

POSTOPERATIVE CARDIAC ARRHYTHMIAS

JOSEPH J. BUCKLEY, M.D., AND J. ALBERT JACKSON, M.D.

POSTOPERATIVE cardiac arrhythmias can be expected to occur with increasing frequency for several reasons. First, the average surgical patient tends to be older than in the past, due to the remarkable increase in life expectancy. In addition, the rapid advances in ancillary surgical areas have extended the safety of surgery to the point that age is no longer a deterrent to surgical therapy. Secondly, cardiovascular surgery is now undertaken for a wide range of congenital and acquired defects which were previously considered inoperable.

When viewed upon this background a consideration of cardiac arrhythmias occurring in the postoperative period seems appropriate. The reduced cardiac reserve characteristic of aging makes rhythm disturbances more likely and more hazardous. Prompt recognition and skillful treatment may determine the success of the surgical procedure.

GENERAL INCIDENCE

Information on the incidence of *postoperative* arrhythmias is limited. The first report dealing specifically with rhythm disturbances after operation was published by Levine²⁰ in 1920. He cited 7 cases observed within a short period of time; no incidence is mentioned however. In 1941, Taylor²¹ reported upon complications following 39,000 cyclopropane administrations and described postoperative arrhythmias in about 5 per cent of the cases. Two years later, Currens and associates¹² studied arrhythmias *following thoracic surgery*, and stated that statistics were not available on the incidence of postoperative arrhythmias, but that they probably occurred infrequently and were usually of little consequence. Turville and Dripps²² in a discussion of postoperative complications following anesthesia and surgery in over 1,000 aged patients stated that disorders of cardiac rhythm were common but quali-

fied their remark by pointing out that geriatric patients commonly exhibit rhythm disturbances in the absence of surgical disease. Fisher and Winsor¹⁷ recorded electrocardiograms on 52 patients after thoracic and abdominal operations and found that six (11.5 per cent) exhibited abnormal rhythm. This high incidence must be viewed in the light that 63 per cent of their patients had abnormal preoperative electrocardiograms and 11 per cent had arrhythmias before operation. Rogers *et al.*²³ studied the incidence of *supraventricular tachycardia* among 28,000 patients after operation. They discovered 43 instances of this irregularity, an incidence of less than 0.2 per cent. This figure is probably low since the authors admit to knowledge of additional cases of postoperative tachycardia (not confirmed by electrocardiograms) not included in the report. Keating²⁷ claims that disturbances of cardiac rhythm occur after operation in about 1 per cent of patients; he reported no data. Recently, Wheat and Burford²⁴ surveyed 439 patients recovering from thoracic operations and noted that 20 to 30 per cent exhibited cardiac arrhythmias.

There are less data relative to the incidence of arrhythmias in the *immediate* postanesthetic period. Buckley *et al.*¹¹ in an electrocardiographic survey of 100 consecutive patients in the recovery room observed 45 instances of arrhythmia (table 1). Sinus tachycardia was present in 32 cases; the next most common arrhythmia was occasional premature ventricular contractions. One case of trigeminy was seen.

It becomes obvious that with the exception of those figures available in patients subjected to pulmonary operations (to be discussed below) we have very little statistical information upon the incidence of cardiac irregularities associated with or incident to surgical intervention. In all likelihood however, if we overlook the sinus tachycardias, the over-all incidence of serious postoperative arrhythmia is low; but predisposing factors such as heart disease, age, site of operation, posture and

Dr. Buckley is Professor of Anesthesiology, and Dr. Jackson is Clinical Instructor, Department of Anesthesiology, University of Minnesota College of Medical Sciences, Minneapolis, Minnesota.

TABLE 1
INCIDENCE OF CARDIAC ARRHYTHMIAS
IMMEDIATELY AFTER SURGERY
IN 100 PATIENTS

Type of Arrhythmia	Patients (Per Cent)
Sinus arrhythmia	3
Sinus bradycardia	2
Sinus tachycardia	32
Premature ventricular contractions	
Occasional	7
Trigeminy	1

respiratory insufficiency can be expected to provide exceptions to the rule.

TYPES OF POSTOPERATIVE ARRHYTHMIAS

Arrhythmias are most commonly detected as a result of the aberrations which they produce in the peripheral pulse. Therefore a classification of abnormal rhythms based upon rate and rhythmicity has been prepared (table 2) with the hope that it may be of greater value at the bedside than a more complex classification based upon abnormal sites of impulse formation or pathways of conduction.

In the sections below each arrhythmia will be discussed with respect to incidence and diagnostic highlights. Emphasis will be placed upon surgical and anesthetic events known to contribute to the incidence of arrhythmias. Some therapeutic considerations will be made, especially for those arrhythmias considered serious enough to warrant prompt treatment. Except for special instances, arrhythmias which produce only subtle derangements in the electrocardiogram will not be discussed.

ARRHYTHMIAS CHARACTERIZED BY A RAPID, REGULAR PULSE

Sinus Tachycardia. Sinus tachycardia is not an arrhythmia by definition but it does represent a disturbance of the normal rhythm of the heart. The cardiac impulses arise normally at the sinus node and are distributed in an orderly, normal pattern throughout the heart; the only unusual feature of this rhythm is its rapidity. Most authorities consider an adult heart rate in excess of 100 beats per minute to represent tachycardia. In children there is less unanimity of opinion; table 3 lists the maximum rates considered normal by Katz²³ and Kaplan,²⁵ above which, tachycardia is said

to exist. Usually, the heart rate in sinus tachycardia does not exceed 150 in adults, but a rate of 180 or higher may be observed.¹⁴

Sinus tachycardia is the most common arrhythmia observed in the immediate postoperative period. It occurred in 32 per cent of the reviewers series.¹¹ The diagnosis usually presents no difficulty; a rapid, regular pulse, usually below 150, which alters in rate and regularity with the phasic changes of respiration is usually sinus tachycardia. The arrhythmia is frequently accompanied by other clinical signs such as fever, and pallor, which relate to the etiology of the tachycardia and make the diagnosis easy. With rates above 150, sinus tachycardia can be confused with auricular or nodal tachycardia, ventricular tachycardia, or auricular flutter with block. In such circumstances, the electrocardiogram will usually separate the entities. Other differential diagnostic points will be considered

