there is an excessive excretion of sodium so that the urine has a greater osmotic pressure than the plasma. Unlike the other two varieties, the patients do not suffer from dehydration, hypotension, or uraemia, and there is no clinical evidence of oedema. The condition has been attributed to inappropriate secretion of antidiuretic hormone (A.D.H.),[1] because it resembles the state produced experimentally by administration of vasopressin to persons who have drunk large quantities of water, and who would normally pass large volumes of dilute urine.[2] The low sodium level in the plasma is believed to be due to retention of water with resultant dilution of plasma solutes and expansion of the extracellular fluid: this leads to increased excretion of sodium and further decrease in the osmotic pressure of the plasma. Secretion of aldosterone is normally inhibited in these circumstances, but occasionally increased secretion may compensate for the sodium loss, though clearance of free water remains diminished and provides a reliable guide to the presence of the syndrome.[3] No explanation has yet been found for the continued secretion of A.D.H. in spite of a hypotonic plasma.

Of conditions associated with inappropriate secretion of A.D.H. the two which have received most attention are bronchial carcinoma[1 4 5] and intracranial disease.[6 7] The syndrome has also been described in myxoedema,[8] porphyria,[9] and advanced tuberculosis,[10] and in one patient episodes occurred over a period of five years without any clinical evidence of underlying disease.[11] In nearly all the cases of bronchial carcinoma the growths were of the anaplastic oat-celled variety, so it seems unlikely that the tumour exerts its effect by mechanical interference with the volume receptors thought to be present in the mediastinum. The tumour may produce an A.D.H.-like compound or some substance which stimulates A.D.H. production. If this were the explanation, however, it would be expected that carcinomata in other sites would have the same effect, as with some of the other carcinomatous syndromes. Cerebral metastases are not the cause, although "dilutional hyponatraemia" and "cerebral salt wasting" have been described in cerebral trauma, encephalitis, pituitary tumours, and cerebrovascular disease.[6 7] Here the mechanisms may more readily be explained by an irritant action on the hypothalamus and the posterior pituitary.

It is uncertain whether the hyponatraemia is responsible for any specific symptoms in this syndrome, though mental confusion and irritability have been attributed to it, and a general improvement in well-being may follow appropriate treatment.[5] Some patients develop signs of water intoxication, with headaches and convulsions. Skin pigmentation has also been noted, and it is important to exclude the possibility of adrenocortical insufficiency or disorders of renal tubular function. Improvement may follow treatment of the underlying condition, but in the case of bronchial carcinoma this is often only partially successful. It is sometimes possible to inhibit A.D.H. secretion by nicotine or alcohol, but this is unpleasant for the patient. Restriction of water intake may help, but administration of 9α-fluorohydrocortisone is more effective and constitutes less of a hardship.[5] The use of A.C.T.H. and corticosteroids has been generally unsatisfactory, but French workers[3] have recently found that a slow-release preparation of oxytocin together with small doses of A.C.T.H. restored free water clearance to normal, especially in patients with cerebral lesions.

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Aberdeen Typhoid Outbreak of 1964

A factual record of the clinical, epidemiological, and public-health aspects of the Aberdeen typhoid outbreak of 1964[1] is of special interest, for the outbreak had several notable features. There were 507 cases, derived from 309 households in the city and 33 in the surrounding districts, with three deaths. All but four patients were treated in hospital; the diagnosis was confirmed bacteriologically in 403 and clinically in 66; in the remaining 38 typhoid was not confirmed. Problems of the carrier state were discussed in these columns last week[2] with reference to a report on the Aberdeen outbreak by Elizabeth M. Russell and her colleagues,[3] while J. C. M. Sharp[4] described the psychological disturbances that may result from it.

The outbreak appeared as one single wave and there were no proved secondary cases. The first patient, who became ill on 12 May 1964, was admitted to hospital on 16 May. A sample of blood submitted for bacteriological examination was reported positive for *Salmonella typhi* on 20 May, and four members from that family were immediately admitted to hospital. Since 38 of 41 patients admitted up to 23 May gave a history of having eaten cold meat purchased from the supermarket, and as three-quarters of them remembered eating sliced corned beef, suspicion centred on this article of food as the probable source. Strong support for this hypothesis was forthcoming when the Public Health Laboratory at Colindale reported that the organism from the first isolations was phage type 34, a variety common in South America and Spain, but virtually unknown in Great Britain. Up to 26 May the outbreak seemed to be confined to persons who had eaten corned beef from a single large tin which had been sold in slices between 6 and 9 May. Thereafter the picture changed and more and more cases appeared with a history of having eaten other cold meats from the supermarket. It then became apparent that the slicing machine used for the corned beef had infected other cold meats on 8 and 9 May and that the organisms had multiplied when the lightly contaminated meats had been stored in an uncooled display window exposed to sunlight. The Health and Welfare Department took action to cleanse the supermarket between 20 and 23 May and this coincided with complete cessation of spread from that source. Intensive investigation failed to reveal a carrier among the staff nor had any member ever visited an area in which phage type 34 had been found. The "all clear" was given on 17 June, 28 days after the first notification of suspects. In all, 391 patients had given a history of eating food from the infected shop, 373 having eaten cold meats and 18 fruit.

