Experiment III: Sodium Gluconate.—Of 28 rats fed on stock diet and subjected to the injection of carbon tetrachloride into the mesenteric vein, half were given 50 mg. of the monosodium salt of glutamic acid eight-hourly by subcutaneous injection for 48 hours after operation.

Experiment IV: Cortisone.—Of 40 rats fed on stock diet and subjected to injection of carbon tetrachloride into the mesenteric vein, 20 were given 2 mg. of cortisone per 100 g. body weight twice a day by intramuscular injection for two days after operation.

Results of Treatment

Treatment	Treated Animals		Controls	
	Surviving	Dead	Surviving	Dead
Vitamin supplements ..	10	7	11	6
Liver extract ..	5	7	4	8
Sodium glutamate ..	4	10	5	9
Cortisone ..	6	14	7	13

Results

In none of these experiments was there any significant difference between the mortality of the treated rats and that of the controls (see Table). Vitamin supplements by mouth, large doses of liver extract parenterally, sodium glutamate, and cortisone all separately failed to improve the chances of survival of rats with experimentally induced hepatic failure.

Discussion

Polyvitamin therapy (Latner, 1950), large doses of liver extract (Snell and Butt, 1941), glutamic acid (Walshe, 1953), and cortisone (Ducci and Katz, 1952) have all been advocated for widely differing reasons in the treatment of hepatic failure. Though these measures may sometimes be followed by symptomatic improvement, claims that they increase the patient's chances of survival should be examined critically. Although there are possible dangers in applying the results of animal experiment to man, no adequately controlled human therapeutic trial has been done, and one is obliged to draw conclusions from human observations which are relevant but inconclusive and from animal experiments which are conclusive but of uncertain relevance.

The clinician faced with a patient suffering from acute liver failure has to deal with a liver which is already severely damaged, and treatment is probably effective only in so far as it will protect the organism against the effects of liver damage until regeneration can occur. Since the metabolic effects of liver injury are so extensive, it is unlikely that any single chemical substance will do this completely, and, as in acute renal failure, the object of treatment must be to maintain chemical homoeostasis until spontaneous recovery occurs. There is a need for more fundamental knowledge of the biochemistry of liver failure in order to plan rational therapy.

Summary

Acute hepatic failure has been produced in rats by the injection of small quantities of carbon tetrachloride into the mesenteric vein.

Vitamin supplements, large doses of liver extract, sodium glutamate, and cortisone all failed to reduce mortality in this type of experimental liver failure.

I am grateful to Miss Shirley Thomas for technical assistance and to Professor L. J. Witts, in whose department this work was done.

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SURGICAL EMPHYSEMA AS A COMPLICATION OF ANAESTHESIA

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Surgical emphysema is that condition in which gas is present in the tissues. This gas may be air or, during anaesthesia, any of the gases used in induction. The first case of surgical emphysema occurring as a complication of anaesthesia was reported by Woolsey in 1912. Thereafter sporadic reports of cases have been published.

Two factors are concerned in the aetiology: (a) the presence of gas under pressure, and (b) some form of trauma permitting entry into the tissues. During anaesthesia, trauma may be produced on any portion of the surface lining of the respiratory tract, including the nose and pharynx, and is usually due to instrumentation (Barrett and Thomas, 1944; Gordon, 1950). Laceration of the mucosa permits gases in the lumen of the respiratory tract to gain access to the submucosal connective tissue and to spread from there.

Gas under pressure in the lumen of the respiratory tract may itself produce the trauma and spread in the connective tissue. Normally the weakest point of the respiratory tract is the alveolar wall, and raised pressure will cause trauma here. In man, a case has been reported in which an intrabronchial pressure of only 12 mm. Hg caused emphysema (Heidrick *et al.*, 1940; Marcotte *et al.*, 1940), and a pressure above 80 mm. Hg will almost always rupture the alveolar walls and their contained vessels, causing air embolism (Haldane and Priestley, 1935). This excess pressure may be exerted by the anaesthetist with his machinery easily enough nowadays, when controlled respiration is so commonplace. Tuffier (1906) showed that a pressure of 6 mm. Hg was enough to maintain distension of the lungs, and 15 mm. Hg has been advocated as the maximum pressure that should be employed in controlled respiration (Thornton *et al.*, 1944). The patient may himself raise the intrabronchial pressure, as by coughing or straining (Hamman, 1945).

The mechanism involved is (Macklin, 1937): The alveolar wall ruptures and gas passes perivascularly along the vessels to the hilum and mediastinum, producing mediastinal emphysema, which becomes the focal point for the further spread of the gas. The gases may (a) spread with the large vessels up to the neck and present as subcutaneous emphysema, (b) spread with the vessels down into the retroperitoneum, or (c) rupture the mediastinal pleura, thus producing pneumothorax. The important point is that pneumothorax does not result directly from rupture of the alveolus.

Clinical signs and symptoms depend on the site and the amount of gas present. Mediastinal emphysema (Hamman, 1945) causes obstruction to the return of blood to the heart and thus lowers the cardiac output, showing clinically as dyspnoea, cyanosis, engorged veins, obliteration of the cardiac dullness, and crackling sounds

on auscultation of the heart. These signs of mediastinal emphysema are generally not marked, the gases usually spreading with comparative ease and presenting as subcutaneous emphysema, with or without pneumothorax. With mediastinal emphysema an associated pneumothorax is present in one-third of the cases.

