to Kyaukpin and allowed them to bite the leg of the case which occurred on the 16th and was still alive. This case had a well marked bubo and a temperature of 102°.

The bugs were allowed to bite for ten minutes and were not removed until it was seen that they actually contained blood. They were then brought back to Meiktila. On August 22nd they were applied to the shaved abdomen of a rat but all refused to bite, owing, I fancy, to the soap used in cleaning the rat. On August 23rd they were again applied to the rat's abdomen and bit freely. The rat was carefully fed and watered, but on the night of the 26th-27th it died. Smears made from this rat's spleen showed plague bacilli in very large numbers, and of most typical appearance, showing well-marked bi-polar staining. It may be noted here that an interval of 48 hours elapsed between the time when the bed-bugs bit the plague patient and the time when they bit the rat, and that after the rat was bitten only some 60 hours elapsed before it died of plague.

Following out Verjbitski's methods, I collected bed-bugs from the clothing and bedding of infected cases and crushed them into agar tubes in order to get a culture of B. Pestis. This was tried on three occasions, but each time the growth of non-pyogenic organism, moulds, etc., was so vigorous that no growth of B. Pestis was detected. There was no proper laboratory available, and it was difficult to carry out this part of the investigation with any hope of success. As regards the epidemic, single cases kept on occurring up to September 16th. The Civil Surgeon, Meiktila, and myself decided to allow the people to return to the village on 18th September. No further cases occurred after this which is curious, as between 10th and 16th September, seven cases occurred. With the return to the village the epidemic ceased absolutely, and there have been no cases since December 3rd. Verjbitski used very large numbers of bed-bugs in his series of 18 experiments, and amply proved that the bed-bug can transmit plague from guinea-pig to guinea-pig. He also explains how infected bugs might convey the disease to man either by "directly introducing bacilli adherent to its proboscis, or owing to the irritation it produces it may be crushed and the infective contents rubbed into the slight puncture occasioned by its bite." This point is proved in his experiment No. XLII in which the hind legs of six guinea-pigs were slightly scratched and the contents of crushed infected bugs spread over the place. All six guinea-pigs died of plague.*

Verjbitski's experiments were conducted in 1902-1903 and first published in English in 1908.† His work proves absolutely the transmission of plague in guinea-pigs by bed-bugs, but no one so far seems to have considered these results as having a more than theoretical value, or to have reported transmission of plague during all epidemic by such means. In view of the conclusion of the Plague Commission that plague is transmitted by the rat-flea and by it alone, and the great amount of evidence that has been recorded in proof of this statement, one feels a certain amount of diffidence in offering a contrary opinion. Still, transmission of plague by bed-bugs has been amply proved under elaborate conditions. it is *possible* in natural surroundings, and if it can be proved to occur in nature it is a factor of some importance in plague prevention. I trust that I have made my position on this point quite clear, I have seen the chain of events involved in rat-flea transmission only too often; it is only the unique and lonely position accorded to the rat-flea to which I object, and I have recorded my experiences above to show that it is possible for another parasite to transmit plague, and to transmit it directly, without infected rats being present.

I am fully aware that these observations are very incomplete, but I hope that other observers who have material available may be able to investigate this question further and settle whether the rat-flea is the only transmitter of bubonic plague, or whether there are not other possible channels of transmission.

I am indebted to Captain J. Good, I.M.S., for valuable assistance and advice.

* *Journal of Tropical Medicine and Hygiene*, May 1908.
† *Ibid.*

A Mirror of Hospital Practice.

A CASE OF HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY.

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THE following case of a disease which is very rare was admitted into the Mayo Hospital recently. The man had a very small cranium, but the facial development was not abnormal, though his face looked large from in front, owing to the poor development of the frontal part of his skull.

His hands and arms below the elbows, and his feet, ankles, legs and knee-joints, were all markedly hypertrophied. He was suffering from longstanding cough and had a cavity of fairly large size in his right apex. A complete and detailed description of the case is appended,

for which I have to thank my Clinical Assistant, Dr. Guranditta Kapur.

M. B., a male, aged about 30, Mohamedan by caste, resident of a village in the Lahore District, was admitted into the medical wards of the Mayo Hospital, Lahore, on 12th November 1909. He was a *faqir* by birth and profession. He was suffering from cough, and complained of pain in the joints, particularly on movement. The duration of the cough was eight months and pain in the joints began a month later.

