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TRIBUTE TO HEAD AND RIVERS. UPDATE FROM THEIR ORIGINAL DESCRIPTION OF SENSORY LOSSES IN INTERMEDIATE AND AUTONOMOUS ZONES OF SEVERED INDIVIDUAL NERVES, TO OUR FINDINGS IN MULTIPLE MONONEURITIS AND NON-TRAUMATIC MERALGIAS DUE TO SYSTEMIC DISORDERS.

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ABSTRACT

Introduction. Head and Rivers reported sectioning their radial nerves resulted in larger zone loss of touch (intermediate) than pain (autonomous). Contrariwise, our examining cold instead of touch was reported in Multiple Mononeuritis(MM), with more frequent loss for pain than cold (2013).

Method. To justify presently Heads original epicritic/protopathic theory, we have examined pain vs cold zones in 1248 new cases, beside performing tests for 13 common etiological factors of peripehral neuropathy.

Results. From 101 male/ 143 female, 163 were MM: 69 Polyneuritis with bilateral Meralgia. Age average was 51.8 yrs. There were 700 damaged sensory nerves: 365 with larger Intemediate pain zones: 335 with cold (P<00.5). In 415 etiology factors: 93 were Iron loss, down to 6 MS/GB secuelae. Older Syphilitic, younger Charcot-Marie.

Conclusion. Lack of significant difference between pain and cold indicates similar fine fibre damage vulnerabitlity at 51. Head had not reached sucha age at his self experiment.

KEYWORDS : Age in fine Aδ and C fibre damage. Head´s Theory. Cytokines in Neuropathy.

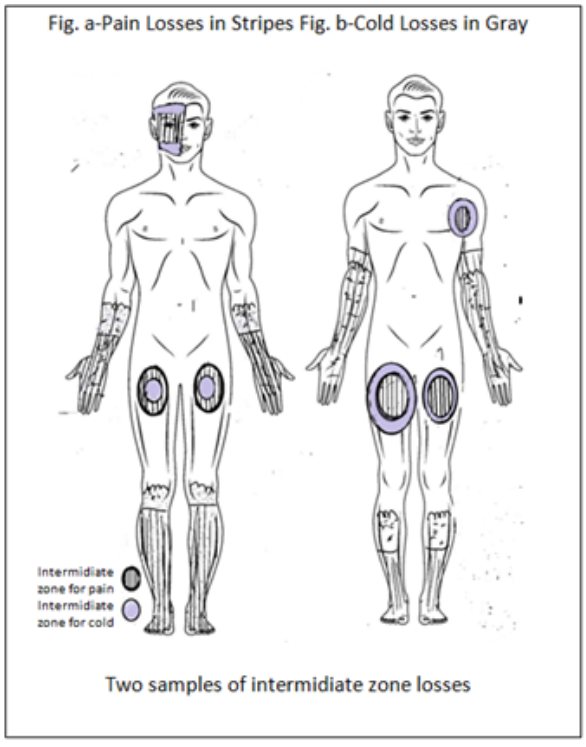
Introduction. Over 109 years ago Sir Henry Head and Rivers´ paper was published. It showed that sections of their own peripheral nerves produced different areas of sensory loss: for pain less than touch or temperature. Photographs presented depicted outer marginal zones larger for tactile or temperature sensory loss; these were named intermediate. Pain sensory loss zone occupied the centre and was called autonomous. This work appeared in Brain in 1908[1], resulting thereafter in Head´s epicritic versus protopathic theory which suggested nature created one sensation more important than the other. It was commented adversely multiple times by various authors such as Trotter, and Walshe, who delivered a definite denial in 1942, much later by Compston[2,3,and4].

After years of investigating the peripheral nervous system and its clinical aspects, we realized it was easier to compare the two zones by using cold sensation instead of touch. In 2013 we published in Neurologia Argentina[5] a paper on 87 cases of Multiple Mononeuropathy complicated frequently by glove and stocking polyneuropathy. For that paper we reported intermediate zone loss for pain in 58 per cent of the cases, only 42% for cold, apparently disagreeing with the original description published by Head and Rivers. Suspecting this disagreement could dim our admiration for these authors, we decided to settle the matter by reporting our inventory of Intermediate and Autonomous zones clinical graphics, of all individual nerves found abnormal in new patients seen since November 2013, at the Rehabilitation Centre (CR), or at our private practice attached to the Universidad Central del Este (UCE). The main purpose of this work is clinical, in order to explore if aetiology may influence in evoking more intermediate zones for cold rather than pain: thus justifying Head´s theory.

Nerve conduction velocities in these polyneuropathies had been extensively studied and included in the Argentinean paper[5], apart form a later presentation as a poster at the XXX International Congress of Clinical Neurophysiology in Berlin, March 2014[6].

