

Clinical Workshop

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Pulse-rate Changes in Elderly Patients during Deliberate Hypotension

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Ganglionic blockade usually results in tachycardia due to inhibition of vagal tone.¹ It has been suggested that with the use of pentolinium (Ansolysen®), tachycardia is less common.² Decreased response to continued administration of ganglion-blocking agents (tachyphylaxis) is almost invariably associated with tachycardia.^{1,3,4} Methods to avoid tachyphylaxis are those directed toward lowering the pulse rate.

In contrast to the frequent occurrence of tachycardia in healthy adults following deliberate hypotension, I have observed that elderly patients commonly develop bradycardia. The purpose of this report is to evaluate the possible interrelationships of pulse rate, arterial blood pressure and right atrial pressure in elderly patients during the induction of hypotension.

Six patients between the ages of 61 and 75 years were chosen for the study. Following pentothal sodium induction, succinylcholine was administered to facilitate intubation. Anesthesia was maintained with 0.4 to 0.5 per cent halothane in oxygen and intermittent doses of *d*-tubocurarine were given as required. Respiration was controlled at a rate between 6 and 10/min, with a tidal volume varying between 600 and 900 ml. When, by clinical estimation, a steady state was achieved, pentolinium (Ansolysen) was administered intravenously in an initial dose of 2 mg. When the blood pressure started to fall, the patients were tilted 10 degrees at a time (head-up tilt). Pulse rate and arterial systolic pressure were

determined by oscillogram. In four patients, central venous pressure was measured simultaneously.

RESULTS

The results obtained are shown in Figures 1 and 2. Figure 1 shows the relationship between arterial systolic pressure and pulse rate in the six elderly patients represented by symbols. A drop in arterial systolic pressure was accompanied by slowing of the pulse rate during the induction of deliberate hypotension. Figure 2 shows the relationship between central venous pressure and pulse rate in four of the patients from Figure 1. A decrease in central venous pressure was also associated with a decrease in pulse rate.

COMMENT

The results of this study suggest a correlation between arterial systolic pressure, pulse rate, and right atrial pressure (central venous pressure) during induction of hypotension in elderly patients. Similar observations following spinal analgesia have been reported. It has been suggested that bradycardia following spinal anesthesia is related more to the development of hypotension than to the level of anesthesia.⁵ Raising the legs or placing the patient in the head-down position will elevate both the arterial blood pressure and the pulse rate during spinal analgesia.⁵ It might be expected that spinal hypotension should cause tachycardia reflexly mediated through the baroreceptors of the carotid sinus and aortic arch. The reason this does not happen during spinal analgesia is probably that the re-

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FIG. 1. Pulse-rate changes during induction of hypotension in six elderly patients. Four values are plotted for each patient: one prior to hypotension, and the other three at different levels of hypotension.

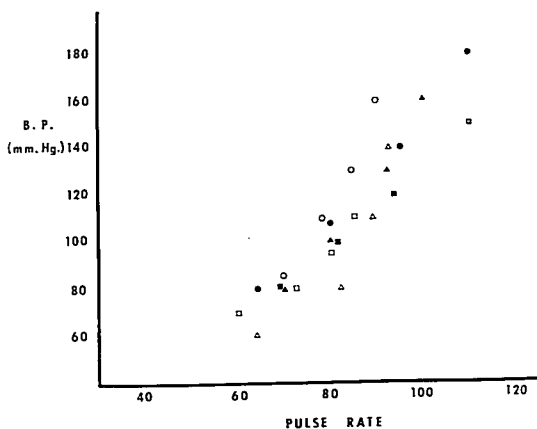
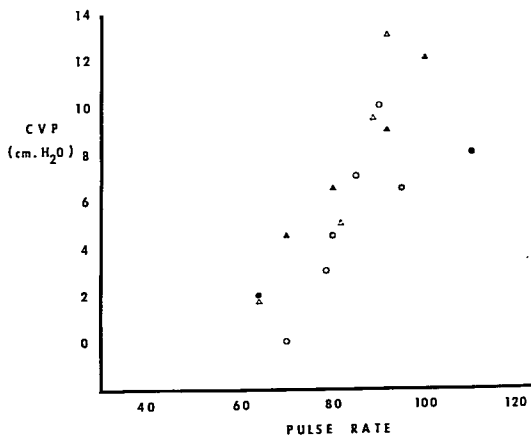


FIG. 2. Changes in central venous pressure and pulse rate during induction of hypotension in four patients. Four measurements are plotted for each patient: one prior to hypotension, and the other three at different levels of hypotension.



sponses activated by the Bainbridge reflex are dominant over those from the baroreceptors.⁵ As a result, elevation of right atrial pressure is accompanied by tachycardia and a decrease in atrial pressure results in bradycardia during spinal analgesia.

With the induction of hypotension, peripheral pooling following ganglionic blockade and

head-up tilt is accompanied by a drop in right atrial pressure in elderly patients. This, in turn, results in reflex slowing of the pulse rate. It is probable, then, that the Bainbridge reflex plays an active role in influencing pulse rate during deliberate hypotension in elderly patients, as it does during spinal analgesia. The common occurrence of bradycardia in elderly

patients may explain the infrequent incidence of tachyphylaxis in this age group.

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Use of the Columbia Pediatric Circle Valve for Respiratory Studies in the Neonate

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Respiratory studies in newborn infants have been hampered by the difficulty of collecting expired gas samples. Golinko and Rudolph¹ designed a nonbreathing valve based on the fact that most quiet healthy infants breathe through the nose. This valve, which is inserted into the infant's nostrils, has minimal resistance to respiration, a deadspace of 0.8 ml, and permits collection of expired air via an open-circuit system. However, when we attempted to measure the tidal volume in neonates at 10, 20, and 30 minutes after birth, many nondepressed infants reacted to even the most gentle insertion of the nasal valve by prolonged bouts of crying, and establishment of a quiet state was markedly delayed. Recently, Rackow and Salanitro² designed a pediatric unidirectional valve (Columbia pediatric circle valve)³ which can be used with either a mask or an endotracheal tube. This valve has a deadspace of only 0.5 ml and, at the peak air velocity of a full-term infant breathing quietly, its resistance is negligible. Combined with a single canister, the valve

was employed satisfactorily in several thousand infants and children receiving inhalation anesthesia.³

We have used the Columbia pediatric circle valve successfully for respiratory studies on the neonate by adapting the tail-end of a three- or five-liter anesthesia bag to the expiratory valve opening. The mouth of the anesthesia bag is closed by a stopper into which a two-way stopcock is incorporated for sampling (fig. 1). Two operators are necessary to complete the collection. The valve, with the expiratory unit lowermost and the pop-off tightly closed, is attached to a Rendell-Baker mask of proper size, which is placed gently but firmly over the infant's mouth and nose. After a quiet state has been obtained (usually less than one minute), the empty bag, with the tail closed by a clamp, is connected to the expiratory-valve opening, and the clamp is released on the first inhalation. Exhaled air is collected for two or three minutes as desired. Then the tail of the bag is reclamped and the mask removed. Ten ml of air are withdrawn for carbon dioxide (and oxygen) tension determinations. The remaining gas volume is measured in a spirometer.

To date, 40 unselected clinically nondepressed neonates have been studied. At 10-12

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