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**Abstract**

Research objective and rationale: The role unmyelinated low-threshold mechanosensitive afferent (LTMA) fibers, C-tactile afferents (CTs), play in the expression of dynamic mechanical allodynia (DMA) is unclear. CTs fire optimally to slow gentle dynamic touch but this preferred stimulus unavoidably co-activates A-beta LTMA fibres. A method differentially blocking CTs would facilitate future human pain models assessing the role of LTMA sub-types in DMA. To address this a model utilizing non-invasive administration of lidocaine was developed. Methods: Lidocaine (0.2mg/cm2) and adrenaline (2.5µ/cm2), were co-iontophoresed on a 16cm2 area of the volar forearm of 17 healthy participants (9 female). Adrenaline was given to increase the depth and duration of local anesthesia. Adrenaline-only was administered contralaterally. Thermal and tactile detection thresholds as well as pleasantness and intensity ratings to slow (3cm/s) and fast (30cm/s) dynamic touch were performed bilaterally. In separate investigations the underlying neural mechanisms were explored using single unit recordings of LTMA fibres in forearm hairy skin. Assessment of mechanical threshold and firing pattern to mechanical skin stimulation were performed before and after lidocaine and adrenaline co-iontophoresis over the receptive field. Results: Compared to control, lidocaine iontophoresis resulted in significant impairment in warm (p<0.008), cold (p<0.007) and, most evidently, tactile detection thresholds (p<0.001). Touch pleasantness was unaffected but there was a significant reduction in intensity ratings for dynamic touch (p<0.01). All but one slowly adapting type 1 (SA1) fiber (6/7) showed preferential block of firing during the sustained indentation phase of mechanical skin stimulation post-lidocaine. This partial blockade temporally matched an elevation of tactile detection threshold. One slowly adapting type 2 LTMA (1/9) showed a similar pattern but other sub-types (hair follicle afferent (n=10), field unit (n=2) and CT (n=1)) showed no appreciable change in firing. Conclusions: No evidence of differential blockade of CTs following lidocaine iontophoresis was seen and the model is unlikely to be of use in deciphering their role in DMA. The lack of blockade could reflect the ion channel population, yet to be clarified, present on these fibers. Instead, the impairments to dynamic and punctate mechanical stimuli following lidocaine are associated with a phenotypic switch of SA1 fibers to fast adapting. The mechanisms underlying SA1 firing during the sustained phase of skin indentation appear more sensitive to sodium channel blockade than at onset or offset.

**Abstract Citation**

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