

# Subarachnoid Block Associated Atrial Fibrillation

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## Abstract

We describe a case, where after subarachnoid block, atrial fibrillation (AF) was precipitated despite the heart was normal structurally and functionally. Since the only risk factor present was increased vagal tone due to subarachnoid block, we attributed it to the causation of AF.

## INTRODUCTION

Parasympathetic and sympathetic stimulation are known to cause AF, by macroreentry and microreentry(triggered ectopics) respectively.It is important to recognize this,since missing the etiology might result in improper treatment,might even be detrimental <sup>1,2</sup>.

## CASE REPORT

A 65 yrs old female came for grafting of post burn contracture. Her medical history was conspicuous by the absence of symptoms of thyrotoxicosis, pneumonia, structural heart diseases, electrolyte imbalance etc which are the known precipitants of AF. Preoperatively her pulse was 80/min and blod pressure was 140/90mm Hg. Preloading WAS done with 1000 ml ringer's lactate. Subarachnoid block was achieved with 3.5ml of injection bupivacaine 0.5% heavy, after 10 minutes a sensory anaesthesia level achieved was T6. Her pulse was 75/min and B.P.120/80 mm Hg.

After about 45 minutes, the cardioscope started showing gradual decrease of heart rate and15 minutes later, sudden tachycardia of 140 bpm with replacement of "P" waves by "f" waves was noted. Pulse was irregular. B.P.80/50 mm Hg. Injection mephenteramine 6 mg was given intravenously. Blood pressure rose to 90/60 mmHg. Injection metoprolol 5mg was given, but no change in pulse or B.P. was noted.15 minutes later an injection of diltiazem 5 mg was given intravenously. Heart rate fell to 110 bpm. B.P. rose to 106/70 mmHg. Though we were ready with a defibrillator, the patient's hemodynamic stability and lack of unconsciousness rendered defibrillation unnecessary.

After the surgery of a duration of 4 hrs, the patient was

shifted to the intensive care unit, where a complete cardiology work-up including 2D-echocardiography was done by the cardiologist. Except for the AF, no other abnormality was noted. Serum electrolytes remained normal.

6 hrs after giving spinal anaesthesia she regained complete motor and sensory functions, but A.F. persisted. After another 5 hrs she reverted spontaneously to sinus rhythm. She was stable hemodynamically.B.P.130/90mm Hg. Pulse 80 bpm, when shifted to the ward the next day.

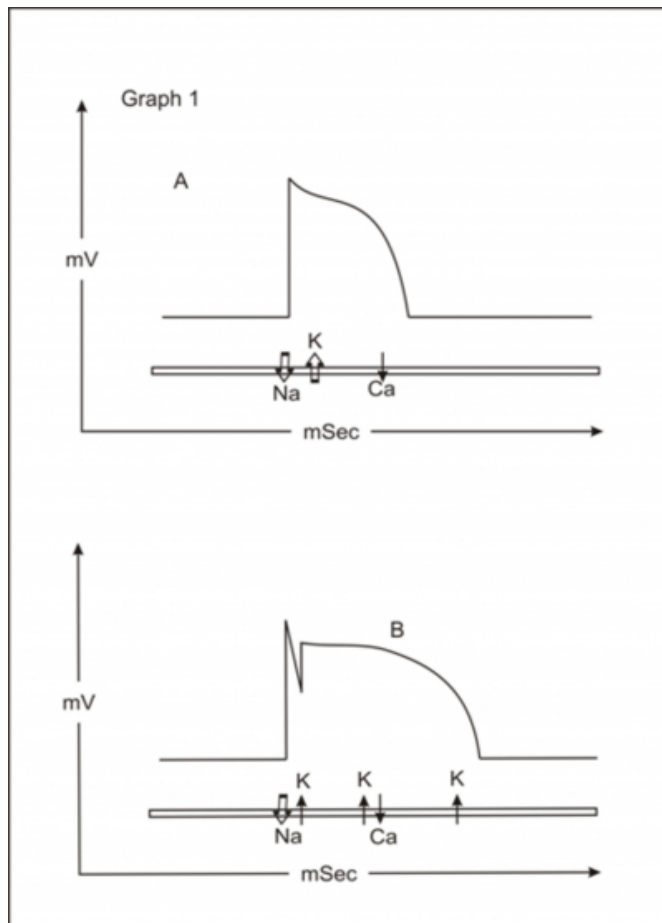
## DISCUSSION

Among the known factors precipitating atrial fibrillation, vagal stimulation assumes a special importance, because it is less commonly identified etiology. Since treatment of atrial fibrillation due to other causes differs radically from vagal mediated atrial fibrillation, conventional treatment may not be effective or even be detrimental <sup>1,2,3,4</sup>

Electrophysiological properties of ventricular and atrial muscles are very different. Compared to ventricles atria have lesser amplitude of phase 0, almost nonexistent phase 1, very short phase 2.This correlates well with the shorter action potential duration and mechanical systolic time of atria compared to the ventricles (Graph 1).

**Figure 1**

Graph 1: Electrophysiological differences between Action potentials of atrial (A) and ventricular (B) myocardia.

