Diagnosis and Management of Tachycardias After Myocardial Infarction

Charles R. Webb

Follow this and additional works at: https://scholarlycommons.henryford.com/hfhmedjournal

Part of the Life Sciences Commons, Medical Specialties Commons, and the Public Health Commons

Recommended Citation
Available at: https://scholarlycommons.henryford.com/hfhmedjournal/vol39/iss3/15

This Article is brought to you for free and open access by Henry Ford Health System Scholarly Commons. It has been accepted for inclusion in Henry Ford Hospital Medical Journal by an authorized editor of Henry Ford Health System Scholarly Commons.
Diagnosis and Management of Tachycardias After Myocardial Infarction

Charles R. Webb, MD∗

The patient with acute myocardial infarction (MI) may present with acute arrhythmias due to ischemia as well as chronic arrhythmias such as chronic atrial fibrillation or ventricular ectopy. The only way to distinguish acute, chronic, or paroxysmal arrhythmia is by a careful history from the patient and review of available old records. Guidelines for the diagnosis and determination of the significance of arrhythmia after acute MI are presented herein.

Rationale for Therapy

The three purposes for treating a patient for arrhythmia are: 1) relief of symptoms due to the arrhythmia, 2) suppression of arrhythmia causing hemodynamic impairment, and 3) prevention of related and potentially lethal arrhythmias.

The management of an arrhythmia begins with determination of its prognosis and hemodynamic implications and detection of any potential causative factors before initiation of specific antiarrhythmic pharmacologic therapy.

Antiarrhythmic therapy should be individualized regarding choice of agent, dosage, and length of therapy. Some cases require acute therapy for arrhythmia termination. Others require chronic arrhythmia prevention or suppression. For some arrhythmias, such as chronic atrial fibrillation or chronic premature ventricular complexes, there may be no effective means of termination. Therefore, control of arrhythmia frequency or rate must be accepted.

Determination of such individualized therapeutic goals or targets will allow use of minimal effective drug dosages, thereby minimizing side effects, toxicity, and cost.

Recognition of Precipitating or Exacerbating Noncardiovascular Factors

For diagnostic and therapeutic purposes, arrhythmias may be called primary if due to cardiac pathology, or secondary if reactive to a systemic or myocardial process. To determine if and when to treat the arrhythmia, the first question which must be answered is whether the arrhythmia is a primary illness, merely reflects more basic cardiovascular dysfunction, or is the result of systemic or metabolic imbalance.

Hypoxia can cause or exacerbate almost any atrial or ventricular arrhythmia (1). Arrhythmia often results from membrane instability which reflects more generalized electrolyte imbalance. Hypokalemia must be appropriately corrected particularly for patients taking diuretics (2). Since potassium is primarily an intracellular ion, merely correcting the serum potassium may not be adequate and days to weeks may be required for complete correction. In the interim period, specific antiarrhythmic drug therapy is not likely to be effective. Anecdotally, it seems that a serum potassium level of 4.0 mEq/L or higher is necessary to stabilize arrhythmias in certain patients with cardiovascular disease.

Hypomagnesemia also predisposes to arrhythmias and tends to make antiarrhythmic drugs less effective (3,4). Like potassium, magnesium is an intracellular ion, and correction of the serum level may precede total body equilibrium by several days. Occasionally, ectopy and tachyarrhythmias, including refractory or incessant ventricular tachycardia (VT), appear to be suppressed by magnesium, even when the serum level is already normal.

Hypokalemia and hypomagnesemia should be suspected in any patient with chronic or acute diuretic therapy, particularly without ionic supplementation, or patients with poor or restricted diets, malabsorption, or diarrhea.

Heart failure is associated with arrhythmia (5,6). Ventricular ectopy and nonsustained or sustained VT often are improved or abolished with appropriate diuresis, inotropic therapy (7) (digoxin), and/or vasodilator therapy (8). In the cardiac intensive care setting, the pulmonary capillary wedge pressure, pulmonary and vascular resistances, and cardiac output can be optimized by therapy guided by right heart catheterization (Swan-Ganz catheterization).

Arrhythmia preceded by chest pain or transient electrocardiographic ST or T-wave changes is often due to myocardial ischemia. These arrhythmias often respond to antiischemic drug therapy (nitrates, β-adrenergic blockade, calcium channel antagonists), coronary angioplasty, or bypass surgery. Paradoxically, in the period immediately after angioplasty (9), thrombolytic therapy, or relief of Prinzmetal’s vasospastic angina (10), arrhythmias may recur or appear de novo. These are called reperfusion arrhythmias. They should be suppressed with lidocaine or procainamide intravenously and should resolve within...
24 hours. When arrhythmias occur later after angioplasty, the possibility of ischemia due to reocclusion of a dilated vessel must be considered.

Ventricular arrhythmias commonly complicate acute MI. Acutely, any significant ventricular ectopy should be suppressed by a lidocaine bolus of 50 to 100 mg intravenously followed by an infusion titrated between 2 to 4 µg/min. Sustained arrhythmias such as ventricular fibrillation within the first 24 hours do not portend chronic arrhythmic propensity, and once arrhythmia is resolved and the patient leaves the critical care unit, routine cardiac monitoring and rehabilitation guidelines should be followed (11,12).

Pericarditis (postinfarction or Dressler’s syndrome) may mimic, complicate, or follow acute MI. It usually presents as chest pain, classically with a pericardial friction rub and a small pleural effusion. Pericarditis is frequently associated with atrial arrhythmias and less commonly ventricular arrhythmias, which usually respond to antiinflammatory therapy and natural resolution of the acute process. Because of the proximity of the sinus node to the epicardium, sinus node suppression and bradycardia may also occur (13).

