Nov. 1906.

with the relations of the vessels to the separation of fibrin *intra vitam*. Death may well alter this condition in some degree, and in any case the time factor is of importance as well as the foreign manipulation which examination involves. During life no portion of the blood remains in a supersaturated state for more than the time required for it to return from the tissues to the lungs. We have already seen that the duration of this period is probably of very great importance as regards risk of bubble formation. On cessation of the circulation the blood remains in a supersaturated state for an indefinite period. In one instance at least we have actually observed such post-mortem separation of bubbles: a rabbit was killed immediately after 75 lbs., 2 hours, 31 minutes stages, opened up at once and no bubbles found[1]; an hour later a few bubbles were found in the inferior *vena cava*. In another case bubbles in the bladder were seen to increase considerably in number and volume during the progress of the examination.

The possibility of air being introduced from without into the veins must also be considered. Ewald and Kobert showed that air might be forced into the pulmonary capillaries by an increase of intra-pulmonary pressure such as may occur in severe dyspnoea. We have seen bubbles in large quantities in the meningeal veins, and in small numbers in the superficial veins of a fore-foot, under circumstances which left no reasonable doubt that they had been sucked into the vessels during the somewhat violent manipulations used in opening the skull and skinning the leg respectively.

There is not much doubt that some of our animals which showed no symptoms must have had bubbles present in the blood. Catsaras decompressed a dog in 1 minute after 2 hours exposure at 65 lbs.: it showed no symptoms and was killed 6 hours later, when fine bubbles were found in the blood. Heller, Mager and von Schrötter (pp. 790, 882) record two dogs which were killed 10 minutes after sudden decompression after 65 and 52 minutes at 15 and 18 lbs. respectively: in both cases bubbles were found in the heart[2], though there is abundant evidence to show that dogs never show any symptoms after decompression from such low pressures. In our own animals attempts were made to see bubbles in the retinal vessels during life. Though an

[1] The vessels and bladder in the rabbit are so thin-walled that bubbles can be seen with certainty if they are present.

[2] On the other hand dogs (showing no symptoms) killed after sudden decompression after 16 minutes exposure at 2·8 atmospheres, 5+ at 3·5, 12 at 4·5 and 5 at 4·7, showed no bubbles in the blood: these were however found in three other dogs after 10+ minutes at 4·0 atmospheres, 16 and 72 at 4·5.

excellent view of the fundus may easily be obtained, no bubbles were ever seen even in animals with severe dyspnoea, so that the method cannot be taken as giving any indication of the absence of bubbles in the blood. Some of these animals died and plenty of free gas was found in the retinal vessels post-mortem. Four animals which showed no symptoms were killed within ten minutes after decompression with the following results:

TABLE XXIV.

Series and number of goat	Pressure lbs.	Exposure (actual) minutes	Decompression minutes	Bubbles in blood	Results of similar experiments in other goats			
					Number	Bends	Severe symptoms	Death
3:XXVI A	25	120	½	absent	23	2	0	0
1:II	45	26	6	absent	15	3	0	0
2:XIV	75	19	31 stages	present	29	5	0	0
1:1	78	30	9	absent	4	0	0	1

These goats were killed before the expiration of the appropriate period for the development of bends. The experience of similar experiments indicates that they might have shown symptoms if they had not been killed. Yet three out of four had no bubbles in the blood. A few observations on rabbits on the other hand gave rather different indications. Seven rabbits in seven different experiments were exposed to 75 lbs. for periods of from 15 to 120 minutes and killed immediately after decompression in 31 minutes by stages. There is no question but that it would be the very rarest occurrence for a rabbit to have any symptoms under these circumstances, but in four of the animals we found bubbles in the heart or great veins. These may however have been formed post-mortem, and in one such case they were observed to appear some time after death.

The post-mortem appearances observed may now be shortly described. It should be remembered that most of the animals dealt with here died under circumstances of experiment less severe than those of other observers. The pressure was in almost all cases 75 lbs. and in a majority of instances the decompression was not instantaneous. These considerations probably afford the explanation of the somewhat less emphatic changes which we have noted. The naked eye appearances were in nearly every case supplemented by microscopical examination of many of the organs.

Lungs. The amount of blood in the lungs depends upon the condition of the heart: in severe cases, with the pulmonary artery choked with bubbles and the right heart distended, they are pale and bloodless; in other instances the quantity of blood appears about normal.

Haemorrhages may occur and small blood clots are not infrequently found in the trachea. A very marked, scattered, lobular emphysema is almost constant; the only explanation appears to be that some bronchioles are more or less impervious during decompression. Examination of fresh material showed that bubbles may burst out of the capillaries into the interstitial tissue of the lungs, and presumably therefore also into the alveoli. The same was found in rabbits which were killed by the injection of small quantities of air into the veins. Nothing resembling the exudative process seen in oxygen poisoning was ever found in animals exposed to simple air pressures.

In all fatal cases (with one exception) more or less abundant bubbles were found in the blood. In severe, rapidly fatal cases, the right heart is much distended with bubbles, the pulmonary vessels plugged with froth and the left heart nearly empty. The block in the pulmonary artery may indeed be so complete that the left auricle is collapsed and puckered up. In other animals, which have lived for 20, 30 or 60 minutes or longer, the two sides of the heart are equally full of blood and the right heart is not distended. The immediate cause of death, in all but three cases, which died many hours after decompression, was clearly pulmonary air embolism, and this is doubtless the cause of the urgent dyspnoea already noted. In two cases death ensued without any dyspnoea. Both these animals were intentionally killed by very severe experiments, viz. 75 lbs. for 1 hour (goat XXI) and 3 hours (XVIII A) with decompression in $1\frac{3}{4}$ and 1 minutes. Both showed no symptoms for 5 minutes and collapsed and died quietly in 10 and 12 minutes respectively. Such animals must be regarded as being so overwhelmed by sudden asphyxiation that they exhibit only the symptoms of deficiency of oxygen and not those of the accumulation of carbon dioxide.

The fatal case in which no bubbles were found in the blood post-mortem was in goat XXVII A. After 75 lbs., 15 minutes, 42 seconds it showed pain, ate part of a note-book, and became paraplegic. It was found dead next morning[1]. In several other cases of delayed death, and in one (XVI A) in which the animal died in 3 hours[2], the quantity of bubbles found seemed to be altogether inadequate to produce a fatal result. One may suppose that they had been previously more

[1] Heller, Mager and v. Schrötter (p. 852) record a case in a dog fatal in 6 hours after decompression in which no free gas was found in the vessels.

[2] This animal had however been recompressed and died under a state of recompression: see protocol *e*, Appendix III.

numerous and that oxygen starvation resulted in death at a time when the aeration of the blood had been restored. This form of delayed death from deficiency of oxygen is well-known in *e.g.* carbon monoxide poisoning. It might be for instance that temporary obstruction of the coronary arteries or portal capillaries caused fatal degenerative changes in the heart muscle or liver cells.

The distribution of the bubbles in the different parts of the vascular system shows several peculiarities. If only a few are present, all may be collected in the smaller branches of the pulmonary artery with none in the right heart. The left heart generally contains a few bubbles; the amount there and in the arteries roughly varies inversely with the rapidity of death unless decompression has been very quick. Smallness in the amount of bubbling affords the heart the best chance of being able to pass on the froth to the arteries, and cases which die slowly seem to show distinctly more arterial bubbles than those which expire almost at once. The veins contain variable quantities of bubbles, but always more than the arteries. They are especially abundant in the mammary, mesenteric, spermatic and portal veins, coronary vessels, and notably few in the veins on the surface of the stomach and in those of the brain and spinal cord. In several instances we have noticed great accumulations of froth in the liver while the spleen at the same time showed no bubbles in the blood flowing out on section. This massive portal embolism is probably the cause of the multiple small capillary haemorrhages which are frequently seen in the omentum and mesentery. Blocking of the portal circulation might also give rise to general symptoms of a very serious character.

It should be noted that the liver is particularly badly situated for getting rid of excess gas during and after decompression; nearly all the blood reaching it is already partly saturated by passing through the intestines, &c. The liver also contains much fat.

Lymph. The lymph in the thoracic duct has been noted to be full of froth on several occasions.

Other liquid areas. Bubbles have very seldom been found in the *aqueous humour*; the blood supply is considerable, so that their absence is probably to be attributed to the excess gas being carried off during decompression and the period which the supersaturation phenomenon adds, for practical purposes, to the actual time occupied in reducing the pressure to normal. The *vitreous humour*, on the other hand, has a poor blood supply and its consistence is such that any bubbles forming there would remain *in situ*. On only one occasion have bubbles been found

(goat XXVI, 75 lbs., 2 hours, 31 minutes stages), when they were seen in a layer close against the ciliary body. Their absence is explicable on the ground that the vitreous humour would take a very long time to saturate. The *bile* often contains bubbles: they were noted in one goat exposed at 75 lbs. for 15 minutes and killed immediately after decompression in 31 minutes stages, and in 8 animals exposed to the same pressure for 1—4 hours, but not in two animals exposed for 15 minutes which died 30 minutes after decompression in 10 minutes and 30 minutes uniformly respectively. The *urine* found in the bladder post-mortem is remarkably free from bubbles; on two occasions only has free gas been found. We have evidence here that the phenomenon must be due to supersaturation and the absence of "points," since we have very frequently observed goats pass urine after decompression which frothed freely on coming into contact with foreign surfaces. This is often seen in animals which show no symptoms. Thus in one experiment, seven goats were exposed at 45 lbs. for one hour and decompressed in 30 seconds. One had bends 19 minutes later. Within 24 minutes after decompression four animals passed urine; in two cases this frothed up freely as it ran over the pavement while in the other two (including the goat which had bends) no bubbles could be observed. It is somewhat striking to observe the transparent bladder of the rabbit containing urine quite free from bubbles while the vesical veins coursing over its surface are full of froth. The *cerebro-spinal fluid* rarely shows any bubbles: they have been seen only three times, in all cases after long exposures (1 to 3 hours) at 75 and 51 lbs. with sudden decompression. *Synovial fluid* is almost always full of bubbles; exposure for 15 minutes at 75 lbs. is sufficient to cause their presence, while decompression in 100 minutes uniformly is not enough to prevent their formation. In animals which have died within 3 hours of decompression, we have found them in every case. *Amniotic fluid* is dealt with below. Bubbles have been seen after very severe experiments in the *pericardial* and *peritoneal fluids* when present, and in the serous contents of a mammary cyst, but not in the milk. We have not seen any accumulations of gas in the serous cavities.