TABLE 2

POSTOPERATIVE ARRHYTHMIAS

Arrhythmias Characterized by a Rapid Regular Pulse
Sinus tachycardia
Supraventricular tachycardia
(1) atrial paroxysmal tachycardia
(2) nodal paroxysmal tachycardia
Atrial flutter
Ventricular paroxysmal tachycardia
Arrhythmias Characterized by a Slow Regular Pulse
Sinus bradycardia
Atrioventricular nodal rhythm
Atrial standstill with A-V nodal escape
Incomplete heart block
Complete heart block
Arrhythmias Characterized by an Irregular Pulse
Sinus arrhythmia
Sino-atrial block
Premature Contractions
(1) atrial premature contractions
(2) nodal premature contractions
(3) ventricular premature contractions
Atrial fibrillation
Ventricular fibrillation
Second degree A-V block (Wenckebach phenomenon)

TABLE 3

MAXIMUM NORMAL HEART RATES IN CHILDREN

Age	Katz ²⁰	Kaplan ²¹
Birth	150	170-180
2 years	125	130
4 years	115	120
6 years	105	115

in the sections dealing with the ectopic tachycardias.

Sinus tachycardia is the result of increased sympathetic tone or decreased vagal tone. Perhaps the three most common etiologic factors observed in the postoperative period are hypoxia, pain and hypovolemia due to blood loss. All provoke increased sympathetic activity.

Hypoxemia is a classical cause of sinus tachycardia in the postanesthetic patient. The hypoxia may be a consequence of hypoventilation,^{20, 21} pneumothorax, atelectasis,¹⁹ or respiratory obstruction. *Hypercapnia* due to hypoventilation may produce sinus tachycardia through sympathetic nerve stimulation.²² *Dehydration* frequently produces sinus tachycardia. Recently a sinus tachycardia of 180 was observed in a 3 year old child who had undergone a relatively bloodless tonsillectomy. The child had received virtually no fluid for 18 hours before operation; dehydration had produced hypovolemia sufficient to invoke the tachycardia; infusion of fluid lowered the rate to 115 in 2 hours.

Drugs are commonly the cause of sinus tachycardia. Meperidine, gallamine and atropine are perhaps most frequently encountered in this relation. The reviewers attribute the high incidence (32 per cent) of sinus tachycardia in their series to the use of gallamine, although postoperative pain may have participated. Residual adrenergic effects of ether may be responsible for postoperative tachycardia. Epinephrine or other chronotropic cardiac stimulants and antihypertensive drugs such as dibenzylamine, the nitrites and hydrazinophthalazine (Apresoline) produce rapid heart action. Occasionally digitalis intoxication produces sinus tachycardia. *Hyperthermic states, extreme toxicity and burns* are almost routinely accompanied by tachycardia. *Thyrotoxicosis*, especially thyroid storm, characteristically is associated with extreme tachy-

cardia and should always be suspected when an elevated cardiac rate is encountered. Finally *early congestive heart failure* will sometimes manifest itself by tachycardia.

Sinus tachycardia is a protective homeostatic response to other abnormal clinical states. Therapy necessarily should be directed toward the latter; attempts to treat the symptom of rapid heart action will either be ineffectual or may be harmful.²

Supraventricular Paroxysmal Tachycardia. Under this heading atrial paroxysmal tachycardia and nodal paroxysmal tachycardia will be discussed. In the strictest sense, this heading should include atrial flutter and atrial fibrillation; however, the latter arrhythmias will be discussed separately because their incidence is higher in postoperative patients and they have greater therapeutic and prognostic importance.

Atrial and nodal tachycardia are difficult to distinguish from one another even electrocardiographically. The clinical description which follows should be considered applicable to both of these rhythms.

These arrhythmias are characterized by an *abrupt onset*, which aids in differentiating them from sinus tachycardia. The heart rate is usually more rapid as well, ranging between 140 and 220 beats per minute. The rhythm is almost always *regular*, in contrast to ventricular tachycardia, which usually has a slight but definite irregularity. Associated with the change in pulse rate, about 16 per cent of patients complain of precordial anginal pain.²⁴ Occasionally cyanosis appears as a result of pulmonary congestion; if the rate exceeds 180 to 200 per minute signs of vascular collapse may ensue if there is a reduction in cardiac output.

As discussed earlier the incidence of supraventricular paroxysmal tachycardia in the postoperative period is apparently low. Atrial and nodal paroxysmal tachycardia may be caused by digitalis toxicity, quinidine overdosage, congestive heart failure and rarely, as a result of myocardial infarction.²⁴ Occasionally it is a manifestation of *thyrotoxicosis*; it occurred in about 5 per cent of 9,950 cases of thyroid disease.²⁴ *Hypoxemia* produced by atelectasis, pneumothorax and sudden mediastinal shift has been suggested as an etiologic factor.¹²

*These arrhythmias are more likely to occur postoperatively in patients with a history of previous attacks.*⁴³

Paroxysmal supraventricular tachycardia (nodal and atrial) does not usually demand emergency therapy unless a very rapid ventricular rate is produced. Classically, this arrhythmia responds to *strong vagal stimulation* accomplished through the application of digital pressure over the carotid sinus.^{21, 40, 41, 42} Ocular pressure is also effective in stopping attacks. Reynolds⁴⁰ suggests that augmentation of the vagal stimulation may be obtained by "priming" the patient with 0.5 mg. of neostigmine given subcutaneously fifteen to thirty minutes prior to carotid sinus or ocular pressure. Methacholine chloride (Mechoyl) is occasionally used to produce vagal stimulation but toxic reactions are frequent.⁶ These "vagal maneuvers," so effective in controlling this arrhythmia in the nonsurgical patient, often fail when it is encountered postoperatively.⁴² *Vasopressor drugs* such as phenylephrine or methoxamine may be tried;^{40, 43} the elevation of blood pressure produced by these drugs increases vagal tone through carotid and aortic sinus reflex stimulation. *Procaine amide* is said to terminate 80 per cent of cases through a quinidine-like action.⁶ When vagal stimulation or procaine amide fail to arrest the arrhythmia, digitalis is indicated.^{21, 23, 42} Quinidine^{42, 44} and the simultaneous administration of digitalis and quinidine^{18, 28} have been recommended for resistant cases. When digitalis intoxication is the precipitating factor, infusion of potassium solution may be effective.^{21, 23, 47} The problem of digitalis intoxication and its associated arrhythmias will be discussed below.

