Correspondence

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Succinylcholine Reverses Spasm of the Sphincter of Oddi

To the Editor:—Naloxone reversal of spasm of the choledochoduodenal sphincter associated with fentanyl anesthesia1 entails partial or complete reversal of analgesia. In our opinion this is an unpractical solution during an operation. By means of roentgenographic image intensification and cinecholangiography we have studied the effects of muscle relaxants on the sphincter of Oddi during fentanyl-supplemented anesthesia for routine operations on the biliary tract.29 Succinylcholine, 100 or 150 mg, is effective in reversing spasm induced either by opiate drugs or by surgical manipulation. Using this technique one can distinguish fentanyl-induced spasm from closure due to inflammation or sclerosis where motility is reduced or abolished. Pancuronium will often reverse opiate-induced spasm, but d-tubocurarine will not. In our hospital the use of succinylcholine for reversal of fentanyl-induced spasm of the sphincter of Oddi is a standard procedure.

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References


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PEEP and PaCO2

To the Editor:—We were most interested in Dr. Dueck’s article about the effects of PEEP on the variations in PaCO2.1 It has generally been assumed that PEEP had little effect on PaCO2.

During the last year we have treated 14 patients with severe adult respiratory distress syndrome (ARDS) with intermittent positive-pressure ventilation (IPPV) and PEEP (shunt: 41 ± 3 per cent, x ± SE during IPPV). In all patients PEEP was increased progressively by 5 cm H2O to the level of optimal PEEP. For our clinic this level required a shunt of less than 15 per cent, cardiac output being supported whenever necessary. Tidal volume and respiratory frequency were always held constant at each level of PEEP. Under these conditions the average PaCO2 did not change (38.7 ± 1.7 torr during IPPV; 38.8 ± 2.8 torr during optimal PEEP).

However, we found that our patients could be divided into two groups: Group I (six patients), in whom PaCO2 did not change or increased with each level of PEEP; Group II (eight patients), in whom PaCO2 decreased at each level of PEEP (fig. 1). We found no relation between PaCO2 variations induced by PEEP and the values of PaCO2 during IPPV or the values of PaCO2, or static and dynamic compliance, during IPPV or PEEP. The prognoses were the same for the two groups. On the other hand, with IPPV the airway resistance values in Group I were higher than those in Group II. We want to emphasize that no patient in either group had chronic obstructive pulmonary disease. In Group I, four patients had Mendelson’s syndrome and two had viral pneumonia, diseases that usually affect airways and pulmonary parenchyma. In Group II, the patients had ARDS related to peritonitis, septic shock, and fat embolism, which mainly affect pulmonary parenchyma like oleic acid-induced acute pulmonary edema. So, in Group II, the decrease in PaCO2 with PEEP can be explained by the decrease in low VA/Q areas without augmentation of high VA/Q regions by holding cardiac index constant. Why PaCO2 values were increased in Group I patients is not clear. Perhaps in