allowance of nitrogenous food so long as the excretory functions of the kidneys are well maintained. The thin, spare man with no excess of oxidizable material in his blood does best. The fat, pot-bellied individual cannot make proper use of his diaphragm, and there is an excessive demand for oxygen which his emphysematous lungs cannot supply. He can have a fair amount of hot water. Two or three pints of milk form a light diet if it be not supplemented by much else. If there be any excess of lime salts in the blood, milk and gelatine are objectionable, but frequently the lime salts are rather deficient, so I think it is always well to examine the blood. I prohibit the use of much table salt, but I allow plenty of fruit, and a fair amount of green vegetables. The latter have the advantage in helping to regulate the bowels, which are usually confined. Alcohol and tobacco should be strictly interdicted. Tea and coffee may be allowed in moderation.

Hygiene.

The patient should live in a clean, dry, moderately warm atmosphere, as pure and free from dust as possible. He should practise respiratory gymnastics, and be taught to expire rather than inspire; the difficulty is in getting rid of the stagnant supplemental air. Establish a negative intrapulmonary pressure, and the atmospheric pressure will soon drive some fresh air in. Teach the patient to keep his costal cartilages soft and pliant, give his lungs as much play as possible, and thus maintain their elasticity, and adopt thoracic breathing rather than abdominal breathing; from faulty teaching and long acquired habit the latter method of breathing is too deeply ingrained to require any special instruction. In order to give the lungs plenty of play I think there is no exercise equal to hill climbing; it calls all the respiratory muscles into play, and makes the individual expire as well as inspire; it has also a beneficial effect on the heart, and the amount of exercise can be easily regulated according to the capacity of the individual. In summer the patient can be sent to an elevated region up to 4,000 or 5,000 feet above sea level, provided the vital capacity be not below 150 cubic inches for the male and 100 for the female. A few years ago I saw an emphysematous man foolishly attempt the ascent of Mont Blanc, but he was very glad to turn back when he reached the Grands Mulets. For the injurious effects of very high altitudes on the circulation I cannot do better than refer you to the writings of the late Dr. Alfred L. Loomis and Dr. Theodore Zangger.*

Numerous mechanical devices and pneumatic cabinets have been invented to assist respiration in these cases, but they have all practically gone out of vogue, except perhaps some simple elastic bands to assist expiration. Some patients find it more comfortable to sleep in the prone posture on account of the assistance afforded to expiration. Nasal and thoracic breathing should be encouraged; in this way the costal cartilages are not so apt to become rigid, but a watch should also be kept on any excess of lime salts in the blood. Cold sponging in the mornings, the Aix douche with massage, and other hydropathic measures can be commended, but the patient should not be left long in a moist chamber.

Drugs.

Their name is legion, but comparatively few of them are of much value; the most generally useful are the iodides and the various preparations of iodine. When there is any deficiency in metabolism or the patient is too stout, I frequently add thyroid to the iodine. If there be any excess of lime in the blood, the citrates of sodium and potassium should be freely administered, or lemonade made with citric acid and cream of tartar may be freely given. Of course, in such cases milk should be forbidden. The bowels should be kept open with a dose of sulphate of sodium in the morning. Bronchitis should be treated as previously directed; if there be much cough, it should be assuaged by some preparation of opium. In the later stages, when there is cardiac failure, special attention should be directed to that organ. If the costal cartilages have been allowed to get very rigid, the surgeon may be called in to perform the operation recommended by Freund.

REFERENCES.

* Notnagel's *Encyclopaedia of Practical Medicine*. * *An Inquiry into the Causes of Respiration*, 2nd edition, 1833. * Schäfer's *Physiology*. * *Selected Researches in Pathology*, 1901. * *New York Medical Journal*, June 12th, 1886. *Liverpool Medico-Chirurgical Journal*, 1888, p. 369. * *Lancet*, June 20th, 1903.

Arris and Gale Lectures

ON THE PHYSIOLOGICAL AND PATHOLOGICAL EFFECTS WHICH FOLLOW EXPOSURE TO COMPRESSED AIR.

DELIVERED IN THE THEATRE OF THE ROYAL COLLEGE OF SURGEONS ON MARCH 18TH AND 20TH, 1908.

BY M. GREENWOOD, JUN., M.R.C.S., L.R.C.P.,

SENIOR DEMONSTRATOR OF PHYSIOLOGY IN THE LONDON HOSPITAL MEDICAL COLLEGE; DIRECTOR OF THE LONDON HOSPITAL STATISTICAL DEPARTMENT; LATE RESEARCH SCHOLAR OF THE BRITISH MEDICAL ASSOCIATION.

