5. Stuart GG, Merry GS, Smith JA, Yelland DN: Severe head injury managed without intracranial pressure monitoring. J Neurosurg 59:601–605, 1983

6. Carmichael FJ, Lindop MJ, Forman JV: Anesthesia for hepatic transplantation: Cardiovascular and metabolic alterations and their management. Anesth Analg 64:108–116, 1985

7. Kang YG, Gelman S: Anesthesia for liver transplantation, Anesthesia for Organ Transplantation. Edited by Gelman S. Philadelphia, W. B. Saunders, 1987, pp 139–185

8. Hanid MA, Davies M, Mellon PJ, Silk DBA, Strunin L, McCabe JJ, Williams R: Clinical monitoring of intracranial pressure in fulminant hepatic necrosis. Gut 21:866–869, 1980

9. Ware AJ, D'Agostino AN, Combes B: Cerebral edema: A major complication of massive hepatic necrosis. Gastroenterology 61: 877–884, 1971

10. Paulsen AW, Valek TR, Blessing WS, Johnson DD, Parks RI, Pyron JT, Ramsay MA, Simpson BR, Swygert TH, Walling PT, Klintmalm G: Hemodynamics during liver transplantation with veno-venous bypass. Transplant Proc 19:2417–2419, 1987

11. Canalese J, Gimson AES, Davis C, Mellon PJ, Davis M, Williams R: Controlled trial of dexamethasone and mannitol for the cerebral edema of fulminant hepatic failure. Gut 23:625–629, 1982

12. Shapiro HM: Anesthesia, Neurosurgical Anesthesia and Intracranial Hypertension. Edited by Miller RD. New York, Churchill Livingstone, 1986, pp 1592–1593

Treatment of Isorhythmic A-V Dissociation during General Anesthesia with Propranolol

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Isorhythmic A-V dissociation is a common cardiac dysrhythmia during anesthesia that usually occurs in patients anesthetized with one of the potent volatile anesthetics.[1–3] With continuous monitoring of the electrocardiogram, the P-R interval is observed to gradually shorten as the P wave approaches the QRS complex and then disappears within it. The QRS morphology does not change, and the SA node and AV node continue to depolarize independently of each other but at similar rates.[4,5] The P wave usually remains hidden within the QRS complex giving the appearance of an A-V junctional rhythm, but it may be observed to immediately follow it or to move in and out of it. When observed the P wave morphology is not altered, unlike in true A-V junctional rhythms where the atria are depolarized from the A-V node, usually causing inverted P waves in leads II, III, and AVF.[6]

Isorhythmic A-V dissociation is usually tolerated without hemodynamic compromise and its diagnosis is frequently missed. In healthy patients anesthetized with halothane, isorhythmic A-V dissociation has been found to cause a 17% decrease in systolic blood pressure[7] and a 14% decrease in mean arterial blood pressure[8] with a relatively constant heart rate. Isorhythmic A-V dissociation can, however, occasionally result in a significant decrease in arterial blood pressure.[9] In patients with compromised cardiac function the loss of the normal atrial contribution to left ventricular filling can significantly reduce cardiac output.

Decreasing the inhaled anesthetic concentration can convert isorhythmic A-V dissociation to sinus rhythm.[7] Previous clinical reports have demonstrated that both atropine[9] and succinylcholine[10] may be effective treatments in some cases of A-V junctional rhythm during anesthesia. The incidence of A-V junctional rhythms in patients anesthetized with fentanyl and nitrous oxide was reduced by pretreatment with practolol.[11] Intravenous propranolol was reported to successfully convert an apparent A-V junctional rhythm to a sinus rhythm in one patient.[12] Some of these rhythm disturbances were probably isorhythmic A-V dissociation.[3,13]

Four cases of patients who developed isorhythmic A-V dissociation during cardiac surgery are presented. In each case the P wave was observed to gradually approach the QRS complex and then disappear within it. The administration of propranolol was successful in treating each episode.

