HAEMORRHAGE INTO THE LUNGS IN CASES OF DEATH DUE TO TRAUMA

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When changing circumstances bring a lesion to the notice of a morbid anatomist as a manifestation of a new aetiological factor it is well to review the past experience of the lesion and to detail, so far as possible, the variations that have been observed. Under war conditions fatalities have occurred in which the outstanding post-mortem finding has been haemorrhage into the tissue of the lung. Reviewing the peacetime incidence of haemorrhage into the lung in association with death from trauma, it must be recalled that such a lesion may occur in various conditions.

Peacetime Injuries

Blood may be inhaled after haemorrhage from lesions of the upper air passages or nasopharynx, or even from a fracture of the base of the skull. The blood in these cases will be found in the bronchi, bronchioles, and filling the respiratory bronchioles and alveoli in a lobule, giving the appearance of haemorrhagic areas approximately wedge-shaped but with lobulated margins. These bloody foci are likely to be more numerous in the lower lobes. The lung tissue is undamaged, and in the absence of previous pulmonary disease the microscopical appearance is that of normal respiratory bronchioles and alveoli filled with fresh blood. In cases in which the inhalation is not immediately fatal coagulation of the blood will have taken place.

A lacerated wound of the lung associated with haemorrhage may occur after penetration of the chest wall by a sharp object—the spike of an iron railing, a part of a motor-car, or some weapon: a similar condition in the lung is produced by a broken rib if driven inwards. The appearances here vary according to whether the penetrating object or rib remains in the lung while respiration continues. The pleura being damaged, there will probably be a pneumothorax and probably also a haemothorax. The injury to the pleura and gross disruption of the tissue of the lung are usually apparent on naked-eye examination. The resultant haemorrhage into the lung varies with circumstances. It may spread for some little distance beyond the margins of the wound.

Haemorrhagic lesions (contusions or bruises) of the lung may be found in cases which show no penetrating wound of the chest and even those in which no ribs are broken and no injury to the chest wall is apparent. Such cases are comparatively frequent in road accidents; they may be found after the violent impact of a moving vehicle or when the victim is hurled against a hard obstacle such as a wall. In children it is most often associated with a history of the rapid passage of a wheel over the thorax. The presence of extensive internal injuries without external evidence of violence is much more common in children than in adults. The haemorrhagic lesion in the lung takes the form of a contusion or localized bruise of some part of the organ, the localization being determined by the direction of the force and the situation of the maximum point of impact on the chest wall. Quite often there is a separation of the lung tissue where the organ has been nipped against the vertebral column. Scattered multiple haemorrhages are infrequent. As a rule there is no associated emphy-

sema, but patients surviving for a few minutes may show all grades of interstitial emphysema when the lung is severely contused. Haemothorax is quite common. Microscopically there is rupture of the lung tissue and haemorrhage into the damaged area.

In another group of cases death may be said to be due to asphyxia. It has been recognized since Tardieu's time that asphyxia from any cause produces haemorrhagic lesions in the lungs. The lesions consist of punctiform subpleural haemorrhagic patches. They are dark in colour and vary in size from 1 to 5 mm. in diameter. Sometimes they are larger. These ecchymoses—Tardieu's spots—are sometimes separated, sometimes in groups. The outline is generally distinct and well defined on the surface. They are most common about the root of the lung at the lower margin and on the pleural surfaces between the lobes. They are usually associated with similar lesions on the other serous surfaces—the pericardium and the peritoneal surface of the diaphragm, etc.—and in the thymus in children. On suffocation the lungs will show congestion as well as Tardieu's spots. In asphyxia due to pressure on the trachea (strangulation) there will be frothy blood-stained mucous fluid in the upper air passages and intense congestion of the lungs, and, in addition to Tardieu's spots, larger patches of haemorrhages may be present in the substance of the lung. Where there has been continued compression of the thorax the haemorrhage into the tissue of the lung will be greatest where the ribs have pressed against the pleura.

Another type of case in which localized haemorrhagic lesions have been said to occur is that in which fat embolism is the cause of death. With reference to this it may be stated that it is not the experience of most morbid anatomists who have dealt with any numbers of deaths of a violent nature that fat embolism of the lung produces any localized haemorrhagic lesions which can be distinguished macroscopically. The lungs of patients dying of fat embolism following crushing injury to the bones, joints, or adipose tissue are typically deeply congested throughout, with no localized haemorrhagic patches.

Wartime Injuries

Having reviewed the haemorrhagic lesions of the lung due to trauma as they were seen in peacetime, it remains to compare the lesions produced as the result of the detonation of high explosive. It is possible to place some cases in the groups already summarized.

Flying missiles—bomb-casing and secondary mobilized fragments of masonry, metal, or wood—produce penetrating wounds of the chest and lacerated wounds of the lung differing in no way, except in degree, from those lesions described above. Impact damage from these missiles, or forcible hurling of the victim by the force of the explosion ("blast"), may produce contusions of the lung such as are all too familiar in road accidents. These contusions may presumably be found without visible external injury.

Cases of asphyxia are probably frequent under conditions due to bombing, where casualties may be buried under debris. These cases may be considered in two groups: in the one there has been inhalation of a large amount of dust; in the other the victim has been pinned under a pile of debris. In the former group the post-mortem findings are those of asphyxia only. Cyanosis is present; frothy mucus may be mixed with the solid inhaled material; and the lungs show congestion and some small scattered haemorrhages throughout and Tardieu's spots on the pleural surface. These ecchymoses are found in varying numbers under the parietal pleura and on the

peritoneal surface of the diaphragm. The other group is of considerable interest as it is possible to construct from examination of these cases a picture of the lung changes in compression asphyxia.

