

Clinical Workshop

S. G. HERSHEY, M.D., *Editor*

Junctional Rhythm Induced by Halothane Anesthesia— Treatment with Succinylcholine

ANIBAL GALINDO, M.D., PH.D.,* STEVEN R. WYTE, M.D.,†
J. W. WETHERHOLD, M.D.‡

The arrhythmias most frequently seen during surgical operations and prolonged administration of halothane involve the phenomenon of A-V dissociation.¹ This arrhythmia appears when there is: 1) a slowing of the primary pacemaker; 2) an acceleration of a subsidiary pacemaker; 3) an A-V or S-A block; or 4) a combination of 1 and 3.^{2,3} Junctional rhythms (formerly nodal)² and, in general, uncoupling of atrial and ventricular contractions, are easily reverted to sinus rhythm by decreasing the inspired concentration of the offending anesthetic. These arrhythmias are associated with decreased systemic arterial blood pressure, increased central venous pressure, and reduction in cardiac output as a result of inefficient hemodynamics.⁴ Despite the above effects, they rarely compromise the circulatory status of the patient and are frequently overlooked unless the ECG is monitored. However, the decrease in cardiac output produced by a junctional rhythm can assume significant proportions when the circulatory system is stressed in response to hemorrhage, the sitting position, intermittent positive-pressure respiration, hypothermia, etc. The objective of this communication is to report a simple treatment

of junctional rhythm caused primarily by halothane anesthesia.

CLINICAL OBSERVATIONS

Subjects of the study were ten consecutive patients, 16 to 68 years old, undergoing mainly neurosurgical operations and anesthetized with halothane (0.5 to 1.5 per cent) and N_2O-O_2 (40 per cent–60 per cent). Succinylcholine chloride (SCh), 10 to 20 mg, was given intravenously in the presence of junctional rhythm. There was conversion to sinus rhythm in all patients. Normal sinus rhythm lasted 10 to 20 minutes, and in two cases there were no subsequent arrhythmias. In one patient being anesthetized with fluroxene, morphine, and N_2O-O_2 , succinylcholine (20 mg) administered twice had no effect on a junctional rhythm; in another patient the junctional rhythm had a reflex origin and was not converted by SCh but by the administration of atropine. Figure 1 illustrates a typical example of conversion. The patient was a 23-year-old woman undergoing a combined thoracotomy and thoracic laminectomy for resection of a recurrent fibrosarcoma. After succinylcholine, 20 mg iv, there was a 30-second period of relative sinus bradycardia, then an increase in heart rate, which subsequently led to conversion to sinus rhythm (ECG traces). This conversion lasted 20 minutes, at the end of which time the junctional rhythm reappeared. Succinylcholine was given a second and a third time, with similar results. Blood gases at the time of the first conversion showed some degree of hyperventilation.

* Associate Professor.

† Assistant Professor.

‡ Resident. Present address: Tucson Medical Center, Tucson, Arizona 85716.

Received from the Department of Anesthesiology and the Anesthesia Research Center, University of Washington School of Medicine, Seattle, Washington 98195. Supported by USPHS Grant GM 15991-03 from the National Institute of General Medical Sciences, National Institutes of Health.

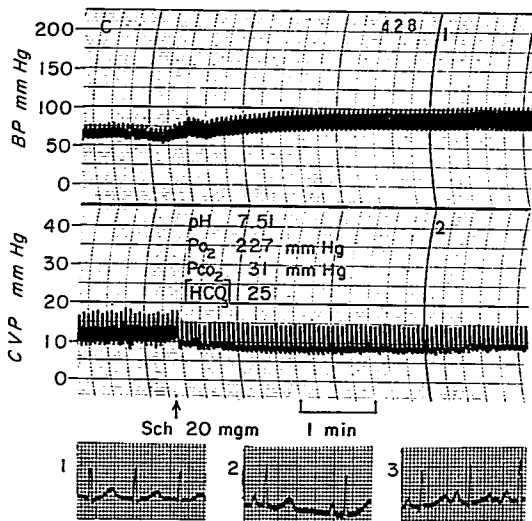


FIG. 1. Conversion of junctional to sinus rhythm by intravenous administration of 20 mg of succinylcholine (Sch). Effects of this conversion on intra-arterial blood pressure (BP) recorded from the radial artery and on central venous pressure (CVP) are shown. The lower ECG traces are shown (1) immediately before, (2) a few seconds after, and (3) five minutes after administration of Sch.

DISCUSSION

Experimental studies during halothane anesthesia reveal a conduction delay through the AV node and an increased internal longitudinal resistance of Purkinje fibers⁵ that may explain junctional rhythm during clinical administration of this anesthetic. Vasoactive drugs such as Isuprel, metaraminol, mephentermine, and ephedrine speed conduction through the specialized myocardial pathways and can be used for the treatment of delayed conduction.⁶⁻⁸ Unfortunately, the administration of these adrenergic stimulators during halothane anesthesia may have undesirable effects. On the other hand, for conversion of a junctional arrhythmia to a sinus rhythm in the anesthetized patient, small doses of succinylcholine are simple to use, readily available, and usually short-acting. Succinylcholine may act by its direct effect on the myocardium and/or by its sympathetic ganglionic stimulating properties.⁹

REFERENCES

1. Kuner J, Enescu V, Utsu F, et al: Cardiac arrhythmias during anesthesia. *Dis Chest* 52: 580-587, 1967
2. Pick A, Langendorf R: Recent advances in the differential diagnosis of A-V junctional arrhythmias. *Am Heart J* 76:553-575, 1968
3. Pick A: A-V dissociation. A proposal for a comprehensive classification and consistent terminology. *Am Heart J* 66:147-150, 1963
4. McIntosh HD, Kong Y, Morris JJ Jr: Hemodynamic effects of supra-ventricular arrhythmias. *Am J Med* 37:712-727, 1964
5. Hauswirth O: Effects of halothane on single atrial, ventricular, and Purkinje fibers. *Circ Res* 24:745-750, 1969
6. Oppenheimer MH, Lynch PR, Ascanio G: Action of mephentermine on arrhythmias due to pulsus alternans, rapidly discharging single atrial foci and prolonged P-R intervals. *Am J Physiol* 191:461-486, 1957
7. Stewart GH III, Lynch PR, Barrera F, et al: Changes in properties of heart muscle due to mephentermine. *Am J Physiol* 186:513-517, 1956
8. Burn JH, Gunning AJ, Walker JM: Effects of noradrenaline and adrenaline on the atrial rhythm in the heart-lung preparation. *J Physiol* 137:141-153, 1957
9. Galindo A, Davis TB: Succinylcholine and cardiac excitability. *ANESTHESIOLOGY* 23:32-40, 1962