

recumbent position, if the diaphragmatic movement also is restricted either through post-anæsthetic shallow breathing or through tight abdominal bandaging, the respiratory excursions of the lower lobe will be rendered relatively smaller than that of the upper lobe. Hence stasis of secretion occurs more easily in the lower lobe bronchioles. Moreover the lie, direction and inclination of the lower lobe bronchus and its branches are more favourable to the production of a ball-valve action of the secretion inside them. Lower lobe atelectasis is therefore less likely to happen if the patient after operation is propped up or turned to one side. Perhaps by keeping the patient in the Trendelenburg position until normal breathing is established, pulmonary atelectasis might be prevented, as it will prevent accumulation of secretion in the lower reaches of the bronchioles.

Summary

(1) Three cases of lower lobe atelectasis are described.

(2) A short review is given of the available literature on the pathogenesis of atelectasis.

(3) The opinion is expressed that the establishment of 'one-way traffic' for the air in the bronchioles by the ball-valve action of secretion inside is the cause of pulmonary atelectasis. Respiratory movements are none the less necessary.

(4) Experiments are described to substantiate the theory.

(5) Reasons for relative frequency of lower lobe atelectasis are cited.

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MEDIASTINAL EMPHYSEMA

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THE total number of cases of spontaneous mediastinal emphysema reported so far is 23; of these, 22 cases were reported by Hamman (1934), but none of these 22 cases was admitted to hospital unconscious and with high temperature. The following case report will therefore be of interest:—

A male, aged 45 years, was admitted to hospital unconscious, restless and dyspnoic on the 4th May,

1945. There was a generalized erythematous blush of the skin over the face, neck and chest. The skin on palpation gave a distinct feeling of crepitus. The temperature was 102°F.

History.—The patient, an average individual with well-developed musculature, was apparently in good health and had never suffered from any chronic cough. Three days before admission he was out fishing on a boat, and suddenly developed fever with pain in the chest, particularly marked in the precordial region. There was no history of constipation, drowsiness, vomiting, headache, convulsions, twitching, fits or paralysis, or addiction to alcohol. He did not complain of any acute pain in the head before losing consciousness such as is seen in profound shock.

Clinical examination.—On pinching the skin on the back of the hand it was withdrawn; on stimulation there was voluntary movement. The corneal reflexes were present. The pupils were equal and dilated and reacted to light. Examination of the fundus oculi showed slight blurring of the disc and congestion of the retinal vessels. The ocular tension was normal. There was no rigidity of the neck; the neck veins were found congested.

The temperature continued from the onset of fever and shot up to 104.8°F. within half an hour of admission. The pulse rate was about 130 to 140 per minute; the pulse was full, rapid and easily compressible. The respiration rate was about 40 per minute. The blood pressure was 125/65.

The apex beat could not be palpated properly owing to surgical emphysema. On percussion, the normal area of cardiac dullness was found obliterated and the note was particularly hyper-resonant. There was a rough, to and fro, grating sound heard anteriorly from the left margin in the 4th and 5th intercostal spaces up to half an inch internal to the mid-clavicular line.

The breathing was hurried but not stertorous or irregular; the breath sounds could not be heard distinctly and were diminished. The percussion note was resonant. Crepitus could be felt from the temple down to the upper half of the abdomen. The nasal sinuses were congested. The upper abdomen was prominent and on palpation gave a distinctive crepitus. The spleen and liver could not be palpated.

The lips were cyanosed, the tongue was red, and the throat congested; a slough was seen over the tonsil; the slough was removed easily and sent for bacteriological examination. Both tonsils were inflamed.

There was no hemiplegia or hemiparesis. Tendon reflexes were sluggish.

Laboratory examinations.—Blood examination showed moderate anæmia (hæmoglobin 60 per cent) and some polymorphonuclear leucocytosis. No malaria parasites were found. Blood culture was sterile. Urine was normal. Friedlander's pneumobacilli were isolated from culture of the throat swab. Lumbar puncture showed the cerebro-spinal fluid under some pressure but clear in general appearance.

Treatment.—Morphine and atropine injections were given immediately on admission, and penicillin 20,000 units immediately and 15,000 units every three hours for 6 days, the daily dose being 100,000 units. An alkaline mixture was given thrice daily and glucose injections twice daily.

For the first two days no food could be given orally. Fluid diet was given from the third day, and soft rice and fish from the 11th day.

Course and prognosis.—The patient's fever continued varying from 99°F. to 102°F. for more than a week (see temperature chart). His general condition was grave for two days, during which period he was unconscious. He became conscious on the third day of admission and talked sensibly; but the temperature and the condition of surgical emphysema persisted, as also the sound in the cardiac area. A few râles and crepitations were heard at the bases of the lungs.

The temperature came down to normal on the 14th day. Crepitus disappeared first from the face, then from

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;

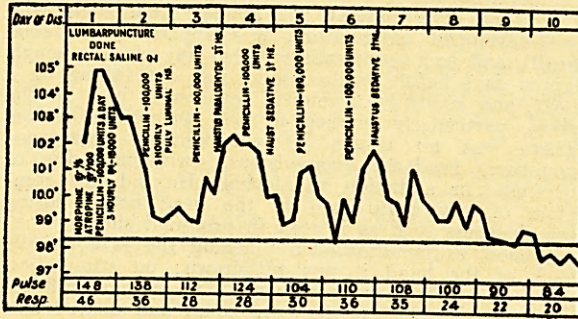


Fig. 3.

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 hyperaemia 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 uraemia 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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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 2¼ 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 11¾ 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.

