ACID-BASE BALANCE DURING EPIDURAL ANALGESIA

BY

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SUMMARY

Capillary blood pH, Pco₂ and standard bicarbonate were measured in twenty unpremedicated patients before and after induction of epidural analgesia to the level of the fourth thoracic dermatome or higher. Twelve patients had normal respiratory systems and eight had chronic bronchopulmonary disease. No patient developed any significant degree of respiratory acidosis and it is concluded that alveolar ventilation is unimpaired by epidural blockade as performed here. These results provide further evidence that epidural block can produce effective analgesia and muscular relaxation without impairing respiratory function.

It is commonly held that a differential block affecting the sensory pathways while sparing the motor fibres can be achieved by epidural injection of local anaesthetic agents. In the opinion of Bonica (1958) the production of a differential block appears to depend upon the concentration of local anaesthetic drug and high concentrations of local anaesthetics may cause complete motor block. The drug most commonly used in Great Britain for epidural block is 1.5 per cent lignocaine, but there is no agreement in the literature concerning the concentration of lignocaine which results in a differential epidural block; the varying opinions expressed do not always appear to have been based on objective measurements (Moir, 1963).

Some aspects of ventilatory function during epidural analgesia have been previously studied. Bromage (1955) found that when epidural analgesia with 0.8 per cent lignocaine and conventional analgesics such as pethidine and amidone were administered for the relief of postoperative pain the increase in vital capacity was assumed to be proportional to the degree of pain relief and was greatest after epidural block. Simpson and his associates (1961) used epidural analgesia with 1.5 per cent lignocaine to relieve pain after abdominal surgery and found that the vital capacity could be restored to an average of 69 per cent of its pre-operative value after upper abdominal surgery and to 84.8 per cent after lower abdominal operations. Following effective coughing the vital capacity could sometimes be further increased. It is concluded that the marked reduction in vital capacity normally found after abdominal surgery is due partly to the effect of pain in inhibiting movement of the diaphragm and chest wall, and partly to the exclusion of air from parts of the lungs by bronchial plugs.

Moir (1963) studied the effects of high epidural block in patients who were pain-free and had not received depressant drugs. Tidal volume, minute volume, vital capacity and peak expiratory flow rate were measured before and during epidural block with 1.5 per cent lignocaine and it was concluded that only minimal changes occurred in these aspects of ventilation. From the absence of any noteworthy reduction in peak expiratory flow rate it was inferred that the ability to cough was unimpaired.

It can be concluded that epidural block with 1.5 per cent lignocaine does not reduce ventilatory function to any important degree and that following abdominal surgery the analgesia provided by epidural block can substantially improve ventilation. Indeed, continuous epidural analgesia is probably the most effective method of relieving pain and improving respiratory function after surgery.

*This work was carried out while the authors were members of the Department of Anaesthetics, Stobhill General Hospital, Glasgow.

Based on a paper read before the Scottish Society of Anaesthetists, April, 1964.
The present investigation was designed to confirm these findings by measurement of pH, Pco₂ and standard bicarbonate values of capillary blood.

**METHOD**

Twenty patients (fifteen males; five females) were studied prior to elective abdominal surgery. Their ages ranged from 28 to 74 years (mean 49 years). Eight patients had chronic bronchopulmonary disease which in one case was classed as severe and in seven was classed as moderately severe. This assessment was based on a history of chronic productive cough, sometimes of wheezing and dyspnoea, and on physical examination and chest radiographs. The other twelve patients had no detectable respiratory disease. No patient showed significant abnormality of the cardiovascular system.

All patients were visited on the day before operation and the procedure explained to them. No premedication was given until all estimations had been completed. On arrival in the anaesthetic room capillary blood samples were obtained by stab puncture of the thumb. Whole capillary blood pH, Pco₂ and standard bicarbonate were

<table>
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<th>Level of analgesia</th>
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<th>pH</th>
<th>Standard bicarbonate (m.equiv/1.)</th>
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<td>24</td>
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</table>

*(a) = before epidural blockade; (b) = during epidural blockade.
determined by the micro-technique of Astrup and associates (1960) using the nomogram of Siggaard Andersen and Engel (1960).

Epidural analgesia was then induced with a single injection of 1.5 per cent lignocaine with adrenaline 1:200,000 at the interspace between the second and third lumbar vertebrae. The total volume of solution used varied with the age, build and general health of each patient but was deliberately large to ensure a high level of analgesia. The average volume was 33.7 ml (505 mg lignocaine) and the range was 28 to 40 ml (420 to 600 mg lignocaine).

Between 25 and 30 minutes after injecting the lignocaine solution the upper level of cutaneous analgesia to pinprick was assessed and the spread of solution assumed to be complete when there was no alteration in this level after 5 minutes. The upper level of cutaneous analgesia was above the fifth thoracic dermatome in all patients, the range being from the fourth to the second thoracic dermatome. A second capillary blood sample was then taken for the estimation of pH, Pco₂ and standard bicarbonate before the commencement of surgery.

