

the abdomen and finally from the neck and chest. The 'rasping' sound continued for a few more days. He was discharged on the 28th day after admission;

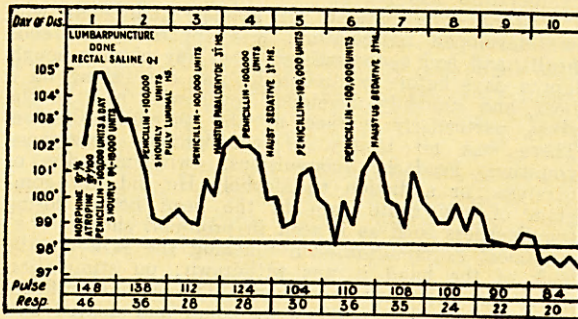


Fig. 3.

there was no abnormal sound in the heart or lung, and the apical impulse was in the 5th intercostal space half an inch internal to the mid-clavicular line. The case was followed up for two months after discharge, and the heart and lungs were found to be normal.

#### Discussion

The skin all over the body from the temple to the upper half of the abdomen was elevated and the crepitus was so evident that the case was at once diagnosed as interstitial emphysema. The cause for such a condition, in the absence of any history of injury, operative procedure or previous tuberculosis, and with the high temperature and unconsciousness, was not so easy to detect. The blood examination showed no malaria parasites, and it is impossible to explain surgical emphysema by a malignant tertian infection. The sudden onset of fever and hyperæmia of the mucosa of the nose and mouth and the raised pulse rate might suggest influenza, but the course of fever was long and there was no leucopenia. The history of the case, the physical examination and the laboratory tests do not suggest any cerebrovascular lesion or uræmia or diabetes as the cause of coma.

The high temperature with rapid pulse and respiration was suggestive of streptococcal infection from the throat, the toxins affecting the brain and causing unconsciousness till the infection was brought under control by penicillin treatment. The streptococcal infection causing congestion of the lung was possibly responsible for the rupture of the pulmonary alveoli; the skiagram of the chest (see figure 1, plate IV) shows an air track arising from the root of the right lung, escaping upwards along the bronchi and the blood vessels into the mediastinum, and passing up to the subcutaneous tissues of the neck.

Hamman (1937) reported that interstitial emphysema may occur spontaneously without injury, for example, during mild walking, sitting on a chair, or resting in bed, and in this condition there may be pain in the chest. When the leakage is greater, the air may pass into the pleural cavity and cause spontaneous pneumothorax, or it may track into the mediastinum and the symptoms may be severe and may

simulate coronary thrombosis or pericarditis. Hamman described the physical signs as a peculiar cracking bubbling sound heard over the heart with each contraction, not lasting long. The case under report showed the same physical signs as described by Hamman but the signs persisted for more than two weeks.

#### Acknowledgment

I express my grateful thanks to Lieut.-Colonel R. Linton, I.M.S., Principal, Medical College, Calcutta, for allowing me to make use of the hospital records and to report this case.

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### AN UNUSUAL DEVELOPMENTAL ABNORMALITY OF THE ARMS

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and

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THE abnormality was discovered accidentally when the patient came to a dispensary in Ahmedabad for treatment of a small ulcer on his left leg.

*Clinical findings.*—The patient is a male, aged about 20. His general appearance is healthy for his age (figure 1, plate IV), and the mental development is in conformity with his social status and education. He comes from Nagpur.

In the right arm the lower end of the humerus cannot be distinctly made out though the two epicondyles can be felt: the medial condyle is unusually prominent. There is no elbow joint. A single bone, probably representing both the radius and ulna and only 2½ inches long, is attached to the lower end of the humerus by a synostosis. The wrist joint also appears to be missing. In the palm which is small and oblong, two metacarpals can be felt. The thumb is absent and only two fingers are seen. It is not possible to say which fingers these are. The total length of the arm is 19 inches, that of the humerus being 11½ inches.

In the left arm the lower end of the humerus projects as a point. At about 8 inches from the acromion, a mass is seen attached laterally to the humerus. This mass represents the wrist joint, the palm and two digits, one of which appears to be the thumb. The forearm and elbow joint appear to be completely absent. The humerus is shorter than its fellow, being 10½ inches long.

In spite of the handicap, the patient can feed himself, clothe himself and wash himself. He can pick up a coin from the floor and lift a bucketful of water.

No other abnormality is seen in any other part of the body except that the forehead is rather sloping, and the frontal and parietal eminences are not felt.

*Radiological findings (figure 2, plate IV).*—Right superior extremity, lateral and antero-posterior views show two projections simulating two epicondyles, and a long projection about 2¼ inches long appearing like the lower end of the radius. The medial epicondyle is unusually prominent. The carpal bones consist of four bones which appear to represent the lunate, triquetral, the pisiform and a fused mass representing the rest. Two complete fingers with their metacarpals are seen and are normal.

The elbow joint is absent; the wrist joint is present; and also the carpo-metacarpal, metacarpo-phalangeal and the interphalangeal joints.

