ACIDOSIS FOLLOWING THORACIC SURGERY

Sir,—Dr Pandit and his colleagues suggest that patients develop a mixed respiratory and metabolic acidosis following a thoracotomy (Brit. J. Anaesth. (1973), 45, 79). However, the results they present do not necessarily support this conclusion. The investigation would have been more convincing if measurements had been made at each stage in all the patients. Incidentally, there are some discrepancies between the figures; for example, for the preinduction phase there are 20 values for pH and base excess but only 18 for the carbon dioxide tension. We are not certain which patients were given sodium bicarbonate, for whilst figure 3 divides the base excess values for the 60-min stage into those who did and those who did not receive bicarbonate, figures 1 and 2 show no such classification for the pH and Pco2 values.

It is well known that indices of a metabolic acidosis such as base excess will show an apparent metabolic acidosis as the carbon dioxide tension of a patient is increased (e.g. Holaday, Ma and Papper, 1957; Stoker et al., 1972). The mean values for the pH and Pco2 for the “pre-reversal” and the “15-min postoperative” stages presented by Dr Pandit would fit such an in-vivo carbon dioxide titration curve. Thus there appears to be no need to invoke an additional metabolic acidosis.

Whilst in individual patients sodium bicarbonate may be of therapeutic benefit following a thoracotomy we do not feel that its routine administration is supported by the results given in this paper.

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Sir.—Dr Norman and his colleagues are quite correct in pointing out that there are discrepancies in the number of dots displayed in the scattergrams contained in our recent paper. We admit that we did not count these meticulously after the design was drawn; however, we would assure them that the mean values obtained were checked and are valid.

Perhaps we did not make it clear that this finding was a side issue to two more detailed studies involving other aspects of controlled ventilation: (1) the comparison of three relaxants by Pandit, Dundee and Stevenson (1971), and (2) the M.D. thesis of Galway entitled, “A clinical investigation of postoperative pain”. For this reason each patient was not investigated in detail at each time interval as one might have liked and it was only when our initial cases showed a consistent postoperative acidosis that we felt it necessary to demonstrate that there was no preinduction acid-base imbalance. Under these circumstances we felt that 20 cases were adequate.

There seemed little point in dividing the 60-min data for all three parameters into those patients who did and those who did not receive bicarbonate. Firstly, it will be seen that this did not affect base excess values and that the mean values obtained were checked and are valid. However, our argument is based on the readings obtained 15 min after the end of surgery and it is from this data that we have concluded that patients suffered from a mixed respiratory and metabolic acidosis following thoracotomy: “This acidosis is mainly respiratory but does have a metabolic element”.

Figure 3 shows only that the base excess levels at 60 min were not significantly influenced by bicarbonate therapy given after the first postoperative observation. However, both the pH and Pco2 levels remain in the acidotic side of normal up to 60 min. Although the standard bicarbonate and the buffer base values were not shown in the paper, these as well as the base excess values were much lower than normal 15 min after operation. From this finding together with an increased Pco2 and a reduced pH we concluded that patients have a mixed acidosis immediately after thoracic surgery.

Dr Norman and his colleagues state that “it is well known that indices of a ‘metabolic’ acidosis such as base excess will show an apparent metabolic acidosis as the carbon dioxide tension of a patient is increased”. One does not get this information from reading the paper by Holaday, Ma and Papper (1957) in which the immediate effects of respiratory depression on acid-base balance in patients anaesthetized with a variety of techniques are reported. These authors concluded that “respiratory acidosis was accompanied by a metabolic acidosis which tended to be proportional to the extent of carbon dioxide retention”. We appreciate that Stoker and his colleagues (1972) have since reported the in vivo carbon dioxide titration curves. However, Stoker’s paper and ours were submitted for publication within a few days of each other and Stoker’s appeared in print after we had corrected the proofs of our own so we could not have known about this. We would like to plot our figures on the curves given by Stoker and see if our metabolic acidosis was real or apparent, but the three authors of our paper are not working together now and this may take some time.

It should be pointed out that nowhere in our paper do we advise routine bicarbonate therapy after operation. On the contrary we have shown (fig. 3) that the metabolic element of the acidosis is likely to revert back to normal within 60 min after operation, with or without bicarbonate therapy. In contrast, we have put up a case in favour of elective ventilation of certain categories of patients and our view is supported by the work of Stoker and his colleagues. In our discussion of the above point one must not lose sight of the object of the paper. One of the main findings of our study was the differing response after thoracic and upper abdominal surgery and also the effects of even small doses of parenteral narcotics.

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