Solid organs. Fat commonly shows bubbles, often in extreme abundance. They are more numerous in the abdominal than in the subcutaneous fat; the latter is much more vascular. Other solid organs for the most part show no bubbles outside the blood vessels; a few are sometimes found in the liver and the spinal cord may contain large numbers. In the liver it is very difficult to determine whether any

bubble is inside a capillary or not, and we have failed to find clear evidence of bubbles outside blood-vessels, still less actually within tissue cells, in cardiac or skeletal muscle, spleen, kidney, suprarenal, salivary glands, thymus, thyroid and parathyroid, pancreas, lymphatic glands, haemolymph glands, nerves, posterior root ganglia, testis, ovary or mammary gland, though an extensive systematic histological examination has been made of more than 20 goats exposed at 75 or 45 lbs. for from 10 minutes to 4 hours and decompressed in from 30 seconds to 100 minutes.

This condition has no very obvious explanation. It is curious, for instance, to see the spermatic vein (and sometimes artery as well) full of froth and yet find no evidence of bubbles in the tissues which it drains: the same thing is also shown most strikingly in the mammary gland and vessels. There can be no doubt that these tissues must be fairly completely saturated in 4 hours and it is impossible that the excess should be removed from the tissues more quickly than from the blood. It follows that the blood must stand in an unfortunate relation towards bubbling in that it effervesces with a smaller difference of pressure, within and without, or with the same difference of pressure in a shorter time, than do the more solid organs. It seems unlikely that this difference depends on the motion of the blood. Rhythmical pulsating circulation through a smooth elastic system can hardly function as a shaking which would be efficient in bringing out free gas. Even if it did, it is not easy to see why the tissues are not affected by the pulsations in the same way, though perhaps not to the same degree, since isolated collections of fluid, as in the joints, may bubble very easily. One can only suppose that the dissolved particles of gas find in the tissues obstacles, visible and invisible, more obstructive to their aggregation into bubbles than those occurring in the blood.

Bubbles once formed in the blood will also increase in size more readily, since their movement will continuously keep them in contact with fresh portions of supersaturated liquid.

Among the solid organs, bubbles outside the vessels are found most frequently in the central nervous system. The fatty nature of this tissue is probably important in this respect. The brain is singularly free, both by direct examination and by the study of secondary degenerations. The cord may however contain numerous bubbles, and a study of their occurrence and distribution gives interesting results. In the first place they may occur in areas of softening after comparatively mild experiments (*e.g.* 45 lbs., 2 hours, 10 minutes): in this case

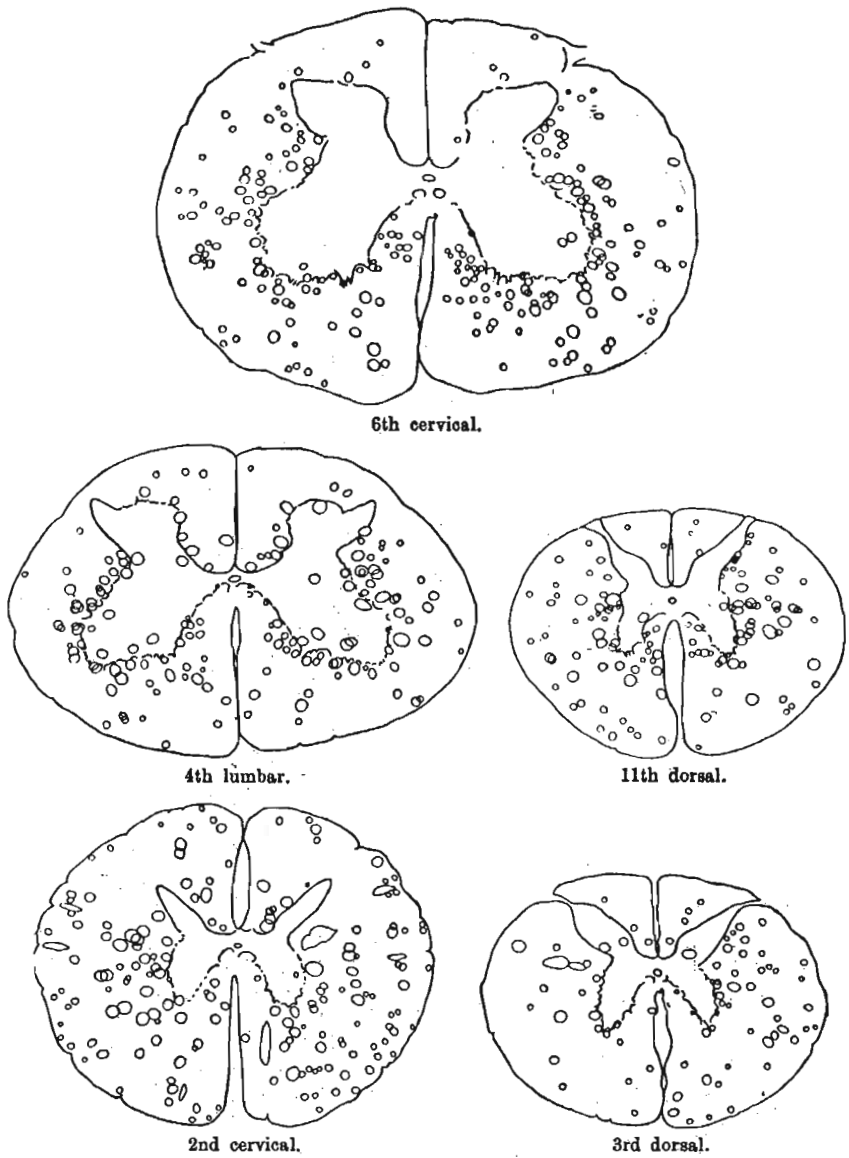


Fig. 7. Shows the distribution of extravascular bubbles in five regions of the spinal cord of goat 3 (series IV). The animal died of oxygen poisoning soon after the beginning of a decompression of 133 minutes duration by stages after 3 hours exposure at 81 lbs. in an atmosphere containing 36 % oxygen. The bubbles are practically confined to the white matter and are there especially concentrated in the boundary zone where the circulation is least good. Each diagram is a composite drawing showing all the bubbles in 0·4 mm. length of cord.

they are confined to the area in which the circulation has been brought to a sudden standstill by a collection of gas. On the other hand they may be found in the cords of animals which have died immediately after a drastic decompression. This is however rather exceptional. Thus the cords of three animals decompressed in less than a minute after 1 hour at 100 lbs. and 1 and 2 hours at 75 lbs. contained numerous bubbles, while in two animals treated in the same way after exposure at 75 lbs. for 1 and 3 hours respectively, none were found.

The distribution of the bubbles when numerous is in harmony with the theoretical conclusions derived from the blood supply. They are for instance least numerous in those segments with an abundant blood supply (lumbar enlargement), and are almost confined to the white matter, those found in the grey substance being distributed along its periphery towards the boundary zone between the superficial and deep vessels. Thus one cord (goat XXI: 75 lbs., 1 hour, 1¾ minutes) contained the following bubbles in 412 cubic millimetres in different parts:

Segment	Grey matter	Posterior columns	Ant.-lat. columns	White matter	Total
2nd cervical	14	141	215	197	175
5th dorsal	11	23	95	87	79
1st lumbar	0	32	161	140	140
4th lumbar	2	15	37	28	19
Average	7	53	127	113	103

Fig. 7 shows the distribution of bubbles in another case: note the paucity of bubbles in the grey matter and their concentration in the boundary zone.

The distribution of the areas of softening is also important. With one exception, these are most marked in, and usually confined to, the lower dorsal and upper lumbar segments where the blood supply of the cord may on many grounds be surmised to be at its minimum. They affect only the white matter. Now the only parts of the body in which we have found appearances resembling embolic infarction are the white matter of the spinal cord and the fat. The latter has on several occasions been found to contain large and small areas of necrosis. We have obtained no evidence of infarction of the spleen, kidney, heart-muscle, &c. The distribution of small bubbles by the arterial blood stream must be universal. They probably lodge in many places: while they are rapidly pushed forward in the grey matter and in most other tissues, if they lodge among the fatty surroundings of the capillaries of the white matter, or in actual fat, they quickly increase in size to such

an extent that their removal becomes impossible. It is also clear that in consequence of the slow circulation in the white matter, and especially in such inactive parts as the lower dorsal segments, bubbles have plenty of time to increase in size in the circulating blood. The condition of supersaturation will also last much longer in the white than in the grey matter. The cause then of these areas of softening in the cord is not ordinary embolism, but embolism which becomes effective to produce infarction by reason of the effect on the size of the embolus of the local conditions of the circulation rather than from any of those peculiarities in the resistance of the different tissues to lack of oxygen, or in the freedom of collateral circulation, which determine the topography of common infarcts.

