

Atrial fibrillation part I: pathophysiology

Christine Cottrell discusses the physiological basis of atrial fibrillation and considers its causes and risk factors

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Atrial fibrillation (AF) is the commonest sustained heart rhythm disorder, and affects about 1.3% of the population in England and Wales (Wallentin et al, 2010) and 1–2% of the population in the UK (Go et al, 2001; Stewart et al, 2001; Camm et al, 2010). The risk of developing AF increases with age with more than 10% of cases diagnosed in people over the age of 75 years, rising to 23% over the age of 80 years (Stewart et al, 2002; Camm et al, 2010). AF is associated mainly with stroke, heart failure and sudden death.

Up to 90% of AF events may be symptomless (Page et al, 1994). Therefore, it is vitally important to ensure that practitioners understand what AF is, its consequences, and the need to screen and identify people who may be at risk of developing AF.

Prevalence and incidence

A report based on the Framingham study suggests that from the age of 40 years there is a lifetime risk of developing AF of one in four, independent of gender, and of one in six in the absence of congestive heart failure or

myocardial infarction. This equates to about 600 000 people in England with diagnosed AF and it appears to be more common in males than females (National Collaborating Centre for Chronic Conditions (NCC-CC), 2006).

It is proposed that this figure has been underestimated as a result of poor screening and many people being unaware that they have asymptomatic AF (NHS Improvement, 2009). About 46 000 people in the UK are reported to be diagnosed with AF annually (Iqbal et al, 2005).

AF is thought to be responsible for about 12 500 strokes a year at an estimated cost of £11 900 per stroke in the first year after stroke occurrence (NHS Improvement, 2009). This places a large burden on the NHS.

To identify AF in patients presenting in general practice, it is important to first understand the normal physiology of the electrical system of the heart.

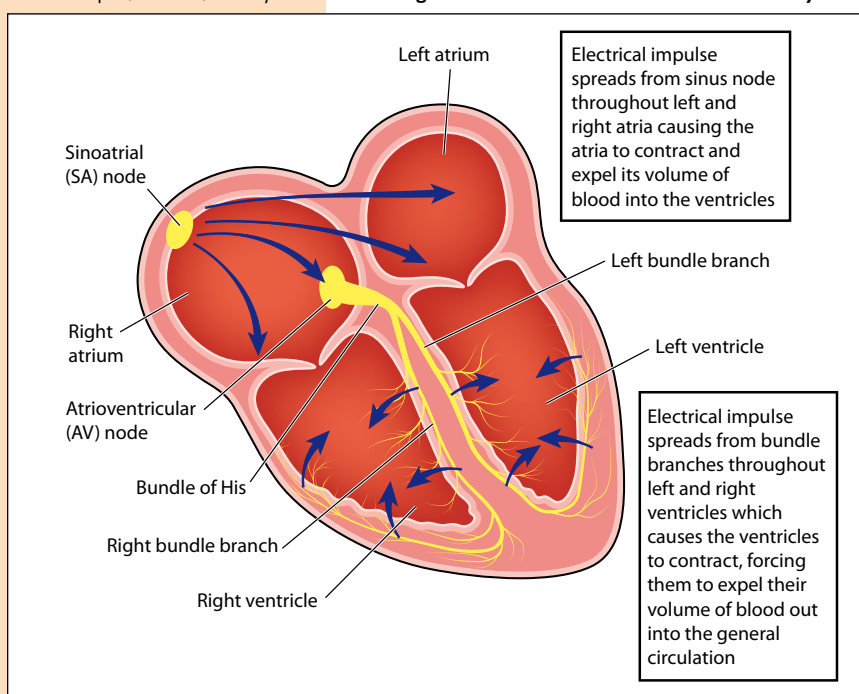
Normal conduction pathway

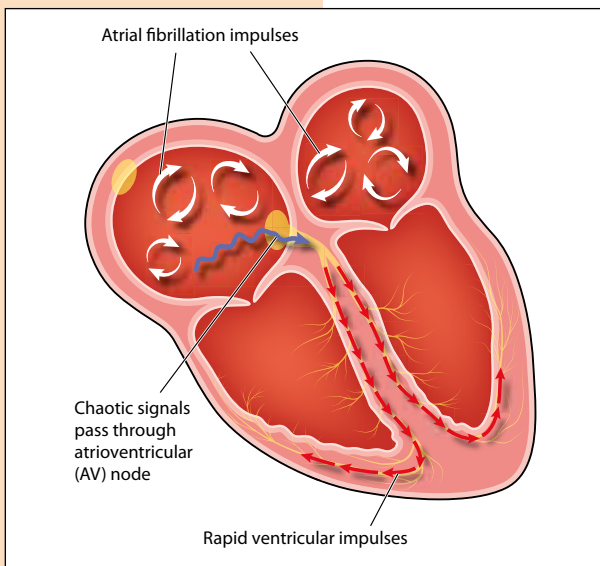
The rate and rhythm of the heart beat is primarily controlled by the electrical conduction system of the heart.

The heart's 'natural' pacemaker comprises a group of specialized cardiac cells that lie at the top of the right atria called the sino-atrial (SA) node (*Figure 1*). The exchange of electrical ions across the cardiac cells (myocytes) produces an electrical signal (impulse) that is initiated in the SA node. This causes a regular wave of electrical activity across the atria causing them to contract and eject blood into the ventricles. The wave of activity (depolarization) across the atria causes them to contract allowing evacuation of the content of blood into the ventricles.

The atrioventricular (AV) node or 'junction box' is known as the heart's secondary pacemaker and its function is to delay the number of impulses passing through to the ventricles and slow down the ventricular rate. There is a slight delay when signals travel through the AV node, which allows the atria time to empty their content of blood adequately and allows the ventricles time to fill. About 60–70 signals will pass through the AV node. The electrical

Figure 1. The heart's electrical conduction system





impulses pass down the bundle of His and across the ventricles via the Perkinje fibres causing a wave of activity (depolarization) across the ventricles causing them to contract and empty their contents. The heart then pumps the blood to the lungs (right ventricle) and to the body via the general circulation (left ventricle). The whole process repeats itself in a regular co-ordinated rhythm.

What is atrial fibrillation?

Atrial fibrillation is defined as a tachy-arrhythmia characterized by predominantly uncoordinated atrial activation with consequent deterioration of atrial function (NCC-CC, 2006).

Atrial fibrillation occurs because of an abnormality of the electrical signalling pathway (Figure 2). Instead of the signals following a regular co-ordinated pathway, signals are not systematically triggered via the SA node, but instead, are generated from all over the atria, resulting in a quivering or fibrillating uncoordinated atrial activity.

In the left atrium of the heart, the area around the pulmonary veins appears to be the site where multiple impulses are generated in most cases of AF (Figure 3). The impulses generated can be fired at a rate of about 300–600 beats per minute. The AV node will not be able to filter the number of signals coming from the atria. This is because the signals may be too fast, chaotic and irregular in nature or coming from multiple areas (foci) within the atria. This will lead to inadequate emptying of the atria.

The ventricular rate may increase as a result of an excess number of signals passing through the AV node. This increases ventricular activity (heart beat) to 60–130 beats per minute or more, affecting the emptying of the ventricles. If this continues then the general circulation of blood will be reduced leading to symptoms, e.g. light headedness, fatigue, breathlessness and chest pain, depending on the rate of ventricular activity, severity and duration of AF.

Within the left atrium of the heart is an area called the atrial appendage. This is an increased atrial muscle mass that forms a small sac or pouch (Figure 4). In AF, the atria are not able to empty their contents adequately because they are fibrillating. This allows ‘pooling’ of blood within the atrial appendage. Blood stagnates in this pouch and this leads to clot formation. When a part of the clot breaks away, an embolus is formed. An embolic stroke occurs because the embolus travels and occludes a blood vessel in the brain (Westerby and Cottrell, 2011). A feature of AF is that, as a result of inadequate emptying, the atria become enlarged over time and this may be a predisposing factor to permanent long-standing AF in adults.

In the UK, the incidence of newly diagnosed AF is about 46 000 per year (National Institute for Health and Clinical Excellence, 2010) and it is known that the incidence of stroke associated with AF increases with age at a rate of

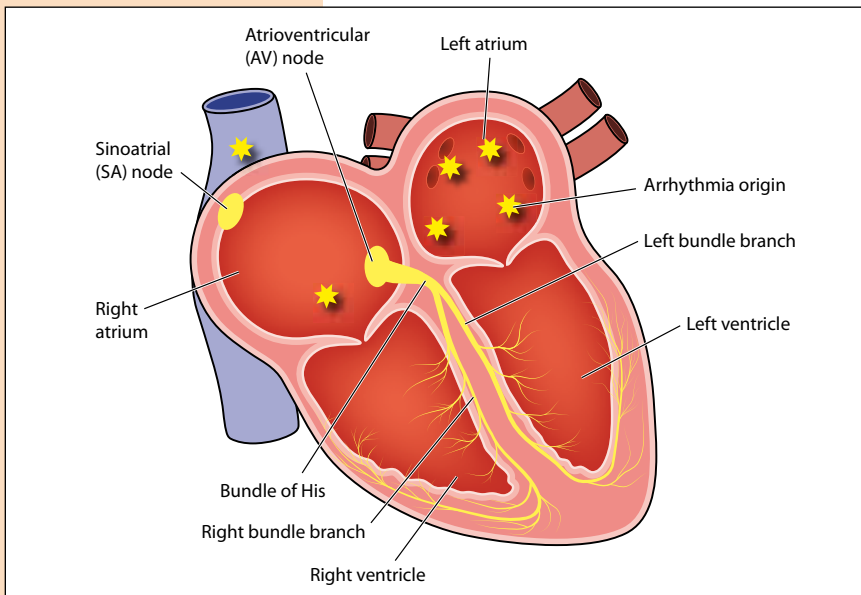


Figure 2 (top). Disturbed electrical conduction in a heart with atrial fibrillation.

Figure 3 (middle). Generation sites of multiple electrical impulses in atrial fibrillation

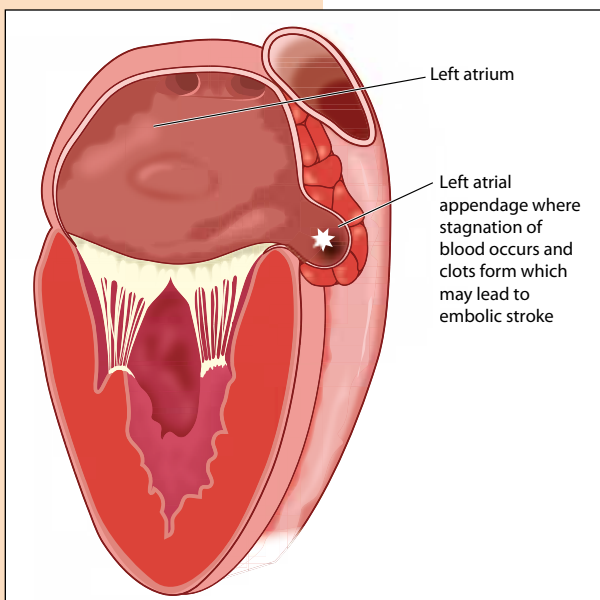


Figure 4 (left). Clot formation in an atrial appendage

1.5% between 50–59 years increasing to 23.5% at 80 years and older (Stewart et al 2001; ESC 2010). About 90% of strokes in cases of AF are found to have occurred from emboli from within the left atrial appendage (Blackshear and Odell, 1996).

About one in five people presenting with a stroke are found to have AF. Mortality is doubled in patients with AF compared to those with a normal heart rhythm and of similar age. In general, an embolic stroke attributable to AF results in greater disability and has a poorer prognosis and greater risk of death.

Symptoms

Patients may experience a variety of symptoms of AF. The most common include:

- Palpitations
- Shortness of breath
- Tiredness or fatigue
- Generalized weakness
- Poor exercise intolerance
- Dizziness or light-headedness
- An irregularly, irregular pulse.

Some of these symptoms may be associated with natural ageing but careful history taking in the consultation may lead to suspecting AF. Any person presenting with palpitations, shortness of breath and light-headedness should be investigated further as these are warning signs that the heart may be showing strain. Assessment and diagnosis is crucial so that a risk assessment can be done and appropriate management planned to avoid complications associated with AF.

More severe symptoms include:

Table 1. Causes of AF

Cardiac	Non-cardiac
Ischaemic heart disease	Chronic lung disease, e.g. asthma, carcinoma of the lung, pleural effusion, pulmonary embolus
Rheumatic heart disease (valvular)	Thyroid disease
Valve disease (mitral valve stenosis)	Electrolyte disturbance
Hypertension	Acute infections, e.g. pneumonia, pericarditis
Cardiomyopathy	
Heart failure	
Sick sinus syndrome	
Pre-excitation syndrome	
From: Kannel et al, 1998.	

Table 2. Some risk factors for AF

Advancing age
Obesity
Stimulants e.g. alcohol, smoking, medication, drug use
High intensity exercise
Stress
Latent hypertension
Sleep apnoea
Inflammation
Cardiac surgery
Long PR interval
Metabolic syndrome
Genetic factors
From: Schoonerwoerd et al, 2008; Rosiak et al, 2010.

- Hypotension
- Chest pain (angina)
- Decompensated heart failure (causing respiratory distress).

These are definitive signs that show the patient is being compromised and warrants rapid assessment and management, and, probably, emergency referral to secondary care.

Causes

AF is more commonly associated with underlying established disease including structural abnormalities of the heart. Causes can be defined as cardiac and non-cardiac (*Table 1*). AF can also occur as a result of thoracic surgery and coronary artery bypass grafts (NCC-CC, 2006).

The causes are likely to be the result of damage to the heart tissue itself, which may generate extra impulses or stimuli. Stretching and remodelling of cardiac muscle tissue appears to increase atrial pressure, which exerts pressure on the pulmonary veins. This is thought to be a contributing factor in other causes associated with AF, e.g. valve disease, heart failure, left ventricular hypertrophy (associated with hypertension), atherosclerosis and obesity (Larson, 2009). Inflammation damages the heart muscle tissue; electrolyte imbalance may contribute to over-stimulation and excitation of the myocytes, and these are all factors precipitating atrial fibrillation.

Risk factors

Age is a known risk factor for AF, with approximately 0.5–1% of the population being diagnosed over the age of 50 years and the incidence rising with each decade to 23% over the age of 80 years (Westerby and Cottrell, 2011). AF is thought to be the result of degenerative heart disease and/or atherosclerosis. Therefore it is vital to ensure that these people are screened for AF as figures estimating the incidence of AF indicate that opportunities are being missed to detect AF especially in a society where people are living longer (NHS Improvement, 2009).

There is evidence to show that obesity, metabolic syndrome, hypertension and diabetes are risk factors for cardiovascular disease and all of these are risk factors associated with AF (NCC-CC, 2006) (*Table 2*).

Although there is no definitive cause of AF, research shows that genetic factors may be associated with AF (Schoonerwoerd et al, 2008; Rosiak et al, 2010). Some familial pre-

dispositions to developing AF, especially in younger patients, include long- and short-QT syndromes, Brugada syndrome, and some cardiomyopathies (Westerby and Cottrell, 2011).

Classification and diagnosis

A useful way of thinking of the classification of AF in practice is the ‘three Ps’: paroxysmal; persistent and permanent (*Table 3*). The difficulty is in diagnosing AF into these categories in general practice. (The diagnosis of AF will be discussed in more detail in the next article in this series.)

In general, patients diagnosed with paroxysmal AF tend to develop persistent AF because episodes become more frequent, lasting longer in duration, and requiring intervention to terminate it. Likewise, people diagnosed with persistent AF are likely to develop permanent AF. The longer the duration and increased frequency of events, the more the heart is remodelled precipitating AF.

Lone or idiopathic AF

AF can occur in those with no specific cause or underlying disease; this is known as lone or idiopathic AF. It appears to be more common in younger people and affects about 50% of those diagnosed with paroxysmal AF (Allessie et al, 2001). Further research is needed to understand the aetiology of lone AF but some research suggests that inflammation is a factor that may trigger it (Frustaci, 1997).

Triggers associated with AF

Stimulants such as alcohol, smoking, caffeine, medication, drug use, high-intensity sport and psychological and physical stress are thought to be triggers of AF, possibly as a result of their affect on the autonomic nervous system (ANS) (Katan and Schouten, 2005; Conen et al, 2008; Heeringa, 2008; Schoonderwoerd et al, 2008).

The ANS controls involuntary actions such as heart beat. It comprises two parts: the sympathetic nervous system, which gears the body for action (e.g. the ‘fight or flight’ response), and the parasympathetic nervous system, which brings the body back to rest and stimulates digestion. The ANS influences the rate at which the SA node generates impulses in the heart. In particular, parasympathetic control of the heart is mediated via the vagus nerve which releases acetyl-

Table 3. Classification of AF

Class	Definition
Paroxysmal atrial fibrillation	A self-terminating arrhythmia that lasts for less than 7 days. It can be difficult to assess and so it is essential to obtain a good history during consultation to determine this as a paroxysmal AF
Persistent atrial fibrillation	When the arrhythmia lasts for more than 7 days but either self-terminates or is terminated via cardioversion then it is diagnosed as persistent AF. Persistent AF may eventually become permanent AF
Permanent atrial fibrillation	Once the arrhythmia has been long standing for more than 1 year it is classed as permanent AF. Usually, other treatments to restore AF rhythm back to sinus rhythm are unsuccessful

From: Levy et al, 2005.

KEY POINTS

- Atrial fibrillation (AF) is a serious condition
- AF accounts for one in five strokes; if not fatal, embolic strokes incur severe disablement
- People with asymptomatic AF are at a greater risk of having an embolic stroke
- Symptoms of palpitations, shortness of breath and an irregular pulse on palpation should be investigated further
- AF is prevalent in older people, those with heart disease and people with chronic disease. These at-risk groups should be actively screened to avoid complications

choline to suppress the release of the stress hormone noradrenaline (norepinephrine).

Neurogenic AF has been described as a type of paroxysmal AF associated with the ANS, usually in the absence of structural heart disease (Siotia and Muthusamy, 2004). It takes two forms.

Adrenergic AF is associated with the sympathetic nervous system and occurs in response to stress and exercise (Siotia and Muthusamy, 2004). It is less common than vagal AF.

Vagal AF is associated with the parasympathetic nervous system and occurs at night. The age of onset is typically between 30–50 years, and it is much more frequent in males (Siotia and Muthusamy, 2004). There appears to be an association between sleep apnoea and vagal AF (McNicholas et al, 2007).

In vagal AF, stimulation of the vagus nerve may lead to increased intra-atrial pressure and stretching of the heart tissue, which over time may result in stretching and remodelling of the heart.

Conclusions

AF is a serious condition which can lead to heart failure and embolic strokes, which if not fatal, incur severe disablement which impacts greatly on the individual, his/her family and is costly to the NHS. Up to 90% of people with AF may show no symptoms. Therefore recognition and diagnosis are critical.

It is important for practice nurses to know the risk factors associated with AF so that patients can be identified for assessment and management. Triggers of AF include lifestyle issues which need to be discussed with the patient presenting with symptoms of AF. The majority of AF cases are asymptomatic so it is essential that practice nurses obtain a good history during a consultation.

Diagnosis of atrial fibrillation will be discussed in more detail in forthcoming articles.

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