A QUESTION FOR ANÆSTHETISTS.

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It must have occurred in the experience of almost every hospital surgeon that a day or two after an operation of very trivial character, where an anæsthetic has been administered, the patient's temperature has been observed to rise very high, the wound has been examined and found perfectly quiescent, devoid of every indication of inflammation, and completely healed, yet the skin is burning and the patient obviously ill; next a little cough is noticed and soon rusty sputum is coughed up, indicating pneumonia. These cases have occurred too frequently in my experience to be explained either upon the hypothesis of infection prior to admission into the hospital or upon the conjectured basis of chill taken at the operation, where every precaution has been taken to obviate unnecessary exposure. I have noticed it happen on several occasions after operations for hernia, when it might be suggested the patient's resistance might have been lowered by previous vomiting; but I think I may say that I have observed it as frequently after many other operations of more trivial character. The most recent case, at present under my care in Guy's Hospital, is that of a healthy young man admitted for what is now one of the commonest and simplest operations, the aseptic excision of a varicocele. Gas and ether were administered on Feb. 4th and the varicocele was excised, and the wound closed and dressed with aseptic pads. His temperature rose and on the 6th it reached 103° F. His wound was examined and found to be perfectly quiet. On the 7th he began to expectorate rusty, blood-stained sputum, and the expectoration on being examined was found to contain the pneumococcus.

It may, of course, be said that this young man was in the incubation period of pneumonia when admitted, and that he simply developed it in the ordinary course after the operation, but I think these cases, though occurring apparently sporadically, do occur too frequently in hospital practice not to have some other explanation. Gas and ether, it may be urged, are powerful stimulants to the lungs, and sufficient to account for subsequent inflammation. Undoubtedly bronchial conditions frequently follow their administration of sufficient severity in old people to prove fatal. But here is a young, healthy man clearly infected with the infective germ of pneumonia. He was operated on in a well-warmed theatre and placed in a bed on the most protected side of the ward. But apart from this, exposure to cold can no longer be regarded as a sufficient cause of pneumonia. It can only predispose thereto. One might as well argue that cold would cause phthisis without any exposure to the tubercle bacillus.

The question I wish to ask of anæsthetists is whether the apparatus used by them may not sometimes be responsible for germ infection of the lung? I speak of hospital practice, now, where some dozen apparatuses are being constantly employed, carried from patient to patient without any attempt at sterilisation. Considering the enormous advance made of late years in every department of surgery for the better disinfection of everything brought into contact with a wound, it has struck me as somewhat marvellous that so little improvement in this direction has been made by those responsible for the inhalers used for giving anæsthetics. I have often thought it not altogether pleasant to see an anæsthetist blow out and distend the large bag used for ether administration by the breath of his own body, however sweet that may be, and then apply it directly over the patient's mouth. But as matters stand now there is no apparatus used in hospital practice, so far as I am aware, for the administration of either the A.C.E. mixture, or gas, or ether, which is capable of being boiled, or which is otherwise disinfected before being applied over the mouth. If, as I am suggesting (and it may be capable of bacteriological proof), that the apparatus used for giving the anæsthetic, and not the anæsthetic itself, may be responsible for lung infecting inflammations, then some radical change will soon have to be made in the apparatus, which, going from mouth to mouth as at present, seems capable of carrying infection of a kind such as I have indicated.

Wimpole-street, W.

Clinical Notes:

MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

RARE CONGENITAL MALFORMATIONS.

By C. KESSICK BOWES, M.B. OXON.

ON Dec. 30th, 1896, a woman was delivered of a female child at full term after a natural labour, this being her second confinement. Directly the child was born I noticed that the hands presented a very unusual appearance, the fingers pointing upwards towards the elbow, and on examination I found that there was entire absence of both radii and both thumbs. When at rest the outer side of each hand—i.e., the index finger and its metacarpal bone—rested against the forearm, but by manipulation the hands could be brought down at right angles to the forearm, but no further, and there was great resistance to supination. The ulna in each arm was a little curved and seemed shorter than normal, but in all other respects the child seemed to be perfectly developed. On the third day, as soon as the mother's breasts became full of milk, she noticed that as the baby sucked the nipple, after drawing two or three times, the milk returned through the nose, and she had to take her from the breast. I examined the child's mouth and found nothing to account for it, and as this regurgitation continued, and the child began to waste, I suspected some obstruction in the œsophagus. I did not pass a bougie, as the nurse told me that the child had frequent attacks of cyanosis with much choking, and she seemed so fragile and weak that I did not think it would be wise to attempt it. In this way she continued to live, but gradually wasted, and eventually died from exhaustion when thirteen days old.

