ARRHYTHMIAS IN MYOCARDIAL INFARCTION (1996)

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Algorithm for the Management of Bradyarrhythmia in Myocardial Infarction

FIGURE 1
A. Sinus node dysfunction (sick sinus syndrome) constitutes a spectra of cardiac arrhythmias including sinus bradycardia, sinus arrest, sinoatrial block, and paroxysmal supraventricular tachycardia alternating with periods of bradycardia or even asystole (1-3).

B. By its parasympatholytic (anticholinergic) activity, atropine sulfate reduces vagal tone, enhances the rate of discharge of the sinus node and facilitates atrioventricular (AV) conduction. During the early moments of acute ischemia or acute MI, atropine is particularly useful in treating sinus bradycardia with associated reduced cardiac output and signs of peripheral hypoperfusion, including arterial hypotension, confusion, faintness and grayish pallor, or frequent premature ventricular contractions. As a rule however, in the absence of hemodynamic compromise, treatment of sinus bradycardia is not indicated (4).

The recommended dosage of atropine for bradycardia is 0.5 mg intravenously, repeated if needed every 5 minutes to a total dose of no more than 2mg, the amount that produces complete vagal blockade. The peak action of atropine given intravenously is observed within 3 minutes (4).

C. Pacemaker insertion is the treatment of choice for symptomatic bradycardia not responding promptly to atropine administration (drug resistant) (4).
Algorithm for the Management of AV Conduction Disorders in Myocardial Infarction

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**FIGURE 1A**
NOTES

A. AV dissociation disturbances of varying degrees may be observed in acute MI. The incidence of AV block varies markedly depending upon the location of myocardial infarction; occurring in up to 30% of patients with inferior wall MI, typically AV nodal in origin. The significance of AV block in AMI must be assessed in the light of clinical setting, the location of the block, the patient’s hemodynamic status. The use of cardioactive medications and other factors such as autonomic tone may be more important than the degree of block (5-6).

B. High-grade or complete AV block usually occurs within the first 24 hours and almost all of the blocks appear within the first week following MI. Complete AV block appearing after this period is extremely unusual unless further extension of the infarction occurs. In anterior wall infarction, AV block is due to permanent damage in the interventricular septum. The site of the block is below the bifurcation of the bundle with marked hemodynamic alterations. Adams-Stokes syndrome is very common (4,5,7).

C. Whether temporary transvenous pacing improves survival of patients with acute myocardial infarction remains controversial. It is generally agreed however that artificial pacemaker is required in all patients with complete AV block complicating MI regardless of site of block or infarction. A permanent pacemaker is usually indicated when complete AV block is associated with anterior wall MI (4).

D. Third degree AV block in inferior wall infarction results from a block above the bifurcation of the bundle. Damage to the AV junction is usually reversible, resolving in a week, with only slight hemodynamic alterations (4,5,7).

E. Second degree AV block type II is considered to be due to infranodal block and is frequently a precursor of AV block. It is almost always associated with anterior wall MI. Because of its potential for progression to complete heart block, temporary pacing has been recommended in most studies. In our setting however, we chose to observe these patients first; ready to pace in case of progression to complete AV block or hemodynamic instability.

F. Atropine is the drug of choice for the occasional treatment of type I 2nd degree AV block, especially when complicating inferior MI. When AV block is associated with congestive heart failure, hypotension or frequent and complex ventricular arrhythmia, atropine may improve AV conduction and increase the sinus rate and may exert the need for immediate insertion of transvenous pacemaker. As a rule, however, in the absence of hemodynamic compromise, treatment is not indicated (4).

G. First degree AV block is relatively common but is often unrecognized in acute MI because of its transient nature. First degree AV block generally does not require specific treatment, except when it is a manifestation of excessive vagotonia with sinus bradycardia and hypotension in which atropine may be helpful (4,5,7).
Algorithm for the Management of Tachyarrhythmias in Myocardial Infarction

FIGURE 2
Algorithm for the Management of Supraventricular Tachyarrhythmia in Myocardial Infarction

**Figure 2A**

1. **Atrial Fibrillation?**
   - If yes, proceed to class I/III antiarrhythmic drugs (B).
   - If no, proceed to cardioversion.

2. **Atrial Flutter?**
   - If yes, proceed to ß-blocker.
   - If no, proceed to cardioversion/pacing.

3. **Atrial/Junctional Non-Paroxysmal Tachycardia**
   - If yes, proceed to digitalis assay.
   - If no, see Figure 2A1.

4. **Digitalis Toxicity?**
   - If yes, discontinue digitalis.
   - If no, treat as AVNRT.

5. **Sinus Rhythm Restored?**
   - If yes, observe.
   - If no, rate control (C).