Atrial Flutter. Atrial flutter is the result of rapid formation of ectopic auricular stimuli at a rate varying from 200 to 380 per minute. In most cases, only a fraction of these auricular impulses are transmitted to the ventricles so that palpation of the radial pulse often yields a rate of 70 to 160 beats per minute. Thus, if atrial flutter with a 2:1 block occurs, the atria contract 300 times a minute while only 150 ventricular responses result; the pulse at the wrist is 150. If the block is 4:1, then the radial pulse is only 75. In the former instance, the differential diagnosis must be made between flutter and sinus tachycardia, atrial or

nodal tachycardia, ventricular tachycardia and paroxysmal auricular fibrillation. When a slow ventricular rate is present (as in the 4:1 block) the arrhythmia may not be noticed and normal sinus rhythm be thought to exist or auricular fibrillation with a slow ventricular rate may be erroneously suspected. Rarely atrial flutter without A-V block occurs; the ventricular rate then may be 200 or above.

The application of carotid sinus pressure is a helpful diagnostic aid in cases of flutter with a rapid ventricular rate; the ventricular rate slows markedly but speeds up again when the carotid pressure is released. In contrast, carotid compression will usually permanently convert an atrial or nodal paroxysmal tachycardia to sinus rhythm; it will have no effect on ventricular tachycardia or auricular fibrillation.

In cases with a slow ventricular rate auscultation of the heart may reveal the rapid, faint heart sounds due to auricular flutter. The rapid auricular rate can usually be observed in the jugular vein pulsations. In flutter, the ventricular rhythm is usually regular, which helps to differentiate the condition from auricular fibrillation. The electrocardiogram will almost always make the diagnosis clear.

Atrial flutter is encountered more frequently in patients over 60 years of age. Pre-existing cardiovascular disease, especially arteriosclerosis, increases the likelihood of postoperative atrial flutter.^{12, 29} Intrathoracic surgery is associated with a significantly greater incidence of this arrhythmia;^{29, 44} carbon dioxide accumulation and electrolyte imbalance, which so frequently accompany thoracic disease and operation, have been cited as predisposing factors.²⁹ Hypoxemia,^{29, 32} vagal nerve stimulation,^{29, 32, 44} hypotension²⁹ and mediastinal shift^{12, 29, 44} are thought to contribute to the occurrence of this arrhythmia. Patients with mitral stenosis,¹⁸ hyperthyroidism¹⁸ and diabetic acidosis⁶ are subject to flutter when subjected to surgical procedures.

This arrhythmia may be precipitated by the injection of epinephrine or ephedrine; digitalis rarely produces flutter.

Atrial flutter has serious prognostic implications for two reasons: (1) it is usually superimposed upon a diseased heart and (2) it frequently causes congestive heart failure.⁴⁴ The

likelihood of this complication is heightened when the ventricular rate produced by the flutter is rapid. Therefore, most authorities recommend termination of the arrhythmia. The drugs most useful for this purpose are digitalis, quinidine and procaine amide in that order.^{6, 12, 22} Digitalis usually converts the flutter to atrial fibrillation from whence normal sinus rhythm will usually return following withdrawal of digitalis. Quinidine is a less desirable treatment because of its tendency to decrease the degree of A-V block (by vagolytic action) at the same time that it is slowing the auricular rate; the ventricular rate consequently may rise temporarily as a 4:1 block changes to a 2:1 block; this may worsen rather than improve the patient's clinical condition. Procaine amide is sometimes useful in converting flutter to sinus rhythm but the high dosage often required may produce serious cardiac depression.

Obviously precipitating factors such as hypoxia or mediastinal shift should be corrected simultaneously.

Ventricular Paroxysmal Tachycardia. Fortunately, this is a rare arrhythmia. When it occurs, it has serious implications because it is almost always superimposed upon a seriously damaged heart; in addition, it may progress to ventricular fibrillation if not treated promptly and correctly.

The pulse rate is usually between 140 and 180. The patient may complain of anginal pain; if coronary occlusion has precipitated the arrhythmia, vascular collapse and a shock-like state may be present. There is usually a detectable irregularity in the rhythm but it is slight and must be sought for carefully (usually on ECG measurement). Of greatest help in the diagnosis is the fact that it is unaffected by carotid sinus pressure.

This arrhythmia may simulate supraventricular tachycardia closely and proper therapy requires accurate electrocardiographic differentiation between the two disturbances. Even then the diagnosis may be difficult.⁶

Data upon the incidence of this arrhythmia in postoperative patients are limited. Kroznick²⁹ mentioned one case among 10 serious arrhythmias in 82 postoperative patients; Likoff³¹ refers to its frequency following aortic valve surgery but provides no figures. Refer-

ence is made to its occurrence in digitalis intoxication but no surgical relationship is made.²⁶

Ventricular tachycardia may be precipitated by sudden reduction in coronary blood flow in a patient suffering from myocardial ischemia; thus hypotensive episodes in the postoperative period may provoke the arrhythmia. Postoperative myocardial infarction may be heralded by ventricular tachycardia.^{34, 42} Johnstone²⁴ has suggested that atropine, administered in conjunction with relaxant antagonists, may foster ventricular tachycardia, especially when the patient is hypercapnic because an excessive sympathetic nervous system activity exists under these circumstances which is further heightened by the atropine vagolysis. Richardson *et al.*¹¹ reported ventricular tachycardia as a sequel to electroshock therapy; usually the arrhythmia disappeared abruptly after the convulsive response subsided but occasionally it persisted for 18 minutes. Severe digitalis intoxication may result in ventricular tachycardia;^{12, 24} this problem will be discussed below. Epinephrine, quinidine or procaine amide, especially when administered intravenously, may precipitate ventricular tachycardia.

