matter of ventilating the lungs; it is a trinity of *ventilation*, *circulation*, and *warmth*. Essentially it is an attempt to revive asphyxiated nerve cells in the medulla and spinal cord by a circulation of warm blood oxygenated by moving lungs.

Ventilation.—When asphyxiated by failure of respiration or circulation these nerve cells gradually die—usually in a few minutes, but they may remain viable for an hour, rarely more. When they are dead all hope is gone. As they die, the elastic tone of the muscles—which they maintain—progressively declines. When the diaphragm has in this way lost all its tone the lungs contract by their own elasticity; they pull the diaphragm right up and become deflated (as in the warm cadaver). At this stage (1) Schäfer's method has become impotent because the elastic recoil of the diaphragm (for inspiration) has gone; (2) Silvester's method will still produce some ventilation owing to elasticity in the thorax independent of its pectoral muscles; (3) the rocking method will effectively shift the flaccid diaphragm up and down independently of tone, and hence should save cases in which loss of tone had gone too far for the Schäfer method to revive.

Circulation.—The heart muscle itself does not die quickly, for after drowning it can be revived by perfusion with oxygenated saline solution—a baby's heart after several hours. The diagram shows how well the head-down tilt perfuses the coronaries. But in shock (hypotonia of muscles) the heart may also fail from mere emptiness because insufficient venous blood gets back to it through veins and capillaries unsupported by tone of muscles. Hence in drowning cases (always shocked) it is important to encourage venous return, as by the compression of abdominal veins which occurs in Schäfer's method or, far more potently, by the rocking method—as already explained. To fill the heart and respiratory centres with blood a head-down slope of about 10 degrees (1 in 6) should be chosen (as on a beach) for manual resuscitation when possible. To fill an empty heart is a quick way to restart it beating. When you tilt steeply to empty out water you incidentally fill the heart and change the coronary blood.

Warmth.—This is indispensable in shock treatment generally—e.g., by hot-water bottles and blankets; also by impervious coverings, to prevent heat loss from evaporation and cold winds. Nerve cells are paralysed by cooling but revive at once on re-warming. With a crippled circulation the indispensable nerve cells in the upper spinal cord will quickly cool and their blood vessels constrict. Hence it may prove very important to warm the back of the neck by a rubber bag early in the resuscitation.

Finally, transcending all else, it is essential to start Schäfer's method instantly: if ventilation (checked at the mouth) is satisfactory, persevere; if not, try Silvester plus expiratory pressure on the upper abdomen, or, better, proceed to rocking if available.

Confronted with so many lamentable failures of Schäfer's method our Naval medical officers seem entirely justified in advising for preference the rocking method, which is now included in BR 25/43, "First Aid in the Royal Navy."

Explanation of the Diagram

Since gravity in rocking affects only the longitudinal components of the blood vessels they may be represented as straight tubes. For clarity, the vessels of the arms (extended on the stretcher handles) and head are omitted: they are similar to the legs. Natural circulation is assumed to have stopped. The piston of this air-pump is formed by the weight of the abdominal viscera (capped by the flaccid diaphragm) sliding up and down. The patient is prone on the rocking stretcher.

When *head-down*, venous blood from the legs and abdomen falls past the right heart into the lung capillaries—reflux prevented by valves in the right heart and leg veins. Veins in the head and extended arms fill. The right heart fills (which may restart it). The aortic valves are banged shut by a pressure of 2 to 3 feet of blood: hence blood is forced into the coronary arteries and joins the stream flowing from the right heart into the lungs (course indicated by the dotted line C).

When *feet-down*, the venous blood which has just been oxygenated in the lungs cannot flow back into the right heart or into the legs (valves closed), but is free to flow past the open aortic valves into the leg capillaries. Blood from head and extended arms refills the right heart.

Thus analysed, slow rocking appears able to aid, or even to maintain unaided, both circulations in the drowned—including the all-important coronary circulation. Silvester's method—unless the epigastrium is compressed—seems to be ineffective on the circulation: Schäfer's method should push some abdominal blood into the lungs but perhaps no further.

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BLAST INJURY TO THE ABDOMEN BY DEPTH CHARGE

BY

D. DENHAM PINNOCK, M.B., F.R.C.S.

Lieut.-Col., R.A.M.C.