Cardiac manifestations may be the only physical evidence of thyroid disease in the elderly or critically ill patient. Patients with persistent sinus tachycardias in the absence of congestive heart failure should have thyroid disease excluded by a serum thyroid-stimulating hormone determination (14).

Many drugs may cause or exacerbate cardiac arrhythmias. Diuretics may induce hypokalemia and/or hypomagnesemia as discussed above. Digoxin may suppress arrhythmias if cardiac function is improved; however, an excess may exacerbate or cause almost any cardiac arrhythmia. Bradycardia and heart block are the most obvious of these, particularly when digoxin is combined with β-adrenergic blockade or calcium channel blocking drugs. However, ventricular ectopy, VT, or ventricular fibrillation may occur. It is important to recognize that these arrhythmias are due to digoxin toxicity because the appropriate therapy is optimization of electrolytes, correction of hypoxia, cessation of digoxin therapy, and occasionally dialysis or intravenous administration of digoxin antibodies. On the other hand, specific antiarrhythmic drug therapy is unlikely to be effective. Cardioversion for atrial arrhythmias or VT is hazardous because digoxin lowers the ventricular fibrillation threshold. The digoxin therapeutic toxic dose ratio is small and variable. Although markedly elevated serum levels correlate well with toxicity, there are usually obvious clinical symptoms or signs in such cases. In more subtle cases, the serum drug levels are likely to be borderline. This is probably due to variable cardiac sensitivity related to the underlying type and severity of cardiac disease and variations in the electrolytic and metabolic milieu (15,16). When an antiarrhythmic drug is necessary for suppression of ventricular arrhythmia, diphenhydantoin has the advantage of moderate efficacy as a membrane stabilizer and the least negative dromotropic effect on the cardiac conduction system. It is the least likely to exacerbate heart block.

Bronchodilators, particularly the methylated xanthines, commonly exacerbate arrhythmias. In the critically ill patient, previously well tolerated agents tend to accumulate and serum levels should routinely be assessed. The only reliable antidote appears to be reduction of dosage; specific antiarrhythmic therapy is unlikely to be useful.

The patient’s hospital and clinic charts must be reviewed for any “antiarrhythmic” therapy. Indeed, there is no antiarrhythmic agent which uniformly decreases arrhythmic potential. Most agents are effective for suppression of ectopy in about 60% of patients and for prevention of sustained arrhythmia in about 30% of patients. In general, any antiarrhythmic agent has the propensity to worsen arrhythmias in 10% to 15% of patients (17). Obviously, for the patient who has arrhythmia while on antiarrhythmic medication, the drug is not antiarrhythmic at the current dosage level. When consulted because of arrhythmia in a patient already receiving chronic antiarrhythmic therapy, first review the history to determine why the therapy was instituted. If the patient has a history of lethal arrhythmias and only ectopy is noted currently, perhaps the therapeutic goal of prevention of lethal arrhythmia is indeed being fulfilled and therapy should be continued. However, if a patient has potentially lethal or lethal cardiac arrhythmia while on chronic antiarrhythmic therapy, peak and trough drug levels should be evaluated. Subtherapeutic levels would require an increase in dosage and reassessment of antiarrhythmic efficacy after a new steady-state blood level is attained (after administration of the new dose for five drug half-lives). On the other hand, levels above the usual therapeutic level call for reduction of dosage and reassessment of arrhythmia after five drug half-lives. If the drug level is within the therapeutic range, it is usually advisable to discontinue the current agent and employ a different agent. In difficult cases, combination drug therapy is beneficial. Success has been reported with combinations of Vaughan-Williams class IA and IB agents (18). Combination of IA and IC agents is contraindicated as arrhythmic deaths have been reported (19). In fact, when switching between these subclasses of drugs, it is safest to eliminate the first agent entirely, before initiation of the second agent.

The Supraventricular Arrhythmias

The most common arrhythmia accompanying acute MI is sinus tachycardia. Sinus tachycardia is recognized by P waves which precede each QRS complex in a 1:1 ratio with an appropriate PR interval. Each P wave is upright in electrocardiographic leads II, III, and AVF, indicating a cranio-caudal sequence of atrial depolarization. Carotid sinus pressure results in transient, gradual slowing with return to the previous rate seconds after release.

Usually sinus tachycardia is due to anxiety or is an appropriate cardiovascular response to pain or disease and seldom should be suppressed with antiarrhythmic drugs. Digoxin at therapeutic levels is rarely effective in suppressing a normal sinus node (20). However, β-adrenergic blockers or calcium channel blockers may blunt an appropriate chronotropic response and result in hemodynamic collapse.

Hypovolemia is a common cause of sinus tachycardia and the heart rate should diminish gradually as plasma volume is restored. Relative hypovolemia may be due to decreased left ventricular compliance or ventricular infarction which can be de-
ected by Swan-Ganz catheterization. Other causes are anxiety, fear, pain, anemia, fever, hyperthyroidism or thyrotoxicosis, overdosage of thyroid replacement, volatile drugs (atropine), vasopressors (isoproterenol, dobutamine, dopamine), vasodilators (nitrates, procardia), and antihypertensives.

Some degree of sinus arrhythmia is common. Unless long symptomatic pauses occur, it is not an indication for specific therapy.

**Atrial premature complexes (APCs)** are common in any hyperadrenergic state that is commonly associated with sinus tachycardia. Usually the P-wave morphology is different from sinus P waves indicating that the focus is ectopic, not the sinus node. However, some foci are near or within the sinus perinodal tissue and may appear similar to sinus node-initiated P waves. Generally APCs are asymptomatic and the therapy should be directed at correction of the systemic disease process as, more often than not, the heart is merely an innocent bystander. Caffeine and methylated xanthines and vasopressors commonly are responsible and their dosages should be minimized. Hypoxia, hypovolemia, anemia, and electrolyte imbalance should be corrected. Rarely is specific antiarrhythmic therapy indicated unless APCs are symptomatic or perceived as harbingers to atrial flutter or fibrillation in patients with a history of such arrhythmia.

