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## Some aspects of atrial fibrillation

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SOME ASPECTS OF ATRIAL FIBRILLATION

by

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A THESIS

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## ANATOMY OF CARDIAC CONDUCTION

The conduction system of the human heart is made up of the following:

- (1) The sinoatrial node: The sinoatrial node is a small strip of specialized muscle approximately 3 mm by 1 mm situated in the crista terminalis at the junction of the superior vena cava and right atrium. The fibers of this strip though only 3 to 5 microns compared to 15 to 20 microns for the rest of the atrium, are continuous with these larger fibers.
- (2) The atrioventricular node: The atrioventricular node lies near the orifice of the coronary sinus in the septal wall of the right atrium. It is composed of branching and irregularly arranged fibers, originally about the same size as the SA node fibers, but getting progressively larger until the terminal branches are normal sized again and continue on into the atrioventricular bundle.
- (3) The atrioventricular bundle (Bundle of His): The atrioventricular bundle is a continuation of the fibers from the AV node. It follows the membranous septum towards the mitral valve, but at the middle of the septum it splits into the right and left bundle branches. (a) the right bundle branch continues under the endocardium to the apex, spreading to all parts of the right ventricle. (b) the left bundle branch penetrates the fibrous septum and lies just under the endocardium of the left ventricle. The fibers of the bundle branches are the terminal conducting fibers of the Purkinje system and become continuous with the regular heart muscles (36,37).

## PHYSIOLOGY OF CARDIAC RHYTHM

With the development of membrane physiology and micro electrode technique, muscle fibers in general, were shown to have the properties of excitability, conductivity and contractility.

Excitability is the ability to react to a stimulus. Contractility is a function of the myosin and actin filaments in the sarcomeres. Conductivity is the function of the propagation of an action potential or the release of an electrical potential across a membrane created by selective concentration of ions on both sides of the membrane (See Figure A).

The ionic shifts that produce the action potentials have been well described and are not pertinent here. In the resting state there is normally a difference in potential across the membrane of 80-90 mv (the resting potential). Any stimulus that reduces this above the threshold of roughly 50-60 mv will cause a discharge of the potential of about 105 mv or from -85 to +20. The speed of which this discharge (action potential) can be repeated is limited by the length of time it takes for the potential to go below the threshold again. This is called the repolarization time. When the charge reaches the resting potential it awaits another stimulus to elevate it above the threshold and trigger another action potential. The trigger mechanism usually follows the all or none law and the stimulus must be of sufficient strength to reach the threshold before an action potential fires. Increasing the stimulus above that necessary to reach the threshold adds nothing to the action potential because it is already "all" fired. From this,

Figure A.

