bacteriology of the disease is not well marked. Dr. Batten informs me in this respect that the distinction between these two types is not very definite, and it may very well be that the acute infective type is merely an aggravated form of the catarrhal.

The presence of a certain bacillus in the stools, or in the intestines, in cases of disease, even although that bacillus be not found in the stools of healthy individuals, hardly proves it to be the cause of the disease. If, however, it has been found to invade the blood or the spleen, this evidence is very strongly in its favour. Unfortunately, neither the blood nor the spleen of fatal cases were examined bacteriologically, so that the presence or absence of bacteria in these localities are matters for future investigation by me next summer.

My thanks are due to Dr. F. E. Batten, who took the greatest interest in this research, and supplied me with the material from the Hospital for Sick Children, Great Ormond Street; also to Dr. Allan Macfadyen, at whose suggestion and in whose laboratory the work was done, for his most valuable assistance and advice.

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REPORT XCVI.

THE INFLUENCE OF INCREASED BAROMETRIC PRESSURE ON MAN.*

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ALTHOUGH the idea of employing compressed air for submarine work is at least as old as the sixteenth century (Sturmius, Halley, etc.), the subject hardly possessed much industrial importance until the application of the principle to well-sinking and pile-driving by Triger, a French engineer, towards the middle of the last century. Triger's memoir was presented to the Academy of Sciences in 1841, his methods were speedily applied on a large scale, and since his time caisson works have been in operation all over the world. The pathological effects of living in compressed air are therefore of practical importance, and their scientific investigation worthy of attention.

The earliest description of caisson disease by medical men is that of MM. Pol and Wattle, published in 1854, but written in 1847, and founded on observations of caisson works in connexion with mining operations at Douchy. The theories of these authors, being destitute of experimental foundation, are erroneous; their practical conclusions, on the other hand, are much sounder than those of many subsequent writers. Thus they noted the importance of slow decompression in obviating accidents, a factor neglected or even derided by other workers. They say:¹

Compression up to 4½ atmospheres is not in itself dangerous; it is quite well borne, far better than a proportionally much less rarefaction. The return to normal pressure is alone the dangerous part, this danger being proportional at once to the amount of compression and rapidity of decompression; the rate of the latter should accordingly be much reduced.

For the next twenty years although a good deal was written little of it has any value. In 1877, Paul Bert's treatise, *La Pression Barométrique*, was published. This work deserves to be regarded as one of the classics of physiological science, and proves its author to have been

an experimenter and reasoner worthy to be ranked with his illustrious countrymen—Claude Bernard and Louis Pasteur. By a series of well-planned and accurately-conducted experiments Bert proved that:

1. Increased air pressures up to +7 or +8 atmospheres do not produce any unfavourable symptoms.

2. Pathological accidents occur during decompression only, and are due to the liberation of nitrogen bubbles in the blood and other tissue fluids, such bubbles leading in some cases to embolism, in others to transitory nerve irritation.

3. These symptoms can be prevented if the rate of decompression be sufficiently slow—that is, at the rate of about twenty minutes for each atmosphere of positive pressure.

These conclusions have received abundant confirmation from the more recent experiments of Hill and Macleod in England, and Heller, Mager, and von Schrötter in Austria.

In view of the attitude taken up by a small section of the British public with regard to experimental physiology, it is worth noting that Bert's epoch-making discoveries—discoveries which, if adequately recognized, would be the means of saving many lives—resulted from numerous experiments on animals.

It is somewhat discreditable to our intellectual culture that Bert's work has been little regarded. Those responsible for caisson works since his time have almost invariably allowed far too short periods for decompression, and many preventable deaths have occurred. Even in Austria, where scientific research is in closer touch with commerce than in many countries, Heller, Mager, and v. Schrötter speak bitterly of the obstacles placed in the way of an attempt to adopt suitable hygienic measures at the caisson works to which they were attached. They say²:

Unfortunately during our work we were able to convince ourselves how difficult it is, without possessing special authority, to carry out the necessary arrangements, with the result that well-thought-out and energetically-urged advice remains merely a pious expression of opinion, in that it is frustrated by external factors and opposition.

This and other experiences indicate that the co-operation of pure and applied science is still lamentably incomplete.

If we consider the effects of pressures greater than those employed in caissons, we shall see that the same conclusions hold. Deep-sea divers have been exposed to very high pressures, and in practice accidents similar to those already noted have occurred.