LECTURE I.

No races of mankind—indeed, no species of terrestrial animals—are habitually exposed to an atmospheric pressure which exceeds by more than a few millimetres of mercury that normally obtaining at sea level. Accordingly, our experience of the phenomena produced by such exposure is comparatively modern, not antedating the sixteenth century (Sturmius, Halley), and has only become wide since technical improvements in the diving-dress and the invention of subaqueous work chambers, or caissons, have rendered possible extensive aquatic engineering and marine salvage feats.

EFFECTS OF RAPID DECOMPRESSION: CAISSON DISEASE.

Whether we consider caisson workers or divers, we are faced by the same condition—exposure to compressed air. Broadly, the distinction between the two classes is that while caisson workers are usually exposed to a lower total pressure than divers, they remain under that pressure for a considerable time, while the diver commonly returns to the surface after a relatively short exposure. In both cases disease and death have been, and unfortunately still are, of frequent occurrence. The following are some caisson statistics:

Douchy Mines: 63 cases.
Kehler Bridge: 133 cases.
St. Louis Bridge: 119 cases out of 352 workers; 50 cases of paralysis; 14 deaths.
Brooklyn Bridge: 110 cases, 3 deaths.
Toulon Dry Dock: 43 cases, 2 deaths.
Culsac Bridge: 104 cases, 3 deaths.
Elsler Bridge: 38 cases, 2 deaths.
Nussdorf: 320 cases among 675 workers.
Felesti Bridge: 55 cases and 5 deaths among 154 workers.
Hudson Tunnel: 3 deaths a month until the conditions were improved.

Since May, 1906, 10,000 exposures to compressed air have been associated with 16 deaths.

It would be unnecessary, even were it possible, to give a clinical description of the cases, varying from simple joint pain to severe paralysis and death. One of the best accounts we have of a rapidly fatal case is due to McKinlay (1900).

The subject was a man of 33, exceptionally strong, and in good health. He descended 24½ fathoms in search of a torpedo, being therefore exposed to a positive pressure of between 4 and 5 atmospheres. He remained below forty minutes, and ascended in half that time. He entered the boat without any trouble, and when the helmet was removed, chatted unconcernedly about his work. He had suffered no inconvenience in the water, and only returned because the light was insufficient for his purpose; his comrades agree that he had never before come up in better condition. All was well for some eight or nine minutes, when suddenly he experienced pains in the stomach, and asked the attendants to get his dress off quickly, as he wished to go on board. In a few seconds he said, "Send for the doctor," and immediately collapsed. Fleet Surgeon McKinlay, who was in attendance at once, found the man cyanosed and breathing stertorously, the lips covered with foam; he was undressed and taken to the sick bay, but expired before arriving there, fifteen minutes after gaining the surface and six or seven counting from the first symptoms.

A careful autopsy revealed the following state of affairs: The surface of the brain was deeply engorged with dark fluid blood; many air bubbles were detected in the veins of Galen and choroid plexus, also in the superficial cerebral vessels themselves when the latter had been partly emptied of blood. The veins on the surface of the heart were beaded with air, while that organ felt like a bladder half full of water, gurgling loudly when pressed. On incision of the right ventricle air escaped with a puff; the left ventricle was empty. The mesenteric vessels were likewise filled with air and the liver frothed on section.

In its sudden onset, rapid termination, and *post-mortem* findings, this case gives a more harmonious picture than an exhaustive catalogue of the symptoms recorded by different observers.

We must next examine the interpretations of these results which passed current before 1879 and are still occasionally repeated.

The large majority of medical writers held that exposure to compressed air mechanically altered the distribution of blood within the body. They believed that blood was forced inwards, congesting the viscera, and that its subsequent return on decompression was the cause of symptoms. Others attributed the mischief to super-oxygenation of the blood, accompanied, as they believed, by extravagant metabolism. One writer held that the intestinal gas, expanding on decompression, acted like a huge cupping glass so that congestions and even haemorrhages were produced.

Amid this confusion two writers published statements not diametrically opposed to the principles of hydrostatics. These were Hoppe-Seyler and Bucquoy. The former wrote in 1857:

If, after an animal has remained for some time in compressed air, the pressure is suddenly lowered, the lungs have no time to eliminate the excess of gas; accordingly gas will be evolved in the great venous trunks themselves, and the symptoms described above will be induced. This is the explanation of the sudden deaths which have been observed in the French coal mines without anatomical lesions.

