### INDIAN MEDICAL SERVICE.

The result of the January examination was announced on Feb. 1st. There were 58 candidates, of whom 51 ultimately entered for the examination; of these, 48 qualified, the first 16 being admitted as lieutenants-on-probation, while one withdrew during the examination. The names of the successful candidates, with the marks obtained by each out of a possible maximum of 5100, are given below:—

| Name. | Marks. | Name. | Marks. |
|---|---|---|---|
| W. E. Brierley | 3988 | F. R. Coppinger | 3610 |
| R. Knowles | 3962 | A. de C. C. Charles | 3575 |
| J. B. Lapsley | 3796 | J. W. Barnett | 3574 |
| J. A. Shorten | 3722 | F. Stevenson | 3554 |
| R. B. S. Sewell | 3680 | S. H. West | 3534 |
| W. L. Watson | 3652 | Madan Lal Puri | 3506 |
| C. H. Fielding | 3627 | Satya Charan Pal | 3501 |
| W. J. Simpson | 3621 | R. S. Townsend | 3483 |

### THE ROYAL NAVY LIST.[1]

This most excellent handbook has now been before the public for more than 30 years and each year demonstrates the care with which the book is compiled while everything is done to facilitate ready reference. Over 200 pages are devoted to the record of the war and meritorious service of officers both active and retired, and there is a complete list of ships of the Royal Navy with details relating to each ship. Under the heading of the "Naval Recorder" are an article on the current history of the Royal Navy, a list of the commissions and services of first- and second-class ships on the active list, a list of the fleets and squadrons in commission since 1878, and a chronological table of notable events from the year 1219 to the present time. A valuable feature of the Navy List is a bibliography of naval literature. The book should find a place in the library of everyone who is interested in our first line of defence.

# Correspondence.

"Audi alteram partem."

## PLEURAL EFFUSION AND ITS TREATMENT.

*To the Editor of* THE LANCET.

SIR,—Sir James Barr affects to think that in agreeing to accept the term "pulmonary traction" and to substitute for my hypothetical case of fibroid phthisis with non-adherent pleuræ an actual one with adherent pleuræ I have made important admissions. This is a little ungallant of him, as these concessions were for the purpose of humouring Sir James Barr and with the full knowledge that they did not affect one way or the other the main issue between us, which is whether it is, or is not, necessary for the student of pulmonary physics to keep separate in his mind the conception of pulmonary tautness and that of pulmonary elasticity. I may at once say that after doing my best to get at Sir James Barr's meaning I do not find that he advances in his last letter a single valid argument against any of the statements made in my two previous letters. I shall once more endeavour to convince him that my contentions are as easy of proof as they are simple.

I, of course, recognise that the factor of resistance is an essential ingredient in our conception of pulmonary elasticity, that the lungs cannot be made to manifest their elasticity as regards a stretching force without being stretched, and that when they are stretched they pull on the visceral pleura and so exert traction on the circum-pulmonary structures; but it is not by virtue of their elasticity that the lungs exert this traction but by virtue of their tautness. It is quite true, as Sir James Barr insists, that the elasticity of the lungs enables them to retain their tautness and thus to exercise a continuous traction during the entire respiratory cycle, and I am ready to admit that this is a prime function of pulmonary elasticity, but we have no more right to say that the elasticity causes the traction than we have to say that the elasticity in the hangman's rope causes the death of the condemned man, or that the elasticity of the traces attached to a waggon causes the waggon to be pulled along.

[1] The Royal Navy List and Naval Recorder, No. 121, January, 1908. Whitherby and Co., 326, High Holborn, London, W.C., and 4, Newman's-court, Cornhill, London, E.C.

Even Dr. D. W. Samways, for whose support, though qualified, I am grateful, fails, eminent physicist though he is, to make the necessary distinction between tautness and elasticity. "Consider," he writes, "the thorax and the respiratory muscles at rest. The lungs remain stretched; they support by their elastic recoil a certain fraction of atmospheric pressure to which the intrapleural space (if space it may be called) is not subjected in consequence. In that sense, as Sir James Barr states, 'there is a slight negative pressure in the pleuræ owing to the elasticity of the lungs.'" I submit that the negative pressure at this moment when the chest is kept fixed and when we may assume the intrapulmonary pressure to equal the extra-corporeal pressure is due to the tautness of the pulmonary tissue pure and simple and that the factor of "elasticity" or "elastic recoil" has nothing to do with it. Let us for argument's sake suppose the lungs in the case in question suddenly to be rendered perfectly non-elastic, their tautness, however, remaining unaltered. In such a case the negative pressure in the pleuræ produced by pulmonary traction would undergo no change. Is it not therefore evident that we must keep the idea of pulmonary tautness and that of pulmonary elasticity distinct? I shall presently show that this is not merely theoretically necessary but that in actual life it is possible to get a great increase in pulmonary tautness and a corresponding augmentation of pulmonary traction though pulmonary elasticity be considerably subnormal.

