is the central impediment; its working is as retrograde as the flight of the Parthian arrow: is it not well named?

To see the above-named effects as *pressure* effects, it will be simplest to observe the sequence of events which arises when a vein is tied or otherwise obstructed. What happens? The movement of the blood in the veins is stayed at the obstruction and that portion of the *vis a tergo*, which until that moment had been expending itself, first, in developing the liquid momentum of the flowing blood, secondly, in overcoming the surface resistance of the vessel walls, now, as the blood comes to a standstill, appears as a distending force and the vein swells immediately behind the block. This process repeats itself, as, successively, the oncoming blood is held up, until the venules and capillaries are reached. If the distending force is able to push past the obstruction, well and good; if it cannot the rise of pressure and distension go on increasing until a maximum¹ is reached and the *vis a tergo* is counterbalanced by the stretch of the tissues. Under the raised pressure the capillaries, in particular, ooze in excess of the drainage capacity of the lymphatics, and the part becomes more or less water-logged. In this area of engorgement, of stasis, of high blood pressure and of exudation (oedema), the functions of the part are brought more or less to a standstill and disintegration preponderates, completing the picture of relative death occasioned by the block.² Fortunately for the part there are few if any closed vascular circuits, in the sense of being quite shut off from neighbouring areas; in the vast majority there are collateral channels which interconnect more or less freely. These anastomotic channels constitute a reserve mechanism which safeguards the vitality of the blocked area by relieving the congestion therein and gradually re-establishing the circulation. The operation beginning at the periphery proceeds centrally, and we may note that it is the raised blood pressure, itself a secondary effect of the venous obstruction, which now comes to the rescue and opens up the collateral vessels.³ Incidentally, we may note that this raised blood pressure is not always so intelligently directed, for whilst it always tends to open up the collateral paths it also tends at the same time to force the blockade, and sometimes with success, as in the case of thrombosis. But the local relief thus secured is at the imminent peril of the whole, since the potentiality for evil of the embolus is of far greater moment than the local congestion. But this is by the way. After such manner does the venous obstruction work, and it matters not how small or how large is the vein involved the process is always the same—viz., by the backward projection of a raised intravenous pressure.

To come back now to the obstruction placed centrally in the heart, again the *modus operandi* is exactly the same, and under its influence the right heart and the whole systemic venous system tend to become congested and stagnant. As a preliminary to this (the block being at the mitral orifice) there will be the congestion of the veins, capillaries, and arteries of the pulmonary circulation, in particular of the hinder and lower parts of this circuit. In one respect, however, an important difference obtains between the effects of the block in the vein and the block at the heart, for whereas in the former, even though the obstruction be in the inferior or superior cava, anastomosis always gives local relief, more or less, in the latter, anastomosis cannot serve, since the whole venous system is involved. There is one exception to this statement: it holds when the cardiac obstruction arises in foetal life, before the completion of the interventricular and interauricular septa; in that case a kind of anastomosis is secured by the prevention of the completion of the septa and by the maintenance of the patency of certain foetal channels. By these means life may be made possible though upon a lower level.

I am unable to quote figures as to the exact values of the intravenous pressures in heart failure from valvular disease. In health physiologists put the pressure in the large veins at the root of the neck at 0 to minus 0.8 mm. Hg¹; in disease, in the absence of precise records, we may, I think, assume

safely that the pressure is always raised whenever there is fullness of the veins at the root of the neck—e.g., a distended external jugular; for if there were the smallest negative pressure at the outflow of the external jugular, or even a zero pressure there, the blood could not be dammed back so as to distend the external jugular and lift the skin over the vein; there must be a positive pressure in the vein to do this. The distended external jugular will therefore always indicate a plus intrathoracic venous pressure unless there be evidence of local compression of the vein.

II. *Back pressure as a cause of heart failure.*—In the ordinary way we do not speak of back pressure as the cause of heart failure, but as its expression or consequence, and in the main this is its most important aspect; now, however, we are to consider how it may be a definite cause of cardiac incompetence. The first roots of the systemic venous system are constituted by the coronary sinus and the coronary veins which open into it, and when back pressure develops as a result of cardiac obstruction these veins will, in a sense, be the first to feel the effects of back pressure, the first to suffer from the general venous stasis. But we have seen that vascular stasis involves a lowering of the vitality of the congested tissues, this can hardly be called in question, consequently the heart muscle, impaired in its nutrition, must suffer in its driving-power, and therewith another form of heart failure adds itself to the pre-existing failure from *mechanical obstruction*. We have here a good example of the vicious circle, so-called.

It is strange that a modern school of cardiologists would restrict the essential cause of heart failure to a functional impairment of the heart muscle; this is about as reasonable as to say that bowel failure can only result from functional impairment of the intestinal musculature, whereas we know that a mechanical obstruction—e.g., a strangulation—of the intestine or an impacted faecal mass may defy the most healthy and vigorous peristalsis, threatening death unless the obstruction be removed. Here the failure is as mechanical as is its relief. True, a paralysis of the bowel may bring about a failure as complete and as fatal in the absence of all mechanical obstruction, but this is no argument against the other variety of bowel failure.