I obtained permission to make a post-mortem examination and found that the upper part of the œsophagus was simply a cul-de-sac extending about three-quarters of an inch below the larynx, and that the lower part, as it came up from the stomach, opened into the trachea near the point of its bifurcation. The stomach and intestines were quite empty and very thin, just as one would expect to see them in a case of starvation. On opening the heart I found that the interventricular septum was not complete, being deficient in its upper part.

The case is an extremely interesting one, not only on account of the rare malformations, but also on account of the length of time the child lived without any nourishment whatever.

Herne Bay.

POISONING FROM EATING ROOT OF SCARLET RUNNER BEAN.

By J. S. MACPHERSON, L.R.C.S., L.R.C.P. EDIN.,
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THE following account of poisoning from eating the root of scarlet runner bean may be of interest to the profession, as I am not aware of any similar previously recorded case.

On the night of Nov. 18th, 1896, a large number of our garrison, comprising Soudanese soldiers and their wives and Swaheli porters, partook of a small quantity of the root of the scarlet runner bean, which had been collected by our native gardener. Several of the men had the root roasted, while others, probably the majority, simply ate it in the raw state. In a short time—i.e., within a couple of hours—they were all seized with symptoms of poisoning, twisting pain in the stomach and violent vomiting, accompanied by vertigo and prostration. As there was nothing to indicate the precise nature of the poison I had at once recourse to an emetic of thirty grains of zinc sulphate largely diluted with hot water, resulting in prompt emesis with immediate beneficial results. The following morning most of them simply complained of vertigo with an uneasy, twisting stomachic pain, though several suffered more than others from prostration and weak

cardiac action. The administration of a purgative dose of sulphate of magnesium with the addition of a few drops of diluted sulphuric acid removed all remaining symptoms, though the vertigo lasted in several cases for from twelve to eighteen hours from the time of eating.

There could be no mistake as to the root in question being that of the scarlet runner bean, as it was planted by ourselves from London seeds and grown in the garden amongst peas, cabbages, and cauliflower, and I cannot conceive unless the root is *per se* poisonous, how it could have absorbed poisonous material from any neighbouring plant. I may mention that the bean itself was largely partaken of by ourselves and we never observed any noxious effect. I forward you a specimen of the root in question, also the plant and bean for analysis. Possibly the deleterious substance may be analogous to that contained in the poisonous variety of cassava or deadly manioc root which is referred to in Mr. Stanley's book "In Darkest Africa."

Ravine Station, Uganda Protectorate.

CHILD IN UTERO CRYING DURING LABOUR.

By E. FITZGERALD FRAZER, L.R.C.S., L.R.C.P. IREL.

I WAS in attendance on a case of twin labour here recently where I applied the forceps to the head of the first coming child. Whilst using gentle traction, and the vertex being still upon the perineum, I was surprised, as was also the nurse, to hear the child cry. At first we thought we were deceived, but in a very short time after the sound was repeated, and before any part of the head was born the same phenomenon occurred several times. Nothing unusual happened during the birth of the second child, which came away naturally. I cannot say whether the pressure of the forceps had anything to do or not with causing the crying. There were no marks of it on the head and the boy is doing well. This instance illustrates the possibility of uterine breathing, as otherwise I cannot see how the vocal cords could have been called into play. I attribute the entry of air into the lungs as having taken place during the time I was putting the forceps into place in doing which no difficulty presented itself. Thinking the particulars may be of interest to the readers of THE LANCET I have pleasure in submitting them.

Brighton.

A Mirror

OF

HOSPITAL PRACTICE, BRITISH AND FOREIGN.

Nulla autem est alia pro certo noscendi via, nisi quamplurimas et morborum et dissectionum historias, tum aliorum tum proprias collectas habere, et inter se comparare.—MORGAGNI *De Sed. et Caus. Morb.*, lib. iv. Proœmium.

ROYAL UNITED HOSPITAL, BATH.

A CASE OF ULCERATIVE ENDOCARDITIS TREATED BY
ANTI-STREPTOCOCCIC SERUM.

(Under the care of Dr. A. E. W. FOX.)

