the health of the people and that it is its duty to define the environmental conditions necessary for the higher state of health which medical science considers can be attained. There is not the slightest doubt that the brave words of Churchill and Bevin are spoken in earnest and that they and other national leaders who are anxious to reorganize our social and economic system for the benefit of the common people would welcome an authoritative statement from the medical profession on the kind of conditions we should plan for. If the profession is willing to make its full contribution to the national effort for victory and for the equally important post-war reconstruction it should begin to do so now.

A CASE OF PULMONARY CONCUSSION ("BLAST") DUE TO HIGH EXPLOSIVE

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It is now well recognized that a proportion of individuals who are exposed to the detonation of high explosive develop pulmonary haemorrhages without any significant injury to the thoracic wall. The number of recorded cases in which such patients have been closely observed and have subsequently died is still quite small. Such a case is recorded below.

Case Report

In November, 1940, a bomb fell on a wooden hut in which the patient, a soldier aged 23, was asleep. No details are available of the immediate effects of the explosion or of the condition of the patient at this time except that he was given morphine 1/4 grain at 4.40 and 1/3 grain at 5.20 and 3,000 units of anti-tetanus serum at 5.30, when he was transferred to hospital.

On admission he was "shocked," dyspnoeic, and cyanosed, and rales could be heard scattered throughout the chest. He had pain in the chest and abdomen and vomited some blood-stained fluid. The abdomen was tender and rigid. The temperature was 100°, and the pulse rate 130 and the respiratory rate 30 per minute. He was given heroin grain 1/12 and atropine grain 1/100, and oxygen was administered. His condition gradually improved. On the morning after admission tubular breathing with an impaired percussion note was found at the left base, but the scattered rales were less conspicuous. A radiograph of the chest showed a diffuse, mottled, woolly shadow throughout the left lung. On the right side there was a similar shadow in the mid-zone and extending out to the periphery from the hilus. The abdomen was still tender and vomiting continued. The temperature was 98.6°, and the pulse rate 120 and the respiratory rate 24 per minute.

It was decided in consultation that the patient should be sent to another hospital, and he was first seen by us thirty-nine hours after the explosion. He was restless and looked ill with high colour and slight cyanosis. There was considerable dyspnoea, with dilatation of the alae nasi on inspiration, but no orthopnoea. He was extremely irritable and resentful of interference, drinking greedily but vomiting large amounts of thin brownish watery fluid which gave a faintly positive guaiacum reaction. His temperature was 99.6°; the pulse rate was 120 and respirations 36 per minute. The tongue was dry and furred. Blood pressure was 126/76. There was generalized tenderness, which made accurate percussion and examination of the back of the chest inadvisable. Movement was diminished on the left side and the breath sounds were obscured by loud rhonchi and scattered coarse rales. The abdomen was distended with flatus and the abdominal wall was rigid and tender. An acute intra-abdominal lesion was suspected, although there was no dullness in the flanks, the liver dullness was not impaired, and bowel sounds were audible. Attempts were made to administer oxygen by means of a B.L.B. mask and spectacle frame, but the patient resisted and would tolerate neither. At 8.30 p.m. his general condition appeared unchanged, although the pulse rate had risen to 140 per minute and there was some congestion of the veins in the neck. His blood pressure was 136/84 and the haemoglobin 125% (Hellige colorimeter).

At this time the indications for treatment seemed somewhat confusing. The furred and dry tongue, the haemoglobin percentage of 125, and the rapid rate of the pulse suggested that plasma should be given. On the other hand, the height of the blood pressure, the distended veins in the neck, the bubbling rales in the chest, and the general appearance of the patient strongly suggested that a venesection should be performed. We decided to do neither, hoping this was a transient crisis precipitated by the journey from the first hospital. As the patient was quiet if left undisturbed, morphine was withheld and he was closely watched. At 11 p.m. it was obvious that his condition was deteriorating. He developed a distressing hiccup, the bubbling rales in his chest could be heard at the end of the bed, his cyanosis was increasing, and the veins in his neck were distended up to the angle of the jaw. The temperature was 102.2°, the pulse rate 144, and the respiration 48. Morphine grain 1/6 was given, but the condition of the patient began to deteriorate even more rapidly. 600 c.cm. of blood was then withdrawn, and this was followed by spectacular improvement. The bubbling rales in the chest rapidly diminished; the patient was quieter, was less restless, and was breathing more easily, and he fell into a light sleep. His blood pressure was not measured lest it disturb him, but the quality of his pulse suggested that it was being maintained at its previous level.

