HÆMOTHORAX.

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Hæmothorax cases are occasionally met with in civil life but their frequency incidence is insufficient to warrant discussion. It is a war-time tragedy and in this article I will confine my remarks to cases caused by penetrating wounds in the chest—the so-called traumatic variety—and make no further reference to the non-traumatic type in which rupture of a diseased blood vessel is usually the cause.

Should perforation occur in the middle line of the thorax and involve the rupture of the great vessels at the root of the lungs or at the base of the neck, a fatal hæmorrhage is almost certain to follow. Such cases as these need not be discussed since no treatment is likely to be of any avail.

Broadly speaking it may be said that hæmothorax is the result of a gun shot wound of the chest of the penetrating type though very occasionally it may occur in consequence of a non-penetrating chest wound.

As the name implies, hæmothorax denotes the presence of blood in the pleural cavity due in the majority of instances to injury of the vessels of the lung itself rather than to that of rupture of the arteries or veins of the thoracic wall as was previously believed.

It would not be inappropriate here to discuss in a few words the *mode of production* of a hæmothorax. It may be due to a penetrating wound, though very occasionally a non-penetrating one has been known to cause a slight reactionary hæmorrhage into the pleural cavity. Equally there are times when a missile, notably a rifle bullet, may enter and leave the thorax having penetrated the lung without causing any hæmorrhage into the pleural cavity.

Some knowledge and appreciation of what is known as ballistics is necessary to appreciate the effects of war injuries as applied to the chest. It must be realised that the rifle bullet in its flight, in addition to a forward movement, has a rotatory one *around its long axis* from the spin imparted by the rifling. A further rotation takes place *around its short axis* should any resistance be met. The result is that the bullet is at one moment travelling broad side on after it has encountered the resistance, and this will greatly increase its destructive powers. This rotation takes place at short ranges when the velocity is very high, and disappears after about 600 yards when the bullet has settled into its flight, reappearing towards the end of the trajectory at long range.

Hence the severity of the wound may vary considerably with the distance of the firing point from the man injured.

One other possible factor to remember is the cushion of compressed air which surrounds the head of the bullet and which is visible in instantaneous photographs. This cushion may be responsible for some cases of penetration with no apparent lesion.

On more than one occasion during the last war I saw cases at post-mortem examinations without any apparent macroscopical lesion in the lung where the entry and exit wounds proved beyond any doubt that the missile had traversed the lung. Death in these cases was, of course, due to other injuries. It must be assumed, therefore, that penetration of the lung need not necessarily in all cases

produce damage sufficient to warrant anxiety or surgical interference, but it must not be lost sight of that the striking force of the modern missile, particularly the fragment of shell, is enormous and little wonder it is that the damage it may inflict is often very considerable.

The formula $\text{Energy} = \frac{\text{mass} \times \text{velocity}^2}{2}$ when applied to the striking force of the modern bullet or shell shrapnel will give us an idea of what amount of damage to the thorax could take place, realising that the initial velocity of a modern projectile is usually at least 2,900 ft. per second.

Hæmothorax is the commonest type of penetrating chest injury. Of a total series of 139 consecutive cases of penetrating chest wounds during the last war I recorded 80 in which this condition had occurred.

The *size of the hæmothorax* may vary within wide limits from so small an amount as to be perceptible only on the closest examination, up to a complete filling of the pleural cavity. As a rough guide, I suggest that a medium sized hæmothorax be one in which the fluid extends up to, but not further than, the angle of the scapula in the sitting position. The apparent severity of the wound is no criterion of the amount of the effusion. What, therefore, is the factor which determines the limitation of size? This explanation cannot be answered on purely physical grounds, for the highly complex physiological action of the lung and its still less understood innervation must be regarded as important contributory factors. The mechanical effect of pressure of the effusion in a case of hæmothorax will tend to check hæmorrhage but not until that effusion is large enough to collapse the lung and so occlude the bleeding points.

When the base of the lung is involved by injury, a small effusion may be quite sufficient to cause deflation of that area, but where the upper part of the lung has been wounded a very extensive effusion would be required to achieve the same result. To account for the limited effusion so often seen with the latter kind of wound active collapse either of the type of massive or partial deflation must be assumed.

Misleading ideas as to the amount of fluid present in a pleural cavity may arise unless the closest attention be paid to the interpretation of these signs. One would emphasize the importance of the *position of the apex beat and that of the diaphragm*. When possible the determination of these must lie with the radiologist.