AND

PAUL WOOD, M.D., F.R.C.P.

Lieut.-Col., R.A.M.C.

As little information has been published concerning the effects of blast injury to the abdomen the following cases are recorded in order to supply additional data. Under conditions of active service it is not possible to do justice even to the very scanty literature. However, it can be mentioned that, in Zuckerman's (1940) experiments on animals exposed to air blast, haemorrhagic pulmonary lesions were the chief feature, and that according to Hamilton Bailey (1942) blast injury to the abdomen may be followed by melaena. That author also states that, although laparotomy has been performed with negative findings on several occasions, rupture to the colon has been revealed by timely operation. Breden, d'Abreu, and King (1942) record a number of cases of abdominal blast injury from depth charge, and they refer to papers by Atkins (1940) and by Gordon-Taylor (1940). These records show that persons swimming in the water close enough to an exploding depth charge may suffer blast injury to the abdominal viscera, characterized by shock, haematemesis, melaena, and pyrexia; recovery may take place without surgical intervention; perforation of the gut may sometimes be successfully treated by surgical repair; many of these patients have died before conditions were suitable for careful study or surgical help. Associated blast injury to the lungs is common.

In December a naval vessel was torpedoed and sank rapidly. The crew took to the water and were swimming away from the ship when two depth charges exploded in quick succession. Some who had managed to clamber on to rafts escaped all injury; others who had swum further afield felt the shock but suffered no ill effect; while about 15 felt as if kicked in the stomach or pelvis and were sufficiently injured to be taken to hospital. Of these 15, five were seriously ill and form the subject of this report. The other ten did not appear ill and had no symptoms suggesting serious trauma to internal organs; they recovered uneventfully.

The history given by four of the seriously ill patients was very similar (the fifth was more or less unconscious owing to a head injury received on the ship): they were all swimming in the water with their heads out and supported by life-jackets. Three felt as if kicked in the stomach, the other in the back of the pelvis. After receiving the injury they were still able to swim until they were picked up. They were all seen in hospital within four hours of the event.

Case I

This patient had most of his chest out of the water when the depth charge exploded, for he was trying to lift himself on to

a raft. He complained of increasing abdominal pain and had vomited blood, but he developed no pulmonary signs or symptoms.

Examination revealed board-like rigidity with considerable tenderness all over the abdomen, normal lungs, and no fractures or external injuries. His general condition seemed good: the pulse rate was 90 beats a minute and the pulse volume fair. The following day he still seemed fairly well, the haematemesis having stopped and the pulse rate and blood pressure remaining in the region of 90 and 110 respectively, but the board-like rigidity and tenderness of his abdomen had lessened very little. A radiograph of his lungs confirmed the fact that there was no pulmonary blast injury. On the third day it was decided to operate, as his general condition was deteriorating and the abdominal signs were not improving. He was given a pint of plasma before and a second pint during the operation. Laparotomy revealed perforation of the transverse colon with localized peritonitis and two small tears in the peritoneum of the floor of the pelvis. The perforation was repaired and a proximal colostomy tube inserted. The peritoneum was drained.

Rather unexpectedly the patient went steadily downhill, with signs of increasing circulatory collapse, despite repeated infusion of plasma, rectal saline, and fluid by mouth. He died on the fifth day. No necropsy was performed.

Case II

This patient gave the typical history and noted that there was no pain in his chest immediately after the explosion, but that within the next four hours pain around the lower thorax increased steadily. He vomited a good deal of blood and may have coughed up some also.

On examination he was found to be considerably shocked, with a pulse rate of 136 and a small pulse volume. There was board-like rigidity and marked tenderness over the whole of his abdomen, and, although it was thought he had ruptured some viscera, he was too ill for surgical intervention. Repeated infusion of blood plasma and continuous oxygen administration failed to improve his condition, and he died with signs of increasing shock in about 24 hours. No necropsy was performed.

Case III

The history in this case was similar to that of the others: after being withdrawn from the water the man felt increasing pain across the lower thorax and began to cough up blood; there was no haematemesis.

