Atrial fibrillation: the most common form of arrhythmia

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Interpreting ECGs
In our continuing series on ECGs, Mel Humphreys reviews the disorder of atrial fibrillation.

BACKGROUND

Atrial fibrillation is the most common form of arrhythmia, affecting more than two million people annually. Its prevalence increases with age, and is found in more than 10% of patients aged 75 years or older, and affects men slightly more often than women (Lake and Thompson, 1991; Palazzo, 1999). Atrial fibrillation can occur in a wide variety of clinical settings. Although it is more likely to occur in patients with underlying heart disease, it can also occur in patients without any demonstrable cardiac disease (Thompson, 1997).

It is most often precipitated by left atrial enlargement (e.g. underlying mitral stenosis or regurgitation) or occurs in other pathological states, including pulmonary disease and thyrotoxicosis (Lilly, 1993). In the case of coronary artery disease, it occurs in 10–15% of patients following myocardial infarction and is associated with an adverse prognosis with increased rates of in-hospital death and stroke (Connaughton, 2001).

Classification

According to Sopher and Camm (1996), atrial fibrillation can be classified as acute or chronic. Acute is the first onset of atrial fibrillation and lasts less than 48 hours. Chronic is atrial fibrillation that recurs after the first onset has been terminated; it can be subclassified as paroxysmal (intermittent), persistent or permanent.

Since atrial fibrillation is an unpredictable condition, it can completely disrupt the patient's life, causing significant emotional and physical distress. This condition is therefore a major clinical challenge to the nurse working within an acute care area, with the management focus being individualised and based upon the degree of disability and symptoms associated with the disorder (Palazzo, 1999).

Aetiology

In the broadest sense, atrial fibrillation represents the loss of synchrony between the atria and the ventricles. A definitive cause has yet to be uncovered, but suggested causes include:

- increased atrial stretch (and/or atrial enlargement)
- increased circulating catecholamines
- metabolic or electrolyte disturbances
- atrial premature beats (Thompson, 1997; Smith and Kampine, 1990; Palazzo, 1999).

ECG IDENTIFICATION

Whatever the cause, however, atrial fibrillation can be characterised as multiple independent wandering wavelets of different size and travelling in different directions, which become fractionated as they divide around islets or strands of refractory tissue (Allessie *et al.*, 1996; Waldo, 1993). Indeed, the re-entering impulse proceeds around multiple circuits, which causes the atria to quiver (fibrillate) at 350–600 times per minute. As a result, discrete atrial P waves are not discernible. This continuous activation is represented by *fibrillatory waves* (f waves) on the electrocardiographic (ECG) tracing (Box 1).

f waves

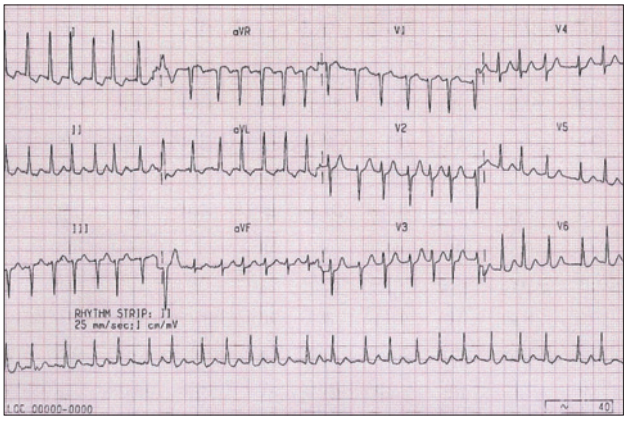
Fibrillation varies from coarse to fine. In coarse fibrillation, prominent f waves are clearly visible in many leads, but in fine fibrillation there may be no visible atrial activity at all (Wagner, 1994). In this instance the best leads to observe are II and V_1 as these give greater clarity of the atrial waveforms. Although the size of the f waves has not been found to correlate with the size of the atria or the type of heart disease, large f waves are unlikely to occur in the presence of a normal-sized left atrium.

Role of AV node

When atrial fibrillation is present, the innumerable f waves compete for penetration of the atrioventricular (AV) node, making it difficult for any impulse to reach the common bundle. The ventricles respond only to those impulses that make it through the AV node. The AV node blocks some of the 350-600 erratic atrial impulses per minute from reaching the ventricles. However, the AV node itself does not receive all the impulses. If myocardial tissue surrounding the AV node is in a refractory state, impulses from other areas of the atria cannot reach the AV node. This further reduces the number of atrial impulses conducted through to the ventricles (Ambrose *et al.*, 1997). These two factors help to explain the characteristic, wide variation in R-R intervals in atrial fibrillation.