Quinidine^{6, 12, 40, 42} and procaine amide^{31, 34, 40} are the drugs of choice in the therapy of this arrhythmia. However when digitalis intoxication is the underlying cause potassium therapy is indicated.³¹ These compounds are relatively safe when given orally; if the urgency of the situation demands intravenous administration, electrocardiographic monitoring should be employed to facilitate recognition of toxicity and overdosage. Other therapy such as magnesium sulfate, atropine, papaverine and morphine have been suggested but probably are of limited value. Digitalis should be used to combat congestive failure unless digitalis toxicity is the cause of the tachycardia.⁶

ARRHYTHMIAS CHARACTERIZED BY A SLOW, REGULAR PULSE

Sinus Bradycardia. Arbitrarily, when a sinus rhythm produces a pulse rate of 60 per minute or less in an adult, sinus bradycardia exists. In children, this lower limit varies inversely with age, being about 110 at birth and

about 65 at the age of six.²⁰ While the rhythm may at times be regular, more often there is an associated sinus arrhythmia. No symptoms are produced by this slow rhythm. Sinus bradycardia may be confused with other slow rhythms such as nodal rhythm, 2:1 or 3:1 incomplete A-V block, complete heart block or auricular fibrillation with a slow ventricular rate; in such cases observation of the jugular vein pulsations and precordial auscultation will usually provide additional diagnostic information about the activity of the auricles.¹⁸

Sinus bradycardia is a normal finding in a few young individuals, primarily in athletes conditioned to prolonged strenuous exercise. In others it is a manifestation of increased vagal tone. Increased intracranial pressure, jaundice and myxedema are frequently associated with bradycardia. Mechanical vagal stimulation, especially when carried out in the presence of hypercapnia may result in severe bradycardia.^{18, 49, 50}

Drug-induced bradycardia is observed frequently in postoperative patients. The injection of edrophonium and neostigmine produces bradycardia by impeding the normal hydrolysis of acetylcholine and heightening vagal tone. Patients on maintenance doses of procaine amide or quinidine may exhibit bradycardia. Digitalis slows A-V conduction and through a vagotonic action, tends to inhibit the rate of impulse formation at the sinus node.¹⁵ The Rauwolfia alkaloids (reserpine and Raudixin) and the veratrum alkaloids (Veriloid and Protopoveratrine) cause marked bradycardia through vagal stimulation.¹⁵ Vasopressor agents such as methoxamine and phenylephrine commonly produce bradycardia through reflex vagal stimulation.² Levaterenol may produce a similar response.

Spinal anesthesia of sufficient height to block sympathetic cardioaccelerator fibers will produce bradycardia. *Carotid sinus* pressure produced by tight surgical dressings or plaster casts may provoke reflex sinus bradycardia.

This arrhythmia usually does not require treatment. However, if there is evidence that cardiac output is decreased by the slow rate, atropine or ephedrine will usually increase the rate temporarily. Removal of identifiable causative factors is of course indicated.

Atrioventricular Nodal Rhythm. When the

normal sinus pacemaker is sufficiently depressed, impulse formation shifts to the atrioventricular node. This tissue has an inherent rate of rhythmicity of 40 to 50 per minute. The slow pulse may be the only clinical sign of this disturbance. The same differential diagnostic possibilities exist as described above in sinus bradycardia. A pronounced jugular vein thrust is characteristic and is attributable to the fact that a regurgitant filling of the neck veins results when the auricle contracts against the closed tricuspid valve.

Since this is usually a temporary disturbance of cardiac rhythm caused by vagal inhibition of the sinus node, it occurs with relative frequency in postoperative patients but is probably not recognized often because of the apparent "normalcy" of the pulse. Strong vagal stimulation such as instrumentation within the pharynx or tracheobronchial tree can produce it; carotid sinus pressure, breath-holding, or vomiting may provoke it. The initial vagotonic action of atropine can produce nodal rhythm.⁵¹ Administration of neostigmine or other cholinergic compounds may be followed by nodal rhythm. Wasserman²² reported nodal rhythm as an associated finding in postoperative myocardial infarction. Vasopressor drugs occasionally provoke nodal rhythm.¹⁴ Digitalis, through its vagotonic action, may be the cause of atrioventricular nodal rhythm.

No treatment is necessary; it is usually self-limited and brief in duration. It produces no symptoms.

Atrial Standstill with Nodal Escape. Rarely, the sinus node will cease formation of impulses temporarily. After a short pause, the A-V node assumes the role of pacemaker, and a slow regular ventricular beat results. In contrast to atrioventricular nodal rhythm, auricular beats do not occur. This condition should be considered whenever a slow pulse is encountered. It cannot ordinarily be diagnosed without the electrocardiogram.

Brief episodes of atrial standstill with nodal escape may be produced by strong reflex vagal stimulation such as carotid compression; under this circumstance the arrhythmia is self-limited. More prolonged episodes of atrial standstill occur as a result of digitalis and quinidine poisoning and in patients suffering from hyperkale-

min; it also may be seen with auricular infarction.⁶

Treatment must be directed at the etiologic factor; vagal stimulation should be terminated; quinidine or digitalis should be stopped if they are causative; high serum potassium levels should be reduced.

Incomplete A-V Block (2:1, 3:1, 4:1 Type). Incomplete A-V block is not a common postoperative problem but organic disturbances of the conduction pathway or increased vagal tone may produce this arrhythmia. Fleeting types of this phenomenon occur during and after operation probably more frequently than realized; no record of its true incidence is available.

In this condition, the A-V node fails to respond to the sinus stimuli at regular intervals; only every second, third or fourth auricular impulse reaches the ventricle. The pulse is slow and regular. The neck veins show small auricular pulsations which occur two, three, or four times more frequently than the radial pulse, depending upon the frequency of A-V nodal transmission of the sinus impulse. Auscultation may reveal the disproportion between the auricular and ventricular beats.

Direct prolonged vagal stimulation, such as carotid sinus pressure by tight surgical dressings may produce this arrhythmia. Vagovagal reflexes from the gastrointestinal or respiratory tract are alleged to produce this phenomenon.¹⁴ Drug effects are probably more common, or at least more easily recognized because they tend to persist longer; digitalis, quinidine and morphine may effect the A-V node sufficiently to produce incomplete A-V block.¹⁵ This arrhythmia is occasionally seen in uremia. Myocardial infarction can involve the A-V node and produce A-V block.

The treatment necessarily must be directed at the cause. If it is reflex in origin, atropine or similar vagolytic compounds will relieve the block. Block induced by drugs is relieved when the offending agent is withdrawn; block associated with digitalis intoxication may be relieved by potassium administration. Depressant drugs such as quinidine and procaine amide are contraindicated.

