of *B. sporogenes* and *B. bifementans* subcutaneously, produced no visible effect on the development of rickets in rats.

4. The deficiency of the diet in calcium alone can produce changes in the skeleton of rats which present some resemblances with rickets, especially when the young rats have originated from a mother kept on the same diet during lactation.

5. Usually deficiency of food in vitamin A produces in rats impoverishment of the bones in calcium, enrichment in water, and osteoporosis with deficient osteogenesis, and in some cases a picture resembling slight rickets. Changes in the skeleton more similar to rickets, and in some cases typical of rickets, were observed in young rats on —A diet, provided their parents had been fed on —A diet during conception, pregnancy, and lactation.

6. Vitamin A has a relation to the metabolism of calcium in the organism and particularly in the bones, and therefore to the development of rickets.

7. My few experiments on the feeding of parents on food deficient in vitamin A or calcium during conception, pregnancy, and lactation suggest that this may start disturbances of metabolism in the child which, if the deficient dietary be continued after birth, result in serious disorders of the skeleton.

8. The changes typical of rickets occur most readily and most frequently in rats kept on a diet deficient both in vitamin A and calcium.

9. Castration performed before the commencement of feeding has no marked influence on the chemical and histological changes in the skeleton of rats fed on a normal diet, on diets deficient in calcium or vitamin A, or deficient in both.

In conclusion, it is my pleasant duty to record my gratitude to the Medical Research Council for affording me a grant which has enabled me to carry out this investigation, and to the Lister Institute for the hospitality of its laboratories. I am also deeply grateful to Professor C. J. Martin for his continuous sympathy and support. To Dr. R. Robison I express my thanks for advice connected with the chemical investigations; to Miss E. Luce for assistance in operations; to Miss S. Rutherford for help in the work of feeding the animals.

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THE SITE OF OPERATION FOR EMPYEMA.

BY

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THE best method of treating empyemata has always been, and probably always will be, discussed from time to time in the medical press. As a surgeon attached to a general, a children's, and a consumption hospital, consulting surgeon to a second consumption hospital, as well as having seen over five years of war, perhaps I might be allowed to express my views. In the first place, there is a sharp distinction to be drawn between the cases seen as the result of warfare and those which develop as the result of disease. There is nothing in civil practice which can reproduce what was seen during the war. Even stabs or bullet wounds in civil life are treated so soon and so

thoroughly that the sepsis we saw during the war is not seen in these cases. The methods of dealing, therefore, with war cases do, and should, differ from the methods we use in civil life, and anyone who draws on his war experience too largely will be apt to err if he applies his ideas to ordinary cases. I shall only deal here with ordinary cases. It has been my fortune twice in my life to see the condition known to our fathers as empyema necessitatis—that is to say, an empyema which had not been opened but which was bursting by itself. It was interesting to note that in both cases it was bursting between the second and third ribs in front. Both cases were in children.

In dealing with a large abscess cavity with only one movable wall—namely, the lung—it is obvious that the drainage must be so arranged as to allow the lung to come up to the other walls gradually and evenly, and so obliterate the cavity. Opening at “the most dependent attainable part of the cavity” is the best way to defeat this object. The lowest and the highest points will be those where the lung on expanding will first touch the chest wall, and when that happens drainage from these points must be embarrassed. The same thing applies when the opening is either too far back or too far forward. The opening should be opposite, roughly, the deepest part of the cavity, and at that place which will be closed last. Where this place is, is not always easy to say, especially in purely local collections. The diagrams perhaps illustrate my meaning best.

One always teaches that the seventh rib, in front of the posterior axillary line, is the best rib to resect, but there should be no objection to coming forward to the mid-axillary, or even to the anterior axillary line, if the same level were maintained—that is to say, resecting the sixth, fifth, or even the fourth rib, as one came further and further forward.

It is the ninth rib we resect in cases of abscess or hydatid of the liver on the right side and subphrenic abscess on the left side. The ninth rib is wholly below

FIG. 2.—Cross-sections of the chest, showing posterior part of cavity obliterated by expanding lung.

the normal lung level in front of the posterior axillary line, and the eighth rib is below that level in front of the mid-axillary line. Only right up behind, near the erector spinae, would it be of any use resecting this rib, and then only in such a position that the expanding lung would soon render the opening useless. In spite of the authority lent to this teaching, the resection of the ninth rib for empyemata has had a very limited vogue.

In many cases the pouch of pleura between the chest wall and the diaphragm becomes obliterated below the lung level, and after resection of the ninth rib the diaphragm may be opened under the impression that it is the thickened pleura. I once saw Mr. Keetley do this, and the medical registrar, to whom he had not been very polite over the case, acidly remarked that he had wished the thoracic and not the peritoneal cavity explored.

Small operations for necrosed pieces of ribs will always be needed. The maintenance of big unhealed cavities inside the chest wall is due to certain very well known

causes. The commonest of these is bad drainage, where the lung itself has blocked the free outlet. The commonest condition found is a narrow passage into the cavity. It is these cases which, when allowed to continue too long, present for us the thickened pleura which calls for such severe methods as decortication.

In the majority, the vast majority, of such cases, which require a more or less extensive resection of ribs to allow the chest wall to fall in upon the stationary lung and so obliterate the cavity, the opening is situated too low down for good drainage. I can only now recall one, an old tuberculous case, which was as high as the sixth rib.

I am fully in accord with the three essentials laid down by Dr. Pearson, but would strongly advocate the superiority of the older method laid down by John Marshall, and brought again to our notice by Dr. Cameron Kidd, over the newer ninth rib route.

THE INTRAVENOUS INJECTION OF ANTIMONY TARTRATE IN JAPANESE BILHARZIA DISEASE.

BY

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I HAVE been awaiting for many months now the publication of accounts of the treatment of Japanese schistosomiasis by antimony tartrate. I have not seen any record in the British medical journals. It is possible that the remedy has been tried in Japan and that the account of the trial has been published in the local medical press and so has escaped my notice.

In 1919 I sent copies of two of my papers to various Japanese medical authorities notifying the success which I had obtained in the treatment of Egyptian bilharzia disease at Khartoum by antimony. The worms of Egyptian bilharzia disease and Japanese schistosomiasis are so similar anatomically and biologically, and the life-history of the Japanese bilharzia is so like that of *Schistosoma mansoni*, that one would expect that the remedy which has been found to be a specific cure in the one would have the same effect in the other disease.

I venture to think, therefore, that the following case which supports this hypothesis will be of interest to medical men, and I trust that Japanese and Chinese investigators after seeing this account will write and publish, in an English medical periodical, the conclusions, favourable or unfavourable, at which they have arrived in cases where they have applied the antimony treatment. It is in the last degree unlikely that antimony tartrate has not yet been tried in Japan and China by Japanese, Chinese, or by European medical men working in districts where bilharzia disease is prevalent.

T. A. R., a British engineer aged 50, was infected by *Schistosoma japonicum* during the years 1898-1901 whilst surveying in the "paddies" in the Hunan Province of China. Although he felt weak and pulled down and had constant colicky pains in the bowels, the disease was not diagnosed till 1914, when the ova of *Schistosoma japonicum* were found in the faeces in a specimen which was being examined for *E. histolytica* (at Nairobi); ova of *Schistosoma japonicum* were again found in the faeces in 1916 by Dr. J. A. Thomson of Harrogate, and in October, 1920, by Dr. Cawston of Durban, when the diagnosis was confirmed by Dr. Murray.

Dr. Cawston, in October, 1920, gave him injections (intravenous) of antimony tartrate. Before treatment the blood was examined and gave a positive reaction with the complement deviation test (*Schistosoma haematobium*). The symptoms noticed before injection appear to have been constant colicky pains in the abdomen, the passage of blood and mucus with the stools, and attacks of diarrhoea; in fact, the symptoms of the chronic dysentery of schistosomiasis. He came under my notice on April 14th, 1921, six months after injections, when there was nothing to note on physical examination. All the organs were natural; there was no enlargement of the liver or abdomen. During the time he was under my observation, from about April 14th to June, 1921, the faeces were examined half a dozen times. They were formed, looked natural, and contained no blood or mucus; only on one occasion was an ovum found, and it was granular and dead. The bowels acted regularly once a day after breakfast, and there were no symptoms of rectal bilharzia disease or any other disability, excepting that he complained of constipation and flatulence and defective digestion of starch. Whether these symptoms resulted

from or were aggravated by the injections I am not prepared to say. There is evidence that they existed in some degree before he had the injections, and it is not impossible that his digestive troubles are due to the non-functioning of the liver, damaged by an abscess for which he had been operated on by Dr. Thomson some years ago, or alternatively by the invasion of the liver by the *Schistosoma japonicum*, whose habitat is that organ, which invasion may have permanently affected its function.

According to a written record which the patient showed me whilst he was under my care, Dr. F. G. Cawston at Durban on October 6th, 1920, commenced injections of potassium antimony tartrate, beginning with a dose of $\frac{1}{4}$ grain. Injections were given daily at first, later on every other day approximately. The maximum dose was $1\frac{1}{2}$ grains, and the whole course extended over thirty-one days; the total amount injected was 21 grains. The ova are stated to have become granular in the same way as in the case of Egyptian bilharzia disease, and disappeared in the same way as *Schistosoma mansoni* ova disappear from the faeces during a course of antimony.

The same complement deviation reaction which had been carried out previous to injections and found positive, was found to be negative after the injections.

I publish this account because I have, with Mr. N. H. Johnson, watched the case for about three months, and we have examined the faeces six times.

There appears to be no doubt of its having been a case of Japanese schistosomiasis (Japanese bilharzia disease), contracted in the Hunan Province of China, which has been cured by the intravenous injection of antimony tartrate.

It will be interesting to learn what effect antimony compounds have on other trematode infections of the Far East—*Fasciolopsis buski*, *Clonorchis sinensis*, *Paragonimus westermani*, and perchance of *Heterophyes heterophyes* of the Far East and of Egypt and other parts of Africa, and, lastly, on *Fasciola hepatica* (sheep rot), whether occurring erratically in man or as a parasite of herbivorous animals.

EIGHTY-NINTH ANNUAL MEETING

OF THE

British Medical Association.

Held at Newcastle-on-Tyne, July, 1921.

SECTION OF DERMATOLOGY.

JOHN FARQUHAR CHRISTIE, M.A., M.B., President.

DISCUSSION ON THE ASSOCIATION OF SKIN TUBERCULOSIS WITH VISCERAL AND OTHER TUBERCULOUS MANIFESTATIONS.

OPENING PAPER

BY

GEORGE H. LANCASHIRE, M.D.,

Physician, Manchester and Salford Hospital for Diseases of the Skin. I MUST at the outset disclaim any special qualifications for the honour done me in asking me to introduce this subject, beyond an extensive clinical acquaintance for many years past with all forms of cutaneous tuberculosis, which is a painfully frequent feature of our hospital clinics in Manchester.

The question of the relationship of cutaneous to visceral and other forms of tuberculosis is very wide and highly speculative, but it is one of scientific and economic importance. Our discussion will be concentrated, I assume, on this practical point—How far is a subject of primary cutaneous tuberculosis liable to secondary visceral infection? We will first briefly review the other side of the picture—secondary infection of the skin from primary visceral disease, for here we are on surer ground, with a well-established sequence of events familiar to all clinical workers. The simplest form of skin tuberculosis in this class is seen in invasion of the cuticle by underlying disease of glands, bones, or joints, with the production of scrofuloderma or, at times, true lupus vulgaris. Here, of course, there is actual extension of disease by contiguity from one organ to another, in a manner analogous to malignant invasion. In the other forms of secondary skin