Examination revealed coarse rales over the left lower lobe, with diminished air entry and normal percussion note. There was moderate rigidity of the abdomen, but in less degree than in the previous two cases. The pulse rate was 100 beats a minute, with fair volume. The patient's general appearance was good and he did not look as if he had suffered serious intra-abdominal injury. When seen 24 hours later, however, his condition was worse, shock having increased and signs of injury to the bowel being more evident. There was board-like rigidity of the abdomen with considerable tenderness, but still no haematemesis; haemoptysis was less, but the pulmonary signs were more extensive.

After adequate resuscitation laparotomy was performed and seven perforations of the small intestine were found. The appearance of the gut suggested that many loops of small intestine were severely bruised at the point where they crossed the pelvic brim. There were localized areas of simple bruising, localized areas where bruising had progressed to gangrene, and seven places where a gangrenous section had perforated. In several areas loops of intestine obviously damaged had become stuck together and were not separated; fistulae could well have formed between these adjacent loops. There was widespread peritonitis. Repair was effected by sewing up the perforations and inverting the gangrenous areas that had not yet perforated. The peritoneum was drained, but jejunostomy was not carried out.

The subsequent course was steadily downhill, and was characterized by fever and extending signs of blast-lung, by increasing circulatory collapse—despite almost continuous intravenous infusion of blood and plasma—by complete paralysis of the gut, and by evidence of increasing peritonitis.

After a gallant but hopeless fight the patient died five days after the injury. Necropsy showed extensive haemorrhagic lesions in the lung, with secondary bronchopneumonia, widespread peritonitis, and gross distension of the gut, but no new perforations. A long section of the small intestine was gangrenous, possibly from mesenteric thrombosis following the injury, as no damage to the mesentery was found at the operation.

Case IV

This patient was the only one who received the shock of the explosion in the back of the pelvis, instead of in the stomach. After being dragged out of the water he complained not only of pelvic pain but of increasing raw bilateral lower thoracic pain; he then developed a painful cough with haemoptysis, and each time he coughed it was stated that red blood was forced from the rectum, but the amount was not noted. He tried repeatedly to pass water, but was unsuccessful for four hours, when a catheter was passed with difficulty and normal urine was withdrawn.

On examination he had obvious signs of blast-lung with widespread rales and diminished air entry over the right lower lobe. A radiograph of his chest confirmed the diagnosis and also revealed changes at the left base. In order to avoid further damage the rectum was not examined (what bleeding there was having stopped), nor were there further signs to suggest serious trouble here.

This patient differed from all the others in that his abdomen was soft and not tender. His general condition was good—the pulse rate 90 to 100, with fair volume. He received no special treatment, had no infusion of blood, plasma, or fluid, and made a steady and fairly rapid recovery, so that within 48 hours he was obviously out of danger.

Case V

This patient was unable to give an adequate history owing to the fact that he had received a head injury when the ship was torpedoed and was unconscious in the water when the depth charge exploded. When first seen he was extremely ill, with haemoptysis and physical signs of blast-lung, haematemesis, and board-like rigidity of his abdomen with extreme tenderness. His mental state was confused and drowsy, but there were no localizing signs in the C.N.S. He was badly shocked, and died within 48 hours. He was far too ill to stand surgical intervention. No necropsy was performed.

Commentary

It is not the purpose of this report to discuss the features of blast-lung, but attention is drawn to the fact that in the one patient (Case IV) who had a proved lung lesion the abdomen remained perfectly soft but without gut injury within the peritoneum. Bleeding from the rectum in this case may have been due to damaged haemorrhoids or to small tears in the bowel wall, and it will be remembered that it was this same patient who felt the shock in the back of the pelvis instead of in the abdomen. In contrast, Case I, in which there was a proved gut injury but no symptoms, signs, or radiographic evidence of blast-lung, had marked rigidity of the abdomen. In considering the management of these cases the significance of the abdominal rigidity was questioned, for it has been stated that blast-lung may produce such rigidity. These cases suggest that board-like rigidity of the abdomen should be regarded as evidence of perforation of the gut, and should not readily be put down to blast-lung.

All four patients who had gut injury died, which suggests a worse prognosis than that indicated in the literature described above. When first seen, and for 36 hours afterwards, the condition of two of them was deceptive in that they did not look severely shocked or as ill as one might expect in patients with perforation of the intestine: the facies of moderate well-being was completely unlike the aspect of the common abdominal catastrophe. The rigidity was much more pronounced than we had previously seen in any case of perforation of the intestinal tract; we had only once seen it equalled, and that in a man with rupturing dissecting aneurysm of the abdominal aorta.