BOX 1

Undertake a systematic analysis of the following 12-lead ECG



COMMENTARY

The features shown on this ECG indicate atrial fibrillation:

- Heart rate: mean ventricular rate 150 bpm (This is calculated by counting 15 QRS complexes in a 6-second period, then multiplying by 10.)
- Rhythm: irregular with no apparent pattern to its irregularity
- P waves: no discernible P waves, baseline undulating, demonstrating fine fibrillatory waves of low amplitude (best seen in Lead II)
- PR interval: not apparent
- QRS: normal, but occurs at irregular intervals.

Most of the atrial impulses are blocked, but those that do pass through are conducted normally through the ventricles. Thus, atrial fibrillation is manifested electrocardiographically, on this occasion, by the absence of P waves and the presence of normal QRS complexes that occur at irregular intervals.

Ventricular rhythm

Consequently, fibrillation always produces an irregularly irregular, ventricular rhythm – this means that there appears to be no pattern to the irregular ventricular rhythm. Indeed, when atrial fibrillation is accompanied by a regular ventricular rate, there is dissociation between the atrial and ventricular rhythms (as in the case of excessive AV block). Since the ability of the AV node to conduct the atrial impulses may vary dramatically, the ventricular rate may also vary. At times, it may change abruptly from rapid to slow, incorrectly suggesting a change in the basic underlying rhythm (Wagner, 1994). When the ventricular response rate is below 100, the rhythm is considered controlled and of relative low risk to the patient. When it exceeds 100 bpm, the rhythm is considered uncontrolled and poses an intermediate risk. Rates above 150 bpm pose a high risk of patient instability (Resuscitation Council, 2001).

Effect on patient's lifestyle

Given its unpredictable nature, atrial fibrillation can completely disrupt the patient's life, causing significant psychological morbidity, arising from justified or unjustified concerns regarding implications for long-term health. In many patients, activity is severely restricted by fear of provoking or suffering from an atrial fibrillation episode. Patients often feel insecure because of their inability to control or even predict attacks (an 'external locus of control') (Waktare and Camm, 2000).

PHYSIOLOGY

The major dangers of atrial fibrillation are concerned with haemodynamic stability and the risk of thromboembolism. With regards to haemodynamic stability, there is a reduction in the pumping efficiency of the heart (decreased cardiac output). The potential rapid ventricular rates may compromise stroke volume, resulting in hypotension and pulmonary congestion, particularly in individuals with hypertrophied or 'stiff' left ventricles, in whom organised atrial contraction contributes significantly to left ventricular filling and cardiac output (Lilly, 1993; Dracup, 1995).

It is well established that about 70% of blood flows passively from the atria into the ventricles, with contraction of the atria forcing the remaining blood into the ventricles. The final push accounts for about 30% of the blood passed into the ventricles (Tortora and Anagnostakos, 2000). The resultant haemodynamic consequences only serve to compromise the patient's haemodynamic stability further, and may lead to left ventricular failure and worsen existing myocardial ischaemia (Humphreys, 2001).

Adverse signs include:

- systolic blood pressure (BP) of less than 90 mmHg
- palpitations
- chest pain
- breathlessness
- syncope
- poor peripheral perfusion (Resuscitation Council, 2001; Campbell, 2000).

When caring for a patient with atrial fibrillation, it is not uncommon to find that the radial pulse rate is slower than the apical rate. This is because the weaker contractions of the heart do not produce a palpable peripheral pulse; only the stronger ones do (Ambrose, 1997). For this reason, it is essential to monitor the patient's heart rate, initially in the acute admission period using a cardiac monitor, so that an accurate measure of heart rate can be obtained.

Risk of stroke and its prevention

The loss of efficient atrial contraction predisposes to stasis of blood in the atria; this encourages the formation of thrombi, which in turn pose the threat of systemic embolisation (Prosser *et al.*, 2000). Clot formation in the left atrium can become a primary source of stroke in patients with atrial fibrillation. One type of stroke called thromboembolic cerebral vascular accident (CVA) occurs approximately six times more often in the elderly, while atrial fibrillation is found in about 15% of all stroke patients (Sherman *et al.*, 1986). Since atrial fibrillation is the most common arrhythmia and is becoming increasing prevalent in the elderly, it therefore poses a substantial and potentially devastating risk (Palazzo, 1999).