CASE REPORTS

Case 1. A 74-yr-old man with class IV angina, a history of palpitations, a previous myocardial infarction (MI), and a traumatic right lower leg amputation was admitted to the hospital for evaluation of his angina. His electrocardiogram (ECG) showed a normal sinus rhythm at a rate of 75 beats/min and an old inferior MI. Cardiac catheterization revealed significant three vessel coronary artery disease, an ejection fraction of 55%, and aortic stenosis with a pressure gradient of 40 mmHg across the aortic valve. His preoperative medications, including 30 mg of diltiazem and 20 mg of isorbid dinitrate every 6 h, were continued to the time of surgery. He was scheduled for coronary revascularization

curred in 4–46% of patients in several clinical investigations.[1,2,19–24] Many of these were actually examples of isorhythmic A-V dissociation. Kuner *et al.*[2] and Alexander *et al.*[2] found that over 50% of the rhythms appearing to be junctional were isorhythmic A-V dissociation. Rhythms of junctional appearance have also been observed during anesthesia with nitrous oxide and fentanyl,[25] following administration of pancuronium as in the first case,[26] and frequently after reversal of muscle relaxants with anticholinesterase drugs.[27–30]

The four patients discussed in this report were all anesthetized primarily with opioids following preoperative sedation with a benzodiazepine. Inhaled anesthetics were used in low concentrations. None of the patients were given preoperative beta adrenergic blockers, which reduce the incidence of junctional rhythms during anesthesia.[11] All were taking calcium channel blockers, which may have increased the chance of isorhythmic A-V dissociation. The appearance of junctional rhythms has been reported in patients treated with verapamil.[31] In all of the present cases the P wave was seen to gradually approach and then hide within the QRS complex, giving the appearance of an A-V junctional rhythm. Treatment with small doses of iv propranolol was effective in converting isorhythmic A-V dissociation to sinus rhythm in all four patients, which is consistent with this author's clinical experience. During conversion to sinus rhythm, the P wave gradually receded from the QRS complex. Restoration of sinus rhythm increased systolic blood pressure by an average of 18%, which is similar to previous reports.[7,8]

Propranolol slows conduction and increases the effective refractory period of the A-V node. It reduces the rate of spontaneous diastolic depolarization and therefore the rate of impulse formation in cells that exhibit automaticity.[32] Propranolol may preferentially slow the spontaneous rate of depolarization of accelerated A-V nodal tissue, thus allowing the sinus node to reassume control of heart rate with a conversion to sinus rhythm.

Propranolol should be used to treat isorhythmic A-V dissociation only after the diagnosis is certain. Beta adrenergic blockers are contraindicated for treatment of junctional escape rhythms, which result from default of normal SA node pacemaker function as in SA block, sick sinus syndrome, or high AV block.[6] Junctional escape occurs after a longer beat-to-beat interval than in the normal dominant sinus rhythm, producing a slower rhythm, usually from 30 to 60 beats/min. It functions as a safety mechanism and should be not suppressed. Isorhythmic A-V dissociation, however, generally results from acceleration of the A-V node, giving rise to a rhythm that is faster than the dominant sinus rhythm. Propranolol should be used with a great deal of caution in patients with disease, such as bronchospasm or poor ventricular function, which is aggravated by the known actions of the

drug. A short-acting drug with more beta 1 receptor specificity such as esmolol may be preferred in situations in which the side effects of beta adrenergic blockade are potentially detrimental.

In conclusion, isorhythmic A-V dissociation is a common rhythm disturbance during general anesthesia. Small doses of iv propranolol are effective in converting this rhythm to normal sinus rhythm.

