THE EFFECTS OF ADDING ADRENALINE TO ETIDOCAINE AND LIGNOCAINE IN EXTRADURAL ANAESTHESIA I: BLOCK CHARACTERISTICS AND CARDIOVASCULAR EFFECTS

T. M. MURPHY, L. E. MATHER, M. D’A. STANTON-HICKS, J. J. BONICA AND G. T. TUCKER

SUMMARY

The addition of adrenaline 5 μg/ml, 1 : 200 000 to 1% etidocaine hydrochloride administered extradurally (L2–3) shortened significantly the onset time for sensory blockade, particularly with respect to the spread of the analgesia from the injection site, and shortened the already rapid onset of motor block. Etidocaine hydrochloride 1% plain caused a slower onset of block, lasted longer and produced more profound analgesia over the caudal dermatomes than did 2% lignocaine hydrochloride. The motor block from plain etidocaine was more profound in its extent and lasted longer than that caused by lignocaine. With regard to cardiovascular variables, there were no significant differences between subjects receiving the plain etidocaine and the plain lignocaine. However, subjects receiving etidocaine with adrenaline exhibited increased cardiac stimulation and a decrease in total peripheral resistance over the first 150 min.

Since adrenaline was added to local anaesthetic agents 50 years ago (Braun, 1914), its effects upon the pharmacology and physiology of regional anaesthesia have been studied widely. The advantages and disadvantages of its addition to those local anaesthetic agents with an intermediate time duration, such as lignocaine, have been described both with regard to its influence on the distribution of the block (Bromage et al., 1964) and on the cardiovascular sequelae (Bonica et al., 1971). The effect of the addition of adrenaline to the longer-acting local anaesthetic agents has yet to be established. This study was undertaken to determine the effects of adding adrenaline 5 μg/ml, 1 : 200 000 to solutions of etidocaine hydrochloride (Duranest) 1%, in a controlled crossover study of extradural anaesthesia in human volunteers. These results were compared with extradural anaesthesia using lignocaine hydrochloride 2% in a separate group of subjects matched for height and weight.

METHODS

Informed consent was obtained from five healthy male volunteers between the ages of 21 and 33 yr. The subjects were unpremedicated and had fasted for 8 h before the commencement of the study. Under procaine local anaesthesia in the antecubital fossa, catheters were introduced into the brachial artery and, via the basilic vein, into the central venous conduit, for the measurement of cardiac output, and arterial and central venous pressures. An extradural catheter was placed via a paramedian approach at the L2–3 interspace and the catheter tip was advanced to the level of L2. Inflatable cuffs were placed for the measurement of limb blood flow by venous occlusive plethysmography using a silastic and mercury strain gauge. After a 30-min rest period, the arterial pressure, cardiac output (by dye dilution), heart rate, central venous pressure, and upper and lower limb blood flow were recorded. The stroke volume, total peripheral resistance, stroke work and minute work were calculated from the primary measurements as described previously (Bonica et al., 1971). The subjects received 20 ml of either 1% etidocaine HCl with 5 μg/ml adrenaline or 1% etidocaine HCl plain solution via the extradural catheter. The other solution was given on the subsequent study (with a 2-week interval between studies).
The progress of the sensory block was assessed by measuring the onset time which was the time when analgesia could be detected first at any dermatomal level, the spread of the agent (the time at which four segments had been blocked, and the times required for complete rostral and complete caudal spread from the injection site at L2), and the maximum number of dermatomes blocked (with regard to both total number of dermatomes and also those dermatomes blocked above the injection site at L2 and those dermatomes blocked below the injection site at L2). The duration of the sensory block was assessed as the time for two segment regression to occur and the time for complete return of all sensation. The motor characteristics of the block were assessed by noting the time for the onset of motor blockade and the time to reach maximal motor blockade. The intensity of motor blockade was assessed on the scale described by Bromage and others, (1964) and the duration of motor blockade was assessed by noting the time at which regression commenced from the maximum intensity block, and for complete return of motor function. The cardiovascular data were recorded before the injection of the local anaesthetic agent and throughout the study. Arterial Po2, Pco2 and pH were monitored also. To compare the effects of etidocaine with a standard local anaesthetic drug, a separate group of five volunteers, matched for age, height and weight, was studied in a similar procedure using 2% lignocaine HCl 20 ml, with and without the addition of adrenaline 5 μg/ml, 1 : 200,000, and injected at the same extradural level.

