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Peripheral tissue injury alters dramatically the relation between a cutaneous stimulus and the sensation experienced by producing both a decrease in the threshold necessary to elicit pain and an increase in the pain resulting from suprathreshold stimuli (hyperalgesia). We have now investigated whether these injury-induced changes result from alterations in the properties of primary afferents (sensitization) (Lynn 1977) or whether the injury triggers a change within the c.n.s. (Woolf 1983).

Two types of peripheral injury have been used; either acute radiant heat (75 °C for 90 s) or a subcutaneous injection of turpentine into the hindpaw. The experiments have been performed in chronic decerebrate rats (Woolf 1984). These animals have intact brainstem and spinal cord reflexes indicating that the effects of injury on reflex behaviour can be studied as well as changes in the electrophysiological properties of primary afferents.

Both the radiant heat injury and the turpentine injection in the chronic decerebrate rat result in a marked swelling and erythema, which is accompanied by a consistent fall in the mechanical threshold necessary to elicit the withdrawal reflex and an alteration in its responsiveness (sustained flexion, limping, etc.). These changes have been studied both behaviourally and electromyographically.

We have attempted to correlate this change in the output of the spinal cord resulting from peripheral injury with possible changes in the primary afferent input. Single primary afferents (particularly C-nociceptors) innervating the skin of the foot have been studied in control rats and in chronic decerebrates with injured skin. Thermal injury and turpentine inflammation did not alter the mean mechanical threshold of C-afferents even though these animals exhibit reflex hypersensitivity. Individual afferents when studied during the course of the radiant heat injury showed decreases in threshold (30% of units), increases (20%), or no change (50%). One aspect of mechanosensitivity that was altered by the injury was the development of afterdischarges to mechanical stimuli in 40% of C-afferents following radiant heat burns and in 25% of afferents innervating turpentine inflamed skin.

The lack of correspondence of the changes in reflex behaviour and in the properties of C-primary afferents following injury implies that hyperalgesia is not merely a reflection of a change in afferent sensitivity.

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