The following are some "record" dives. Lambert, the famous diver employed by Messrs. Siebe and Gorman, salved £100,000 at a depth of 160 ft., staying twenty minutes below and occupying about the same time in his ascent. On his last journey he stayed rather longer below, and became affected subsequently with symptoms pointing to a lesion of the spinal cord—permanent incontinence of urine, etc. Another diver, Walker, has descended to a depth of 189 ft. This man, to whom I have spoken, attributes his immunity from accidents to his habit of ascending slowly. Perhaps the greatest depth ever attained is one of 204 ft. (approximately equal to +88½ lb. air pressure); the diver in this case expired on returning to the surface, probably owing to a very rapid ascent.

The experimental study of these higher pressures, as applied to man was first undertaken by Hersent, who, in 1893, compressed a workman to +71.2 lb., and subsequently to +76.8 lb., without misadventure. As the subject of these experiments was not a trained observer, it seemed desirable to reinvestigate the subject, and to furnish yet another proof of the accuracy of Paul Bert's work. This has been done in the course of some researches by Leonard Hill and the writer.

Messrs. Siebe and Gorman kindly placed their workshop at our disposal and constructed the pressure chamber, of which a photograph is reproduced. In addition to the fittings represented in the figure, the chamber was provided with a mattress, blankets and electric light; further, it was of such a size as to permit the observer to sit up or lie down comfortably.

We first studied the effect of air pressures greater than those formerly employed.

In an experiment carried out in November last, the

* A full account of the experiments summarized in this essay will be found in a memoir by L. E. Hill, F.R.S., and M. Greenwood, jun., read at a meeting of the Royal Society on February 16th, 1906, and subsequently published in the *Proceedings*.

writer was compressed to +92 lb. (that is, a total pressure of seven atmospheres) in fifty-four minutes, and subsequently decompressed in two hours, seventeen minutes, without any inconvenience, beyond a little neuralgic pain in the arms which had completely disappeared within one and a half hours of quitting the pressure chamber.

Subsequently L. Hill and the author were compressed to +75 lb. on more than one occasion without any serious ill-effects.

A positive pressure of +92 lb. corresponds to a water depth of about 210 ft., and these experiments entitle us to conclude that it is possible for a man to endure a total pressure of seven atmospheres without unpleasant results, provided that:

1. At least twenty minutes are allowed for each atmosphere during the period of decompression—the decompression being carried out at a uniform rate.

2. All parts of the body are moved in turn, the joints being flexed and extended, and the muscles contracted, so as to maintain the capillary circulation in every part during compression. The importance of (2) will appear from the following considerations: In the experiment referred to above, which was followed by some neuralgic pain in the fore-arms, I had remained perfectly quiet during the period of decompression. In another experiment I moved every part of the body except the knees; after leaving the chamber, there was some pain and tenderness in these parts only. Still more demonstrative is the experience of Dr. Leonard Hill; on one occasion he omitted to massage his chest, and the same evening a well-marked purpuric eruption appeared in the anterior thoracic region, associated with local tenderness. This rash did not fade for some days.

Clearly, it is of importance to aid the sluggish capillary circulation by muscular movements and massage, thus avoiding any local stagnation of blood containing a large quantity of nitrogen in solution. It is the sudden liberation of this excess in areas where the circulation is feeble that leads to the joint pains frequently experienced by caisson workers, and called "bends" by them. In the case of the spinal cord, where, of course, such bubbling would produce grave lesions, the action of the respiratory pump is sufficient to avoid any stasis, under suitable conditions as to rate of decompression.

It has often been asserted that quantities of carbon dioxide, innocuous at ordinary pressures, become toxic in compressed air. Our researches do not confirm this. We have frequently allowed the percentage of CO₂ to rise considerably above the normal limit without any embarrassment of respiration or other inconvenience. Thus, in one experiment, at a pressure of +16 lb., the chamber contained 1.2 per cent. CO₂, equivalent to 2.4 per cent. at normal pressure; in another, at +30 lb., the percentage was 0.5, equivalent to 1.5 per cent. at +0; in another it was 0.6 per cent. at the same pressure, equivalent, therefore, to 1.8 per cent.

The ventilation of caissons is, for reasons which need not be discussed here, a problem of some difficulty, but it does not appear to be necessary to go to great expense in

ventilating for the sake of keeping the CO₂ percentage abnormally low.

It has often been observed that the power of whistling and whispering is lost in compressed air, while the voice undergoes peculiar changes, becoming nasal and metallic in quality. I can confirm these observations, and, in addition, Dr. Hill and myself experienced a peculiar lip anaesthesia, which is, we believe, dependent upon the loss of fine vibrations of the lips, owing to the density of the in-going and out-going air currents, that is, the sensory change depends upon a partial motor paralysis. Apart from this, we think there are no subjective impressions by means of which the increased pressure can be gauged. We have been unable to observe the marked diminution in pulse frequency, described by many writers.