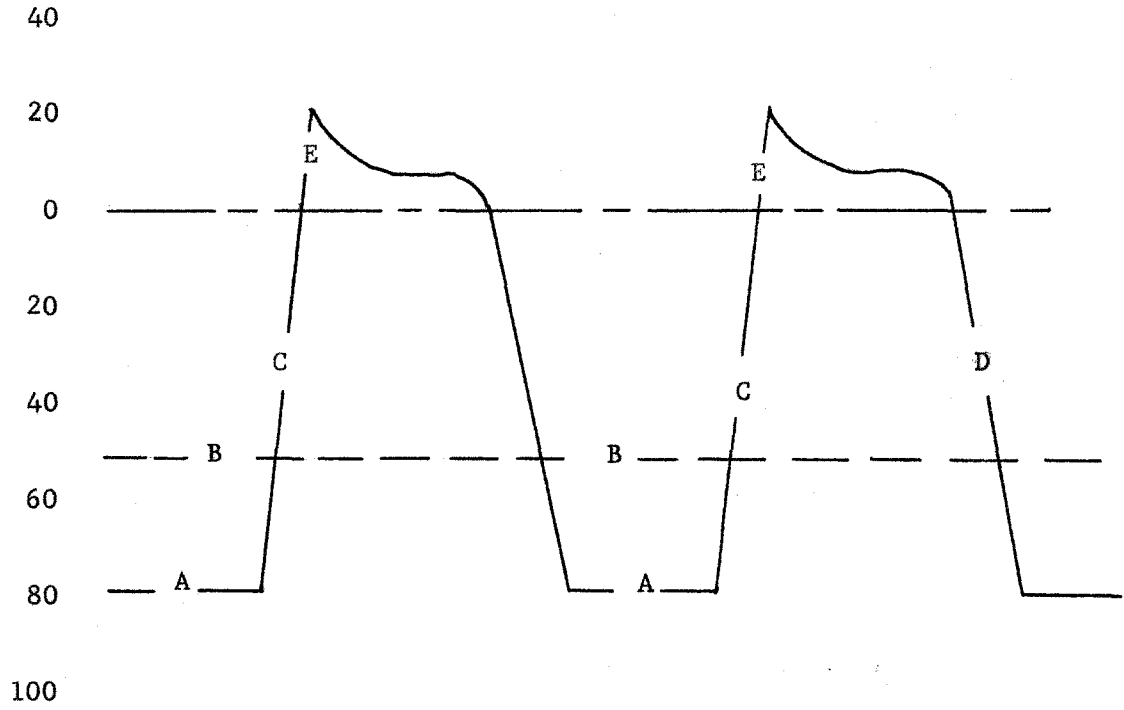


Diagram of Cardiac Muscle Action Potential (38)

- A = Resting Potential
- B = Threshold
- C = Depolarization
- D = Repolarization
- E = Overshoot

it can be easily seen that the closer the resting potential is to the threshold the smaller the stimulus required to initiate an action potential (See Figure B).

The specialized muscle cells of the SA, AV and Purkinje system all have another property, self-excitation or automaticity. In these cells the resting potential is not stable, but spontaneously decays toward the threshold until it reaches it and triggers another action potential (See Figure C).

The most rapidly firing of these automatic tissues will force its rhythm upon the others and will become the pacemaker of the heart.

The SA node normally performs this function. The resting potential of the SA node is only 55-60 mv, compared to 80-90 mv in other cardiac tissues. Under normal conditions of uninterrupted conduction, a constant decaying rate, constant threshold, and constant resting potential, the SA resting potential only decays from 60 to about 55 mv and therefore this tissue will fire more rapidly than tissues in which the resting potential must decay from 85 to 55 mv in order to trigger an action potential.

When one of the special fibers of the SA node fires itself, the impulse is spread throughout the atrium providing the stimulus for the firing of action potentials from the non-automatic cells. As this impulse spreads through the atrial muscle, the atrium contracts, forcing blood through the mitral and tricuspid valves into the ventricles.

Figure B.

