

Ventricular Rate Control for Atrial Fibrillation – When and How?

Dorian, Paul

Introduction:

Many of the symptoms that patients suffer during atrial fibrillation are caused directly or indirectly by a rapid and irregular ventricular rate. These include the subjective discomfort and unpleasantness of the sensation of rapid heart action (variously described as an unpleasant awareness of heart action – “palpitations”; fluttering or butterflies in the chest or abdomen; or symptoms caused by tachycardia but without patient awareness of rapid rates e.g. shortness of breath, weakness, nonspecific chest discomfort, etc.).

Hemodynamic consequences of rapid ventricular rates which cause symptoms include a fall in cardiac output with or without a drop in blood pressure, leading to weakness, fatiguability, lightheadedness, and occasionally presyncope or loss of consciousness (syncope). Because the diastolic period during atrial fibrillation is short, and because many patients with atrial fibrillation also have pre-existing left ventricular diastolic dysfunction, most commonly from hypertension or valvular disease, there is insufficient time for appropriate ventricular filling during short diastoles. This leads to an elevation in left atrial pressure, transmitted to the pulmonary venous circuit, and leading to dyspnea, chest tightness, and occasionally cough. Elevations in left atrial pressure can also cause release of atrial natriuretic peptide (ANP) which can cause polyuria.

Irregularly irregular ventricular rates can also lead to beat-to-beat changes in left ventricular filling, and thus beat-to-beat changes in aortic pressure, as a consequence of variable ventricular loading conditions and Frank-Starling forces. Beat-to-beat changes in aortic pressure cause beat-to-beat changes in aortic arch baroreceptor function, which leads to variable parasympathetic and sympathetic mediated reflex feedback to the heart and blood vessels, potentially also responsible for the surges, drops, and variabilities in blood pressure and vagal tone which can be subjectively very unpleasant.

It should be noted that the above abnormalities are independent of any drop in cardiac output that may be the result of the loss of atrioventricular synchrony which of course accompanies atrial fibrillation.

Although there are no large scale controlled clinical trials which have compared patient well-being and clinical outcomes in the presence of and the absence of any form of ventricular rate control, there is of course incontrovertible clinical evidence as well as short term studies which verify that administering drugs to slow ventricular response during atrial fibrillation in symptomatic patients, yields patient benefit.

There are however a number of questions which need answering with respect to the strategies to be used for rate control:

First, when is therapy to control ventricular response necessary – is it in all patients; or in some patients? If rate control is advisable, is this to primarily treat symptoms related to rapid rate, or to slow the ventricular response, or both? (the two are not necessarily synonymous)

If rate control is advised, what treatments should be used, in what order, and how should their effect be measured?

When is rate control necessary?

There is universal agreement that when a patient has atrial fibrillation and a rapid ventricular response accompanied by symptoms likely caused by tachycardia, then rate control is advisable. Usually, such patients will have ventricular rates of greater than 90 minutes at rest, and greater than 110 or so beats/minute with mild or moderate activity. If a patient has ventricular rates slower than this value, and there are not unequivocal symptoms related to tachycardia, the decision to administer rate control is more difficult. For example, in a patient with a resting ventricular rate of 75 beats/minute (in the absence of any rate controlling medications), as well as fatigue, and a heart rate of 100 beats/minute with moderate exercise, rate control is not necessarily advisable. Such patients usually have underlying AV nodal disease. In particular, patients who have a relatively normal profile of heart rate in the absence of rate controlling drugs, rate controlling medications are generally not advisable as symptomatic bradycardia can ensue. When symptoms are exclusively described as weakness or dyspnea as opposed to palpitations, but diastolic dysfunction is present or there is mitral stenosis, the decision may be somewhat more difficult. In such patients it may be advisable to attempt a careful therapeutic trial of a rate control medication to maximize diastolic filling time, but it should be noted that such patients may improve, not have benefit, or may even get worse with rate control therapies.