**Atrial tachycardia** is an arrhythmia which originates in the atria. Therefore, each ventricular complex is preceded by an atrial complex with an appropriate PR interval. The P-wave morphology is uniform, usually ectopic, and different than sinus P waves. Carotid sinus pressure may terminate the arrhythmia abruptly. Ectopic atrial tachycardia is rarely of hemodynamic significance and does not require specific therapy.

Multifocal atrial tachycardia is a common cardiac manifestation of serious acute cardiac or pulmonary disease. It is an irregularly irregular arrhythmia characterized by the triad of varying P-wave morphologies, varying P to P intervals, and varying PR intervals (21). Carotid sinus pressure usually has no effect on the arrhythmia. Its distinction from atrial flutter or fibrillation is important. Digoxin is contraindicated because the factors which perpetuate multifocal atrial tachycardia also predispose to digoxin toxicity and proarrhythmia, i.e., hypoxia and electrolyte imbalance. Some investigators report relative success with calcium channel blockers. However, the most effective therapy is detection and correction of the underlying cause.

**Atrial flutter** is characterized by uniform P waves at a rate of 300 to 350 beats/min. It is associated with rapid mechanical atrial activity. There is discrete electrical and mechanical systole and diastole. Usually the atrioventricular (AV) node is effective as a filter resulting in a physiologic 2:1 or higher AV nodal block and a moderated ventricular rate of about 150 to 200 beats/min. Any rhythm at a rate of 150 beats/min should result in consideration of atrial flutter with examination of rhythm strips and a 12-lead ECG for P waves. (Caution: P waves may be small and hide on the QRS complex or in the T waves. They tend to become more visible at early morning staff rounds than at night.) Careful examination of the neck veins for A waves may make the diagnosis. Movement of the V leads an interspace higher or lower than usual or even to the back may detect P waves and make the diagnosis. Carotid sinus pressure may transiently increase the AV nodal block and make the P waves visible. Rarely, insertion of an intraatrial lead is necessary to make the diagnosis.

Atrial flutter may be a primary arrhythmia in patients with no other evidence of cardiac or systemic disease. More frequently, however, it is secondary to congestive heart failure, cardiomyopathy, pericarditis, pulmonary disease, cardiac surgery, hypertension, alcohol, smoking, or caffeinated beverages. Occasionally patients present with heart failure secondary to a sustained episode of paroxysmal atrial flutter.

Three basic forms of therapy are available for termination of sustained atrial flutter. Atrial overdrive pacing is an invasive procedure involving insertion of an intratrial electrode which is useful to confirm the diagnosis and for termination (22, 23). When the atrium is carefully entrained and pacing abruptly stopped, sinus rhythm should prevail, but there is always the possibility of proarrhythmia, resulting in atrial fibrillation which often converts to sinus rhythm spontaneously within 24 hours. The advantage of atrial overdrive pacing is that it is rapid and avoids administration of drugs which are potentially negative inotropic agents in critically ill patients. A brachial vein approach is safe for patients who are on low-dose anticoagulation therapy, even in outpatients whom I send to work after two hours of observation. Digoxin toxicity is not a contraindication.

When the rate of the atrium during flutter is greater than 210 beats/min, entrainment of the atrium may not be possible. In such cases, the refractory period of the atrium may be increased by prior oral or intravenous administration of a Vaughan-Williams type IA antiarrhythmic agent.

Intravenous procainamide, 500 to 1,000 mg, by intravenous infusion over 20 minutes, is often effective. This dosage requires constant patient supervision and blood pressure monitoring at least every 5 minutes. Hypotension frequently results, occasionally because of negative inotropic effect, but more commonly because of peripheral vasodilatation which responds to reduction of infusion rate by half and administration of 100 to 200 mL of normal saline rapidly. Depending on the estimated likelihood of early recurrence, the infusion may be either terminated upon restoration of normal sinus rhythm or reduced to 2 to 4 mg/min until oral loading is initiated. Oral loading of digoxin and subsequently a quinidine preparation may also be effective, but it takes longer (days rather than minutes). Quinidine has a potent vagolytic effect on the AV node and can accelerate the ventricular rate to 200 beats/min or higher due to 1:1 AV nodal conduction unless the AV node is first blocked with digoxin or a β-adrenergic blocker. However, the vagolytic effect of procainamide is not of clinical significance and previous administration of digoxin is not a prerequisite.

The third method of acute termination of atrial flutter is cardioversion. It is often said that low energy cardioversion (10 to 25 joules) is effective for atrial flutter, but frequently higher energy is necessary. Cardioversion is uncomfortable with sedation and there is hazard of respiratory suppression in the critically ill patient. Brief general anesthesia with the ultrashort-acting agent, methohexital, 40 to 100 mg intravenously, titrated under supervision of an anesthetist is our preferred method of car-
dioversion. Cardioversion should not be attempted if there is electrolyte imbalance or suspicion of digoxin toxicity.

If an acute precipitating factor for atrial arrhythmia can be identified and corrected, the patient is usually followed chronically without antiarrhythmic therapy or given digoxin to control the ventricular response if atrial flutter recurs. When chronic therapy is desired, digoxin is the basis and a type IA antiarrhythmic agent such as quinidine gluconate, sustained-release procainamide, or disopyramide is given. Preliminary data suggest that flecainide, encainide, and propaphenone are effective for chronic prevention and by slowing the ventricular rate if flutter should recur. However, according to the results of the Cardiac Arrhythmias Suppression Trial, these agents must be avoided in the patient with recent or acute MI. Amiodarone is also highly effective, but its delayed onset of action, long half-life, and potential toxicity prevents recommendation unless other agents have failed or are not tolerated.