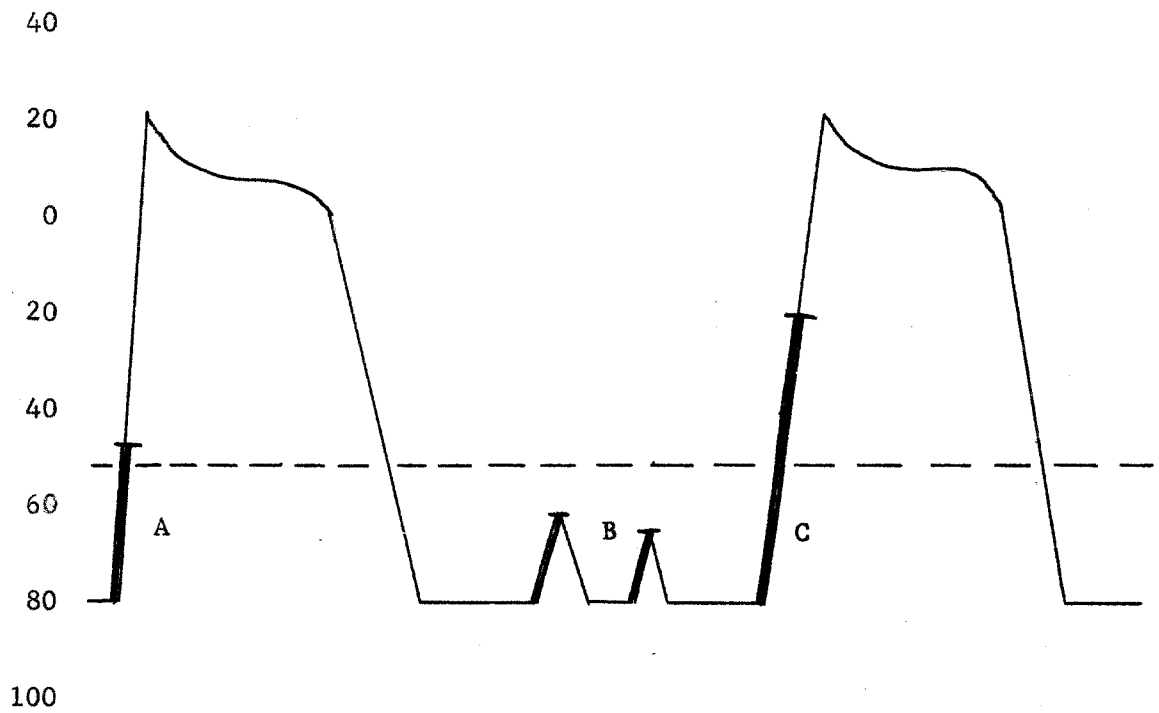


Illustration of Stimulus and All or None Law (38)

- A = Stimulus Above Threshold
- B = Stimulus Below Threshold
- C = Stimulus 2x Threshold



Figure C

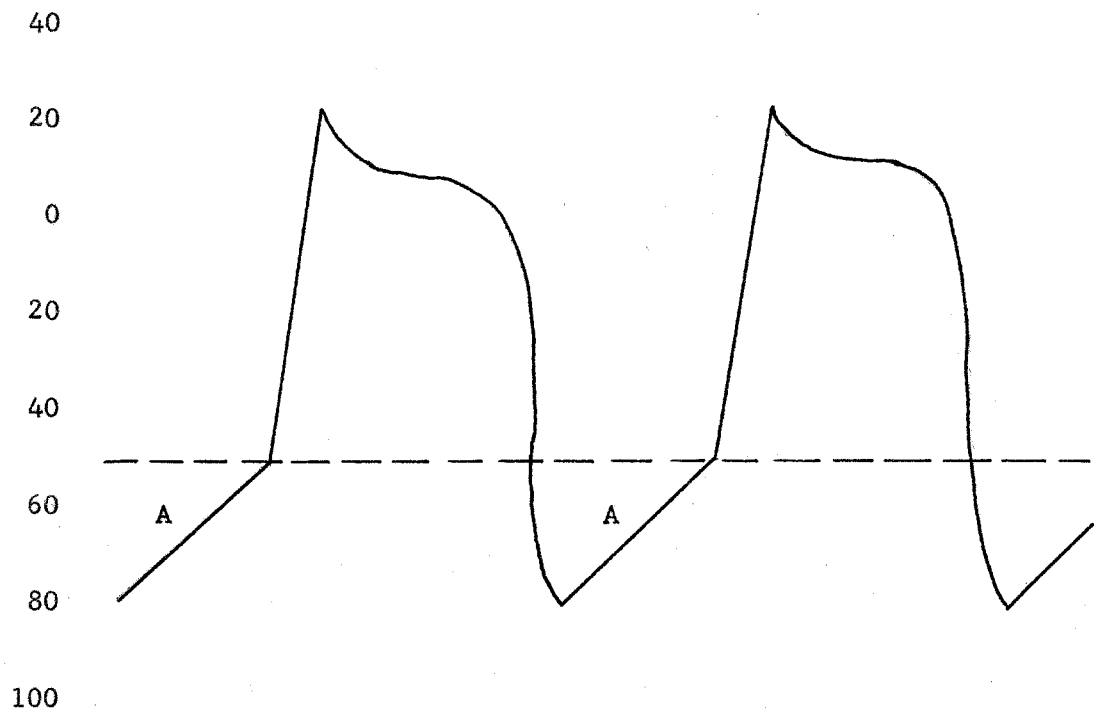


Diagram of Automatic Tissue Action Potential (38)

A = Decay of Resting Potential

This impulse travels at a rate of about 0.3 m/s reaching the AV node in about 0.04 seconds and reaching the farthest part of the atrium in about 0.09 seconds.