The data were analysed statistically using Student’s t test for paired data.

RESULTS

The characteristics of the extradural anaesthesia produced by the agents in the study are summarized in table I and figure 1.

Characteristics of nerve block

When adrenaline was added to etidocaine there was a significant decrease in the time of onset of analgesia. Although there appeared to be an earlier spread of the solution containing adrenaline, the complete upward spread of the block (the spread in a rostral direction from the point of injection at L2) was not significantly different for the two preparations. However, the complete caudal spread downward from L2 was significantly longer with the plain solution (P<0.001). Previous authors (Bromage et al., 1964; Bridenbaugh, et al., 1973; Lund, Cwik and Pagdanganan, 1973) have taken two-

### Table I. Comparison of extradural anaesthesia with plain and adrenaline (1 : 200,000) containing solutions of 1% etidocaine HCl and 2% lignocaine HCl. Columns (2), (4) and (6) indicate the probability values for the significance of differences between data in immediately adjacent columns. Mean values ± SEM

<table>
<thead>
<tr>
<th>Sensory</th>
<th>1% Etidocaine solutions</th>
<th>2% Lignocaine solutions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>With adrenaline</td>
<td>P (paired)</td>
</tr>
<tr>
<td>Onset time (min)</td>
<td>5 ± 3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Four-segment spread (min)</td>
<td>8 ± 3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Complete rostral spread (min)</td>
<td>23 ± 4</td>
<td>n.s.</td>
</tr>
<tr>
<td>Complete caudal spread (min)</td>
<td>15 ± 9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Two segment regression (min)</td>
<td>113 ± 20</td>
<td>n.s.</td>
</tr>
<tr>
<td>Max. dermatomes blocked</td>
<td>16 ± 4</td>
<td>n.s.</td>
</tr>
<tr>
<td>Anaesthetic dermatomes above L2</td>
<td>10 ± 3</td>
<td>n.s.</td>
</tr>
<tr>
<td>Anaesthetic dermatomes below L2</td>
<td>6 ± 3</td>
<td>n.s.</td>
</tr>
<tr>
<td>Complete return (min)</td>
<td>326 ± 30</td>
<td>n.s.</td>
</tr>
</tbody>
</table>

Motor

| Onset (min) | 4 ± 1 | <0.01 | 13 ± 5 | n.s. | 24 ± 21 | n.s. | 11 ± 4 |
| Complete (min) | 18 ± 10 | n.s. | 32 ± 14 | n.s. | 29 ± 17 | n.s. | 27 ± 11 |
| Max. intensity (0–3 scale) | 2.8 ± 0.4 | n.s. | 2.4 ± 0.9 | <0.05 | 1 ± 0.2 | <0.01 | 2.6 ± 0.5 |
| Regression (min) | 186 ± 11 | n.s. | 148 ± 64 | n.s. | 74 ± 32 | <0.005 | 122 ± 20 |
| Complete return (min) | 298 ± 59 | n.s. | 244 ± 30 | <0.001 | 98 ± 31 | <0.001 | 184 ± 30 |
EFFECT OF ADRENALINE IN EXTRADURAL BLOCK

Comparison of lignocaine with and without adrenaline. A significantly greater number of segments was blocked with the adrenaline-containing solution. Although the rostral spread of lignocaine seemed relatively uninfluenced by the addition of adrenaline, the caudal spread was affected markedly, yielding a caudal spread of only two segments with the plain solution compared with eight segments with the adrenaline-containing solution. There was a greater length of time until two-segment regression of block, and a more profound and longer-lasting motor blockade, with the adrenaline-containing solution.

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Etidocaine with adrenaline v. etidocaine without adrenaline. The cardiovascular data are shown in figure 2. The adrenaline-containing solution caused significantly larger values of cardiac output during the

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first 150 min of the block. There was a corresponding decrease in the total peripheral resistance for the first 120 min of the block. There were other statistically significant differences in heart rate, stroke volume, stroke work and minute work (fig. 2).

There were no significant differences in cardiovascular variables when the etidocaine plain was compared with the lignocaine plain solution.

There were no significant changes in the blood-gas variables measured throughout the studies.

Sympathetic vaso-motor blockade. As indicated by a change in the temperature of the great toe, the duration of the sympathetic blockade following etidocaine was longer than that following lignocaine.

