Medical Memoranda

A Clinical Sign of Vagotonia

One of the most difficult decisions a physician has to make is in the small hours of the morning at the bedside of an elderly patient who is in or who has just recovered from an attack of orthopnoea. There is little clinical evidence of disease to help; indeed, extensive clinical examination is too distressing to be employed, and one must be content with a rapid and superficial search for abnormality. Frequently in such a case the chest is emphysematous, there are no accessory breath sounds, and expiration is prolonged and the heart sounds are distant. Temporarily the heart muscle is indicted, and the case is labelled one of cardiac dyspnoea, with all its attendant fears, until a more detailed examination is possible.

Some 10 years ago I noticed in many of these cases the following physical sign, which I have found most helpful in making an immediate diagnosis. When present it is, I claim, an unmistakable sign of vagotonia, producing the symptoms of a paroxysm of bronchial asthma in its initial, waxing, or waning stages. It is often the only indicator of the true cause of the breathlessness complained of by middle-aged or elderly patients. Listening close to the patient's mouth, one hears, without the use of the stethoscope, a fine effervescing bubbling sound during expiration, and in marked cases to some extent in early inspiration. The sound resembles that heard at the neck of a full bottle of soda-water from which the cork has recently been withdrawn. So similar are the two sounds that I have come to call it "the soda-water sign" when speaking of it to others. It cannot be heard through the stethoscope, but its audibility can be increased by folding the morning newspaper for use as a megaphone by the patient or as an ear-trumpet by the physician.

The sign can be banished by the subcutaneous injection of an adrenaline salt, and with it will go the dyspnoea. Further, those cases which have not already developed frank bronchial asthma will do so to a greater or less extent before long: it may be a slight bronchial catarrh with dyspnoea, it may be only dyspnoea on exertion, or it may be a characteristic attack. I have seen it so in many cases, but two stand out in my memory. In the first the sign preceded the asthma by two years, and in the second by five years.

The sign is clearly due to the contraction of the bronchial muscles gradually allowing the inspired air to escape through tubes the lumen of which is narrowed by the spasm and by turgescence of the mucous membrane covered by a liquid exudate. The rales thus formed are at this stage too delicate to transmit vibrations through the chest wall, but they are conducted up the trachea and amplified by it and the buccal cavity. The recognition of the sign clinically will quickly bring a full realization of its value.

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Penicillin in Ophthalmic Therapeutics

The following investigation was carried out in conjunction with Major S. T. Cowan, R.A.M.C., officer in charge of a military laboratory. Its object was to find out what, if any, was the effect on the activity of penicillin of the drugs most commonly employed in clinical ophthalmology.

The drugs tested were: (1) atropine, (2) eserine, (3) adrenaline, (4) cocaine hydrochloride, (5) decicain (amethocaine hydrochloride), (6) argyrol (argent. prot. mite), (7) fluorescein. With the exception of adrenaline, which was used as liq. adrenalin. hydrochlor. 1/1000, the drugs were employed and tested in double the customary strength, so that when diluted with penicillin solution the resultant strength was as desired. Our endeavour was to stimulate as nearly as possible the conditions maintaining in the conjunctival sac.

Procedure.—Penicillin (2 units per ml.) and the drug under test were mixed in equal volumes, and were kept at 37° C. for three hours. The controls were: (1) penicillin (2 units per ml.) + water; (2) each drug + water. After incubation the mixtures were pipetted into porcelain cylinders on plates sown with the Oxford staphylococcus H. The plates were incubated overnight, and the diameter of the zone of inhibition was measured.

Findings.—Argyrol + water diffused into the agar and inhibited the growth of the test organism almost as much as did argyrol + penicillin. Of the other drugs tested none had any inhibitory action on the staphylococcus; only one—adrenaline—inactivated the penicillin in the mixtures.

Additional experiments were made with two solutions of adrenaline hydrochloride. Equal volumes were mixed with penicillin (10 units per ml.) to give a final concentration of

5 units per ml. in the mixture. After incubation at 37° C. for one hour the activity of the penicillin was determined as before. With one solution, said to be several years old, the penicillin in the mixture was reduced to about 0.5 unit per ml. With a more recent sample of adrenaline the penicillin was *completed* inactivated.

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Susceptibility to Acute Nephritis

The following case history may be of some interest as illustrating a familial susceptibility to acute nephritis.

Three girls of the same family, aged 10, 7, and 6 years, were admitted to hospital on Oct. 9, 1944. On attending a child welfare clinic three weeks before admission all three children were found to be infested with *Pediculus capitis*. A nit comb was supplied to the mother, and a week later the children's faces became puffy and swollen. This was more marked in the morning on waking up. It was not until a week later when they attended the child welfare clinic again that their heads were seen to be covered with crusts. Castor-oil and zinc ointment was applied to their heads, and bread poultices to their faces. Three days later the children lost their appetites and their urine was noticed to be red in colour. Of the two younger children, one developed oedema of the legs and back and the other of the legs alone. There was no history of scarlatina or sore throat. The mother gave a history of having had nephritis at the age of 18, and a sister of the father had died of "kidney trouble."

On admission all three showed a considerable degree of impetigo, involving the scalp and limbs. There were still many nits present. The two younger children showed oedema of the face, legs, and back. The blood pressure in all three was raised, being 172/120, 152/115, and 160/110. The urine in all three was smoky, loaded with albumin, and contained many epithelial cells, pus cells, and red cells, with a few hyalogramular casts. There was no oliguria, and during their stay in hospital the oedema and blood pressure subsided, and the impetigo rapidly cleared up with dressings of gentian violet. Examination of swabs taken from one of the impetiginous lesions showed *Staphylococcus albus* and a non-haemolytic streptococcus.

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Open Safety-pin in an Infant's Gullet

I beg to report a case of impacted open safety-pin in the gullet of an infant aged 7 months, and enclose an x-ray negative showing the pin *in situ*. [The illustration is reproduced from part of a print.] On account of the tender age of the patient and consequent smallness of the throat, the smallest endoscope had to be used in such a way that its beak kept the

soft tissues away from the sharp point. The forceps devised by the late Dr. D. R. Paterson were passed down the throat outside the lumen of the endoscope in order to grasp the sharp point. General anaesthesia was necessary. The infant was able to leave hospital (Southern General, Glasgow) apparently unscathed after a few days' observation to rule out hidden trauma.

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