Complete Atrioventricular Block. The slow pulse rate in this arrhythmia is the result of complete blockage of transmission of auricular

beats to the ventricles. Consequently a ventricular pacemaker provides ventricular rhythm at a rate usually between 30 and 60 times per minute. The sinus node usually continues to propagate impulses at about 70 times per minute. The extremely slow, forceful regular character of the pulse associated with this arrhythmia is usually sufficient to suggest it. Observation of the neck vein pulsations will indicate the presence of independent auricular activity at a faster rate. The electrocardiogram will confirm the diagnosis.

This arrhythmia is usually chronic and in such cases will be recognized before operation. Occasionally it follows open heart surgery as a result of direct surgical trauma to the conduction system. Septal infarction may produce A-V block in the postoperative period. Prolonged, severe hypoxia is capable of producing complete block through profound depression of the A-V node; block under this circumstance soon gives way to asystole. Digitalis intoxication can result in complete heart block but this is rare.

Treatment, if indicated at all, is largely dictated by the cause of the block. In infarction, supportive therapy of the heart and administration of isopropylarterenal to increase contractility and rate are indicated.¹⁴ Block which is surgically produced is usually treated with an electronic pacemaker.²²

ARRHYTHMIAS CHARACTERIZED BY AN IRREGULAR PULSE

Sinus Arrhythmia. This arrhythmia is commonly encountered in children and young adults. It is due to a disturbance in the rhythmic production of impulses at the sinoauricular node; it is usually produced or accentuated by alterations in vagal tone. Classically, the irregularity is associated with the phases of respiration; inspiration, by diminishing vagal tone, is usually accompanied by an increase in heart rate, while expiration results in increased vagal tone and slowing of the pulse. The variations in vagal tone are related to right auricular filling (Bainbridge reflex) and to pulmonary distention. Sinus arrhythmia is usually evident only when the heart rate is relatively slow. Activity, emotion or atropine will quickly abolish it. It is a "normal" arrhythmia, relatively easily recognized by its

association with the respiratory phases and it produces no symptoms and requires no treatment. Occasionally, it is observed in an older patient as a result of digitalis administration; here again, heightened vagal tonicity produces the irregularity.

Sino-atrial Block. This arrhythmia is also known as sinus arrest or sinus block. It is characterized by a sudden standstill of the entire heart for one or more beats. Single beats may drop out with a regular sequence (such as in an alternating fashion) or runs of two or three dropped beats may occur. If the sinus arrest is prolonged, nodal or ventricular escape occurs as described previously. Sino-atrial block may be suspected when a regular pulse is suddenly interrupted by a long pause, after which the regularity of the pulse is reestablished. It must be differentiated from premature contractions which are followed by a compensatory pause and from the Wenckebach type of A-V block; the electrocardiogram may be necessary for diagnosis.

Sino-atrial block is due to increased vagal tone, with resultant depression of sinus node impulse formation. Although no figures are available on its incidence in the postoperative period, it probably occurs more often than realized, considering the frequency of increased vagotonia during and after operation.

Suctioning of the tracheobronchial tree, carotid sinus pressure or the administration of a vagotonic agent such as neostigmine may produce it. Sinus arrest may accompany the state of hyperkalemia; digitalis and quinidine have been known to produce it.

The treatment is not urgent; atropine will reverse most instances of this arrhythmia. When digitalis intoxication or hyperkalemia are responsible, withdrawal or reduction of these agents will usually result in re-establishment of normal cardiac rhythmicity.

Premature Contractions. Premature contractions constitute the most common cause of irregularity of the pulse. The arrhythmia results when the auricles or the ventricles are stimulated prematurely, that is, before the next regular sinus impulse is due. The premature contractions may have their origin in the auricles, the A-V node or in the ventricles; they may occur infrequently, or they may recur in a regular sequence, such as every second

or third beat, in which case bigeminy or trigeminy is produced. A premature beat can be recognized by the fact that it occurs earlier than expected, often quickly after the previous beat, and is usually followed by a pause, due to the failure of the ventricle to respond to the next normal sinus impulse. It is difficult to distinguish clinically, the origin of the premature beat (whether it is auricular, nodal or ventricular), but observation of the jugular pulsations is helpful; the electrocardiogram is the only reliable means of identification of premature beats. Clinically, premature beats must be differentiated from sinus arrhythmia; this can usually be done by noting the association of the latter with the phrases of respiration. When premature beats are frequent, differentiation from atrial fibrillation may be difficult; dropped beats in A-V block also resemble premature contractions and should be considered when pauses are detected in the pulse rhythm.

The incidence of this arrhythmia was reported as less than 2 per cent by Massie and Valle²² among 120 postoperative patients studied. This tabulation included both auricular and ventricular premature contractions. Richardson⁴¹ on the other hand reported a 20 per cent incidence, following electro-shock therapy. This high occurrence was observed in spite of atropinization. Fisher and Winsor¹¹ observed a 3 per cent rate of premature ventricular contractions among 52 patients studied postoperatively. Wasserman²³ collected 25 cases of postoperative coronary occlusion and found that 3 exhibited premature auricular contractions. Jacoby²⁴ noted that premature contractions were the third commonest arrhythmia in hypoxic patients subjected to vagal stimulation. Among the reviewers series of recovery room patients occasional premature contractions were observed in 8 per cent.

The cause of premature contractions is not completely understood. They appear to be a manifestation of localized irritability of the heart. Circumstances arise during and after anesthesia which tend to foster the formation of premature beats. Posture, especially recumbency, has been identified as predisposing to premature contractions.⁶ Certain drugs, such as ephedrine and epinephrine may precipitate the arrhythmia. Bennett⁷ reported

frequent premature contractions after the administration of nikethamide and caffeine for central nervous system stimulation; Anderson and Scott¹ made similar observations after amphetamine. No reference to sequelae of this type following β -ethyl- β -methylglutarimide (Megimide) or methyl phenidate (Ritalin) could be found. Digitalis toxicity commonly causes ventricular premature contractions, often with coupling.¹⁸ The onset of postoperative congestive heart failure may be heralded by auricular premature beats; eventually auricular fibrillation supervenes. Myocardial infarction frequently invokes premature contractions.²³ Sympathetic overactivity, such as occurs with hypercapnia may produce premature ventricular contractions;²⁴ Brown and Miller¹⁰ observed premature ventricular contractions in 15 of 17 hypercapnic dogs *after washout of carbon dioxide*.

