Macintosh (1949) has employed a gum-elastic catheter in difficult intubations. This is pushed 2 in. (5 cm.) beyond the end of the tube, which is threaded over it and guided by it into the larynx. Barnard (1952) has used a malleable bougie in the same manner.

Medical literature is full of complaints when old facts are rediscovered. No original claim is made regarding the above application of a curved introducer (Chaussier, 1780-1; O'Dwyer, 1885).

Summary and Conclusion

Five cases in which direct laryngoscopy and subsequent intubation were difficult are reported. The causes of the difficulty are described and the value of a curved introducer is reaffirmed.

As no report has hitherto appeared in the literature concerning this type of case and its exact abnormalities, these few examples encountered during three years' anaesthetic practice at a busy general hospital may serve to draw attention to the causes of this relatively rare but embarrassing problem for patient and anaesthetist alike.

We are grateful for the interest and ready co-operation of Professor A. Amies and Professor H. Atkinson, both of the Melbourne University Dental School, who made available detailed reports on the abnormalities shown in the x-ray films.

REFERENCES

- Adriani, J. (1947). *Techniques and Procedures of Anaesthesia*, p. 117. Thomas, Illinois.
Ballantine, R. I. W., and Jackson, I. (1954). *Anaesthesia*, 9, 9.
Bannister, F. B., and Macbeth, R. G. (1944). *Lancet*, 2, 651.
Barnard, J. (1952). *Anaesthesia*, 7, 119.
Caine, C. W. (1948). *Anesthesiology*, 9, 553.
Chaussier (1780-1). *Hist. Soc. roy. Méd.*, 4, 346.
Deutsch, E. V. (1951). *Anesthesiology*, 12, 667.
Gillespie, N. A. (1948). *Endotracheal Anesthesia*. Madison, Wisconsin.
Lee, J. A. (1953). *Synopsis of Anaesthesia*, 3rd ed., p. 173. Wright, Bristol.
Macintosh, R. R. (1943). *Lancet*, 1, 205.
— (1949). *British Medical Journal*, 1, 28.
— and Bannister, F. B. (1952). *Essentials of General Anaesthesia*, 5th ed., p. 257. Blackwell, Oxford.
O'Dwyer, J. (1885). *N.Y. med. J.*, 42, 145.
Rowbotham, S. (1920). *British Medical Journal*, 2, 590.

SURGERY IN ADDISON'S DISEASE

BY

H. R. C. RICHES, M.B., M.R.C.P.

Senior Medical Registrar, Harefield Hospital, Harefield, Middlesex

Before the introduction of deoxycortone acetate (D.C.A.) in 1938 surgery was so hazardous in patients with Addison's disease that it was seldom attempted. A few survivals after surgery were reported prior to 1938, however (Rowntree *et al.*, 1931; Green *et al.*, 1933), but they were infrequent and usually followed minor procedures. Fortunately, modern substitution therapy with D.C.A. and cortisone so improves patients with adrenal insufficiency that the risks of surgery are no longer prohibitive and major operations can be performed with confidence, although careful management is still essential.

The following case of Addison's disease, with active pulmonary tuberculosis, is reported in detail because it is believed to be one of the earliest in which major thoracic surgery has been successfully completed under cortisone and D.C.A. cover and because it also clearly illustrates the management of a patient during two major operations.

Case Report

The patient, a male clerk aged 38, became ill in 1950 with progressive asthenia, anorexia, pigmentation, decreasing weight, loss of libido, and periodic attacks of diarrhoea and

vomiting. After six months of slow deterioration in health he became desperately ill and was admitted to hospital as an emergency in Addisonian crisis. On admission he was restless, confused, dehydrated, and pigmented, and exhibited rapidly progressive ascending flaccid paralysis which had begun to affect the respiratory and pharyngeal muscles. Other clinical abnormalities were buccal pigmentation, deviation of the trachea to the right, flattening of the anterior chest wall of the right hemithorax with diminished breath sounds in the upper zone, and a marked hypotension of 76/60 mm. Hg.