Systolic blood pressure was maintained above 90 mm Hg in all cases, methoxamine being administered if required for this purpose. Neither oxygen nor any drug other than methoxamine was given until the second sample had been obtained.

**RESULTS**

The values for pH, Pco₂ and standard bicarbonate in capillary blood taken from twenty patients before and during high epidural blockade are listed in table I. In table II the values found in twelve patients who had no respiratory disease are summarized, while table III summarizes the findings in eight patients who had chronic bronchopulmonary disease. The significance of these results is discussed later, but it is apparent that high epidural block did not produce any important degree of metabolic or respiratory acidosis.

**DISCUSSION**

The following ranges of normal values for pH, Pco₂ and standard bicarbonate are proposed by recent workers:

<table>
<thead>
<tr>
<th>pH</th>
<th>Pco₂ (mm Hg)</th>
<th>Standard bicarbonate (m.equiv/l)</th>
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<td>Mean and SD</td>
<td>Range</td>
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<tr>
<td>7.36 - 7.44</td>
<td>36 - 44</td>
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<tr>
<td>7.36 - 7.42</td>
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<td>35 - 49</td>
<td>23 - 29</td>
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</tbody>
</table>

The mean values and standard deviations for pH, Pco₂ and standard bicarbonate in the present series all fall within any of these proposed ranges of normality. The highest observed Pco₂ was
48 mm Hg before anaesthesia and this fell to 44 mm Hg after epidural blockade (case 1). In three patients (cases 3, 8 and 20) $Pco_2$ was below normal. This may have been due to hyperventilation caused by anxiety. It is unlikely that such figures would be a cause for worry if observed during general anaesthesia, where a respiratory acidosis is not uncommon during spontaneous ventilation and where deliberate hyperventilation of varying degree is often practised during controlled ventilation.

In a steady state isolated readings of $Pco_2$ should reflect respiratory function (Nunn, personal communication, 1964). The patients in this series were at rest and so the production of carbon dioxide by metabolism would have been low and reasonably constant. Before taking the second blood sample there had been no alteration in the upper level of cutaneous analgesia for at least 5 minutes so that a rising level of epidural blockade could not have caused an unsteady anaesthetic state. Previous work has shown that the resting tidal and minute volumes are little altered during high epidural analgesia (Moir, 1963). It is probable, therefore, that in the present series the capillary blood $Pco_2$ may be taken as a reasonably accurate reflection of alveolar ventilation. Constant serial readings of $Pco_2$ over a period of time would have confirmed the existence of a steady state but as the patients were being prepared for routine surgery time did not permit the making of these estimations. Under normal resting conditions the $Pco_2$ of capillary blood is very closely related to the $Pco_2$ of arterial blood (Cooper and Smith, 1961) and so in the conditions existing in this study the use of capillary blood appeared to be acceptable and unnecessary arterial punctures were avoided. Since no significant degree of respiratory acidosis was observed in any patient during high epidural block it can be concluded that alveolar ventilation was adequate because alveolar hypoventilation always causes carbon dioxide retention (Comroe et al., 1962).

Carbon dioxide elimination is influenced by the functioning of the respiratory muscles, by the pulmonary ventilation-bloodflow ratio and by the diffusing capacity. Vital capacity and peak expiratory flow rate are little altered by high epidural block (Moir, 1963). Campbell (1958), however, states that in normal subjects the ability to perform tests of maximal ventilatory function is not limited by the power of the respiratory muscles, since these muscles can be shown to be contracting with less than maximum force during the performance of these tests. Two possibilities would therefore appear to exist during high epidural block. Either the motor innervation of the intercostal and abdominal muscles is intact or these muscles are partly or totally paralyzed but the diaphragm has sufficient reserves of power to compensate for this. Electromyographic techniques would be required to distinguish between these two situations but the available evidence does not suggest any important degree of paralysis.

No change occurred in the ratio of pulmonary ventilation to pulmonary bloodflow sufficient to alter the $Pco_2$. Diffusion defects are not thought to be a barrier to the transfer of carbon dioxide.