THIS case illustrates one of the more severe types of malignant endocarditis. It is published to show the results of the injection of anti-streptococcic serum, the use of which was decided upon from a perusal of the notes of a successful case which we brought forward in the Mirror of Hospital Practice. The duration of illness before treatment by the serum was about the same in both cases, but the symptoms had been more severe in this patient. The value of the treatment will most probably be found to depend largely on the time the disease has existed before the treatment is commenced. The difficulty consists in making an early diagnosis; but when the diagnosis has been successfully made the method of treatment by the serum has proved of great value in other forms of septicæmia. The publication of cases in which it has been tried, if they illustrate important points, is of much value, although the ultimate result may not be satisfactory. For

the notes of this case we are indebted to Dr. W. H. Cooke, resident medical officer.

A man, aged thirty-six years, was admitted to the Royal United Hospital, Bath, on Nov. 17th. He stated that he had not had rheumatism or syphilis; he had been an abstemious man and always lived in England. His illness had commenced seventeen days previously with cold shivers and pains, which commenced in the back. These pains disappeared from the back with the aid of strong turpentine embrocations, but reappeared in the limbs and head. He had had cold shivers daily and became very feverish. There had been no vomiting or diarrhoea. His urine was examined three days before his admission; it smelt strongly of violets. Cold nitric acid produced a cloud, which appeared at the top of the urine first and cleared up on boiling. This was considered as due to turpentine absorption. There was no albumin. On admission to hospital on Nov. 17th he was found to be well-nourished. His complexion was pallid and muddy. The conjunctivæ were slightly jaundiced. The tongue was slightly coated, white, and moist. The temperature was 101·8° F.; the left pulse 100, of good strength; and the respiration 20. There was no distress of any kind, but he complained of some pain in the right arm and right occipital region. There was no rheumatic odour, no œdema of the legs, and no effusion into any joint. The right radial artery was not pulsating and a clot could be felt in the middle third of the brachial artery. It was not tender. The anterior and posterior tibial arteries of both legs were pulsating. The chest was well formed, resonance was good, and the breath-sound normal. As to the heart, relative cardiac dulness commenced at the third left rib and was absolute at the fourth. The apex was in the normal position. At the apex the first sound was reduplicated and the second part of it ended in a slight whiff; the second sound was pure and distinct. At the base, over both the pulmonary and aortic valves, a faint soft systolic murmur was audible. With regard to the spleen, dulness commenced at the upper border of the eighth rib and continued to the lowest border of the ribs; it could not be felt. The liver dulness was increased downwards. The urine was of normal colour, 1020, acid, with no albumin. On the 18th the temperature at 8 P.M. rose to 104·6°. He did not shiver, but sweated profusely. There was no rheumatic odour. On the 19th there was no change in the cardiac signs. On the 20th he had had much pain in the calves during the preceding night. There was no blocking of the anterior or posterior tibial arteries. He was sweating profusely. The bowels were rather constipated. He had been given an injection of 10 c.c. of anti-streptococcic serum on the previous day at 1 P.M. There was no rash or any other discomfort from the injection. On the 21st the pain in the legs had gone. He was sweating profusely. He took nourishment well. The urine was 1020, acid, with a trace of albumin. The systolic murmur at the apex of the heart was becoming more distinct. On the 22nd he had had a better night, but felt rather faint in the morning. He had had some return of the pain in the calves. On the 23rd he had two injections of 10 c.c. each, and similar injections were now made every day. The systolic murmur at the aortic orifice was soft, whereas at the pulmonary orifice it was rough and loud (? two separate murmurs). The pulmonary was louder than the aortic second sound. A loud, rough systolic murmur was heard in the mid-sternal region, probably exocardial. On the 24th he was not so bright in the morning; he had had a bad night. The murmur at the apex of the heart was becoming louder and rougher each day. On the 25th he sweated profusely. There was no pulsation in either of the tibial arteries or the popliteal of the left leg. The leg was only slightly cooler than the right. On the 26th there was no change. On the 27th he felt a little better. The systolic murmur at the base of the heart was now loudest over the aortic orifice, the second sound being short and sharp. The British Institute of Preventive Medicine reported of blood taken from the left arm three days previously: "Streptococci not found either microscopically or by cultivation. No micro-organism has been isolated." On the 28th he felt and looked a trifle better. He had not had a good night. About the lumbar and lower scapular regions much boggy œdema had appeared from the numerous injections in the scapular regions. The left leg and right arm were of the same temperature as the corresponding limbs. On the 29th the injections were left off. On the 30th the temperature, which fell to 100·4°, had risen again