CHAPTER 11

BLAST INJURIES AND POISONING BY FUMES

The clinical effects of blast could usually be explained in terms of the positive (compression) and negative (rarefaction) phases of a wave caused by an explosion. While most of these effects were capable of simple explanation, some of the observed phenomena were curious.

The effects of aerial blast were notably capricious, owing no doubt to the labile and compressible nature of the medium in which we live and breathe. The front of an advancing wave was often irregular, so too were its effects. It could be readily understood that the caprice of aerial blast might produce less severe damage in persons blown into the water by a bomb explosion than in those affected by submarine blast. The power unleashed by the underwater explosion of a depth-charge might be sufficient to erupt a vast column of water with such violence that the effect would be communicated to the hulls of ships two or three miles distant.

In April 1941 during the evacuation of Greece a merchant ship anchored in Piraeus just ahead of *Perth* was hit and sank in a few minutes. Some of the survivors were suffering from bomb blast and it was noted that those who had actually been blown into the water did not suffer severe injury. On the other hand, when *Perth* was torpedoed many men who were already in the water were killed by blast when the ship was hit again: one succumbed to a ruptured viscus which could not be treated.

Chest injuries due to aerial blast were not uncommon and were explained by the nature of the thoracic cage whose response to powerful waves of compression and rarefaction made damage to the lungs possible. The multiple lung injuries found after fatal cases of blast might resemble those occasionally seen after accidents where a severe fall or violent propulsion of the body had occurred. Aerial blast gave rise to severe shock, pain, restlessness, difficulty in breathing and cyanosis, with underlying lesions of patchy haemorrhage and consolidation. Visceral bleeding also occurred in the abdominal organs when these were affected by blast, and sanious diarrhoea and haematemesis might appear. These were often accompanied by haemoptysis.

Thoracic injury after underwater blast was less common than after aerial bombing: abdominal injury occurred more frequently, probably owing to the lack of protection afforded by the abdominal wall when exposed to the violent thrust communicated to the water by the force of the explosion. A life-jacket gave more protection to the thorax than to the abdomen, but a certain amount of damage might be done to the bases of the lungs through the transmission of explosive force from the abdomen to the diaphragm leaflets and thence to the lungs. This mechanism might suggest the co-existence of abdominal injury also when there were signs of pulmonary injury. Some protection could be afforded to the abdominal organs of persons surviving the sinking of a ship subjected
to submarine explosion, provided they swam or floated on the back, thus saving the abdomen from the impact.

Abdominal blast caused bleeding under the serous coat of the organs, and severe damage might cause deeper lesions. Immediate treatment was for shock, morphine and oxygen being of particular value. Different types and degrees of severity in abdominal blast were recognised. In the least severe forms pain and rigidity would perhaps be the only obvious signs for several days, but bleeding from bowel or urethra might indicate more severe injury, and this could be followed by contusion or abscess. Perforation of hollow organs might be manifest early, and if unrelieved would be rapidly lethal. The question of laparotomy had to be decided by these signs, but it was found unwise to defer decision until the issue was clear: such clarity was only too often attended by dangerous risk. It was pointed out that signs confined to the upper part of the abdomen might be caused by a chest injury, but when these spread to the abdomen a severe abdominal lesion was highly probable. Signs confined to the abdomen from an early stage were usually associated with a fatal injury.

Surgeon Commander C. Keating, R.N., who contributed an article to a joint discussion on blast injuries, pointed out that combined intra-abdominal and infra-thoracic injury often occurred, and that basal pulmonary damage was accompanied by a brief relaxation of the abdominal muscles at the end of the expiratory phase. Patients suffering from the effects of immersion blast described sudden pain in the back, often with a sensation of constriction and numbness of the lower limbs which might make self help difficult.1

Naturally there was a wide disparity of symptoms, ranging from those of severe shock and pain, due to multiple vascular or visceral injuries, to milder manifestations caused by intra-mural bleeding in the hollow organs. The misleading period between injury and the signs of peritonitis needed special emphasis. It was well illustrated by an experience of the medical officers of Hobart, who in February 1942, as mentioned, treated survivors from the British ship Norah Moller, which had been attacked by bombers.

In describing the incident, Surgeon Commander L. Lockwood wrote:

Another type of injury dealt with was due to the effect of blast at the time of bombing. Two cases received the blast injury while in the bombed ship; the third was stated to have been injured by the compression wave in the water after having jumped overboard, due to a bomb bursting in the water not very far away. At first sight this type of the injured does not appear to be very ill. The patient complains of some pain and discomfort in the abdomen; one had a moderate degree of haematemesis. A characteristic feature of all three was their inability to micturate; all had to be catheterized. Abdominal rigidity did not develop until some hours after admission to hospital next day, that is until at least twenty-four hours after receipt of the injury. What made things more difficult was the fact that all three cases were Chinese and could not speak any English.

When abdominal rigidity developed, Professor Reddinghuis (of the Central Civic Hospital, Batavia) at once operated. Two cases were found each to have a rupture of the ileum; in the third the sigmoid colon was ruptured. In the latter case a

left sided colostomy was performed. The first two cases were found to have large tears of the ileum; owing to their shocked condition resection and anastomosis were inadvisable, and exteriorisation of the affected loop of ileum was performed in each case; that is, the ruptured segment of bowel was brought outside the abdominal wall and sutured to the peritoneum. After the patients had recovered from the initial shock and their general condition improved, it was proposed to resect an appropriate amount of small intestine. In the case of the sigmoid colon lesion, it was hoped that this would heal and that later the colostomy could be closed. However, all three cases died, and autopsy showed that in each general peritonitis had developed.