Investigation showed the urine to be normal; 24-hour urinary ketosteroids, 4.1 mg.; haemoglobin, 15.4 g./100 ml.; P.C.V., 44%; E.S.R. (Westergren) 3 mm./hour; serum sodium, 122 mEq/l.; serum potassium, 10.5 mEq/l.; serum chlorides, 84 mEq/l.; blood urea, 133 mg./100 ml.; alkali reserve, 17.3 mEq/l.; blood sugar, 65 mg./100 ml.; Wassermann and Kahn reactions, negative; cerebrospinal fluid, normal. An electrocardiogram showed changes consistent with the high serum potassium, and x-ray examination of the chest revealed a fibrotic lesion at the right apex with pleural calcification at the base. Sputum cultures were negative for tubercle bacilli. There was no radiological evidence of suprarenal calcification.

Intensive treatment with intravenous saline, "eucortone" (total 90 ml.), and D.C.A. produced dramatically rapid improvement, and after six weeks the patient was discharged home to continue with D.C.A., 5 mg., and salt, 10 g., daily. By this time the blood biochemistry had returned to normal and the blood pressure was 115/80 mm. Hg.

After a short convalescence he returned to clerical work, but throughout the next two years his health was only moderate and he was finally readmitted to hospital in November, 1953, complaining of persistent lethargy and failure to gain weight. The blood biochemistry proved normal except for a fasting blood sugar which was persistently low, varying between 65 and 73 mg./100 ml., and the blood pressure was 110/65 mm. Hg. In view of these findings, and as the chest x-ray film was unchanged, cortisone, 25 mg. daily, was added to the regimen of treatment, and this brought immediate subjective improvement. After only three months of good health, however, he relapsed again, and on readmission to hospital there was clinical and radiological evidence of extensive cavitation in the upper lobe of the right lung and many tubercle bacilli were found in the sputum.

Antibacterial treatment with streptomycin and *para*-aminosalicylic acid (P.A.S.) was started at once; the D.C.A. and salt were continued as before, but the cortisone was reduced to 12.5 mg. daily. When he was subsequently transferred to Harefield Hospital on July 1, 1954, having completed three months' chemotherapy, the sputum was negative for tubercle bacilli but cavitation was still present in the right upper lobe. Bed rest and chemotherapy were therefore continued (streptomycin, 1 g. thrice weekly, P.A.S., 15 g., and isoniazid, 400 mg. daily) and the Addison's disease was controlled with cortisone, 25 mg., and D.C.A., 5 mg., daily. After a further ten months' treatment, cavitation persisted in the right upper lobe and surgery was therefore advised, a right seven-rib thoracoplasty being completed uneventfully in two stages (T. Holmes Sellors) under local analgesia (A. I. Parry Brown). Anaesthesia was entirely satisfactory with "omnopon," 1/3 gr. (22 mg.), and scopolamine, 1/150 gr. (0.43 mg.) as premedication, chlorpromazine, 25 mg. intravenously, brachial plexus and paravertebral blocks with 0.4% lignocaine hydrochloride and infiltration of the operation site with 0.2% lignocaine hydrochloride. Throughout both operations 4.3% glucose with 0.18% NaCl was given by intravenous drip.

There were no post-operative complications, and twelve weeks after completion of the thoracoplasty D.C.A. was implanted subcutaneously and the daily injections were discontinued. Cortisone, 25 mg. daily, and long-term chemotherapy with isoniazid and calcium 4-benzamidosalicylate ("therapas") were being continued with entirely satisfactory results. Therapas was prescribed because, unlike some of

the commonly used forms of P.A.S., it does not contain sodium. This was regarded as an advantage because sometimes in the early period after the implantation of D.C.A. there is sodium retention with consequent hypertension and oedema. It therefore appeared more satisfactory to use a sodium-free preparation of P.A.S. and later to supplement the diet with salt if required.

All the relevant details of management throughout the critical period of the operations are shown in the diagram.

Details of management during the period of the operations. In addition to cortisone therapy, the following were given daily: D.C.A., 5 mg.; streptomycin, 1 g.; isoniazid, 400 mg. Penicillin, 0.5 mega unit twice daily, was also given for ten days after each operation.

Discussion

Addison's disease is the sequela of destruction and hypofunction of the cortex of the adrenal glands. Most cases are caused by tuberculosis and idiopathic atrophy, but rare causes are amyloidosis, neoplasms, vascular lesions, pyogenic infections, syphilis, and trauma (Hartman and Brownell, 1949).

