LOWER LIMB REFLEX CHANGES IN SEGMENTAL EPIDURAL ANALGESIA

P. R. BROJAGE

SUMMARY

Sensory changes and lower limb reflexes were observed in 35 patients receiving segmental epidural analgesia in the mid-thoracic region. Sensory blockade was confined to the thoracic and upper lumbar segments. The lower limb reflexes changed to an "upper motor neurone" pattern, with the onset and development of segmental blockade. The sequence of changes observed was hypertonic tendon reflexes, then the onset of ankle clonus, and finally the plantar response changed to a positive Babinski sign. With regression of analgesia the reflexes returned to normal in the reverse order.

The significance of these findings is discussed in relation to the anatomy of descending spinal pathways, and to the pattern of penetration of local anaesthetics into the substance of the spinal cord.

Corning's original attempts at spinal analgesia in 1885 were based on the concept that medications injected close to the cord would be taken up by the substance of the spinal cord (Corning, 1885). Subsequent clinical and laboratory data discredited Corning's hypothesis, and led to the idea that subarachnoid and epidural analgesia acted primarily upon elements of the peripheral nervous system, such as spinal roots, ganglia and nerve trunks, and not upon the neuraxis itself. However, animal experiments with radioactive tracers have shown that injected local anaesthetic agents can be demonstrated in the peripheral parts of the spinal cord after both epidural and subarachnoid injection (Bromage, Joyal and Binney, 1963; Cohen, 1968). While similar direct experiments are not feasible in humans, indirect evidence of blockade of major descending pathways within the cord should be demonstrable by suitable neurological tests.

Segmental epidural analgesia confined to the cervical or thoracic regions presents the interesting possibility that the effects of any spinal cord involvement might be observed in unanaesthetized segments remote from the area of blockade. Thoracic blockade of descending pathways would then produce characteristic reflex changes in the legs, below the lowest limits of sensory anaesthesia. This paper is concerned with changes of lower limb reflexes that have been observed after segmental thoracic epidural block, and that strongly suggest partial interruption of long descending pathways. These observations were part of a larger enquiry into the clinical qualities of a new local anaesthetic agent, etidocaine (2-(N-Ethylpropylamino)-2',6'-butyroxylidide hydrochloride) a drug with unusually high lipid solubility (Lund, Cwik and Pagdanganan, 1973).

METHODS

Lower limb reflexes were observed in 35 patients aged between 23 and 87 years (average: 49 years) undergoing abdominal surgery after segmental epidural analgesia induced at the 6th or 7th thoracic interspace. All the patients in this series were free from neurological disease. Epidural puncture was performed using an 18-gauge thin-walled Bromage needle, with the patient in the sitting position. The epidural space was identified by the hanging-drop sign, and the calculated dose of local anaesthetic was injected through the needle in a single bolus. The following solutions were used: 1% etidocaine (30 patients), 2% lignocaine hydrochloride (3 patients), 2% CO₂-lignocaine (2 patients). All solutions contained adrenaline (1:200,000). The dose was calculated in relation to age, and was approximately 40% less than the calculated dose for administration at the 2nd lumbar interspace (Bromage, 1969a). The doses used were inversely proportional to age, and varied between 40 mg and 160 mg for etidocaine, and 120 mg and 220 mg for lignocaine.

A vinyl plastic catheter (external diameter 1 mm)
was passed until a length of 4 cm lay within the epidural space, and the needle was withdrawn, and the catheter taped in place. The patient was then placed in the supine position.

The onset of cutaneous analgesia, and its subsequent regression were determined by pinprick, and the area of analgesia plotted against time in a segment-time diagram. Subsequently, blockade was allowed to regress for a few segments before being reinstated by a reinforcing dose of local anaesthetic solution injected through the epidural catheter. The reinforcing dose was half the initial induction dose.