In 1861 Bucquoy wrote:

During and after decompression all the gas dissolved in the blood owing to the condensation of the air tends to escape from the liquid with efforts so much the greater, for equal times of exposure, the greater the pressure to which the subject has been submitted. This is a necessary consequence of the physical laws of the solution of gases in liquids, and we have a familiar and common illustration in the rapidity and force with which carbonic acid escapes from an aerated water when the bottle is uncorked.

These were, however, voices crying in the wilderness, as is shown by the approbation with which a memoir by Foley, published in 1863, was received. This monograph, characterized by Paul Bert, with delightful irony, as the most original work from the theoretical standpoint which he had ever encountered, reproduces and exaggerates all the absurdities of the "mechanical" school. I will quote one sentence in illustration:

When one enters the caisson one is flattened out, the arteries diminish in calibre, and one sweats easily. By all these effects the skin is emptied and dried up. On the other hand, the moment one leaves, by the mere mechanical force of its elastic fibres the envelope expands. Unfortunately the retractility of their yellow tunics preserves to nutrient vessels their minimum diameter. As a result, a sort of vacuum is formed around them:

If it be concluded that the early history of caisson disease reflects but small credit upon the logical powers of our profession, certain extenuating circumstances may fairly be pleaded. It is easy to make merry over these physical crudities, but we have to remember that the divorce of experimental science from clinical medicine was then nearly as complete as certain educational authorities desire to make it now. The victory of error over truth is attributable to that divorce. That the real cause of compressed air illness could be inferred from a consideration of known physical principles is true—in theory; but Hunter's advice to Jenner must be followed before such truth can incorporate itself as a real part of man's stock of knowledge.

RESEARCHES OF PAUL BERT.

To Paul Bert, and to him alone, is due the credit of making this great advance, of patiently and accurately employing the experimental method. It is a fact that the true pathology of caisson disease was suspected by at least two workers before Bert's time; it is also true that, since Bert's publications, some problems which he did not completely elucidate have been solved; it is not less certain that Paul Bert first placed the question on the firm basis of experiment, and that all subsequent advances are, in comparison with his work, trifles. His book has been exposed to searching and, in at least one instance, malicious criticism, yet not one important statement contained in it has been shown to be incorrect.

The relation of Bert to the physiology of atmospheric pressure closely resembles that of Pasteur to the science

of microbiology and that of Lister to the practice of aseptic surgery. Merely as a literary composition, *La Pression Barométrique* is a model some physiologists could study with advantage. As an illustration, let me quote our author's criticism of an absurd experiment performed by Vivenot:

The curious part of the business is that an experiment so strangely conceived and so unhappily performed has been accepted and applauded on both sides of the Rhine. Vivenot has done an experiment! It is said, and this is enough for many people; for there exists quite a school of medical men the secretaries of which have not, be it understood, haunted the laboratories, but for whom the word "experiment" takes the place of everything like the cream tart in the comedy (Bert, 1878, p. 516).

By a series of well-planned and accurate experiments Bert proved that:

1. Increased air pressures up to +7 and +8 atmospheres do not in themselves produce any unfavourable symptoms. No serious modifications of the vital phenomena occur until the tension of the oxygen dissolved has attained a degree at which this gas itself behaves as a tissue poison, especially with respect to nervous elements. In other words, the "mechanical" effects of pressure upon which so much eloquence has been vainly spent by Foley and others, simply do not exist apart from an unimportant increase of the thoracic diameters resulting from a diminution in volume of the intestinal gases.

2. Pathological accidents occurring after decomposition are due to the liberation of nitrogen bubbles from the blood and tissue liquids, such bubbles leading in some cases to embolism, in others to transitory nerve irritation.

3. These effects can be prevented if the rate of decompression be sufficiently slow.

Since Bert's death Phillipon (1894-5) and others have carried out some interesting researches in France; in Austria and Germany, v. Schrötter, Heller, and Mager have published the results of work in which experiment and clinical observation are happily combined, publications which confirm Bert's conclusions in every detail. In England Professor Lorrain Smith (1899) has much advanced our knowledge of the poisonous effects of oxygen, showing that prolonged exposures to even comparatively low tensions are inevitably followed by pneumonia.

