ture is more likely to minimize ketosis than is complete starvation. A return to *insulin* and the usual diet should be made *as soon as possible*, and as an emergency is likely to arise the situation should be reported to a doctor. Extra doses of insulin may be necessary to re-establish the previous state of health, and some patients may require frequent doses of insulin and carbohydrate (perhaps four-hourly) to rescue them from serious ketosis.

If a person's insulin supplies are running short it would probably be fairly safe for a few days to economize by taking half the usual dose with the usual diet, except that *no fat* should be eaten. More value will be got out of a limited amount of soluble insulin given in three or four small doses than if it is given in one or two bigger doses per day—for instance, 10 units eight-hourly has a better effect than 30 units once a day. This does not apply to protamine-insulin, and is one advantage of the latter.

Many diabetics are very conservative about the brand of insulin they use, but they should realize that these are all the same, and should not hesitate to use any brand (that is, A.B., Boots, B.W.) if they cannot obtain temporarily their usual one. Also they may have to use a different strength of insulin for the time being—perhaps 20 units per c.cm. strength instead of 40 units per c.cm.

If insulin is available but for any reason no carbohydrate food can be got to balance it another problem arises, because the usual dose of insulin without food would cause hypoglycaemia, and the complete omission of insulin would allow ketosis and coma to develop. As a general rule a subject of severe diabetes should take one-half (certainly one-third) of the usual dose of insulin without food. It is unlikely that food would be lacking for long, and as soon as available the usual meal and insulin should be taken, or only a half-dose of insulin if within six hours of the previous half-dose.

PHRENICO-COSTAL SINUS PNEUMONIA

BY

G. R. OSBORN, M.B., B.S.

By "phrenico-costal sinus pneumonia" is meant a peculiar condition of the lateral portion of that part of the base of the lung which enters the phrenico-costal sinus of the pleura on deep inspiration. It will be remembered that during expiration the base of the lung reaches only the eighth rib in the mid-axillary line. During deep inspiration the base moves freely downwards but does not reach the tip of the phrenico-costal sinus. Many muscular efforts are made with the breath held in full inspiration—that is, with this part of the lung fixed in the phrenico-costal sinus. The portion of the lung thus fixed may be

Diagram showing part of lung affected and the mechanism of production.

"caught in a pincers movement" consisting, on the right side, of the elbow and liver and, on the left, of the elbow and the spleen (see accompanying diagram, in which these forces are indicated by arrows and the part of the lung involved is shaded black).

Although this condition does not appear to have been reported previously it is probable that it is not infrequent. In the first two cases here recorded healthy men sustained a relatively trivial but peculiar injury. Their accidents were similar and were well described, and there can be no reasonable doubt that the accident was the cause of the lung condition found later. The second two cases show the same lesion in delirious patients, in whom it is probable that a similar injury took place but was not observed. No great degree of violence is needed to produce the condition. The fifth case is an air raid fatality.

Case 1.—This patient was a well-developed healthy man aged 49, weighing about 12 stone. While having his lunch at work on June 23, 1937, some boys kicked a football near him. He got up and gave it a kick to send it back, but in so doing he tripped over a heap of iron castings and came down on his right side with his elbow in by the side of his chest. The shop ambulance man dressed the elbow and sent him to the hospital, where two sutures were inserted just above the elbow. The patient then resumed work. On the 25th the ambulance man noticed he was sitting down, so he went to him "and found he was in a semiconscious condition." He was taken to the first-aid room, where he "started to groan and complain of pain in the right shoulder and down the right side." He then coughed up some blood and was again taken to the hospital, where he died from acute pulmonary oedema six hours after admission. At the post-mortem examination the injuries above the right elbow laterally were noted; there were multiple abrasions, and a deep wound was closed with two silk-worm-gut sutures. There was no sign of infection, and the axillary glands were not enlarged. The heart weighed 12 oz. Except for slight dilatation it was perfectly normal; there was no valvular or coronary disease. The blood was dark and fluid. The air passages were filled with deeply blood-stained froth; a similar fluid poured from the cut surface of the lung. The lateral part of the base of the right lung (in a position corresponding with the arm injury) showed a peculiar wedge-shaped consolidation. This was very much harder than the hepatization of pneumonia, and was cyanotic, with a dry cut surface; the edges were very sharp all around and stood up from the rest of the lung. Sections revealed that there had been free haemorrhage into this part of the lung. Some phagocytic cells laden with haemosiderin granules were observed among the red cells, but most of the erythrocytes were well preserved. There was no relative increase in white cells, but some of the bronchi had many polymorphonuclear leucocytes in their lumen. The pleura over this solid part showed a light fibrinous exudate; elsewhere it was shiny. There was a large blood-stained exudate in the right pleural cavity, but the left side was clear. The phrenico-costal sinus was normally deep on the right; on the left it was obliterated by old adhesions. There was no significant lesion in the abdomen or the brain.