In simple hæmothorax the dome of the diaphragm is at a higher level than the normal, contrary to what would be expected on physiological grounds, since experimental injury to the lung is followed by increased diaphragmatic tone and hence a lowering of the dome. It is difficult to say why gun shot wounds of the chest should show this difference from those produced in the laboratory but whatever the reason the elevation of the dome of the diaphragm can only mean that instead of an increase of pressure within the pleural sac which would be expected as a result of an effusion the pressure is actually diminished and, therefore, the lung collapsed. The collapse obviously is not the result of the pressure of the effusion in all cases, it must often be due to some independent process. Its effect is to stop hæmorrhage since the vessels collapse with the lung tissue. It must be nature's pulmonary hæmostatic.

A small hæmothorax, therefore, may often be and is often associated with a considerable degree of deflation of lung indicating the controlling action of the latter process.

All stages of pulmonary deflation may be met with up to the massive collapse originally described by Pasteur, the physical signs naturally varying with the degree of the process. The occurrence of this phenomenon is not frequently met with in cases of pleural effusion of the non-traumatic type, but it must be regarded as by no means infrequent in hæmothorax cases resulting from war injuries. In my series of cases during the last war, I noted some degree of deflation in 48 per cent. That this pulmonary collapse is not always the result of pressure within the pleural cavity from the effused blood and serum is proved by the fact that on many occasions considerable degrees of it may exist when the evidence of hæmothorax is slight.

The question has been discussed as to whether the condition may not be one of pneumonic consolidation, but the absence of the constitutional signs of a pneumonia and the rate of disappearance contra-indicate this. Moreover the displacement of the heart to the side of the lesion and the rising of the arch of the diaphragm are additional points against such a supposition.

Just as the amount of hæmothorax fluid present in the pleural cavity may be difficult to determine on account of the deflation present, so also may the signs of collapse be obscured where the amount of fluid effused is abundant. Here the employment of X-rays is of great importance, particularly with regard to the position of the diaphragm and the displacement of mediastinal contents.

Pressure of the intrapleural fluid can certainly not explain the occurrence in all cases of this collapse which is so commonly associated with hæmothoraces. It may be that in many instances the explanation of such an occurrence can be found in assuming that a large branch of the bronchial tree has been blocked in the course of aspiration of mucus or blood. The sudden disappearance of the signs of collapse occurring after a fit of coughing lends weight to this possibility.

It is interesting also to remember that during the last war accurate notes made at a casualty clearing station often indicated that a severe amount of deflation had taken place, no such evidence of this being found when the patient was admitted to a base hospital a few days later. The aspiration theory of the cause of collapse might equally well explain the frequent occurrence of what we came to regard as a not infrequent complication—*contralateral collapse*. It may be, however, that a nerve influence may play a part in the production of the collapse process. A. B. Soltau and I put forward a suggestion some years ago that, owing to the injury which the terminals of the vagus in the wounded lung sustained, a reflex action was set up and that reflex path was from the terminals, via the vagus to the respiratory centre and thence by the phrenic nerve to the diaphragm, causing a diaphragmatic paralysis.

The value of collapse in limiting hæmorrhage can often be appreciated. I have notes of one case in which the lung was so extremely deflated that resonance was impaired as far up as the spine of the scapula but only a very small amount of fluid was present. The degree of collapse would have permitted at least three times the amount of fluid to occupy the pleural cavity.

The important question of *clotting of the hæmothorax* must be briefly considered in that the proper treatment to be adopted in hæmothorax cases is closely associated with it. That clotting does take place must be admitted, both the degree and the nature of the clotting varying considerably. The usual experience on examining a chest post-mortem is to find a blood-stained fluid containing small particles of clot in the dependent areas; the remaining fluid usually does not clot, or only partially so, even after standing, a proof that the fibrin has been deposited.

I have not infrequently found when withdrawing blood from a hæmothorax for purposes of examination that after prolonged standing no clotting has taken place, suggesting that we are dealing with blood already defibrinated and therefore not capable of forming a massive coagulum. It has been suggested that the presence of sepsis may be one of the factors in preventing a complete coagulation, though the constant movement of the lung may act as another deterrent. My own observations have led me to believe that a partial clotting, or at any rate a deposition of fibrin, is constant but that a true massive clot is extremely uncommon. The process of clotting would appear to commence at a very early time after wounding and the absence of any such coagulum at a later date would favour the presence, or at least advent, of sepsis. This partial clotting or fibrin deposition does not, I believe, usually lead to extensive adhesions likely to bring about great limitation of lung expansion.