With regard to caisson disease itself, however, the most valuable and extensive researches are those of Dr. Leonard Hill (1900-8). Dr. Hill was assisted in his earlier work by Professor J. J. R. Macleod and Dr. C. E. Ham; in his later researches I have been allowed to help, and I now wish to describe some of the more recent observations. Although I adopt the conventional "we," no one acquainted with modern physiological literature can fail to recognize the sagacity in devising experimental methods, at once simple and effective, which characterizes all Dr. Hill's contributions to biological science.

DIRECT EFFECTS OF HIGH PRESSURES.

A description of our work in chronological order would be tedious. I shall instead attempt to prove that each of our results is logically deducible from Bert's original hypothesis, or from a necessary extension of that hypothesis.

First of all, air pressure *qua* pressure should have no effect upon man, provided the oxygen tension do not attain a certain maximum, and equalization of pressure within and without the middle ear be not impeded by catarrh of the Eustachian tubes.

Although this is logically deducible from known physical considerations—is, indeed, obvious—many narratives of caisson workers and visitors to compressed air constructions suggest the contrary. As a matter of fact, the peculiar environment of, for example, the tunnel worker is calculated to enhance that subconscious tingling of our bodily sensations which, even in the least imaginative, so powerfully contributes to any complex of feeling. A visitor to the tunnel now in process of construction at Rotherhithe cannot but be impressed by his surroundings, which are, in the eyes of a tyro, sufficiently strange to account for most "sensations of pressure." Observations uncomplicated by these modifying circumstances are not numerous for pressures greater than +45 lb. In 1894 M. Hersent, an engineer, in collaboration with five members of the Bordeaux Medical Faculty, exposed workmen to pressures varying from +68 to +77 lb.

in a steel chamber. No ill-effects were produced; but we have not an account of the workmen's own sensations, so that Hill and I reinvestigated the matter with apparatus constructed by Messrs. Siebe and Gorman, whose assistance has throughout been of the greatest value. We used a steel boiler having an internal volume of 42.2 cub. ft., so that the subject could adopt a sitting or recumbent posture, and fitted with an electric bell and telephone, allowing him to communicate with the outside world.

In this chamber we have been exposed on eleven occasions to pressures greater than +60 lb., once attaining +92 lb. As a result, we found that no real "sense of pressure" is to be detected. Of course, even under such favourable conditions some degree of anxiety, especially in the earlier experiments, could not be avoided, but this soon ceased to trouble us. Two changes only are noticeable, which may, perhaps, be referred to an increased density of the atmosphere. The first is an alteration in the quality of the voice, which becomes nasal and metallic, losing its individuality; the second is an inability to whistle, no doubt because the necessary fine vibratile movements of the lips and tongue are damped; an apparent consequence of this inability is that the lips appear to be anaesthetic, the sensation being similar to that following an application of cocaine, although direct sensitivity to stimulation is unaltered. This would seem to be a state of pseudo-anaesthesia, mainly due to a lack of normal movement; but the power of whistling is to some extent regained by practice, at least for moderate pressures, and no doubt nervousness is influential even here.

The absence of direct action of increased air pressure upon the vital processes is shown by our observations on the pulmonary output of carbon dioxide.

Haldane and Priestley (1905) have demonstrated that the tension of CO_2 in the alveolar air varies normally within narrow limits only, being in most persons about 5 per cent. of an atmosphere. If highly compressed air do not change the output, this percentage must vary inversely as the pressure, being 2.5 per cent. at +15 lb., 1.67 per cent. at +30 lb., etc. We ascertained that this is actually the case. Samples of alveolar air were collected and analysed while under pressure, and the percentage of carbon dioxide found, multiplied by the total pressure in atmospheres, gave a constant value, as shown in the table:

Percentage of CO_2 Found.	Pressure.	Percentage Multiplied by Pressure (in Atmospheres).
	Subject M. G.	
5.3	+ 0 lb.	5.3
0.9	+ 75 "	5.4
1.0	+ 60 "	5.0
1.8	+ 30 "	5.4
2.7	+ 15 "	5.4
5.4	+ 0 "	5.4
	Subject L. H.	
4.7	+ 0 lb.	4.7
0.9	+ 60 "	4.5
0.7 and 0.8	+ 75 "	4.5
0.95	+ 60 "	4.75
1.2	+ 45 "	4.8
1.8	+ 30 "	5.4
2.5	+ 15 "	6.0
5.0	+ 0 "	5.0

So far, therefore, we have no evidence that gaseous metabolism is at all affected by a sojourn in compressed air.