Individuals who remain in chronic, persistent or permanent atrial fibrillation are therefore at increased risk of stroke and should be considered for anticoagulant therapy (Lilly, 1993). Evidence suggests that anticoagulation with warfarin (Coumadin) is effective in reducing the risk of thromboembolism, but does not totally eliminate the risk. The therapeutic range of the INR (International Normalised Ratio) is usually between 2.0 and 3.0

for the prevention of stroke. Anticoagulation therapy confers a risk in itself and any treatment regime should be individual and flexible in its approach (Sopher and Camm, 1996).

MANAGEMENT

The treatment of atrial fibrillation is highly complex and requires considerable skill and judgement. Generally, the appropriate treatment for atrial fibrillation is determined by the patient's relative risk from arrhythmia in terms of haemodynamic stability (Resuscitation Council, 2001). Further considerations include the classification of the atrial fibrillation, i.e. acute or chronic (paroxysmal, persistent or permanent), and the current best evidence concerning long-term health prognosis within that classification framework (Waktare and Camm, 2000). However, it is beyond the scope of this article to discuss these treatment options in great detail.

Terminating or 'breaking' the fibrillation may be accomplished using either drugs or electrical stimulation. A drug is capable of suddenly terminating the re-entrant tachyarrhythmia either by increasing the speed of the recycling impulse so that it encounters only cells that are still refractory (e.g. amiodarone) or by prolonging the refractory periods of the cells (e.g. soltalol) (Prosser *et al*., 2000). External electrical stimulation (cardioversion) is capable of suddenly breaking the fibrillation by depolarising all cardiac cells that are not already in their depolarised state. The external stimulus eliminates the receptivity to reentry that is required to maintain the tachyarrhythmia (Wagner, 1994).

However, if there is significant haemodynamic compromise as a direct consequence of rapid atrial fibrillation, the arrhythmia should be terminated without delay and normal sinus rhythm restored by means of cardioversion (Thompson, 1997; Connaughton, 2001). If possible, heparinisation should be administered first because a successful cardioversion to normal sinus rhythm will cause forceful atrial contractions to resume abruptly. If a thrombus has formed in the atria, the resumption of contractions can result in systemic emboli with potentially disastrous results. Anticoagulation is also necessary after elective cardioversion of chronic atrial fibrillation to avoid thrombus formation in stunned atria, following successful cardioversion, as their mechanical function may take 4–6 weeks to return (Sopher and Camm, 1996).

Patients cardioverted from recurrent paroxysmal or from chronic persistent atrial fibrillation have a very low likelihood of remaining in sinus rhythm without active drug therapy. In controlled trials, it has been shown that only 20–40% of patients remain in sinus rhythm after 12 months following successful cardioversion (Hohnloser and Li, 1997).

With regards to medical treatment, the three major goals are the restoration of normal sinus rhythm, maintenance of sinus rhythm, control of the ventricular rate during atrial fibrillation, and prevention of blood clot formation (Sopher and Camm, 1996). Other current treatment options include ablations, pacemakers, atrial defibrillators and surgery (Palazzo, 1999; Murgatroyd and Camm, 1995) (Box 2).

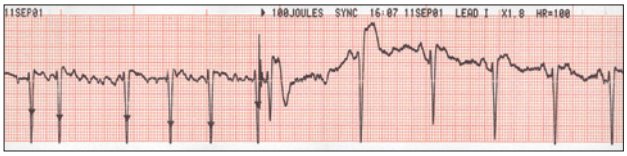
SUMMARY

Recognition and appropriate management of atrial fibrillation is a major challenge for the critical care nurse. Atrial fibrillation may cause symptoms of palpitation, angina and syncope, and often causes concern because of haemodynamic dysfunction. This presentation may necessitate immediate intervention (Sopher and Camm, 1996). Given its unpredictable nature, atrial fibrillation can completely disrupt the patient's life, causing significant psychological morbidity, and patients often feel insecure because of their inability to control or even predict attacks (Waktare and Camm, 2000).

Early recognition is vital. It is a common dysrhythmia affecting 2–5% of the population over the age of 60 (Kannel *et al*., 1982). It is also a common cause of thromboembolic events, including disabling stroke, and an occasional cause of death. A greater understanding of the physiological processes involved and management options can only serve to enhance individualised care delivery within the acute care setting.

