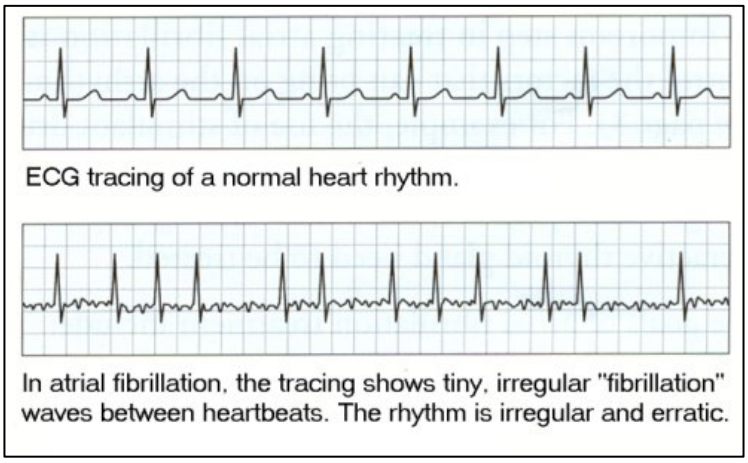
Atrial Fibrillation

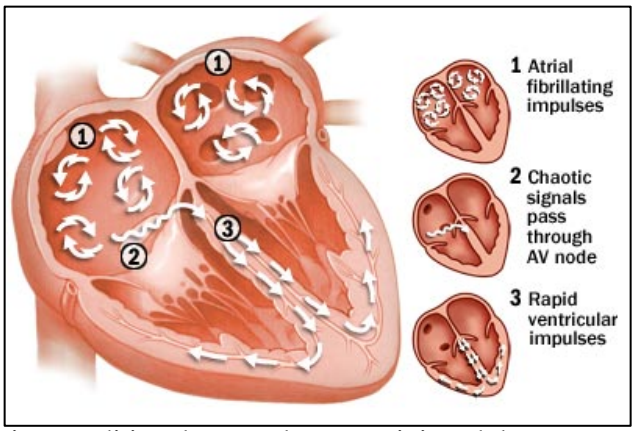
Anesthetic Pearls: Anesthetic Implications and Management of Atrial Fibrillation



Atrial Fibrillation (A-Fib) by definition is a rapid chaotic rhythm of the atria involving atrial conduction rates exceeding 350 beats per minute. The classic A-Fib moniker is "irregularly irregular." A-Fib can cause significant hemodynamic effects due to decreased diastolic filling time with the rapid heart rate and the loss of atrial kick to fill the ventricle. CHF and hypotension may occur acutely, especially with mitral stenosis.

Etiology of Acute Atrial Fibrillation:

1. Metabolic disorders: acid / base disturbances
2. Acute binge drinking
3. Vascular accidents
4. Sympathomimetic drugs
5. Electrolyte disturbances (hypo-kalemia & hypo-magnesimia)
6. Surgical interventions (recent cardiac surgery have post-op A-Fib rates >30%)
7. Sudden volume overload to the atria (R & L)
8. Chronic heart disease / failure (may develop acute A-Fib)
9. Acute thyroid crisis



Treatment: The hemodynamic response to A-Fib determines whether treatment is necessary.

Acute Atrial Fibrillation must be treated if there is acute onset of significant hypotension or rapid ventricular response (RVR). The acuteness of the A-Fib leaves the patient at high risk for ischemia or CHF. The optimum treatment is to correct the underlying condition that may have precipitated the A-Fib. If treatment by pharmacologic needs is deemed necessary, many choices are available. Slowing the ventricular response (**Rate Control**) is the primary goal of acute therapy.

Rate Control Agents:
Digoxin (purified cardiac glycoside: 0.5 mg IV bolus until rate controlled)
Esmolol (beta blocker: 0.5 mg/kg bolus until rate controlled, then begin infusion at 0.05-0.2 mcg/kg/min)
Verapamil (calcium entry channel blocker: 0.1 mg/kg bolus until rate controlled)

Atrial pacing that overrides the supraventricular arrhythmia may help convert back to sinus rhythm if this modality is available. Another option is **synchronized cardioversion**, if the patient is a candidate anesthesia. Pharmacologic therapy is generally preferred if the patient is awake and not in serious hemodynamic jeopardy.

If the patient has underlying Wolf Parkinson White (**WPW**) physiology or rapid re-entry phenomena (challenging to distinguish from A-Fib), treat with **Adenosine** (5 mg IV bolus) or **Verapamil** (0.1 mg/kg bolus). Digoxin is contraindicated in patients with WPW.

Other adjuvant therapy that may be considered are **vagal maneuvers** (carotid massage, Valsalva, ocular pressure) to slow the heightened ventricular response rate. If the patient is near the end of an anesthetic and neuromuscular blocking reversal is being given, the side effects of **Neostigmine** (bradycardia induction) will also help slow the ventricular response. The use of **Phenylephrine** to treatment the underlying hypotension of A-Fib is also appropriate and may help slow the heart rate reflexively.