CORRESPONDENCE

Reprints

To the Editor.—It has been brought to our attention that a large number of unsolicited reprints of a paper entitled: "Promethazine: its influence on the course of thiopentone and methohexital anaesthesia,"—*Anaesthesia,* 1961, vol. 16, page 61—have been circularized to anaesthetists in the United States.

These were purchased by permission of the authors and the Editor of *Anaesthesia,* but it did not occur to the authors when permission was granted, that the order would be for 10,000 reprints.

We wish it to be known, however, that we were not personally responsible for, nor do we condone, this mass circularization. Our American colleagues may ascribe this to us since the reprints were sent from Dublin bearing a Republic of Ireland postage stamp.

JAMES MOORE, M.D. and
JOHN W. DUNDEE, M.D.
The Queen's University of Belfast

Tourniquet Pain During Spinal Anesthesia

To the Editor.—A recent article in ANESTHESIOLOGY ("Cause of Pain from a Pneumatic Tourniquet During Spinal Anesthesia" by Drs. Egbert and Deas)[1] prompts me to write, since I believe the author's conclusions are based on an incorrect interpretation of their data.

Egbert and Deas have based their conclusions on the grounds that tourniquet pain, ". . . is carried to the spinal cord by nerve fibers larger than those transmitting pricking pain." The authors' interpretation of the writings of Heinbecker, Bishop and O'Leary[2, 3] is different than mine. I believe this group had already shown in the early 1930's that pain is carried by *small,* not large, myelinated fibers, a fact that has been corroborated repeatedly. Pain in unanesthetized man is carried only in the small myelinated A-delta fibers and in the even smaller, nonmyelinated slow-conducting C-fibers. These fibers are blocked by relatively low concentrations of local anesthetics which are insufficient to affect the larger and more resistant proprioceptive and motor fibers. The presence of complete cutaneous analgesia to pin-prick is, therefore, an indication of complete block of *all* pain fibers entering the spinal cord below the corresponding dermatomal level, even though touch and motor function may still be intact in the extremity. The reverse is true of pressure block of a nerve. Compression of a nerve trunk will differentially block the largest sensory fibers before smaller ones, and pain can still be felt after tourniquet pressure has abolished touch and proprioceptive senses in 30 minutes or less. That tourniquet pain characteristically does not arise until at least 45 minutes after inflation of the cuff, points to the likelihood of small fiber involvement. This is especially true since by that time larger myelinated sensory fibers will have become completely blocked by tourniquet compression.

Electrical stimulation of a nerve in unanesthetized man has been one of the best tools in assigning charactersitic sensory modalities to specific fiber sizes. Electrical stimulation of large fibers alone is interpreted as touch, *but never as pain.* Not until the smaller A-delta fibers are also stimulated is pain felt and interpreted as a sharp pricking. Additional increases in stimulus strength to C-fiber threshold will then add a quite uncomfortable deep aching component to the subject's pain interpretation.[4] This writer's ulnar nerve has been stimulated for hours by a current sufficient to cause muscle contractions, yet each shock was felt only as a "thump" and was never painful before, during, or after upper-arm-tourniquet compression for up to 45 minutes.

Since pain then is carried exclusively by smaller fibers, which we may assume to be completely blocked if adequate cutaneous analgesia is present, an alternative explanation must be presented to understand the occurrence of tourniquet pain. The most logical hypothesis I can offer is that some pain fibers, especially nonmyelinated, slow-conducting, deep pain, C-fibers, travel, sometimes for a considerable distance, in the sympathetic trunks. Thus, such sensory fibers may completely bypass their expected dermatomal entry level into the cord to enter at a higher unblocked level via the white communicating rami and be centrally interpreted as pain. Kuntz [5] has demonstrated afferent fibers in the sympathetic trunks arising rostral to the spinal representation of the sciatic nerve. Such pathways also offer a plausible explanation for other bizarre pain phenomena seen during spinal analgesia. The sudden onset of causalgia or pain on surgical stimulation of the sciatic nerve following a solid spinal block are examples. The reason that pain is not immediately perceived after tourniquet inflation may well be that only prolonged compression or anoxia of the nerve, or both, will lead to spontaneous irritative discharge of the fibers under the tourniquet.

That Egbert and Deas were able to reduce the incidence of tourniquet pain by increasing their dose of spinal anesthetic agent (from 12 to 16 mg. of tetracaine) might be explained by the fact that larger doses of agent will be effective over a wider area of cord. The volume of spinal fluid remains the same but the concentration of agent increases, thus blocking sensory fibers to a higher level and reducing the number of unblocked pain fibers. A graph showing the distribution of level of pin-prick analgesia in patients with and without pain after 60 minutes of tourniquet inflation might be instructive here. Surprisingly enough, their incidence of tourniquet pain, even following an increase in dosage, was still one out of three patients. Had they obtained cutaneous analgesia to the first thoracic level, then I believe their incidence of pain would have been more dramatically reduced. The undesirable total sympathetic block following such high levels would seem, to me at least, to be an unnecessarily high price to pay for complete comfort of the patient.

From personal experience in the Army Medical Service, with a patient population similar to that of Egbert and Deas, and in the University of California service, with an older patient distribution, we have not seen the high incidence (63.6 per cent) of tourniquet pain reported; even though we ordinarily use smaller doses of agents (10 mg. of tetracaine or less) and a small volume of solution in an attempt to limit analgesia to the tenth thoracic level or below. When we do encounter tourniquet pain we manage to treat it successfully with additional analgesic drugs or nitrous oxide inhalation.

Finally, I would like to emphasize the relative rarity of tourniquet pain if the nerve trunks themselves are blocked (*e.g.*, sciatic-femoral or brachial plexus block). This is probably because the nerves are interrupted distal to the sympathetic trunks and spinal cord, so that the entire afferent content of the extremity is blocked, rather than only the spinal component.

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

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