Material and methods. Throughout the 2013-2017period,1428 new patients were recorded for their gender, race and age, and were examined using the Medical Research Council aids for peripheral nerve injuries to identify affected individual nerves[7]. To delimit sensory losses, pain was first studied by pricking from midpoint outwards with a plastic straw section, cut (with scissors) at a 45° angle. Cold loss measuring followed, asking patient to report when the edge of a steel tuning fork (1 cm wide arm) began to feel colder, while applying it from oval midpoint towards its periphery (Consulting and commercial office air-conditioner thermostats in Dominican Republic are usually set at 16° Celsius). Both zone extensions were drawn with a crayon: the external, intermediate, and the central, autonomous. The same technique was used on thighs to determine Meralgia Parestesica, common to many Polyneuritis. Graphics were stored in body maps (Graphic 1), though always expressed in the file text. Inclusion required at least 3 damaged nerves for diagnosing Multiple Mononeuropathy (MM), while bilateral Meralgia alone with Polyneuritis also justified inclusion. Polyneuropathies with only glove and stocking, and no individual nerve affected, were excluded.

Graphic 1



Laboratory tests were performed in every case to determine if aetiology could relate to the prevalence of pain over cold in the intermediate zone. For such battery of tests, we selected first the four causes already studied and published by us: i) Disturbances in the iron metabolic profile DIMP[2], ii) Antibody excess of Helicobacter Pylori AEHP[8], iii) HiperIgEemia[9], iv) Presence of FTA ABS in late syphilis[10]. The next group included eight previously known causes of neuropathy such as v) B12 deficit[11], vi) Diabetes Mellitus[12], vii) Streptococcal related and other infections[13], viii) Thyroid diseases[14], ix)

Hiperuricemia[15], x) Carcinomatous neuropathy (CN)[16], xi) Neurological degenerative diseases like Parkinson´s (PD)[17], xii) Charcot Marie Tooth (CMT)[18], and, finally xiii) Secuelae of immune syndromes like Miller-Fisher and Guillain Barré, recently associated with mosquito borne viruses such as Zicka[19].

Results. Included clinical cases studied totalled **244**: 163 classified as Multiple Mononeuritis, 69 as Polyneuritis with Meralgia Paresthetica; 12 polyneuritis with only glove/stocking losses were excluded. There were more women than men, 141 vs103, more Caucasians than non-Caucasians 143 vs.101 but there was no significant difference for gender (P 0·001). There is some significant difference between race and intermediate zone loss of temperature (P 0·322).

Average age in the total sample was 51·85, (Median 52 years, standard deviation 18, most frequent 45, 12 youngest, 96 eldest). Regarding age there is an important significant difference between age and intermediate loss of pain (P 0·478). A result much less important, practically neutral, is of non significance between age and intermediate loss of temperature (P 0·300). These results found for race and age, and in particular, average age of 51, are very similar to those 87 cases due to iron profile disturbances published in Neurología Argentina[5].

In Mononeuritis Multiplex, except for 13 facial motor nerves and 12 auditory nerves, the distribution of 700 affected sensory peripheral nerves was: 398 lateral femoral, 68 trigeminal, 65 musculo-cutaneous, 47 peroneal, 46 ulnar, 36 axillary, 16 median, 9 radial, 7 cervical, 6 intercostals and 2 saphenous. Signs of autonomic disturbance with Myosis were recorded in 12 patients. Intermediate zone loss for pain appeared in 365 (52·1%) nerves, more frequent than cold zone, which prevailed in 335 (47·9%). With no obvious significant difference: there is a compelling suggestion of equal damage occurrence for small Aδ and C fibres.

Distribution of the 13 aetiological factors was led by a disturbance of iron metabolic profile (DIMP) in 93 cases, followed by 85 Helicobacter, 51 B12 deficits, 43 HiperIgEemia, 41 Streptococcal and other infections, 23 Diabetes M, 20 late Syphilis, 12 Thyroid disorders, 15 Gout, 8 Parkinson diseases (PD), 11 Carcinomatous neuropathy, 7 Charcot Marie T., and 6 secuelae of MFS or GBS.

The following had average ages older than the 51·8 years quoted above: Helicobacter, Diabetes, late Syphilis, Gout, Parkinson and Carcinomatous neuropathy. Disturbed Iron Metabolic Profile had the same age of 51, while B12 deficit, HiperIgEemia, Streptococcal and other infections, Hypothyroidism, Charcot Marie and MF/ GBS secuelae had less age. Our results confirm the evidence of multiple causes of extensive peripheral nerve damage in MM and polyneuropathy with bilateral Meralgia. Regarding its pathology we suspect that, along with age, Cytokines mentioned in our publications[5, 8, 9, 10] could damage the distal small diameter sensory fibres, as well as the central somato-sensory and nociceptive pathways.

Conclusions. The results in this paper indicate that ageing seems to be an important aetiological factor, turning fine Aδ delta and C fibres vulnerable after 51 years of age. We could postulate for Sir Henry, that when conducting his self experiment, he was only in his early forties and thus his fine fibres had not become vulnerable, therefore the difference in his results from ours.

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