But first a word as to the factors which determine "pulmonary traction." They are two—pulmonary tautness and intrapulmonary air pressure. Pulmonary traction varies directly with the one and indirectly with the other. These two factors are not only distinct but may at one and the same time work in opposite directions. Thus if at the end of a deep inspiration when pulmonary tautness is at the maximum a powerful expiratory effort be made with closed glottis, the increment in intrapulmonary pressure thus produced more than obliterates the traction effect of pulmonary tautness, and consequently the lungs no longer exert traction upon, but actually press against, the surrounding parts. Contrariwise, if at the end of a deep expiration, when the pulmonary tissue is relaxed and the lungs cease to exercise traction, a powerful inspiratory effort be made with closed glottis, the greatly lowered intra-pulmonary pressure thus effected will give rise to considerable pulmonary traction.

Pulmonary tautness then plays an important part in determining pulmonary traction, the two tending to rise and fall together. Pulmonary tautness, however, bears no constant relation to pulmonary elasticity. I shall prove this proposition by reference to two diseases—hypertrophous emphysema and fibroid phthisis. Dr. Samways remarks on the fact that the elastic tissue of the lung "strangely chooses from birth onwards never to assume the unstretched conditions." This stretched condition of the pulmonary tissue is effected by the inspiratory muscles. As I have elsewhere pointed out, the inspiratory muscles are throughout life ever on the watch to maintain pulmonary tautness at a certain mean level, with the object of maintaining a constant suction or (as Sir James Barr prefers to call it) "traction" on the heart, and thus facilitating diastole; the inspiratory muscles, in fact, play a considerable part in effecting cardiac diastole—constitute, in fact, an important diastolic force. Now let us take a simple uncomplicated case of hypertrophous emphysema insidiously coming on, as it so frequently does, without any bronchitis or cough about middle life. The lungs gradually lose their elasticity much in the same way as the skin loses its elasticity. Suppose, now, the mean size of the chest to remain the same; it is obvious that the tautness of the lungs will fall below the normal and that there will be a corresponding fall in pulmonary traction. But the mean size of the chest does not remain the same. The ever-watchful inspiratory muscles, in obedience to the physiological necessity just referred to, cause an increase in mean thoracic capacity and thus tighten the pulmonary tissue up to the normal, much as the violinist is compelled from time to time to tighten up the strings of his instrument. And thus, as the pulmonary tissue loses in elasticity with every advancing month and year, the thorax is made to increase in size and for a long time *the tautness of the pulmonary tissue, in spite of steadily diminishing elasticity, is kept at the normal and with it the resulting pulmonary traction* until at length a point is reached at which the increase in the size of the thorax can no longer keep pace

with the decrease in the elasticity of the lungs, with the result that pulmonary tautness and pulmonary traction sink below the normal. What better instance can I give of the need to distinguish between tautness and elasticity?

I now come to the case of fibroid phthisis. It is quite easy to prove that in this disease—in which all will admit there is an enormous diminution of elasticity—there may be a great increase in pulmonary tautness with a corresponding increase of pulmonary traction. Sir James Barr denies that the lungs are taut in fibroid phthisis. "Try and you will find that the fish does displace the water and the lungs are not taut and the intrathoracic pressure is not lowered." If the lungs are not taut, and if pulmonary traction is not increased, how are we to account for the "sinking in" of the upper part of the chest not infrequently met with in chronic phthisis as well as in the more acute forms of the disease? Instead, i.e., of the lungs exercising moderate traction on the upper part of the chest so that the inspiratory muscles have no difficulty in keeping that portion normally expanded, the lungs exercise a traction so great that the inspiratory muscles, strive how they may, are powerless to contend against it and the chest flattens in consequence.