The presence of bubbles in the uterine contents. We may group together here a number of casual observations which have been made on the distribution of bubbles in the foetus and amniotic fluid of pregnant goats dying of caisson disease. The pressure was in all cases 75 lbs.

TABLE XXV.

Number of goat	Exposure minutes	Decompression minutes	Time of death after decompression minutes	Bubbles present in the maternal blood	Bubbles present in the foetus	Bubbles present in the amniotic fluid	Development of foetus
XXIII	15	31 stages	30	+	0	0	advanced
XXII	120	31 ,,	75	+	0	+	,,
XX	240	31 ,,	40	+	live + dead 0	+ +	6 inch 4 inch
XXI	60	1¾	10	+	+	+	advanced
XI	240	31 uniform	25	+	0	0	1 inch
XIV	15	31 stages	killed at once	+	0	0	½ inch
XVIII A	180	1	12	+	+	+	advanced
XXVIII A	180	4¼	24	+	0	0	½ inch
XXXII A	180	4	27	+	0	0	4 inch (dead)

These observations seem to be fairly concordant. In 15 minutes the uterine contents have not taken up much excess of gas (XXIII), nor does a dead foetus absorb any (XX, XXXII A). In one hour both foetus and amniotic fluid have taken up abundant excess (XXI), which may, if death be long delayed after a rather slow decompression, be discharged from the foetus more quickly than from the amniotic fluid (XXII)[1]. With a very young foetus, the circulation is probably too

[1] Two pregnant guinea-pigs were exposed for 1 hour at +100 lbs. and decompressed in 34 minutes by stages: they showed no symptoms. On being killed 5 hours later, numerous bubbles were found in the amniotic fluid but nowhere else.

active and the bulk of fluid too small to favour bubbling (XI, XIV, XXVIII A).

The amniotic fluid, which contained in this case only a faint trace of proteid, may show the phenomenon of supersaturation to an exquisite degree. In goat XXI it was especially noted that a large bubble was present in the amniotic fluid on removing the uterus from the body. After free shaking to bring out any more gas, the uterus was opened and the contents poured into a glass vessel. Contact with this foreign surface immediately produced a great froth of fine bubbles.

The free gas runs together into one large bubble. Advantage was taken of this convenient circumstance in two instances to make analyses. The samples were collected over water and in XXII analysed at once; in XX they were kept for 20 hours over water before examination and in this case therefore the figures for CO_2 represent minimal and those for oxygen maximal values.

	XXII	XX Live foetus	XX Dead foetus
Total gas c.c.	16	27	10·5
CO_2 per cent.	16·23	3·55	2·73
O_2 "	1·10	2·14	0·85
N_2 "	81·90	94·14	96·21
Combustible gas (calculated as CH_4 and H_2)	0·77	0·17	0·21

These results correspond with those of Bert (pp. 955, 961) of the free gas in the heart: they are not in accord with those of Heller, Mager and von Schrötter (p. 800) who found 15·31 and 7·18 per cent. of oxygen in the free gas collected from the hearts of dogs. It is somewhat significant that if this excess of oxygen is calculated as an air leak, the figures of Schrötter correspond exactly with those of Bert and our analysis XXII.

Duration of bubbles. It is difficult to say how long bubbles may remain in the vessels and tissues after their first formation in animals which survive[1]. The question is much complicated by the fact that we have reason to believe that bubbles may continue to form for a long, and quite unknown, time after decompression. This is probably especially marked in cases in which either local blocking of the blood supply has occurred, or the circulation has been slowed generally by a greater or less degree of cardiac and pulmonary obstruction. It would

[1] Zografidi (*Revue de Médecine*, 1907, p. 159) records the finding of numerous bubbles in the peripheral vessels, but not in the heart, of a diver who was paralysed and died 33 days after decompression!

appear likely that bubbles once lodged in the lungs would probably stop there for a considerable time, since their gaseous composition would quickly approximate to that of the alveolar air and there would be no considerable difference of tension to encourage their removal. Such results as we have which bear on the matter are collected in the next table. It will be seen that bubbles have been found in the blood of one animal which died two days after decompression (and that in an animal which had shown no dyspnoea) and in the joints up to 26 hours. In the substance of the spinal cord bubbles may persist far longer: in two cases we have found them 15 days after the last exposure to pressure and in one 27 days after the last occurrence of symptoms.

TABLE XXVI.

Goat	Pressure lbs.	Exposure minutes	Decompression minutes	Result hours	Bubbles present in	
					Blood	Joints
XXII (Series I)	75	35	40 stages	died 39	+	–
XXV A	75	120	100 uniform	,, 16½	+	0
XXVII A	75	15	¼	,, 15*	0	0
XV A	45	120	½	killed 24	0	0
4	45	120	½	,, 26	0	+
XIII A	45	120	10 uniform	,, 72	0	0
X A	75	10	¼	,, 96	0	0
XXIII A	45 to –6	180	6	,, 144	0	0

* Found dead next morning.

A histological point of some practical importance arises in connection with the size of the bubbles found in the blood. The bubbles soon run together into large bullae after death so that it is necessary to make the examinations immediately after death in order to observe approximately the true state of affairs. It will then be found that there are no bubbles so small as to be of strictly microscopic dimensions. Nor are any very small bubbles found in the spinal cord; in any one case all the bubbles are about the same diameter, commonly some 25 microns. The same is true of the bubbles given off on decompressing water, salt solutions, serum, blood, and even such thick solutions as gelatine or agar. At the same time however it is possible to produce bubbles which are truly microscopic and which last some hours in some sticky solutions such as gum and treacle. The energy required to aggregate particles of dissolved gas into a bubble is evidently considerable, and there is the same difficulty in the formation of free gas bubbles from solution in liquids as there is in the separation of liquid particles from solution in gases and of solid particles from solution in liquids.

Extremely minute bubbles are unstable in the same way as extremely minute droplets of water condensing from supersaturated air, or salt crystals forming in a supersaturated solution in water: in all cases the tendency is to reduce the free energy (surface tension) by reducing the ratio of surface to mass, and accordingly the smallest bubbles, droplets or crystals as the case may be, are rapidly, in the case of bubbles practically instantaneously, abolished to produce macroscopic masses. This is well seen on watching under the microscope a stream of bubbles coming off some "point" in soda water. It follows that if the concentration of dissolved molecules of gas is not higher than some unknown point, bubbles will not be formed. It is possible that the absence of bubbles from most of the solid tissues is to be explained by this non-existence of very small bubbles and the mechanical difficulties of the rapid aggregation of a sufficient number of molecules to produce large bubbles. It is also doubtless connected with the period of delay in bubble formation whereby an animal, for practical purposes, gains several minutes over the actual time of decompression.

It is reasonable to suppose that the temporary paralyses are due to temporary ischaemia from air bubbles in the vessels. The more lasting palsies are undoubtedly caused by obstruction sufficiently complete to produce softening and necrosis. As already mentioned, the change is confined to the white matter and in nearly all instances affects only the lower dorsal or upper lumbar region. In these segments the bulk of cord destroyed may be very extensive: thus in goat XXIII A fully three-quarters of the lateral columns were destroyed from the eighth dorsal to the second lumbar segment over a length of rather more than five inches, and in goat XV A the softening involved nearly the whole of the lateral columns and parts of the anterior and posterior columns for a length of four and a half inches. Such cases may recover to a remarkable degree, and eventually show objective signs of paralysis so slight as to be hardly perceptible except to those familiar with the individual animals.

The only other tissue in which we have found any signs of the results of circulatory obstructions is the intra-abdominal fat. Large masses of necrosed fat have been occasionally met with, especially in the fat lying below the kidneys. Only late stages have been seen: the necrosed areas are then surrounded by a well-marked ring of giant-cell reaction, and the surface layers are mostly converted into a calcium soap.

No evidence of infarction in other organs has been seen: the rarity

of such changes seems to afford pretty good evidence that the duration of any obstruction in such organs as the spleen or kidney cannot be very long.

The pathological changes underlying the chief symptoms have been already sufficiently noticed except as regards bends. The exact cause of bends is not known. They have been attributed to bubbling in the central nervous system, chiefly on the ground that human experience shows that they are very frequently bilaterally symmetrical. This fact however cannot be taken as indicating any such origin in view of the complete symmetry of the limbs (where the symptoms occur) and the uniform symmetry of the causative agent throughout the body. In two animals which were killed soon after decompression when they showed bends only, we could find nothing abnormal in the cord, posterior root ganglia, or nerves, and there is abundant evidence from a number of goats that the cause of bends does not produce such lesions in the nervous system as are followed by secondary degeneration which can be revealed by the methods of Marchi or Weigert. The two following goats may be cited in detail as to this point: in neither was any degeneration found. Goat XXI (Series II) was used in seven experiments between November 26th and January 18th: it had bends on December 5th, 11th and 18th (the last being noted as "bad bends"), and dyspnoea, nearly fatal, on January 2nd: it was killed on January 18th. Goat XV A (Series III) was exposed 27 times between February 2nd and June 10th: it had bends on February 2nd, 20th, 22nd, March 3rd, 5th, May 13th, 15th, 27th and June 6th: it was killed on June 11th. Thorough examination of the pons, medulla and cord showed no secondary degeneration in either animal. There are therefore reasons for thinking that the cause of bends is peripheral rather than central. The constant presence of bubbles in the joints has been already mentioned, and they seem to afford a fairly probable explanation of most of the cases. Even in those cases in which the muscles are the seat of pain, it is quite possible that a sensation actually originating in or around the joints is referred to other parts. The joint pains in man are often relieved by flexion, and goats evidently try to obtain ease in the same way (see Plate VI). This fact adds strong confirmation to the conclusion that the origin of the pain is in or about the joints.