Case 2.—The second instance of this condition was that of a man aged 55 who on May 24, 1939, slipped at work while carrying a heavy object with another workman. He came down on his left side and "strained" his lower left chest; because of this he had to sit down for a time. He then resumed work and carried on for several days, despite his "strain." He then had to visit his doctor, who strapped his lower left chest and sent him to bed. He appeared to be making good progress until, quite unexpectedly, he suddenly collapsed and died on the seventh day after the accident. At the post-mortem examination it was noted that the left leg was moderately swollen because of a femoral thrombophlebitis. Clot from this had become detached and had caused his death by pulmonary embolism. The clot embolus was 20 cm. long and passed from the right ventricle into the right branch of the pulmonary artery, where it turned sharply across to enter the left pulmonary artery. The heart was moderately dilated and hypertrophied; it weighed 15 oz. There was no valvular disease, but moderate coronary arteriosclerosis was present. Except for the clot described the content of the heart was dark fluid blood. The left pleural cavity revealed an effusion of about one pint of blood-stained fluid; the right had no exudate, but there were some old adhesions. Both lungs showed some oedema. At the lower

part of the base of the left lung laterally there was a hard wedge-shaped lesion similar to that noted in the previous case; it had sharp outlines and felt heavy, and the cut surface was dark red. It was possible to demonstrate the mechanism of the lesion very convincingly (among others to the three doctors who attended to represent the different financial interests of the case) because there were a number of small bruises in the adjacent part of the diaphragm, and the anterior surface and margin of the spleen showed contusions and some blistering. The spleen weighed 7 oz. Nothing of interest was seen in the remaining abdominal organs and in the brain.

In the next two cases showing the lesion the patients were delirious for some days before death and had multiple small surface bruises. While these bruises show that injuries of the type described above may have taken place they were not specially observed, and so these lack some of the interest of the first two cases.

Case 3.—A man aged 62 developed neurasthenia during the war of 1914-18 and became psychotic at the outbreak of the present conflict. Several weeks before he died he was admitted to hospital with a diagnosis of "neuro-circulatory asthenia." During the course of the second week he developed an increasing delirium. On October 23, 1939, I made a post-mortem examination. The heart weighed 16 oz. It was markedly dilated, especially on the left side. The muscle of the left ventricle was wasted and fibrous, and the papillary muscles were completely atrophic. There were a few recent small vegetations on the mitral valve: no emboli from these were demonstrated. The valves otherwise were normal. Moderate coronary arteriosclerosis was present. The upper air passages were clear. The lungs showed a patchy bronchopneumonia, and at the right base laterally there was a hard wedge-shaped zone of consolidation which was heavy and stood out sharply from the surrounding lung tissue. This was of similar size, shape, and consistency to that described previously. Its measurements were: 6 cm. high, 8 cm. in the antero-posterior direction, virtually nothing at the distal tip, increasing to a maximum of 4 cm. at the upper end. The pleural surface over this wedge was shiny. Sections showed that free haemorrhage into the lung had occurred here; there were phagocytic cells filled with haemosiderin among the red cells but no collections of pus and no evidence of infection. There was no blood-stained effusion into the pleural cavity. A small haemorrhage into the neighbouring part of the liver made it clear that this case was similar to the first two described. Apart from gall-stones the remainder of the examination did not reveal anything of significance.

Case 4.—A man aged 63 had had heart trouble for a year and had taken to bed six weeks before admission to hospital. He was sent there on a "fourteen-day order" because of his delirium. On December 12, 1939, I made a post-mortem examination. His heart weighed 23 oz. and showed general hypertrophy and dilatation. The valves were normal except for great dilatation of the aortic ring from a gross aortitis of syphilitic type. There was little coronary disease and little general arteriosclerosis. Both lungs showed a sharply defined wedge-shaped lesion at the extreme base laterally. Except that it was double in this case the appearances were the same as those described in Case 3. There was only a little blood-stained fluid in the two pleural cavities. No contusions were demonstrated on the diaphragm or abdominal organs, but the chest wall was bruised over the phrenico-costal sinuses laterally. Changes in the remaining organs were of little interest.