That the general appearance was so good misled us to the extent that we were loath to accept perforation as the cause of the rigidity (recollecting what we had read in the Army Medical Bulletin No. 5 of the great rigidity encountered in cases of blast-lung), and the opinion was formed that the condition was due to blast-lung, with probable actual damage to the abdominal muscles. Even 24 hours later, when there was obvious deterioration in the condition of these patients and when operation was decided upon, one of our surgical colleagues remarked that he had not seen a patient with a perforated viscus of so many hours' standing with so good a facial aspect, and could not believe that such a catastrophe had occurred.

It is probable that these patients would have done better with immediate surgical intervention. On the other hand, the lesions

found at laparotomy in Case III make it clear that perforation results from gangrene supervening on gross bruising, and that many patches of bruising do not lead to gangrene and perforation, so that too early laparotomy might defeat its purpose if perforation were to occur subsequently. However, the findings point to the necessity for exploration of the abdomen in cases of this type, and in future one would prefer to explore—even in the early hours—and find nothing more than bruising of the viscera, and would be prepared to operate a second time if a later perforation occurred.

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INTOLERANCE TO LIVER EXTRACT IN PERNICIOUS ANAEMIA

BY

ELIZABETH DELIKAT, M.D.Bratislava

Assistant Pathologist, the Royal Hospital, Wolverhampton

It is a matter of common experience in clinics devoted to the treatment of pernicious anaemia that a certain small proportion of patients treated with liver by the parenteral route in course of time become "sensitive" to the injections. In such cases treatment may successfully be carried on by the oral route; this method of administration of liver extract is, however, extremely wasteful, and the Ministry of Health has made an Order, dated May 21, 1941, under Regulation 55 of the Defence (General) Regulations, 1939, which lays it down that liver extracts shall not be administered otherwise than by injection. At the suggestion of Dr. S. C. Dyke, Director of the Pathological Department of the Royal Hospital, an inquiry has therefore been undertaken into the possibility of inducing patients who had become sensitive to liver to tolerate their requirement by parenteral injection.

Out of about 200 patients with pernicious anaemia attending the department over the past fifteen years 14 were known to have become intolerant to liver by parenteral injection. None of these gave any previous history of allergic manifestations, but in all cases the symptoms of intolerance were those usually associated with allergic hypersensitivity. These ranged from slight local manifestations at the site of injections, such as redness, induration, and itching, through all gradations up to severe constitutional reactions associated with generalized urticaria, vomiting, failure of vision, and attacks of typical spasmodic asthma. In some cases the symptoms were alarming, and necessitated the detention of the subjects in hospital; in the milder cases they responded readily to ephedrine by mouth, and in the more severe to adrenaline hypodermically. It was found that once sensitization had been established it extended to all brands and types of liver extracts; attempts to carry on treatment with preparations other than those responsible for the sensitization were uniformly unsuccessful. In some cases the allergy was only transient; in others it was persistent and had necessitated a change-over to administration by the oral route. Three cases of this latter type formed the subject of this investigation.

Treatment of Three Cases

Case 1.—Man aged 52. Under treatment for pernicious anaemia since May, 1939. Maintenance dosage, 4 c.cm. of liver extract parenterally every six weeks. In Oct., 1940, after the usual injection he developed generalized urticaria, with puffiness of the face and failure of vision; thereafter he was treated with liver extract orally, 1 oz. daily, until Dec., 1941, when it was decided to attempt desensitization. Intradermal tests with 0.1 c.cm. of two different brands of liver extract gave strongly positive skin reactions. The attempt at desensitization was begun with a dose of 0.1 c.cm. of liver extract, which was tolerated well; increments of 0.1 c.cm. were made at each successive dose, given every second day until a

dose of 1 c.cm. was reached; thereafter an increment of 0.2 c.cm. was used. The patient attended every third day, and remained in hospital for two hours after each injection until he could tolerate 3 c.cm. of liver extract in one dose. This dose was increased by 0.5 c.cm. at two-weekly intervals until 4 c.cm. could be given in a single injection. The patient now tolerates this dose every four to five weeks, and on it a normal blood picture is maintained. On some occasions after this injection of 4 c.cm. of liver extract he complains of slight headache and shows some swelling of the forehead; this disappears on giving ephedrine. He is admitted to hospital for two hours for each monthly injection; probably it will be possible to dispense with this precaution in the future. The intradermal tests with liver extract are still strongly positive, though less so than before desensitization.