We have already seen that bends, while not the first symptom to appear as the duration of exposure to high pressure is increased, are the last symptom to disappear as the decompression is extended, that bends in short arise in parts of the body which saturate and desaturate

rather slowly. The synovial fluid satisfies this criterion; on the other hand the tendons and other dense tissues about the joints are not in disagreement with it.

Bends occur with a lower degree of supersaturation with air than any other symptom of compressed-air illness. In goats they are readily produced after exposures to 30 lbs. or less. The fact that only a moderate degree of supersaturation is needed to produce them seems to explain the fact that although they are not the first symptom to appear as the duration of exposure to very high pressure is increased, yet a moderate duration of exposure suffices to produce them, in spite of the fact that they occur in parts of the body with a slow rate of circulation, as shown by the fact that they are the last symptom to disappear as the duration of decompression is prolonged.

Summary.

1. The time in which an animal or man exposed to compressed air becomes saturated with nitrogen varies in different parts of the body from a few minutes to several hours. The progress of saturation follows in general the line of a logarithmic curve and is approximately complete in about five hours in man and in a goat in about three hours.

2. The curve of desaturation after decompression is the same as that of saturation, provided no bubbles have formed.

3. Those parts of the body which saturate and desaturate slowly are of great importance in reference to the production of symptoms after decompression.

4. No symptoms are produced by rapid decompression from an excess pressure of 15 pounds, or a little more, to atmospheric pressure, *i.e.* from two atmospheres absolute to one. In the same way it is safe to quickly reduce the absolute pressure to one-half in any part of the pressure scale up to at least about seven atmospheres: *e.g.* from six atmospheres (75 pounds in excess) to three (30 pounds), or from four atmospheres to two.

5. Decompression is not safe if the pressure of nitrogen inside the body becomes much more than twice that of the atmospheric nitrogen.

6. In decompressing men or animals from high pressures the first part should consist in rapidly halving the absolute pressure: subsequently the rate of decompression must become slower and slower, so that the nitrogen pressure in no part of the body ever becomes mor-

than about twice that of the air. A safe rate of decompression can be calculated with considerable accuracy.

7. Uniform decompression has to be extremely slow to attain the same results. It fails because it increases the duration of exposure to high pressure (a great disadvantage in diving work), and makes no use of the possibility of using a considerable difference in the partial pressure of nitrogen within and without the body to hasten the desaturation of the tissues. It is needlessly slow at the beginning and usually dangerously quick near the end.

8. Decompression of men fully saturated at very high pressures must in any case be of very long duration: and to avoid these long decompressions the time of exposure to such pressures must be strictly limited. Tables are given indicating the appropriate mode and duration of decompression after various periods of exposure at pressures up to 90 pounds in excess of atmospheric pressure.

9. Numerous experiments on goats and men are detailed in proof of these principles.

10. The susceptibility of different animals to compressed-air illness increases in general with their size owing to the corresponding diminution in their rates of circulation.

11. The average respiratory exchange of goats is about two-thirds more than that of man; they produce about 0·8 gram. of CO_2 per hour per kilogramme of body weight.

12. The mass of the blood in goats is six and a half or seven and a half per cent. of the "clean" body weight.

13. The individual variation among goats in their susceptibility to caisson disease is very large. There is no evidence that this depends directly on sex, size or blood-volume: there is some evidence that fatness and activity of respiratory exchange are important factors.

14. Death is nearly always due to pulmonary air-embolism, and paralysis to blockage of vessels in the spinal cord by air. The cause of "bends" remains undetermined; there are reasons for supposing that in at least many cases they are due to bubbles in the synovial fluid of the joints.

15. In our experiments bubbles were found post-mortem most freely in the blood, fat and synovial fluid; they were not uncommon in the substance of the spinal cord, but otherwise were very rarely found in the solid tissues.

APPENDIX I.

Details of the experiments made on Lieutenant Damant and Mr A. Y. Catto, Gunner, R.N., in the pressure chamber at the Lister Institute.

These experiments were undertaken in July, 1906, as a preliminary to actual diving experiments in very deep water.

In the first three or four the decompression was controlled from inside the chamber; in the rest from outside. The subjects remained closed in the chamber for half an hour after each experiment, the engine being also kept running so that recompression could be at once begun if any serious symptom developed. In addition to the actual period of exposure to each pressure, we have noted the virtual period of exposure calculated on the assumption that about half the time occupied in compression must be added (see above, p. 362).

In view of the results with goats, the occurrence of decompression symptoms seemed probable in the more severe experiments. No symptoms were, however, observed, except considerable itching of the skin of the fore-arms where it was uncovered. In the compressed air the well-known alteration in the voice, and corresponding abnormal sensations about the lips and mouth, were very marked at pressures exceeding 60 or 70 lbs.

I. July 25th. Actual exposure to 39 lbs. for one hour. Virtual exposure 69 minutes, decompression in 24 minutes:

Compressed to	39 lbs.	in 17	minutes.
Waited at	39 ,,	for 60	,,
Decompressed to	9 ,,	in 7	,,
Waited at	9 ,,	for 5	,,
Decompressed to	4 ,,	in 1	,,
Waited at	4 ,,	for 9	,,
Decompressed to	0 ,,	in 2	,,

24.

II. July 26th. Actual exposure to 50 lbs., 27 minutes. Virtual exposure, 39 minutes. Started at 10.37 a.m. Decompression in 34 minutes:

Compressed to	50 lbs.	in 24	minutes.
Waited at	50 ,,	for 27	,,
Decompressed to	17 ,,	in 4	,,
Waited at	17 ,,	for 6	,,
Decompressed to	13 ,,	in $1\frac{1}{2}$	,,
Waited at	13 ,,	for $3\frac{1}{2}$	,,
Decompressed to	9 ,,	in 2	,,
Waited at	9 ,,	for 3	,,
Decompressed to	4 ,,	in 2	,,
Waited at	4 ,,	for 8	,,
Decompressed to	0 ,,	in 4	,,

III. Same day, 3.3 p.m. Exposure to 55 lbs. for 19 minutes. Virtual 33 minutes. Decompression in 31 minutes:

Compressed to	55 lbs.	in 28	minutes.
Waited at	55 ,,	for 19	,,
Decompressed to	17 ,,	in 4	,,
Waited at	17 ,,	for 5	,,
,, ,, ...	13 ,,	,, 5	,,
,, ,, ...	9 ,,	,, 5	,,
,, ,, ...	4 ,,	,, 10	,,
Decompressed from	4 to 0 ,,	in 2	,,

The time taken for decompressing from 17 to 13 lbs., &c., was counted as time at 13 lbs.

IV. July 27th, 10.29 a.m. Exposure to 60 lbs. for 20 minutes. Virtual exposure 36 minutes. Decompression in 37½ minutes:

Compressed to	60 lbs.	in 30½	minutes.
Waited at	60 ,,	for 20	,,
Decompressed to	22 ,,	in 5	,,
Waited at	22 ,,	for 5	,,
Decompressed to	17 ,,	in 1	,,
Waited at	17 ,,	for 4	,,
Decompressed to	13 ,,	in 1½	,,
Waited at	13 ,,	for 3½	,,
Decompressed to	9 ,,	in 1	,,
Waited at	9 ,,	for 4	,,
Decompressed to	4 ,,	in 1½	,,
Waited at	4 ,,	for 8½	,,
Decompressed to	0 ,,	in 2½	,,

V. Same day, 3.37 p.m. Exposure to 67 lbs. for 18 minutes. Virtual exposure 36 minutes. Decompression in 36 minutes:

Compressed to	67 lbs.	in 36	minutes.
Waited at	67 ,,	for 18	,,
Decompressed to	22 ,,	in 3	,,
Waited at	22 ,,	for 5	,,
Decompressed to	17 ,,	in 1	,,
Waited at	17 ,,	for 4	,,
Decompressed to	13 ,,	in 1	,,
Waited at	13 ,,	for 4	,,
Decompressed to	9 ,,	in 1	,,
Waited at	9 ,,	for 4	,,
Decompressed to	4 ,,	in 1½	,,
Waited at	4 ,,	for 8½	,,
Decompressed to	0 ,,	in 3	,,

VI. July 30th, 10.57 a.m. Actual exposure at 74 lbs., 15 minutes. Virtual exposure 35 minutes. Decompression in 42 minutes:

Compressed to	...	...	...	...	74 lbs.	in	39 minutes.
Waited at	...	...	...	...	74 ,,	for	15 ,,
Decompressed to	...	...	...	...	26 ,,	in	4 ,,
Waited at	...	...	...	...	26 ,,	for	5 ,,
Decompressed to	...	...	...	...	22 ,,	in	1 ,,
Waited at	...	...	...	...	22 ,,	for	4 ,,
Decompressed to	...	...	...	...	17 ,,	in	1½ ,,
Waited at	...	...	...	...	17 ,,	for	3½ ,,
Decompressed to	...	...	...	...	13 ,,	in	1 ,,
Waited at	...	...	...	...	13 ,,	for	4 ,,
Decompressed to	...	...	...	...	9 ,,	in	1 ,,
Waited at	...	...	...	...	9 ,,	for	4 ,,
Decompressed to	...	...	...	...	4 ,,	in	1½ ,,
Waited at	...	...	...	...	4 ,,	for	8½ ,,
Decompressed to	...	...	...	...	0 ,,	in	3 ,,

VII. July 31st, 11.0 a.m. Actual exposure to 80 lbs. for 12 minutes. Virtual exposure, 34 minutes. Decompression in 51 minutes:

Compressed to	...	...	...	...	80 lbs.	in	44 minutes.
Waited at	...	...	...	...	80 ,,	for	12 ,,
Decompressed to	...	...	...	...	31 ,,	in	3 ,,
Waited at	...	...	...	...	31 ,,	for	5 ,,
Decompressed to	..	...	...	...	22 ,,	in	1 ,,
Waited at	...	...	...	...	22 ,,	for	4 ,,
Decompressed to	...	...	...	...	18 ,,	in	1 ,,
Waited at	...	...	...	...	18 ,,	for	4 ,,
Decompressed to	...	...	...	...	15 ,,	in	3 ,,
Waited at	...	...	...	...	15 ,,	for	2 ,,
Decompressed to	...	...	...	...	13 ,,	in	1 ,,
Waited at	...	...	...	...	13 ,,	for	4 ,,
Decompressed to	...	...	...	...	9 ,,	in	1 ,,
Waited at	...	...	...	...	9 ,,	for	9 ,,
Decompressed to	...	...	...	...	4 ,,	in	2 ,,
Waited at	...	...	...	...	4 ,,	for	8 ,,
Decompressed to	...	...	...	...	0 ,,	in	3 ,,

APPENDIX II[1].

A diary of the deep diving experiments carried out off Rothesay, Isle of Bute, from H.M.S. *Spanker*, August, 1906.

Monday, 20th August.

H.M.S. *Spanker* arrived at Rothesay about 7 p.m., and was met by Drs Haldane and Rees and Mr Catto, Gunner, R.N. Arrangements were made to commence experiments the following day.

Tuesday, 21st August.

All the pumps to be used in the experiments were tested up to a pressure of 200 feet, and the leakage at this pressure measured. The pressure gauges, which had been specially graduated for these experiments, were tested and found to give correct readings. The method of testing employed was to attach the free end of the diving hose to a lead line, and lower it over the side into the sea to the required depth. The pumps were then hove round until there was a free supply of air, and then stopped whilst the reading of the gauge was taken.

The re-compression chamber was tested on the Whitehead torpedo charging column, and it was found that the pressure could be brought up to 40 lbs. on the gauge in 3 minutes. There was a leak of 1 lb. per minute, or, roughly 3 cubic feet. Afterwards Drs Haldane and Rees were compressed up to about 30 lbs. in order to further test the working of the chamber.

In the afternoon both divers made a trial dive in 15 fathoms:

	Lieutenant Damant	Mr Catto
Time of descent	2 minutes	1¼ minutes.
,, on bottom	1 hour	1 hour.
,, of ascent	18½ minutes	17½ minutes.
No. 5-minute stops	1 at 30 feet	1 at 20 feet.
,, 10 ,, ,,	1 ,, 10 ,,	1 ,, 10 ,,

Two double pumps were used for each diver in these and the subsequent dives. The divers were perfectly comfortable in moving about on the bottom. It may be mentioned that Lieutenant Damant had not dived previously beyond about 19 fathoms, and had no experience in diving except what he had gained in his course of instruction as a gunnery officer and in experimenting at Portsmouth for the Committee. Mr Catto had much previous experience in diving work, but had never dived beyond 23 fathoms.

[1] Reprinted from the *Report of the Admiralty Committee on Deep Diving*, 1907.

Wednesday, 22nd August,

H.M.S. *Spanker*, off Rothesay.

In the forenoon Mr Catto descended in 23 fathoms, and in the afternoon Lieutenant Damant did the same:

	Mr Catto	Lieutenant Damant
Time of descent	2 minutes	2½ minutes.
,, on the bottom	20 ,,	20 ,,
,, of ascent	35½ ,,	32½ ,,
No. 5-minute stops	4 at 50, 40, 30, 20 feet	2 at 50, 40 feet.
,, 10 ,, ,,	1 ,, 10 feet	2 ,, 20, 10 ,,

Thursday, 23rd August,

H.M.S. *Spanker*, off Rothesay.

After testing the pumps each diver made a descent to 25 fathoms:

	Lieutenant Damant	Mr Catto
Time of descent	2 minutes	2 minutes.
,, on the bottom	18¾ ,,	19¼ ,,
,, of ascent	37¾ ,,	39¾ ,,
No. 5-minute stops	3 at 60, 45, 30 feet	3 at 50, 40, 30 feet.
,, 10 ,, ,,	2 ,, 20, 10 feet	2 ,, 20, 10 feet.

Friday, 24th August,

H.M.S. *Spanker* was taken through the narrows of the Kyles of Bute and anchored off the entrance of Loch Riddon.

In the morning, after the usual tests had been applied to the pumps, Mr Catto descended in 27 fathoms, and in the afternoon Lieutenant Damant went down in a similar depth:

	Mr Catto	Lieutenant Damant
Time of descent	2 minutes	1 minute 20 seconds.
,, on the bottom	16½ ,, ...	16¾ minutes.
,, of ascent	55½ ,, ...	44¼ ,,
No. 5-minute stops	4 at 60, 50, 40, 30 feet	4 at 60, 50, 40, 30 feet.
,, 10 ,, ,,	1 at 20 feet. (Diver was employed just under the ship's bottom in examining a propeller which had been slightly injured, for 19½ minutes before coming up.)	2 ,, 20, 10 feet.

Saturday, 25th August,

H.M.S. *Spanker*, off Loch Riddon.

The *Spanker* shifted her position slightly, and, after the usual tests of the pumps, both divers descended in 29 fathoms of water:

	Mr Catto	Lieutenant Damant
Time of descent	3 minutes	1½ minutes.
,, on the bottom	14½ ,,	13½ ,,
,, of ascent	46 ,,	48½ ,,
No. 5-minute stops	4 at 70, 50, 40, 30 feet	4 at 66, 54, 40, 30 feet.
No. 10 ,, ,,	2 at 20, 10 feet	2 at 20, 10 feet.

Monday 27th August,

H.M.S. *Spanker*, off Loch Riddon.

Thirty fathoms of water were obtained. Mr Catto was the diver in the morning. The pumps used were Nos. 3604 and 3593. Six men were told off for each pump, in reliefs of 5 minutes. Details of the descent:

Time	Remarks
11.22	Glass screwed up. Depth by lead line 30½ fathoms.
11.23¼	Diver under water.
11.23½	,, down 50 feet.
11.23¾	,, ,, 70 ,,
11.24¼	,, ,, 110 ,,
11.24½	,, ,, 150 ,,
11.24¾	,, ,, 180 ,, on the bottom. 1 min. 30 secs. in descending. Revolutions averaged 32 per min., but fell to 24 for a short time, owing to the great exertions that were necessary to keep the pumps going at the higher speed. Diver quite comfortable while moving about on the bottom.
11.36¾	Diver called up.
11.38½	,, started up.
11.39¼	,, at 160 feet.
11.39¾	,, ,, 140 ,,
11.40¼	,, ,, 120 ,,
11.41	,, ,, 100 ,,
11.41½	,, ,, 70 ,, 1st stop. Diver employed in gymnastic exercises. One pump stopped.
11.46½	Diver at 50 feet. 2nd stop.
11.51½	,, ,, 40 ,, 3rd stop.
11.56½	,, ,, 30 ,, 4th stop.
12.1½	,, ,, 20 ,, 5th stop.
12.11½	,, ,, 10 ,, 6th stop. There were no ill-effects. Water jackets gained 20 degrees F.
12.22½	Diver called up.
12.23¾	Glass off.

Afternoon. Lieutenant Damant.

2.14½	Screwed up glass.
2.15¾	Diver under water.
2.16	,, down 70 feet.
2.16½	,, ,, 120 ,,
2.16¾	,, ,, 160 ,,
2.17	,, ,, 186 ,, on the bottom. 1 minute 20 seconds in descending. Revolutions averaged 30 per minute.
2.29	Diver called up.
2.30	,, started up.
2.31	,, at 170 feet.
2.33	,, ,, 120 ,, Diver stopped 1¼ minutes.
2.33½	,, ,, 70 ,, 1st stop.
2.38½	,, ,, 50 ,, 2nd ,,

Time	Remarks
2.43½	Diver at 40 feet. 3rd stop.
2.48½	,, ,, 30 ,, 4th ,,
2.53½	,, ,, 20 ,, 5th ,,
3.3½	,, ,, 10 ,, 6th ,,
3.13½	,, called up.
3.15¼	Glass off. There were no ill-effects. Later in the afternoon the pumps were tested at different temperatures of the water jacket, to see how the leakage was affected.

Tuesday, 28th August.

In the same locality, Lieutenant Damant made a second descent in 30 fathoms in order to obtain samples of the air in the helmet. The pumps used were Nos. 3588 and 3592:

Time	Remarks
10.18½	Diver under water.
10.20¼	,, on the bottom, 1 minute 40 seconds in going down.
10.34½	,, started up.
11.21½	Glass off. Whilst on the bottom, diver took two samples whilst at rest. There was a distinct tide on the bottom, which affected the diver.