**Figure 2A1**

- [ ] Y
- [ ] N

**FIGURE 2A**
A. Atrial fibrillation is the most common new supraventricular arrhythmia in patients with acute myocardial infarction. It usually appears during the first 48-72 hours and in 90% of patients within the first 4 days but it is believed to be rare during the earliest stage of the infarction. Atrial fibrillation is also frequently associated with right ventricular infarction. In about 1/2 of patients with AF, the onset can be traced to an atrial premature complex and in another half to atrial flutter. Factors believed to contribute to the appearance of AF in setting of AMI included atrial infarction; release of catecholamine, acute pericarditis, preexisting atrial muscle disease, chronic lung disease, acute hypoxia, drugs and hypokalemia. In about half of the patients, the episodes of AF are single and in another half, multiple. AF complicating AMI has adverse prognostic implications. There is no reason to expect that prognosis will be ameliorated by treating AF; the arrhythmia merely seems to be marker of a phenomenon which in itself carries the prognostic burden.

B. Treatment is indicated and may take one of 2 approaches: ventricular rate control or restoration of sinus rhythm. Restoration of the sinus rhythm is the ideal. This could be achieved with Class I agents if there is no contraindication, or Class III antiarrhythmic agents (5,7-9).

C. Adenosine, beta-blockers or verapamil may be given quickly to slow the ventricular rate. In patients with left ventricular dysfunction, digoxin is often the drug of choice. If arrhythmia persists and causes hemodynamic instability, electrical cardioversion (100-400 joules) is applied followed by preventive therapy and anticoagulation (5,7,9).

D. Atrial flutter occurs as the first arrhythmia in 1-3% of patients with AMI. Because of clinical deterioration associated with persistence of this arrhythmia, intravenous drug administration or cardioversion is frequently indicated (5,7-9).

E. This arrhythmia is attributed to an abnormal automaticity or to triggered activity associated with delayed afterdepolarization. This arrhythmia appears to be an independent prognostic marker for cardiogenic shock. The rate of tachycardia is more rapid and mortality rate is higher in patients with anterior than in those with inferior infarction. Digitalis toxicity should be ruled out first prior to any intervention. The general approach is the same as for AV nodal reentrant tachycardia (5,7,9).
Algorithm for the Management of Supraventricular Tachyarrhythmia in Myocardial Infarction

**FIGURE 2A-1**

- **AV nodal reentry?**
  - Y: adenosine, β-blockers, amiodarone
    - Y: responsive?
      - Y: observe
      - N: cardioversion
    - N: multifocal atrial tachycardia? (B)
- N: frequent PACs? (C)
  - Y: observe
  - N: sinus tachycardia (D)
  - Y: β-blockers (E)
  - N: multifocal atrial tachycardia? (B)

**Notes:**
- ß-blockers
- Amiodarone
- Adenosine
- Multifocal atrial tachycardia
- Frequent PACs
- Sinus tachycardia
- Treat underlying disease

**Legend:**
- Y: Yes
- N: No
A. Tachycardia dependent on dual AV nodal conduction or a concealed AV bypass are uncommon in patients with AMI. When present this arrhythmia may contribute to hypertension and low cardiac output. Adenosine is now the initial drug of choice unless it is contraindicated. If ineffective, beta-blockers may be given. If arrhythmia remains refractory, amiodarone is given intravenously. If the condition is hemodynamically unstable, electrical cardioversion may be necessary (5,79).

B. Atrial tachycardia is usually benign, and is not associated with increased incidence of congestive heart failure, pericarditis, or atrial infarction. Because of the transient nature and short duration, treatment is seldom required. The underlying cause, usually pulmonary insufficiency should be treated. The drug of choice if needed, is verapamil (5,7,9).

C. Isolated premature atrial complexes occur commonly in patients with AMI, but because of a high prevalence of this arrhythmia in the general population, the role of MI in their pathogenesis is not always obvious. It may indicate atrial dilatation, excessive autonomic tone, or occult heart failure. Treatment is seldom required even with frequent premature atrial complexes. Occasionally, one of the Class I anti-arrhythmic drugs may be used (5,7,9).

D. Sinus tachycardia occurs in about 1/3 of patients and is attributed to sympathetic overactivity particularly when associated with hypertension. Persistence of tachycardia for more than several days usually signifies ventricular failure and represents unfavorable prognostic sign. It is an undesirable rhythm because it results in augmentation of myocardial oxygen consumption and reduction in time available for coronary perfusion (5,7,9).

