Literature Briefs

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Briefs were submitted by Drs. Vagn Askrog (Denmark), C. M. Ballinger, Peter P. Bosomworth, M. T. Clarke, R. B. Clark, Deryck Dun calf, J. E. Eckenhoff, E. A. Frayworth, Martin Helrich, G. Hohmann (Germany), J. J. Jacoby, F. C. McPartland, W. H. Mannheimer, A. S. Paterson, R. E. Ponath, Alan D. Randall, Norman Rosenbaum, and H. S. Roe. Abstracts of Japanese articles were obtained from Excerpta Medica Foundation. Briefs appearing elsewhere in this issue are a part of this column.

CARDIAC ARRHYTHMIAS Ventricular arrhythmias were produced by the inhalation of 1 per cent halopropane, 1 per cent halothane plus 10 per cent carbon dioxide, 1 per cent halopropane plus 1 µg./kg. of norepinephrine intravenously. The halopropane arrhythmia was abolished by decerebration or 0.01 mg./kg. of Hydergine but not by vagotomy or 2 mg./kg. of atropine. The halopropane-CO₂ arrhythmia was not abolished by any of the above procedures. It was abolished by spinal transection, 0.3 mg./kg. of Hydergine, 5 mg./kg. of dibenamine, or 2.5 mg./kg. of nethalide. Neither depletion of myocardial catecholamines nor adrenalectomy abolished the arrhythmia but a combination of these procedures did. The halopropane-norepinephrine arrhythmia was not blocked by spinal transection, vagotomy, 2 mg./kg. of atropine or depletion of myocardial catecholamines and adrenalectomy. Inhalation of 20 per cent cyclopropane resulted in arrhythmias when 10–15 per cent carbon dioxide was added to the inhaled mixture or 2–10 µg./kg. of norepinephrine was injected. These arrhythmias were similar to those seen with halopropane but were more difficult to produce, were at a lower frequency of ectopic beats and were easier to abolish. (Katz, R. L.: Cardiac Arrhythmias With Halopropane and Cyclopropane in the Cat, Fed. Proc. 23: 180 (March–April) 1964.)

VENTRICULAR ARRHYTHMIAS Recordings of the electrogram of the intact right atrium, bundle of His and both ventricles, as well as of isolated anterior papillary muscle, right ventricular wall and false tendons of the latter, indicated that arrhythmias induced in cyclopropane anesthetized dogs following appropriate doses of epinephrine always originated distal to the His bundle. Vagal stimulation did not convert these arrhythmias. Although the exact mechanism of the bigeminal beats could not be analyzed by this study, the monofocal tachycardias appeared to result from increased pacemaker activity in the ventricles. (Moore, E. N., Morse, H. T., and Price, H. L.: Cardiac Arrhythmias Produced by Catecholamines in Anesthetized Dogs, Circ. Res. 15: 77 (July) 1964.)

ARRHYTHMIA Petroleum ether, halothane and possibly other sensitizers interfere with the stabilizing action of endogenous acetylcholine on the ventricle and allow the stimulating action of adrenaline to produce severe fibrillatory rhythms by an increase in ventricular automaticity. (MacLeod, D. P., and Reynolds, A. L.: Effect of Acetylcholine on Adrenaline-Induced Subatrial Rhythms in the Sensitized Cat Heart, Canad. J. Physiol. 42: 431 (July) 1964.)

ARRHYTHMIAS The incidence of postoperative arrhythmias in 60 consecutive patients following 62 open heart procedures for correction of cyanotic tetralogy of Fallot was studied by electrocardiographic monitoring. The incidence of arrhythmias following repair of tetralogy of Fallot was 40 per cent, the most common types being supraventricular tachy-
cardia, A-V dissociation and nodal rhythm; the incidence was only 20 per cent in patients over 18. By comparison, patients who had repairs of uncomplicated interventricular septal defects had an incidence of only 9 per cent arrhythmias (41 per cent in those with associated pulmonary hypertension), indicating the right ventriculotomy per se was not the primary causative factor. Precipitating factors fell into three categories: (1) acidoses and/or anoxia, (2) drugs (digitalis intoxication usually) and (3) operative trauma. Depending on the blood gas values, the management of acidosis involved assisted ventilation and/or intravenous administration of sodium bicarbonate. Digitalis intoxication enhanced by cardiopulmonary bypass occurred in 8 instances with one death; now digoxin is seldom given before the fourth postoperative day. Arrhythmias due to "operative trauma" usually followed atrioptomy for closure of a large patent foramen ovale or atrial septal defect. (Popper, R. W., and others: Arrhythmias After Cardiac Surgery. II Cyanotic Tetralogy of Fallot, With Comments in Regard to Ventricular Septal Defect, Amer. Heart J. 68: 32 (July) 1964.)

CARDIAC ARRHYTHMIA In dogs anesthetized with chloroform, infusion of epinephrine caused bigeminy, irregular ventricular ectopic beats, or multifocal ventricular tachycardia. Vagal stimulation with resultant cardiac slowing could return sinus rhythm. Restoring the heart rate by driving the atria or stopping vagal stimulation induced arrhythmias. Heart rate is a factor in the production of ventricular ectopic activity in dogs during chloroform anesthesia. Although such activity occurs more readily when the A-V node and the upper bundle are intact, it can occur when that area is functionally disconnected from the ventricles. Ectopic foci or re-entry excitation might be the mechanisms responsible for this phenomenon. (Vick, R. L.: Role of Heart Rate in Chloroform-Epinephrine Induced Cardiac Arrhythmia, Fed. Proc. 23: 326 (March–April) 1964.)

ATRIAL FIBRILLATION Resistance of isolated rabbit atria to arrhythmic factors such as low potassium medium, acetylcholine, and electrical stimulation was determined before and after contact with reserpine and following large doses of adrenaline. Animals pretreated with reserpine were more sensitive to arrhythmic factors than untreated controls. Adrenaline abolished the induced arrhythmias or lowered the frequency of atrial firing rate. This antiarrhythmic property of adrenaline at the atrial level is the result of an antagonism with acetylcholine, presumably through a lengthening of the duration of the action potential. (Lavalle, M., Tremblay, G., and Beaudnes, A.: Effects of Reserpine and Adrenaline On Atrial Fibrillation, Canad. J. Physiol. 42: 385 (July) 1964.)

VENTRICULAR FIBRILLATION Two cases are reported in which electroshock proved ineffective but in which procaine amide reestablished sinus rhythm. Electroshock should remain the standard treatment of ventricular fibrillation but if it fails procaine amide may be useful. The recommended dose is 100 mg. intravenously with continuous electrocardiographic control. Administration should be continued until the fibrillation yields or heart block occurs. Series of electroshocks should be interposed between doses. (Kristoffersen, M.: Electroshock-Resistant Ventricular Fibrillation Treated With Procaine Amide, Danish Med. Bull. 11: 127, 1964.)

CARDIAC OUTPUT A new water-soluble organic dye of low toxicity, Coomassie blue, has been used in the indicator dilution method for the determination of blood volume and cardiac output. Mean plasma volume was determined to be 46.3 ml./kg., mean blood volume 77.4 ml./kg. and mean cardiac output 6.1 liters/minute. Effects of various anaesthetic agents on cardiac output was determined. Although previous investigations have suggested that the hypotension associated with halothane is due to a direct depressant effect of the drug on the heart, this study showed that, even in the presence of hypotension, the cardiac output is often raised, and it proved impossible to establish a direct relationship between the blood pressure and cardiac output. Only when the inspired concentration of halothane was raised well above that required for clinical anaesthesia for prolonged periods was there any