Cardiac Arrhythmia Induced by Negative Phase in Artificial Ventilation

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The hemodynamic effects of the negative phase during intermittent positive-negative ventilation have been well documented. For example, in certain neurologic disorders such as fracture of cervical spine with quadriplegia, venous return is improved and arterial blood pressure better maintained if a negative phase is introduced during artificial ventilation. Watson et al. also showed that in such disorders subatmospheric pressure applied in expiration during IPPR increased transmural CVP and often increased mean arterial pressure also. We are not aware, however, of any reports of cases in which arrhythmias were induced by the negative phase in intermittent positive-negative ventilation. The following is a report of such a case occurring during hypothermia.

Case Report

The patient, a 42-year-old man, was admitted with a history of sudden hemiplegia. He was conscious on admission. He had been on antihypertensive therapy (guanethidine) for two years. Blood pressure was within normal limits and there was no evidence of cardiac disease clinically. The ECG was interpreted as normal. The respiratory system was normal. Cerebral angiograms revealed an arteriovenous malformation in the thalamic region, with an intracerebral hematoma. A left frontal craniotomy was scheduled, to be done under hypothermia. Atropine 0.6 mg. was given 45 minutes prior to induction of anesthesia with thiopental, 400 mg. and Anectine® 80 mg. Four per cent Xylocaine® spray was applied topically to the glottis and a 9.5 mm. armored cuffed tube inserted. Maintenance consisted of N₂O-O₂ (2:2 L/min.), halothane, and d-tubocurarine with methoxyflurane given for the first 45 minutes after inflation of the scalp with 1:250,000 adrenalin (while the temperature was still 34° C). Central venous pressure was monitored via a catheter introduced through the left basilic vein. Continuous arterial pressure was monitored via a cathether in right radial artery. Blood gases were determined at intervals. Controlled ventilation was achieved with a Bird Mark IV plus Mark VIII respirator. Positive pressure was kept at 15 cm. H₂O and negative pressure at −5 cm. H₂O to obtain a tidal volume of 550 ml. at 16/minute.

The course of the anesthesia was uneventful until a temperature of 30° C was reached. At this point, ventricular bigeminy appeared. This was attributed to the hypothermia; blood gas values were close to normal, with pH 7.44, $P_{\text{CO}_2}$ 33 mm. Hg, $P_{\text{O}_2}$ 175 mm. Hg, HCO₃⁻ 21.7 mEq/l. and base deficit 2.0 mEq/l. Lidocaine, 100 mg. (1 per cent solution, intravenously), given slowly, rapidly corrected the arrhythmia to sinus rhythm. Twenty minutes

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later, however, ventricular bigeminy reappeared (fig. 1A). Manual ventilation was
done at this stage. Within 1–2 minutes the
bigeminy disappeared, although a nodal rhythm became evident (Fig. 1B). The patient
then was put back on the ventilator, this time with the negative phase off, and there was no
further episode. However, as soon as a negative phase was reintroduced, ventricular bigeminy recurred within 60 to 90 seconds (fig. 1C). When the negative phase was turned off again, sinus rhythm occurred within 60 seconds (fig. 1D). This sequence of events was reproduced and recorded six times consecutively; there was no fall in arterial pressure at any time. Thereafter the patient was maintained on positive pressure only without any cardiovascular abnormalities. Emergence from anesthesia and rewarining were uneventful; the operation terminated successfully with a total anesthesia time of seven hours.

**COMMENT**

The ventricular bigeminy appeared so consistently when a negative phase was introduced into the cycling pattern, and disappeared when positive phasing only was applied, that we had to conclude that the subatmospheric tracheal pressure was triggering a ventricular arrhythmia. It is likely that this occurrence was evident only because the heart was rendered irritable by hypothermia. The actual mechanism is open to speculation. We believe that the subatmospheric pressure in the trachea initiated a vagal reflex such as is commonly seen during manipulation of the tracheobronchial tree during pulmonary surgery.

**REFERENCE**

1. Watson, W. E., Smith, A. C. and Spalding, J.