In the foregoing the obstructing valve has received chief attention, but the leaking valve leads to exactly the same results, for the regurgitation develops a reactive pressure as effective as the simple passive resistance of a narrowed orifice in holding up the circulation.

In conclusion, it must be said that however much modern advances in cardiology may have in store for us, they have not so enriched us up to the present that we can afford to forgo those older standards which have stood the test of time. Amongst these standards we must place those back-pressure effects with which we are all familiar; and we must insist that to belittle their significance is not in accordance with clinical experience, that *argumentum ad aegrotantem*, which, in dealing with the sick man, his griefs, their cure or their relief, must ever speak the final word.

I am, Sir, yours faithfully,

HARRINGTON SAINSBURY.

Wimpole-street, W., Nov. 21st, 1917.

THE VALUE AND LIMITATIONS OF SANATORIUM TREATMENT.

To the Editor of THE LANCET.

SIR,—In your issue of Nov. 24th two important matters were dealt with.

(1) In Professor Sims Woodhead's and Mr. Varrier-Jones's article a statement was made, to which you referred in your leading article, on which I should like some further evidence.

"It is stated on very good authority that the results of sanatorium treatment vary according to the class of person treated, and that amongst the well-to-do the results of treatment are three times as good as are those obtained with the working men."

I should be very glad if the facts or statistics could be published on which this statement was founded, as it certainly does not accord with my general impression and is absolutely contradicted by the Australian Commission's figures, published in a recent number of your journal, where it was shown that in Australia 222 per 100,000 persons of independent means died from tubercle in 1911, as against only

¹ According to Halliburton, the complete closure of the veins leading from a capillary area may quadruple the capillary pressure (Physiology, edition 1917, p. 278); this pressure, since we are now dealing with a problem in hydrostatics, should represent the venous pressure also.

² Dr. Charles Bolton (Observations on the Pathology of Cardiac Dropsy, Oliver Sharpey Lectures, 1917) attributes the chief part of the oedema to the disintegration of the capillary wall.

³ With the re-establishment of the circulation the raised blood pressure, of course, falls. Cf. the experiments of C. Bolton, loc. cit.

⁴ Starling: Physiology, edition 1915, p. 817.

200 per 100,000 among miners, the highest death-rate among the working-class, and only 36 per 100,000 amongst dependents. A statement so far-reaching and important as that the rich recover easier demands very accurate proof.

(2) Concerning Dr. Louis Cobbett's letter *re* Success and Failure in the Treatment of Tuberculosis. While quite agreeing with him that the time has not yet arrived for the general use of prophylactic injection, and believing, as he does, that it must rest entirely on its own foundations, there are, in addition to the points he has suggested, two further points which at the present time give one a hope that it may be a definite remedial procedure. The first is, so far as I have been able to observe, that lupus tissue which has been attacked and has recovered seems to be immune from further invasion, the invasion always occurring at the free edge of the disease. Secondly, the application of tuberculin to the infected conjunctiva in repeated doses, at least in my hands, has produced such a condition of resistance that ulceration, or breaking down, though frequent previously, does not recur. Freedom from relapse has been demonstrated practically in all cases tested, most of them for a period of over two years.

I am, Sir, yours faithfully,

Middlesbrough, Nov. 27th, 1917.

HENRY A. ELLIS.

HYSTERICAL DEAFNESS IN SOLDIERS.

To the Editor of THE LANCET.

SIR,—The questions raised in the interesting paper by Major A. F. Hurst and Captain E. A. Peters in THE LANCET of Oct. 6th are of the greatest importance. It is certain that many applications for compensation will be made by soldiers who claim that their hearing has been injured or destroyed by the explosion of mines, shells, or hand grenades, or by prolonged gun-fire. There may be great difficulty in adjusting these claims as the hearing power of recruits has not been systematically examined. Thousands of men suffering from deafness—with or without otorrhœa—have been admitted to the Army. Sohler Bryant¹ gives some startling figures regarding the number of "ear" cases in the various zones of the French Army.

Major Hurst and Captain Peters state that organic disease or injury of the ear of sufficient intensity to produce severe cochlear deafness is invariably accompanied by loss of vestibular reaction. They apparently apply this law not only to cases of shell deafness but also to those of neuro-labyrinthitis due to epidemic cerebro-spinal meningitis. The logical conclusion is that if, in testing a case of shell deafness, or neuro-labyrinthitis we find that the vestibular reactions to the rotation and caloric tests are normal, we may conclude that the deafness is either hysterical or malingered. If this is really true it immensely simplifies the work of otologists in examining such cases. I, however, agree with Dr. P. McBride in thinking that we must hesitate a little before accepting this delightfully simple test as a reliable one for the purpose of excluding organic injury or disease of the sound-perceiving apparatus. It is a pity that Major Hurst and Captain Peters do not give an account of any cases of "explosion" injury of the ear in which they have found loss of the vestibular reaction.