Atrial fibrillation is characterized by a chaotic electrical baseline on the ECG. There is no distinct electrical systole or diastole and no effective mechanical contraction of the atria. Rarely, fibrillation is isolated to one atrium. Because there is no effective mechanical activity, there is a high propensity to thrombus formation and cerebrovascular embolization is one of the most severe complications. The cardiac output may be diminished by 10% to 25% depending on atrial size and effectiveness and the ventricular compliance. In cardiovascular disease with a stiff left ventricle such as asymmetric septal hypertrophy, restrictive cardiomyopathy, or severe aortic stenosis, development of atrial fibrillation may result in hemodynamic collapse. Thus the assessment of atrial fibrillation should include a complete cardiovascular evaluation including an echocardiogram to evaluate atrial size and to search for obvious intramural atrial thrombi.

There are two potential mechanisms of atrial fibrillation. One is a rapid chaotic atrial discharge which suppresses a normal sinus node; the other is an escape mechanism in the presence of a diseased sinus node which results in severe bradycardia or long pauses. In the former instance, if the abnormal atrial fibrillation is electrically, pharmacologically, or spontaneously terminated, normal sinus rhythm supervenes. In the sick sinus syndrome, however, the abnormal atrial arrhythmia may be a vital escape mechanism and its termination may reveal impaired or absent sinus node activity. Whenever this situation is predicted by previous ECGs demonstrating significant bradycardia, sinus pause or arrest, or junctional or lower escape mechanism, electrical or chemical cardioversion should be preceded by placement of a temporary or permanent transvenous pacemaker.

Acute versus chronic arrhythmia

It is essential to determine the premorbid arrhythmia to determine appropriate therapy and predict outcome. All old ECGs should be reviewed to determine the time of onset of atrial fibrillation, the underlying baseline rhythm, and whether atrial fibrillation is a lone event that is unlikely to recur for a long time after successful restoration of normal rhythm or is paroxysmal and likely to be recurrent without therapy in the future. An old Holter monitor may document significant 3-second pauses or symptomatic pauses with a lower junctional escape rhythm demonstrating that a pacemaker should be implanted.

As a “grandfathered” standard of clinical care, the recent literature is devoid of precise statistics on the “odds” of successful cardioversion to normal sinus rhythm. In the individual patient, clinical experience dictates that the acuteness of onset is a good predictor of success, whereas cardioversion is only occasionally successful for atrial fibrillation of six months duration. Atrial size by echocardiography also plays a role, but there are major limitations to accurate atrial measurements by echocardiography. Treatment and reversal of a precipitating cause is a good predictor, i.e., thyroxine excess, congestive heart failure, alcohol consumption, exposure, electrolyte imbalance, antiarrhythmic drug noncompliance. High amplitude “flutter waves” in the baseline imply that the atrium is electrically active and suggest more recent onset and easier cardioversion.

Once these matters have been considered, the basic alternatives for conversion are electrical and pharmacologic. The choice should be based upon informed patient preference and availability of personnel and facilities.

Pharmacologic therapy has the advantage of determining effective long-term therapy and can be initiated on any telemetry-monitoring hospital floor with oral or intravenous drug administration.

If atrial fibrillation is of recent onset, correction of precipitating causes and restoration of normal sinus rhythm is desirable.

If rapid pharmacologic termination is desired, intravenous procainamide is administered, just as described for atrial flutter (24). Verapamil may slow the ventricular response, but seldom, if ever, terminates atrial fibrillation to restore normal sinus rhythm (25). Use of verapamil may be complicated by hypotension and impaired cardiac output.

If an oral pharmacologic approach is preferred, digoxin is usually administered to control the ventricular response. Oral quinidine sulfate, quinidine gluconate, or sustained-release procainamide is then added. I do not advocate high dosage therapy as the likelihood of conversion to sinus rhythm is not assured, but risk of side effects or toxicity is likely.

Cardioversion is effective for atrial fibrillation of recent onset after exacerbating factors are resolved (26). If atrial fibrillation has persisted for two or more weeks, four to six weeks of anticoagulation therapy is required to prevent microthrombi which might be dislodged during cardioversion. The technique is similar to the description for atrial flutter, except that higher energy levels are used, beginning with 100 joules. Our laboratory makes an attempt at 100, 200, and 360 joules with sufficient time for recovery and monitoring of vital signs between shocks.

If precipitating factors can be identified, chronic antiarrhythmic therapy may not be required for the first episode (27). However, if arrhythmia is recurrent, digoxin is useful to help control the ventricular rate. Type IA antiarrhythmic therapy is useful for prevention of atrial fibrillation. In the absence of recent MI or ischemia, flecainide, encainide, and propaphenone alone or in combination with digoxin have been successful in prevention or modulation of paroxysmal atrial fibrillation.

For atrial fibrillation of more than six months duration or which has failed to respond to pharmacologic or electrical car-
dioxigen, control of the ventricular rate is accomplished with
digoxin which may be titrated safely according to ventricular
rate in atrial fibrillation, occasionally requiring unusually high
dosages. Addition of other AV nodal blockers may be required
to modulate the ventricular rate including β-adrenergic blocking
agents, calcium channel blocking agents, or amiodarone (28).

In patients refractory to antiarrhythmic drug therapy, catheter
or surgical modulation of the AV node may be required (29). If
AV nodal ablation is required or results inadvertently, a perma-
nent ventricular pacemaker is required. This is not all bad. Im-
plantation of a newer rate-responsive pacemaker may restore a
degree of appropriate chronotropic responsiveness simulating a
sinus node which the chronic atrial fibrillator has never before
enjoyed. Guiradon and coworkers (30,31) are developing surgical
pacarding techniques to restore sinoventricular communica-
tion. This technique can restore chronotropic function but not
atrial mechanical function. Cox et al (32) are developing a
"maze" procedure to disrupt the reentrant electrical pathways of
atrial fibrillation and restore normal sinus rhythm and transport
function.