The following reflexes were tested in each limb: (1) Knee jerk. (2) Ankle jerk. Tendon reflexes were scored on a 4-point scale of absent, 0; normal, 1; brisk, 2; and very brisk, 3. (3) Ankle clonus. Ankle clonus was scored as the number of tonic beats following sharp passive dorsiflexion of the ankle. More than 20 beats was scored as "sustained clonus". (4) Plantar reflex, elicited as Babinski, or Oppenheim reflex. Observations were made in the conscious patient before induction of epidural analgesia, and at frequent intervals for 20–45 min after induction of blockade, and then for a period of 2–6 hours in the postoperative period. All patients received light thiopentone and nitrous oxide anaesthesia during the operation, in addition to the epidural block, and so observations of reflex changes were discounted during the 1st hour after operation, in view of the emergence pattern of reflex change that lasts for 20–40 min after general anaesthesia (Soliman and Gillies, 1972). The lower limb reflexes were tested again on the following day, 24 hours after regression of anaesthesia.

RESULTS

Figure 1 shows the distribution of upper and lower levels of analgesia to pinprick in the 35 patients. Motor power and sensation to touch and pinprick were normal in all segments below the lower limit of segmental analgesia. The knee jerks tended to be inhibited in all instances where the lower level of blockade extended caudally beyond the first lumbar dermatome; in these cases the tendon reflexes were scored after the sensory block had receded to the groin.

The lower limb reflexes began to show signs of upper motor neurone suppression within 10–15 min of epidural injection. The intensity of this change was usually symmetrical, but occasionally one leg would show more profound changes than the other. In some instances sustained clonus and an upgoing toe were present in one leg while reflexes remained normal in the other.

Increased briskness of the knee and ankle jerks were usually the first signs to appear, except in those patients where the lower level of blockade spread rapidly downwards to involve the 2nd and 3rd lumbar segments, when the knee jerks became obtunded at an early stage. Occasionally this was followed by the onset of ankle clonus a few minutes later. The incidence and intensity of clonus is shown in figure 2, and it can be seen that this sign was
often absent. Even when present it was only mildly developed, except in three patients in whom sustained clonus was obtained.

The plantar reflexes were the last to change, and the toes became upgoing about 20 min or longer after the induction of epidural block. In 6 patients the plantar reflexes did not change after the induction dose, but they did become upgoing after the second or reinforcing epidural dose given 2-3 hours later. In all 35 patients the reflexes had returned to normal on the following day.

DISCUSSION

The results of this study demonstrate that certain descending pathways in the spinal cord are reversibly affected by segmental epidural analgesia. The sequence of reflex changes described in this series is a mirror-image of the pattern seen on emergence from general anaesthesia uncomplicated by the addition of muscle relaxants. The first sign on emergence from general anaesthesia and areflexia is a positive Babinski sign, followed shortly after by clonus and exaggerated tendon reflexes. The positive Babinski then progresses to a normal downgoing toe in 5-10 min but a condition of hyperreflexia persists for 15-25 min and then gradually subsides (Soliman and Gillies, 1972).

The pattern of reflex emergence after general anaesthesia is thought to be related to events occurring at intracranial levels, and acting upon inhibitory pathways restraining corticospinal impulses. In segmental epidural blockade the reflex changes could conceivably be the result of local anaesthetic absorbed by the blood stream in sufficient amounts to cause a degree of cerebral depression comparable to that seen on emergence from general anaesthesia. However, this explanation seems improbable, since the dosage of local anaesthetic was small, and insufficient to cause any signs of central depression or impairment of alertness and intellectual activity. Although blood concentrations of local anaesthetic were not estimated in this investigation, published figures from other studies strongly suggest that the doses used could not have caused significant concentrations in the blood (Bromage and Robson, 1961; Lund, Cwik and Pagdanganan, 1973).

Thus, it seems reasonable to assume that the reflex changes observed were the result of events at a spinal site, and probably within the segmental area of blockade. The question arises, what sites in the cord are involved, and why does the progression of reflex changes so closely resemble the reverse of the pattern seen after general anaesthesia?