While a mere child, the patient was left an orphan and did not know when, and of what, his parents died; he had no sister nor brother, so that it is difficult to say whether he inherited any predisposition to this disease or not.

He was unmarried and addicted to tobacco and *charas* smoking to excess. There was no evidence of syphilis, though he confessed having had an attack in his early life.

There was very little known about the course of the disease during the eight months of its duration, except that he had an attack of fever and cough associated with pain in the side of the chest, followed, a few days later, by hæmoptysis which was fairly profuse. The acute attack subsided gradually, but the cough still persisted and assumed a chronic form. Ever since the attack the patient had been gradually losing flesh and growing weaker day by day. A month after the attack of fever he felt pain in both the elbow joints almost simultaneously, and the joints became swollen, tender and somewhat stiff. The condition extended down and involved the wrists and then the joints of the fingers. Similarly, in the lower extremities, starting in the knees, the inflammation appeared in the ankles and then in the small joints of the toes. The shoulder and the hip joints were very little affected, the hips, however, more than the shoulders.

Physical examination on admission.—Weight, 115 lbs.; height, 5′ 6½″. Could not stand or walk owing to pain in the joints of the lower limbs.

He was distinctly emaciated, skin dusky, lips somewhat cyanosed; conjuntivæ, rather pale; tongue firm, dry, coated with thick dirty greyish white fur, clean at the tip and edges; marked clubbing of the fingers and to a less extent of the toes. Temperature, 100° F. Pulse, quick, 104 per min., rhythmic (both in force and frequency), of moderate volume and tension, and the vessel wall somewhat thickened.

His chest was broad and flat, ribs prominent, marked hollowing above and below the clavicles, more on the right than on the left; expansion of the lungs defective, especially on the right side. Breathing was quick, about 28 per min., and abdomino-thoracic in type. Slight diminution of vocal-fremitus at the right apex. *Percussion*—dulness in the supra- and infra-clavicular and upper parts of mammary and axillary areas on the right side. *Auscultation—Breath sounds* distinctly bronchial in character over the area of dulness and amphoric in the axilla. *Accompaniments*—only a few coarse crepitations at the right apex. *Vocal resonance* increased with bronchophony and whispering pectoriloquy in the first interspace, and the upper part of axilla on the right side. The left lung had nothing particular except that the breath sounds were harsh vesicular in character.

Heart was a little dilated and the heart-sounds rather weak. There was a little accentuation of the pulmonary second sound.

Abdomen was full and resistant; *spleen* palpable, only on deep inspiration, and the *liver* was only a finger's breadth below the costal margin in the right nipple line.

Urine contained nothing abnormal.

The smallness of the patient's head associated with enormous enlargement of his hands and feet caused a suspicion to arise of the case being one of acromegaly, but absence of any hypertrophy of the lower jaw and presence of pain in the swollen joints of the extremities were sufficient to set this diagnosis aside.

On taking measurements of the different parts of the body it was noticed that in the head the diminution was particularly in the lateral diameter, and that in the extremities the enlargement was fairly symmetrical on both sides. The forearms and the legs appeared much longer, and the upper arms and the thighs looked comparatively shorter than a person of his height and age would ordinarily possess.

The ends of long bones were enlarged and the joints swollen, stiff, and painful (except the shoulder and the hip joints). There was some effusion in the knee-joints; very little in the others.

The hands and feet were much hypertrophied, and the small joints thickened, especially the metacarpo-phalangeal and inter-phalangeal joints.

The patient stopped in the hospital for about three weeks. The fever he had, on admission came down to normal the next day, and it remained normal all along, except on the 10th day, when it again went up to 100° F.; but was again normal the following day.

Cough was rather a troublesome symptom to deal with; it was worse at night and early in the morning. Expectoration was fairly easy. *Sputum* large in quantity, muco-purulent and occasionally blood-stained, and under the microscope it revealed a number of pus-cells, leucocytes and epithelial cells. There were also some staphylococci and tubercle-bacilli (mostly single or in groups of two or three). On the 20th day after admission the patient had a slight attack of hæmoptysis.

