An inspection shows that while in the case of mitral stenosis there is an increased percentage of those who indulge in heavy exercise as we pass from a history of rheumatic fever to no history suggestive of rheumatism, the increase is in no way comparable with the increase in the case of pure aortic lesions; and, further, that as we pass from pure mitral lesions to pure aortic lesions where no history of rheumatism exists, there is again a steady increase in the percentage engaged in heavy exercise. Another remarkable fact that this table reveals is that the distribution between light, medium, and heavy exercise is practically the same for both aortic and mitral cases where there is a history of preceding rheumatic fever. The deductions which we are inclined to draw from these tables are that while strain has possibly some effect in producing mitral stenosis, it is nothing like so important a factor as it is in the production of aortic regurgitation. From the extremely similar numbers in aortic and mitral cases in the various strain groups, we cannot help suspecting that the disability that rheumatism causes, and which determines the amount of exercise or work the patient does, is more dependent upon some common factor, such as damage to the cardiac muscle, than on the particular valve affected.

When we were dealing with aortic regurgitation we compared the number of cases engaged in heavy, light, and medium labour who gave no history of acute rheumatism with numbers calculated on the assumption that strain and rheumatic fever were the two chief causes of aortic regurgitation, and we found a remarkably close agreement between the calculated and the actual figures; when we applied this method to the mitral stenotics the difference between the actual and calculated numbers was so great as to negative such an assumption, which again leads us to the same conclusion as the other figures in the present communication—namely, that there is another cause of mitral stenosis besides rheumatic fever, which is not strain.

SUMMARY.

Taking aortic regurgitation and mitral stenosis as the two typical valvular lesions which can be diagnosed with comparative certainty during life, the results of the present statistical inquiry lead us to the following conclusions:

1. The most important etiological factor in the production of organic lesions of the heart valves in men between the ages of 18 and 41 is rheumatic fever.
2. Strain can independently produce aortic regurgitation.
3. While strain may be a cause of mitral stenosis, it is a much less important factor than in the production of aortic regurgitation.
4. There is a third agent giving rise to symptoms simulating influenza, which is an efficient cause of mitral stenosis, and can possibly affect the aortic valves as well, but is not nearly so likely to do so.

We have been unable to find any statistical evidence that syphilis, scarlet fever, diphtheria, pneumonia, or gonorrhoea, are causes of valvular lesions, nor can we find statistical evidence that a history of growing pains points to affections productive of valvular lesions. The same may be said, with slight reservations, of a history of rheumatism where there is no clear account of pain, swelling of the joints, fever, or confinement to bed. The evidence in favour of chorea as an efficient cause of mitral stenosis is strong, and we have found some, though not very striking, evidence in confirmation of the view that chorea and rheumatic fever are both manifestations of the activity of a common cause.

THE IMMEDIATE CLOSURE OF EMPYEMATA.

BY

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THERE is nothing new under the sun, and I was very much surprised, after I had drafted this paper, to be told by a friend of mine that twenty-five years ago it was the custom at King's College Hospital, under Lord Lister, to wash out their cases of empyema and sew them up. The fact remains, however, that when I returned from France late in 1917, it struck me that in the treatment of

empyema it was not sufficient merely to open the abscess and put in a drainage tube, but that something more should be done and the wound immediately sutured.

These short notes are the result of my experience since I published my first successful case in a paper on the abuse of drainage tubes¹ in 1918, when I thought I had struck something original and really good. Although I am not a pioneer in the immediate closure of empyemata, I hope that the success I have met will stimulate others to follow the lines of treatment I find by experience to give the best results.

It stands to reason that the simple resection of rib, letting out of pus, and putting in of a drainage tube is not the ideal and complete treatment of empyema. Something more is wanted, because re-expansibility of lung is far more important than the mere evacuation of pus, and this re-expansibility can only be produced in two ways: (1) By introducing the whole hand into the pleural cavity and freeing the lung of all adhesions and removing all fibrinous clots; (2) by immediate closure of the wound.

Those who have had experience of chest surgery in France learnt that the first thing was to do away with a "sucking wound." Why, then, should we by the use of a drainage tube deliberately establish one in the treatment of empyema? By using an open tube the atmospheric pressure in the pleural cavity must be greater than that in a collapsed lung, and in order to get a lung to re-expand as quickly as possible it is essential to produce a vacuum in the pleural cavity. This result can only be attained by immediate closure of the wound, after filling up the cavity with some fluid.

