MANAGEMENT OF CARDIAC ARRHYTHMIAS DURING ANAESTHESIA*

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WHEN a continuous electrocardiogram is recorded during surgical operations which involve general anaesthesia, 70 to 80 per cent of the patients will show disorders of cardiac rate and rhythm. Most of these arrhythmias have little clinical importance. To be feared are those which lead to a decline in blood pressure or those which may be a prelude to ventricular fibrillation or cardiac arrest. Specific treatment with antiarrhythmia drugs requires first an accurate diagnosis, reached by interpretation of the electrocardiogram. Observation of the pulse by palpation or auscultation during blood pressure measurements provides little information on the presence or nature of cardiac arrhythmias.

There are two situations which focus the attention of the anaesthetist on cardiac arrhythmias. First, "poor-risk" patients are not denied needed surgery because they have cardiovascular disease. Then, more recently, the manoeuvres devised for repair of cardiac valves or septa are accompanied by ominous changes in cardiac rhythm. The anaesthetist is accustomed to coping with rapid changes in the patient's physiological status, and is in the best position to provide therapy when there is deterioration of cardiac function. While the cardiologist should be consulted freely, he rarely can be present throughout surgical operations.

ANAESTHETIC AGENT AND DISTURBANCES OF CARDIAC RHYTHM

During ether or nitrous oxide anaesthesia cardiac arrhythmias are usually of the innocuous type arising chiefly from the supraventricular centres. Sino-atrial or atrioventricular nodal tachycardias are the most common. Atrial extra systoles may also appear. Serious arrhythmias, whose origins are in the ventricles, are seldom evident. During induction of anaesthesia with chloroform, slowing of cardiac rate (bradycardia) and cardiac arrest may occur. With the thiobarbiturates also, Pentothal, Surital or Kemithal, there are episodes of sinus arrhythmia and slowing of cardiac rate. These arrhythmias, seen with chloroform, trichloroethylene, or the thiobarbiturates, may be provoked in experimental animals by stimulation of the vagus nerve. It has been assumed that, until an adequate depth of anaesthesia has been reached, vago-vagal and tracheocardiac reflexes are elicited on passing an endotracheal tube or caused by the irritating nature of the anaesthetic gas mixture. During deep chloroform anaesthesia the vagus is quiescent and aberrant ventricular systoles may appear at that time. However, the ventricular fibrillation, which occurs with animals who are given adrenalin during chloroform administration (1), is seldom observed in man. As more experience has been gained with cyclopropane, there has been a greater facility in the control of the ventricular arrhythmias which accompany its use. Nevertheless, ventricular tachycardia (and possibly ventricular fibrillation) can occur with

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any duration or depth of anaesthesia with cyclopropane if adrenalin is released or administered. In experimental animals a dose of 10 microgm./kgm. of adrenalin suffices to cause ventricular arrhythmias—it is believed that this amount can be released from a patient's adrenals by manipulations in that area. Thus, chloroform or cyclopropane anaesthesia is hazardous during operations for pheochromocytoma.

HYPOXIA AND HYPERCARBIA AS FACTORS INITIATING CARDIAC ARRHYTHMIAS

Denson and Joseph (2) attribute the majority of the arrhythmias which occur during induction to hypoxia. The arrhythmias consisted of premature ventricular systoles and ventricular tachycardia. All arrhythmias reverted to normal sinus rhythm on improvement of pulmonary ventilation. The duration of apnea which resulted from endotracheal intubation was only 18 to 71 seconds. They suggest that tracheal-initiated reflexes are not involved because the appearance of the arrhythmias was delayed for several minutes after passing the tube. Johnstone (3) also reports that hypoxia provoked arrhythmias in all patients and in varying stages of anaesthesia. Again, the predominant arrhythmias seen were couplet, ventricular extrasystoles and ventricular tachycardia. He stresses the view that spontaneous respirations during cyclopropane should be assisted at all times. Johnstone also reports that the accumulation of carbon dioxide (hypercarbia) is accompanied by ventricular arrhythmias in most patients. Removal of the soda-lime absorber for periods of time (as short as 10 to 15 minutes) will stimulate changes in cardiac rhythm. Hypoxia and accumulation of carbon dioxide are so frequently the cause of cardiac arrhythmias that some anaesthetists teach that no antiarrhythmia drugs are needed, provided due attention is paid to maintaining an adequate oxygen-carbon dioxide balance.