Analysis of Samples.

No. of sample	CO_2 per cent.	O_2 per cent.	CO_2 production in cubic feet per minute
1st	·32	20·86	·025
2nd	·50	20·43	·041 (? tide)

In the afternoon Mr Catto was in the dress. Pumps Nos. 3588 and 3592 were used:

Time	Remarks
2.17	Glass screwed up.
2.18¼	Diver down 60 feet.
2.19	,, ,, 100 ,,
2.19¾	,, ,, 180 ,, on the bottom. The diver took down with him a wire hawser to shackle on to a sinker.
2.31¾	Diver called up, but could not come up as he was foul, until—
2.48½	,, started up.
2.50½	,, at 140 feet.
2.53	,, ,, 100 ,, 1st stop.
2.56	,, ,, 80 ,, 2nd ,,
3.1	,, ,, 60 ,, 3rd ,,
3.7	,, ,, 50 ,, 4th ,,
3.12	,, ,, 40 ,, 5th ,,
3.22	,, ,, 30 ,, 6th ,,
3.37	,, ,, 20 ,, 7th ,,
3.52	,, ,, 15 ,, 8th ,,
4.0	,, ,, 10 ,, 9th ,,
4.18½	,, on the surface.

Mr Catto attempted to shackle a hawser on to the sinker. He found the sinker without the slightest difficulty, and then, having tied his distance line to it, went back to the hawser. He found this in bights, and he seems to have got within the coils, and in trying to find the end of the wire to have fouled his life line. When called up he could not get away, and it was 20 minutes before he could clear himself. In all he was down 28¾ minutes in 30 fathoms of water. The rate of the pump could not be kept up above 24 revolutions per minute, and the supply of air was not adequate to his exertions to free himself, so that he was almost overcome by the excess of CO_2. On account of his long exposure during heavy work, great care was taken in decompressing him, 1½ hours being allowed. There were no ill-effects.

Thursday, 30th August,

H.M.S. *Spanker*, off Loch Riddon.

Mr Catto made another descent under the same conditions, and shackled on the hawser to the sinker in 4 minutes after reaching the bottom. The revolution of the pump averaged 24 to 30 per minute. The day was very bright, with the sun shining on the water, so that the diver saw with comparative ease in the water.

In the afternoon Lieutenant Damant, at the same depth, took three samples of the air in the helmet, and the pumps were tested at 180 feet pressure. He suffered from no ill-effects:

	Mr Catto	Lieutenant Damant
Time of descent	3 minutes	1 min. 20 secs.
,, on the bottom	12¾ ,,	13 minutes.
,, of ascent	46¼ ,,	46¼ ,,
No. 5-minute stops	4 at 70, 50, 40, 30 feet	4 at 70, 50, 40, 30 feet.
,, 10 ,, ,,	2 at 20, 10 feet	2 at 20, 10 feet.

Analysis of Samples obtained by Lieutenant Damant.

Per cent.	First sample	Second sample	Third sample
CO_2	·43	·39	·36
O_2	20·56	20·52	20·47
Deficiency of oxygen	·48	·52	·57
CO_2 produced in cubic feet per minute	·035	·029	·027

Friday, 31st August,

H.M.S. *Spanker* moved down to the entrance of Loch Striven, where 35 fathoms of water could be obtained.

In the morning Lieutenant Damant was the diver. Pumps Nos. 2593, 3604 and 3592 were tested and used. Six hands were told off for each pump in reliefs of 5 minutes:

Time	Remarks
11.8	Glass screwed up.
11.8¼	Diver under water.
11.9	,, down 80 feet.
11.9¼	,, ,, 120 ,,
11.9½	,, ,, 150 ,,
11.9¾	,, ,, 180 ,,
11.10¼	,, ,, 200 ,,
11.10½	,, ,, 216 ,, on the bottom. Revolutions kept at 30 per minute, and the diver had a good supply of air.
11.13½	Diver took samples seated on the shot at the bottom of the rope.
11.15½	,, called up.
11.16½	,, started up.
11.17	,, at 190 feet.
11.18	,, ,, 110 ,, Diver stopped to blow off sampling tube.
11.20½	,, ,, 90 ,, 1st stop.
11.23½	,, ,, 70 ,, 2nd ,,
11.28½	,, ,, 52 ,, 3rd ,,
11.33½	,, ,, 42 ,, 4th ,,
11.39½	,, ,, 32 ,, 5th ,,
11.44½	,, ,, 22 ,, 6th ,,
11.54½	,, ,, 11 ,, 7th ,,
12.4½	,, called up.

There was no light on the bottom, which was of soft mud. The depth by the shot rope was 210 feet. Pressure was 93½ lbs. The gauge showed a pressure of 216 feet of fresh water with the pumps stopped, and 220 feet whilst they were heaving. The actual depth, as carefully measured on the shot rope against the ship's standard measure, was just over 35 fathoms, 210 feet.

In the afternoon Mr Catto made the same descent, and reached 35 fathoms. He found that the air supply was more than ample. He walked out to the end of his distance line, and then took a sample of the air in his helmet:

Time	Remarks
2.12	Screwed up glass. Same pumps as last.
2.12¾	Diver under water.
2.14¾	,, on the bottom. Revolutions reduced to 24, as the diver found the supply too much. He proceeded to the end of his distance line before taking his sample.
2.20½	Diver started up.
2.27¾	,, at 90 feet. 1st stop.
2.30½	,, ,, 70 ,, 2nd ,,
2.35¾	,, ,, 50 ,, 3rd ,,
2.40¾	,, ,, 40 ,, 4th ,,
2.45¾	,, ,, 30 ,, 5th ,,
2.50¾	,, ,, 20 ,, 6th ,,
3.0¾	,, ,, 10 ,, 7th ,,
3.10¾	,, called up.

Analysis of Samples.

	Lieut. Damant	Mr Catto
CO_2 per cent.	·14	·53
O_2 ,, ,, ...	20·89	20·34
Deficiency of O_2 per cent	·15	·70

Monday, 3rd September.

Experiments on rest and measured work were carried out, by means of an arrangement of rope and pulleys by which the diver on the bottom raised and lowered a 56 lb. weight suspended in view of those on deck. The heavy rope and blocks used caused great friction and resistance.

Time	Remarks	
2.26	Diver, Mr Catto, descended.	
2.27½	,, on bottom, 142 feet.	
2.31	,, took sample sitting on the shot.	(No. 1.)
	Two pumps at 30 revolutions per minute.	
	Raised the weight 4 times 5 feet, at the rate of one lift per minute.	
2.36	,, took sample.	(No. 2.)
	Raised weight 7 times 5 feet in 5½ minutes.	
2.42	,, took sample.	(No. 3.)
2.45	,, started up.	
3.23	,, on surface, no ill-effects.	

3.3½	,, Lieutenant Damant, started down.	
3.4½	,, down 100 feet.	
3.5	,, on bottom, 139 feet.	
4.0	,, took sample sitting on the shot.	(No. 4.)
	Two pumps at 26 revolutions.	
	Raised weight 5 times in 1¾ minutes.	
4.3	,, took sample.	(No. 5.)
	Raised weight 3 feet 18 times in 6¼ minutes.	
4.10	,, took sample. Pump 24 revolutions.	(No. 6.)
4.13½	,, started up.	
4.52½	,, at the surface. No ill-effects.	

Analysis of Samples.

CO_2 ... ... ·30 per cent. O_2 ... ... 20·72 ,,	Mr Catto.	Sample No. 1.
CO_2 ... ... ·70 ,, O_2 ... ... 20·29 ,,	,,	,, No. 2.
CO_2 ... ... ·71 ,, O_2 ... ... 20·23 ,,	,,	,, No. 3.
CO_2 ... ... ·18 ,, O_2 ... ... 20·73 ,,	Lieutenant Damant.	Sample No. 4.
CO_2 ... ... ·73 ,, O_2 ... ... 20·12 ,,	,,	,, No. 5.
CO_2 ... ... ·81 ,, O_2 ... ... 20·36 ,,	,,	,, No. 6.

Tuesday, 4th September.

The *Spanker* was anchored in six fathoms of water, and experiments were made on the bottom by Dr Haldane, Lieutenant Damant, and Mr Catto on the risks of blowing up. After being compressed in the air chamber to teach them to open their Eustachian tubes, Lieutenant and Commander E. V. F. R. Dugmore, Lieutenant G. N. Henson, Jack Haldane (age 13) all made descents in six fathoms of water. This was the first time that these had ever dived in a diving dress, which illustrates the usefulness of the re-compression chamber in the practical teaching of divers.

Wednesday, 5th September.

Exhaustive tests were made as to the leakage of the pumps and composition of the air, with the water jackets at various temperatures. The results are embodied in the Report. These experiments concluded the work undertaken for the Committee.

APPENDIX III.

We give here some illustrative protocols of certain important animal experiments.

1. *Comparison of stage* (93 *minutes*) *and uniform* (100 *minutes*) *decompression after* 2 *hours exposure at* 75 *lbs.*

(*a*) 15.3.07. Goats 3, 4, X A, XI A, XVI A, XIX A, XXV A.