AV nodal reentrant tachycardia is a common tachycardia
usually characterized by a narrow QRS complex with retrograde
P waves which are more often than not buried within the QRS
complex or distorting the terminal portion of it. Occasionally a
rate-dependent (usually right) bundle branch QRS pattern re-
results. Carotid sinus pressure may transiently slow or terminate
the tachycardia abruptly. Usually the atrial rate is 140 to 210
beats/min with the symptoms and hemodynamic consequences
dependent on the rate. It is common in patients with no evidence
of cardiovascular disease. Intravenous digoxin or procainamide
are effective for acute termination, as are atrial or ventricular
paering techniques which may also be of diagnostic value. Inter-
estingly, although the reentrant arrhythmia circuit is confined to
the AV node (animal studies have shown that resection of atria
and ventricles does not terminate the tachycardia), type IA anti-
arrhythmic agents are effective in blocking the retrograde limb
of the tachycardia (33,34).

Generically, tachycardias may be classified into three groups
based on duration alone: paroxysmal, persistent, or chronic.
Arrhythmias which are paroxysmal may be of any mechanism,
including VT. By definition, torsade de pointes is paroxysmal.
Paroxysmal atrial tachycardia describes any atrial arrhythmia
which lasts only seconds to hours. It is a mistake to develop a
management plan for paroxysmal atrial tachycardia, because it
is not a specific diagnosis but a syndrome which may include
AV nodal reentrant tachycardia, sinus tachycardia, or atrial fi-
brillation. When the mechanism is determined, specific therapy
follows logically.

Wide QRS Tachycardia

A wide QRS tachycardia associated with hypotension during
acute MI is considered to be VT and is treated as such until
proven otherwise.

In the setting of acute MI or acute ischemic syndrome, wide
complex tachycardia must be terminated immediately. How-
ever, in the absence of acute MI if the hemodynamics and symp-
tomatology permit, appropriate recording, documentation, and
assessment are essential because the arrhythmia is likely to re-
cur, but perhaps never again in a controlled monitored setting
such as the cardiac intensive care unit. The initial electrocardio-
graphic recording during the acute event facilitates appropriate
in-hospital evaluation and long-term outpatient management.
Termination without adequate documentation can result in un-
certainty of diagnosis, delay hospital discharge, and confound
the chronic management plan.

Wide complex tachycardia is defined as a cardiac dysrhythm-
ia having a rate greater than 100 complexes per minute (or R-
R cycle length less than 600 msec) and a QRS duration greater
than 120 msec in any lead on a standardized 12-lead ECG. For
descriptive purposes, the QRS morphology during tachycardia
is assigned a bundle branch block pattern (left or right or nor-
mal). A right bundle branch block pattern is assigned if the QRS
complex is predominantly positive in lead V1; prominent termi-
nal negative S waves in leads I and V5 or V6 are supportive find-
ings. A left bundle branch block pattern is assigned when the
intrinsicsoid deflection is delayed in the lateral electrocardio-
graphic leads (I, and V5 or V6); usually V1 is predominantly
negative. The tachycardia may be further characterized by the
predominant or mean QRS axis during the tachycardia: normal
axis if it lies between 0 and 90 degrees; left axis deviation if it
is less than 0 degrees; or right axis deviation if it is greater than
90 degrees. This classification scheme does not necessarily imply
mechanism or origin of tachycardia but serves as a catalog to de-
termine whether there is recurrence of the same tachycardia or
perhaps tachycardias of different types in the same patient, each
of which may require specific therapy. For example, a patient
may have a polymorphous tachycardia due to ischemia or elec-
trolyte imbalance which will not require chronic therapy and an-
other monomorphic tachycardia due to a reentrant focus (either
supraventricular or ventricular) which is likely to recur after
hospital discharge and requires chronic therapy.

A 12-lead ECG machine, cardiorespiratory resuscitative
drugs and equipment, and a defibrillator should be brought to
the bedside in case cardioversion is required for emergent ter-
ation of life-threatening arrhythmia.

The differential diagnosis of a regular wide complex tachy-
cardia includes: 1) supraventricular tachycardia with a func-
tional bundle branch block, 2) supraventricular tachycardia with
a preexistent bundle branch block, 3) supraventricular tachycardia
with antegrade AV conduction over an accessory AV path-
way (antidromic supraventricular tachycardia), or 4) VT.

Age, heart rate, blood pressure, level of consciousness, and
duration or presence or absence of heart disease are not accurate
determinants of the tachycardia mechanism. VT is the most com-
mon mechanism of wide QRS tachycardia at any age, although
a heart rate of 150 complexes/min suggests atrial flutter with 2:1
heart block. There is no particular heart rate which excludes VT
or supraventricular tachycardia. Either arrhythmia may be fast
or relatively slow. Occasionally patients with fast tachycardia
are alert and conversational despite rapid VT and underlying
ventricular dysfunction. Presumably this results from an opti-
mal balance of a low cardiac output and adequate peripheral
the QRS and P-wave morphologies are distinct and ventriculoatrial association may occur in either supraventricular have supraventricular arrhythmia. Some patients have both.

Ventriculoatrial dissociation is diagnostic for VT, but ventriculoatrial association may occur in either supraventricular tachycardia or VT with 1:1 retrograde conduction. A single-lead electrocardiographic monitoring strip is diagnostic of VT when the QRS and P-wave morphologies are distinct and ventriculoatrial dissociation is apparent. Since wide QRS complexes are usually tall, as well as wide, it is often difficult to discern the smaller P waves on emergency or bedside monitoring equipment because they are buried within the QRS complex or a broad T wave. A fortuitously timed sinus beat may capture the ventricle resulting in occasional ventricular capture beats which are narrow and look like sinus beats (because they are) during the tachycardia. Such sinus beats which occur later in electrical diastole may result in ventricular fusion beats which are a morphologic hybrid of sinus and tachycardia QRS complexes.