Remedies to relieve cough were administered, but his condition remained more or less stationary; on the whole, however, he improved, and gained one pound in weight before he left the hospital. The further progress of the case

could not be watched, as the patient advanced all sorts of excuses, on plea of domestic affairs, to leave the hospital, so he was discharged on 2nd December 1909.

The point worth noting in this case is that Marie's sign-group, which is diagnostic of hypertrophic pulmonary osteo-arthropathy was well-marked, namely :—

1. A chronic and primary lesion in the lung.
2. Hypertrophic enlargement of the extremities with swelling, pain and limitation of movement of the various joints of the limbs.
3. Clubbing of the fingers and toes.

EPILEPSY NOCTURNAL,

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THE following case will be very interesting for publication as very few, I believe, have had the opportunity of observing a fit in a patient during sleep at night. This is not my only plea for publication, but the main object is the enlightenment of my own knowledge as regards its prognosis and treatment, if anybody amongst our profession, on reading this, condescends to drop a few lines (either in your journal or privately to me) in order to assist me by his practical experience in the treatment of such a case.

The patient is a lad of 15 and of strong and robust health, with no other complaints nor defects whatsoever, except that he is a little dull in intelligence and has not a very retentive memory. By this I mean, his intelligence and memory are a little below the average. He has been subject to this malady probably from his infancy but, however, it was detected only five years ago, and from that time onward he has been under observation and was found now and then to be attacked with convulsive fits always during sleep at night. For the last three years he has been living with me and sleeps in the next room to mine with a door of communication open all night, so that I may be aroused by the noise of the "cry," announcing the fit, which is very loud and high-pitched. The character of the fit is exactly like the description given in the text-book of a typical epileptic fit, *i.e.*, an epileptic 'cry' followed by tonic and clonic convulsion; the first portion (very short) if the 'cry' is expiratory and the rest inspiratory in character. The whole thing only lasts two to three minutes at most, and as soon as the respirations become regular, after several groans and sighs of extreme agony, he falls again into a deep sleep for several hours. When he is awake he says, if asked, he does not remember anything the matter with him at night but only that he feels very sore in all his body and limbs and has a headache. One thing to be noted in connection with the fit is that he is bathed in profuse perspiration when the respirations become regular: He never passes urine or fæces involuntarily. During my observation in the last three years this fit has been found to come on during sleep and at night, once at the interval of about a month. On four or five occasions it was noticed during the day when the boy fell asleep. In the previous and family history there are two things to take notice of. One is that being an inhabitant of a highly malarious village of Burdwan, he suffered much from malaria and his health remained shattered by the repeated attacks, up till he was twelve years of age. But for the last three years with me here he has been keeping excellent health. The other is that his mother was subject to convulsive fits all her life (whether epileptic or hysterical not exactly known) and died of broncho-pneumonia, consequent on food materials going down the trachea during one of her fits immediately after a heavy meal. He has no disease of the ear or nose, no phimosis, no intestinal parasites, no troubles of digestion and nothing of the kind that may be regarded as one of the reflex causes.

In the way of treatment in the last three years I never allow him to go to bed until at least four hours after his last meal, and give occasional courses of bromides (10—15 grains) once in the evening, combined with liq. arsenicalis and tinct. belladonnæ. In the first two years each course never exceeded three weeks at a stretch, and I gave sometimes pure pot. bromide and at other times, combined pot. sodi, and ammon. bromides, in equal parts in the mixture. I used to begin a course a few days before the fit was anticipated, as the fits used to come on, at the first part of my observation, at almost regular interval of a month. There was no fit during the period of his taking the draught. But as soon as the draught was withdrawn the fit would come on as usual. Being afraid to continue the bromides for a long time, I stopped the evening draught altogether for five months from the beginning of the third year. But now the fits began to come at a much shorter intervals—first of 15—20 days (for two or three months), then once (even twice) a week. I had, of necessity, to revert to the evening draught of tri-bromides with tinct. belladonnæ and liq. arsenicalis and continued this time for five months without a break. Again it had the desired effect and there were no fits for this long period. But again I stopped the draught and a fit came on just three weeks after its stoppage. There are no skin eruptions nor did the patient feel anything the worse for stopping the draught after a course of five months. Now the question is what course to follow in the way of treatment? How long can the bromides be continued without doing permanent injury to the nervous system? Is there any probability of these nocturnal fits becoming diurnal and when the patient is awake, if it is allowed to