Radioassay studies of cord, spinal roots and meninges in dogs have indicated that epidural analgesia produces the highest concentrations of local anaesthetic in spinal roots and pia-arachnoid, but in addition significant amounts of local anaesthetic enter the substance of the cord itself. Autoradiographs show that distribution of local anaesthetic is in the peripheral parts of the cord, and penetration does not extend very far into the deeper layers (Bromage, Joyal and Binney, 1963). Nevertheless, penetration is probably deep enough to affect a significant proportion of ascending and descending tracts. Therefore, the pathways involved in the observed lower limb reflex changes probably are relatively close to the surface of the cord, where they are likely to be included in the peripheral field of local anaesthetic diffusing inwards from the perimeter.

Identification of the long pathways implicated in the observed reflex changes can only be speculative at the present time. Lower limb reflexes might be affected by conduction changes in either ascending or descending pathways, or both. Interruption of ascending pathways may modulate distant segmental reflexes indirectly by long rostral circuits affecting corticospinal or propriospinal pathways. On the motor side, two major and separate descending pathways terminate in the grey matter of the cord (fig. 3). First, a medial system originating in the brain stem (the ventromedial subcortical spinal pathway), and running downwards close to the surface.

Fig. 3. Descending pathways in the spinal cord, and their termination on Rexed's laminae. After Nyberg-Hansen (1965, 1969), and Kuypers (1964). Pyr=Pyramidal tract; MR-Sp=Medullary reticulospinal fibres; PRSp=Pontine reticulospinal fibres; VST=Vestibulospinal tract; AFP=Anterior funicular pyramid.
of the anterior median fissure to terminate in the
dorsomedial and adjacent parts of the intermediate
zone of the spinal grey matter, that is Rexed's
laminae VIII and IX. This comprises the pontine
reticulospinal fibres, and the vestibulospinal tract
and the anterior funicular pyramidal fibres. Second,
a lateral system consisting of the corticospinal and
medullary reticulospinal fibres running relatively
deeply in the lateral white matter to terminate on
cells in the lateral and dorsal parts of the inter-
mediate zone of the spinal grey matter, that is
Rexed's laminae V, VI, and VII (Nyberg-Hansen,
1965, 1969). In addition, some rubrospinal fibres
lie relatively superficially in the lateral white matter
of the lower thoracic cord (Kuypers, 1964).

The most superficial, and most vulnerable would
seem to be the reticulospinal fibres, the vestibulo-
spinal tract, and the anterior direct pyramidal tract
lying close to the anterior median fissure in the
sulcomarginal zone, together with the superficial
fibres of the lateral corticospinal and rubrospinal
tracts in the lateral columns (fig. 3). Wall has shown
that the crossed pyramidal tract in the dorso-lateral
funiculus of the cat is markedly affected by reversible
cold block applied to the surface of the cord
(Wall, 1967). It is possible that some of the more
superficial of these fibres in man are also affected
by local anaesthetics diffusing inwards from a
segmental epidural block.

It is tempting to suggest that the observed
sequence of reflex change, hyperreflexia—clonus—
positive Babinski, is the consequence of blockade of
progressively deeper layers of descending fibres. If
this is so then it is suggestive that superficial fibres
in the sulco-marginal fasciculus may be largely con-
cerned with the earlier hyperreflexia and clonus,
while the lateral and deeper pyramidal fibres are
cconcerned with the positive Babinski that appears
later in the sequence of reflex changes.

This simple concept of inward diffusion affecting
fibre tracts sequentially at increasing depth is sup-
ported by the author's original tracer studies and
autoradiographs that suggested a relatively sym-
metrical pattern of peripheral uptake from the sur-
face of the cord (Bromage, Joyal and Binney, 1963).
However, this view may be unduly naive in the
light of Cohen's subsequent work on the quantita-
tive regional distribution of local anaesthetics in the
cord after subarachnoid injection. Cohen found sub-
stantial differences in the concentration of local
anaesthetic in different parts of the spinal white
matter, with about twice as much drug present in
the dorsal and lateral columns as in the anterior
columns. He has suggested that this uneven distri-
bution may be the result of different lipid charac-
teristics and local anaesthetic affinities in the various
regions of the cord (Cohen, 1968).

