$P_{ET}CO_2$ remained unchanged and $PaCO_2$ decreased significantly following caudal block with 0.5 per cent bupivacaine, although $\dot{V}_E$ and respiratory frequency decreased significantly after both caudal blocks. The reductions in $\dot{V}_E$ and respiratory frequency were clearly observed in all the groups, and the increases in $P_{ET}CO_2$ and $PaCO_2$ were observed in half the groups in our two reports.

Afferent and efferent nerve blocks would be a main cause of impairment of resting ventilation following caudal epidural injection of two per cent mepivacaine or 1.5 per cent lidocaine. However, motor nerve block of the lower chest wall by one per cent mepivacaine or 0.5 per cent bupivacaine is weaker than that by two per cent mepivacaine or 1.5 per cent lidocaine. The decrease in physiological dead space and no change of alveolar ventilation may be a cause of the reduction of $\dot{V}_E$ with no increases in $P_{ET}CO_2$ and $PaCO_2$ following caudal block with one per cent mepivacaine.

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End–tidal CO_2 monitoring in spontaneously breathing adults

To the Editor:

The measurement of end–tidal carbon dioxide measurement ($ETCO_2$) is commonly used as an approximation of arterial CO_2 ($PaCO_2$). However, it is difficult to obtain end–tidal gases in non-intubated patients. Cannulae threaded into the posterior nasopharynx obstruct frequently[1] and are uncomfortable. Canopy ventilation monitors are bulky and may interfere with the nursing care of postoperative patients. Nasal oxygen cannulae have been modified to form "oronasal cannula" which give reliable tracings of $ETCO_2$, although these were not examined quantitatively.[2] In agreement with Urmey[3] and Dunphy,[4] we do not believe that modified nasal cannulae with oxygen flowing[5] may be utilized for accurate $ETCO_2$ sampling.

We have developed a system of measuring $ETCO_2$ in spontaneously breathing patients using Intertech nasal oxygen cannulae into which a 16-g IV cannula is inserted 3 cm from one nasal prong, allowing the tip to lie midway between the two prongs. This is then taped in place (see Figure). The catheter is Luer-locked to the standard sampling tubing of the $ETCO_2$ analyzer. The extension tubing normally connected to the oxygen source is cut and knotted approximately 15 cm from the bifurcation to prevent aspiration of room air.

FIGURE Modified nasal cannula; note presence of 16-gauge IV cannula and cut oxygen delivery line.

We have found this device meets with patient acceptance, is free from obstruction and fits easily under an oxygen mask if an increased FiO_2 is desirable. The majority of our experience has been in patients requiring oxygen by face mask, although removal of the face mask in individual patients has not been associated with changes in $ETCO_2$ measurement. It does not appear to be affected by mouth breathing vs nose breathing though complete nasal obstruction prevents its use. It is convenient, simple and inexpensive. Most importantly it seems to provide good sampling of end–tidal gases as evidenced by capnograph tracings and correlation with $PaCO_2$. This will require further clinical comparison which we are now performing. This method may provide further assistance in respiratory monitoring of non-intubated patients both in and outside of the operating room setting.

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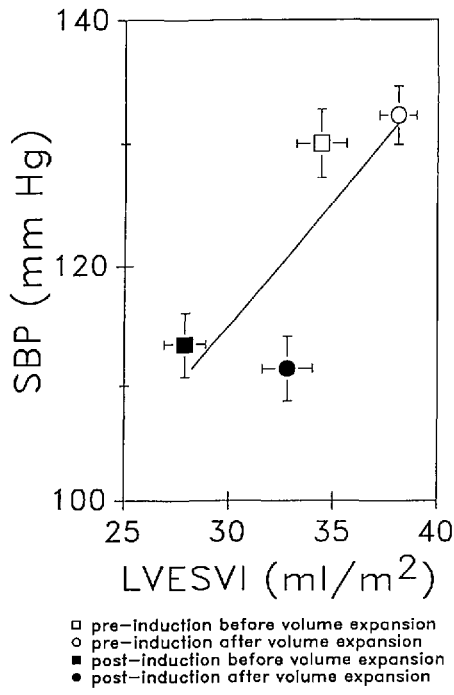
Fentanyl, sufentanil, alfentanil and myocardial function

To the Editor:

We congratulate Miller *et al.* for their careful and sophisticated study of the effects of narcotic anaesthesia on myocardial function and metabolism.[1] Based on systolic pressure-volume (PV) relationships obtained by volume loading, before and after induction of anaesthesia, Miller *et al.* conclude that "all of the narcotics studied depress contractility." They further conclude that, based on leftward shift of the diastolic PV relationship, "the anaesthetic sequence reduced diastolic compliance." These conclusions are at variance with traditional views of narcotic effects on myocardial function. We dispute these conclusions and would like to propose an alternative interpretation of their data.

We feel strongly that the average change in systolic blood pressure (SBP) produced by blood volume expansion (approximately ± 2 mmHg) was much too small to allow any valid conclusions to be drawn regarding the slope of the pre-induction and post-induction systolic PV relationships of the left ventricle. In contrast, the change in SBP associated with induction (approximately −20 mmHg) was large enough to allow limited conclusions to be drawn regarding the average systolic PV relationships of Miller's patients.

The Figure shows the systolic PV points for all the patients both prior to and after induction of anaesthesia, and before and after volume expansion. Since both post-induction points lie below and to the left of the pre-induction points, we see no convincing evidence of impaired contractility. In fact, we suggest that all four



FIGURE

points lie on the single PV curve which we have drawn, and that contractility was in no way altered by induction of anaesthesia. The slight change in position of the PV points prior to and following volume expansion probably reflects imprecision in the determination of left ventricular end systolic volume index (LVESVI), and the fact that systolic BP rather than end–systolic BP was used to define the PV relationship. The LVESVI calculation depends on several measurements (heart rate, radionuclide ejection fraction and thermodilution cardiac output) and individual errors may be compounded by calculation. We also believe that the small leftward shift in the diastolic PV relationship after induction of anaesthesia simply reflects change from spontaneous to positive pressure ventilation, rather than an ischaemia-induced decrease in compliance, as suggested by the authors.

We cannot comment on the effect of the individual narcotics on PV relationships because the authors did not