Recording three or more electrocardiographic leads simultaneously allows comparison of barely perceptible P waves in several leads and may clarify presence or absence of any temporal relationship to the QRS complexes. If there is a 1:1 relationship with normal P waves preceding each QRS complex, sinus rhythm or other atrial rhythm is present. If there is AV dissociation (i.e., atrial and ventricular activity are independent), then VT is diagnosed. If there is a 1:1 relationship with inverted P waves following each QRS complex, either VT with retrograde conduction or supraventricular tachycardia may be present.

A standard 12-lead ECG should be recorded during tachycardia whenever possible. It provides 1) a better “fingerprint” of the arrhythmia (35,36), and 2) greater likelihood of detecting P waves, and 3) the QRS morphology may be useful to discriminate tachycardia of supraventricular or ventricular origin. A QRS width greater than 140 msec is highly suggestive of a VT. Leftward deviation of the mean QRS vector is also highly correlated with VT. Capture beats and fusion beats, although diagnostic of VT, are relatively uncommon (37). A “concordant pattern” of precordial complexes (i.e., all leads in V1 to V6 are entirely upright or entirely inverted) is highly suggestive of VT, but is not common. In the presence of antegrade conduction over an accessory AV pathway, the ventricle is activated ectopically and such supraventricular tachycardia cannot be distinguished from VT by surface electrocardiography. Furthermore, presence of delta waves in the resting ECG by no means excludes the possibility of VT.

If the mechanism of the arrhythmia remains unclear, positioning of chest leads one interspace higher or lower or on the back (nearer to the left atrium) may reveal P waves. When stable, a “pill electrode” may be swallowed into the esophagus during the tachycardia to record at the level of the left atrium. Otherwise a temporary pacing wire may be passed through a nasogastric tube.

The most reliable method of detection of atrial activity during tachycardia is to record the right intraatrial electrogram by inserting a temporary pacing wire transvenously with fluoroscopic guidance, or a balloon-tipped electrode catheter with electrocardiographic monitoring while the proximal portion of the lead is connected to lead V1. A quadripolar catheter allows simultaneous recording and pacing.

In addition to passive recording, a pacing lead allows other diagnostic and therapeutic interventions. Atrial pacing at rates exceeding the tachycardia rate can artificially produce the phenomena of atrial capture beats and fusion beats with transient normalization or near-normalization of the QRS complexes. The latter method may also allow for termination of the tachycardia by pacing in the atrium. Theoretically, atropine or isoproterenol, by enhancing AV nodal conduction, may facilitate capture and fusion beats but could cause degeneration of the tachycardia to ventricular fibrillation. Many supraventricular tachycardias can be terminated by atrial entrainment, but this is not absolutely diagnostic as relatively slow VTs may also be terminated by impulses conducted across the AV node. If the tachycardia is ventricular in origin, the electrode catheter may be advanced across the tricuspid valve to the right ventricular apex to provide a stable position to attempt to entrain and terminate VT.

Carotid sinus massage may slow or terminate AV nodal or AV reentrant supraventricular tachycardia. It may result in AV nodal block and dissociate the ventricles from the atria during supraventricular tachycardias which originate in the atrium or high within the AV node (sinus node reentry, intraatrial reentry, atrial flutter or fibrillation, or automatic atrial tachycardia). P waves may become apparent. A functional (rate-dependent) bundle branch block may resolve.

Careful observation of any change in the tachycardia rate or termination by an antiarrhythmic drug can be of diagnostic and therapeutic value. Intravenous lidocaine is uniquely effective for VT; it has no efficacy for atrial tachycardias nor reentry over an accessory AV pathway. This specificity has diagnostic value.

Intravenous propranolol, verapamil, diltiazem, or digitalis glycosides slow AV nodal conduction. Hence, a tachycardia whose mechanism depends on anterograde or retrograde AV nodal conduction will become slower and more tolerable. If intermittent AV nodal block is produced, the tachycardia will terminate.

Digitalis glycosides are the least likely to exacerbate hypotension and are preferred if there is evidence of heart failure. The disadvantage is the slow onset of action, even when administered intravenously, which makes them undesirable if the patient is rapidly deteriorating.

Calcium channel antagonists (verapamil or diltiazem) and β-adrenergic blockers (propranolol) are contraindicated if the patient is hypotensive or has evidence of heart failure (35,38). However, if there is no evidence of fluid overload or heart failure, it is valuable to administer about 200 mL of normal saline (0.9% aqueous sodium chloride) intravenously to achieve an adequate intravascular volume and decrease baroreceptor drive for tachycardia. This often corrects hypotension, relieves anxiety, slows supraventricular tachycardia rate, and occasionally allows spontaneous resolution of tachycardia. If the blood pressure is normal or high, calcium channel antagonists or β-adrenergic blockers may be safely given with negative dromotropic effect similar to the digitalis glycosides but more rapid in onset and easier to titrate.
Adenosine, when administered intravenously, blocks AV nodal conduction rapidly. It is metabolized rapidly in plasma and human tissues. Therefore, its duration of action is brief (about 5 minutes). Advantages are rapid termination of supraventricular tachycardia and brief duration of hypotension or other side effects like facial flushing (39).

Esmolol, an ultrashort-acting β-adrenergic blocking agent, also offers the opportunity to observe a brief effect on AV nodal conduction and termination of supraventricular tachycardia while minimizing any residual side effect.

