literature. The clinical and experimental data concerning the anterior and posterior lobes of the hypophysis are briefly discussed.

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TEST FOR THE POWER OF FLEXOR DIGITORUM SUBLIMIS

BY

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As a result of poliomyelitis the opponens pollicis is sometimes paralysed, severely disabling the hand. The function of the hand can be greatly improved if the flexor digitorum sublimis to the middle or ring finger is disinserted, wound round the tendon of flexor carpi ulnaris, threaded subcutaneously across the wrist, and attached to the distal end of the first metacarpal. This valuable operation should not, however, be carried out unless the finger selected as a donor has (1) a flexor digitorum sublimis muscle powerful enough to perform its new function, and (2) a flexor digitorum profundus capable, even without the sublimis, of flexing the finger strongly. It is easy to estimate power in the profundus, but to estimate the power of the sublimis is much harder, and the following test has proved useful.

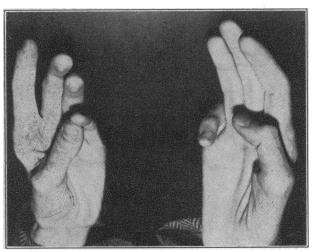


Fig. 1.—Patient is unable to oppose the right thumb, the opponens pollicis having been paralysed by poliomyelitis.

Method

The patient's hand is placed palm upwards on a table. The finger to be tested is left free, while the surgeon—with his own hand—anchors the patient's remaining fingers to the table, keeping them fixed and straight. The patient is now asked to flex the free finger. If he has a strong sublimis

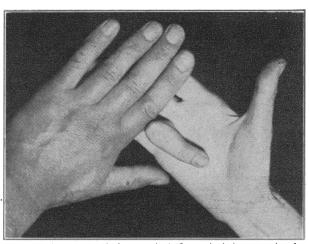


Fig. 2.—The proposed donor (ring) finger is being tested: the terminal joint is nearly straight, and it also feels flail.

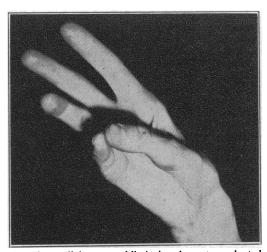


Fig. 3.—Flexor digitorum sublimis has been transplanted from the ring finger to the thumb; it can be seen standing out under the skin and opposing the thumb.

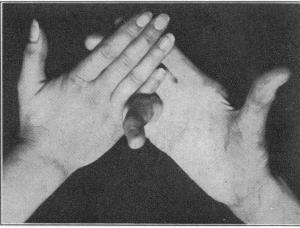


Fig. 4.—Test is being repeated on the finger from which the subiimis is known to have been removed; the terminal joint is flexed and is also rigid. It is this combination of flexion and rigidity which shows that the sublimis is either absent or paralysed.

he will use it to bend at the proximal interphalangeal joint, leaving the distal joint straight—or nearly so: moreover, the distal joint can be freely waggled by the surgeon and to him feels flail. If, however, the sublimis is paralysed or weak, the profundus bends the finger at both interphalangeal joints; in consequence the terminal joint is bent and not flail.

The explanation is as follows. In a normal hand the flexor profundus tendon usually acts en masse, to bend all fingers at all joints. In the test position described above the surgeon's hand prevents the mass action of the profundus; only the sublimis is available to flex the finger, and consequently the terminal joint remains almost straight and flail. If the sublimis is weak, the patient employs what is, in effect, a trick movement, using the profundus to the affected finger as an isolated muscle, and therefore the terminal joint will be bent and rigid.

The efficiency of this test is demonstrated in the photographs.

Medical Memoranda

Two Cases of Amphetamine Poisoning

Amphetamine is easily obtainable by the public in the form of "benzedrine" inhalers, which can be bought freely without prescription. The ingestion of the contents of such inhalers is a practice which appears to be increasing owing to the widely known stimulant action of the drug. The public at large, however, is unfortunately not aware of the dangerous hypertensive properties of amphetamine and of the possible sequelae. The two cases reported below illustrate the grave dangers accompanying benzedrine overdosage.

CASE 1

A stage artist aged 42 was admitted to a medical ward under the care of Dr. Gainsborough in May, 1955. Two days previously he had felt very tired just before he was due to appear on the stage, and had decided to take some benzedrine. He had never taken the drug previously, but had learnt of the practice from his associates. The contents of a benzedrine inhaler were dissolved in a glass of "cocacola," and he drank the resultant mixture. Fifteen minutes later he felt extremely well and very wide awake, but after half an hour he began to feel weak and confused, and developed an intense occipito-frontal headache. He became unable to walk, and was taken home. A doctor who was called two days later found that the patient had a left hemiplegia, and referred him to hospital.

