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Epidemiology of Arrhythmias and Conduction Disorders in Older Adults

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Abstract

Normal aging is associated with a multitude of changes in the cardiovascular system, including decreased compliance of blood vessels, mild concentric left ventricular hypertrophy, an increased contribution of atrial contraction to left ventricular filling, and a higher incidence of many cardiac arrhythmias, both bradyarrhythmias and tachyarrhythmias. Conduction disorders also become more common with age, and may either be asymptomatic, or cause hemodynamic changes requiring treatment. The epidemiology of common arrhythmias and conduction disorders in the elderly is reviewed.

Keywords

Aging; arrhythmia; atrial fibrillation; atrial flutter; conduction disorders

EFFECTS OF AGING ON THE CONDUCTION SYSTEM, ASSOCIATED CONDUCTION DISORDERS, AND BRADYARRHYTHMIAS

Aging affects the cardiovascular system in multiple ways, including a decrease in compliance of blood vessels through arterial stiffening and thickening, mild left ventricular thickening, and a shift in the balance of early versus late diastolic filling. Many of these changes result, in part, from cardiac cell enlargement with apoptosis of neighboring cells and subsequent fibrofatty infiltration of the myocardium.[1] The conduction system of the heart is also affected by the latter, producing changes that may result in conduction disorders or arrhythmia.

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SINOATRIAL NODE

Aging is associated with increased fat and collagen deposition surrounding the sinoatrial (SA) node, which may result in delay of action potential propagation or even complete electrical separation of the node from surrounding tissue. During the course of normal aging, the number of pacemaker cells in the sinoatrial (SA) node declines significantly after age 60 years, with < 10% of the cells seen in young adults remaining by age 75. Paradoxically, although older adults generally have fewer SA nodal pacemaker cells, they also have a lower prevalence of sinus bradycardia.[2] This counterintuitive observation is likely due to an offsetting age-related reduction in parasympathetic activity, which is also responsible for decreased heart rate variability and reduction in sinus arrhythmia.[3,4]

Although normal aging alone does not change the normal resting heart rate (HR) range of 60 – 80 beats per minute (bpm) in adults, it causes a predictable decrease in peak HR, with resultant decrease in maximal oxygen delivery during exercise. For each year following the onset of adulthood, peak heart rates decrease at a rate of approximately 0.7 – 1.0 bpm per year. For routine stress testing, maximum age-predicted heart rate (MPHR) is commonly estimated as $220 - \text{age (years)}$. [5] However, several groups [6–8] have recently called into question the accuracy of this formula, as its initial publication appears to have been based on a linear best fit to a series of observational data in 1971.[6] Several revised formulae for calculating MPHR are now available although none has yet been promoted in a widely-recognized practice guideline.

Sinus node dysfunction

The umbrella terms ‘sinus node dysfunction’ (SND) and ‘sick sinus syndrome’ may be used in reference to any condition in which the atrial rate is inappropriate for physiological requirements.[9] These include: 1) symptomatic sinus bradycardia, sinus pauses, or arrest; 2) chronotropic incompetence, and 3) alternating periods of atrial tachyarrhythmias and bradyarrhythmias (“tachy-brady syndrome”). Although the overall prevalence of SND in the elderly is unknown[10], it is estimated that 70–80% of all pacemakers implanted for the indication of SND occur in patients over the age of 65.[11,12]

Sinus bradycardia—Sinus bradycardia is a common, usually normal and asymptomatic finding, defined by a sinus rate of less than 60 bpm. It should be recognized that this cutoff point is arbitrary. Although classified as an arrhythmia, it may be a normal variant in patients whose parasympathetic system is particularly dominant, such as competitive athletes. Although the prevalence of sinus bradycardia does not increase with age, *symptomatic* sinus bradycardia due to sick sinus syndrome occurs almost exclusively after the age of 65, at an estimated rate of one per 600 elderly cardiac patients.[9]

In the Baltimore Longitudinal Study of Aging (BLSA), 4.1% of 1172 healthy, nonendurance-trained, unmedicated participants aged 40 or greater were found to have sinus bradycardia (defined as a HR < 50 bpm) on resting ECG. Prevalence of unexplained sinus bradycardia was similar between men (3.9%) and women (4.5%), and was associated with an increased prevalence of conduction system abnormalities (43% vs 19%, $p < 0.05$), including first-degree AV block, left axis deviation, incomplete or complete RBBB; however, none experienced syncope, high-degree AV block, or symptomatic sinus node dysfunction. [13] There was no significant difference in major adverse cardiac events or deaths between groups during an average 5.4 year follow-up.