Haldane and Priestley, in a recent paper,³ have shown that the percentage of CO₂ in the alveolar air is normally constant. Now if high air pressure do not modify the respiratory metabolism, one would expect the percentage of CO₂ in the alveolar air to vary inversely as the pressure, so that the actual quantity of that gas in the alveoli may remain constant.

A numerical illustration will make this clear. At one atmosphere, our lungs hold about 3,000 c.cm., and we respire

about 500 c.cm. at each breath, while the alveolar percentage of CO₂ is about 5, which gives about 5 × 30 c.cm. of carbon dioxide in the alveolar air. At three atmospheres we find that our lungs hold about 3,000 c.cm., and that the tidal air is 500 c.cm., and the alveolar percentage of CO₂ about 1 $\frac{2}{3}$. Reducing these values to terms of one atmosphere, the tidal air is equivalent to 500 × 3 c.cm.; and, as at one atmosphere, the volume of CO₂ in the lungs is $\frac{1}{3}$ × 3 × 30 c.cm.—that is, the same as before.

Our experiments are in perfect agreement with the above hypothesis, as the following records of two typical results show.

VIEW OF PRESSURE CHAMBER (CLOSED).

A. Manhole. B. Electric bell. C. Observation window. (This was closed by a steel shutter during most of the experiments.) D. Decompression tap. (A second decompression tap is seen at the right end of the chamber.) E. Telephone. F. Tube leading to compressing pump. G. Wire to electric lamp inside chamber.

Subject.	Pressure.	Percentage of CO ₂ in the Alveolar Air.	Percentage Reduced to Terms of One Atmosphere.
M. G.	0 lb.	5.3	5.3
	75 ..	0.9	5.4
	60 ..	1.0	5.0
	30 ..	1.8	5.4
	15 ..	2.7	5.4
	0 ..	5.4	5.4
L. H.	0 ..	4.7	4.7
	75 ..	0.7 and 0.8	4.5
	60 ..	0.9	4.5
	45 ..	1.2	4.8
	30 ..	1.8	5.4
	15 ..	2.5	5.0
	0 ..	5.0	5.0

These results, so far as they go, indicate that the respiratory exchange is not affected by an increase of barometric pressure.

Many points connected with the influence of high air pressures on the physiological functions of man urgently need investigation. For example, the rate at which the body fluids are saturated with gas is a question of much theoretical and practical importance which has not yet

been elucidated. The results described above do, however, show the prophylactic importance of slow decompression, and suggest that with careful attention to various details the upper limits of pressure, both for caissons and diving operations, can be much extended.

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A CASE OF EXOPHTHALMIC GOITRE,

WITH REMARKS ON

THE PROBABLE NATURE OF THE DISEASE.

By HENRY TEMPEST DUFTON, B.A.CANTAB., L.S.A.,
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THE following case has been selected (chiefly because the patient is at present under treatment) from amongst some forty to fifty cases of goitre, both simple and exophthalmic, which have been treated successfully upon the same lines during the past twelve years.

Mrs. R., aged 51, married, of the poorer class, was first seen by me on July 12th, 1905; since then I have seen her eleven times in all.

SYMPTOMS.

A uniformly enlarged right lobe of the thyroid was easily seen and felt, and rather soft to the touch, measuring not quite 4 in. across; on the left side was a prominent hard knot, 1½ in. in diameter, with the appearance and feeling of fibrous or tense cystic tissue. The enlargement on the right side had been noticed by the patient for certainly fifteen months; that on the left had existed for about fifteen years, and had been professionally treated many years ago. The pulsation of the carotids gave an impulse to the swelling on the right side.

Exophthalmos existed in a moderate degree, and von Graefe's sign was present. There was oedema of the eyelids in varying amount at different times. In addition to a general restlessness, shown in fidgety movements of hands, arms and head, a fine tremor of the hands was plainly seen.

The patient complained of palpitation, and it was observed that the action of the heart was rapid, and was increased by excitement or emotion. The pulse, usually regular, has not been below 112. The patient, intolerant of strangers, was chiefly in a state of subdued excitement. She was emaciated, and stated she had lately become thinner, but there was no loss of strength. She had to make a great effort to raise her voice above a whisper. She drove four miles in an open cart, on some of the coldest days, to the surgery, and did not complain of the cold; perspiration was, however, free upon slight exertion. There were pigmentary changes on her face, neck, and hands, and she stated that she was changed in colour. There was no itching. Appetite was good, and the bowels naturally regular.

RESULTS OF TREATMENT.

The treatment given was, I confess, almost entirely symptomatic. Rest, in the horizontal position, during a portion of the day, and in the open air when possible. The drugs given were tincture of belladonna and liquor arsenicalis, of each 5 minims; potassium bromide, 10 gr.; and tincture of digitalis, 8 minims—three times a day, in water, after meals. Liniment of iodine was applied externally, eleven times altogether.