Case Report

A boy aged 8 years was admitted to hospital for enucleation of an eye. Pre-operative examination revealed a plump child, weight 58 lb. (26 kg.), height 3 ft. 9 in. (114 cm.), nose blocked, pigeon chest, emphysematous vesicular breathing. Premedication consisted of chloral, 8 gr. (0.5 g.), at 9.25 a.m., and atropine, 1/100 gr. (0.65 mg.), at 10.15 a.m.

At 11 a.m. 200 mg. of thiopentone sodium as 2% solution, followed immediately by 20 mg. of succinylcholine chloride ("scoline"), was given intravenously. After the usual fibrillations of the muscles, a size 8 "portex" endotracheal tube was passed easily through the mouth into the trachea under direct vision, using a Macintosh laryngoscope. The endotracheal tube was connected to a Boyle machine, and inflation of the lungs with oxygen was begun pending the return of spontaneous respiration.

As the bag was compressed, the sudden rapid appearance of subcutaneous emphysema of the face and neck was noticed. The Boyle machine was immediately disconnected from the endotracheal tube, the laryngoscope was inserted, and the tube was removed from the larynx and immediately replaced. The patient was still apnoeic as a result of the succinylcholine chloride, so the tube was reconnected to the Boyle machine and very gentle rhythmic compression of the oxygen-filled reservoir bag was restarted. The subcutaneous emphysema had spread superiorly up to the eyebrows and inferiorly on the anterior abdominal wall down into the scrotum. At this time the pulse could not be palpated, but it rapidly returned to full volume in less than two minutes at a rate of about 150 beats a minute. The mucous membranes were pink throughout, except for a period of about one minute following the appearance of the subcutaneous emphysema, when they were cyanosed. Spontaneous respiration began again in about five minutes.

In view of the patient's condition the operation was cancelled and he was returned to the ward with the endotracheal tube *in situ*. At about 11.15 a.m. he started to object to the presence of the endotracheal tube, but was restrained from removing it, and amethocaine 2% solution was sprayed down and round the tube. Portable x-ray examination of the chest revealed a bilateral pneumothorax, retroperitoneal gas, and subcutaneous emphysema. Bilateral puncture of both pleural cavities was performed with needles attached to tubes leading under water. Gas bubbled out rapidly and x-ray examination showed re-expansion of both lungs. The patient was then sedated with paraldehyde, and three and a half hours after the onset of the surgical emphysema the endotracheal tube was removed. Penicillin, 500,000 units, and streptomycin, 0.5 g., were given twice daily as a prophylactic against the possible development of mediastinitis, and the patient made an uneventful recovery, the subcutaneous emphysema being almost entirely reabsorbed by the following day.

Comment

The cause in this case appears to be the institution of positive pressure, leading to the sequence of events described by Macklin: rupture of the alveoli and perivascular tracking of the oxygen to the mediastinum, with extension from there to the neck to present as subcutaneous emphysema, and rupture of the mediastinal pleura to give bilateral pneumothorax with collapse of the lungs.

Trauma to the tracheal mucous membrane, as has been described by others (Barrett and Thomas, 1944; Gordon, 1950), seems to be unlikely, in view of the ease with which the endotracheal tube was passed.

Summary

The causes, mode of production, and treatment of surgical emphysema as a complication of anaesthesia are discussed, and a new case is reported.

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Medical Memoranda

Winged Scapula Caused by a Solitary Exostosis

Solitary exostoses of the scapula of sufficient size to cause symptoms occur very rarely. Du Mortier and Halpert (1934), who reported two cases, could find only 18 further cases in the literature covering the previous 100 years; Mouchet, who showed one case to the Société d'Anatomie de Paris in December, 1927, had personal knowledge of two further cases (quoted by Pouzet and Labry, 1929). Five other isolated cases have since been reported (Pouzet and Labry, 1929; Hartmann, 1930; Lunardi, 1934; Taylor and Schaffer, 1937; Leffmann, 1947). In a survey of the causes of "snapping scapula" collected by Milch and Burman (1933), subscapular exostosis was mentioned, these authors citing the case described by McWilliams (1914), which is included in the series of Du Mortier and Halpert.

In 15 of the cases in the series of Du Mortier and Halpert the exostosis grew from the subscapular surface, thus causing winging of the scapula or interference with shoulder movement. Of the remaining five cases, four were inadequately described by the original writers, and in one instance (Heurtaux, 1886) the exostosis arose from the spine of the scapula. In the five further cases now collected from the literature, the situation of the exostosis was at the medial angle or the vertebral border of the scapula, except in one, in which it was attached to the axillary border (Taylor and Schaffer, 1937). There appears to be no sexual predominance, 14 being male and 12 being female.

In all but four cases symptoms occurred before the age of 20, and in the remainder it is likely that the condition had existed for some years before advice was sought. Because of its rarity it was thought worth while reporting a case of subscapular exostosis with some unusual features.

CASE REPORT

A bus driver aged 23 attended the out-patient clinic complaining of aching pain in the left shoulder and limitation of shoulder movement.

On examination an extensive deep-brown pigmented naevus was found to be situated over the left shoulder and a pronounced winged scapula which could not be passively corrected. The deformity had apparently been present for about 10 years, recently becoming more noticeable. The arm could be abducted to 160 degrees, this being accompanied by a creaking sensation over the scapula. Palpation deep to the vertebral border of the scapula revealed a large bony mass. X-ray examination showed this to be an exostosis growing from the subscapular fossa and impinging on the uppermost three ribs, which bore slight indentations at the points of contact. At operation the exostosis was approached by a longitudinal incision over the vertebral border of the scapula. The exostosis was easily excised and was found to be pear-shaped, 3½ in. (9 cm.) in length