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Atrial Fibrillation (AF): Causes, Sequela, Risk Factors, and Management

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Atrial Fibrillation(AF): Causes, Sequela, Risk Factors, and Management Strategies

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Importance

Atrial fibrillation(AF) is a common cardiac disturbance encountered in the healthcare field (January et al., 2014, p. 6). Nurses ought to remain up to date with the current understanding and treatment of AF.

Definition

A rapid, irregularly, irregular atrial rhythm (Gutierrez et al., 2016, p.442).

Diagnosis by ECG

- No p waves
- Irregularly, irregular R-R interval
- Possible presence of fibrillatory waves

Adapted from Gutierrez et al, 2016, p. 442.

Classifications

- Paroxysmal AF: Typically lasts <1 week and spontaneously converts to sinus rhythm.
- Persistent AF: Continuous AF that lasts >1 week.
- Long-standing: >1 year, but may still convert.
- Permanent: Sinus rhythm cannot be restored.

Adapted from Mitchell, 2017, p. 2-3

Symptoms

- May be asymptomatic
- Palpitations
- Presyncope
- Syncope
- Chest pain
- Shortness of breath
- Weakness
- Irregular pulse

Adapted from Gutierrez et al., 2016, p.446

Risk Factors

- Age > 65
- Hypertension
- Coronary artery disease
- Cardiomyopathy
- AV Valve disorders
- Hyperthyroidism
- Lung disease

Adapted from January et al, 2014, p.7

Terms to Know

Anatomical re-entry features a refractory core (typically scarred or ischemic tissue), a "slow" pathway, and a "fast" pathway. Signals travel around the core through the fast pathway and are slowed enough by the slow pathway to give the fast pathway time to become excitable again just before the slowed signal arrives. This forms a loop of re-entry (Zaman et al., 2014, p. 49-50). See below.

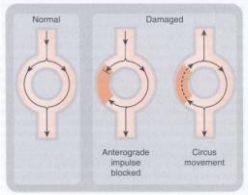


Fig. 17.3 Generation of a re-entrant rhythm by a damaged area of myocardium. The damaged area (brown) conducts in one direction only. This disturbs the normal pattern of conduction and permits continuous circulation of the impulse to occur.

Shante, 2017.

Functional re-entry occurs with a region of myocardial tissue that is heterogenous in terms of its conductive properties. When an electrical signal passes through this area it may be slowed or even blocked in some areas while being conducted normally in other areas. The blocked signal often fragments in different directions, sometimes circling back on itself and forming a re-entrant loop (Zaman et al., 2014, p. 49-50).

The distinction between anatomical and functional re-entry is best understood as anatomical being fixed (scar tissue, valvular tissue, etc.) and functional as being variable and often unstable (sometimes it will conduct normally, other times erratically). This often makes locating functional re-entrant circuits much more difficult than anatomical circuits.

Excitable gap: The period of time between when a cardiac myocyte has repolarized (meaning it is now available to depolarize) and the time the next signal would normally arrive. In sinus rhythm, this would essentially be the time between the end of the T wave and the beginning of the next QRS complex in ventricular tissue (Surawicz & Knilans, 2008, p. 433).

The excitable gap is a period where there is opportunity for a different circuit to take over. This can mean that a rhythm such as atrial flutter is started via a PAC while some arrhythmias are actually stopped because of other premature beats that strike during an excitable gap, making the tissue refractory to the arrhythmia's impulse.

Sequela

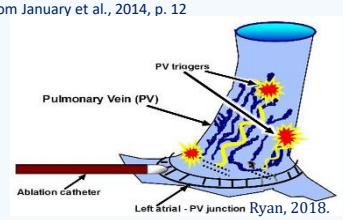
- Thromboembolism (Schmid et al., 2011, p. 220)
- Heart failure (Gutierrez et al., 2016, p. 442)
- Treatment-associated problems such as bleeding risk with anticoagulation or exercise intolerance from beta blockers and/or nondihydropyridine calcium channel blockers.

Pathophysiology

Vein Signals

- Myocardial sleeves are known to extend into veins connected to the myocardium (pulmonary veins, superior/inferior vena cava, coronary sinus, and Marshall's vein). The pulmonary veins(PV) play a much more significant role in AF than the other veins.
- The sleeves are capable of generating ectopic electrical signals and initiating AF.
- They display variability in conduction properties and refractoriness which may enable them to maintain AF through re-entry.
- Vein signals appear to be the primary contributors in paroxysmal AF.
- Re-entry in AF is believed to be primarily *functional.* However, this functional source is usually isolated to a common anatomic region (the pulmonary veins).

Adapted from January et al., 2014, p. 12

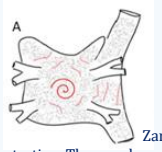


Ryan, 2018.

- As an example, suppose patient Bob has rapid signals arising from 3 of the 4 pulmonary veins. As these signals enter the atrium, they depolarize it from several different directions at slightly different intervals. Some of these signals run around each other in a race for non-refractory tissue. Eventually, one of two things happens…
  - The signals travel around the atria in discordant patterns along with additional signals still arising from the pulmonary veins, and AF perpetuates.
  - All the signals dead end into refractory tissue, signals cease to arise from the PV's, and AF is terminated.