CLASSIFICATION OF DISTURBANCES OF CARDIAC RHYTHM

Since identification of the cardiac arrhythmia must precede rational therapy, a review of the classification of arrhythmias is pertinent. Arrhythmias may arise from the sino-atrial or atrioventricular nodes or be atrial or ventricular in origin. Activity of the vagus will inhibit the sino-atrial node; bradycardia, sinus pauses, and even complete asystole may result. If preanaesthetic medication with belladonna alkaloids is inadequate or the patient's vagal reflex unduly sensitive, the intravenous administration of a Banthine-like drug is suggested. Vagal-induced asystole apparently does not occur without cardiac ischemia or excessively deep anaesthesia. The prophylaxis and management of this cardiac emergency has been admirably discussed in a recent issue of this journal by Fortin and Letienne (4). The term supraventricular tachycardia focuses attention on a rapid cardiac rate. It includes rapid rhythm and arrhythmias arising from either node or the atrial myocardium (atrial flutter or fibrillation). Supraventricular arrhythmias generally require no treatment unless the patient's cardiac reserve is minimal. If a supraventricular tachycardia is allowed to persist in these patients, evidence of a "strain" pattern may appear in the electrocardiogram. As a result of cardiac ischemia a coronary infarct may be precipitated. Then, too, the rapidly beating heart may exceed its compensation and acute congestive failure can be the result. A rapidly acting digitalis glycoside may be employed, the object being

to ensure a slow, albeit still irregular, rhythm. Although cardiac output and efficiency usually follow successful conversion to normal sinus rhythm from atrial fibrillation or flutter, quinidine therapy during anaesthesia is too dangerous. In fact, because of the high oxygen tensions possible during anaesthesia, auricular fibrillation may disappear. Aberrant ventricular systoles, even if coupled into bigeminal or trigeminal rhythms, are treated only because it is believed that they are premonitory of ventricular tachycardia or ventricular fibrillation. During ventricular tachycardia blood pressure falls and with ventricular fibrillation there is circulatory failure. Procaine amide is the drug of current choice to arrest ventricular extrasystoles as prophylaxis against the serious ventricular arrhythmias. Ventricular fibrillation requires opening of the thorax, cardiac massage, and use of electrical defibrillators.

PHARMACOLOGY OF ANTIARRHYTHMIA DRUGS

Procaine has for some years been applied topically by the surgeon prior to manipulations of the heart and great vessels, with a reduction of cardiac arrhythmias. When it was realized that procaine could be given intravenously, this route of administration was adopted for prophylaxis of cardiac arrhythmias. Since procaine is rapidly hydrolysed and inactivated by serum esterases, it must be given by continuous drip to be effective. Procaine amide (Pronestyl) was synthesized as a substitute for procaine as it is slowly destroyed in the body—its duration of action may be as long as 3 hours. Diphenhydramine (Benadryl) is chemically quite similar to procaine and has a very stable ether linkage. Benadryl has proven effective in preventing ventricular tachycardia or ventricular fibrillation in experimental animals (5) and clinical trial is indicated. Pronestyl is primarily of value in the control of ventricular arrhythmias. Peters, Newman, and Burstein (6) employed a dose of 300 mgm. of Pronestyl and found a significant reduction in the number of arrhythmias which occurred during induction of anaesthesia with cyclopropane. Joseph and coworkers (7) recommend its use for prophylaxis and therapy of ventricular arrhythmias during thoracic surgical procedures. There is, however, doubt that Pronestyl can actually prevent ventricular "flutter" or ventricular fibrillation. If it cannot, then there is little justification for its use in control of alarming but not serious premature ventricular systoles. Experimentally induced ventricular fibrillation in animals may be controlled only by doses of Pronestyl in excess of 40 mg./kg. (a dosage in excess of that permitted in patients). Pronestyl is to be given intravenously no more rapidly than 200 mgm. (2 cc.) per minute and no more than 0.5–1.0 gm. at any one time. Even with this dose hypotension may occur, although the drop in systolic pressure does not usually exceed 40 mm. of Hg. An additional side action of Pronestyl is the result of slowing of myocardial conduction. Following an overdose, bundle branch block may appear and proceed to ventricular fibrillation. If the QRS interval is prolonged more than 20 per cent of the initial value, drug injection should be stopped. Pronestyl also may prolong the QT interval and flatten, notch, or invert the T wave. Despite its defects, Pronestyl is currently preferred to quinidine gluconate. Hypotension and conduction disturbances with intravenously administered quinidine are common.

