PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

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The Royal Society

Phil. Trans. R. Soc. Lond. B 1985 **308**, 269-270
doi: 10.1098/rstb.1985.0026

Phil. Trans. R. Soc. Lond. B **308**, 269–270 (1985)
Printed in Great Britain

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The very first point for discussion was about the meaning of the word 'nociception'. This problem came up throughout the meeting and was the main topic considered in the general discussion at the very end. Dr Nathan reminded the audience that the term 'noci-receptive' was due to Sherrington and was not equivalent to 'pain'.

Dr Nathan's account of human beings inducing trance states so as not to feel pain drew some anecdotes from the audience: of people lying on beds of nails (who did not feel pain after naloxone administration) and of sympathetic labour pains, as well as the state of fire-walkers' feet. To a question about whether there was any evidence for animals controlling pain, animal hypnosis and acupuncture analgesia were discussed, although it was agreed that the mechanisms are not understood. Finally, it was noted that stress-induced analgesia in animals can be conditioned so that withdrawal reflexes and escape behaviour may be suppressed.

Dr Torebjörk's presentation drew congratulations on the results obtained from his technically demanding experiments, especially the achievement of recording from Aδ axons in man. There was considerable discussion about possible temporal and spatial summation in the central nervous system in response to activation of the polymodal nociceptors, especially in view of the fact that pain ratings remained elevated after the C fibre discharge had ceased. Also, the point was made that stimulating the receptive field (adequate stimuli) or the nerve (electrical stimuli) would activate several fibres rather than a single axon. In reply to these points, Dr Torebjörk said he was aware of them but it was difficult to escape the conclusion that activation of a single human C fibre could lead to a sensation of pain. Cramp-like muscle pain seemed to be due to activation of Aδ and C afferent fibres.

The beautiful correlation between pain and neural firing was remarked upon and a question asked concerning whether this could be changed by drugs. So far, little has been done in this area but a prostaglandin synthetase inhibitor, used to by-pass the receptors, seemed to have a central analgesic effect.

The fact that Aδ nociceptors seem to have thresholds at about the pain threshold whereas C nociceptors have thresholds below the pain threshold was noted and it was suggested that the Aδ fibres were responsible for the sensation. Dr Torebjörk thought it was not as simple as that: heat pain mainly depends on temporal (and spatial) summation since after pressure block of A fibres the pain threshold stays the same.

The main thrust of the discussion on Professor Iggo's presentation returned to the problem of nociceptive specific neurons and their possible role in sensation. It was claimed in discussion that flexor motoneurons are 'nociceptive specific' and that under conditions of anaesthesia and also strong descending inhibitory controls there is always the possibility that the experimenter is only seeing part of the whole. Professor Iggo replied that nociceptive specific neurons have been shown to project to the brain, e.g. to the thalamus, and that careful searching fails to reveal any input to these neurons from sensitive mechanoreceptors. Descending tonic inhibition was very powerful and selective for the nociceptive component of the response of the deeper, lamina V, neurons. But there was some evidence that there was no descending tonic inhibition

of the neurons in substantia gelatinosa, rather these neurons were facilitated by electrical activation of the dorsolateral funiculus in the descending direction.

Dr Willis's paper led to a discussion on the effects of anterolateral cordotomy in man with disagreement between members of the audience as to whether or not the original pain could return after cordotomy (as opposed to a pain problem returning, such as a lowering of the level of analgesia or the appearance of dysthesias). To a question on whether there is any suggestion of an increased number of fibres after experimental cordotomy Dr Willis had no information, but Dr Nathan said that he and Dr Marion Smith had a large amount of human material that showed no evidence for regeneration.

The problem of species differences was raised, with the suggestion that the cat should be re-classifed as a 'red herring' and that primates and rats were similar. The abiding impression left with this rapporteur was that all species have several parallel ascending neuronal pathways handling information from nociceptors and that our present knowledge makes it difficult to consider any one of these pre-eminent as a 'pain pathway'.

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