Some determinations of the respiratory quotient from samples collected by the method of Zuntz testify in the same sense; thus, in one experiment, the respiratory quotient at +0 was 0.85 and 0.9 under a pressure of +45 lb., but the results are neither numerous enough

nor sufficiently accurate to allow me to attach much importance to them.

Panum, G. Liebig, Pravaz, and others have published researches which seemed to indicate that oxidation is increased at pressures of about +15 lb. Bert inclined to the view that some such increase did occur, but pointed out that "nothing is more difficult than such experiments, and the deductions are always hazardous" (Bert, p. 1139). Hill and Macleod (1903) exposed three dogs to +6 and +7 atmospheres for several hours a day in a series of observations extending over more than a week. The urine was collected, the total nitrogen, the urea nitrogen, the sulphates, and the phosphates were determined, but no distinct change was discovered.

A matter of greater importance is the diminished frequency of the pulse which has been noted by almost every writer as a constant phenomenon. The following observations are extracted from Heller, Mager, and v. Schrötter's treatise (p. 653):

Before Exposure.	At + 1.5 Atm.	On Decompression.	Fifteen Minutes Later.
72	59	62	69
66	58	63	72
77	68	70	78
75	62	60	73
88	73	79	80
82	68	74	80
81	73	75	80
70	65	70	75
80	75	71	79
82	72	74	86
86	67	110	72
88	73	79	85

These observers could not, however, find any definite relation between the increment of pressure and the lowering of the pulse frequency. Our own observations do not reveal any marked diminution. In one experiment, my pulse-rate was 43 at +65 lb., 41 at +60, 41 at +45, 39 at +15, 42 at +10, and 41 at +2.3 lb. My pulse frequency is normally low, rarely exceeding 60 in the sitting and 45 to 50 in the recumbent posture; the effect produced was not, therefore, great. Hill was also unable to detect any change in his pulse-rate at +75 lb.

The interest of this point resides in the fact that saturation of the body with nitrogen is, ultimately, a function of the blood stream, so that if the circulation is less rapid under pressure, we could not draw any conclusions as to the time required for saturation from a knowledge of the normal pulse-rate. I shall refer to this in some detail later on, but would draw attention to it now as being, perhaps, one of the factors in the individual variations upon which stress has rightly been laid. Experiments on animals confirm the views just expressed, but there are influences at work which cause some results to appear contradictory. Hill and Macleod (1903) discovered the water output of mice to be diminished and their heat loss increased by exposure to moderately-compressed air. At first sight this would seem difficult to reconcile with my previous statement, but the observers were able to prove that the interference with metabolism was due to the saturation of the pressure chamber with water vapour, and was not a real consequence of exposure to compressed air itself.

The presence of carbon dioxide has been accused of exerting a specially unfavourable influence upon calsson workers, particularly by Snell, who published a good account (1896) of his observations at the Blackwall Tunnel. If carbon dioxide be regarded merely as a criterion of impurity, suggesting the presence of noxious substances, perhaps due to soil conditions, the accusation is just; but, as applied to the action of CO_2 , *per se*, it is groundless. We have never observed any bad effects upon ourselves, although we have allowed the percentage of CO_2 in the cylinder to rise to a value corresponding to 1.8 per cent., measured at the ordinary pressure. I have, more than

once, exposed cats and dogs to as much as 4 per cent. CO₂ without untoward results, beyond increased frequency of respiration. Hill has made many similar observations.

We have now established the truth of Bert's assertion that compressed air, as such, does not affect the bodily functions, and may pass to the more interesting and important facts relative to changes of pressure.

EFFECTS OF DECOMPRESSION.

It is scarcely necessary to adduce direct evidence in support of Bert's statement that bubbles of gas are liberated in the tissue liquids when the pressure is suddenly lowered. I wish, however, to describe some observations which make the proof logically complete, being of the nature of a control experiment. If the liberation of gas from circulating liquids be a principal factor in decompression effects, it follows that any animal devoid of a circulating liquid *respiratory* in function, must be immune.