Rapidly acting digitalis glucoside preparations are frequently needed to slow

the heart rate with supraventricular tachycardias. Usually lanatocide C (Cedelanide) or ouabain are employed. With these glycosides the onset of action may require 10 to 30 minutes, the peak effect being delayed for 45 to 180 minutes. With drugs having such a long lag period before full action develops, overdosage or underdosage will be a common occurrence. Recently, acetyl strophanthidin has been introduced. This drug has a rapid but brief action, the new digitalis substance, when injected intravenously, has an onset of action in $\frac{1}{2}$ to 5 minutes and peak effects within 10 to 12 minutes. Potentially dangerous effects from an overdose persist but 30 minutes. Currently (8) this new drug is being employed as a "tolerance test" to ascertain the degree of digitalization of patients whose digitalis status is in doubt. Enselberg and coworkers (9) have found it valuable to control the rapid arrhythmias arising during surgical operations. The myocardial sensitivity to digitalis may change abruptly as a result of stresses, fluid and ionic changes which occur during surgery. Acetyl strophanthidin should prove a valuable aid to the anaesthetist in the control of rapid rhythms or acute congestive failure which may develop.

Digitalis overdosage results in a variety of cardiac arrhythmias, the most common being ventricular extrasystoles and ventricular tachycardia. It is conceivable that during surgery the patient's myocardial sensitivity to digitalis may increase and account for these and other arrhythmias. Toxic doses of digitalis liberate potassium from within the myocardial cell. Loss of potassium will occur through the kidney or through fluid balance shifts. Administration of potassium promptly reverses the ventricular arrhythmias of digitalis overdosage (10). Potassium chloride may be injected slowly intravenously until the arrhythmia ceases or evidence of hyperkalemia appears in the electrocardiogram. Toxic doses of potassium, hyperkalemia, produce a characteristic high, peaked T-wave and beginning signs of heart block. If administration of potassium be continued, complete electrical depolarization of the heart will take place. This toxic action has been adapted for therapeutic purpose. Ventricular fibrillation may occur during hypothermic anaesthesia. Swan and Virtue inject potassium into the fibrillating heart until complete depolarization occurs—the excess potassium is flushed into the circulation by direct massage of the heart. If normal spontaneous rhythm does not appear as a result of the massage the injection of calcium gluconate usually produces the desired effect. In their experience the use of electrical defibrillators has been disappointing. The role of potassium on the actions of the heart is still poorly understood but should be considered in evaluating clinical cardiac arrhythmias

SUMMARY

Disorders of cardiac rhythm may be described as supraventricular or ventricular in origin. The former for the most part are innocuous. Ventricular premature systoles may be the forerunner of ventricular tachycardia or ventricular fibrillation. Ventricular tachycardia if persistent is accompanied by failing circulation. The majority of the arrhythmias observed during anaesthesia can be attributed to hypoxia or accumulation of carbon dioxide. In the opinion of certain anaesthetists, if these two causes of arrhythmia are avoided there is no need for an antiarrhythmia drug. Procaine amide has proven of value in the control of the

ventricular arrhythmias which occur during cyclopropane anaesthesia. Rapidly acting digitalis preparations, particularly acetyl strophanthidin, may be needed to slow a rapidly beating heart (supraventricular tachycardias). The ventricular arrhythmias which accompany digitalis toxicity are caused by loss of intracellular potassium. Potassium chloride is effective in the control of digitalis-induced arrhythmias and should be considered in other instances as well.