Started up	10.23	
Reached 75 lbs.	11.3	
Started from 75 lbs.	12.43	Stage decompression. Total = 93 mins.
,, 31½ ,, ...	12.49	
,, 27 ,, ...	12.59	
,, 22 ,, ...	1.14	
,, 18 ,, ...	1.29	
,, 13½ ,, ...	1.44	
,, 9 ,, ...	1.59	
,, 4½ ,, ...	2.14	
Reached 0 lbs.	2.16	

XIX A had bends left hind-leg at 2.20. X A passed urine not frothy at 2.27. Rest nil.

(*b*) 18.3.07. Goats XII A, XIII A, XV A, XVIII A, XX A, XXI A, XXIII A.

Started up	10.53	
Reached 75 lbs.	11.35	
Started from 75 lbs.	1.14	Stage decompression. Total = 93 mins.
,, 31½ ,, ...	1.20	
,, 27 ,, ...	1.30	
,, 22 ,, ...	1.45	
,, 18 ,, ...	2.0	
,, 13½ ,, ...	2.15	
,, 9 ,, ...	2.30	
,, 4½ ,, ...	2.45	
Reached 0 lbs.	2.47	

XX A urine no froth at 2.54. XIII A at 2.55 seemed uneasy in hind-legs and lay down but nothing definite; at 3.0 right hind-leg slight limp and foot-drop; foot-drop very marked at 3.10 and could hardly walk; alright at 4.0. Rest nil.

(*c*) 14.3.07. Goats XXIV A, XXVI A, XXVII A, XXVIII A, XXIX A.

Started up	10.35	
Reached 75 lbs.	11.14	
Started from 75 lbs.	12.55	Stage decompression. Total = 93 mins.
,, 31½ ,, ...	1.1	
,, 27 ,, ...	1.11	
,, 22½ ,, ...	1.26	
,, 18 ,, ...	1.41	
,, 13½ ,, ...	1.56	
,, 9 ,, ...	2.11	
,, 4½ ,, ...	2.26	
Reached 0 lbs.	2.28	

XXIV A showed bends left fore-leg during decompression from 4½ to 0; XXVII A passed urine no froth at 2.32; XXVIII A bends left hind-leg at 2.40. Rest nil.

(*d*) 12.3.07. Goats XXIV A, XXVI A, XXVII A, XXVIII A, XXIX A.

Started up	12.53	
Reached 75 lbs.	1.1	
Started from 75 lbs.	3.0	Uniform decompression. Total = 100 mins.
Reached 60 lbs.	3.20	
,, 45 ,, ...	3.40	
,, 30 ,, ...	4.0	
,, 15 ,, ...	4.20	
,, 0 ,, ...	4.40	

XXIX A bleated at about 1 lb., gnawed side, lay down; both fore-legs completely paralysed and hind-legs unsteady; kept head bent round on left side; bleated continually; no dyspnoea till 4.55, when it was moderate; seemed like to die. At 5.10 no dyspnoea, stopped bleating, could just stand. Could walk at 5.35. XXVII A passed very frothy urine at 4.41; both hind-legs bad bends at 4.50, right fore-leg at 5.10. XXIV A left hind-leg partial foot-drop and bends at 4.50; left fore-leg bends at 5.0. XXVIII A bad bends right fore-leg, won't stand up; at 5.0 could not stand, had constant nystagmus, bleated; at 5.10 left hind-leg bad bends, nystagmus stopped, no bleating; walked very badly at 5.30. XXVI A no symptoms.

(*e*) 19.3.07. Goats 3, 4, X A, XI A, XVI A, XIX A, XXV A.

Started up	10.30	
Reached 75 lbs.	11.14	
Started from 75 lbs.	12.52	Uniform decompression. Total = 100 mins.
Reached 60 lbs.	1.12	
,, 45 ,, ...	1.32	
,, 30 ,, ...	1.52	
,, 15 ,, ...	2.12	
,, 0 ,, ...	2.32	

XVI A came out with bad bends left hind and right fore-legs; could hardly walk and kept head twisted round to left; much better at 2.50. XIX A urine at 2.34 full of froth; bends right fore-leg. X A began bleating at 2.38 but showed nothing till 2.44 when he had complete foot-drop right fore-leg and bends left hind-leg; at 2.50 right fore-leg paralysed, could not stand up, left fore-leg also weak; urine at 2.50 a little froth. XXV A cried out a bit, belly very tight, refuses to move, evidently far from well: died between 8 and 8.30 a.m. next day: a good many bubbles in right heart. Rest nil.

(*f*) 20.3.07. Goats XII A, XIII A, XV A, XVIII A, XX A, XXI A, XXIII A.

Started up	11.5	
Reached 75 lbs.	11.47	
Started from 75 lbs.	1.26	Uniform decompression. Total = 100 mins.
Reached 60 lbs.	1.46	
,, 45 ,, ...	2.6	
,, 30 ,, ...	2.26	
,, 15 ,, ...	2.46	
,, 0 ,, ...	3.6	

A goat unknown aborted two foetuses 2 in. long; they were quite warm when found, so probably during decompression. XXIII A very

frothy urine at 3.8. XIII A dyspnoea, both hind-legs dragging; at 3.25 lying down, moaning bleat, tongue and lips getting cyanosed, dyspnoea not violent. Made sure it was going to die, but at 3.55 it got up and showed only bends right fore-leg and weakness in both hind-legs. At 5.0 seemed all right. Rest nil.

(2) *The effects of a sudden drop of* 51 *lbs. in different parts of the scale of absolute pressure.*

(*a*) 26.3.07. Goats XXIV A, XXVI A, XXVII A, XXVIII A, XXIX A.

Started up	10.0	
Reached 75 lbs.	10.46	
Left 75 lbs.	1.23	
Reached 24 lbs.	1.24½	
Left 24 lbs.	2.25	
,, 14 ,, ...	2.55	
,, 8 ,, ...	3.18½	
Reached 0 lbs.	3.21½	Total = 118½ mins.

No symptoms during decompression. XXVIII A passed frothy urine at 3.23; at 3.31 had bad bends, evidently very uneasy generally; better at 4.0. XXIX A urine no froth at 3.31. Rest nil. (The immediate object of the experiment having been attained, an unwise quickening of the end of decompression gave XXVIII A bad bends.)

(*b*) 23.5.07. Goats 7, 9, XXX A, XXXII A, XXVII.

Start up	9.55	
Reach 75 lbs.	10.35	
Left 75 lbs.	1.15	
Reach 24 lbs.	1.15.40″	
Left 24 lbs.	2.15	
,, 14 ,, ...	2.45	
,, 8 ,, ...	3.5	
,, 4 ,, ...	3.25	Total = 131 mins.

No symptoms during decompression. 7 limped right hind-leg on coming out; urine 3.30 no froth. 9 right hind-leg bends at 3.35. XXXII A urine 3.35 no froth. Rest nil.

(*c*) 27.3.07. Goats XXIV A, XXVI A, XXVII A, XXVIII A, XXIX A.

Started up	11.0
Reached 51 lbs.	11.30
Left 51 lbs.	2.16
Reached 0 lbs.	2.20¼

XXVIII A very unsteady on hind-legs at 2.25, passed urine full of froth; legs gave way; at 2.30 lying down grunting, constant nystagmus, mucous membranes not pale; at 2.38 respiratory movements almost stopped; died 2.44 p.m. Ordinary moderate bubbling. XXVII A bends left fore-leg at 2.27, bad; a little left next day. XXIV A left hind-leg bends at 2.29; had pretty marked dyspnoea at 2.40. XXIX A urine 2.40 no froth, seemed uneasy, kept lying down but could make out nothing definite. XXVI A no symptoms.

(*d*) 24.5.07. Goats 7, 9, XXVII A, XXX A, XXXII A, XXVII.

Started up	...	...	...	...	9.51
Reached 51 lbs.	...	...	...	...	10.15
Started down	...	...	...	...	1.3
Reached 0 lbs.	...	...	...	...	1.7

XXX A, urine 1.10 much froth, no symptoms. XXVII A, bad bends left fore-leg, jumpy hind-legs. 7, bends right fore-leg. 9, bends right fore-leg; slight dyspnoea, bends bad, both hind-legs wobbly; dyspnoea gone by 1.40 and legs alright. XXXII A bleating, won't stand up, dyspnoea; died 1.34 p.m. Bad general bubbling. XXVII bends right fore-leg.

(*e*) 5.6.07. Goats XII A, XVI A, XXIII A.

Started up	...	...	...	...	9.52
Reached 45 lbs.	...	...	...	...	10.14
Left 45 lbs.	...	...	...	...	12.3
Reached 0 lbs.	...	...	...	...	12.3.33″
,, −6 lbs.	...	...	...	...	12.10

XVI A uneasy at −5 lbs., paraplegic at 12.10, struggling and bleating, dyspnoea. XII A bends right fore-leg 12.19, bleating at 12.28. XXIII A tried to get up at 12.25 but failed once; then got up, right hind-leg paralysed; both gone just afterwards, could just crawl across tank; dyspnoea at 12.28. Raised pressure to atmospheric and opened tank at 1.10. XII A got up and seemed alright. XXIII A and XVI A lay log-like, conscious, breathing slightly and slowly. At 1.40 XXIII A could rest on fore-legs, hind-legs completely paralysed, ate hay; seemed pretty well except for paraplegia at 4.0. (Condition did not improve and it was killed six days later.) XVI A died at 3.20 p.m. A few small bubbles in right auricle and right femoral vein.

(*b*) 12 . 6 . 07. Goats 7, XXIV A, XXIX A, XXX A.