When the impulse reaches the AV node it is radically slowed down so that it takes about .11 seconds for it to get through the AV node into the Bundle of His. Most of this delay occurs in the very small junctional fibers with their velocity of less than 0.05 m/s. However, once the impulse is into the larger nodal fibers, the rate is still only about 0.1 m/s though it increases as the nodal fiber size increases until it reaches the quite large Purkinje fibers.

The purpose of this delay is to allow the atrium to complete their emptying before the ventricles begin to contract.

As the impulse reaches the Purkinje fibers of the Bundle of His the rate is rapidly increased, these fibers are even larger than the normal ventricle muscle and conduct at about 1.5 to 2.5 m/s spreading the impulse over the entire endocardium in about 0.03 seconds. The Purkinje fibers are continuous with the normal muscle fibers of the ventricle. It takes about another 0.03 seconds for the impulse to be transmitted from endocardium to the epicardium. The effect of the very rapid transmission is to have all the ventricle fibers contract almost in unison, giving a short, but very strong, push to the circulating blood.

To summarize the normal: The resting potential of the automatic tissue began decaying toward the threshold. The fibers of the SA node having the shortest distance to go, reach the threshold first and

start an impulse. This spreads over the atria, contracting the muscle fibers and forcing blood into the ventricles. The impulse pauses in the AV node to allow completion of the atrial contraction, then is rapidly delivered to the ventricle muscles by the Purkinje system, allowing the ventricle to contract almost as a unit forcing blood into the greater vessels. (37,38)

#### MECHANISMS OF DISTURBANCES IN CARDIAC RHYTHM

The rate and rhythm of the heart are sensitive to many influences. The mechanism is an alteration in the firing frequency of one or more automatic cells. This alteration is accomplished by changing the slope of decay, changing the resting potential, changing the threshold, or some combination. The following influences have been studied in isolated preparations. Expansion to a clinical situation is not feasible at this time (39,40).

Norepinephrine increases the slope of decay and therefore increases the rate. Its effect is essentially the same on all automatic cells.

Acetylcholine decreases the slope of decay and therefore decreases the rate. Its effect is selective for the SA node. The selectivity is both intrinsic and a function of distance from the autonomic terminals.

Decreased extracellular potassium increases the slope of decay. Increased extracellular potassium decreases the slope of decay but also elevates the resting potential. The Purkinje system is more sensitive than the SA node.

Increased calcium shifts the threshold away from the resting potential and vice versa. The SA node is more sensitive than the Purkinje system.

Hypoxia and increased CO<sub>2</sub> pressure both increase the slope of decay.

Excessive stretch or trauma both increase the slope of decay.

Electrical current if depolarizing increases the slope of decay, if hyperpolarizing it decreases it.

#### MECHANISMS OF ATRIAL FIBRILLATION

Atrial fibrillation occurs when the rate of stimulation exceeds the rate at which the atria can respond. This results in the absence of regular or coordinated atrial contraction and in irregular atrial conduction. The rapid irregular stimuli to the AV node occur during various states in nodal depolarization and repolarization resulting in irregular nodal transmission and therefore irregular ventricular contraction even though the ventricular conducting system functions normally.

There are three theories of the mechanism of atrial fibrillation: (1) the unifocal theory of Rothberger, (2) the circus theory of Lewis, and (3) the decremental conduction theory of Hoffman.

According to the unifocal theory, the rate of stimulation from an ectopic focus exceeds the rate at which the atria can respond resulting in the chaotic rhythm (41).

The circus theory states that the wave of excitation follows an irregular path around the vena cava. This path is roughly circular

and is perpetuated by self reexcitation. The wave gives off daughter waves that rapidly but irregularly stimulate the atria (41).