Case 5.—A well-developed girl aged 17 was brought to hospital dead at about 3.30 a.m. The house in which she lived had been demolished by a high-explosive bomb. She was covered with the dust of the falling house. There was a fracture of the left anterior fossa of the skull. The back of the right hand showed terminal abrasions from protecting herself. There was a blood-stained effusion in the left pleural cavity and a smaller one in the right. The large air passages were filled with blood-stained froth. There were multiple small haemorrhages throughout both lungs; one in the lower part of the right middle lobe was up to 2 cm. in diameter. The most interesting haemorrhage was into the lateral part of the base of the left lung. This had the characteristic wedge shape

described for the previous cases. The antero-posterior extent was 9 cm.; it extended up the lateral surface 3 cm. and over the diaphragmatic surface 6.5 cm. The lesion felt hard, had sharp outlines, and on section was dark red and solid. There were no external injuries to the chest or abdomen to be seen about here, but the abdominal component described was demonstrated by extensive tearing of the spleen with haemorrhage into the peritoneal cavity. There was no other abdominal injury. In this case the father came downstairs ahead of the mother and daughter; he had reached the head of the cellar while the two women were at the foot of the stairs. He was unhurt. The women were buried. The girl was dead when found; the mother was unconscious with a fractured skull and fractured ribs. She died next day.

Discussion

The diagram should make the site of the lesion and its mode of production clear. The movements of the diaphragm are important in the production of phrenico-costal sinus pneumonia. In expiration the lateral muscular fibres of the diaphragm lie against the chest wall and obliterate the sinus, leaving it a potential space only. Inspiration involves active contraction of the diaphragm, which therefore descends and opens the phrenico-costal sinus, the outer parts of the base of the lung being drawn into the sinus. If the breath is held at the end of this movement it is done so by a spasm of the glottis, and the diaphragm and other respiratory muscles are in the position for the beginning of expiration—that is, relaxed. In this condition the phrenico-costal sinus contains part of the base of the lung, but this lung tissue is not protected by contracted muscles. The lung thus held in the phrenico-costal sinus can then be caught between two opposing forces. The external force is the lower part of the arm (elbow) being driven into the chest as the man falls to that side; the internal force is the weight of the liver in a fall to the right and the weight of the spleen (probably aided by other organs in the vicinity) in a fall to the left. No great degree of violence is needed to injure the lung caught between these two forces. Obviously this "pincers movement" can only be applied laterally with any ease; hence the peculiar wedge-shaped lesion described is not likely to be met with anteriorly or posteriorly.

The lesion is probably fairly common, and should be diagnosed during life by radiographs if not by physical signs alone. Doubtless most of the cases recover after running the course expected of a "strain" and the opportunity of seeing the lesion post mortem must be rare. The first two cases described here are the convincing ones, because there was a good history; the death in each instance was by an unexpected complication, so that in the ordinary course of events both men would have recovered. There is no certainly fatal feature in the lesion, and death when it occurs will be due to complications.

The fifth case enables us to arrive at the mechanism of blast injuries to the lungs. To produce the phrenico-costal sinus pneumonia there has been an external force with chest and abdominal components; the latter in this case is proved by the ruptured spleen. This means that the blast effect "is due to the impact of the pressure wave on the chest wall" (Zuckerman, 1940) and, I believe, the abdominal wall as well. Zuckerman arrived at this conclusion by experiments which excluded the effects of pressure and suction waves acting through the air passages, and showed that the blast effect could be minimized or prevented by sponge rubber, etc., around the chest. The study of phrenico-costal sinus pneumonia suggests that blast effects on the lungs would be minimized if people could inhibit the tendency to take a deep breath and hold it in emergencies. This could be tested experimentally. Alternatively, it will be noted that if we accept Zuckerman's results these tend to prove the mechanism described for the production of phrenico-costal sinus pneumonia.

Strictly speaking, the lesion described is not a pneumonia but a contusion; in spite of this the most appropriate name appears to be the anatomical one of "phrenico-costal sinus pneumonia." The lung lesion has such definite features that it can be distinguished from the various types of pneumonia, etc., with ease. These features are: (1) position—lateral part of extreme base; (2) wedge shape; (3) exceptional hardness and heaviness; (4) sharply defined edges which project from the surrounding lung; and (5) dry and dark red cut surface. Other signs of contusion should be looked for. These are: (i) injuries to the elbow and chest wall near by; (ii) a blood-stained pleural effusion with or without flakes of fibrin on the pleura over the lung lesion; (iii) contusions of the diaphragm; and (iv) contusions of the liver on the right side and of the spleen on the left.

The first four post-mortem examinations recorded here were made on the instructions of the Coroner, Major T. H. Bishop, to whom I am indebted for permission to publish the reports. Inquests were held on the first two cases, verdicts of "Death from misadventure" being returned.

REFERENCE

Zuckerman, S. (1940). *Lancet*, 2. 220.

Clinical Memoranda

Obstructive Jaundice due to Stricture at the Junction of Right and Left Hepatic Ducts

Years ago I described a case of simple obstructive jaundice with huge dilatation of the common duct (Burman, 1912, and am now satisfied that it was similar to the cases described by Sir James Walton (1939).

The following case is of interest because post-mortem findings cleared up the pathology—namely, a stricture at the junction of the right and left hepatic ducts, with dilatation of these ducts to the extent of admitting two fingers.