The right lobe is now reduced to little over one half of its former size. The knot on the left is softer and smaller. The exophthalmos is scarcely noticeable. The patient of her own accord has frequently said she was better. There is still the tremor of the hands, but she says she does not notice it when at home. She feels much better when she is quite alone. She complained of dryness of the throat after taking the medicine (belladonna), but not before. Twice I have omitted this ingredient, and twice the arsenic. The pupils are moderately dilated, the left more than the right. She does not complain of palpitation, and the pulse is now just 100. She now has to make no effort to raise her voice.

NATURE OF THE DISEASE.

In certain parts of East Anglia simple goitre is very common. The exophthalmic form is, I think, also frequently seen. In the textbooks the disease is classed with those of glands, but is it not probable that it is essentially a nervous disease? It is well known that many of the symptoms are suggestive of a nervous origin, and that the disease is sometimes associated with other nervous disorders; and, may I ask, is not that nervous origin intimately connected with the disturbances of the circulation? Let us inquire what nervous factors would produce tachycardia, palpitation, pulsation of the carotids, and from these possibly cardiac hypertrophy or dilatation.

The patients are readily upset by a loud noise, or good or bad news; they are irritable, peevish, and easily given

to tears. These symptoms certainly suggest the sympathetic system as the origin, but not more than the others. To what symptoms would disease of the sympathetic ganglia connected with the cardiac plexus, for example, be expected to give rise? To an inhibition of function, certainly; to a loss of tone in the large arteries. This loss of tone, produced by disordered action of vaso-motor nerves, would cause a general dilatation of the large arteries; it would be first felt on the right side and in the innominate artery, the first large vessel arising from the aorta; it would be followed in a direct line into the common carotid, the external carotid, and into the first branch of the external carotid—that is, the superior thyroid artery. Similarly, the loss of tone producing dilatation in the thyroid axis would be continued into the inferior thyroid artery. This would account for the usual prior enlargement of the right lobe of the thyroid gland. The proximity of the middle cervical ganglion which gives off the middle cardiac nerve; the sympathetic nerve at the back of the sheath of the common carotid artery; the upper and lower cervical ganglia with the internal carotid and subclavian arteries respectively are to be remembered.

It seems reasonable to argue that the tachycardia, and possibly the palpitation, may be caused by communication from the last cervical ganglion, through the accelerator nerves, as it is well known that when stimulated these nerves produce a quickening of the heart's beat. It is well known, also, that division of the cervical sympathetic causes vascular dilatation of the face, which is accompanied by increased perspiration.

As regards the exophthalmos, a similar argument will obtain from the contiguity of the sympathetic root of the lenticular ganglion with the ophthalmic artery.

The persistent or enlarged thymus, the enlarged lymphatic glands, the increased connective tissue in the neck, are not these due simply to increased vascularization?

What actual evidence is there that these diseases really originate in the sympathetic ganglia? All the evidence of which I am aware is that found by Dr. Greenfield. He has described swellings of the sympathetic ganglia, invasion of their tissue by leucocytes, and degenerative changes in the ganglion cells. If these morbid changes are constant, what more can we want? In short, may not the hypertrophy and over-activity of the thyroid gland, the exophthalmos, the persistent thymus, the enlarged lymphatic glands, the increased connective tissue in the neck, the tachycardia, palpitation, and tremor, besides the other subjective symptoms, be all the symptoms of a disease of some of the chain of sympathetic ganglia? If so, I see no reason why ordinary simple goitre and the exophthalmic form should not be varieties of the same disease, and that a disease of the sympathetic ganglia.

A CASE OF WIDESPREAD MOTOR PARALYSIS
DUE TO MULTIPLE SYMMETRICAL
PERIPHERAL NEURITIS,FOLLOWING AN UNUSUALLY MILD ATTACK OF SCARLET
FEVER.By FREDERICK W. PRICE, M.B., C.M., M.R.C.P.,
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It is well known that peripheral neuritis may occur as a sequela in many of the specific febrile affections, and Dr. Buzzard, among others, has pointed out that a motor paralysis having a very widespread distribution may be caused by a lesion of the periphery of the nerve trunks. The following is a remarkable example of such a case occurring in the sequel of an unusually mild attack of scarlet fever. When the patient first came under observation it was difficult to make a diagnosis between peripheral neuritis and acute anterior poliomyelitis, but the subsequent progress put the matter beyond all doubt. The case is also interesting, because during its course it presented the features of what looked like a very brief attack of acute nephritis.

REPORT OF THE CASE.

A girl, aged 11 years, was admitted to Westminster Hospital on November 25th, with practically complete loss of power in the legs, and some paresis of both arms.