At 5.30 a.m. the patient became much worse and was restless and delirious. The rate of the pulse was 140 per minute, and its volume was poor; there was a systolic blood pressure of 44 mm. Hg, but no congestion of the veins in the neck. The respiratory rate again increased to 40 per minute, but no rales could be heard in the chest anteriorly except in the left axilla. The haemoglobin was 100%. Oxygen was administered with a B.L.B. mask and, as no plasma was immediately available, a transfusion of blood was begun. After the patient had received one-third of a pint clotting occurred. He was then moribund, and died at 7 a.m.

In a sample of blood taken at 11 p.m. the blood urea was 86 mg. per 100 c.cm. and the alkali reserve 60.5 c.cm. per 100 c.cm. plasma.

Post-mortem Examination

The body showed no external signs of injury except a small superficial abrasion on the bridge of the nose. The upper air passages were normal except that they contained a moderate amount of lightly blood-stained thin and frothy mucous fluid. The pericardial fluid was lightly blood-tinged. There were some petechial haemorrhages between the parietal pleura and the thoracic wall and a few larger haemorrhagic areas up to the diameter of a five-shilling piece in size. These were composed of thin laminae of dark blood and were not associated with any recognizable fracture of the ribs or trauma of the thoracic parietes. There was no excess of fluid in the pleural sacs.

Both lungs were large. At least two-thirds of the bulk of the left and about half the bulk of the right lung were consolidated. The visceral pleura showed no laceration, but there were several extensive dark irregular haemorrhagic areas beneath the pleura of both lungs, chiefly disposed about the posterior and basal regions; the largest was roughly rectangular, and measured about 6 by 5 cm. All these lesions were subsequently shown to be continuous, with large foci of deeply seated resolving haemorrhage.

Most of the consolidation in the left lung was basal, posterior, and deeply placed, but there were solid areas deep in

the substance of the upper lobe. The colour of the consolidated parts was that of dark venous blood clot, but their ill-defined edges were bright red, fading off gradually into normally coloured lung. Throughout this dark-coloured consolidated lung there were many scattered areas, ashen-grey in colour and sharply demarcated from the surrounding tissues. The larger areas were about 2 by 1½ cm., and were roughly polygonal in shape. The light red margins of the consolidated areas were firm and elastic; the deeply coloured solid lung was wooden in consistency and quite elastic; the ashen-grey foci were firm but friable.

The lesions in the right lung were similar to those in the left but not so extensive, and whereas a large portion of the left lower lobe appeared to be occupied by a lesion roughly resembling a massive organizing pulmonary infarct, the lesions in the right lung were smaller and more widely scattered. The sharp demarcation of the ashen-grey areas was just as noticeable in the right as in the left lung.

A few small submucous haemorrhages were found in the body of the stomach and there were recent symmetrical subarachnoid haemorrhages over the convexity of each occipital lobe extending forwards for about 4 cm. from each occipital pole. These haemorrhages were not severe in degree and there was no naked-eye evidence of damage either to the overlying skull and dura or to the underlying brain.

On histological examination of the consolidated lungs the macroscopic changes were seen to be largely the consequence of widespread intra-alveolar capillary haemorrhage. In the dark firm elastic areas the appearances bore a striking resemblance to those found in haemorrhagic infarction. There was the same preservation of the outline and structure of the alveolar walls, the same tight packing of the alveoli with partly haemolysed and rather poorly staining red cells, and the same lack of free tracking of blood through the substance of the lung. There was no microscopic or macroscopic evidence of tracking of air through the lung, as one might expect to find if there had been alveolar rupture of any degree. The general resemblance to infarction and the lack of convincing evidence of widespread alveolar rupture incline us to the belief that the haemorrhage in this case arose from diapedesis or from the rupture of grossly dilated capillary vessels. This view receives some confirmation from the fact that a considerable degree of generalized capillary dilatation was found in sections taken from air-containing parts of the lung unaffected by haemorrhage.