It is needful to remember that pulmonary tautness is not necessarily equal throughout the entire extent of the lung. Under normal conditions, with non-adherent pleuræ and normal pulmonary texture, it tends to be. Thus when a purely abdominal breath is taken the lower part of the lungs expand first, but owing to the mobility of the lungs, rendered possible by the pleuræ, and owing to the uniform elasticity of the pulmonary tissue, the augmented tautness tends rapidly to diffuse itself throughout the entire lungs. When, however, the pleuræ are adherent, or when the structure of the lungs is profoundly modified as by the abundant deposit of fibrous tissue, one portion of the lung may be supernormally stretched while another part is only moderately taut. Suppose, for instance, that in its upper part the lung is highly fibrotic and the pleura is adherent, while in the lower parts there is emphysema, pulmonary tautness might be supernormal in the one part and subnormal in the other.

"Try and you will find that the intrathoracic pressure is not lowered." It is not clear whether Sir James Barr here means intrapulmonary or intrathoracic extrapulmonary pressure—two very different things. In another passage he refers to intrathoracic *or* intrapleural pressure, and presumably Sir James Barr is here referring to intrapleural pressure. Sir James Barr appears to assume that it is possible to measure pulmonary tautness and pulmonary traction by means of intrapulmonary pressure, but we have already seen that the two factors which determine pulmonary traction—pulmonary tautness and intrapulmonary pressure—may simultaneously operate in opposite directions. Sir James Barr not only denies that the lungs may be supernormally taut in fibroid phthisis but actually argues, or appears to argue, that pulmonary traction is necessarily lowered in this disease because the intrapulmonary pressure cannot in Müller's experiment be reduced to anything like the normal extent, say, only — 8 Hg mm., as against — 80 Hg mm. I freely admit that it may be possible to produce a higher degree of pulmonary traction in the normal individual by Müller's experiment than in the case of a patient suffering from fibroid phthisis. But people do not spend their lives in making Müller's experiment. Further, if Sir James insists upon dwelling upon the difference just referred to, I may point with equal force to the fact that in the contrary experiment of expiring with closed mouth and nares, the normal individual is capable of producing a very much higher intrapulmonary pressure than the patient with fibroid phthisis, and that therefore pulmonary traction is much more effectually obliterated and converted into pulmonary pressure in the former case than in the latter. Sir James's argument here cuts, in fact, both ways. What we have to do with, however, is not a temporary condition, voluntarily induced, but with habitual conditions, and, as a matter of fact, there is very little difference between the habitual mean intrapulmonary pressure of the normal individual and of the patient with fibroid phthisis. If, therefore, the average degree of pulmonary tautness is greater in the latter case than in the former the average degree of pulmonary traction must be greater.

Regarding my assertion that in cases of phthisis the mean size of the thorax may be increased, Sir James Barr says: "I always thought that fibroid tissue occupied less space than the portion of the lung which it replaced. I am afraid there is some confusion in Dr. Campbell's mind between fibroid tissue and the accompanying emphysema." Of course, fibroid tissue tends to occupy less space than the portion of lung which it replaces. For this reason, if the atrophied fibroid lungs are to increase in size and thus adapt themselves to the thoracic cavity, the mean size of which is constantly tending to be increased by the powerfully acting inspiratory muscles, something must give, and this giving involves not only the alveoli with the production of the emphysema to which Sir James refers, but also (what Sir James neglects to mention) the bronchi (causing bronchiectasis) and often also the tubercular cavities which tend to be pulled out by the taut lung tissue into spheroidal form; these yieldings manifestly all result from supernormal pulmonary tautness.

Sir James Barr asks why in fibroid phthisis my "heightened tautness of the lungs should allow the fingers to become bulbous"? the inference being (I presume) that by augmenting pulmonary traction it should facilitate, not impede, the circulation. The answer is surely obvious: in so far as the bulbous condition of the fingers in fibroid phthisis is due to obstructed circulation it results from the widespread destruction of the pulmonary blood-vessels. No amount of augmented traction on the heart could adequately compensate for this.

Commenting on my assertion that "comparatively non-elastic lungs, such as those seamed with scar-tissue, are capable of being rendered more taut, and thus of exerting more traction on circumjacent parts than normal, highly elastic lungs," Sir James Barr says: "This reasoning is that of the academician in his study and not that of the clinician who views facts and reasons therefrom. I shall now," he continues, "proceed to prick the bubble." This is his *modus operandi*: "Non-elastic fibroid lungs cannot be taut both in inspiration and expiration and therefore a constant negative pressure cannot thus be maintained." Quite true. Lungs wholly non-elastic could only become taut at the extreme limit of inspiration, and this to all intents and purposes means that with such lungs there would be no pulmonary traction, but, on the contrary, a positive pressure on the heart, and this would so interfere with the circulation, already sorely embarrassed by the block in the pulmonary circuit, that death would soon ensue. I admit that Sir James has pricked the bubble, but please note, Sir, that I am in no way responsible for the blowing of that poor bubble. Sir James himself is responsible for that, for it will be observed that in the passage he quotes from me I refer not to "non-elastic" but to "*comparatively* non-elastic" lungs—two very different things. While I believe it is possible for certain portions of the lung in fibroid phthisis (the apex, for instance) to be at one and the same time practically non-elastic, supernormally taut, and immobile, yet other portions must, if life is to continue, retain sufficient elasticity to remain taut during the ordinary respiratory movements.

