LETTERS TO THE EDITOR

The City of New York,
Department of Hospitals,
Bronx Municipal Hospital Center,
October 3, 1962

SIR:

I think it is important to clarify some of the discussion in the article "A Hazard of Epidural Anaesthesia in Obstetrics" which appeared in the September issue of your journal. The author postulates that the explanation for the extremely high epidural block he obtained using a standard technique and recommended dose of anaesthetic agent was due to the increased cerebrospinal fluid pressure coincident with *painless* uterine contraction and the increased amount of blood distending the epidural space and diminishing the volume of the epidural space itself. Both Vasicka, Kretchner, and Lawas[1] and Marx, Zemaitis and Orkin[2] have shown, using slightly different techniques, that the cerebrospinal fluid pressure does *not* rise with a painless uterine contraction, the rise being dependent upon the straining associated with pain or the bearing-down effort. Marx, Zemaitis, and Orkin also pointed out that the amount of blood shunted to the anastomatic venous channels in the epidural space by the inferior vena caval obstruction of a large uterus must be insignificant because the cerebrospinal fluid pressure did not decrease rapidly after delivery and uterine decompression. Since the test dose abolished the pain and presumably the bearing-down of the patient, the injection of the main dose of the local anaesthetic during a painless contraction could not account for the level of anaesthesia becoming high. In reality, instead of being a reason for omitting the test dose, the abolition of the pain of uterine contraction with consequent reduction of the risk of increasing level is just another reason that the test dose should not be omitted.

The reason for the unusually high level of anaesthesia in this case may be found by exploring another possibility. Moore and associates[3] and later Mostert[4] have shown that a local anaesthetic can sometimes be given as easily intraneurally as intravenously. The solution will then spread centrally to the cord and then to the cerebrospinal fluid creating clinically the picture of spinal anaesthesia. This mechanism has been used to explain late onset (15–40 min.) of high spinal anaesthesia following epidural block. It may be the mechanism in the case under discussion.

1. VASICKA, A.; KRETCHNER, H.; & LAWAS, F. Cerebrospinal Fluid Pressures during Labor. Am. J. Obstet. & Gynecol. *84*: 207 (1962).
2. MARX, G. F.; ZEMAITIS, M. T.; & ORKIN, L. R. Cerebrospinal Fluid Pressures during Labor and Obstetrical Anesthesia. Anesthesiology *22*: 348 (1961).
3. MOORE, D. C.; HAIN, R. F.; WARD, A.; & BRIDENBAUGH, L. D. The Importance of the Perineural Spaces in Nerve Blocking. J.A.M.A. *156*: 1050 (1954).
4. MOSTERT, J. W. Unintentional Spread of Epidural Analgesia. Brit. J. Anaesth. *32*: 334 (1960).

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The University of Leeds,
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Leeds, England,
October 18, 1962

SIR:

Thank you for the opportunity of replying to Dr. Edelist's letter [above]. The basis of the postulated explanation of the high spread of epidural anaesthesia in the case described was that uterine contractions may cause an influx of blood into the epidural veins, thus diminishing the volume of epidural space available to accommodate injected fluid, and perhaps diminishing the patency of the outlets. Bromage's observations on epidural pressures in labour tend to support this supposition.[1,2]

The relationship between cerebrospinal fluid pressure and uterine contractions is less directly pertinent to considerations of epidural anaesthesia. The cerebrospinal fluid pressure presumably tends to remain constant under physiological conditions and is regulated by a balance between formation and absorption, most of which occurs in the cranium. It varies directly with general venous pressure, being raised by forced expiration against the closed glottis in the expulsive reflex of the second stage of labour. The effect of local variations in venous pressure such as may occur in the epidural veins during uterine contractions is less well defined. Thus it is quite possible that a tendency to change in cerebrospinal fluid pressure secondary to alterations in vertebral haemodynamics may be compensated by a variation in the rate of absorption or formation of cerebrospinal fluid in the cranium. Furthermore, the epidural veins are thin-walled structures which may accommodate a relatively large volume of blood before distension produces appreciable pressure changes.

I cannot accept the suggestion that the case was one of high spinal block. Consciousness and touch sensibility were retained, voluntary muscles were not totally paralysed, and there were no sequelae. It is difficult to visualize the tip of a blunt No. 16 Tuohy needle introduced in the sagittal plane in the mid-line posteriorly entering a nerve root.

1. BROMAGE, P. R. Continuous Lumbar Epidural Anaesthesia in Obstetrics. Canad. M.A.J. *85*: 1136 (1961).
2. ——— Spread of Analgesic Solutions in the Epidural Space and Their Site of Action: A Statistical Study. Brit. J. Anaesth. *34*: 161 (1962).

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