No striking or constant change was observed in the air-conducting system of the lung, but in all sections of the regions showing haemorrhagic consolidation there was a rather striking degree of dilatation of the respiratory bronchioles and alveolar ducts.

An unexpected and rather surprising histological picture was presented by the ashen-grey friable areas lying in the midst of the massive areas of haemorrhagic consolidation. Here every alveolus contained a precisely staining close-meshed fibrin network in the meshes of which considerable numbers of wandering cells and partly haemolysed red cells were lying. The general appearance under the low-power bore a rather striking superficial resemblance to that of lobar pneumonia in the stage of red hepatization.

Closer examination of the wandering cells showed them to be predominantly monocytic. The capillaries in the alveolar walls were considerably dilated, and towards the edges of these areas of pseudo-pneumonic consolidation more and more alveoli were seen to contain coagulated protein with a scantier fibrin network and fewer cells. There was the same general preservation of the alveolar walls in these areas as was found in the frankly haemorrhagic parts of the lung and the same degree of dilatation of the respiratory bronchioles and alveolar ducts.

These appearances bear more than a superficial resemblance to those found in the lung in those cases of rheumatic infection dying of acute cardiac failure which show the condition known as "rheumatic pneumonia."

Sections taken from the peripheral parts of the areas of haemorrhagic consolidation where the lung was firm and airless but bright red in colour showed all alveoli in these zones to be fairly tightly packed with fresh, well-staining, fully haemoglobinized red cells. Wandering cells were absent and there was no fibrin formation. The alveolar walls were for the most part intact.

Discussion

The changes seen in these peripheral areas bore a striking resemblance to the appearances found in the lungs of patients dying soon after exposure to the detonation of high explosive, in which cases recent pulmonary haemorrhages are so often found post mortem. In reviewing a series of post-mortem examinations of seventeen of these cases we have been struck by the variability in extent of these haemorrhages, and it appears reasonable to relate this variation to the period of survival after exposure to detonation. In the case here described the survival period was longer than in any case we have previously observed and the haemorrhagic areas were considerably larger.

These facts should be taken in conjunction with the observation that the areas of haemorrhagic consolidation were surrounded by deep zones of recent bleeding, and we are inclined to believe that patients who develop pulmonary haemorrhages without external trauma to the thoracic wall after the detonation of high explosive continue to bleed into the lung for a period which may amount to forty-eight hours or so.

It would be dangerous to draw any definite therapeutic conclusions from a single case observed for so short a period. It seems probable, however, that on admission the patient was suffering from acute pulmonary congestion bordering on acute pulmonary oedema, and that venesection might well have been performed earlier. The use of concentrated serum was considered, and might have been employed. It also seemed clear that at 5 a.m. the patient passed into a state of "shock" and that the great improvement following venesection had led to a false sense of security. Although he was asleep his blood pressure should have been taken more often, and a plasma transfusion should have been given when it had fallen.

This patient also illustrates the dangers involved in transport by ambulance. When he left thirty-six hours after the explosion his condition had improved and, in the consultation which decided his transfer, he was considered to be quite fit for the journey. When first seen by us his condition was poor and gave obvious cause for anxiety. In the light of the post-mortem finding of continued bleeding it seems clear that these patients should be rested as strictly as if they had had recent severe haemoptysis.

The interpretation of the signs referable to the cardiovascular system is difficult. The bubbling rales in the lungs with dyspnoea and engorgement of the vessels of the neck suggested severe congestion of the pulmonary vascular bed, and this interpretation was supported by the marked improvement after venesection and by the post-mortem appearance of the lung. On the other hand, if there was any change in the blood pressure it was in the nature of a rise, and it is not easy to reconcile this with any serious impairment of blood flow through the lungs.

Summary

A case is reported in which a man aged 23 lived for fifty-one hours after exposure to the detonation of high explosive.

The clinical picture is described and certain suggestions as to treatment are made.

Reasons are given for regarding the pulmonary haemorrhage as being progressive and associated with pulmonary congestion.

Throughout the areas of haemorrhagic consolidation in the lung there were many foci superficially resembling pneumonic consolidation; these are described.

We are greatly indebted to Dr. J. N. O'Reilly for his co-operation in the investigation of this case.