To my contention that the enlargement in the mean size of the chest which may occur in fibroid phthisis is brought about by the inspiratory muscles, Sir James replied in his first letter that any stretching of the lungs that might occur was caused by the pressure of air within them and not by the inspiratory muscles. Referring to this I said in my last letter: "What has the average intrapulmonary air pressure got to do with the stretching of the lungs when the pleuræ are adherent? Manifestly nothing whatever," seeing that the pressure of the intrapulmonary air is counterbalanced by the extracorporeal air pressure. Dr. James Barr thus comments on this passage: "The question and answer coming from a physicist are really very funny. Does he not know that whether the lungs are adherent or not the thoracic parietes intervene between the external atmosphere and the lungs, and any force which expands the thorax beyond the expansile power of the lungs must exercise a force of 15 pounds to the square inch, and this the inspiratory muscles could not do over such a large surface as the chest though they tugged and pulled like Dr. Campbell at Cleopatra's Needle. The inspiratory muscles, like Dr. Campbell, often expend a lot of useless energy. How often do we grieve at the struggles of a poor asthmatic doing nothing?" Surely these remarks are beside the question. I nowhere made any mention of an "expansion of the thorax beyond the expansile power of the lungs," but simply referred to the moderate increase in the mean size of the chest which may sometimes be observed in cases of fibroid phthisis. Sir James's objections apply to such an expansion of the thorax as should separate the chest walls from the lungs

and leave a vacuum between the two, and not only would the inspiratory muscles be incompetent to do this, but no force which can be imagined, not even one equal to 15,000,000 pounds or 15,000,000 tons to the square inch of chest surface, would be competent to do this, for in the process of expansion the thoracic walls or the lungs would rupture long before such a vacuum could be brought about. As a matter of fact, the inspiratory muscles are competent to expand the chest to its potential maximum, as may be observed in advanced cases of hypertrophous emphysema.

May I, Sir, in concluding this long letter venture to express the hope that this time the energy expended may not be entirely lost on Sir James Barr. Certain it is that I shall be more than content if it proves as profitable to him or to others as are the respiratory struggles of the unfortunate asthmatic profitable to the latter, convinced as I am that those struggles, blind and purposeless as they may appear to the casual observer, are yet wisely directed towards a useful end, and that but for them no patient could battle through a severe asthmatic paroxysm. Nature is not always such a fool as some seem to think.

I am, Sir, yours faithfully,

Wimpole-street, Feb. 2nd, 1908. HARRY CAMPBELL.

## THE CAUSE OF THE PREVALENCE OF ADENOIDS.

*To the Editor of* THE LANCET.

SIR,—I am glad Dr. J. Sim Wallace does not "put any importance on" his figures, for even more startling deductions might be made from them. As his five shut-window families have 26 children and his five open-window families but 19 he might have argued that closed windows at night increased the number of children born, and closed windows might be recommended to the President of the United States as a cure for race-suicide! But joking apart, I do not think Dr. Wallace will find that his theory is supported by facts. I think the explanation of his numbers, if one is needed, is that adenoid families see a lot of the doctor and where the doctor rules the windows are open at night. If adenoids are on the increase it cannot be due to open windows, for windows that really shut are of quite recent introduction. The old leaded glass always leaked and it is only since it was superseded that the bed-curtains and night-caps of our parents have been done away with. I am, Sir, yours faithfully,

O. CLAYTON JONES, M.B. Oxon.

Silverton, Exeter, Jan. 29th, 1908.

## THE RESPONSIBILITY FOR THE ANÆSTHETIC.

*To the Editor of* THE LANCET.

