# CORRESPONDENCE

## Solubility of Diethyl Ether

*To the Editor.*—In reviewing the literature prior to our recent studies on ether solubility (ANESTHESIOLOGY 24: 676, 1963), we found no reference to such solubility other than the classic papers of Haggard (J. Biol. Chem. 55: 131, 1923) and of Shaffer and Ronzoni (J. Biol. Chem. 57: 741, 1923) both of which gave blood/gas partition coefficents of about 15. Dr. C. P. Larson has called to my attention two other papers (Jones *et al.*: ANESTHESIOLOGY 14: 490, 1953 and Hattox *et al.*: ANESTHESIOLOGY 14: 584, 1953) on the same subject. These investigators using the mass spectrometer found a partition coefficient of 12 which is confirmed by our work. Recently, Lowe (personal communication) found a coefficient of 10.5 to 11 with flame ionization after separation of gas chromatography. These more recent findings of coefficients of 11 or 12 are obtained with a variety of techniques and, I believe, substantiate these values as opposed to the older one of 15.

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## Tourniquet Pain

*To the Editor.*—A recent paper in ANESTHESIOLOGY, "Theoretical aspects of pain: bizarre pain phenomena during low spinal anesthesia," by Drs. de Jong and Cullen (Sept.–Oct. 1963, pages 628–635) challenge the interpretation of data which Dr. Deas and I published last year (ANESTHESIOLOGY 23: 287, 1962). Dr. de Jong also criticized our conclusions previously (Current Comment: ANESTHESIOLOGY 23: 881, 1962). I must comment upon these two critiques.

They mention our high incidence of tourniquet pain, quoting 63.7 per cent incidence which we reduced to 33.3 per cent by increasing the dose of tetracaine in the spinal injection. As they suggest, the incidence is lower than either of these figures; in our own experience in the Navy, we promptly lowered the incidence by adding epinephrine, by administering doses of tetracaine more proportional to the heights of the patients, and by keeping the level of the spinal around tenth thoracic. Nevertheless, the reason we did the study was that the incidence was appreciable; we cannot consider a phenomenon rare simply because no one has recorded its incidence. Every anesthetist who has given spinal anesthesia has seen this.

Drs. de Jong and Cullen believe that the pain is carried over small fibers which travel "around" the area of spinal block; this of course is the opposite of our theory, namely, that tourniquet pain goes "through" the block carried by fibers which are large and therefore become excitable before pin-prick sensation returns. Certainly, Dr. Deas and I did not prove that the sensation which the patients called pain went through the spinal. But one must note that our patients, at the time they perceived pain, still had analgesia to pin-prick to fifth thoracic level. We are forced to conclude either: (1) that touniquet pain enters the cord below fifth thoracic level upon nerve fibers larger than those transmitting pin-prick, or, (2) that it enters above fifth thoracic level on smaller fibers.

No one has demonstrated the existence of nerve fibers travelling along the sympathetic chain from the legs to enter the cord above fifth thoracic level. Kuntz (South. Med. J. 44: 673, 1951) was only able to find fibers from the legs entering the cord in the lower thoracic and lumbar regions. However, Gasser (Proc. Assoc. Res. Nerv. Ment. Dis. 23: 44, 1943) has reported that fast pain resists cocainization more than slow pain. Also, Arrowood and Sarnoff (ANESTHESIOLOGY 9: 614, 1948) have demonstrated convincingly the relief of pain other than pin-prick type by increasing the dose of local anesthetic. We have merely chosen the theory best supported by data. We do not deny that pain is transmitted through the sympathetic chain, but no one has shown that it travels so far.

If our patients who developed pain had

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.

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*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]

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REFERENCES

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2. de Jong, R. H., and Wagman, I. H.: Physiological mechanisms of peripheral nerve block by local anesthetics, ANESTHESIOLOGY 24: 684, 1963.