Premature contractions are a common finding in patients over 50 years of age. When they occur infrequently they are without clinical significance and require no treatment. If they occur with more than occasional frequency, serious heart disease or toxicity (for example, digitalis poisoning) is usually present and treatment is indicated. Quinidine therapy is usually recommended;^{4, 18, 22, 42} procaine amide has also proved useful.^{6, 14} Digitalis is the drug of choice when congestive failure is present. *However, when digitalis intoxication causes the premature contractions*, the drug should be stopped; the administration of potassium will usually control the arrhythmia.^{18, 24} Care must be exercised in the use of this therapy in patients with renal disease.

Atrial Fibrillation. Atrial fibrillation frequently exists as a chronic arrhythmia in which case its presence in the postoperative period poses no diagnostic problem. However, a paroxysmal form of auricular fibrillation occasionally appears postoperatively; it occurs most frequently in the aged, arteriosclerotic patient and is especially likely to occur after thoracic surgery. It can usually be diagnosed clinically by the complete irregularity of the rate, rhythm and force of the heart beat felt in the radial pulse, and by the pulse deficit observed by simultaneous auscultation of the apex beat and palpation of the pulse. The neck veins show irregular pulsations which are synchronous

with the radial pulse. Early untreated auricular fibrillation is usually characterized by a relatively rapid ventricular rate; when this is present, the arrhythmia may be mistaken for auricular, nodal or ventricular tachycardia, or atrial flutter. However, the gross irregularity of rhythm and force and the lack of response to vagal stimulation which characterize auricular fibrillation usually make the differential diagnosis clear. Occasionally, differentiation from frequent premature contractions may present difficulty. Prolonged palpation of the pulse will usually simplify identification.

When digitalis has been given, the ventricular rate may be slow and almost regular; here nodal rhythm and sinus bradycardia must be considered. The differentiation of these arrhythmias has been mentioned above but electrocardiographic identification may be required.

The causes of paroxysmal auricular fibrillation are numerous. The arrhythmia is frequently precipitated by the stresses of anesthesia and operation in elderly patients; advanced arteriosclerosis and hypertension increase the likelihood of the complication. It is a common sequel to chest operation, occurring in some series in 15 per cent of patients.²⁴ It occurs in about 7 per cent of patients who suffer postoperative coronary occlusion.²⁴ The presence of rheumatic valvular disease predisposes to auricular fibrillation after operation;⁴⁴ the arrhythmia is frequently precipitated by mitral valvotomy. Hyperthyroidism, especially the thyroid crisis following operation, may result in auricular fibrillation. Pleural complications such as effusion,⁴ empyema¹² and shift of the mediastinum may provoke this arrhythmia. It has been described following head trauma.¹⁴

In its paroxysmal form, auricular fibrillation may precipitate congestive heart failure. When this occurs, digitalis administration is indicated.^{12, 21, 22, 40} In the absence of heart failure, expectant treatment is warranted since spontaneous reversion to sinus rhythm is common.^{12, 22} Quinidine has been recommended^{18, 22} to convert postoperative atrial fibrillation not associated with failure.

Ventricular Fibrillation. This arrhythmia has been considered elsewhere in this symposium under sudden cardiac collapse.

Incomplete A-V block—Wenckebach Phe-

TABLE 4
INCIDENCE OF CARDIAC ARRHYTHMIAS FOLLOWING
THORACIC SURGERY

Author	Number of Patients	Over-all Incidence (Per Cent)	Types of Arrhythmia*
Krosnick and Wasserman ²⁹	82	12.2	P.A.F. 7 V.T. 1 A.F.I. 1
Bailey and Betts ³	78	11.0	P.A.F. 5 N.T. 1 A.F.I. 2 Fl.-Fib. 1
Currens <i>et al.</i> ¹³	56	21.0	P.A.F. 8 A.F.I. 4
Massie and Valle ²⁵	120	9.1	P.A.F. 5 A.F.I. 4 PAC 1 PVC 1
Fisher and Winsor ¹⁷	52	12.0	P.A.F. 3 PVC 2 N.T. 1
Wheat and Burford ³⁴	302 139	16.0 9.1	P.A.F. 55 possibly 58 Fl.-Fib. 1 SVT 5

* P.A.F. = paroxysmal auricular fibrillation; V.T. = ventricular tachycardia; A.F.I. = auricular flutter; N.T. = nodal tachycardia; Fl.-Fib. = flutter-fibrillation; PAC = premature atrial contractions; PVC = premature ventricular contractions; SVT = supraventricular tachycardia.

nomenon. The Wenckebach type of incomplete A-V block produces an irregular pulse. This is due to the fact that each successive stimulus arriving from the auricles finds increasing difficulty in traversing the A-V node; finally the impulse is not transmitted at all, and ventricular contraction fails to occur. By the time the next auricular impulse reaches the A-V node, it has recovered and is able to conduct the stimulus to the ventricle as before; the cycle thus repeats itself resulting in a series of dropped beats, which usually occur at regular intervals; for example, every fourth or fifth beat will be dropped. It is obvious that superficially this arrhythmia resembles sinus arrest and premature auricular contractions. In sinus arrest however, examination of the jugular pulse reveals the absence of auricular

activity during the dropped pulse beat. In the Wenckebach block, the blocked auricular beat can be seen in the veins and occasionally heard over the precordium. In the case of premature auricular contractions, the situation is more difficult. Unless the auricular premature contraction is distinctly audible over the precordium and its premature timing clearly identifiable, it may be impossible to separate the two arrhythmias without an electrocardiogram.

ARRHYTHMIAS FOLLOWING THORACIC OPERATION

In previous sections it has been pointed out that arrhythmias occur frequently following operation upon the thorax. Furthermore, disturbances in rhythm are less well tolerated in these patients and are at times resistant to treatment. They represent an obstacle to a successful operative result unless they are managed skillfully. *Incidence*—Table 4 lists the incidence of arrhythmia following thoracic operations at six prominent centers. The range is 9.1 per cent to 21.0 per cent. These values are in sharp contrast to the relatively low incidence (approximately 0.5 per cent) observed by Rogers *et al.*⁴² following non-thoracic procedures.

Types of Arrhythmias. Paroxysmal auricular fibrillation is the commonest arrhythmia encountered; 83 instances occurred among the 827 patients (table 4). Auricular flutter or flutter-fibrillation was noted 13 times. Premature contractions were not tabulated unless they occurred frequently.

