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EDITORIAL

Pain in the Arm and the Carpal Tunnel Syndrome

The subject of pain in the arm is a broad one. In this number of the Journal, several writers, each expert in his own field, have made contributions which must impress the reader with the need for a keen clinical sense in matters of diagnosis and treatment.

Fortunately some of the painful conditions once treated in an extraordinary number of ways are now responding well to simple measures. The injection of hydrocortisone for tennis elbow and division of the transverse ligament for carpal tunnel compression of the median nerve are two excellent examples dealt with in this number. In fact, this most effective minor operation on the wrist, thanks largely to the warm advocacy of the Middlesex School, has become by far the most frequent of all operations for the relief of pain in the upper limb.

Clear thinking on the carpal tunnel syndrome is greatly helped by separating the cases into two groups—a large homogeneous group, nearly all women, with the 'spontaneous' or *primary* disorder, and a small heterogeneous group of men and women with the disorder *secondary* to some condition such as early rheumatoid tenosynovitis, an old-standing lesion of the carpal bones, acromegaly, or any state of fluid retention such as late normal pregnancy, early toxæmia of pregnancy or myxoedema.

It is interesting here in Great Britain to reflect upon the sudden rise to prominence of the carpal tunnel syndrome during the last ten years, since the article of Brain, Wright and Wilkinson appeared in 1947 under the title of '*Spontaneous Compression of Both Median Nerves in the Carpal Tunnel—Six Cases Treated Surgically*'. The outstanding features of the whole syndrome are the insidious onset of partial thenar atrophy from *paralysis* of the median nerve, and acute nocturnal parasthesiae from *irritation* of the nerve. Paralysis is relatively uncommon, and by itself is painless and of little clinical importance. Painful irritation, however, is very common indeed, and may cause broken sleep night after night for weeks or months or years.

Prior to 1947 the motor paralysis and the sensory disturbance were usually regarded as separate entities, though many writers placed the level of the lesion in either case at the thoracic outlet. The paper of Brain, Wright and Wilkinson helped to make the association clear, brought the level down from the neck to the wrist, and showed how the painful symptoms could be relieved almost immediately by division of the transverse ligament. In America a similar service was performed by the paper of Cannon and Love in 1946

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For some time after these papers appeared, thenar atrophy was regarded as a physical sign essential to the diagnosis, which was therefore made rather seldom. But as one might expect on general grounds, the cases of irritation have been found greatly to outnumber the cases of paralysis of the median nerve. This is the main reason why many series have now passed the hundred mark and are mounting rapidly.

A clear history of median nerve irritation is obviously of prime importance, but is often hard to extract from a distraught or vague woman. It is a good plan to insist that the patient should describe her symptoms over a bad twenty-four hours under three headings—*In the middle of the night, On rising in the morning and During the day with repetitive work.* When the characteristic time pattern of symptoms is given, the diagnosis can be made even in the absence of signs such as weakness of grip or numbness of the tips of the fingers.

The site of the nerve lesion is undoubtedly in the carpal tunnel and nowhere else. This was shown away back in 1913 by Pierre Marie and Foix in a fascinating post-mortem study at the Salpêtrière of a woman aged eighty with bilateral thenar atrophy for no detectable cause—no doubt a case of 'spontaneous' compression. Both median nerves were found 'strangled' in the tunnel and completely degenerated beyond it. *Serial sections of the cervical cord and downwards into the median and ulnar nerves showed no lesion above the level of the wrist.* If only the significance of this report had been realised, all the confusing theories of trouble at the thoracic outlet which persist to this day would never have obtained a foothold, and many a cervical rib and scalenus anticus would have remained undisturbed.

With regard to the mechanism of 'spontaneous' compression, an important observation is that when the transverse ligament is divided at open operation, the cut margins are seen to retract, leaving a considerable gap. By the use of markers and serial radiographs it can be shown that the separation persists. One effect of the operation, therefore, is a permanent enlargement of the carpal tunnel.

Almost certainly the primary fault in 'spontaneous' compression is that the carpal tunnel is just a fraction too tight. This theory is supported by the fact that when the synovial sheaths of the flexor tendons are completely resected in a bloodless field, they are found to weigh between two and six grammes instead of the normal one or two grammes, because of a mild general hypertrophy. This could well be the result of a minor degree of frictional irritation of the synovial sheaths from

active use of the hand over a long period of time. The modest increase in bulk of the sheaths is never enough to cause frank bulging of the deep fascia of the forearm; it lurks there unsuspected. Because the hypertrophied synovial membrane, like the normal membrane, is of loose consistency and highly vascular, its content of tissue fluid and of blood can readily change.

The question is, how to relate these findings to the various symptoms and signs of the syndrome. It would appear that *changes in volume* of the thickened paratenon are responsible for irritative phenomena in the median area. *In the middle of the night* the tissue increases in volume from the peripheral vaso-dilatation which accompanies deep sleep; direct pressure on the median nerve then causes a mixture of pain, tingling and numbness. *On rising in the morning* the hypertrophied synovial membrane impedes the action of the flexor tendons; the grip is weak and the fingers are stiff, puffy and held semi-flexed. During the day with repetitive work, increased fluid content of the tissue may cause symptoms like occupational cramp and a return of tingling.

The mechanism of paralysis of the nerve would appear to be quite different, and related to *changes in position* of the paratenon. With every extension movement of the fingers and wrist the hypertrophied synovial sheaths are drawn downwards under the ligament, where they exert most pressure on the relatively fixed median nerve in the narrowest part of the tunnel. The effect of repeated minor stimuli is firstly to cause the nerve to swell proximal to the ligament, and later to provoke the interstitial fibrosis so fully described by Pierre Marie and Foix. A remarkable degree of local constriction may develop from the gradual contracture of fibrous tissue within the nerve deep to the thick central part of the ligament.

With regard to surgical treatment, Mr. Boulter in this number describes very clearly the technique of blind division of the ligament employed with such success at the Middlesex Hospital as an in-patient procedure under a general anaesthetic. The blind operation has to compete with the open operation, which can be performed in a few minutes as an out-patient procedure under local anaesthesia with a pneumatic tourniquet round the upper arm. The ligament can be seen to be completely divided, the superficial palmar arch (and hence the risk of a haematoma in the palm) can be avoided, and the short incision down the middle of the base of the palm heals almost without trace. Whatever the technique employed, complete division of the ligament enlarges the carpal tunnel and so effects a permanent cure.

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