The left superior extremity shows a bone which represents an ulna joined to the humerus, both being undefined. The carpal bones are represented by two bones, the proximal and distal rows. Two metacarpal bones are seen and the outer one has only one phalanx whereas the inner one has two phalanges attached.

*Comments.*—As the condition is bilateral, we are inclined to believe that it is hereditary, though there is no history of any such case among the patient's brothers and sisters nor in either of his parents. His father is dead and his mother is far away and not available for interview. In any case, family histories in India are always difficult to elicit and notoriously unreliable.

Perkins (1936) states as follows:

'The foetal limb bud consists of: (1) a root segment near the trunk, (2) an intermediate segment which separates into two parallel segments, and (3) a terminal segment which separates into five parallel segments. Abnormalities may occur in the growth of the limb buds, resulting in suppression, hypoplasia or hypertrophy; and abnormalities may occur in the segmentation of the limb buds, resulting in either the production of supernumerary segments, or a fusion of segments. Fusion between the root segment and the intermediate segment is rare; more commonly one of the intermediate segments is fused with its fellow, e.g. radio-ulnar synostosis; most common of all, two of the terminal segments are fused together.'

Our case represents both a fusion, that of the humerus with either the radius or the ulna (if our surmise is correct), and a suppression of some carpal bones, many metacarpals and phalanges.

In the view of one of us (K. A. S.), the deformity of the hands is a variety of split-hand or lobster-claw, which is usually due to a dominant gene. It was this fact which induced him to study and investigate the case but the attempt to find sufficient data has not been successful.

REFERENCE

PERKINS, G. (1936) .. *British Encyclopædia of Medical Practice*, 2, 553. Butterworth and Co., Ltd., London.

ANALYSIS OF 242 CASES OF SPRUE IN INDIAN TROOPS\*

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A CONDITION manifesting itself in various degrees of emaciation accompanied by diarrhoea, indigestion, glossitis and anæmia, has of late been described under the labels of parasprue (Chaudhuri and Rai Chaudhuri, 1944), nutritional diarrhoea (Karamchandani, 1944), incomplete sprue (Manson-Bahr, 1940), chronic jejuno-ileal insufficiency (Bennett and Hardwick, 1940), and vitamin B deficiency syndrome allied to sprue (Cook, 1944). Rogers (Rogers and Megaw, 1942) is of the view that unexplained diarrhoea of more than 10 days' duration should be treated as sprue, provided amœbic disease is excluded. We are in agreement with the last view; it is not necessary that the whole picture with glossitis, flatulent dyspepsia, fatty diarrhoea containing high percentage of split fat, emaciation and macrocytic anæmia must be complete before the label of sprue should be applied.

*Pathology.*—In this syndrome, with faulty intake and faulty absorption, the stress initially falls on the delicate columnar epithelium of the intestines and the symptoms depend upon the site and extent of stress. Thus (A) when the duodeno-jejunal region is affected there may be deficiency of bile flow which means fat not emulsified, i.e. fat even when split is not absorbed, hence increase in split fat; or, deficiency of pancreatic secretion, and consequently deficiency of splitting of fats and high total fat content in the stools; or, when both bile flow and pancreatic secretion become deficient, typical white sprue stools occur with great increase in fat. (B) When the ileum is affected, the fat content in stools is normal and the diarrhoea is predominantly of an irritative kind (enteritis). (C) When the colonic area is affected, mucus and even blood are found in the stools (colitis).

The next stage in the stress comes when, in addition to the affection of the delicate columnar epithelium, increased vascular permeability occurs. In the stomach, irritative gastritis is produced, and this is responsible for rapidly developing anæmias and in the intestines, diarrhoea is accentuated.

In the third stage infection is superimposed. These infections are salmonellas in the small intestines and not of importance; but in the large intestines, amœbic infection shows concomitant chronic intoxications and bacillary infection shows ulcerative colitis, with rapid intoxication. At this stage, fatty diarrhoea with progressive and rapid asthenia and anæmia is found.

Finally, complication may extend to the nervous mechanism. These changes when confined to the more superficial Meissner's plexuses manifest themselves in accentuation of the secretory variety of diarrhoea, the villi pouring out watery diarrhoea (easily controlled by chlorodyne); when extending deeper into the Auerbach's plexuses, the motor accentuation with hypermotility of lenteric variety occurs which accentuates mal-absorption and increase of split fat in stools.

The post-mortem findings in two typical cases are given.

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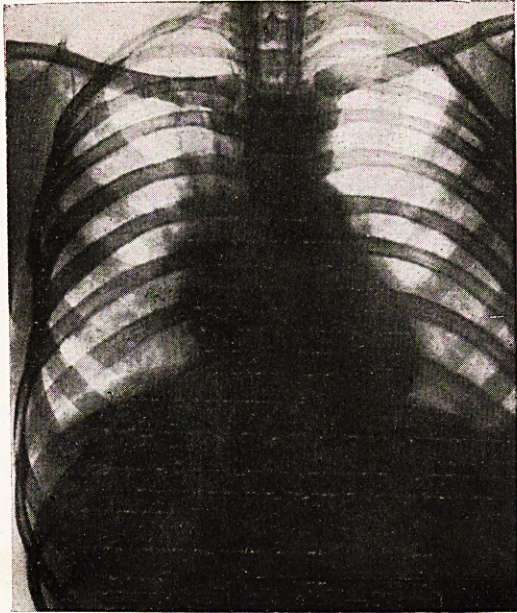


Fig. 1.—Before treatment.