Time of Onset of Arrhythmia. Time of onset of the arrhythmias associated with thoracic operation is variable. Krosnick and Wasserman²⁹ noted that about 30 per cent occurred on the first postoperative day. Wheat and Burford state that 60 per cent of their arrhythmias began within 72 hours of operation.³⁴ The range was from the first to the eighteenth postoperative day. It is difficult in the light of these data to develop a correlation with the anesthetic procedure, since rarely did the arrhythmia occur *immediately* after operation. However, factors *related* to the anesthetic management are doubtless involved and will be discussed.

Predisposing Factors. There is general agreement that *age* is an important predisposing factor. Krosnick²⁹ observed his highest incidence (11.0 per cent) in patients over 60 years. Wheat and Burford²⁴ state that 20 to 30 per cent of patients over 60 may be expected to have postoperative arrhythmias. The presence of *previously existing heart disease* also appears to be important. Arteriosclerosis and hypertensive cardiovascular disease are common findings among patients with arrhythmias. Patients with history of previous rhythm disturbances also are prominent in this regard.²⁹ *The site of the operation and its extent* are also important.^{3, 29, 34} Arrhythmias are common (11 to 32.4 per cent) following pneumonectomy whereas following lobectomy, the incidence is 5.4 per cent or less. When the thoracic lesion involves the mediastinal structures, especially the vagus nerve, arrhythmias occur with increased frequency.²⁹ Blood electrolyte alterations and hypercapnia, which occur commonly in these patients are thought to contribute to the high incidence of arrhythmias.²⁹

Precipitating Factors. *Hypoxemia* occurring as a result of pulmonary collapse is cited as a precipitating factor. Massie and Valle³⁵ and Hood²¹ contend that hypoxia develops commonly in patients after thoracic operation in spite of the fact that most patients receive oxygen therapy. *Vagal irritation*, arising as a result of reaction about the site of operation or provoked by suctioning within the pharynx or trachea has been blamed.^{3, 22} *Hypotension*,²⁹ *mediastinal shift*^{2, 12, 29} and *pleural effusion* are thought to contribute to the onset of the arrhythmias.

Treatment. Prophylactic digitalization of all patients facing thoracic operations has been recommended;²¹ other authors suggest quinidine or procaine amide therapy preoperatively in patients exhibiting premature contractions.²⁹ Obviously, measures which forestall or prevent hypoxia, hypercapnea and hypotension should be employed. These include preoperative postural drainage, breathing training, and adjustment of blood volume and electrolyte balance. When the arrhythmia occurs, the immediate danger is that it will precipitate congestive heart failure. If the patient appears to be tolerating the arrhythmia well, it may be observed expectantly; a high percentage revert

to normal rhythm spontaneously.¹³ However, if congestive heart failure threatens, digitalis is the treatment of choice. Quinidine may be tried if it seems desirable to control the arrhythmia in the absence of congestive failure.²²

Prognosis. Since the arrhythmias occur against a background of advanced age, pre-existing heart disease and the pulmonary disease for which the operation was undertaken, it is not surprising that they carry a high mortality. Krosnick reported 2 deaths among 10 patients who developed arrhythmias in the postoperative period, a mortality of 20 per cent.²⁹ Currens *et al.*¹² reported an 8 per cent mortality; Massie and Valle³⁵ had 3 deaths among 11 patients with arrhythmias (26 per cent).

ARRHYTHMIAS AND DIGITALIS

In the foregoing sections, digitalis has been cited as a possible causative agent of many arrhythmias. It is worth-while to examine further the characteristics of this drug and the circumstances under which toxicity is likely to be provoked.

Lown and Levine³⁴ state that "digitalis is a potent myocardial irritant and depressant. The interaction of these two effects makes possible the gamut of electrocardiographic anomalies." Digitalis produces (1) an increase in myocardial contractile force, (2) a slowing of the heart rate, probably through a caroticoaortic body reflex in response to the increase in stroke output, (3) a shortening of the refractory period of the auricles, (4) a progressive shortening of the refractory period of the ventricles, and (5) a slowing of A-V conduction, even to the point of complete block. (It is this latter action that produces ventricular slowing when digitalis is used in atrial fibrillation.)

Actions (3), (4) and (5) give clues to the types of arrhythmia which may be precipitated by digitalis overdosage. The change in the refractory period of the auricle provokes paroxysmal atrial tachycardia in some patients. The depressant effect of digitalis upon the A-V node results in varying degrees of block with the tachycardia. The progressive shortening of the refractory period of the ventricles is associated with increased excitability and a greater tendency to automaticity; hence, pre-

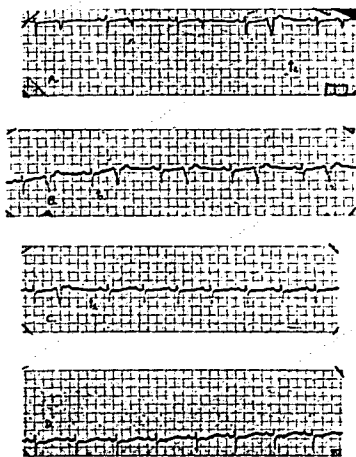


FIG. 1. Postoperative bigeminy due to digitalis intoxication. A. Before treatment with potassium. Note multifocal premature ventricular contractions with coupling. Arrow 1 indicates start of treatment with KCl (see text). B. During treatment. At arrow 2, 16 mEq. of KCl have been infused; premature contractions are now unifocal. C. At arrow 3, arrhythmia ends abruptly after 20 mEq. of KCl have been infused. D. Two hours after infusion of 40 mEq. of KCl; sinus rhythm maintained.

mature ventricular contractions are the most common rhythm disturbances observed with digitalis intoxication in man. The great hazard of digitalis intoxication is that it may precipitate ventricular fibrillation; premature contractions, coupling and ventricular tachycardia are the forerunners of this phenomenon.

Circumstances related to the anesthetic and surgical period may facilitate the onset of digitalis intoxication and as a consequence produce many of the arrhythmias described above. First, the use of rapid digitalization with the purified cardiac glycosides has become commonplace. Surgical patients who show evidence of cardiac incompetence frequently received a "standard" dose of these potent drugs before operation. In many instances, the dose produces subdigitalization but in the debilitated, cachectic patient, it often proves to be excessive. Signs of toxicity

may appear immediately before, during or after operation.