Rotors

- Complicated form of functional re-entry which appears to be more important in persistent AF (Spitzer et al., 2017, p. 31).
- Unlike anatomical re-entry with single wavefronts that encircle a non-excitable core, rotors have "singularities," which are regions surrounded by spiral waves that disperse into the atrial "milieu" (Krummen et. al, 2015, p. 143).
- Rotors display no or almost no excitable gap, so there is essentially no opportunity for the circuit to yield to another signal unless it is overwhelming (Krummen et. al, 2015, p. 143).
- Rotors appear to be capable of shifting within a 2-3cm^2 area of myocardium. This capability has made it challenging to even prove their existence until recently. It also makes them difficult to locate, especially when there are multiple rotors (Krummen et. al, 2015, p. 143).



Zaman et al., 2018

Rotor 2-D illustration. They are known to have filaments extending from endocardium to epicardium, but are difficult to portray in 3-D view.

References

Consequences

The rapid and chaotic electrical impulses in the atria causes many small different areas to contract at different times. The result are atria that quiver, rather than squeeze, and fail to contribute their extra blood volume to ventricular diastolic filling. Thus, cardiac output may decrease and signs/symptoms of heart failure may present.

Some portions of the atria, particularly the left atrial appendage, may have blood stasis with resulting thrombus formation. Risk of a stroke from atrial fibrillation is estimated to be increased 5-7 fold compared to the rest of the population (January et al., 2014, p. 8-9).

Management

Drug Therapy

Rhythm control: Amiodarone, Propafenone, Flecainide, Ibutilide, Dofetilide (January et al., 2014, p. 32)

Rate control: β1 antagonists, nondihydropyridine calcium channel blockers, amiodarone, digoxin (January et al., 2014, p.29)

Anticoagulation: Beyond the scope of this presentation due to the complexity of the recommendations. See Norby et al., 2017, p. 238-249.

DC Cardioversion (Biphasic)

Normal or lower BMI: 100J was equally effective to 200J in first shock success. Overweight or greater BMI: 200J was significantly more effective in first shock success (Glover et al., 2008, p. 884).

"It is reasonable to perform transesophageal echocardiogram (TEE)" to rule out intracardiac thrombus formation (particularly in the left atrial appendage) prior to DCCV in stable AF patients with symptoms >48 hours or unknown symptom duration – Class IIa recommendation (January et al., 2014, p. 33).

Procedures

Ablation: Pulmonary Vein Isolation (PVI) – Venous catheters are advanced to the right atrium and punctured through the atrial septum. An ablation catheter is then used to burn or freeze tissue around ostium of PV's and a multi-electrode catheter is used to verify isolation (Hachem et al., 2018, p. 1).

Ablation: Focal Impulse and Rotor Modulation (FIRM) – A basket catheter and real-time 3D mapping are utilized to identify singularities and ablation catheters are used to burn/freeze them. This is a relatively new and controversial therapy (Spitzer et al., 2017, p. 32-34).

MAZE: A maze of scar tissue is created in the atria around arrhythmogenic zones from which only one path leads to the rest of the myocardium. Techniques include radiofrequency (heat) ablation, cryoablation (cold), and surgical incisions. It is most commonly done during heart surgery for other problems such as CAD and valvular disease, but is also available by itself as a minimally invasive procedure (Sharples et al., 2018, p. 21).

Left atrial appendage closure: The LAA is a common source of thromboembolism in patients with AF. There are multiple options available to close the LAA off from the left atrium such as ligation, Atriclip, WATCHMAN, and the Amplatzer Cardiac Plug (Cleveland Clinic, 2017).

Nursing Implications

- Be aware of the signs and symptoms of atrial fibrillation and expand assessment accordingly. For example, if a patient becomes short of breath, do not stop at a respiratory assessment. Examine the patient's rhythm if on telemetry or the apical/radial pulse and consider ECG if orders are on standby.
- Be able to identify atrial fibrillation on ECG if telemetry is utilized in your work area.
- Ask the patient when symptoms first started because this will be important in establishing the need for TEE if DCCV is considered.
- Obtain order to establish adequate IV access if patient's stability is in question.
- Anticipate orders for the typical medications, dosages, IV push rates, and monitoring required for treatment of AF.
- Anticipate orders for anticoagulation – a "bridge" of a heparin/bivalirudin infusion is common until warfarin is therapeutic. This may not be necessary depending on AF duration and the agents utilized for anticoagulation.
- Be prepared to educate patients and caregivers about atrial fibrillation and be careful to distinguish it from ventricular fibrillation. Let them know that it is often controlled rather than fixed and that many people with AF lead normal lives.

Conclusions

The significance of atrial fibrillation on health cannot be overstated. AF as a primary diagnosis causes greater than 467,000 hospitalizations per year in the United States (January et al., 2014, p. 7). The causes of atrial fibrillation are multifactorial so maintaining control of it can be challenging for healthcare providers. This difficulty is evident in the fact that no single treatment is effective for everyone.

As surgeons and electrophysiologists continue to search for and utilize new treatment options, such as WATCHMAN and FIRM, nurses in the field also need to stay current. A stronger understanding of the mechanisms underlying these options is important to providing excellent care and education to our patients. This author hopes to have enhanced the knowledge of other nurses to that end.

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