Started up	9.55
Reached 39 lbs.	10.13
Left 39 lbs.	12.4.20″
Reached 0 lbs.	12.4.50″
Left 0 lbs.	12.6.10″
Reached − 6 lbs.	12.11.5″

XXX A bends right fore-leg at 12.13, dyspnoea at 12.30. XXIX A bends right fore-leg at 12.14, lay down, dyspnoea at 12.24. 7 lay down, dyspnoea at 12.14. XXIV A no symptoms. Raised pressure to normal and opened up at 1.15. 7 showed bends left fore-leg and had slight dyspnoea. XXX A seemed alright. XXIV A and XXIX A were very quiet but no definite symptoms. All alright at 3.30.

APPENDIX IV.

TABLE I.

Stoppages during the ascent of a diver after ordinary limits of time from surface.

Depth		Pressure	Time from surface to beginning of ascent	Approximate time to first stop	Stoppages in minutes at different depths*						Total time for ascent in mins.
Feet	Fathoms	Pounds per square inch			60 ft.	50 ft.	40 ft.	30 ft.	20 ft.	10 ft.	
0–36	0–6	0–16	No limit ...	—	—	—	—	—	—	—	0–1
36–42	6–7	16–18½	Over 3 hours	1	—	—	—	—	—	5	6
42–48	7–8	18½–21	Up to 1 hour	—	—	—	—	—	—	—	1½
			1–3 hours ...	1½	—	—	—	—	—	5	6½
			Over 3 hours	1½	—	—	—	—	—	10	11½
48–54	8–9	21–24	Up to ½ hour	—	—	—	—	—	—	—	2
			½–1½ hours ...	2	—	—	—	—	—	5	7
			1½–3 hours ...	2	—	—	—	—	—	10	12
			Over 3 hours	2	—	—	—	—	—	20	22
54–60	9–10	24–26½	Up to 20 mins.	—	—	—	—	—	—	—	2
			20–45 mins. ...	2	—	—	—	—	—	5	7
			¾–1½ hours ...	2	—	—	—	—	—	10	12
			1½–3 hours ...	2	—	—	—	—	5	15	22
			Over 3 hours	2	—	—	—	—	10	20	32
60–66	10–11	26½–29½	Up to ¼ hour	2	—	—	—	—	—	—	2
			¼–½ hour ...	2	—	—	—	—	—	5	7
			½–1 hour	2	—	—	—	—	3	10	15
			1–2 hours ...	2	—	—	—	—	5	15	22
			2–3 hours ...	2	—	—	—	—	10	20	32
66–72	11–12	29½–32	Up to ¼ hour	2	—	—	—	—	—	2	4
			¼–½ hour ...	2	—	—	—	—	3	5	10
			½–1 hour ...	2	—	—	—	—	5	12	19
			1–2 hours ...	2	—	—	—	—	10	20	32
72–78	12–13	32–34½	Up to 20 mins.	2	—	—	—	—	—	5	7
			20–45 mins. ...	2	—	—	—	—	5	10	17
			¾–1½ hours ...	2	—	—	—	—	10	20	32
78–84	13–14	34½–37	Up to 20 mins.	2	—	—	—	—	—	5	7
			20–45 mins. ...	2	—	—	—	—	5	15	22
			¾–1¼ hours ...	2	—	—	—	—	10	20	32
84–90	14–15	37–40	Up to 10 mins.	2	—	—	—	—	—	3	5
			10–20 mins. ...	2	—	—	—	—	3	5	10
			20–40 mins. ...	2	—	—	—	—	5	15	22
			40–60 mins. ...	2	—	—	—	3	10	15	30
90–96	15–16	40–42½	Up to 10 mins.	3	—	—	—	—	—	3	6
			10–20 mins. ...	2	—	—	—	—	3	5	10
			20–35 mins. ...	2	—	—	—	—	5	15	22
			35–55 mins. ...	2	—	—	—	3	10	15	30
96–108	16–18	42½–48	Up to 15 mins.	3	—	—	—	—	3	5	11
			15–30 mins. ...	3	—	—	—	3	7	10	23
			30–40 mins. ...	3	—	—	—	5	10	15	33
108–120	18–20	48–53½	Up to 15 mins.	3	—	—	—	2	3	7	15
			15–25 mins. ...	3	—	—	—	5	5	10	23
			25–35 mins. ...	3	—	—	—	5	10	15	33
120–132	20–22	53½–59	Up to 15 mins.	3	—	—	—	2	5	7	17
			15–30 mins. ...	3	—	—	—	5	10	15	33
132–144	22–24	59–64½	Up to 12 mins.	3	—	—	—	3	5	5	16
			12–25 mins. ...	3	—	—	2	5	10	12	32
144–156	24–26	64½–70	Up to 10 mins.	3	—	—	—	3	5	5	16
			10–20 mins. ...	3	—	—	2	5	10	12	32
156–168	26–28	70–75	Up to 10 mins.	3	—	—	2	3	5	5	18
			10–16 mins. ...	3	—	2	3	5	7	10	30
168–180	28–30	75–80½	Up to 9 mins.	3	—	—	2	3	5	5	18
			9–14 mins. ...	3	—	2	3	5	7	10	30
180–192	30–32	80½–86	Up to 13 mins.	3	—	2	3	5	7	10	30
192–204	32–34	86–91½	Up to 12 mins.	3	2	2	3	5	7	10	32

* During each stoppage the diver should continue to move his arms and legs.

TABLE II.

Stoppages during the ascent of a diver after delay beyond the ordinary limits of time from surface.

Depth		Pressure	Time from surface to beginning of ascent	Approximate time to first stop	Stoppages in minutes at different depths							Total time for ascent in mins.
Feet	Fathoms	Pounds per square inch			80 ft.	70 ft.	60 ft.	50 ft.	40 ft.	30 ft.	20 ft.	10 ft.	
60–66	10–11	26½–29½	Over 3 hours	2	—	—	—	—	—	—	10	30	42
66–72	11–12	29½–32	2–3 hours ...	2	—	—	—	—	—	—	10	30	42
			Over 3 hours	2	—	—	—	—	—	—	20	30	52
72–78	12–13	32–34½	1½–2½ hours	2	—	—	—	—	—	—	20	25	47
			Over 2½ hours	2	—	—	—	—	—	—	30	30	62
78–84	13–14	34½–37	1½–2 hours ...	2	—	—	—	—	—	—	15	30	47
			2–3 hours ...	2	—	—	—	—	—	5	30	30	67
			Over 3 hours	2	—	—	—	—	—	10	30	35	77
84–90	14–15	37–40	1–1½ hours ...	2	—	—	—	—	—	5	15	25	47
			1½–2½ hours ...	2	—	—	—	—	—	5	30	35	72
			Over 2½ hours	2	—	—	—	—	—	20	35	35	92
90–96	15–16	40–42½	1–1½ hours ...	2	—	—	—	—	—	5	15	30	52
			1½–2½ hours ...	2	—	—	—	—	—	10	30	35	77
			Over 2½ hours	2	—	—	—	—	—	30	35	35	102
96–108	16–18	42½–48	40–60 minutes	2	—	—	—	—	—	10	15	20	47
			1–2 hours ...	2	—	—	—	—	5	15	25	35	82
			Over 2 hours	2	—	—	—	—	15	30	35	40	122
108–120	18–20	48–53½	35–60 minutes	2	—	—	—	—	5	10	15	25	57
			1–2 hours ...	2	—	—	—	—	10	20	30	35	97
			Over 2 hours	2	—	—	—	—	30	35	35	40	142
120–132	20–22	53½–59	½–¾ hours ...	3	—	—	—	—	5	10	15	20	53
			¾–1½ hours ...	3	—	—	—	5	10	20	30	30	98
			Over 1½ hours	3	—	—	—	15	30	35	40	40	163
132–144	22–24	59–64½	25–45 minutes	3	—	—	—	3	5	10	15	25	61
			¾–1½ hours ...	3	—	—	—	10	10	20	30	35	108
			Over 1½ hours	3	—	—	—	30	30	35	40	40	178
144–156	24–26	64½–70	20–35 minutes	3	—	—	—	3	5	10	15	20	56
			35–60 minutes	3	—	—	—	7	10	15	30	30	95
			Over 1 hour	3	—	—	20	25	30	35	40	40	193
156–168	26–28	70–75	16–30 minutes	3	—	—	—	3	5	10	15	20	56
			½–1 hour ...	3	—	—	3	10	10	15	30	30	101
			Over 1 hour	3	—	5	25	25	30	35	40	40	203
168–182	28–30	75–80½	14–20 minutes	3	—	—	—	3	3	7	10	15	41
			20–30 minutes	3	—	—	2	2	3	10	15	25	60
			½–1 hour ...	3	—	3	3	7	10	20	30	35	111
			Over 1 hour	3	—	15	25	30	30	35	40	40	218
182–194	30–32	80½–86	13–20 minutes	3	—	—	—	3	3	7	15	15	46
			20–30 minutes	3	—	—	3	3	5	10	15	25	64
			½–1 hour ...	3	—	3	5	10	12	20	30	35	118
			Over 1 hour	3	5	20	25	30	30	35	40	40	228
194–206	32–34	86–91½	12–20 minutes	3	—	—	3	3	5	7	10	20	51
			20–30 minutes	3	—	3	3	3	5	10	20	20	67
			½–1 hour ...	3	3	3	5	10	15	20	30	35	124
			Over 1 hour	3	15	20	25	30	30	35	40	40	238