The other difficult situation is that management of patients with a rapid ventricular response but no symptoms. For example, some patients can have ventricular rates of 100/minute or faster at rest, and up to 130-150 beats/minute or faster with mild activity. If such patients are completely asymptomatic, there is no good evidence that rate control is necessarily required. Of course, quality of life will not (in the short term at least) be improved by rate control in these already asymptomatic patients. There is the theoretical risk of developing so-called “tachycardiomyopathy” if tachycardia persists for very long periods e.g. months, but this is only a conjecture. It would seem reasonable to add a rate control agent if there is LV dysfunction, or if the heart rates are dramatically greater than normal for example an average 24 hour heart rate on holter monitoring of more than 100 to 120 beats/minute. Episodic tachycardia in an asymptomatic patient e.g. heart rates averaging 80-100 per minute, but surging to 150 or even more beats/minute with exercise is not necessarily an indication for adding rate control agents. Exceptions would include patients with angina pectoris, known severe coronary disease, or extensive valvular disease or pulmonary hypertension.

If rate control agents are used, what drugs should be used and how should their effect be measured?

Digoxin is the drug most frequently used historically for rate control. Unfortunately, there is very little evidence that digoxin by itself is highly effective at rate control, and tends to slow resting heart rates (when symptoms are generally mild or absent) much better than exercise heart rates (when symptoms are generally more severe). For this reason, digoxin is not generally recommended as a “first line” rate control agent except perhaps in older individuals who have mild to moderate symptoms related to rapid heart rates, and who are not likely to be extremely physically active. It should be noted that digitalis frequently leads to nighttime slowing of ventricular rates, occasionally with long nocturnal pauses.

Most guidelines and clinicians treating atrial fibrillation patients use calcium channel blockers e.g. diltiazem or verapamil, or beta blockers as drugs of first choice for ventricular rate control. There is no clear evidence to prefer one group of the drugs over another, although in patients with coronary disease or heart failure, beta blockers are clearly preferred. Multiple small but randomized and controlled studies have documented that calcium channel blockers are associated with better exercise tolerance than beta blockers in patients with atrial fibrillation, although they may be less effective at controlling the ventricular response. Particularly in younger patients who are more active, if they have normal left ventricular function, calcium channel blockers may be preferable to

beta blockers. In patients with a pre-existing indication for beta blockers, these latter drugs are preferred.

There are no easily applied “universal” targets for rate control in atrial fibrillation. Aiming for resting heart rates of less than 90 beats/minute and activity (for example during a brisk walk) heart rates of less than 110 beats/minute seem reasonable. Drug doses should be titrated to patient well-being, taking into regard the heart rate at rest and with activity (most easily measured using holter monitoring), and potential adverse effects from treatment. It is generally wiser to begin with lower doses and titrate upwards. Substantial anecdotal evidence suggests that the addition of a small dose of digitalis to calcium blockers or beta blockers has added benefit without substantial risk and should be preferred over the strategy of using large doses of a single agent. In rare cases, combinations of beta blockers and calcium blockers are required to control ventricular response.

If patients have persisting very rapid ventricular rates with symptoms despite the use of calcium channel blockers and beta blockers and digoxin in addition, underlying disorders which shorten AV nodal refractory periods should be considered, for example thyrotoxicosis, sepsis, or cardiomyopathy with elevated sympathetic tone and heart failure.

If ventricular rate control cannot be achieved by using combinations of AV nodal blocking drugs, AV node ablation and pacemaker implantation is sometimes considered. This however involves “trading one disease for another”, i.e. treating atrial fibrillation but resulting in surgical AV block and pacemaker dependency. Another concern is a potential long term risk of developing left ventricular failure with chronic right ventricular pacing. For these reasons, AV node ablation is less and less resorted to in most tertiary care electrophysiology practices.

Summary:

It needs to be remembered that the primary goal of treatment in atrial fibrillation is patient well-being and quality of life. Practitioners should be very wary of “treating the ECG” rather than treating the patient.

References:

Dorian P, Connors S. Canadian Journal of Cardiology 2005;21(Suppl B):26B-30B.