The combination of severe burns with blast injuries undoubtedly increases the hazard very greatly.

In July 1943 Hobart herself was torpedoed, and there were many cases of blast injury among her crew. Surgeon Lieutenant R. M. MacIntosh recorded that he was in the wardroom when the torpedo struck the ship. After a time he became conscious that he was in water up to the knees and that wreckage restricted movement. He was undoubtedly suffering from the effects of blast concussion, but managed to escape by the quarterdeck and eventually reached the sick bay, where the injured were being given preliminary treatment by the sick-bay petty officer. The commonest type of injury was that due to blast effect, with concussion or shock. Some patients who were badly shocked were treated with morphine, warmth and intravenous serum, and half-hourly charts were kept. Hobart reached port about 38 hours after the attack and the injured were landed at an American naval hospital; their subsequent progress was satisfactory.

Blast injuries of the brain were perhaps responsible for more damage than was realised, as structural changes might exist unsuspected in the central nervous system of persons dying as the result of bomb explosions. Lesser grades of the same type of damage fell into the sphere of the neurological surgeon, and explorations revealed lesions only detected by careful study of the peripheral effects. Surgeon Captain L. C. Rogers, consultant in neurosurgery to the Royal Navy, referred to the curious apathy and dejection of some patients who were blown up by the force of an explosion. The pathological lesions in the brain were usually vascular in type, due apparently to leakage from blood vessels with possible sequels of more serious damage occurring later. It did not follow, however, that apathy and depression were always due to demonstrable lesions. The National Research Council, Washington, D.C., investigated the nature and incidence of acute emotional disturbances in seamen of the merchant marine who had been torpedoed. Some of these men had "startle" reactions, personality changes and symptoms of psychoneurosis and could not return to sea. It was found that the torpedoing of their ship bore no relation to the disturbance, and in general the most likely causal factors were considered to be endogenous and not exogenous.

Although attack by gas never appeared to be more than a possibility, it was known that the Japanese did possess chemical warfare weapons in some form, probably mustard or lewisite, and precautions had to be taken.
One medical officer, Surgeon Lieutenant T. A. McLean, received special training in the medical aspects of gas warfare, and visited various parts of Australia and New Guinea in order to advise on defensive precautions. A report received by the Naval Board of a tragic mustard gas casualty in Canada illustrated the necessity for adequate instruction being given on (a) the gases themselves, and (b) the procedure to be followed when an accident occurred. A lieutenant in the Canadian forces was demonstrating the use of gas training expedients to his platoon. He ignited a mustard gas bomb but it did not explode; 15 minutes later he approached and kicked it. It exploded and he was drenched from head to foot with liquid. He rinsed his eyes and applied ointment immediately, but did not remove his clothes until 10 or 15 minutes later. He was admitted to hospital, and despite all the efforts of the medical staff, died seven days later.

It was unlikely that any form of chemical warfare would be successful in an attack on ships at sea, but all men were supplied with and trained in the use of gas masks, and certain other items such as bleach anti-gas ointments were provided. On shore more elaborate precautions were necessary, and protective clothing was provided, as well as other necessary equipment and decontaminating centres.

Industrial types of poisoning were sometimes seen in the R.A.N. through contamination with a poisonous substance or vapour. Toxic symptoms could arise by contact with poisonous liquids such as benzol, with an associated affection of the blood cells. Tetryl dermatitis could also arise in susceptible persons from contact, and arsenical poisoning might also be due to the handling of castings. More familiar was the toxic effect produced by burning off lead paint in a confined space: this was occasionally responsible for symptoms of plumbism.

A more important and potent cause of poisoning by fumes was that due to nitrous gases, which could easily give rise to toxic signs when imperfect combustion of nitro-explosives such as cordite had taken place in partly enclosed areas of a ship. In confined spaces such explosions could produce a concentration of gases sufficient to cause not only toxic but lethal effects. Of the fumes arising in this way those of the nitrous type were found to be far more toxic and potentially lethal than carbon monoxide, which was produced at the same time.

In this type of respiratory poisoning there was a long, latent period between inhalation and the appearance of serious symptoms. In fact, grave damage could be inflicted before the real state of affairs was realised. Nitric and nitrous acids were formed on contact of the gases with moisture in the respiratory passages. These acids predominantly affected the bronchial tree, in particular the alveoli, and could bring about a progressive oedema of sufficient severity to cause death. The Naval Technical Instructions included an account of this condition. It laid special stress on the minor early symptoms in contradistinction to those arising after the deceptive latent period was over. These later manifestations were dangerous, and caused a characteristic clinical picture. This was one of grave
respiratory embarrassment with increasing dyspnoea, distressing though productive cough, accompanied by cyanosis, and wide, scattered, moist rales in the lungs.

Treatment was directed to relieve pulmonary oedema and congestion: to do so the administration of oxygen was necessary, using an effective method such as a B.L.B. mask. More important still were the measures based on prevention, all care being taken to limit the possibility of exposure of men to danger. At the time of possible exposure to nitrous fumes danger was always to be suspected, and the presence of an air or oxygen-breathing apparatus was found highly desirable: as a protection against fumes even a service respirator was of value. Further, all men exposed to the hazard of nitrous fumes were regarded as suffering from a serious degree of poisoning, and they were not allowed to be exposed to exertion for several days; no intravenous transfusions were permitted, and all possible means of reducing vascular congestion were taken.

When a ship was exposed to nitrous fumes the possibility of potential nitrous poisoning had always to be kept in mind until this risk had passed. Where bacterial infection supervened a drug of the sulphonamide group was administered, and atropine or adrenalin could be given with advantage. Morphine was also of value, particularly in allaying restlessness.