This epidemic of typhoid, the largest in Britain since 1937, contains many lessons of importance. Epidemiologically, the

Departmental Committee of Inquiry were in no doubt that the source of the infection was a can of corned beef from the canning establishment at Rosario in the Argentine. Despite the fact that the contents of the can were consumed 12 days before the first notification of the disease and the can itself destroyed, the circumstantial evidence incriminating the corned beef was very strong. Additionally disturbing is the fact that in three outbreaks of typhoid at Harlow, Bedford, and South Shields in 1963 the only common factor was again corned beef. In these outbreaks the association was with one particular brand of corned beef produced at a packing establishment in the Argentine at which the water used for cooling the cans of meat after sterilization was unchlorinated river water. Furthermore, an outbreak of typhoid at Pickering in 1954–5 was traced to tongue or ham of South American origin. At that time no canning establishment used chlorinated water for cooling purposes, and it was subsequently learned that the river water used at this particular plant was contaminated with typhoid bacilli of the same phage type E1 which had caused the outbreak at Pickering. Still more damning evidence pointing to contaminated corned beef from South America as a source of infection has been produced by E. S. Anderson, who has re-examined the evidence in connexion with the outbreak of typhoid at an orthopaedic hospital in Oswestry in 1948. He found that the infecting organism, which had fortunately been kept alive since 1948, was of phage type 34, the same type which was responsible for the Aberdeen outbreak. In eight other outbreaks of typhoid between 1929 and 1949 canned beef figured in the case histories.

The Departmental Committee of Inquiry[5] into the outbreak showed that the canning industry's reliance on "blowing" of the can (by gas-producing organisms accompanying the typhoid bacillus) as an indication of contamination was ill-placed. They concluded that such cans may reach retail shops and customers without any warning signs to show that they are contaminated. Nevertheless, the committee believed that canned meat produced under wholly suitable and satisfactory conditions is among the safest foods available and provides a relatively inexpensive and valuable source of first-class protein. It is reasonable to suppose that, by exercising our right of inspection to check that the arrangements at all canneries and meat plants in Argentine, Paraguay, and Uruguay are satisfactory, we should be able to ensure that no further outbreaks of typhoid from this source occur.

While variability in clinical presentation is a well known feature of the disease this epidemic was notable in that the incubation period of the early cases was very short and the clinical pattern more consistent with acute gastro-enteritis with high fever, headache, diarrhoea, vomiting, and sometimes epistaxis and neck stiffness. The later cases, on the other hand, presented with an influenza-like illness with headache, vomiting, and sore throat, but no diarrhoea. The classical picture with rose spots and splenomegaly was seldom seen. The marked contrast between early and later cases could be accounted for by the much heavier infection sustained by those people who had eaten the corned beef and the relatively light infection acquired from the cold meats contaminated by the slicing-machine. An infection in the form of a brisk attack of diarrhoea and vomiting was reported in certain cases; this is a feature of the disease which has sometimes been noted in recent years.

Of outstanding importance was the emergence of the value of "clot" culture—that is, of the clot in the bottles submitted for Widal examination for diagnostic purposes. The finding that previous T.A.B. immunization renders the Widal valueless as a diagnostic criterion is in accordance with general experience and emphasizes the advisability of obtaining a positive culture from blood or faeces as proof of infection. Although it was impossible to make a direct comparison between the antibiotics used in treatment, there was nothing to suggest that ampicillin can rival chloramphenicol in efficacy. It was also notable that all but 18 of the adults received chloramphenicol alone and no case of marrow aplasia was found. There was clearly reluctance to use the drug in the dosage now recommended—at least 50 mg. per kg. daily for the first week or longer—or to continue it for longer than 14 days. There was also reluctance to add corticosteroids in cases showing no response to chloramphenicol, a procedure which has produced satisfactory, and often dramatic, results.

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[4] Sharp, J. C. M., ibid., 1966, **2**, 551.
[5] *The Aberdeen Typhoid Outbreak, 1964*, Cmnd. 2542, 1964. H.M.S.O

Roots of Plastic Surgery

In a timely letter this week (page 641) Mr. Patrick Clarkson draws attention to the unique contributions to surgery, both during and after the two world wars, made by Sir Harold Delf Gillies (1882–1960). His name was well known to the British troops of 1914-18 as was that of his cousin, pupil, and fellow New Zealander, Sir Archibald McIndoe (1900-1960), to the burnt airmen of the 1939–45 war. Before Gillies there had been no specialist plastic surgeons, though many plastic operations had been performed, and their subsequent condensation into one specialty was anticipated by the German bibliographer and surgeon Eduard Zeis, who in 1838 coined the term "plastic surgery."

Gillies made his first tube pedicle at Sidcup in October 1917 for a sailor who had been burnt at the Battle of Jutland. Meanwhile—though it was unknown to him until 1919—Vladimir Filatov had already raised and tubed a pedicle to repair a cancerous lower eyelid in Odessa in September 1916. But the further development and wide recognition of the value of the operation was due to Gillies. At the same time a major stimulus to the development of endotracheal anaesthesia by Sir Ivan Magill was his desire to satisfy the anaesthetic requirements of Gillies and his colleagues.

Raising a tube pedicle entails "surgical delay," thereby increasing local blood supply, a mechanism demonstrated first by Frank H. Hamilton, of Buffalo, New York State, in 1854, when he deliberately postponed the attachment of the first cross-leg flap ever to be raised, and transferred successfully fourteen days later. Another American, John P. Mettauer, of Virginia, was the first anywhere to operate successfully for hypospadias (in 1830) and the first in America to repair a cleft palate (in 1827).

Frenchmen first mended congenital cleft both of the lip—namely, Pierre Franco in 1556—and of the palate—Philibert J. Roux in 1819. In Paris Victor Veau (1871-1949) devoted his surgical lifetime to the study and repair of these clefts and was the first to use a speech therapist in such cases, Madame

[1] Wolfe, J. R., *Brit. med. J.*, 1875, **2**, 360.