Every surgeon in France was surprised to observe how quickly the lung re-expanded after immediate closure for gunshot wounds, and the same holds true for empyemata. Breath sounds will be heard down to the base of the lung within a few days after operation, and the lung will rapidly push out the fluid left, so that it comes to lie under the skin and can be easily and painlessly evacuated.

Therefore we may lay it down as an axiom in chest surgery that the lung will quickly re-expand if only it is given a fair chance. This favourable condition can only be secured by freeing the lung from all adhesions and by closing the wound at once.

If these two main rules are carried out, we shall do away with the old-standing cases of empyema which go on from year to year, with collapsed lung and a persistent sinus, and eventually have an enormous Estlander operation performed. Is it too good to hope that this operation will become a thing of historical interest only?

As I have pointed out elsewhere, there is no reason why a pneumococcal infection should be treated on lines different from a tuberculous infection. In either case, by using an open drainage tube, we are asking for trouble by producing a secondary mixed infection.

It is important to note that a careful bacteriological examination is essential in every case of empyema. If pneumococci or pneumococci and staphylococci are present the wound may be sutured; but if streptococci are present, then I think it advisable to remove the stitches and treat by some open method—that is, Carrel-Dakin, packing with gauze, or by a drainage tube.

Operation.

I have, since my return from France, sewn up all my cases, and my experience up to date has taught me that the following are the best lines:

I have always used a local anaesthetic, but I am inclined to think that when handling the lung a little general anaesthetic with it is useful.

I resect sufficient rib, either a long section of one rib or adjacent parts of two ribs, to allow of the introduction of the whole hand into the pleural cavity. This is the first most important point, because, after evacuation of the pus, it is essential to pass the hand into the chest cavity and strip off the collapsed lung all adherent fibrin and separate the lung from all adhesions. In any early case this is easily done, but an old-standing one will require much freeing of the lung in order that it may recover its re-expansibility. I then wash out the pleural cavity with flavine till the fluid comes away quite clean, and fill up the chest with a 2 per cent. suspension of iodoform in sterilized paraffin. The pleural wound is then sewn up with catgut, not that I think this is very important, because these stitches soon give way. I then carry out immediate

closure of the skin wound with deep sutures, so as to leave no "sucking wound."

The next important point is the after-treatment. When dressing on the next and following days the wound will be bulging; this is due to a mixture of pus and iodoform and paraffin being pushed out of the pleural cavity by the expanding lung under the skin. Daily I introduce the needle of an exploring syringe between the edges of the wound—this is quite painless—and extract all the fluid I can. This process usually takes ten to fourteen days, depending on the re-expansion of the lung. Daily bacteriological report of this fluid shows that the number of organisms in a field steadily diminishes. A certain amount of this mixture of pus and iodoform and paraffin will ooze out also into the dressings.

The two most important points in technique, then, are—

1. Introduction of the whole hand into the pleural cavity to separate adhesions and remove all fibrin. The lung must be made to re-expand.

2. Daily evacuation by an exploring syringe of the pus and paraffin as it is pushed out under the skin by the re-expanding lung.

I have now sutured 9 cases. The ages of the patients were 55, 53, 44, 30, 27, 25, 18, 4, and 2½. All were pneumococcal except two; one was staphylococcal as well as pneumococcal, and one which was my only failure; this was a tuberculous case—a man, aged 44, with two big abscesses pointing under the skin, one behind and one in front. He was very debilitated and ill. As it was one of my early cases I did not sufficiently explore the chest with my hand; had I done so I should have found, as I did *post mortem*, that the lung was tied in a knot at the root. I sewed the incision up, and the patient died in twenty-four hours. After seeing the condition of his chest I am quite sure the result would have been the same had I drained with a drainage tube or sewn up.

Of the other eight cases, all made good and rapid recoveries. The temperature fell at once, and the pulse and respirations came down more gradually.

The duration of the disease in these cases varied from two to six weeks, after the resolution of the primary pneumonia.

A further suggestion has occurred to me, but I have not lately had an empyema to try it on. It is this: Whether it would not be a good plan to insert into the pleura a soft piece of folded rubber—that is, a passage tube—in order gradually to let out the iodoform and paraffin, and to do away with the daily needling of the collection under the skin. This piece of folded rubber will not allow air to enter the pleura, and will yet slowly allow the escape of pleural contents.