In 1987, Kannel et al. reported results from the Framingham Heart Study, in which HR was determined from resting ECG examinations in a supine position.[14] Of the 5070 individuals who entered the study without history of cardiovascular disease, HR tended to increase with

age in both men and women, with a linear increase in overall death rate observed with increasing baseline resting HR.(Table 1) The prevalence of sinus bradycardia was low (<5%) for both men and women older than 65 years..

ATRIOVENTRICULAR (AV) NODE AND HIS-PURKINJE SYSTEM

Aging results in varying degrees of calcification of the cardiac skeleton, particularly in the region including the central fibrous body and the left-sided valves (aortic and mitral valve rings). The AV node, AV bifurcation, as well as the proximal left and right bundle branches are located near the central fibrous body, and are thus vulnerable to slowed signal transmission with increasing age-related changes.

The PR interval undergoes a modest but significant prolongation with advancing age. In 46,129 subjects with very low probability of cardiovascular disease an increase in mean PR interval occurred between the third and ninth decades of life, both in men (from 153 to 182 ms) and women (from 148 to 166 ms).[15] In the BLSA, a similar increase in PR interval prolongation was seen between the ages of 30 and 72 years, in both men (159 to 179 ms) and women (156 to 165 ms), due to prolongation of conduction proximal to the His bundle. [16] Although PR prolongation is often seen in normal aging, exaggeration of this phenomenon may clinically manifest as AV nodal block.

In contrast to the PR interval, QRS duration shows no significant age relationship, although the QRS axis does shift leftward with age. Mason et al. reported a mean QRS axis shift from 56 to 8 degrees between the third and ninth decades, with corresponding lower limits shifting from -3 to -60 degrees.[15] Thus, the prevalence of left axis deviation (defined as a QRS axis <-30 degrees) increases to 20% by the tenth decade.[17] This age-associated leftward QRS axis shift may be due in part to increases in left ventricular wall thickness. Although some longitudinal studies have shown small increases in cardiovascular mortality associated with this isolated ECG finding in the general population [18] or persons referred for exercise testing [19], it remains unclear whether this increased risk extends to the elderly.

Atrioventricular (AV) nodal block

First degree AV block—First degree AV block is defined as a PR interval of > 200 ms, representing a delay in AV conduction within the AV junction, usually within the AV node. The condition is usually asymptomatic and is associated with normal aging. The prevalence of first-degree AV block in healthy older men is approximately 3–4%, which is severalfold greater than in young men.[20]. In 1986, the Manitoba study analyzed the resting ECGs of 3983 healthy airmen who were followed for 30 years, reporting percentage distribution of PR intervals according to age.[21] By the seventh decade, 20% of study participants had a PR interval of at least 200 ms but a PR interval > 220 ms was seen in only 4% of this group. No significant differences in cardiac morbidity or mortality were observed in these latter individuals compared to age-matched controls during 30 years of follow-up. [21]

Although previous cross-sectional and longitudinal studies have generally found no correlation between first-degree AV block and cardiac disease [22] or mortality [21], a recent report using 20-year follow-up data from 7,575 individuals in the Framingham study (mean age 46 ± 15 years at baseline) demonstrated increased risks of atrial fibrillation, pacemaker implantation, and all-cause mortality associated with PR interval prolongation, even within the normal range.[23] In the Heart and Soul study representing 938 patients with known stable coronary disease and mean age 66 years, an association was found between first-degree AV block (defined as a PR interval > 220 ms) and an increased risk of both heart failure hospitalization (OR 2.33, 95% CI 1.49–3.65; $P < 0.01$) and overall mortality (OR

1.58, 95%CI 1.13–2.20; $P<0.01$) over a 5-year follow-up period. [24] Thus, the prognostic significance of first-degree AV block may differ, depending on whether cardiac disease is present.

