The central respiratory stimulant, doxapram, which is used primarily for postanesthetic depression, restored breathing in this patient. It is conceivable that breathing might have been restored later without use of the drug. Because occasional patients take the odd breath after injection of caffeine sodium benzoate, which is less effective as a respiratory stimulant, it seemed justified to test the "central reserve" with doxapram, despite the recommended contraindications to its use in arteriosclerotic vascular disease.

No additional arousal was produced by doxapram. While pain did not prompt respiration before the administration of doxapram, or influence respirations afterwards, pain did produce increased sensorial arousal. Thus, pain stimuli did not pass through the central connections to the respiratory centers but did proceed to higher stations.

In terms of current concepts of central respiratory circuitry, instant restoration of rhythmicity by doxapram might suggest a central disturbance of the modulatory pneumotaxic-apneustic mechanism and therefore imply the drug's action to be in this pontine location.\textsuperscript{2-4}

References


Cardiovascular Effects of Pancuronium

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During the past six months, we have used pancuronium, 0.15 mg/kg, during induction of anesthesia to facilitate endotracheal intubation in 37 patients scheduled for open-heart surgery. Since this dose is somewhat larger than those reported by other investigators, we attempted to determine whether it caused any untoward cardiovascular effects. Accordingly, we performed cardiovascular studies of ten of these patients during induction of anesthesia.

Materials and Methods

Ten adult patients who were to undergo cardiac surgery involving cardiopulmonary bypass were selected for study. Nine patients underwent aortocoronary vein grafting and one patient had an open mitral valvulotomy. Informed consent for administration of pancuronium and determination of cardiac output was obtained from each patient prior to study. The patients were premedicated with morphine sulfate, 8-15 mg, atropine, 0.4 mg, and hydroxyzine, 25 mg, one hour prior to the study.

Using local anesthesia, Teflon catheters were inserted percutaneously into a radial artery and subclavian vein and connected to pressure transducers; the data were displayed on a two-channel recorder and digital meters. Systolic, diastolic, and mean blood pressures, ECG, central venous pressure, and temperature were monitored. Cardiac output was measured by the dye-dilution method employing a Waters densitometer. Systemic vascular resistance was calculated from the ratio of mean arterial pressure minus right atrial pressure to cardiac output.

Prior to the induction of anesthesia, baseline data were recorded and cardiac output was measured. Anesthesia was induced with thiopental, given intravenously until the laryn reflex

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was lost (mean dose 4 mg/kg). The patient was then allowed to breathe 60 per cent nitrous oxide and 40 per cent oxygen and ventilation was assisted to maintain a $P_{CO_2}$ of approximately 40 torr. Repeat cardiac output measurements were made and the cardiovascular variables were recorded. Then, pancuronium, 0.15 mg/kg, was given intravenously and controlled ventilation was instituted. About 5 minutes later, when neuromuscular blockade was judged to be complete by use of a peripheral nerve stimulator, cardiac output and other variables were again determined. The trachea was then intubated and the operation performed. A t test for paired data was used in comparing the values obtained in the awake state with those obtained after induction of anesthesia in each patient. A similar comparison was made between the values obtained after induction with thiamylal and those obtained 5 minutes after the administration of pancuronium. Thus, for each variable studied, each patient served as his own control.

RESULTS

Induction with thiamylal, iv, was associated with significant decreases in systolic blood pressure and cardiac output. Heart rate increased an average of 6 beats/min (8.4 per cent). Diastolic and mean blood pressures were unchanged (table 1). These values were not significantly altered by the administration of pancuronium. No serious ventricular arrhythmia developed during the induction of anesthesia.

The relaxation of the jaw muscles during intubation was comparable to that achieved with succinylcholine. Intubation was performed with ease after the cardiovascular studies were completed, and no patient coughed or reacted unfavorably to intubation.

Anesthesia was continued with N2O/O2 60/40 and fentanyl or halothane. We did not find it necessary to administer any more pancuronium to any patient prior to cardiopulmonary bypass. At the completion of the surgical procedure, the effects of pancuronium were reversed with neostigmine, 3.0 mg, and atropine, 1.2 mg, in seven of the ten patients and the tracheas were extubated. These patients were able to maintain adequate ventilation, as judged by serial blood-gas measurements. We elected not to reverse pancuronium effects in three patients because of the extents or durations of the surgical procedures. There was no operative mortality in the study group.

DISCUSSION

Our clinical experience with adult patients indicates that pancuronium, 0.15 mg/kg, produces consistently satisfactory conditions for intubation. This dose did not cause any deleterious cardiovascular effect even in a group of patients whose cardiovascular status was relatively unstable. We did not observe the increase in cardiac output found by Smith et al. in dogs and Kelman and Kennedy in man. We noted a significant decrease in cardiac output following the administration of thiamylal, but there was no additional change following the administration of pancuronium. We also did not observe any significant change in blood pressure or heart rate after the administration of pancuronium. This observation is in agreement with findings in a recent study by Lyons and Clarke. It should be noted that our study and that of Lyons and Clarke were performed...
under similar conditions, whereas the studies of other authors 2, 5, 7 who report increases in heart rate and blood pressure were performed under different clinical situations, i.e., following endotracheal intubation.

Although reversal of the effects of pancuronium, 0.15 mg/kg, was accomplished without difficulty, we cannot assess whether it would be a problem in operations of shorter duration. For the purposes of open-heart surgery, however, pancuronium, 0.15 mg/kg, was excellent, because we had no postintubation coughing or bucking and transition to our maintenance anesthetic was uneventful.

REFERENCES