Such animals are the larvae of Lepidoptera. In these creatures air is conveyed directly to the body cells by finely branching tracheae, and the vascular system is not believed to take part in the respiratory exchange. I have worked with numerous species (1906), including *B. rubi*, *S. populi*, and *C. cossus*, and have found them completely immune. An active third year's caterpillar of the goat moth was exposed to + 27 atmospheres for two hours forty minutes, and decompressed instantaneously without any ill effects; the larva subsequently pupated normally. A similar experiment on a larva of *Lasiocampa quercus* was followed by the emergence of a normal imago. On the other hand, these insects appear almost as sensitive to the toxic influence of high pressure oxygen as frogs. That the immunity is really produced in the manner described seems clear from the following experiments. Two active larvae of *L. quercus* were used; A. was placed in a small pressure chamber, with an observation window, together with some food plant, and raised to + 30 atmospheres. During the experiment the caterpillar consumed several leaves. After fifteen minutes the pressure was lowered rapidly, and on removal the insect was dead, the gut being enormously distended with semidigested leaves and air bubbles. On B. a similar experiment was performed, except that no leaves were put in the chamber. Sudden decompression did not lead to any unfavourable result, the insect pupating and emerging in the usual way. In the former case death was probably due to the expansion of air swallowed with the food and dissolved in the contents of the gut.

Experiments of this kind afford a pretty demonstration of the absurdity of primitive "mechanical" theories. If an active goat-moth caterpillar be enclosed in a glass-walled chamber, together with a cork, on raising the pressure, the cork, owing to the air contained in its pores, shrinks visibly, while the insect crawls about unharmed. If the pressure be instantaneously lowered the caterpillar is unaffected, but the cork suddenly reassumes its former shape.

NATURE OF GASES LIBERATED.

Bert found that the gas liberated in the tissues was almost entirely composed of nitrogen, and concluded that this gas was alone to be feared, since the avidity of the cells for oxygen is, normally, so great that the latter would never be yielded up in sufficient quantity to form emboli. Bert's analyses of the gas set free in the hearts of dogs rapidly decompressed from + 10 atmospheres, gave:

	Per Cent. Carbon Dioxide.	Per Cent. Oxygen.	Per Cent. Nitrogen.
1	15.2	2.0	82.8
2	15.9	0.0	84.1
3	20.8	A trace	79.2

V. Schrötter, Heller, and Mager found:

	Per Cent. Carbon Dioxide.	Per Cent. Oxygen.	Per Cent. Nitrogen.
1. 4 dogs ...	4.71	15.31	79.98
2. 4 dogs ...	12.45	7.18	80.37

Hill and Ham (1905) decompressed rats from + 10

atmospheres, and dissected their bodies under a funnel filled with acidulated water. They found:

	Per Cent. Carbon Dioxide.	Per Cent. Oxygen.	Per Cent. Nitrogen.
1	10.7	2.1	87.2
2	16.0	4.0	80.0

Hill and Macleod (1903, C) submitted frogs and toads to + 20 atmospheres of oxygen (Brin's oxygen, containing 93 to 95 per cent. pure gas) for five minutes, and observed that after decompression the animals were not only convulsed, but enormously distended with gas. Mice exposed to + 10 atmospheres of oxygen were not so swollen, but went into tetanic spasms. Such convulsions could be evoked by a touch, and animals killed in this state occasionally showed bubbles in the capillaries of the central nervous system. Being composed of oxygen, these emboli supplied the nerve cells with that gas until the circulation was re-established. Accordingly, the nervous inco-ordination was but temporary, and the animals in most cases recovered.

A consideration of these experiments suggests that if the power of the cells to combine with oxygen be destroyed or injured, then not only nitrogen, but oxygen as well, would be liberated from solution and form emboli. In order to test this inference, we have exposed frogs, mice, and rats to high oxygen pressures until they showed signs of paresis, and then rapidly decompressed them. In all cases we have obtained large quantities of free oxygen from the peritoneal cavities, subcutaneous tissues, and blood vessels.

On one occasion 3 rats were exposed to + 70 lb. of oxygen for an hour. Two were convulsed in thirty-five minutes, and at the end of the experiment all were moribund. One, however, still breathed after decompression, which occupied three and a half seconds; an analysis of its peritoneal gas gave 54.5 per cent. oxygen. In another experiment 5 rats were exposed to + 75 lb. of oxygen for twenty-five minutes; all appeared to be affected, and 1 was convulsed. The pressure was reduced to + 0 lb. in four seconds, and several of the others were immediately thrown into spasms. One of the latter was killed for analysis, and yielded 67.9 per cent. oxygen and carbon dioxide, 32.1 per cent. nitrogen. (Hill and Greenwood, 1907, B)

We are therefore entitled to conclude that oxygen bubbles will be liberated when the tissues are damaged by prolonged exposure to that gas. The onset of convulsions immediately after decompression, as observed in the experiment last described, is suggestive, and we attribute it to bubbles, suddenly formed, stimulating the already poisoned tissues.