REFERENCES

1. Kuner J, Enescu V, Utsu F, Boszormenyi E, Bernstein H, Corday E: Cardiac arrhythmias during anesthesia. Dis Chest 52:580–587, 1967
2. Alexander JP, Bekheit S, Fletcher E: Dysrhythmia and oral surgery II: junctional rhythms. Br J Anaesth 44:1179–1182, 1972
3. Sethna DH, Deboer GE, Millar RA: Observations on "junctional rhythms" during anaesthesia. Br J Anaesth 56:924–925, 1984
4. Schubart AF, Marriott HJL, Gorten RJ: Isorhythmic dissociation atrioventricular dissociation with synchronization. Am J Med 24:209–214, 1958
5. Marriott HJL, Menendez MM: A-V dissociation revisited. Prog Cardiovasc Dis 8:522–538, 1966
6. Marriott HJL, Myerburg RS: Recognition of arrhythmias and conduction adnormalities, The Heart, 6th edition. Edited by Hurst JW. New York, McGraw-Hill, 1986, pp 433–475
7. Laver MB, Turndorf H: Atrial activity and systemic blood pressure during anesthesia in man. Circulation 28:63–71, 1963
8. Haldemann G, Schaer H: Haemodynamic effects of transient atrioventricular dissociation in general anaesthesia. Br J Anaesth 44:159–162, 1972
9. Boba A: Significant effects on the blood pressure of an apparently trivial dysrhythmia. ANESTHESIOLOGY 48:282–283, 1978
10. Galindo A, Wyte SR, Wetherhold JW: Junctional rhythm induced by halothane anesthesia-treatment with succinylcholine. ANESTHESIOLOGY 37:261–262, 1972
11. Saarnivaara L, Kentala E, Kautto UM, Yrjola H: Electrocardiographic changes during microlaryngoscopy in practolol-pretreated patients under balanced anaesthesia. Acta Anaesthesiol Scand 30:128–131, 1986
12. Breslow MJ, Evers AS, Lebowitz P: Successful treatment of accelerated junctional rhythm with propranolol: Possible role of sympathetic stimulation in the genesis of this rhythm disturbance. ANESTHESIOLOGY 62:180–182, 1985
13. Sosis M, Cooper PS, Herr G: The diagnosis of "junctional rhythms" with halogenated anesthetics. ANESTHESIOLOGY 63:233–234, 1985
14. Segers M: Les phenomenes de synchronisation au niveau du coeur. Arch Int Physiol Biochim 54:87–106, 1946
15. Marriott HJL: Atrioventricular synchronization and accrochage. Circulation 14:38–43, 1956
16. Segers M, Lequime J, Denolin H: Synchronization of auricular and ventricular beats during complete heart block. Am Heart J 33:685–691, 1947
17. Fletcher E, Morton P, Murtagh JG, Bekheit S: Atrioventricular dissociation with accrochage. Br Heart J 33:572–577, 1971
18. Marriott HJL: Interactions between atria and ventricles during interference–dissociation and complete A-V block. Am Heart J 53:884–889, 1957
19. Wright CJ: Dysrhythmias during oral surgery: A comparison between halothane and enflurane anaesthesia. Anaesthesia 35:775–778, 1980

20. Ryder W, Wright PA: Halothane and enflurane in dental anesthesia. Anaesthesia 36:492–497, 1981
21. Lindgren L: ECG changes during halothane and enflurane anaesthesia for E.N.T. surgery in children. Br J Anaesth 53:653–662, 1981
22. Thomas VJE, Kyriakou KP, Thurlow AC: Cardiac arrhythmias during outpatient dental anaesthesia: A comparison of controlled ventilation with and without halothane. Br J Anaesth 50:1243–1245, 1978
23. Bertrand CA, Steiner NV, Jameson AG, Lopez M: Disturbances of cardiac rhythm during anesthesia and surgery. JAMA 216:1615–1617, 1971
24. Saarnivaara L: Comparison of halothane and enflurane anaesthesia for tonsillectomy in adults. Acta Anaesthesiol Scand 28:319–324, 1984
25. Saarnivaara L, Kentala E: Comparison of electrocardiographic changes during microlaryngoscopy under balanced anaesthesia induced by althesin or thiopentone. Acta Anaesthesiol Scand 24:321–324, 1980
26. Saemund O, Dalentius E: Pancuronium and nodal rhythm. Br J Anaesth 53:780, 1981