Decremental conduction is defined as "a type of conduction in which the properties of the fiber change along its length in such a manner that the action potential becomes progressively less effective as a stimulus to the unexcited portion of the fiber ahead of it." (4). Hoffman states that the factors that cause decremental conduction also cause progressively slower conduction. He believes this conduction slow enough to effectively remove all requirements for a minimum path length in reentry (39).

#### DISCUSSION OF ATRIAL FIBRILLATION

Atrial fibrillation is a common arrhythmia and has been studied extensively. It can occur as either self-limiting paroxysmal attacks or as a sustained arrhythmia (1). The sustained arrhythmia is usually associated with underlying organic heart disease. Nohara (2) in his autopsy study found atrial fibrillation in half the patients dying of rheumatic heart disease, one-fourth of the patients dying of hypertensive vascular disease and one-fifth dying of coronary artery disease. Atrial fibrillation can cause congestive heart failure in some people without underlying heart disease (3). It is possible, though rare, for patients to have it for years without any symptoms (4). Averill and Lamb (5) found only five of 67,375 asymptomatic healthy men with either flutter or fibrillation.

Studies on the hemodynamics, particularly the cardiac output, have been done for the last 40 years. These studies have had widely varying

results. The early studies, before 1962, used quinidine conversion and differed greatly in experimental model and patient selection as well as results (See Table I) Some of the criticisms of these studies are: (1) Weeks and frequently months separated the prereversion and postreversion studies, (2) drug dosages were changed and new medications added during the interval, and (3) little attempt was made to do the followup studies at similar ventricular rates or oxygen consumptions. Hecht (8) could only conclude that the converters fell into three categories: (1) those that did not improve significantly following conversion, (2) those that improved significantly both at rest and at exercise, and (3) those that did not improve at rest but did improve during exercise. Tables I and II were drawn up with this in mind. Rather than report the average percent increase in the cardiac output in all patients that converted to a sinus rhythm, it seems much more practical to report what percent of the converters had a significant increase (0.5 l/m or more) and the percent increase in this group.

Table I shows that about 70 percent of the patients that converted to a sinus rhythm with quinidine had an increase in cardiac output of 0.5 l/m or greater and that the average percent increase in these patients was about 40 percent.

The development of synchronized electrical cardioversion in 1962 (44) made it possible to complete the pre and postreversion within minutes and under similar conditions of ventricular rate or oxygen consumption.

Table II gives the summaries of many of the electrical conversion studies. Though these studies are much more standardized, there are

still appreciable differences in the results. The averages from these studies show that about 55 percent of the electrical converters will have an increase in cardiac output of 0.5 l/m or greater and that the average percent increase of this group is about 35 percent at rest and about 25 percent during exercise.

The heterogeneity of these studies is less confusing if one considers that in a given patient the state of the circulation is the result of; (1) the effects of the arrhythmia, (2) the degree of AV block, (3) the amount of the underlying heart disease, and (4) individual variances in homeostatic mechanisms.

#### Effects of the Arrhythmia:

The pathology of atrial fibrillation is the absence of coordinated or regular atrial contractions and the impulse transmission to the ventricles is unpredictable in timing and intensity. From this, it seems logical that a large contribution to the improved cardiac output with restoration to a sinus rhythm is the addition of the atrial pump.

Staub (23) in 1906 showed with a soap film cardiometer that atrial contraction caused a distinct increase in ventricular diastolic volume. Hirschfelder (2 ) in 1908 studied the effect of atrial contractions in experimentally produced mitral stenosis. He found that progressive constriction of the mitral valve yielded a progressive decrease in the early diastolic filling but an increase in the atrial contribution to the end diastolic volume of 10 percent to 25 percent. This contribution stopped abruptly with the onset of atrial fibrillation.