Guttman (1930), in a review of the literature, found that although 60% of cases had a tuberculous aetiology, Addison's disease was rare in sanatorium and chest hospital practice. In many large series of necropsies on tuberculous patients which he quotes, disease of the adrenals was reported in from 2 to 4.8%, but in the majority of these it was probably insufficient to cause recognizable impairment of adrenal function. If allowance is made for the prevalence of very advanced disease at that time, this is a low incidence and confirms the clinical impression that Addison's disease and active pulmonary tuberculosis are not commonly associated (Benians, 1952).

It has been suggested that to-day more cases have a non-tuberculous aetiology: Thorn *et al.* (1942), in a series of 158 cases, found only 40% definitely tuberculous. Perhaps this change can be related to the decreasing incidence of tuberculosis as a disease and the more accurate interpretation of adrenal pathology.

Many patients can be kept in good health with D.C.A. and salt, but others show profound disturbance of carbohydrate metabolism and tend to become hypoglycaemic. This tendency, which persists in spite of adequate treatment with salt and D.C.A., can be relieved quite readily with extracts

of adrenal cortex and cortisone (Thorn *et al.*, 1950). In clinical practice it has been found that 15 to 30 mg. of cortisone daily, in addition to salt and D.C.A., greatly improves many patients with severe Addison's disease by preventing hypoglycaemia and returning the electroencephalogram to normal (Thorn *et al.*, 1950).

The wisdom of prescribing cortisone to patients with any disease which has, or may have, a tuberculous aetiology is sometimes questioned, since it is well known that this hormone has a deleterious effect on tuberculosis in experimental animals (Michael *et al.*, 1950; Spain and Molomut, 1950). There are also in the literature many reports of latent tuberculosis being activated in patients having cortisone treatment for other unrelated diseases (King *et al.*, 1951; Popp *et al.*, 1951; Fred *et al.*, 1951). However, it seems possible that cortisone given in physiological doses as substitution therapy for adrenal insufficiency may be without serious risk (Browne *et al.*, 1954; Fourman and Horler, 1954), although our patient developed florid pulmonary tuberculosis whilst on a maintenance dose of 25 mg. daily, which is within the estimated human physiological range.

Cortisone therapy presents no difficulties in tuberculous patients receiving chemotherapy, for it has already been shown by Houghton (1954) that under cover of adequate chemotherapy corticotrophin can be safely given to patients with active pulmonary tuberculosis, provided the bacilli are sensitive to the antibiotics being used. The problem which will arise with our patient, however, will be in deciding when to stop the chemotherapy, since his present daily requirement of cortisone (25 mg.) is the same as that associated with the original deterioration of his chest disease. Because of this difficulty it may perhaps be suggested that resection of the disease would have been more logical than thoracoplasty. This approach was rejected because pneumonectomy would have been required, and this was not thought justified. Moreover, Browne *et al.* (1954) have treated five patients with Addison's disease and pulmonary tuberculosis by long-term chemotherapy with good results. In their cases cortisone was continued after stopping the antibacterial drugs and all maintained good health: the follow-up, however, was short—varying between one and three and a half years. In the hope of preventing further relapse of our patient it is intended to continue chemotherapy for some years.

Surgery caused no untoward disturbance to our patient, the only abnormalities noted being slight hypotension during and immediately following both operations and moderate hyperglycaemia after the second operation. The hypotension was attributed to the analgesic, and the hyperglycaemia was undoubtedly an effect of cortisone, since it disappeared as the dose of the drug was reduced. Wound-healing was entirely satisfactory both clinically and on histological examination of the excised scar. Although cortisone and corticotrophin are reputed to depress wound-healing in experimental animals (Ragan *et al.*, 1949), it is now believed that healing is normal in patients if the dose of these drugs is kept within the accepted therapeutic limits (Thorn *et al.*, 1950).

Summary

A case of Addison's disease with active pulmonary tuberculosis is described. After thirteen months' chemotherapy in hospital a two-stage thoracoplasty was successfully completed under cover of cortisone and D.C.A. The aetiology of Addison's disease, its association with pulmonary tuberculosis, and its treatment are briefly discussed.

It is concluded that there is no contraindication to major chest surgery in a tuberculous patient with Addison's disease.

