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PRELIMINARY COMMUNICATIONS

New and Simple Test of Nerve Function in Hand

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Summary

A simple objective test of innervation and regeneration of sensory nerves in the hand by immersion in warm water is described. The test results are shown to correspond to operation findings.

Introduction

Moberg (1958) recently focused our attention on the type of sensation in our fingertips which enables us to recognize unseen objects by touch alone. Sensation in the hand has been tested by such methods as the coin test (Seddon, 1954) and two-point discrimination (Weber, 1835), which are subjective tests and therefore depend on the patient's co-operation and intelligence. Moberg (1958) devised an objective test depending on the sweating of the skin of the fingers—the ninhydrin fingerprint method—and with it he was able to show objectively which parts of the skin had intact sensation and which parts were defective.

Patients and Method

Over two years ago the mother of a child who had had a nerve repair in the hand reported that she noticed at bath time the skin of the denervated fingers did not "shrink" like the normal fingers, and that after repair shrinking of the skin returned progressively from palm to tips.

Since then a number of patients have been tested by immersing their hands and forearms in water at approximately 40°C for a period of 30 minutes and the normal shrinking of the skin of their fingers has been observed. It has been noted that after this procedure denervated skin does not shrink; it remains smooth.

FIG. 1—Patient with ulnar nerve lesion. Skin of little finger remained smooth after immersion in water.

FIG. 2—Anaesthetic index finger after tendon graft and multiple nerve repairs.

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For example, a child who had a deep laceration of the palm treated primarily by skin closure only and who was later referred to us had his hand immersed in water for half an hour at 40°C. The skin on the thumb and index fingers and the ulnar border of the little finger shrivelled normally, while the skin on the fingers with loss of sensation—that is, middle, ring, and radial border of little fingers—remained quite smooth. There was also a typical damaged area on the tip of the anaesthetic middle finger. At exploration we found that the situation in the palm was exactly as expected.

Another patient had an ulnar nerve lesion after a motor cycle accident, resulting in pain and tenderness at the elbow. The hand and forearm were immersed in water, resulting in shrivelling of the skin on four digits, the little finger remaining smooth (fig. 1). At exploration about 3 cm above the medial epicondyle an ulnar nerve neuroma was found.

The result of an extensive injury in the palm requiring tendon graft and multiple nerve repairs in a third patient is shown in fig. 2. His index finger at this stage was quite numb, and it remained smooth after testing, while the skin on the other fingers shrivelled normally.

Discussion

An objective method of testing and assessing innervation in the hands is of much more value than traditional subjective tests, which depend not only on the patient's co-operation and concentration but also on his intelligence. Moberg's (1958) finger-printing test is objective and accurate, but requires special equipment. The present test requires nothing more than a basin of warm water for 30 minutes. It is objective, accurate, and shows not only sensory loss, but also the progress of sensory recovery.

I wish to acknowledge the patient's mother's astute observation of this phenomenon in her son's hand and to thank her for drawing my attention to it. I am also indebted to Mr. Scully and the photographic staffs in the hospitals concerned.

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MEDICAL MEMORANDA

Lassa Fever in Britain: An Imported Case

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The diagnosis of Lassa fever has been made in a patient at the Hospital for Tropical Diseases, London, who arrived in Britain by air from West Africa and was at the time of admission suffering from pyrexia of unknown origin. The infection is among the most dangerous of those currently known; mortality rates of 36-52% have been reported for hospitalized cases in the few outbreaks so far known. This report is therefore made with the object of bringing to the notice of practitioners the main features of the disease, particularly in so far as they influence the recognition and management. The complement fixation tests on serum from this patient and on other sera that were examined at the time enabled it to be shown that an outbreak of fever present in Sierra Leone was caused by the Lassa virus.

Another patient who recently arrived in Britain from West Africa has been admitted to the Hospital for Tropical Diseases, London, and it is probable that she has been infected

Case Report

A 35-year-old nursing sister at the Catholic Mission Hospital at Panguma, Sierra Leone, developed generalized aches and pains. Four days later she felt exhausted, developed a temperature of 101°F (38.3°C), and her temperature did not finally settle for another 20 days. The day after onset of the fever she became nauseated and had a headache. There were no other symptoms or physical findings. Malaria parasites were not seen in blood films, but a full course of chloroquine treatment was given without observable effect. One week before the onset of her symptoms the patient had pricked her finger with the needle of a syringe she had used for taking blood from a patient with pyrexia of unknown origin who had died the next day. There had been at least eight deaths among patients suffering from pyrexia of unknown origin in the Panguma area during the preceding six months.

On admission to hospital in London the patient's salient symptoms were severe pains in the limbs, described "as though the bones were breaking," together with headache and excruciating back pains confined to a narrow level around the fourth and fifth lumbar vertebrae. The day after admission she vomited on two occasions; she was anorectic and nausea persisted on and off throughout her illness. Prostration and weakness were severe. She was febrile and, apart from two days, remained so for two weeks with relative bradycardia (see chart). The blood pressure was 90/60 mm Hg and thereafter fluctuated between this level and 115/75 mm Hg. There was no rash, sore throat, faucial injection, lymphadenopathy, oedema of face or neck, or abnormal bleeding.

Findings on admission were: haemoglobin 12.6 g/100 ml; white cells 4,700/mm³ (normal differential); platelets plentiful; reticulocytes 0.4% E.S.R. (Westergren) 13 mm in the first hour; prothrombin ratio 1. Twelve days after admission these were haemoglobin 12.4 g/100 ml; white cells 4,200/mm³ (normal differential); reticulocytes 1.4%; E.S.R. 58 mm in the first hour. Twenty-five days after admission: haemoglobin 11.7 g/100 ml; packed cell volume 35.3%; white cells 6,500/mm³ (normal differential); reticulocytes 4.4%; E.S.R. 23 mm in the first hour. A midstream specimen of urine contained 30 mg albumin/100 ml and a few pus and red cells in the centrifuged deposit. Cultures yielded no significant growth. Stool examination by microscopy and culture showed no protozoal, helminthic, or pathogenic bacterial infection. No malaria or other parasites were found on blood examination, and blood culture on two occasions produced no growth after three weeks' incubation. The Widal test gave titres of less than 20 to the H and O antigens of *Salmonella typhi*, *S. paratyphi A*, *S. paratyphi B*, and *S. para-*

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