Skinner (25) showed that in dogs the decrease in cardiac output has multiple causes. When the ventricular rate was constant the stroke volume and, therefore, the cardiac output decreased with the introduction of atrial fibrillation. By increasing the ventricular rate stepwise to 180 bpm, there was both an inordinate decrease in stroke volume and an increase in left atrial pressure. Finally, as the ventricular rate increased, he found the development of mitral insufficiency during the isovolumetric contraction period of the left ventricle. Daley (34) had shown the mitral incompetence earlier by injecting dye into the left ventricle and withdrawing it from the fibrillating left atrium. Sarnoff and Skinner (35) using a hydrogen electrode showed a similar reflux of ascorbic acid into the left atrium and has even defined the optimal AV interval in dogs and found that regurgitation occurs outside the range of 60 to 120 milliseconds.

Corliss (21) and others (19,22) have found a decrease in mitral regurgitation with restoration of a sinus rhythm in some but not all of the cases in their patient studies.

Degree of AV Block:

The unpredictable impulse transmission found in atrial fibrillation results in a marked irregularity of ventricular contractions. The degree of AV block will determine the rate of ventricular response. The irregular atrial rate is in the range of 400 to 800 bpm. An uncontrolled ventricular response may be around 140-160 bpm but with treatment can usually be maintained in the 60-100 bpm range. The irregular timing of ventricular contractions give a marked beat to beat



variation in arterial systolic pressure, pulse pressure and duration of ventricular systole. Wiggers (28) with his animal studies and Dodge (29) on patients illustrated the importance of the duration of the preceding R-R interval on this variation. They also showed that there is an inverse relationship between any given pulse pressure and the preceding pulse pressure.

The above aspects become clearer by considering them separate. If the R-R interval is constant then (systole + diastole = 1) and they are, therefore, inversely proportional to each other. With a large pulse pressure and stroke volume the time required to complete the systolic ejection will take up a larger portion of the R-R interval, less time will be available for diastolic filling resulting in a smaller end diastolic volume. The second systole will require less time to complete the ejection of the smaller volume giving a longer period of time to the second diastole. The second diastole will present the larger end diastolic volume to the third systole.

If the R-R interval is not constant then the limiting factor on the diastolic filling time will often be the following systole rather than the preceding. If the R-R time is shortened then the filling time will be decreased and the following stroke volume will be small. If the R-R time is elongated there will be more time for ventricular filling and the following stroke volume, systolic arterial pressure and pulse pressure will be increased.

Combining the two factors gives us a picture of the chaotic nature of ventricular contractions, the occurrence of frustrate

ventricular beats unable to generate sufficient force to eject blood into the aorta and some normal appearing beats. In the absence of effective atrial contractions in a given patient in a steady state the above factors can be responsible for considerable variations in the end diastolic volume (27).

Underlying Heart Disease:

The amount of underlying heart disease in atrial fibrillation becomes extremely important in those patients with increased left atrial pressure, especially those with mitral stenosis. In mitral stenosis, there is a decrease in the early diastolic filling but in sinus rhythm the atrial contribution to the end diastolic increases to compensate (24). With atrial fibrillation, there is no atrial component and the end diastolic volume is completely dependent on the diastolic time available. Atrial fibrillation, therefore, presents a double insult to the circulation by increasing the ventricular rate and by stopping the atrial contraction.

Atrial contraction is important in aortic stenosis for different reasons. The pressure gradient caused by the aortic stenosis causes a secondary hypertrophy if the left ventricular muscle in an attempt to compensate for the added work necessary to overcome this gradient. The hypertrophied muscle mass infringes on the left ventricle cavity resulting in a smaller volume. Starlings law says the force of contraction is directly proportional to the fiber length (42). The atrial pump significantly increases the end diastolic volume and therefore fiber length. This results in a greatly increased force of contraction assisting in overcoming the pressure gradient across the aortic valve.

By contrast the 12 patients on Table II that had no organic heart disease ("benign fibrillators") had essentially no improvement in cardiac output with conversion.

The hemodynamic effects of atrial fibrillation can be greatly altered by emboli, either systemic or pulmonic. This is a well known complication of atrial fibrillation especially in rheumatic hearts (30) and is thought to be a combination of stasis caused by ineffective atrial contractions and rheumatic changes in the atria.