There is a need to extend both clinical observa-
tions and Cohen's quantitative analyses to determine
whether a similar pattern of selective uptake of local
anaesthetic occurs within the cord after epidural
injection. The emergence of a coherent chart of
selective affinities within the spinal cord would be
of importance in designing appropriate anaesthetic
molecules for selective inhibition of spinal pathways,
and for specific clinical tasks. Such a chart of
neuraxial local anaesthetic affinities might throw
light on some presently unexplained paradoxes of
performance in epidural blockade. For example,
certain local anaesthetics show an unexplained lack
of correlation between the intensity of motor block
and the quality of sensory anaesthesia that they
provide. Thus, tetracaine produces a more profound
degree of motor blockade than bupivacaine, and yet
the quality of sensory analgesia produced by bupi-
vacaine is superior (Bromage, 1969b). Recent studies
with etidocaine show that etidocaine is somewhat
similar to tetracaine in this respect (Bromage and
Datta, 1974). This dichotomy between the
quality of motor and sensory performance is not
explicable in terms of neurophysiological considera-
tions of fibre size within the nerves and nerve roots.
However, these differences of motor and sensory
performance might be explained by specific patterns
of regional affinities and local anaesthetic distribu-
tion within the architecture of the cord. After the
passage of nearly a century, the process of investi-
gation would then come a little closer to Coming's
original concept of intraspinal medication and spinal
anaesthesia proposed in his paper entitled "Spinal
anaesthesia and local medication of the cord"
(Coming, 1885).

ACKNOWLEDGEMENTS

The author wishes to thank Miss Lorna Dunford, R.N.,
for technical assistance. Supplies of etidocaine were
provided by Astra Chemicals Ltd (Canada). The work was
partially supported by M.R.C. Canada, Grant No.
MA.1008.

REFERENCES

Bromage, P. R. (1969a). Ageing and epidural dose re-
—— (1969b). A comparison of bupivacaine and tetracaine
J., 16, 37.
—— Datta, S. (awaiting publication). Clinical evaluation
of etidocaine for epidural analgesia in obstetrics.


---

**NORTH OF ENGLAND SOCIETY OF ANAESTHETISTS**

1974–75

**President:** Dr. Larry B. Dunkin

**PROGRAMME**

1974

**FRIDAY, OCTOBER 11.** President’s Night.

**FRIDAY, NOVEMBER 8.** Dr J. G. Whitwam, Reader in Anaesthesia, Royal Postgraduate Medical School, London: “Acute Cardiovascular Compensation”.

**FRIDAY, DECEMBER 13.** Professor Reginald Hall, Professor of Medicine, University of Newcastle upon Tyne: “Endocrine Disease in Relation to Anaesthesia”.

1975

**FRIDAY, APRIL 11.** Dr J. C. Stoddart, Consultant Anaesthetist, Royal Victoria Infirmary, Newcastle upon Tyne: “Lessons from Intensive Care”.

**FRIDAY, MAY 9.** Dr Cedric Prys-Roberts, Senior Lecturer in Anaesthesia, Nuffield Department of Anaesthetics, University of Oxford: “Hypertension, Ischaemic Heart Disease and Anaesthesia”.

Arrangements are in hand with the University Department of Anaesthesia for a one-day Symposium on “Teaching and Training in Anaesthesia in Britain and the E.E.C.” on Saturday, March 22, 1975.

Meetings are normally held in the New Lecture Theatre, R.V.I., Newcastle upon Tyne, at 8 p.m. Buffet suppers will be available in the Board Room from 6.30 to 7.30 p.m. and coffee in the ante-room to the New Lecture Theatre from 7.30 to 8 p.m.

All communications should be addressed to the Honorary Secretary: Dr C. J. Hull, Department of Anaesthesia, Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP.