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EDITORIAL

PAIN

Pain is a very common manifestation of injury and disease and for the physician it is a useful symptom, since without it many a diagnosis would be difficult or delayed. Much information is available on the subject, though little of the knowledge has passed into general circulation, and there is still much scope for research into the many problems which the subject presents. The problems of pain are well set out, especially from the point of view of the practising doctor, in one of the 'Practitioner' handbooks.[1]

Pain is produced by stimulation of free nerve-endings of fine terminal branches of nerve fibres. By means of stimuli applied to various parts of the body it has been shown that there are two waves of pain, with an interval between them corresponding to the difference in the rate of conduction in myelinated and non-myelinated afferent fibres. However, stimuli applied to the surface of the body are likely to stimulate the endings of many different fibres; thus, however localized the stimulus, the message which reaches the central nervous system will produce sensation representing the summation of effects from many different fibres. The precise mechanism of stimulation of the pain nerve-endings in the skin is not known with certainty, although some change in the permeability of the surface membrane of these nerve-endings appears to be essentially involved.

The effects of pain on the higher levels of the central nervous system are hardly confined to precise channels but are diffuse and generalized. There are no regions of the cerebral cortex specially concerned with reception of pain. The effect of pain is mainly on the central regions which regulate cortical activity; the pain signals play little part in building up cortical or mental patterns, although the cortex does apparently contribute to the perception of pain as a mental event. The ventrolateral nucleus of the thalamus is almost certainly the main perceptive centre for the affective aspects of pain. Lesions of the cerebral cortex do not abolish perception of pain permanently nor do they reduce materially the sensibility to pain-producing stimuli; on the other hand destruction of the lateral part of the thalamus produces analgesia of the opposite half of the body.

VAN DIE REDAKSIE

PYN

Pyn is 'n baie algemene manifestasie van besering en siekte en vir die geneesheer is dit van belang want daarsonder sou menige diagnose moeilik wees of vertraag word. Heelwat inligting is oor die onderwerp beskikbaar, maar dit is nie algemeen bekend nie. Daar is ook heelwat geleentheid vir navorsing oor die menigte vraagstukke wat die onderwerp oplewer. In een van die ‚Practitioner'-handboeke[1] is daar 'n deeglike uiteensetting van die probleem veral soos dit die praktiserende geneesheer raak.

Pyn word veroorsaak deur prikkeling van die vrye senu-ente van die fyn eindvertakkings van die senuweedrade. Deur prikkelings op verskillende dele van die liggaam toe te dien, is bewys dat daar twee pyngolwe is met 'n tussenpose wat gelykstaan aan die verskil in spoed van geleiding tussen toevoersenuweedrade met murgskedes en dié sonder murgskedes. Prikkelings wat egter op die oppervlakte van die liggaam toegedien word stimuleer moontlik die ente van 'n aantal verskillende senuweedrade. Dus, hoe gelokaliseer die prikkeling ookal mag wees, sal die boodskap wat die sentrale senuweestelsel bereik 'n gewaarwording skep wat die totale uitwerking van verskillende drade verteenwoordig. Die juiste meganisme waardeur die pyn-senu-ente in die vel geprikkel word, is nie met sekerheid bekend nie, alhoewel dit waarskynlik is dat een of ander verandering in die deurdringbaarheid van die oppervlakkige vlies van hierdie senu-ente hoofsaaklik daarby betrokke is.

Die uitwerking van pyn op die hoër sentrums van die sentrale senuweestelsel is nie juis tot presiese kanale beperk nie maar is versprei en algemeen. Geen deel van die breinskors is spesiaal by die opneem van pyn betrokke nie. Pyn affekteer veral die sentrale dele wat die skorsaktiwiteit beheer. Die pynprikkels speel nie 'n groot rol in die vorming van skors- of begripspatrone nie, alhoewel die skors waarskynlik bydra tot die gewaarwording van pyn deur die verstand. Dit is amper seker dat die ventrolaterale kern van die talamus die hoofwaarnemingsentrum vir die gevoelsaspekte van pyn is. Letsels van die breinskors neem nie die persepsie van pyn permanent weg nie, ook verminder hulle nie wesenlik die gevoeligheid vir pynverwekkende prikkels nie. Vernietiging van die laterale deel van die talamus veroorsaak egter gevoelloosheid van die teenoorgestelde helfte van die liggaam.