Homeostatic Mechanisms:

Neuroregulatory controls of the peripheral circulation also may alter the expected hemodynamic changes. Lewis (31) and Skinner (25) both showed that animals with intact baroreceptors had an acute drop in arterial pressure and flow but these returned toward normal as the fibrillation continued. By sectioning the vagi and controlling carotid sinus pressure, they were able to decrease or prevent this return toward normal. Nakano (32) studying reserpinized dogs during atrial tachycardia found a similar picture. The unreserpinized dogs acutely decreased their mean arterial pressure, cardiac output and myocardial contractile force. After one to two minutes, these returned toward, but did not reach, control. The myocardial contractile force stabilized above control. Atrial pressures rose and remained high. On termination of the tachycardia the arterial pressure and cardiac output increased markedly above control. Within two minutes all parameters returned to control.

In the reserpinized dogs, arterial pressure, cardiac output and myocardial contractile force all fell abruptly then continued to fall

slowly. After termination, they very slowly returned to control without showing an overshoot phase.

Corday and Irving (33) have studied more distal homeostatic mechanism during arrhythmia. They used flow meters and an integrater-computer analyzing renal and mesenteric blood flow. During atrial fibrillation, they found an average drop in renal blood flow of 20 percent (range 9-60 percent) and a 34 percent decrease in mesenteric blood flow (range 9-50 percent). The renal flow stabilized and remained constant for several hours after termination. The mesenteric flow returned to near normal values rapidly and then became very labile. Since roughly 25 percent of the cardiac output is used to perfuse the kidneys and another 15 percent goes to the mesentery, this makes relatively large amounts of blood available for the more critical coronary and cerebral circulation.

#### PRINCIPLES OF MANAGEMENT OF ATRIAL FIBRILLATION

Synchronized electrical defibrillation has presented us with a safe and very successful method of reverting the rhythm in patients with atrial fibrillation. Initial successful conversions are accomplished in 90-95 percent of the cases (41,42). With adequate prophylactic therapy about 80 percent will remain in sinus rhythm for one month and about 50 percent will remain for longer than four months. In the absence of prophylaxis only 10-20 percent will remain in sinus rhythm.

When the diagnosis of atrial fibrillation is made in the absence of organic heart disease, a careful search should be made for the

cause. Such things as thyrotoxicosis, undiagnosed mitral stenosis, acute rheumatic fever, and systemic infection should be investigated.

Digitalis in doses sufficient to maintain the heart rate between 60 and 80 bpm is indicated. When the ventricular rate is 60 or less in the absence of digitalis it probably should not be given in the absence of signs of cardiac decompensation (41).

Quinidine 0.2mg every six hours should be given for two to four days prior to conversion and prophylactically following conversion. Quinidine is potentially a quite toxic drug but most people can tolerate this dose with little trouble. McIntosh (27) found no hemodynamic evidence of cardiac toxicity in 13 patients studied before and after quinidine was given in doses to give blood levels of 3 to 5 mg/100 cc. Sokolow (45) found no toxic effects in levels less than 3 mg and only 1.6 percent in levels less than 6 mg/100 cc. Corday (41) reports that 10-15 percent of the patients will revert to sinus rhythm after digitalis and the maintenance quinidine have been given.

Anticoagulation is indicated in all patients with a history of embolization. Many groups use short term anticoagulation in all reversion and long term in anyone with an embolic history.

Thind (42) has stressed the importance of correction of electrolyte imbalance prior to reversion. He found postconversion arrhythmia in 100 percent of a group with hypokalemia but in only 11 percent of a group with normal electrolytes.

The decision to convert patients with atrial fibrillation should always be individualized. The indications for conversion are: (1) recent onset of atrial fibrillation in mild heart disease, (2) young

or middle-aged patients without known heart disease, (3) persistence of atrial fibrillation after successful treatment of hyperthyroidism or successful surgery for mitral stenosis, (4) chronic congestive failure not well controlled by drug therapy, (5) history of frequent embolic phenomena, (6) rapid ventricular response not controlled by digitalis, and (7) when palpitation is intolerable (43).