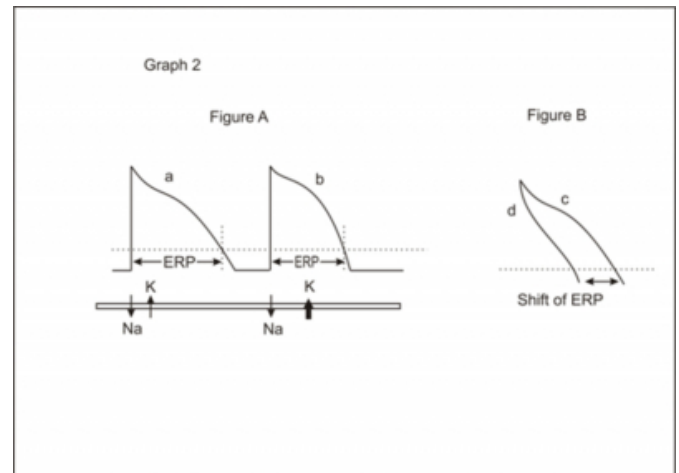


Note shorter action potential duration of atrial myocardium.

Influence of autonomic stimulation is also different between atria and ventricles. In the ventricles, because parasympathetic innervation is poor, vagal stimulation has almost insignificant change of either level of resting membrane potential (RMP) or action potential (AP) morphology. Whereas in the atria, vagal stimulation enhances Ach-gated K channels, hence reducing the duration of phase 2 further<sup>5,6,7</sup>. Also the slope of phase 3 is increased. This hastens the attainment of RMP of -55mV. So the inactivated sodium channels are activated by opening of “h gate”, much earlier. This decreases effective refractory period (ERP)<sup>7,8,9</sup>. More importantly these effects are non uniform. Some fibers are affected more than others. Therefore temporal dispersion of inhomogenieties is strongly enhanced<sup>9,10</sup> (Graph 2).

**Figure 2**

Graph 2: Fig A. The transmembrane potential changes of atrial musculature pre and post vagal stimulation (a and b respectively). Enhancement of Ach gated K channels increases the slope of repolarisation, Fig B. Change in the slope of repolarisation resulting in reduced ERP, pre and post vagal stimulation (from c to d respectively).

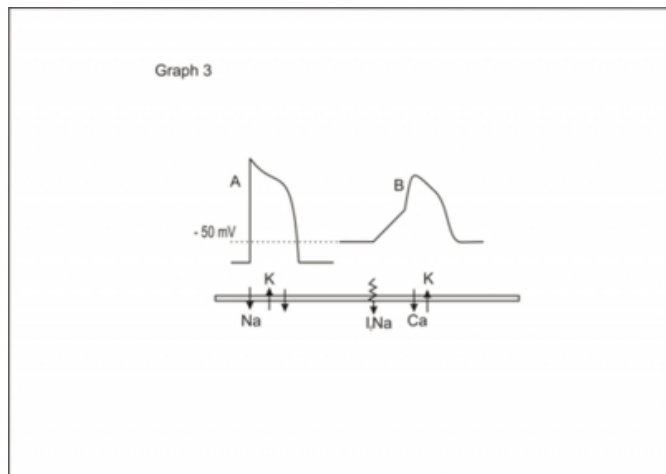


This can be proved experimentally by eliciting AF by intravenous injection or topical application of acetylcholine. Acetylcholine increases the liability of the atria to respond with fibrillation to a single electrical stimulus which in absence of the drug would elicit only one extrasystole<sup>7</sup>. Microelectrode studies show that acetylcholine shortens refractory period from 100ms to 10ms or less. Due to nonuniform affection, conduction velocities vary between the fibers. This contributes to inhomogeneity in refractoriness and spread of excitation and favour “functional fragmentation”<sup>11,12,13,14</sup>

Electrophysiological properties of SA node and AV node are different from the rest of the atria. The main differences are SA,AV nodes can function as pacemakers by triggering action potentials regularly and spontaneously. This is possible by the existence of “ $i_f$ ” current, “inward funny current” of sodium channel. The slope of Phase 0 is less steep, dominant ion responsible for the AP is calcium, not sodium as in atria. These differences are fundamentally due to less negative RMP of SA-AV nodes in contrast to the atria. Due to less negative RMP~-50mV, “h gate” of voltage gated sodium channels are inactivated hence the dependence of AP on calcium channels, also the extreme sensitivity of SA and AV nodes to be blocked by the calcium channel blockers (Graph 3).

**Figure 3**

Graph 3: Electrophysiological differences between atrial (A) and nodal cells (B).



Note the dependence of AP generation on  $i_{Na}$  and  $i_{Ca}$ , not voltage gated  $i_{Na}$ .

While treating the patient, heart rate control assumes paramount importance in case of hemodynamic instability. Decreasing the heart rate, improves ventricular filling and B.P. increases.

Digoxin and beta-blockers compound the problem by increasing the vagal tone further due to sympathetic blockade, are avoided<sup>2,3,4,15</sup>. For rate control calcium channel blockers and for reversion of rhythm sodium and potassium channel blockers might be the preferable option<sup>16,17</sup>.

In the end, we conclude that, understanding the electrophysiology of vagal mediated atrial fibrillation, rather than the protocol, helps the anaesthesiologist deliver better patient care.

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