In cases of compression asphyxia where the victim has been buried under debris death has presumably been slow, with attempts at respiration. In such conditions rib markings have been observed; a dark purple line of the size and direction of the rib is seen, with emphysematous bullae visible at the margin of the dark rib marking. In addition these cases showed some scattered haemorrhages into the lung, with subpleural Tardieu's spots. There are localized areas of collapse. Microscopically there is disruption of the alveolar walls where the rib marking has been sectioned. There is fresh blood in the alveoli, and clotting may have begun in some areas. Elsewhere there is general congestion of all the capillaries in the alveolar walls and a considerable number of thromboses in smaller arterioles and veins. Oedema is present in many areas.

So far it has been possible to correlate these wartime injuries with peacetime parallels. Another group is without parallel. This includes cases in which death may be found to have occurred without or with only trifling external injury, but in which intrapulmonary haemorrhage has been a post-mortem feature common to them all. These cases have all been found near to the site of explosion of a bomb. In view of this fact the condition may well be called "haemorrhagic pulmonary concussion"; it may or may not be due to blast from the high explosive. It may prove to be of interest in the elucidation of the actual causes of this concussion that where these pulmonary lesions have been found and there have been external markings of small splinters, etc., the superficial lesions have all been on the anterior surface of the body.

Subpleural haemorrhages are not conspicuous and haemorrhagic rib markings are not present in the pleura. The haemorrhagic areas are scattered through, and often deep in, the substance of the lung, most often posteriorly and towards the base. The degree of haemorrhage varies considerably, but both lungs are affected to the same degree.

Comparing the histology of these cases with that found in compression asphyxia, it is seen that oedema is not a feature. Thromboses are not present. The haemorrhage in those patients found dead is fresh, and clotting has not occurred. There is widespread and intense capillary congestion in the lungs. There is haemorrhage into the walls of the small bronchioles, but absence of bleeding into the walls of the bronchi. There is some over-distension of air vesicles and respiratory bronchioles in some cases, but there is no anatomical relation of these to the ribs.

At the present time it is not known how many of the air-raid casualties dying with gross fatal injury, apparent externally, show pulmonary lesions such as these, but it is beyond question that these appearances are a definite morbid entity distinct from conditions hitherto familiar, and are distinguishable on careful analysis from lesions due to compression asphyxia or to pulmonary haemorrhage due to impact of solids against the thoracic wall.

Conclusion

The main points of difference may be summarized as follows:

In compression asphyxia the lesions may be symmetrical on both sides, but not necessarily so. The haemorrhages are usually subpleural and in the lines of the ribs. Emphysema may outline these rib markings. There is

generalized congestion of the lungs and some oedema. Tardieu's spots are nearly always found.

In haemorrhage due to impact of a solid the haemorrhage is in relation to the point of maximum impact and may be unilateral. The lung tissue is contused, torn, and filled with blood at or around this point.

In haemorrhagic concussion the lesions are always bilateral and roughly symmetrical. There is general congestion of the lungs. Pleural haemorrhages are present only as an extension from deeper areas.

FAMILIAL PERIODIC PARALYSIS

BY

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The following brief study of two cases of familial periodic paralysis is recorded because of the rarity of the disease. The patients were brothers recently enlisted in different units, and their disability was first noticed after exercise. The chemical investigations were limited by military conditions and are by no means complete.

Familial periodic paralysis is characterized by sudden attacks of flaccid paralysis in one or more muscle groups, generally in the limbs. The paralysis is usually symmetrical. The trunk muscles are seldom involved, and much more rarely the muscles supplied by the cranial nerves. There is a complete loss or diminution of the tendon reflexes in the affected muscles, and usually the electrical excitability to both faradic and galvanic currents is lost or diminished. The duration of the attacks varies from a few hours to three or four days. They vary greatly in frequency, occurring every few days or at intervals of several months. Sphincter control is rarely affected, and very infrequently death occurs from respiratory paralysis. Biernard and Daniels (1934) have described a muscular atrophy developing in later life in the muscles most affected by the paralysis, but this complication is rare. There is usually little difficulty in diagnosing the cases with a familial incidence, but occasional sporadic cases occur which may be regarded as hysteria.

Until recently the factors causing the disease, apart from the inherited predisposition, have remained obscure. The first real advance in the understanding of the condition was made by Aitken, Allott, Castleden, and Walker (1937). These observers found that during the attacks of paralysis there was an abnormally low serum-potassium level and that the paralysis could be cut short by the administration of potassium chloride by mouth. They also found that paralysis could be induced by giving large amounts of glucose by mouth, either alone or together with insulin injections. These observations were confirmed in two further cases by Allott and McArdle (1938).

The fall in the serum-potassium content had been observed by Biernard and Daniels (1934), but they did not realize its significance. Treatment with potassium salts was mentioned as early as 1902 by Mitchell, Flexner, and Edsell, also by Holtzapfel in 1904, and by Herrington in 1937. Holtzapfel used potassium bromide, and ascribed the benefit to the bromide rather than to the potassium. Herrington used potassium citrate in 5-gramme doses with success in two cases.