Absolute contraindications to conversion include: (1) complete heart block in the presence of atrial fibrillation, (2) history of previous reversions with relapse despite adequate prophylactic therapy, (3) hypersensitivity to prophylactic agents, (4) history of previous reversion with a worsening clinical condition, (5) atrial fibrillation resulting from digitalis toxicity, and (6) prior to mitral valve surgery (43).

#### SUMMARY

The anatomy, physiology and pathology pertinent to atrial fibrillation have been discussed. Material has been presented to show that atrial fibrillation causes circulatory abnormalities in the majority of patients. Factors that influence the variability of these abnormalities have been discussed. Finally the principles of management of atrial fibrillation have been briefly discussed.

Table I. Summaries of Studies With Quinidine Cardioversion

Year	Author	Type Heart Disease	Time After Conversion	State	No in Study	No with Increased Cardiac Output*	Ave % of Increase**
1963	Gilbert (12)	Mixed	Unknown	rest	3	2	54%
1957	Broch (11)	Mixed	Unknown	rest	17	?	21%
1952	Hansen (10)	Mixed	1-4 weeks	est	14	9	47%
		Mixed	1-4 weeks	Exercise	12	11	43%
1951	Kory (9)	Mixed	Unknown	rest	8	6	43%
1936	Kerkhoff (7)	Mitral Stenosis	Unknown	rest	8	6	28%
1930	Smith (6)	Mixed	1-2 months	rest	3	2	30%
	Average***	Mixed	1-8 weeks	rest	36	25 (72%)	41%

\* Significant increase = 0.5 l/m or greater

\*\* Percent increase of those with significant increase

\*\*\* Study of Broch not included

Table II. Summary of Studies With Electrical Cardioversion

Year	Author	Type Heart Disease	Time After Conversion	State	No. in Study	No. with Increased Cardiac Output*	Ave. % of Increase	Overall Ave. Increase**
1968	Shapiro (23)	Mixed	1-2 hours	Rest	11	5	35%	
			1-2 hours	Exercise	5	3	23%	
1968	Corliss (21)+	Mixed	Immediate	Rest	16	6	33%	
1966	Killip (19)	Mitral	24 hours	Rest	10	?	?	32%
			24 hours	Exercise	10	?	?	22%
		Aortic Benign	24 hours	Rest	4	?	?	37%
			24 hours	Rest	6	?	?	7%++
			24 hours	Exercise	6	?	?	7%
1965	Reale (18)	Rheumatic	30 minutes	Rest	12	8	27%	
1964	Graettinger(17)	Rheumatic	1-2 hours	Rest	11	4	27%	
			1-2 hours	Exercise	10	3	23%	
		Mixed	1-2 hours	Rest	6	2	53%	
			1-2 hours	Exercise	4	2	23%	
1964	McIntosh (16)	Mixed	3hr-6 days	Rest	6	4	19%	
			3hr-6 days	Exercise	5	4	41%	
1964	Morris (15)	Mixed	3 hrs	Rest	11	7	34%	
			3 hrs	Exercise	5	5	17%	

(continued)



Table II. (Continued)

Year	Author	Type Heart Disease	Time After Conversion	State	No. in Study	No. with Increased Cardiac Output*	Ave. % of Increase	Overall Ave. Increase**
1964	Killip (14)	Rheumatic	24 hours	Rest	12	12	34%	
			24 hours	Exercise	12	12	17%	
		Benign	24 hours	Rest	6	1	10%	
			24 hours	Exercise	6	0	0	
1963	Oram (13)	Mixed	3-16 days	Rest	10	7	70%	
	Average***	Mixed	0-16 days	Rest	101	56 (55%)	35%	
			1hr-6 days	Exercise	47	29 (62%)	26%	

\* Significant increase = 0.5 l/m or greater

\*\* Only given if figures not available for previous column

\*\*\* 1966 study by Killip not included

+ No anesthesia given

++ Six patients that did not convert also had a 7% rise in cardiac output

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