If the patient has a wide complex tachycardia and has a history of the Wolf-Parkinson-White syndrome, delta waves on the ECG in sinus rhythm, or is young without underlying cardiovascular disease, particularly with a left bundle branch block morphology, tachycardia mediated by conduction over an accessory AV pathway must be considered. If an AV nodal blocking agent is administered and atrial fibrillation ensues, rapid conduction over the accessory pathway can be catastrophic and even degenerate to ventricular fibrillation requiring cardioversion (40-43). In such patients, intravenous procainamide is the agent of choice because it may effectively block the accessory pathway and thereby slow or terminate AV reentry or atrial fibrillation or flutter. Procainamide is also effective for termination of VT.

A variety of guidelines have been published to help determine the mechanism of the tachycardia based upon the QRS morphology on the 12-lead ECG. It should be compared side-by-side with the resting ECG in sinus rhythm. If the patient has aberrant conduction with a wide QRS morphology in sinus rhythm, aberration will be present during supraventricular tachycardia. If the QRS morphology is similar during sinus rhythm and tachycardia, the tachycardia origin is probably supraventricular.

The diagnosis can be made electrophysiologically if the patient is stable enough for safe transport to the laboratory during the arrhythmia to allow insertion of a catheter across the tricuspid valve to record a His-bundle electrogram. Otherwise the tachycardia may be electively induced in the laboratory at a later time. A supraventricular tachycardia is recognized electrophysiologically by the presence of a His-bundle potential preceding each QRS complex with an H-V interval equal to or exceeding that recorded when the patient is in sinus rhythm. In contrast, during VT, the H-V interval, if a His-bundle potential is recorded, is shorter than normal. More commonly, the His-bundle potential is buried within the QRS complex during VT and cannot be discerned even on a recording from a catheter properly positioned to record the His-bundle electrogram. This phenomenon may also be observed during antegrade conduction over an accessory AV pathway (preexcitation syndrome) during atrial tachycardia or antiodromic AV reentrant supraventricular tachycardia.

Each patient with sustained monomorphic VT, in the absence of acute MI or other reversible cause, a reasonable quality of life, and a reasonable life expectancy of at least six months, should undergo a diagnostic cardiac catheterization and coronary angiography to determine any correctable anatomic etiologies (44). Thereafter, electrophysiologic testing can determine the mechanism and appropriate therapy (45).

“Wide complex tachycardia” is a syndrome with many clinical presentations and several potential underlying mechanisms. It may or may not be associated with structural underlying cardiovascular disease, may be relatively benign, or may result in sudden death (35,38). The patient must always be evaluated promptly, but critical data must also be gathered emergently in order to assure good long-term diagnostic and therapeutic accuracy. Wide complex tachycardia is a most challenging phenomenon for the cardiologist or electrophysiologist, but alert paramedics, nurses, house officers, and acute care physicians provide the crucial and irreplaceable initial data base which underlies successful long-term management.

Ventricular Arrhythmia

Sustained VT is arbitrarily defined by most experts as a tachycardia of ventricular origin which, if uninterrupted, persists continuously for at least 30 seconds, or is intentionally terminated earlier by cardioversion because of impending hemodynamic collapse. Sustained VT may be terminated by intravenous lidocaine or procainamide, but generally requires cardioversion.

In the patient with acute ischemic syndromes or evolving MI, nonsustained VT should be suppressed because it is frequently a harbinger of ventricular fibrillation. Torsade de pointes, which literally means “turning around the point,” is a descriptive, not a mechanistic, term for a polymorphic VT which appears to turn above and below the isoelectric axis (46). More simply, some QRS complexes appear upright and some appear inverted. It is usually too fast, however, to accurately distinguish the QRS (ventricular depolarization) from the T wave (ventricular depolarization) and hence the negative deflections might be T waves or vice versa. Torsade de pointes, as originally described, is nonsustained or self-terminating, usually within several seconds. This is the major electrocardiographic distinction from ventricular fibrillation which is rarely (some say never) self-terminating.

Torsade de pointes is a syndrome and as such requires further clinical data to determine appropriate therapy (47). It is associated with drug toxicity or proarrhythmia, electrolyte imbalance, acute ischemia, coronary reperfusion, intracranial disease, or congenital long QT syndrome. It is sometimes related to clinical bradycardia. The therapy begins with correction of any reversible exacerbating factors and discontinuation of any antiarrhythmic drugs. If rapidly recurrent, isoproterol infusion at a rate of 1 to 3 µg/min, titrated upward to increase the resting sinus heart rate to a maximum of 100 beats/min, may suppress torsade de pointes. When time permits, placement of a temporary transvenous pacemaker allows more precise control of the heart rate and suppression of arrhythmia. There is some theoretic basis for this therapy if the arrhythmia is due to early afterdepolarizations which are suppressed in cellular preparations by pacing.

Ventricular fibrillation is the most serious of the cardiac arrhythmias and is invariably lethal if not promptly reversed or unless the patient is supported by effective cardiopulmonary resuscitation. The only effective therapy is asynchronous DC cardioversion with 100 to 360 joules. If ineffective, resuscitative...
efforts should continue while improving oxygenation, acid-base status, coronary blood flow, and electrolytes. Cardioversion may then be repeated. Clinically, coarse fibrillation seems more easy to terminate. If fine fibrillation prevails, administration of epinephrine may "coarsen" the undulations on the surface ECG and facilitate cardioversion. Various experts advocate intravenous, intracardiac, or intratracheal administration. Some studies have shown that cardiac levels may be adequate with intravenous or intratracheal administration, avoiding the risks of cardiac, pericardial, or pleural laceration, but controversy prevails and it is common to administer intracardiac epinephrine for otherwise refractory fine ventricular fibrillation or asystole. Calcium chloride, 5 to 8 mg intravenously, may also be useful (48).