DISCUSSION

Bridenbaugh and others (1974) stated that the addition of adrenaline to etidocaine may provide some protection against systemic toxicity, although it has little benefit, if any, on the clinical effects of the drug. In our studies, the onset and initial spread of the agents were significantly shorter with the adrenaline-containing solutions and this was particularly so for the complete caudal spread of the agent (caudal spread being easier to monitor in the volunteer than in the patient). We speculate that the local anaesthetic agent spreads to the same extent in the extradural space, with or without adrenaline, but the vasoconstrictive effect of the adrenaline maintains the drug in closer proximity to the nerves for a greater period of time, enhancing the local anaesthetic effect. Lund (1974) stated that recovery from extradural block by etidocaine occurred in a linear rather than a segmental pattern. Our findings do not support this, since the recovery was clearly segmental.

The traditional view is that the sympathetic block outlasts the somatic block (Greene, 1969), but Lund (1974) has mentioned that the prolonged duration of sensory analgesia following etidocaine is not necessarily accompanied by an equally prolonged duration of autonomic blockade. We found this to be so in the case of the plain etidocaine solution where the complete sensory blockade lasted for 301 (± SD 44) min, whereas the sympathetic vasomotor block, as indicated by increased toe temperature, had dissipated earlier (190 ± 66 min). This was noted also for lignocaine where the complete return of sensation occurred at 132 ± 18 min, whereas the sympathetic blockade had dissipated at 81 ± 37 min. Further, in the lignocaine studies in which we measured psychogalvanic reflexes, the psychogalvanic reflex had returned at 65 ± 22 min, lasting only half as long as the sensory blockade. Further details have been presented elsewhere (Murphy et al., 1975). In contrast to the present report, Bridenbaugh and others (1974) reported no differences attributable to adrenaline, except in the onset of motor blockade. However, they studied premedicated patients in an operating-room setting, compared with our unpremedicated volunteers in the laboratory.

Perhaps our most significant finding was the influence of the addition of adrenaline on the caudal spread of the anaesthesia. With etidocaine, this was manifested in the decreased time to reach complete caudal spread compared with the plain solution. However, with lignocaine not only was caudal spread longer in occurring with the plain solution, but it was less complete than when adrenaline was included. The average caudal spread of anaesthesia with lignocaine was limited to two dermatomes below the L2 injection site, but when adrenaline was included, an average spread of eight dermatomes below the injection site was achieved. “Hypoaesthesia” over the caudal dermatomes occurred for a variable period of time in all subjects who received plain solutions of lignocaine. We speculate that the lignocaine solution spreads caudally in the extradural space but does not cause effective analgesia, unless adrenaline is added, because it is removed by the circulation before effective concentrations are present at the site of action. This brief exposure of the nerve elements in the extradural space to lignocaine is probably sufficient to block the relatively small thoracic nerves, but is insufficient for the, much larger, lower lumbar and upper sacral nerves (Galindo et al., 1975).

With regard to the cardiovascular changes, the beta-adrenergic effects of the adrenaline which is absorbed from the injectate were predominant. These resulted in cardiac stimulation over the first 150 min as indicated by changes in the cardiac output, heart rate, and cardiac minute work, and also in peripheral vasodilatation. In the absence of adrenaline no significant differences between the two local anaesthetic agents could be detected.

We concluded, therefore, that the addition of adrenaline to 1% etidocaine solution will speed the onset of motor block and the onset of analgesia, and increase the caudal spread. When plain solutions of 1% etidocaine and 2% lignocaine were compared, lignocaine had a faster onset and caudal spread, but the caudal spread was less complete. The useful block for surgery (two-dermatome regression) and the time for complete recovery lasted longer with etidocaine.
EFFECT OF ADRENALINE IN EXTRADURAL BLOCK

The incomplete caudal spread of analgesia with plain lignocaine was surprising, and has not been emphasized by previous authors. It is much easier to test the progress of analgesia in lumbar and sacral dermatomes in experimental subjects than in patients who are undergoing surgery and who may be sedated. This is important clinically because it may explain many of the "inadequate blocks" of lower extremities when a lumbar extradural approach is used. An early diminished sensation to pinprick in the caudal dermatomes may give false hopes of a more profound block to follow.

Part II of this study, entitled "Pharmacokinetics", will be published in the October 1976 issue.