BOX 2

Consider the following single-lead ECG recording



COMMENTARY

The features of atrial fibrillation cardioverted to sinus rhythm are shown here:

- The initial five QRS complexes are of normal duration, but irregularly regular
- P wave: absent with coarse fibrillatory waves obvious
- Synchronisation evident on first five QRS complexes and sensing appropriately
- Sixth QRS complex shows delivery of 100 J defibrillatory shock resulting in sinus rhythm i.e. P-QRS-T, rate 75 bpm.

Synchronised cardioversion is similar to defibrillation, except that cardioversion generally requires lower energy levels; a starting level of 100 J would be appropriate (Resuscitation Council, 2001). However, this does vary, and the patient would have been sedated. It is important to establish that the synchronisation is apparent on the R wave, since stimulation that hits a T wave increases the risk of fatal arrhythmias. Once the firing buttons have been pressed, the cardioverter discharges energy when it senses the next R wave.

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REFERENCES

Allessie MA, Konings K, Kirchhoff C, Wijffels M. (1996). Electrophysiologic mechanism of perpetuation of atrial fibrillation. *American Journal of Cardiology*; 77: 10a–25a.

Ambrose Ml, Mauro E, Nash J, Sinovic D. (1997). *ECG Interpretation made Incredibly Easy!* Pennsylvania: Springhouse.

Campbell RW. (2000). How do we achieve optimal cardiovascular function in atrial fibrillation? In: *Royal College of Physicians of Edinburgh Proceedings: Conference on Atrial Fibrillation in Hospital and General Practice.* July 1999. Edinburgh: Royal College of Physicians of Edinburgh Press; 29: 16–18.

Connaughton M. (2001). *Evidence-Based Coronary Care.* London: Churchill Livingstone.

Dracup K. (1995). *Meltzer's Intensive Coronary Care: A Manual for Nurses.5th ed.* California: Prentice-Hall International.

Hohnloser SH, Li YG. (1997). Drug treatment of atrial fibrillation: what have we learned? *Current Opinion in Cardiology*; **12:** 24–32.

Humphreys M. (2001). Patients with inferior MI: right ventricular involvement. *Connect*; **1:** 46–47.

Kannel WB, Abbott RD, Savage DD, Mcnamara PM. (1982). Epidemiologic features of chronic atrial fibrillation: the Framingham study. *New England Journal of Medicine*; **306:** 1018–1122.

Lake FR, Thompson PL. (1991). Prevention of embolic complications in non-valvular atrial fibrillation in the elderly. *Drugs and Aging*; **1:** 458–466.

Lilly LS. (1993). *Pathophysiology of Heart Disease: A Collaborative Project of Medical Students and Faculty.* London: Lea & Febiger.

Murgatroyd FD, Camm AJ. (1995). Atrial fibrillation: the last challenge in interventional electrophysiology. *British Heart Journal*; **73:** 209–211.

Palazzo MO. (1999) *What you need to know: a-fib 101*. Archived at: members.aol.com/mazern/afib101.htm. (Updated 4 March 1999; accessed 14 November 1999).

Prosser S, Worster B, Macgregor J, Dewar K. (2000). *Applied Pharmacology.* London: Mosby.

Resuscitation Council (UK). (2000). Advanced Life Support Course Provider Manual (4th ed.). London: Resuscitation Council (UK) and European Resuscitation Council.

Sherman DG, Hart RG, Easton JD. (1986). The secondary prevention of stroke in patients with atrial fibrillation. *Archives of Neurology*; **43:** 68–70.

Smith JJ, Kampine JP. (1990). *Circulatory Physiology – the Essentials. 3rd ed.* London: Williams & Wilkins.

Sopher SM, Camm AJ. (1996). Atrial fibrillation: maintenance of sinus rhythm versus rate control. *American Journal of Cardiology*; **77:** 24a–35a.

Thompson PL. (1997). *Coronary Care Manual.* London: Churchill Livingstone.

Tortora GJ, Anagnostakos NP. (2000). *Principles of Anatomy & Physiology. 7th ed.* New York: Harper Collins.

Wagner GS. (1994). *Marriott's Practical Electrocardiography. 9th ed.* London: Williams & Wilkins.

Waktare JE, Camm AJ. (2000). How do we maintain sinus rhythm in paroxysmal atrial fibrillation? In: *Royal College of Physicians of Edinburgh Proceedings: Conference on Atrial Fibrillation in Hospital and General Practice.* July 1999. Edinburgh: Royal College of Physicians of Edinburgh Press; 29(3): 5–11.

Waldo AL. (1993). Basic mechanisms of atrial fibrillation and atrial flutter. *Cardiology in Review*; **1:** 16–23.