Premature ventricular depolarizations are exacerbated by the same factors as any other arrhythmia. In coronary care units and in survivors after MI there is a certain correlation with early mortality, usually due to sudden arrhythmic cardiac death (49-51). Such arrhythmias during acute MI should be suppressed with intravenous lidocaine or procainamide. Interestingly, related symptomatology does not serve to distinguish benign from malignant premature ventricular depolarizations. Although premature ventricular complex (PVC) frequency and complexity, particularly in the presence of left ventricular dysfunction, is highly associated with risk of early mortality after hospital discharge, chronic suppression of PVCs has not been shown to diminish the risk.

Originally the clinical diagnosis of acute MI was almost synchronous with sudden cardiac death; survivors were few. The ability to monitor cardiac arrhythmia and to defibrillate and the development of specialized coronary care units and personnel resulted in a significant population of survivors. Many of these patients, however, remained at risk for sudden death over a period of several years despite return to a functional life-style. Studies to determine the predictors of risk were conducted by cardiologists and often funded by insurance firms. All studies showed that the most common cause of sudden cardiac death in survivors of MI was VT or ventricular fibrillation. The most reliable predictor for risk stratification during periods of relative health was presence of PVCs. Left ventricular dysfunction as measured by the ejection fraction was also an independent predictor.

More detailed analysis of substrata, however, showed that the PVC was a somewhat nonspecific marker. Therapy directed at all patients with PVCs would treat far more patients than appeared actually to be at risk. Several algorithms were developed based upon frequency of PVCs and/or complexity. Complexity was defined by the presence of at least one of several criteria. One of these was the "R on T phenomenon" which describes occurrence of an early PVC on top of the T wave of the previous normal beat or in the vulnerable period which had been determined in animal studies. This represented a period of time in the relatively refractory period when the cells had repolarized enough to be captured by such an early trigger, yet the membranes had not yet achieved the normal resting potential. In these studies repetitive ventricular depolarizations, nonsustained arrhythmia, and sustained VT or ventricular fibrillation might result.

Another criterion for complexity was the presence of multifocal PVCs which were identified by varying PVC morphologies. Although there is a possibility that PVCs from a single myocardial focus may have varying sites of exit and/or patterns of conduction resulting in varying QRS morphologies, this would be difficult to prove by surface electrocardiography alone and hence "multifocal" and "multiformed" are synonymous for clinical purposes.

"Repetitive" forms include pairs of PVCs and salvos or runs of nonsustained VT. Nonsustained VT is defined as a self-terminating run of VT consisting of three or more complexes. Some investigators considered a frequency of 30 or more PVCs per hour to be complex.

There are excellent data correlating complex PVCs with mortality in a number of large studies and hence the "PVC suppression hypothesis" was developed. In other words, it was assumed that suppression of these lethal triggers could prevent sudden death in survivors of acute MI. Antiarrhythmic drugs were then defined clinically by their ability to suppress spontaneous PVCs. Without good data, the hypothesis was generalized to justify the suppression of ectopy in other disease states (52) and even apparently normal individuals without statistical proof of risk.

In the absence of clinical trials to determine the effect of PVC suppression on survival, the clinical practice prevailed. There were sporadic clinical data to suggest that suppression of PVCs did not prove efficacy for prevention of the more important clinical goal of preventing sustained lethal tachyarrhythmias.

Josephson and Horowitz (53) popularized the electrophysiologic technique of triggering, or supplying, PVCs in a clinical laboratory to detect a clinical substrate which could sustain a lethal tachycardia in patients with remote MI or inducible sustained VT. Electrophysiologic studies after acute MI are not yet accepted in clinical centers in the United States.

Chronic suppression of such arrhythmias, if frequent and particularly if associated with runs of nonsustained VT, is probably desirable. However, there is no proof that such chronic suppression prolongs survival, and each oral antiarrhythmic agent is associated with side effects and toxicity in a large number of patients.

In the Cardiac Arrhythmia Suppression Trial, suppression of ectopy after MI by type IC antiarrhythmic drugs was associated with increased mortality (54,55). Therefore, neither flecainide nor encainide should be used for chronic PVC suppression after MI.

Because of the uncertainty about the indications, benefits, and risks of chronic antiarrhythmic therapy for high-risk survivors of MI, several randomized institutional trials are being conducted:

The Coronary Artery Bypass Graft/patch (CABG/patch) trial is designed to determine the efficacy of the automatic implantable cardioverter defibrillator (AICD) on all causes of mortality or sustained ventricular arrhythmias in patients with low ejection fractions and no previous episode of cardiac arrest or sustained ventricular arrhythmia who are undergoing coronary artery bypass graft surgery. Quality of life and cost-effectiveness substudies are being performed concurrently.
The Multicenter Automatic Defibrillator Implantation Trial (MADIT) is designed to determine if the survival of high-risk, low ejection fraction, coronary artery disease patients with nonsustained VT and electrophysiologically inducible VT who have not yet experienced sudden cardiac death or sustained VT can be improved with AICD device therapy versus conventional medical management. The study will also include quality of life and cost-effectiveness substudies.

The Multicenter Unsustained Tachycardia Cardiac Trial (MUSTT) is a North American study designed to determine if therapy guided by electrophysiologic testing will reduce the risk of sudden cardiac death in patients with coronary artery disease and low ejection fraction who have nonsustained VT. The AICD device will be used as adjunctive therapy in one limb of the study design.

The AICD cost-effectiveness trial is designed to determine if the AICD device, when used as the therapy of first choice for both MI patients and those who have survived cardiac arrest, is a more effective treatment modality than conventional medical evaluation and therapy.

At Henry Ford Hospital a study is currently being conducted to assess psychological implications of AICD therapy as opposed to electrophysiologically guided medical therapy in patients who are effectively controlled on either regimen. The study is neither randomized nor controlled. The purpose is to determine whether patients can or are likely to have an appropriate sense of well-being with either or both types of therapy and also give insight as to what psychological and teaching interventions might be beneficial for this patient population.

References


109:905-12.