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REFERENCES


EFFETS DE L'ADDITION D'ADRENALINE A L'ETIDOCAINE ET A LA LIGNOCAINE DANS LES ANESTHESIES EXTRADURALES I: CARACTERISTIQUES DU BLOCAGE ET EFFETS CARDIOVASCULAIRES

RESUME

L'addition d'adrénaline 5 µg/ml, 1 : 200 000, à une solution de chlorhydrate d'étidocaine à 1% administrée extraduralment (L2-3) a raccourci d'une manière significative le temps d'atteinte nécessaire pour le commencement du blocage sensoriel, particulièrement en ce qui concerne la dissémination de l'analgesie à partir du point d'injection et elle a raccourci également le commencement, déjà rapide, du blocage moteur. Le chlorhydrate d'étidocaine à 1% sans adrénaline, a provoqué un commencement de blocage plus lent, qui a duré plus longtemps et entraîné une analgesie plus profonde des dermatomes caudaux que la chlorhydrate de lignocaine à 2%. Le blocage moteur obtenu à partir d'étidocaine pure a été plus profond dans sa portée et a duré plus longtemps que celui provoqué par la lignocaine. En ce qui concerne les éléments cardiovasculaires variables, il n'y a eu aucune différence notable entre les sujets recevant l'étidocaine pure et ceux recevant la lignocaine pure. Néanmoins, les sujets auxquels on a administré l'étidocaine additionnée d'adrénaline ont accusé une stimulation cardiaque plus forte pendant les 150 premières minutes, ainsi qu'une diminution de la résistance périphérique totale.

DIE WIRKUNGEN EINER ZUGABE VON ADRENalin ZU ETIDOCAIN UND LIGNOCAIN BEI EXTRADURALER NARKOSE. I: BLOCKIERUNGSEIGENSCHAFTEN UND KARDIOVASCULARE WIRKUNGEN

ZUSAMMENFASSUNG

Die Zugabe von 5 µg/ml, 1 : 200 000 Adrenalin zu 1% Etidocain Hydrochlorid, extradural gegeben (L2-3), verkürzte wesentlich die Zeit des Einsetzens der sensorischen Blockierung, besonders im Hinblick auf die Verbreitung der Analgesie von der Injektionsstelle aus, und verkürzte auch das bereits sehr schnelle Einsetzen der motorischen Blockierung. Etidocain Hydrochlorid (1% gewöhnliches) bewirkte ein langsameres Einsetzen der Blockierung als Lignocain Hydrochlorid (2%), dauerte länger und rief eine profondere Analgesie bei kaudalen Dermatomen hervor. Die motorische Blockierung durch gewöhnliches Etidocain war ihrem Ausmass nach noch länger und hielt länger an als die durch Lignocain. Im Bezug auf kardiovaskulare Werte gab es keine wesentlichen Unterschiede zwischen Patienten, die gewöhnliches Etidocain oder gewöhnliches Lignocain erhielten. Die Patienten allerdings, die Etidocain mit Adrenalin erhielten, zeigten eine erhöhte Stimulierung der Herzfrequenz und ein Absinken des totalen peripheren Widerstandes während die erste 150 Minuten.
LOS EFECTOS DE AÑADIR ADRENALINA A ETIDOCAINA Y LIGNOCAINA EN LA ANESTESIA EXTRADURAL. I: CARACTERISTICAS DEL BLOQUEO Y EFECTOS CARDIOVASCULARES

SUMARIO
La adición de 5 μg/ml, 1:200 000, de adrenalina al clorhidrato de etidocaína al 1% en administración extradural (L2-3) acortó significativamente el tiempo de aparición del bloqueo sensorial, especialmente en cuanto a la difusión de la analgesia desde el sitio de la inyección, y abrevió el ya rápido comienzo del bloqueo motor. El clorhidrato de etidocaína al 1%, por sí solo, causó una aparición más lenta del bloqueo que con clorhidrato de lignocaína al 2%, tuvo mayor duración, y produjo analgesia más profunda sobre las áreas radiculares caudales. El bloqueo motor producido por la etidocaína por sí sola fue más profundo en su extensión y duró más tiempo que el causado por la lignocaína. Respecto a las variables cardiovasculares, no se produjeron diferencias importantes entre sujetos que recibieron etidocaína simple y la lignocaína simple. Sin embargo, los sujetos que recibieron etidocaína con adrenalina exhibieron aumento en la estimulación cardíaca y un descenso en la resistencia periférica total durante los primeros 150 min.