These results also follow naturally from the principles enunciated by Paul Bert, because the power of combining with oxygen has been lessened or abolished.

Having established these fundamental propositions, it becomes of importance to consider the manner in which the tissues are saturated with gas, and, subsequently, the methods by which that gas may be removed without danger.

It is clear that the absorption of gas is effected by the blood as it passes through the lungs. The vast surface exposure of blood in the pulmonary capillaries and the intimate relationship between blood and air in the alveoli favour rapid saturation of this liquid with gas. We have, indeed, no proof that blood, after one passage through the pulmonary circulation, is in equilibrium with the alveolar air, the experimental evidence being imperfect.

Analyses of Bert, v. Schrötter, Hill and Macleod demonstrate, however, that the nitrogen content of arterial blood is, in a relatively short space of time, in moderate agreement with the atmospheric partial pressure of that gas. The following table is an illustration (Hill and Macleod, 1903 B):

	Pressure.	Exposure.	Nitrogen Calculated (Percentage).	Nitrogen Found (Percentage).
Dog	+ 100 lb.	30 min.	11.9	8.41
Dog	+ 100 lb.	2 hr. 45 min.	11.9	11.61
Dog	+ 100 lb.	1 hr.	11.9	9.27
Cat	+ 100 lb.	1 hr.	11.9	10.14

The calculated amounts depend upon the assumption that 1.8 per cent. of nitrogen is dissolved in the plasma at +0. The actual values obtained in the control experiments of this series ranged between 1.46 and 2.16 per cent., a somewhat considerable variation.

In an experiment of v. Schrötter and his colleagues, a dog was exposed to +4.5 atmospheres for forty-five minutes, and the arterial blood yielded 9.2 per cent. of nitrogen as compared with a theoretical 9.9 per cent. on the former assumption. Bert's analyses are not strictly comparable with these, as he did not maintain his animals for long under a constant pressure, but they seem to suggest that the blood is not immediately saturated to the full amount. It is hard to arrive at satisfactory conclusions on this matter, because not only is the analysis of blood gases technically difficult, but the amount of nitrogen taken up by the plasma under normal circumstances is not accurately known. A less direct but simpler method of inquiry, which will be considered later, suggests that arterial blood is more rapidly saturated than the former analyses indicate, but we should be rash to assume that one circuit through the lungs necessarily equalizes the nitrogen tension of the blood with that of the alveolar air.

It is apparent that the time required to saturate the blood is not too easy to measure, but the problem is simplicity itself when compared with the more general question as to how long is required to saturate all the tissue liquids. As a matter of fact, absolutely complete saturation will not occur at all in a finite time. The reason of this is clearly explained by v. Schrötter (pp. 35-7, 1904, and p. 803, 1900).

Let us suppose that $1/p$ of the tissue liquids consists of blood, and that these liquids dissolve v volumes per cent. of nitrogen under normal conditions; let us further assume that under a pressure of n atmospheres all the blood passing through the lungs comes into gaseous equilibrium with the alveolar air at once.

Then, at the end of one complete circulation of the blood we shall have in the whole body

$$\frac{n.v}{p} + \frac{(p-1).v}{p} \text{ volumes per cent. of nitrogen.}$$

After another circulation we shall have:

$$\frac{n.v}{p} + \frac{p-1}{p} \left(\frac{n.v}{p} + \frac{(p-1).v}{p} \right) \text{ per cent. of nitrogen,}$$

and quite generally, after q circulations, we have:

$$n.v - \left(\frac{p-1}{p} \right)^q \text{ volumes per cent. of gas.}$$

Thus to attain full saturation—that is, $n.v$ volumes per cent. of dissolved nitrogen— q must be infinite.

In practice, however, the term $\left(\frac{p-1}{p} \right)^q$ diminishes rapidly at first and is small for relatively low values of q .

Thus, according to Haldane and Lorrain Smith (1900) the ratio of blood mass to body mass is about 1 to 20, and we have:

$$\frac{p-1}{p} = \frac{19}{20}$$

The value of this raised to the sixtieth power is less than 0.05. If, therefore, we assume all the blood to pass through the lungs in one minute, then at a pressure of +6 atmospheres—on the hypothesis that equilibrium is really attained in the lungs after one circuit—the body would be about 95 per cent. saturated in one hour. In other words, saturation is almost complete in a comparatively short space of time.