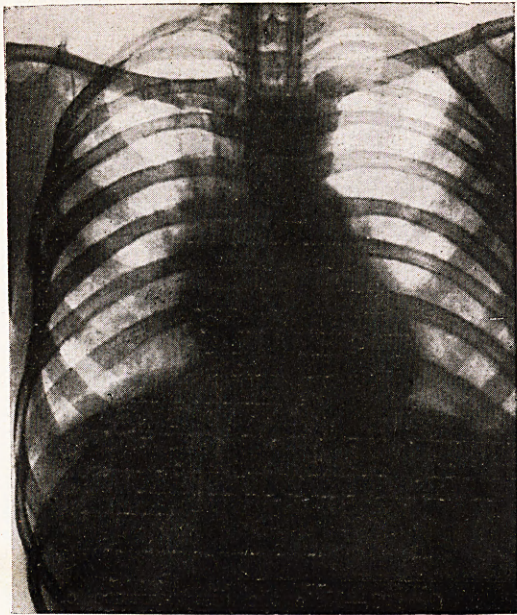


Fig. 1.—Before treatment.

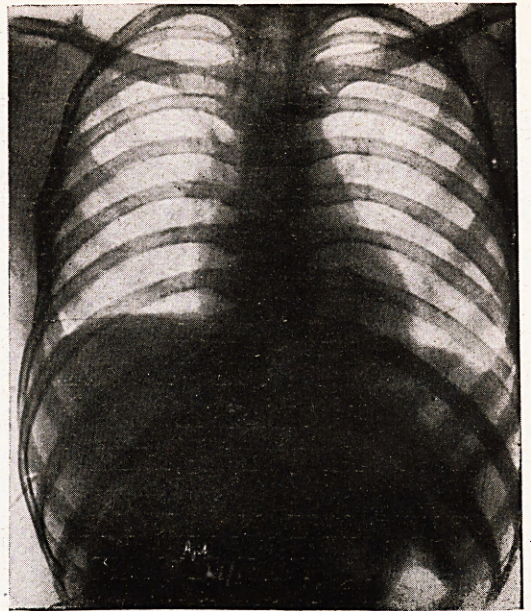


Fig. 2.—After treatment.

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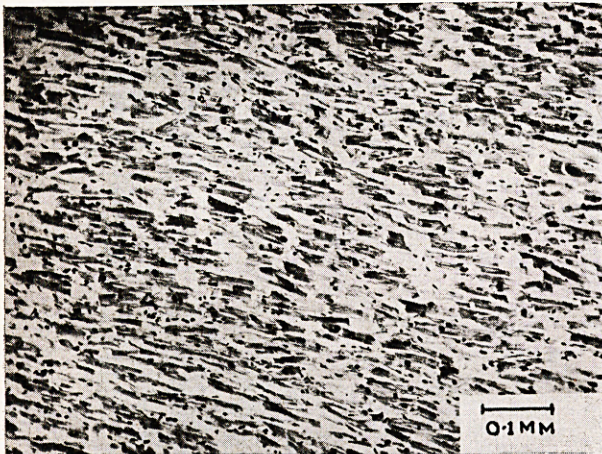


Fig. 1.—Microphotograph of heart muscle ($\times 100$) showing extreme fragmentation and degeneration of the heart muscle fibres; and œdema of the fibrovascular connective tissue.

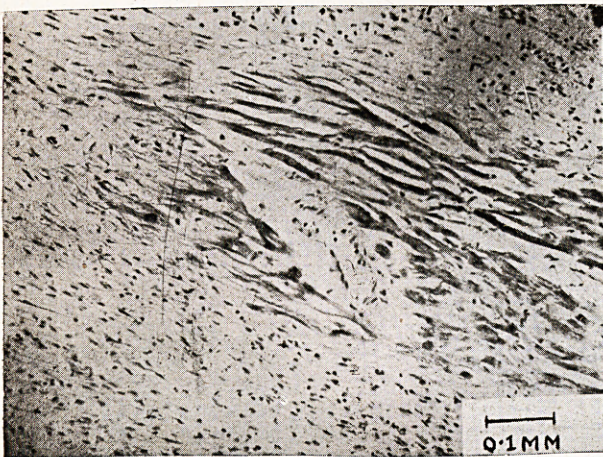


Fig. 2.—Microphotograph of another area ($\times 100$) showing replacement of the heart muscle by œdematous fibrous connective tissue and degeneration of muscle fibres suggesting a chronic myocardial lesion.

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Fig. 1.—Photograph of the patient.

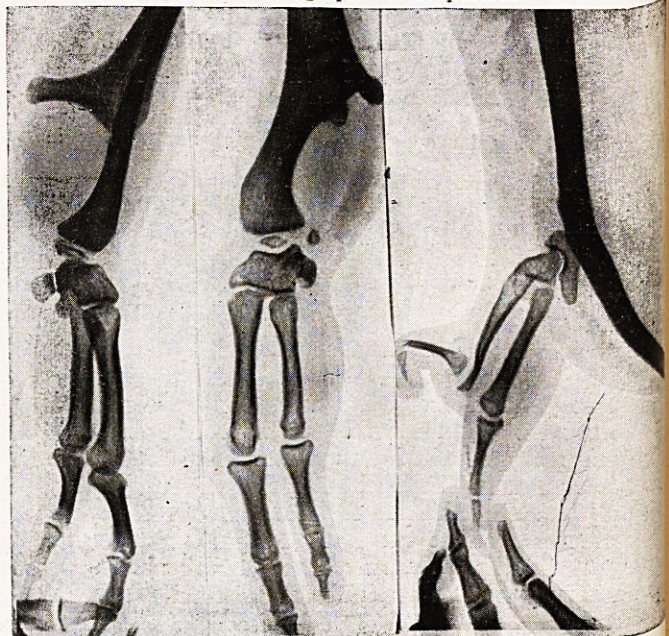


Fig. 2.—Radiograms of upper extremities.