had a lower level of analgesia than those patients who were free from pain (regardless of dose of tetracine), we would have concluded that the pain travelled "around" the spinal. de Jong and Cullen state, "Fortunately, a cutaneous level of analgesia to the tenth thoracic segment provides adequate spinal anesthesia for the vast majority of lower extremity operations." Using the same dose of tetracaine but obtaining a lower level of analgesia causes a higher concentration of local anesthetic in the anesthetized part, a therapeutic result exactly in line with what Dr. Deas and I have written. It seems to me as if Drs. de Jong and Cullen *act* as if they believe Dr. Deas and myself despite what they have written.

LAWRENCE D. EGBERT, M.D.
Department of Anaesthesia
Massachusetts General Hospital
Boston

To the Editor.—Dr. Cullen and I are pleased that the subject of tourniquet pain continues to be of interest. That unanimity of opinion as to its cause has not been reached is apparent from the above communication. We thank Dr. Egbert for giving us the opportunity to reply to his well-documented interpretation.

Summing up the two different explanations for the occurrence of tourniquet pain during spinal anesthesia: (1) Tourniquet pain is transmitted by nerve fibers larger than those transmitting other types of pain (*i.e.*, larger than Aδ and C fibers), but running along the classical anatomical segmental distribution; or (2) tourniquet pain like other painful stimuli is transmitted by nerve fibers which fall into the usual physiological classification for pain fibers (*i.e.*, Aδ and C fibers), but some of which enter the cord at a level cephalad to that of the analgesic block along paraspinal pathways in the sympathetic trunks.

Recent studies in man [1] have shown beyond a reasonable doubt that pain is transmitted by smaller nerve fibers only. Stimulation of larger fibers—which incidentally have a lower threshold, *i.e.*, they fire off more easily—has never been shown to be painful.

Even more pertinent is the well-known observation that tourniquet compression causes progressive fallout of nerve fibers according to size, with largest fibers blocked relatively quickly. Thus by the time tourniquet pain usually appears, which is 45 to 60 minutes after application of the tourniquet, all large fibers are already blocked and the small myelinated fibers, *i.e.*, Aδ fibers) are just beginning to be affected. It seems unlikely to us that impulse conduction can take place in large fibers which have already been blocked by tourniquet compression.

We therefore stick to our premise that tourniquet pain must, of necessity, be associated with impulses transmitted by small amyelinated C fibers, which are unaffected by tourniquet compression at the time of onset of tourniquet pain.

Finally, we would like to show why Dr. Egbert observed a high incidence of tourniquet pain when using relatively low concentrations of local anesthetic in spinal fluid and why he noted a reduced incidence of pain when using higher concentrations of agent.

His observation of apparent "break-through" of a block is an excellent demonstration of Wedensky-type inhibition, which may be seen at near minimum blocking threshold (C_m) concentrations of local anesthetic. Under these conditions a nerve is effectively blocked for single impulses, as for example a pin-prick, yet will pass repetitive stimuli, as for example a surgical incision, but at a reduced frequency. Thus what appears to be a "break-through" of a strong stimulus beyond a block existing for a brief stimulus, in reality is not related to the strength of the stimulus but rather to its duration. Such conducted impulses will, however, be much attenuated after passing through a nerve segment at threshold.

This not too well-known phenomenon is considered in more detail elsewhere.[2]

RUDOLPH H. DE JONG, M.D.
University of California Medical Center
San Francisco

REFERENCES

1. Collins, W. F., Nulsen, F. E., and Randt, C. T.: Relation of peripheral nerve fiber size and sensation in man, Arch. Neurol. 3: 381, 1960.
2. de Jong, R. H., and Wagman, I. H.: Physiological mechanisms of peripheral nerve block by local anesthetics, ANESTHESIOLOGY **24**: 684, 1963.