We must confess that as yet we do not know the exact pathology of "shell" or "explosion" deafness. We can, of course, observe ruptures of the tympanic membrane when these are present, but we know that such ruptures, even when associated with hæmorrhage into the middle ear spaces, will not account for the marked or absolute deafness met with in cases of severe shell deafness. There are three other changes which may account for the condition: (1) Rupture of, or hæmorrhage into, the membranous labyrinth; (2) hæmorrhage into, or rupture of, the cochlear nerve or some of its fibres in the internal auditory meatus; and (3) degenerative neuritis of the nerve structures of the cochlear apparatus—i.e., Corti's organ, spiral ganglion cells, &c. In a paper on this subject at present appearing in the *Journal of Laryngology* (in conjunction with Captain John Fraser, R.A.M.C.) I have shown that, out of four cases of explosion injury of the ear microscopically examined, (1) rupture of the delicate neuro-epithelial sacs and tubes of the membranous labyrinth was not found in any case. (2) Hæmorrhage into the cochlear nerve at the fundus of the internal meatus was present in three out of four cases.

Such a lesion is quite capable of producing deafness. (In one case there was a large plug of wax in the external meatus which seems to have protected the structures of the middle and inner ear from injury.) (3) An early stage of degenerative neuritis was probably present in one case in which the injury was due to the explosion of a hand grenade close to a soldier. (Microscopic examination of a case of shell deafness which dies from other causes some months or years after the onset of the deafness would give more reliable results as to the presence or absence of degenerative neuritis.) From these rather meagre observations we may at least state that there is some pathological basis for explosion deafness. I must, however, admit that, as all the cases examined by Captain Fraser and myself were admitted to the casualty clearing station in a state of profound shock and died shortly after admission, no vestibular or cochlear tests were carried out.

I can, however, bring forward the case of a congenital deaf-mute in which microscopical examination of both inner ears showed that Corti's organ in all coils was so malformed that hearing was almost abolished, while the vestibular structures were normal. The caloric reaction was tested on both sides some days before the death of the patient and a normal response was obtained. Another case which throws some light on this subject was that of a patient who had suffered from chronic middle-ear suppuration and absolute deafness on one side for many years before death. (The vestibular reactions unfortunately were not tested as the case came under observation before such examinations became a routine part of otological work.) Microscopic examination of the labyrinth showed complete obliteration of the whole cochlea by new bone formation and atrophy of the cochlear nerve. The structures of the vestibule and semicircular canals along with the vestibular nerve, on the other hand, were comparatively well preserved. These two cases show that the function of the cochlea may be entirely lost while the vestibular apparatus is normal (Case 1) or shows but slight alteration on microscopic examination (Case 2). Those who have had experience in carrying out the functional tests of the cochlear and vestibular apparatus in cases of otosclerosis will have come across instances in which the patient is extremely deaf (mixed middle and inner ear deafness), whereas the vestibular apparatus responds normally to the rotation and caloric tests (Fraser and Walker²). These observations show that the cochlear apparatus is not always normal when the vestibular apparatus responds to the caloric test.

Professor A. A. Gray has shown³ that in the evolution of the animal kingdom the vestibular apparatus appeared long before the cochlear apparatus. The former is present and fully developed in the lower fishes, whereas the cochlea only begins to appear in the reptiles and amphibians and reaches its full development in the higher mammals. It is usually admitted that organs such as the vestibular apparatus which appear earlier in the scale of evolution have greater resisting powers or greater powers of recovery than those which, like the cochlear apparatus, are developed at a considerably later period. I know that your correspondent Dr. Dan McKenzie does not agree with this view.

Let us, however, look at the matter from the point of view of ordinary anatomy and physiology. There are two moveable areas in the rigid bony capsule which surrounds the labyrinth. These areas—the oval and round windows—are situated in the party wall which separates the middle from the inner ear. The former—the oval window—communicates with the perilymph space of the vestibule just at the point where the scala tympani of the cochlea begins. The latter—the round window—lies at the other end of the perilymph space of the cochlea. As the labyrinth capsule is rigid and as fluid is incompressible we must conclude that movements of the perilymph, produced by vibration of the stapes in the oval window, can only occur in that area which lies between the two movable windows. It would seem therefore that a "blow" to the ear caused by a sudden great movement of the atmosphere following an explosion is transmitted to the oval window and can only affect the column of fluid in the cochlea. This helps to explain the greater vulnerability of the cochlear apparatus. We must, however, admit that a very violent explosion close to a soldier can shatter the skull, including the temporal bone. The "rigidity" of the labyrinth capsule is therefore only a relative term.

¹ *Journal of Laryngology, Rhinology, and Otology*, November, 1917, p. 338.

² *Journal of Laryngology*, November, 1914, p. 519.

³ *Otosclerosis*, Lewis and Co., London, 1917, p. 55.