This investigation and the resultant formula are simple and interesting, but not very helpful in the solution of our real problem. To consider only one difficulty—the total circulation time as determined by experimental methods is misleading. Vierordt estimated (Heinz, 1906, p. 47) that the time required for the passage of a substance from one jugular vein to the other was, in the rabbit, 7.46 seconds; in a small goat, 14.14 seconds; in a dog, 16.7 seconds; in a horse, 31.5 seconds. As the pulse-rates of these animals are approximately 210, 110, 96, and 55 a minute respectively, he inferred that in each animal about the same number of pulsations is required to effect a complete circulation. This appears, at first sight, to afford a basis for calculation; but unfortunately it only gives us the time required for passing through the vascular system

by the *fastest moving* blood. For instance, if a volume equal to the total volume of the blood passes through the lungs of a man in one minute, this is *not* equivalent to saying that all the blood in his body traverses the lungs once a minute. Some portions of the blood will have made the circuit more than once, others not at all. We know roughly the circulation time of the *quickest* blood, not of the *slowest* blood, and the latter is just as important as the former. *A priori* reasoning will not, therefore, be of much assistance, and we can only have recourse to direct experiment.

PRIMARY PNEUMOCOCCIC PERITONITIS.

By ARCHIBALD CUFF, B.A., M.B., B.C., F.R.C.S.,
SURGEON TO THE ROYAL INFIRMARY, SHEFFIELD.

UP to within the last few years the all-prevailing view of the origin of non-traumatic peritonitis was that it was without exception caused by some gross lesion, generally readily recognizable in the course of operation or *post-mortem* examination of one of the abdominal viscera. This opinion, held and taught dogmatically, and almost come to be looked upon as an axiom of surgery, had displaced the older conception of peritonitis as a clinical entity and a frequent primary condition. Recently it has been established beyond all doubt that cases of peritonitis do occasionally come under observation in which the most careful search, both *ante* and *post-mortem*, fails to discover the expected lesion in an abdominal viscus; and not only so, but in the majority of these cases there is no other lesion in any other organ whatever. Such a condition may justly be described as a primary peritonitis.

The cause of many such cases, though probably not all, is the organism of pneumonia, the *Pneumococcus lanceolatus*. Pneumococcal peritonitis, whether "primary" or owing its origin to the existence of some infecting focus elsewhere in the body, is at all times uncommon. It is most often met with in children, and in adults would appear to be extremely rare. Ashdowne,¹ in 1906, when recording a case of the primary diffuse form in an adult, could only find the record of 29 similar ones; while Annand and Bowen,² with 4 cases of their own in the same year collected the records of 47 primary and 44 secondary cases in children. The disease, like that caused by other infecting agents, may be diffuse or localized in the form of an intraperitoneal abscess.

During the last two years the following instances of the disease have been under my care, and their record may, in spite of their small number, help with others to elucidate the nature of these obscure and difficult cases, the existence of which will have to be borne in mind when trying to arrive at the probable cause of peritonitis, and hence to its prognosis, for the diffused forms of pneumococcal peritonitis have a very high mortality.

The first case is an example of the localized form in the adult of very rare occurrence indeed, only 8 cases having been so far recorded.

LOCALIZED PERITONITIS: ABSCESS.

CASE I.—A girl, aged 17, was admitted into hospital complaining of pain on the left side of her abdomen.

History.

Ten days before admission she began to have rather severe pain all over her abdomen, like "stomach ache"; there was no vomiting, and the bowels were regular, being open every day. The pain continued to be diffuse until three days before being seen, when it became slightly less, though still rather severe, and became more localized to the left side. The patient had never had any previous illness, and her family, with the exception of her father, who suffered from heart disease and bronchitis, was a healthy one. Menstruation was in every way regular and normal.

Condition on Admission.

An examination of the abdomen showed that the left rectus muscle was rigid, and that there was little movement of the abdomen on that side. A mass extended vertically on this side from somewhat below the crest of the ilium to slightly above the level of the umbilicus, and transversely from just without the median line into the loin. The mass was extremely tender and dull to percussion. It was apparently firmly fixed to its deeper connexions. Nothing abnormal could be made out by a rectal examination. There was a slight vaginal discharge of muco-pus, but a vaginal examination showed that the uterus and appendages were of normal size, freely movable,