E. The underlying cause of sinus tachycardia (anxiety, fear, pain, low cardiac output, hypertension) should be addressed first. In some cases, administration of low doses of beta-blockers is helpful particularly when the arrhythmia is a manifestation of a hyperdynamic circulation but is contraindicated in the setting of hypovolemia and LV failure (5,7,9).
Algorithm for the Management of Ventricular Tachyarrhythmia in Myocardial Infarction

ventricular tachyarrhythmia in MI

ventricular fibrillation?

(A)

Y

defibrillation

anti-arrhythmic

(B) drugs

N

AVRT?

(C)

Y

transient?

Y

atropine

responsive?

Y

observe

N

N

atropine

pacing

N

N

repetitive VEB’s (couplets, salvos) isolated VEB’s

(D)

Y

lidocaine bolus and drip

(E)

(+)

response

Y

taper drip

N

hemo-
dynamically stable?

N

other anti-arrhythmic drugs except IC

(F)

cardioversion

FIGURE 2B
NOTES

A. Four to eighteen percent of patients with AMI develop ventricular fibrillation. It occurs with equal incidence in anterior and inferior Q-wave infarction and is rare with non-Q wave infarction. Primary VF is responsible for more than 80% of all instances, occurs suddenly and unexpectedly with no or few signs of LV failure. Secondary VF, on the other hand, is the final phase of a progressive downhill course with LV failure and cardiogenic shock.

The effect of primary VF continues to be debated. The MILIS study group showed that it does not have adverse effect while the GISSI trial suggest that excess mortality due to primary VF occurred only during the hospital phase but not thereafter. On the other hand, secondary VF occurring in association with marked LV failure or hypotension clearly entails a dire prognosis with only 20-25% of patients surviving hospitalization (7,11-12).

B. The treatment of VF is electrical countershock implemented as rapidly as possible. Irreversible brain damage may occur within 1-2 minutes particularly in elderly patients. The initial DC shock should be of 200-400 joules (7-8).

C. The potential for random reentry is a feature of the ischemic myocardium that exists in the acute phase MI. Frequent VEBs occurring very soon after the onset of MI, particularly during the first hour, may depend primarily on reentry rather than increased automaticity. At this time, lidocaine which impairs ventricular myocardium and diminishes automaticity may be somewhat less effective. If persistent, atropine may prove to be of benefit (7-8).

D. Ventricular ectopic beats (VEBs) are ubiquitous in anterior MI. Neither their form nor frequency has prognostic significance. They may arise from a variety of mechanisms and may follow a pattern of expression that varies over follow-up. There is no mandate for their treatment. Most studies have shown that prophylactic lidocaine therapy, though it may decrease the incidence of VF, has not improved mortality. In our setting, we do not advocate the use of prophylactic lidocaine treatment of AMI, but we however take an aggressive stand on treating VEBs, even isolated ones on the background of AMI (8-9).

E. Lidocaine is the drug of choice for the management of ventricular ectopy and non-sustained ventricular tachycardia in the setting of AMI. Before deciding to begin administration of lidocaine it is important to consider that ancillary factors may increase the likelihood of ventricular arrhythmia, including inadequate oxygenation, electrolyte imbalance such as hypokalemia and hypomagnesemia, cardiac failure, digitalis toxicity and recurrent myocardial ischemia.

A loading dose of multiple bolus injections are necessary to initiate lidocaine therapy to achieve therapeutic blood levels rapidly. An initial bolus injection of 1 mg/kg not to exceed 100 mgs. Additional bolus of 0.5 mg/kg every 8-10 minutes if necessary to a total of 4 mg/kg. Maintenance of blood level is accomplished by administration of 20-50 ug/kg/min (1.4-3.5 mg/min). Elimination is almost exclusively by the liver. Half life averages 1-2 hours in normal subjects, more than 4 hours in patients with relatively uncomplicated MI and more than 20 hours in MI complicated by cardiac failure and even longer in cardiogenic edema. Patients treated with an initial bolus injection followed by a maintenance infusion may experience transient subtherapeutic plasma concentration at 30-120 minutes after initiation of therapy. A second bolus injection of 0.5 mg/kg without increasing the maintenance infusion rate reestablishes the therapeutic concentration. If arrhythmia recurs after 6-10 hours (that is at steady state infusion), a similar bolus injection should be given and maintenance infusion rate increased. The half-life of lidocaine increases after 24-48 hours so dose should be reduced by 1 mg/min preferably at 12 hours but at least by 24 hours or blood levels should be monitored or both (1,7-9).

F. The Cardiac Arrhythmia Suppression Trial (CAST) used the drugs flecainide, encainide and moricizine to suppress ventricular arrhythmia in asymptomatic or mildly symptomatic MI patients. Results of the trial suggest that chronic prophylactic anti-arrhythmic treatment of asymptomatic ventricular ectopy is not indicated after MI (7,10).
References