REFERENCE.

¹ BRITISH MEDICAL JOURNAL, 1918, i, D. 718.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

A CASE OF FACIAL TROPHONEUROSIS WITH DEFORMITY OF THE HAND.

As progressive facial hemiatrophy is a rare condition, the notes of the following case observed during the examination of recruits will be of interest.

A boy, aged 17, presented the following structural abnormalities, which are seen in the photograph.

Bones.—There was atrophy of all the bones of the right side of the skull, including the cartilaginous portion of the external ear. The metacarpal and phalangeal bones of the thumb and right index finger were atrophied and deformed.

Muscles.—Wasting was noted on the right side in the muscles of the face and of the thenar eminence, and to a lesser degree in the muscles of the neck; the right sterno-mastoid, however, was well formed. The right upper limb was poorly developed although he was right-handed.

Nerves.—Epicritic and protopathic sensations were everywhere normal; in no muscle was there palsy.

Other points of interest were that the right carotid and radial pulses were perceptibly weaker than those of the left side, and that the skin of the affected side was of a more delicate texture.

The boy is a junior member of a family of fifteen, and I have excluded as far as possible the question of natal trauma with resultant torticollis and facial asymmetry.

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FOREIGN BODY IN BRONCHUS.

THE case reported in the JOURNAL of May 1st, p. 602, by Mr. W. C. Cammock, in which a toy air balloon was present in the left bronchus, recalls a similar one with which I had to deal about three years ago.

A boy, aged 11, when playing in the street, inhaled a small rubber balloon with wooden mouthpiece which caused immediate and complete respiratory obstruction. This was relieved by tracheotomy performed by a doctor near whose house the boy was at the time. He was then taken to the Liverpool Royal Infirmary, and when I saw him there soon after his arrival he was suffering considerably from shock, and surgical emphysema, originating no doubt from the tracheotomy wound, was present over the neck and a great part of the trunk. The boy's condition at the time did not justify any attempt to find and remove the foreign body which was probably then in the larynx. On the following day the rubber balloon without the wooden mouthpiece was coughed out, and the boy could breathe and phonate clearly on closing the tracheotomy opening. The surgical emphysema disappeared and his general condition improved, so that six days after the accident I considered it safe to endeavour to remove the mouthpiece which was presumed to be lying in some part of the bronchial tree, although there were no physical signs in the chest giving any clear indication of its position. Inferior bronchoscopy at once revealed its presence in the right main bronchus, from which it was removed without difficulty. The absence of physical signs of obstruction of the bronchus was due no doubt to the fact that air had passed freely through the lumen of the foreign body as well as between its walls and those of the bronchus in which it lay quite loosely.

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CARBOHYDRATE EXCESS AND BERI-BERI.

CONSIDERABLE interest attaches to the fresh light thrown upon the pathogeny of rickets and beri-beri by Professor Mellanby in his opening remarks at the Section for the Study of Disease in Children of the Royal Society of Medicine on February 27th, 1920. While admitting that administration of the suitable vitamins in rickets and beri-beri will effect a cure, he holds it not improbable that the two diseases are brought about by some other elements in the diet, probably a carbohydrate excess, together with some considerable protein deficiency. Such facts are in agreement with results of observations I made some years ago upon the former diets of beri-beri patients, in which, generally speaking, in addition to the lack of the particular antineuritic vitamin, carbohydrate was found to bulk largely in the diet, with insufficient intake of protein derived principally from polished rice and dried fish.

The epidemiological distribution of beri-beri still, however, to some extent remains unsolved, in its special incidence upon sea-coast places, in which immigrants specially suffer, whilst residents partaking of an identical diet almost invariably escape. In regard to occupation I found a large number of those engaged in sedentary trades, such as the Chinese tailors in Singapore, specially prone to the disease.

Whilst I hesitate to introduce a further causative factor in beri-beri, it may well be that a degree of acidosis is the ultimate determining agent in the production of neuritic symptoms, a condition now known to be liable to arise in new arrivals to the hot moist climate of the seaboard towns of the Straits Settlements, being aggravated by the already mentioned dietary deficiencies and excesses, against which the Straits-born inhabitants have become more or less immune through prolonged residence.