On admission he was fully conscious, and complained of a very severe headache extending over the whole cranium. Examination of the C.N.S. revealed a hemiplegia with almost complete paralysis of the left face, arm, and leg, and a left extensor plantar response. The fundi showed no papilloedema, but many linear and small round haemorrhages were noted, more profuse on the right than on the left. His blood pressure was 180/100, falling in less than 24 hours to 130/70. The urine was sugar-free and blood urea was normal. Lumbar puncture showed a slightly pink fluid with a pressure of 200 mm. The C.S.F. contained 1,200 red cells and 2 white cells per c.mm. Other constituents were normal. The diagnosis was considered to be intracerebral haemorrhage with subarachnoid leak. The aetiology of the cerebrovascular accident was at first puzzling. Here was a fairly young man without hypertension or irregularity of the pulse. The retinal picture was unusual and not suggestive of either uraemia or diabetes. The many small haemorrhages, in the absence of papilloedema or exudates, gave the impression of a sudden hypertension with bursting of the smaller vessels. Amphetamine intoxication, with transient hypertension and

consequent cerebral haemorrhage, was then considered, and a review of the literature strongly supported this hypothesis.

Four days later the patient was transferred to Atkinson Morley's Hospital for neurosurgical investigation. He at first refused ventriculography, but when, after the lapse of a few days, he realized that his condition was not improving he finally agreed. By this time bilateral papilloedema had developed. Ventriculography suggested a posterior parietal parasagittal lesion indenting the body of the right lateral ventricle and the mid-part of the corpus callosum. Operation was performed and 40 ml. of clotted blood was aspirated, after which the patient quickly improved. returned to St. Mary Abbots Hospital five days later and continued to make rapid progress. At the end of five weeks the facial weakness had disappeared and the affected arm had completely recovered. There was still a spastic weakness of the left leg, but the plantar response became flexor, and the patient was able to walk unaided. The retinal picture was returning to normal.

The makers of benzedrine inhalers, Messrs. Menley & James Ltd., inform us that each tube contains 325 mg. of free amphetamine, and about 100 mg. of natural aromatics in the form of lavender oil, this latter constituent being pharmacologically inert. From the patient's story it is probable that he ingested the entire amphetamine content. While it is possible that he might have had a pre-existing aneurysm (carotid arteriogram will be performed at a later date to exclude this), we nevertheless consider that the haemorrhage was initiated by this high dose of amphetamine. We were unable to take his blood pressure until two days after the event, as he did not come under our care until that date, but the fact that the blood pressure can be raised to high levels in such a situation is well illustrated by Case 2.

CASE 2

This patient was not observed personally by us, and we are indebted to Dr. A. Otaki for the clinical details.

A Service man aged 28 became tired after driving his car for three hours, and, still having a long way to go, decided to alleviate his fatigue. He dissolved the entire contents of a benzedrine inhaler in hot coffee and drank the mixture. Fifteen minutes later he felt dizzy and had to lie down. He developed a severe headache extending from the occiput to the vertex, and noticed a tingling in the extremities and difficulty in controlling movement of the fingers. He began to feel that he was "floating on air." Half an hour later he felt sick, and made himself vomit by digital stimulation.

Two hours after taking the amphetamine he was brought to the casualty department of the West London Hospital. where his blood pressure was found to be 160/90. stomach was aspirated immediately, after which he felt a little better, but he still complained that his head was bursting. Examination of his C.N.S. revealed nothing abnormal. There were no changes in the fundi, and the heart was not clinically enlarged. One hour later his blood pressure was found to be 180/110 and he had become very restless. Phenobarbitone sodium, 3 gr. (0.2 g.), was given intramuscularly, but although he became quieter his blood pressure continued to rise, reaching a maximum of 200/125 half an hour later (three and a half hours after taking the drug). Phenobarbitone sodium was repeated twice more during the ensuing eight hours, and at the end of this time his blood pressure had fallen to 145/80. He was discharged to a military hospital, where he made an uneventful and complete recovery. His blood pressure there fell to 125/75 and remained at this level during his two-day stay.

COMMENT

In our search through the literature we have found reports of seven fatalities occurring after large doses of amphetamine during the 20 years that it has been available, but no other record of hemiplegia. In animals the occurrence of cerebral haemorrhage after amphetamine has been clearly established