Case 2.—Woman aged 61. Under treatment for pernicious anaemia since May, 1939. On maintenance dose of 4 c.cm. of liver extract parenterally every two months until 1940. In March, 1940, she collapsed after her injection; the same thing happened again in May, 1940. Treated by liver extract orally, at first 1 oz. and later 2 oz. daily. This dose was not enough to maintain a normal blood picture, and in Sept., 1941, it was decided to attempt desensitization. Somewhat surprisingly, skin tests with liver extract gave negative results. For the next five months she was admitted to hospital for one day every four weeks and received her liver extract parenterally in divided doses, starting with 0.1 c.cm., with increments of 0.1 c.cm. every 20 minutes so long as no reaction occurred; on the occurrence of a reaction time was allowed for the symptoms to subside, when the previous dose was repeated. The reactions consisted of tachycardia, vomiting, and malaise; there were never any dermal manifestations. By Jan., 1942, the patient tolerated a dose of 2 c.cm. of liver extract in two injections of 1 c.cm. at an interval of one hour. At this time the state of the blood was not satisfactory, but an attempt to raise the total dose to 2.5 c.cm. resulted in severe collapse. In March, 1942, owing to the unsatisfactory state of the blood picture she was admitted to hospital. She then received 6 c.cm. of liver extract parenterally in divided doses over two days. After a single dose of 1 c.cm. she developed for the first time generalized urticaria, and intradermal tests with 0.1 c.cm. of liver extract were found to be strongly positive. In spite of this she now tolerated a dose of 0.5 c.cm. of liver extract, and a desensitizing course was started from this level with increments of 0.1 c.cm. daily up to a single dose of 1.5 c.cm. This injection, however, again gave rise to generalized urticaria, and had to be repeated the next day, when it was well tolerated. Desensitization was thereafter continued with increments of only 0.1 c.cm. up to a dose of 2 c.cm., and later of 0.5 c.cm. until 4 c.cm. was tolerated in one dose. A total of 34.5 c.cm. of liver extract was given in 21 days. The patient now receives 4 c.cm. of parenteral liver extract in a single dose every four weeks without disturbance.

Case 3.—Female aged 44. Under treatment for pernicious anaemia since Aug., 1940. Maintenance established on a dose of 2 c.cm. of liver extract parenterally every two months. In May, 1940, after her usual injection, she developed generalized urticaria and collapsed. On three more occasions she developed urticaria after her injection. In Jan., 1942, intradermal tests with 0.1 c.cm. of liver extract were strongly positive. This patient has been unable to spare the time for frequent attendances at the hospital, and in her case no serious attempt at desensitization has therefore been made. She attends every six weeks and receives her requirement of liver extract in divided doses, starting at 0.1 c.cm., with increments of 0.1 c.cm. at intervals of 20 minutes until the first signs of intolerance appear; these in this case are flushing of the face and a feeling of tightness of the throat, and usually appear after a single dose of 0.3 to 0.4 c.cm. She then goes home and returns on the following day, when we have found the injections to be well tolerated. Dosage is started at the level reached on the previous day and proceeds at increments of 0.2 c.cm. at 20-minute intervals. The full requirement of 4 c.cm. for six weeks is usually injected in two days.

In all our cases the symptoms of intolerance to liver injections appeared only after several months or years of parenteral treatment; the interval between the injections was at least six weeks, for the reason that no patients under maintenance treatment in this department receive their injections at shorter periods.

Discussion

In view of the facts there is every reason to suppose that the intolerance is an anaphylactoid phenomenon and due to "allergic" sensitization, the liver acting as an antigen. This is the usually accepted view (Criepe, 1938; Jones, 1939). Our experience, using different brands and types of liver extract, would lead us to believe that none are free from bodies capable of this antigenic action. Various methods of desensitization have been suggested: splenic extract (Schlesinger, 1930); histamine (Corelli, 1935); "quotentin," which appears to be an empirical remedy alleged to contain hypophysial, parathyroid,