To recognise the advent of sepsis in hæmothorax cases too much attention cannot be paid to the consideration of factors concerned. Once the primary shock of the wound is overcome, sepsis must be regarded as the greatest danger which concerns us. A frequent examination of the chest is essential and is of far greater importance than a mere slavish watching of the temperature chart. Bacteriological examination of an aspirated sample of the effusion is often misleading as the infection may be localised and the sample drawn off be free from contamination. X-ray examinations when available are of the utmost importance. The leucocyte count also cannot be relied upon implicitly because of the disturbing factor of the inflamed wounds.

The aspect of the case must be regarded as a whole, namely facies, distress, pulse rate, temperature, alteration in position of apex beat, development of signs indicative of gas, and respiration rate.

Mention here may be made of the typical hæmothorax temperature chart which follows a steadily improving course. The initial fever gradually subsides, each diurnal oscillation being less until the mean is arrived at. A secondary curve is not infrequently seen with hæmothorax after the temperature has approached the normal. It is characterised by what I describe as the periscopic course, in that a steadily increasing morning and evening rise takes place; this, reaching its maximum, similarly decreases until a final normal mean is reached. The regularity of the increase is remarkable and is so characteristic that it has sufficed to differentiate this rise from that of sepsis.

The pulse rate and the respiration rate are of extreme importance when on the outlook for sepsis. A sudden jump in these rates is in my opinion a clearer indication of approaching danger, than a rise in temperature. I believe that a mild degree of sepsis exists in the great majority of cases of hæmothorax and that the rise of temperature in the early stages denotes this, but this mild infection is usually overcome but may reappear when organisms become active. This is particularly true in the case of gas forming bacilli. The following case may illustrate this point.

The patient was at first apparently following a normal course of hæmothorax. Eight days later without any appreciable rise in temperature the pulse rate jumped to 140 and the respiration to 40. Immediate resection showed the presence of gas formation.

The progress of the case may be modified by the presence of some intercurrent disease which may have been overlooked, thus a rise in temperature, pulse rate

and respiration rate may occasionally tend to suggest the development of sepsis and the urgent need for surgical intervention, in spite of the absence of altered physical signs or bacteriological infection. Tuberculosis, presumably a latent infection roused to activity by the trauma, may be met with. This possibility should be borne in mind and a careful examination of the sputum be undertaken.

I have previously recorded two cases of hæmothorax in which progress was unsatisfactory but the ultimate discovery of tubercle bacilli in the sputum elucidated the problem. Again one must not forget that pneumonia may occur, particularly in cases where the wounded person may have been left lying out untended for many hours. It is not surprising that such a complication should occasionally arise nor is it to be wondered at that it may often be undetected owing to its being overshadowed by the traumatic elements of the condition. Infections of other kinds may also complicate the hæmothorax case, notably acute bronchitis or even extra-thoracic conditions. Mention of these intercurrent diseases is made merely to emphasise the importance of studying the hæmothorax patient from every angle so that surgery may not be unnecessarily employed.

In this connection, I think a passing reference should be made to the fact that many injuries of the pleura or lungs, especially when in the neighbourhood of the diaphragm, often occasion the symptoms of sickness, vomiting and pain and even the physical sign of abdominal rigidity. It is important in such occurrences to decide whether such abdominal symptoms and signs referred to are due to the missile having penetrated the diaphragm causing perforation of a viscus and peritonitis, or whether such phenomena can be explained as being in the nature of a referred type.

I have endeavoured in the preceding paragraphs to indicate the means by which hæmothoraces are produced and to indicate briefly the significance of the various phenomena usually associated with them. I feel sure, from experience gained in the last war, that a further appreciation of these facts will tend to limit unnecessary surgical treatment. I will not attempt here to deal with the treatment of these cases since it is not within the province or title of this article. Modern methods of surgical procedure, I believe, will be dealt with in this Journal by my surgical colleagues who are well qualified to discuss such matters.

Medically speaking, however, it may be said that for large effusions, so considerable as to dislocate and embarrass the heart, an operation of some kind is imperative. From what I have said before it must be remembered that the immediate removal of a large amount of fluid is not without risk owing to the possible recurrence of hæmorrhage. Experience gained in the last war suggested that aspiration even on a large scale could be undertaken without any serious risk 72 hours after injury.

Careful determination as to whether the thoracic signs are due to effusion alone or to collapse is essential, and furthermore the importance of contralateral collapse must not be overlooked. A patient exhibiting signs of massive deflation in the opposite lung is far from being a favourable anæsthetic risk.

Let it not be forgotten also that due consideration should be paid to the shocked and collapsed condition of the patient generally, before deciding upon severe surgical treatment.