Soos hierbo aangedui stimuleer pynverwekkende prikkels ook ander soorte gewaarwordings, soos bv. die

As indicated above, pain-producing stimuli excite other forms of sensation, such as touch or pressure or heat, and these sensations produced at the same time as pain are probably responsible for accurate localization and discrimination. In the rare condition known as the thalamic syndrome, caused by softening in the lateral aspect of the thalamus, intense pain occurs in the opposite side of the body. Otherwise the central nervous system is insensitive to injury and no pain arises unless sensory roots, or parts of the meninges, or possibly larger vessels, are also involved.

The relief of pain has always been one of the chief tasks of doctors, and it is a well-established principle that if possible it should not be allowed to interfere with the full investigation and treatment of the condition which is causing the pain. The possibility of pain being psychogenic in origin should always be borne in mind. Pain of apparently non-organic origin may be referred to any part of the body. Careful examination of the history, with appreciation of the behaviour of the patient, may enable a conclusion to be reached and a positive diagnosis to be made. The services of a psychiatrist may be essential when no satisfactory organic aetiology can be found, when pains recur at regular intervals, and when ordinary analgesic measures do not bring relief. The pain of functional disease requires treatment just as obviously as pain due to organic disease; and in the correction of the psychogenic disorder not only medicinal treatment may be of value, but also social and moral help.

1. *Pain and its Problems* (1950): The Practitioner, London: Eyre and Spottiswoode.

tas-, druk-, of hittegevoel, en hierdie gewaarwordings wat tegelykertyd met die pyn ontstaan, is waarskynlik verantwoordelik vir presiese lokalisering en die vermoë om te onderskei. In die seldsame kondisie, bekend as die talamussindroom, wat veroorsaak word deur verweking in die laterale aspek van die talamus, kom daar geweldige pyn in die teenoorgestelde kant van die liggaam voor. Anders is die sentrale senuweestelsel gevoelloos vir besering en geen pyn word gevoel nie tensy sensoriese wortels, of dele van die breinvliese, of moontlik groter bloedvate betrokke is.

Verligting van pyn is nog altyd een van die geneesheer se grootste take. Dit is egter 'n erkende beginsel dat, indien moontlik, die verligting van pyn nie moet verhinder nie dat die toestand wat die pyn veroorsaak, volledig ondersoek en behandel word. Die moontlikheid moet altyd in gedagte gehou word dat pyn psigogeen in oorsprong kan wees. Pyn wat oënskynlik nie-organies van oorsprong is, kan na enige deel van die liggaam uitgestraal word. Noukeurige ondersoek van die geskiedenis, met inagneming van die pasiënt se gedrag, kan mens in staat stel om tot 'n gevolgtrekking te kom en om 'n positiewe diagnose te maak. Die hulp van 'n psigiater is waarskynlik nodig wanneer geen bevredigende organiese oorsaak gevind kan word nie, wanneer pyne met gereelde tussenposes terugkeer, en wanneer die gewone pynstillende middels nie verligting bring nie. Die pyn van funksionele siektes het behandeling net so nodig soos die pyn wat aan organiese siektes te wyte is; vir die verhelping van 'n psigogeniese kwaal is nie net geneesmiddels van belang nie, maar ook sosiale en morele hulp.

1. *Pain and its Problems* (1950): The Practitioner, Londen: Eyre en Spottiswoode.

CLUBBING OF THE FINGERS AND HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

Nothing in medicine is more puzzling than the clubbed finger. How can one hope to explain it when it can apparently occur in such a variety of conditions as cyanotic heart disease, bacterial endocarditis, a tumour in the lung parenchyma no larger than a marble, acute hepatitis within 15 days, aneurysm of the aorta, intrathoracic goitre, myxoedema, and idiopathic steatorrhoea? Hippocrates unfortunately laid stress on the non-essential changes and today clubbing is frequently incorrectly diagnosed. The basic clinical feature, so well described by Lovibond,[1] consists of an increase in soft tissue beneath and around the bed of the nail, without which clubbing cannot be considered present.

None of the various theories that have been proposed to explain the production of clubbing is satisfactory for all cases, though a few more definite clues are now available. There is an anatomical change in the capillary loops in the nail bed, which are wide and tortuous, while the blood flow through the clubbed fingers is increased (this does not apply to the congenital cases). If the causal intrathoracic lesion can be eradicated the clubbing and heightened blood flow may disappear *pari passu*. On the other hand an increase in blood flow can hardly be the primary factor, since clubbing does not occur in hyperthyroidism, and it would be strange to think of hypothyroidism initiating a rapid circulation.