RÉSUMÉ

On peut décrire les troubles du rythme cardiaque comme étant d'origine supra-ventriculaire ou ventriculaire. Les premiers sont pour la plupart inoffensifs. Des systoles ventriculaires prématurées peuvent être les signes avant-coureurs d'une tachycardie ou d'une fibrillation ventriculaires. Si la tachycardie ventriculaire persiste, elle s'accompagne d'insuffisance cardiaque. La majorité des arythmies observée en cours d'anesthésie peuvent être attribuées à l'hypoxie ou à l'accumulation d'anhydride carbonique. Selon certains anesthésistes, si l'on évite ces deux causes d'arythmie, il n'est nul besoin d'employer une médication contre l'arythmie. La procaine amide a été reconnue utile pour juguler les arythmies ventriculaires qui se produisent pendant l'anesthésie au cyclopropane. On peut avoir besoin de préparations à action rapide à base de digitale, en particulier de l'acétyl-strophanthidine, pour ralentir les battements du cœur trop rapides (tachycardie supraventriculaire). Les arythmies ventriculaires qui accompagnent la toxicité de la digitale sont dues à une perte de potassium intra-cellulaire. Le chlorure de potassium est efficace pour juguler les arythmies provoquées par la digitale et son emploi doit être aussi bien envisagé dans d'autres cas.

REFERENCES

- 1 MELVILLE, K I The Protective Action of Atabrine against Chloroform-Adrenaline Ventricular Fibrillation. *J. Pharm & Exper. Therap.* 87: 350 (1946).
- 2 DENSON, J S & JOSEPH, S. I Cardiac Rhythm and Endotracheal Intubation—A Clarification. *Anesthesiol* 15. 650 (1954).
- 3 JOHNSTONE, M Cyclopropane Anaesthesia and Ventricular Arrhythmias. *Brit. Heart J.* 12 239 (1950).
- 4 FORTIN, G & LETIENNE, R Prophylaxis and Treatment of Cardiac Arrest. *Canad. Anaes. Soc J* 1 24 (1954)
- 5 McCORMICK, T E, McCawley, E L, & DAVID, N A. Factors affecting Cyclopropane Cardiac Arrhythmias In press.
- 6 PETERS, N S., NEWMAN, W., & BURSTEIN, C L. Electro cardiographic Studies during Endotracheal Intubation. IV. Effects during Cyclopropane or Cyclopropane-Ether Anesthesia and Intravenous Procaine Amide. *Anesthesiol.* 12 673 (1951).
- 7 JOSEPH, S. I., HELRICHS, M, RAYDEN, H J, ORKINE, L R, & ROVENSTINE, E. A. Procaine Amide for Prophylaxis and Therapy of Cardiac Arrhythmias during Thoracic Surgery. *Surg, Gynec & Obst* 93 75 (1951)
- 8 LOWN, B & LEVINE, S A Current Concepts in Digitalis Therapy. *New Eng. J. Med.* 250. 866 (1954).
- 9 ENSELBERG, C D, ALTCHER, M R, & HELLMAN, E. Action of Acetylstrophanthidin in Rapid Cardiac Arrhythmias. *Am. Heart J* 40. 919 (1950).
- 10 ENSELBERG, C D, SIMMONS, H. G., & MINTZ, A A Effects of Potassium on Heart with special reference to the Possibility of Treatment of Toxic Arrhythmias due to Digitalis. *Am Heart J* 39 713 (1950).