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REFERENCES

- Benians, R. G. (1952). *Brit. J. Tuberc.*, 46, 66.
 Browne, J. S. L., Aronovitch, M., Beck, J. C., Leith, W., and Meakins, J. F. (1954). *Amer. J. med. Sci.*, 228, 491.
 Fourman, P., and Horler, A. R. (1954). *Lancet*, 1, 334.
 Fred, L., Levin, M. H., Rivo, J. B., and Barrett, T. F. (1951). *J. Amer. med. Ass.*, 147, 242.
 Greene, C. H., Walters, W., and Rowntree, L. G. (1933). *Ann. Surg.*, 98, 1013.
 Guttman, P. H. (1930). *Arch. Path. (Chicago)*, 10, 742, 895.
 Hartman, F. A., and Brownell, K. A. (1949). *The Adrenal Gland*, p. 344. Kimpton, London.
 Houghton, L. E. (1954). *Lancet*, 1, 595.
 King, E. Q., Johnson, J. B., Batten, G. S., and Henry W. L. (1951). *J. Amer. med. Ass.*, 147, 238.
 Michael, M., Cummings, M. M., and Bloom, W. L. (1950). *Proc. Soc. exp. Biol. (N.Y.)*, 75, 613.
 Popp, C. G., Ottosen, P., and Brasher, C. A. (1951). *J. Amer. med. Ass.*, 147, 241.
 Ragan, C., Howes, E. L., Plotz, C. M., Meyer, K., and Blunt, J. W. (1949). *Proc. Soc. exp. Biol. (N.Y.)*, 72, 718.
 Rowntree, L. G., Greene, C. H., Ball, R. G., Swingle, W. W., and Pfiffner, J. J. (1931). *J. Amer. med. Ass.*, 97, 1446.
 Spain, D. M., and Molomut, N. (1950). *Amer. Rev. Tuberc.*, 62, 337.
 Thorn, G. W., Dorrance, S. S., and Day, E. (1942). *Ann. intern. Med.*, 16, 1053.
 — Forsham, P. H., Frawley, T. F., Hill, S. R., Roche, M., Staehelin, D., and Wilson, D. L. (1950). *New Engl. J. Med.*, 242, 783.

CONGENITAL ABSENCE OF LEFT PULMONARY ARTERY

BY

JOSEPH SMART, M.D., F.R.C.P.

Physician, London Chest Hospital

AND

J. N. PATTINSON, M.B., F.F.R., D.M.R.D.

Radiologist, London Chest Hospital

The congenital absence of a pulmonary artery is a rare condition, but one which has been recognized for many years. The first case was reported by Fraentzel (1868), this being discovered at necropsy, and, later, Döring (1914), Müller (1927), Ambrus (1936), Miller (1937), and Thomas (1941) all recorded cases of absent pulmonary artery found after death.

Subsequently, further cases have been reported, largely occurring in cyanotic heart disease, the absent pulmonary artery being found at operation. Blalock (1947, 1948) refers to nine such patients occurring in a series of 610 cases of cyanotic heart disease. Findlay and Maier (1951) and Jew and Gross (1952) reported further examples of this condition found at operation.

After the introduction of angiocardiology the diagnosis of this condition became possible without operation, and from 1952 onwards a number of cases of an absent pulmonary artery were described. Madoff *et al.* (1952), Steinberg *et al.* (1953), Madoff (1954), Wyman (1954), and Flynn *et al.* (1954) all described cases of an absent pulmonary artery in patients with a normal heart, diagnosed by angiocardiology. During this same period Nadas *et al.* (1953), McKim and Wiglesworth (1954), Wyman (1954), Campbell (1954), and Maier (1954) reported cases of an absent pulmonary artery associated with congenital heart disease, and some of the cyanotic type. Finally, Read (1954) reported a case during the discussion on Maier's paper in which there was an absent pulmonary artery associated with hypoplasia of the lung; and Waterman (1954), also during the discussion of Maier's paper, reported two cases of very small right pulmonary arteries.

It would appear from the literature that the absence of a pulmonary artery may occur on either side, but is slightly more common on the left than on the right, and

is often associated with congenital heart disease, particularly of the cyanotic type. In those recorded cases where there is no evidence of heart disease, the absence of the right pulmonary artery appears to be more frequent than the absence of the left. The defect occurs more commonly in females than in males. This variation of an absent left pulmonary artery with congenital heart disease and an absent right pulmonary artery with a normal heart has been noted by Emanuel and Pattinson (1956).

The following case is reported in view of the rarity of the condition and its radiological interest.

FIG. 1.—Normal left bronchogram.

FIG. 2.—Normal right bronchogram.