SIR,—It is to be hoped that Dr. Dudley W. Buxton's paper in THE LANCET of Jan. 18th, p. 151, and your leading article thereon have been very carefully read not only by surgeons and anæsthetists but also by general practitioners. It happens far too commonly that a practitioner who calls in a surgeon to perform an operation—whatever the nature of this may be—suggests, and even requests, that he or his partner or assistant may give the anæsthetic, although there may be no adequate reason for not engaging a skilled anæsthetist. When such a request is made to a surgeon it is, of course, not easy for him to refuse, although he may have grave misgivings as to the result of his consent and although he may be well aware that the operator who permits a person of small experience to give an anæsthetic accepts a very serious responsibility.

I have no doubt such an arrangement is usually suggested in all good faith, for the majority of practitioners whom I have met do not seem to realise that for the proper conduction of a large proportion of the operations done at the present time special skill and experience are as needful in the anæsthetist as in the surgeon, and that the average practitioner cannot expect to be any more capable of replacing the one than the other. It is necessary not only to know the kind of anæsthetic best suited to the patient and to the operation but also to be well acquainted with the surgical procedure in order that pitfalls may be avoided. This is especially the case with operations on internal organs and above all with those (even "trivial" ones) on the upper air-passages, where safety and success can only be attained by a knowledge of the operator's methods and a perfect coöperation between him and the anæsthetist. So many instances have come to my knowledge where want of special experience in a self-constituted anæsthetist has led to inconvenience and even disaster that I venture to trouble you with this letter. I am, Sir, yours faithfully,

Jan. 25th, 1908. F.R.C.S.

## THE PRESENT PROSPECTS OF THE MEDICAL PROFESSION.

*To the Editor of* THE LANCET.

SIR,—The remarks of "Inspector-General" in your issue of Jan. 25th are worthy of the very greatest consideration, and call for more than ordinary passing comments in your correspondence columns. Indeed, a change of government in the whole attitude and conduct of modern professional manners is sadly needed. Everything is moving too quickly nowadays and this is not foreign to ourselves. There is a rush, a congestion, and a competition in our ranks which is decidedly tending to disorganise the enthusiasm and high aims of those who formerly filled the ranks of the profession. There is, in fact, disorder. That this is the outcome of education and general social changes is clear. But to stave off this wave of socialism in medicine needs a stronger bulwark than the lean-to wall of your valuable professional journal or any other medical publication of your standard.

Thus, Sir, my fragmentary remarks in THE LANCET in support of "Inspector-General" may possibly only react proportionately as a grain of sand. But medicine is full of hope and, although slow in results, nevertheless a number of grains of sand may affect the whole problem before us—viz., the present, nay probably the future, prospects of the medical profession. We are apt to throw stones at glass windows and to blame others for a great deal that befalls the profession when, if we weighed the matter thoughtfully, perhaps we ourselves are sore. There are hundreds of men in the profession striving to aim at good deeds but they are baffled by those whose methods and practice are not pleasant to behold. If we despise the lay advertiser why should this not apply to ourselves, may I ask? Is not personal advertisement contrary to the ethics of the profession of medicine? Why, then, do gentlemen send open postcards and the like through the post seeking patients? Are there no censors in the profession? And, what is worse, how few of us have the courage of their opinions and perhaps are too shy to utter condemnation on personal advertisement? But is not the canker worm of self-advertisement a sign of stress—an ominous warning for the future of the profession for which some drastic measures will have to be devised sooner or later? It is impossible for a whole body of men to travel along a corduroy road for a length of time without several falling out of the ranks.

Again I will draw attention to another lapse in medicine among the present generation—namely, the using of proprietary drugs and other medicaments foreign to the order of the British Pharmacopœia. What has become of the good old pill may I ask? And if we do not actually practise empiricism do not we sail very close to the wind and encourage that class of the laity who deal in such things? Altogether, from the drug point of view, are we not getting very muddled?

As concerns our practice, there is no definite ruling as to what constitutes the exact duties of the general practitioner, because his need to earn a living wage encourages him to be Jack of all branches of the profession. Hence arises specialism, but truly there is more dabbling in the specialties of the profession by its members than is justified. And it is questionable whether this dabbling by the recently qualified is not indirectly an explanation of the oft-repeated public censure which the profession as a whole has to endure. In the army we see things are differently managed, for there is a stage of probation, then successive periods, including an examination, before the young man reaches the title of a colonel. And in general civil practice we ought to see something similar, so that a junior should not rank as high as his senior who has practised for many years. The licensing bodies might renew by fresh reception, at varying periods, an extension to practise, gradually weeding out those who by age, incompetency, or irregular practice rightly cease as practising members. Above all, before a candidate qualified to practise leaves the threshold of the licensing body, be it the Royal College of Physicians or the Royal College of Surgeons, there should not be the slightest doubt as to