27. Cronnelly R, Morris RB, Miller RD: Edrophonium: Duration of action and atropine requirement in humans during halothane anesthesia. ANESTHESIOLOGY 57:261–266, 1982
28. Urquhart ML, Ramsey FM, Royster RL, Morrell RC, Gerr P: Heart rate and rhythm following an edrophonium/atropine mixture for antagonism of neuromuscular blockade during fentanyl/N₂O/O₂ or isoflurane/N₂O/O₂ anesthesia. ANESTHESIOLOGY 67:561–565, 1987
29. Azar I, Pham AN, Karambelkar DJ, Lear E: The heart rate following edronphonium–atropine and edrophonium–glycopyrrolate mixtures. ANESTHESIOLOGY 59:139–141, 1983
30. Takkkunen O, Salmenpera M, Heinonen J: Atropine vs. glycopyrrolate during reversal of pancuronium block in patients anaesthetized with halothane. Acta Anaesthesiol Scand 28:377–380, 1984
31. Walker WS, Winniford MD, Mauritson DR, Johnson SM, Hillis LD: Atrioventricular junctional rhythm in patients receiving oral verapamil therapy. JAMA 249:389–390, 1983
32. Wit AL, Hoffman BF, Rosen MR: Electrophysiology and pharmacology of cardiac arrhythmias: IX. Cardiac electrophysiologic effects of beta adrenergic receptor stimulation and blockade, Part C. Am Heart J 90:795–803, 1975

Fiberoptic Endobronchial Intubation for Resection of an Anterior Mediastinal Mass

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Growth of a cervical goiter through the thoracic inlet into the retrosternal space is a rare cause of an anterior mediastinal mass.[1] This uncommon lesion frequently presents with life-threatening respiratory obstruction.[2–4] It is not sensitive to radiation therapy and carries a mortality rate approaching 3% if not surgically corrected.[5,6] In common with other types of anterior mediastinal masses, loss of respiratory muscle tone may precipitate complete obstruction of the tracheobronchial tree, which may not be relieved by passage of an endotracheal tube through the vocal cords.[7,8] If this occurs, an intraoperative death may rapidly follow.[9] The case of a patient in whom fiberoptic endobronchial intubation during spontaneous respiration allowed safe induction of anesthesia for excision of a massive intrathoracic goiter is presented.

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CASE REPORT

A 60-yr-old, 178-cm, 120-kg woman was scheduled for urgent mediastinoscopy and median sternotomy for resection of an anterior mediastinal mass. Two months prior to admission she had noticed a progressive hoarseness of her voice, a continual sense of fullness in her upper chest, and a complete inability to sleep on her back. Three weeks prior to admission she developed audible wheezing and shortness of breath, which was so severe that she could not walk more than a few short steps. The patient had a long history of exogenous obesity.

Admission vital signs were as follows: blood pressure 130/80 mmHg, heart rate 80 beats/min, respiratory rate 26 breaths/min, and axillary temperature 37° C. Admission hemogram, serum electrolytes, liver function tests, coagulation profile, urinalysis, and ECG were completely within normal limits. Analysis of arterial blood gases (F_{IO_2} 0.2) revealed the following: Pa_{O_2} 46 mmHg, Pa_{CO_2} 71.4 mmHg, pH_a 7.27, % hemoglobin saturation 73. Analysis of arterial blood following administration of 2 l/min O_2 by nasal cannulae showed: Pa_{O_2} 73 mmHg, Pa_{CO_2} 66 mmHg, pH_a 7.39, % hemoglobin saturation 95.

A chest radiograph obtained at admission showed a left upper mediastinal mass with displacement of the cervical and thoracic trachea to the right; a small mass in the right hilum was also noted. An Iridium-131 thyroid scan revealed a cold nodule in the left lobe of the thyroid with an area of nonhomogeneous uptake extending into the mediastinum. Computerized tomography of the thorax revealed continuity of the cervical, mediastinal, and hilar masses with posterior displacement of the trachea and extrinsic compression of the carina. An aortogram demonstrated compression of the aorta and brachiocephalic vessels. Severe dyspnea prevented measurement of flow–volume loops in the upright or supine positions.