DIFFERENTIAL PERIPHERAL NERVE FIBER BLOCK: UNIT STUDIES IN THE VAGUS NODOSE GANGLION

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Introduction. The sequence of block of large and small nerve fibers by local anesthetics is important for understanding and controlling differential effects but remains unsettled because of ambiguity inherent in compound action potentials. Teased fiber preparations avoid this difficulty but incorporate unstandardized mechanical disturbance. We here report unequivocal data, obtained by extracellular recording of units in the inferior vagal ganglion while treating and stimulating the intact nerve.

Method. Nerve and ganglion excised in continuity were incubated in a three compartment chamber partitioned by vaseline, the first for the ganglion, the second and third for 40 mm of nerve, the third for the distal end immersed in mineral oil. The first and second compartments were perfused independently with oxygenated (95% O₂ - 5% CO₂) bicarbonate buffered glucose-Ringer at 37° - 38° C; lidocaine hydrochloride was applied only in the second compartment, in concentrations (mM) 0, 0.2, 0.4, 0.6 or 0.8 (0 - 0.2 g/dl). Unit action potentials were recorded with a tungsten microelectrode micromanipulated into the ganglion. The distal end of the nerve was stimulated supramaximally at intervals of 1 - 10 min. After a control period of about 1 h, a test solution (one solution per nerve) was applied and its effect observed to equilibrium. Control solution was next restored and the course of recovery charted. The electrode was then withdrawn, inserted elsewhere in the ganglion, and a second set of units observed. Potentials were stored on magnetic disk via a Nicolet digital storage oscilloscope, and reproduced on an XY plotter (Fig. 1).

Results. A total of 99 units were observed in 23 ganglia. Latencies ranged between 1.44 and 90.85 millisecond, equivalent to conduction velocities of 37.5 to 0.55 m/sec (Fig. 2). The dose-response to lidocaine identified three groups. (1) Axons conducting at velocities 37 - 5 m/sec, 50% of which were blocked by lidocaine 0.4 mM (0.01 g/dl) (myelinated Aβ and Aα fibers). (2) A small group in the range 2 to 1.2 m/sec all of which were blocked by 0.2 or 0.4 mM lidocaine ('rapid unmyelinated C fibers'). (3) A large group with velocities 1.25 - 0.5 m/sec or less, resistant to block by 0.2, 0.4 or 0.6 mM, but generally blocked by the 0.8 mM concentration ('slow' unmyelinated C fibers). The time course of the increase in latency indicated that equilibrium was reached within about 20 min. On washing, latency recovery reached a nearly steady state within about 20 min but a residual slowing of conduction averaging 11% (range 1 - 32) of the control velocity was still present after 60 min.

Discussion. The results subdivide the C units into two groups distinguishable by conduction velocity and susceptibility to conduction block by lidocaine. The slower C fibers were the most resistant to block, whether the residual increase in latency after 1 h of washing was a preparation artifact or an effect of the lidocaine remains undetermined. However similar slowing produced with increased ambient KC1 (20 mM) was followed by complete recovery in the case of myelinated axons susceptibility to lidocaine did seem to relate to conduction velocity.

Conclusion. Unit study distinguishes two populations of vagus unmyelinated axons: 'rapid' and 'slow'. The second, many of which type conduct slow pain, is about half as sensitive to lidocaine as the first. (NIH Grant GM-27678-02).

References

Fig. 1.

Responses of C units in ganglion nodosum before and after lidocaine 0.4 mM was applied to the nerve (sheath intact, conduction length = 50 mm). Note changes in spike latencies, extinction of large spike, recovery on washing. Large spike = 320 mV. Sweep width = 205 msec.

Fig. 2.

Conduction velocity of 99 units in ganglion nodosum before treatment of nerve with lidocaine. Effect of one concentration of lidocaine on conduction is also shown: blocked, o, not blocked, x.