Hypertrophic osteo-arthropathy is equally obscure. Here there is pain and stiffness in the joints of the limbs, together with pain, swelling, tenderness and even redness of the limbs themselves, especially of the forearms towards the wrists. The underlying intrathoracic lesion may be silent and rheumatoid arthritis diagnosed. Clubbed fingers, however, are always present—in fact we usually consider osteo-arthropathy as a sort of extension of clubbing. Yet the two are highly dissimilar—clubbing is painless (with rare exceptions), osteo-arthropathy usually painful; in clubbing the new tissue is non-osseous and obvious on inspection, in osteo-arthropathy it is periosteal and bony, and needs an X-ray for its diagnosis.

With the increasing incidence and awareness of bronchial carcinoma it would appear that this may now be regarded as the chief cause of osteo-arthropathy, and Semple and McCluskie[2] have recently described 24 examples of such a combination. They call attention particularly to those cases in which the joint symptoms may be the presenting features of a lung cancer. The moral, presumably, is to X-ray the chest in all cases of

PEPTIC ULCER

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In 1936 Selye[1] in describing the findings in animals submitted to physical and toxic stress stated *inter alia:* 'Gastro-intestinal ulcers and other manifestations of shock were actually more severe in adrenalectomised than in intact animals and could be lessened by treatment with cortical extracts. These lesions are not mediated through the adrenal; in fact they are actually combated by an adequate adrenacortical response to stressor agents.'

To those of us who have come to accept peptic ulcer as a stress-induced pathological entity it surely must become a matter of some concern that even after possessing such knowledge for close on 20 years our preventive and therapeutic approach should bear so little relationship to our adopted concept of causation. There must be a reason for this; it may be that we have become too tied down by prejudice to contemplate that past therapy, dietary and medicinal, has been based on erroneous theory and that our ideas urgently need reorientation.

In this field an admirable summary of modern therapeutic measures has been compiled by Berk.[2] In his review he features the vagaries, the limitations and the benefits associated with the exploitation of banthine, methonium salts, sodium carboxymethylcellulose, cortisone, ACTH urogastrone, enterogastrone, protein hydrolysates, anion-exchange resins, gastric mucin, and atropine. Having done so he concludes: 'It is apparent that a specific cure for peptic ulcer is still not at hand.' It is of extreme interest that in this comprehensive review reference to antacid therapy is conspicious by its absence. What the account does reveal is an earnest attempt to give full value to the stress factor and to evolve a therapeutic approach which might adjust the stress as well as the physical and bio-chemical disharmony engendered by such stress or by failure of systemic anti-stress defence.

THE REACTION TO STRESS IN RELATION TO PEPTIC ULCER

In the past we have been led to devote too little attention to the patient as a whole and too much to his ulcer. This being so, it behoves us in the first instance to crystallize in our minds the picture of what constitutes normal reaction to stress, and having done so we are then much better equipped to assess and rectify systemic disharmony indicative of failure of defence against stress.

Normal reaction to stress should be too well known to require lengthy elaboration in such a discussion as this. Cannon[3] has made it common knowledge that adequate reaction by the human economy is represented by an outpouring of adrenaline and a mobilization of sugar into the blood stream. These reactions represent cause and effect, but before the latter eventuates there are numerous hormonal and metabolic activities to be invoked, all of them contributing to the final physiological result and any one, in failure, conditioning the emergence of pathology such as is represented by peptic ulcer.

All digestive stimuli of whatever nature, be they physical or psychological, motor or sensory, visual, olfactory or gustatory, eventually reach the cerebral cortex, whence responses are relayed to the thalamus and hypothalamus. From the latter, impulses are transmitted along the vagus and sympathetic nerves, the latter catering for the outpouring of adrenaline.

In addition to the neurogenic stimuli arising in the autonomic centres in the hypothalamus Hume[4] has adduced evidence that a hypothalamic hormone is formed and that this exerts a stimulatory effect on the anterior pituitary, which in turn by virtue of secreted ACTH sets the suprarenal cortex into active production of its own particular hormones. Hume has also adduced some evidence suggesting that an injection of adrenaline acts on the anterior pituitary in a manner indistinguishable from that of the hypothalamic hormone. If this be so it becomes a matter of interest that a reaction starting in the suprarenal medulla should induce defence response through devious channels at the ultimate level of the suprarenal cortex.

The above findings have been confirmed by Selye[5] and by Ungar,[6, 7] and all investigators are agreed that the ultimate goal to be reached is the prevention of cell-breakdown and of histamine release. In the *British Medical Journal* of 5 March 1947 a sub-leader appraising