Understanding atrial fibrillation: A historical perspective

“I have tremor cordis on me: My heart dances; But not for joy; not joy”.
[William Shakespeare]

In 1685 Abercromby believed that “the origin of the pulse is as mysterious as the source of the Nile” [1]. However, in 1883 the tall, indefatigable Scotsman and general practitioner Sir James MacKenzie was determined to unravel the mystery [1]. After the unexpected death during childbirth of one of his patients due to a rhythm disorder and congestive heart failure he could not help but ask himself “Would this death have occurred if I had better knowledge of heart afflictions?”[2]. At that time little was known about “fibrillation of the auricles”.

The ancient disease was probably first described by the legendary emperor physician Huang Ti in China between 1696 and 2598 BC; “When the pulse is irregular and tremulous and the beats occur at intervals, then the impulse of life fades” [3]. In 1628 the meticulous observer William Harvey was the first to notice the “undulation/palpitation” of the “auricle” of the dying animal heart [4]. “…but I noticed that after the heart proper, and even the right auricle were ceasing to beat and appeared on the point of death, an obscure movement undulation or palpitation had clearly continued in the right auricular blood itself for as long as the blood was perceptibly imbued with warmth and spirit” [5]. Jean-Baptiste de Senac (1693–1770), the physician to Louis XV, devoted much attention to palpitations and in his necropsy reports related them to mitral valve disease, noting that the “reflux of blood into the auricles causes contractions of greater force, necessarily precipitating palpitations” [6] and that “if the auricles are strained and increased in volume they cause palpitations”. He almost prophetically rationalized the use of quinidine for atrial fibrillation, and he insightfully hinted at an ectopic origin: “the causes of palpitation are not the causes of the natural heart beat” [6].

The discovery of the therapeutic properties of the digitalis leaf (digitalis purpurea) in 1785 by William Withering brought some relief to patients with severe heart failure. It is interesting that Withering recorded a patient who had a weak, irregular pulse that became “more full and more regular” after treatment with digitalis [3].

René Laennec’s experience with irregular heartbeats led him to disparage reliance on palpation of the pulse, considering it an unreliable guide to the state of circulation [4].

Soon after, Robert Adams recognized atrial fibrillation clinically as a “sign of mitral stenosis” (1827) [3, 7]: “Extremely irregular action of the heart is almost pathognomonic of mitral stenosis” [5].

In 1830 Jean Baptiste Bouillaud considered digitalis as a “sort of opium for the heart”. Digitalis decreased the ventricular rate in patients even though the irregularity of pulse persisted [3].

Using heart sounds, Hope (1839) noted that exercise induced an increase in irregularity and a decrease in the “intermittent pulse” [1].

Marey and Chauveau passed metal sounds through the jugular veins of horses and interpreted their observations, and in 1863 were the first to publish pulse tracings of atrial fibrillation from humans with mitral valve stenosis [7].

In light of Marey and Chauveau’s observation, Potain was able to interpret the pulsations of the neck in 1867 [1].

Around 1874 Vaulpian in France and Hoff and Ludwig in Germany observed atrial fibrillation in experiments with animals.

Theodor Wilhelm Engelmann (1843–1909) proposed in 1894 that atrial fibrillation is caused by multiple foci, a theory that was further developed by Winterberg in 1906 and by Lewis [4].

Around 1900, a few clinical investigators, notably James Mackenzie in Scotland and Karel Weenkebach in Holland, studied cardiac arrhythmias with the use of arterial and venous pulse tracings.

Mechanically, Mackenzie (1853–1925) noted the absence of the presystolic ‘a’ wave seen in the jugular phlebogram during “pulsus irregularis perpetua” [8]. He described his findings as the “most
puzzling of all forms of irregularity of the heart, where the heart is never regular in its action, where seldom or never two beats of the same character follow one another” [5].

Electrically, Einthoven published in 1906 an ECG tracing of atrial fibrillation without knowing its nature [9]. He called it “pulsus inequalis et irregularis” [10].

In 1906 Cushing and Edmunds, two pharmacologists from the University of Michigan, convinced Mackenzie that the so-called “nodal rhythm” was atrial fibrillation. They studied atria in the open chest of anesthetized dogs and noticed that they were dilated and motionless when a “myograph” was placed directly on the atrium. They also used the term “fibrillary state” to describe the atrial walls [9].

Sir Thomas Lewis (1881–1945), sometime around 1909, captured atrial fibrillation on an ECG and studied the mechanism of conduction, noting that atrial fibrillation was “continuously and extremely irregular”. He called it a “common clinical condition”, establishing it as a clinical entity [10].

In 1909 Vienne Rothenberger and Winterberger identified a direct connection between the “arrhythmia perpetua” and “fibrillation of the auricles” [5]. Their paper from 1915 proposed that atrial fibrillation resulted from rapidly discharging spontaneously active ectopic foci. They postulated that the irregularity of the rhythm resulted from the interaction between the wave fronts produced by the focal generator and the variable refractory periods of the atrial tissue [7].

The theory of multiple-circuit re-entry was introduced by Garrey, and advanced later by Moe and Abildskov. This was later refined by Allessie in the 1970s and 1980s [7]. Garrey also provided experimental evidence that a “critical mass” of atrium was required to sustain atrial fibrillation [7].

Our understanding of the basic mechanism of atrial fibrillation has improved with the observations of many brilliant scientists, especially noting the modern work of Jalife et al. [11].

“This history of atrial fibrillation underscores the importance of collaboration of scientists, clinical investigators and physicians in the discovery, refinement and application of new knowledge” [10].

“I know that the truth will prevail, when if I am wrong my views will be forgotten and if I am right, time will vindicate them.” — James Mackenzie [2].

References