Second, electrolyte disturbances are common in surgical states. Digitalis provokes toxic responses more readily under these circumstances. Potassium is the most important ion in this regard. Patients receiving the mercurial diuretics or 2-acetylaminio-1,3,4-thiadiazole-5-sulfonamide (Diamox) suffer depletion of tissue stores of potassium through increased renal excretion.²⁴ Sizable fluid and electrolyte losses frequently accompany bowel obstruction. In these instances serious digitalis intoxication may follow in spite of a normal serum potassium. Abrupt reduction of the serum potassium level occurs when hemodialysis is carried out with the artificial kidney; digitalis toxicity is a frequent sequel and arrhythmias are common.²⁴ Also, when hypertonic glucose solution is administered to provoke diuresis, serum potassium depression occurs as glycogen deposition proceeds; arrhythmias are frequently observed in digitalized patients under these circumstances.²⁴ Patients receiving steroid therapy have reduced potassium stores and are thus prone to digitalis toxicity and arrhythmias.

When such circumstances exist, premature ventricular contractions usually are the earliest manifestation of digitalis poisoning. Bigeminy (coupling) is also common. Sagall¹⁶ points out that the ectopic beats of digitalis intoxication usually arise in the right ventricle. They are often multifocal in origin. Figure 1A illustrates a postoperative multifocal bigeminy caused by digitalis toxicity.

Treatment of arrhythmias resulting from digitalis intoxication is fortunately simple, though not without hazard. The drug of choice is potassium, which is best administered intravenously, with continuous electrocardiographic control. Even if circumstances indicate that a potassium deficit does not exist, the cation will still be effective in controlling arrhythmias due to digitalis overdosage. Forty milliequivalents of potassium chloride dissolved in 500 ml. of 5 per cent dextrose solution should be administered in one hour. The electrocardiogram must be watched for signs of hyperkalemia (peaking or tenting of T waves) and the drip stopped if they appear. The infusion may be repeated until 120 mEq. have

been given. Figure 1 (B, C, D) illustrates the reversion of a postoperative digitalis bigeminy with potassium chloride. The arrhythmia developed at the end of the surgical procedure. The history and electrocardiographic findings indicated that digitalis was the cause of the arrhythmia. At arrow 1, the infusion of 40 mEq. of potassium chloride dissolved in 500 ml. of 5 per cent dextrose in water was begun; at arrow 2, 200 ml. of solution (approximately 16 mEq. KCl) had been infused; the arrhythmia terminated abruptly at arrow 3 after approximately 20 mEq. of KCl had been administered. Figure 1D, recorded two hours later, reveals persistence of sinus rhythm.

Procaine amide is useful in combating arrhythmias due to digitalis, principally through its ability to depress excitability and conduction.²⁷ Although its actions are in many ways similar to those of quinidine, procaine amide does not depress myocardial contractility. It is most effective against the ventricular arrhythmias; it is variable in its effectiveness in atrial dysrhythmias, although Lown and Levine reported success in restoring sinus rhythm in 5 of 7 cases of paroxysmal atrial tachycardia with block.²⁴ Procaine amide frequently causes toxic symptoms and may even cause death.²⁹ The most common side-effect is hypotension, which is especially profound in aged patients with advanced cardiac disease. Although effective when given orally, procaine amide is usually administered intravenously in postoperative situations. It should be given slowly at a rate of approximately 25 mg. per minute, while frequent blood pressure determinations are made. The effective dose range is 50-500 mg.

Finally, the relationship between digitalis and calcium deserves mention. Injection of calcium salts in normal man has been shown to produce bradycardia, sinus arrhythmia, shifting pacemaker, heart block and multifocal ventricular premature contractions.⁸ In digitalized patients, calcium appears to act synergistically with the cardiac glycosides;⁶ the likelihood of toxic reactions is thus enhanced. Sudden deaths have been reported following the intravenous injection of calcium.⁹

Information concerning the exact mechanism of such reactions to calcium is lacking, but evidence indicates that the failing, digital-

ized heart is more sensitive to its action. This finding has significance when massive blood transfusions are necessary. Calcium is frequently recommended to offset the effects of citrate intoxication and hyperkalemia with massive transfusions. Howland²² however has recently challenged the need for exogenous calcium administration and reported untoward cardiovascular reactions when the ion was injected. Shock, acidosis or hypoxemia appeared to exaggerate the response. Thus, it would appear that calcium administration should be undertaken with caution, if at all, in the digitalized patient.

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PULMONARY FUNCTION Pulmonary function was studied in 13 hyperthyroid patients before and after therapy. Vital capacity was decreased in some, but not all. Pressures at the mouth on maximal expiratory and inspiratory effort against a closed airway indicated weakness of the respiratory muscles. On exercise the patient's ventilation was increased in excess of the oxygen uptake when compared to a group of controls. This is apparently related to an increase in the dead space ventilation due to an increase in frequency of breathing and alveolar dead space ventilation. Following remission of symptoms produced by thyroidectomy, radioactive iodine, or propylthiouracil drugs, the previously described abnormalities reverted to normal levels. In hyperthyroid patients, apparent diffusing capacity of the lung, pulmonary capillary blood volume, and membrane diffusing capacity at rest were not elevated in the presence of significant elevations of pulmonary capillary blood flow. (Stein, M., and others: *Pulmonary Function in Hyperthyroidism*, *J. Clin. Invest.* 40: 348 (Feb.) 1961.)

ASPIRATION PNEUMONITIS Mendelson's syndrome, or peptic-aspiration pneumonia, results from aspiration of fluid gastric contents. Whenever aspiration is suspected, vigorous treatment should be carried out by intubation of the trachea, suctioning of the tracheobronchial tree, and lavage with normal saline. This treatment should be carried out even if it is necessary to re-anesthetize the patient to accomplish it. If aspiration pneumonia supervenes, treatment must be instituted on an emergency basis with an adequate airway, suction, oxygen, intravenous hydrocortisone (100 mg. adult dose), and aminophylline (500 mg. in 500 cc. 5 per cent dextrose in water by intravenous drip). Aminophylline will relieve bronchospasm, and the hydrocortisone acts in an anti-inflammatory manner, reducing the edema and fluid resulting from the chemical injury to the bronchial tree. In this emergency, the early intravenous administration of hydrocortisone may be life-saving. (Dines, D. E., and others: *Aspiration Pneumonitis—Mendelson's Syndrome*, *J. A. M. A.* 176: 129 (Apr. 22) 1961.)