When 1.5 per cent lignocaine is injected into a mixed peripheral nerve, paralysis typically ensues. Yet after epidural injection there is apparently no paralysis of the respiratory muscles, or if any paralysis exists it is of a minor degree and does not impair the performance of standard tests of resting and maximal ventilatory function, nor does it alter the resting blood pH and $Pco_2$. This may be related to the site of action of local anaesthetics when injected into the epidural space. It may be that the drug is simply diluted in the epidural space until its concentration is insufficient to produce motor paralysis although sufficient to produce analgesia. According to the theory advanced by Nathan and Sears (1963) nerve fibres with a high safety factor are more resistant to the action of local anaesthetics. This safety factor has been defined by Tasaki (1953) for myelinated fibres as the ratio of current developed to current actually required for depolarization. Again, the dura covering the anterior nerve roots is thicker than that covering the posterior nerve roots and so it has been suggested that dural penetration is largely confined to the region of the posterior sensory roots (Dawkins, 1954). There is evidence that the "ink-cuff" area near the junction of the posterior and anterior nerve roots is a major site of dural penetration from the epidural space.
(Brierley and Field, 1948), but from this point solutions might be expected to have equal access to both posterior and anterior roots. Dilution of local anaesthetic solutions in the cerebrospinal fluid is probably not the explanation of the differential block since the drug appears not to reach the subarachnoid space until after analgesia is established (Foldes, Colavincenzo, and Birch, 1956; Bromage, 1962). Recent work suggests that the spinal cord may be an important site of action of local anaesthetics injected into the epidural space (Bromage, 1962; Bromage, Joyal and Binney, 1963). If this is so it must be assumed that the concentration of lignocaine reaching the anterior horn region is inadequate to affect significantly the motor innervation of the respiratory muscles.

Methoxamine was given in all except three of our cases in order to maintain the systolic blood pressure above 90 mm Hg. If this drug had a stimulant effect on respiration it might have masked a potential rise in blood Pco₂. However, methoxamine is said to have no central stimulant action (Goodman and Gilman, 1958; Wood-Smith and Stewart, 1962). Because autonomic disturbances, in particular pallor, sweating and marked alteration of the pulse rate or blood pressure, may affect the normally good correlation between capillary and arterial blood Pco₂ (Cooper and Smith, 1961) it was considered desirable to use a vasopressor in order to avoid these upsets. A free flow of capillary blood was obtained even after the administration of methoxamine, so that it seems unlikely that errors were introduced due to peripheral vasoconstriction.

It is emphasized that although epidural block, as performed here, did not cause respiratory acidosis nor affect significantly the standard tests of resting and maximal ventilatory function, these conclusions were reached in patients who were not at the time undergoing surgery and who had received no depressant drugs.

The negligible effects of epidural analgesia on acid-base balance and ventilatory function may be compared favourably with the effects of general anaesthesia, where spontaneous and controlled respiration may both produce substantial alterations in blood pH and Pco₂.

Acknowledgment

The assistance afforded by the staff of the Department of Biochemistry, Stobhill General Hospital, who performed the biochemical estimations, is gratefully acknowledged.

References


Brierley, J. B., and Field, E. J. (1948). The connexions of the spinal subarachnoid space with the lymphatic system. *J. Anat. (Lond.)*, 82, 153.


L'EQUILIBRE ACIDO-BASIQUE PENDANT L'ANALGESIE EPIDURALE

SOMMAIRE
Mesure du pH du sang capillaire, de la Pco₂, et des bicarbonates standard chez 20 malades non préparés avant et après induction d'une analgésie épiderale atteignant le niveau du 4ᵉ dermatome dorsal au moins. Douze malades présentaient un système respiratoire normal et huit présentaient une affection broncho-pulmonaire chronique. Aucun des malades ne développa une acidose respiratoire significative et on en conclut que la ventilation alvéolaire n'est pas gênée par le blocage épideral comme il a été réalisé ici. Les résultats obtenus montrent d'autre part que le blocage épideral permet d'obtenir une analgésie efficace et une relaxation musculaire sans déranger la fonction respiratoire.

BOOK REVIEW


This volume records the proceedings of a symposium held in May 1963. The contributions come from American, as well as European, laboratories and are arranged in four sections: sodium transport and sodium excretion; persistent hypokalaemia in children; calcium and phosphorus metabolism; and treatment of renal failure.

The second section is concerned with the detailed investigation of absorbing but rare examples of hypokalaemia. The most interesting and best-defined study is that of J. R. Gill and F. C. Bartter, in which they describe a new form of hyperaldosteronism and hypokalaemic alkalosis arising from hyperplasia of the juxtaglomerular apparatus of the kidneys. The third section consists of two excellent articles. The first, by O. L. M. Bijvoet, A. P. Jansen, H. Prenen and C. L. H. Majoor, upon the interpretation of the various indices of phosphate clearance, is for experts in this field only, but S. W. Stanbury's account of calcium and phosphate metabolism in chronic renal failure deserves to be read widely.

The first section is the most comprehensive and has a wide biological interest, but excludes clinical applications. It is, therefore, from the last section that the anaesthetist will derive the most immediate benefit. Three papers discuss the role of androgenic steroids in the management of acute and chronic renal failure and two others describe their authors' experiences with the artificial kidney in chronic uraemia.

There is, rightly, an increasing tendency to deprecate the publication of the omnibus proceedings of symposia, for the component papers are usually of uneven merit and rarely attack a problem comprehensively. By and large this volume avoids these difficulties; and while the book could scarcely be considered compulsory reading for anaesthetists, it does directly illuminate four topics of concern to clinical biochemists in a manner which all medical scientists will find stimulating.  

W. H. Taylor