CASE REPORT

A male European aged 46 consulted me on September 13, 1939, and gave the following history. Six weeks previously he had been suddenly seized with acute generalized colicky pains over the whole abdomen that made him vomit. The vomit was greenish and watery. These attacks occurred daily for a week, then ceased, and were followed by jaundice, which gradually deepened. He noticed that his stools were clay-coloured and that his urine was very dark. Flatulence, heartburn, and abdominal distension were troublesome after food, and he complained of cramp-like pains in the calves and tingling of the fingers and toes. Itching was very troublesome, and incessant scratching had produced minute ulcers on both ankles. He was very depressed and lethargic. On examination deep jaundice was evident, and the liver was considerably enlarged. No clinical signs other than those stated in his history were noted. Tentative treatment having been given for a short while without improvement, he was admitted to Greys Hospital for thorough investigation, under my care.

Further questioning now elicited that the patient had a heavy alcoholic history, and for years had had a peculiar feeling of sickness over the xiphisternum after meals. He had contracted malaria during the war of 1914-18, his last attack occurring five years ago. On admission his weight was 131 lb. and his blood pressure 100/70.

The following data were obtained from tests made: (1) Motions: no unsplit neutral fat, a few fatty acid crystals, and no undigested meat fibres. (2) Urinary diastase,

32 units. (3) Wassermann reaction negative. (4) Van den Bergh test: direct prompt positive. (5) Icterus index, 96 on October 13. (6) Blood count: haemoglobin, 80%; R.B.C., 4,200,000 per c.mm. (halometer reading normal); colour index, 0.95; white cells, 5,800 per c.mm.—polymorphs, 61%; lymphocytes, 27%; monocytes, 5%; eosinophils, 6%; basophils, 1%. (7) Radiographs of the gall-bladder region revealed no evidence of opaque stones.

The patient was put on a fat-free high-carbohydrate diet with glucose and insulin from September 22 to October 2 without any result. Medinal was given for relief of itching. Hexamine, 40 grains daily, was tried for a few days.

Owing to the paucity of clinical findings other than the jaundice, itching, and enlarged liver, my provisional diagnosis lay between an ordinary catarrhal jaundice and a growth (papilloma) in the common duct. Against catarrhal jaundice was the enlarged liver and the entire absence of a palpable gall-bladder, and at the end of four months no evidence of diminution of the jaundice was apparent except that occasionally the urine was not so dark. Weight was gradually lost. Before deciding on exploratory laparotomy the patient was shown at a clinical meeting in the hope that some suggestions might accrue from argument. The diagnosis I had now made was a papilloma causing obstruction in the common duct above the junction of the cystic and common ducts. At the meeting, cirrhosis of the liver due to alcohol was mostly favoured, or the possibility of a stone in the common duct.

On November 21 an exploratory laparotomy was undertaken under oxygen-ether anaesthesia. The gall-bladder was found empty and shrivelled; the head of the pancreas was firm but not unduly hard, and the common duct neither enlarged nor distended; palpation revealed no growth or stone. There were a few enlarged glands in the portal fissure; one of these was removed for examination, and on section appeared to be merely inflammatory. (Later, microscopical section showed hyperplasia only.) It was deemed advisable to close the abdomen in view of the negative findings. The patient died during the early hours of the morning following the operation.

Post-mortem Examination.—After considerable difficulty a limited examination was obtained. The liver, pancreas, portion of stomach, and duodenum were removed *en bloc* and the common duct opened from the duodenum upwards to the junction of the right and left hepatic ducts. A stricture, admitting a small probe with difficulty, was encountered. On division of the constriction a large quantity of bile escaped from the greatly dilated ducts. Two fingers could easily be admitted into either duct. Careful examination of the stricture showed thickened tissue only. The spleen was normal. Two handfuls of clotted blood was found in the abdomen, otherwise no abnormality was noticed.

Pathological Report.—Prof. Ryrrie of Capetown reported as follows: "The hard tissue found around the duct is only fibrous tissue. The section of liver appears to me to be very difficult to interpret. There is the obvious evidence of obstructive jaundice; the bile globules are not in the cells about the portal tract, however, but in irregular foci and often about the area where the central vein should be, and in injured cells. All this may well indicate that the obstruction in the liver followed upon a hepatitis, so called, or liver necrosis. Or is the liver necrosis secondary to the jaundice? The clinical story is against this. There is, of course, also some cirrhosis, but it is hardly significant. The section from around the dilated duct again shows much necrosis, but hardly any other lesion to account for the obstruction."

COMMENTARY

The post-mortem findings and a careful survey of the history, especially the sick feeling in the xiphisternal area and, later, constant flatulence and indigestion, suggest the possibility that the stricture was congenital and that the patient's alcoholic proclivities produced a certain amount of cirrhosis, which as time went on caused narrowing of the stricture and, finally, a sudden block with onset of jaundice, similar to the typical history obtained in cases of stone. It