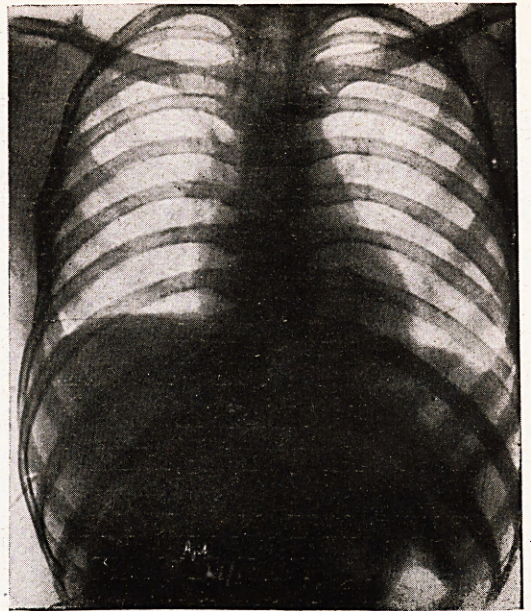


Fig. 2.—After treatment.

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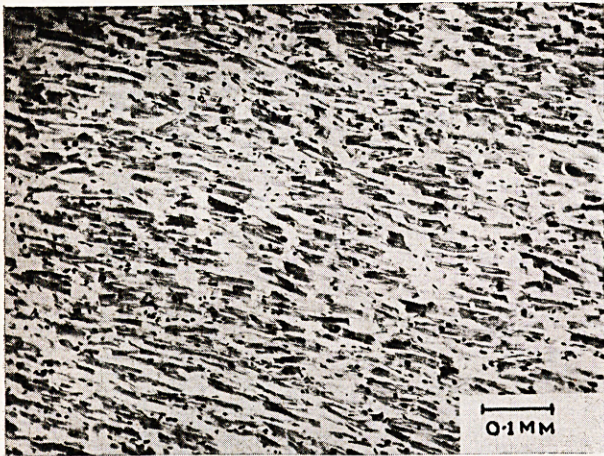


Fig. 1.—Microphotograph of heart muscle ( $\times 100$ ) showing extreme fragmentation and degeneration of the heart muscle fibres; and œdema of the fibrovascular connective tissue.

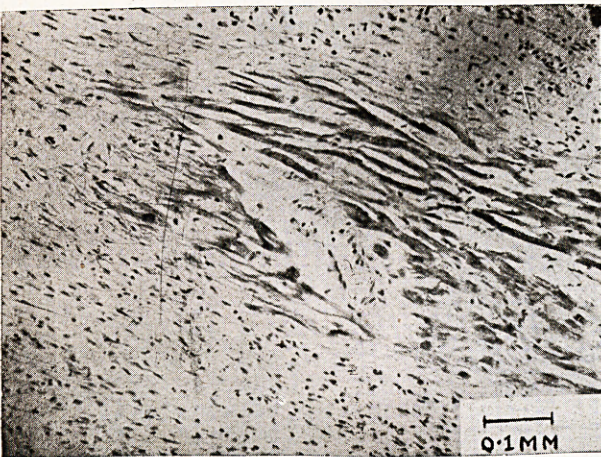


Fig. 2.—Microphotograph of another area ( $\times 100$ ) showing replacement of the heart muscle by œdematous fibrous connective tissue and degeneration of muscle fibres suggesting a chronic myocardial lesion.

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Fig. 1.—Photograph of the patient.

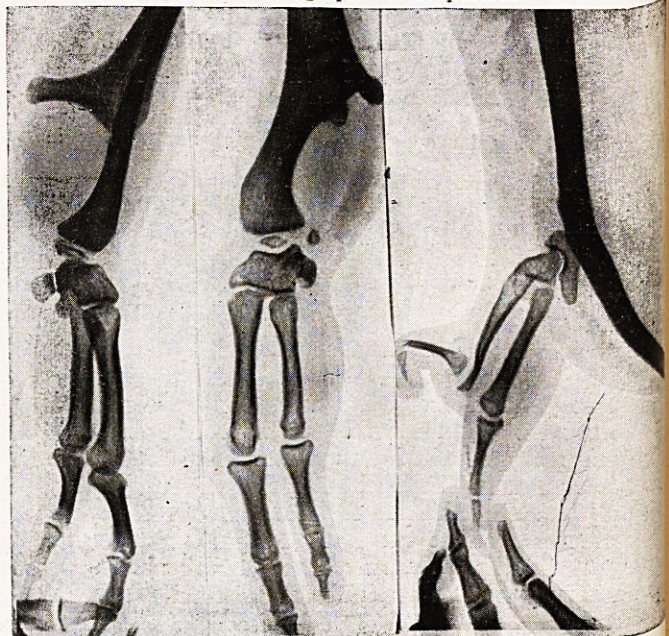


Fig. 2.—Radiograms of upper extremities.