Mobitz I second degree AV block—Mobitz I second degree AV block is characterized by a progressively lengthening PR interval until complete block occurs in the AV node, resulting in a non-conducted P wave. This conduction disorder is often asymptomatic and clinically silent.

Although no large population-based studies specifically report the prevalence of Mobitz I AV block, a study of 625 asymptomatic patients undergoing 24-hour ambulatory ECG monitoring showed transient Mobitz I block in 2.2% of individuals, occurring more frequently in those with a resting HR less than 60 bpm.[27] Of the 14 subjects who experienced this conduction disorder, 64% were men, with a wide age distribution of 22 – 80 years (mean 42 ± 14 years). This wide age range likely encompasses both young subjects with AV block due to high vagal tone and older subjects with AV block due to AV nodal disease.

The natural history of Mobitz I block was examined in 147 patients from the Devon Heart Block and Bradycardia Survey found to have Mobitz I block on resting ECG. Professional athletes and patients with evidence of prior or coincidental Mobitz II or complete heart block, transient block following acute infarction, or drug-induced block were excluded.[26] Pacemakers were implanted in response to either significant symptoms associated with Mobitz I block (i.e. pre-syncope, syncope, or confusion) or the subsequent development of higher degree block. After 1982, elderly patients were offered prophylactic pacemaker implantation if no contraindications existed. Of the 147 study patients, 90 (61%) received pacemakers. Patients ages 45–79 years who received a pacemaker enjoyed a significantly improved 5-year survival rate, while those over 80 years showed a non-significant survival advantage over those not receiving a pacemaker (Table 2).

High degree AV block—The term “high degree AV block” (HAVB) encompasses both Mobitz II second degree AV block and third-degree (or complete) AV block, both of which usually require a permanent pacemaker. HAVB may occur either as an isolated diagnosis in the elderly, often in the setting of hypertension and/or diabetes mellitus, accounting for approximately 40% of all implanted pacemakers in the United States[27], or as a complication following acute myocardial infarction, cardiac surgery, endocarditis, or other cardiac injury.

The overall prevalence of HAVB in the general population is low. For the assessment of third-degree heart block, the Reykjavik Study prospectively evaluated 9139 men and 9773 women aged 33 – 79 years with baseline ECGs over a 24-year period.[28] Complete AV block was found in only 11 individuals (0.04%, mean age 55 years), and was transient in 7 (64%) of these 11 cases.

In the largest pacemaker and implantable cardioverter-defibrillator (ICD) survey ever performed, data on more than 80% of all devices implanted worldwide during 2009 were analyzed by Mond and colleagues.[29] Of the 61 countries participating in the survey, the United States implanted the most devices ($n=225,567$), with Germany implanting the most devices per capita (927 devices per million population). In countries reporting the prevalence of pacemaker implantation for the indication of HAVB, results were highly variable between nations – from 15% (Greece) to 95% (Sudan) of all procedures performed. The mean age of patients receiving a pacemaker also varied significantly (men: mean 44 years (Qatar) to 78 years (Puerto Rico); women: mean 47 years (Qatar) to 80 years (Italy)).

Despite many differences in accessibility of healthcare and availability of pacemakers to the populations of the world, patients older than 60 years consistently comprised the largest group of pacemaker recipients, with the highest proportions in Uruguay (94%) and Italy (95%).

His-Purkinje conduction abnormalities

Right bundle branch block—Right bundle branch block (RBBB) increases in prevalence with age. In the Framingham Heart Study, the incidence of RBBB peaked in men in the seventh decade, while a continued rise occurred in women throughout the study period.[30] In total, 70 new cases of RBBB were detected during the 18-year follow-up period, representing 1.3% of the total study population. Although the initial appearance of RBBB was not associated with adverse clinical events, subsequent incidence of coronary artery disease was 2.5 times greater ($P<0.001$) and congestive heart failure was almost 4 times greater ($P=0.02$) in patients with RBBB compared to those without by the end of the study period.

In the BLSA, RBBB was observed in 39 of 1142 (3.4%) men on resting ECG, of whom 24 (2.1%) had no evidence of associated cardiac disease. Mean age on presentation with, or development of, RBBB was 64 ± 13.5 years. In both the BLSA and Framingham cohorts, the diagnosis of RBBB in persons *without* concurrent clinical heart disease was not associated with major adverse cardiac events.[31] In the Reykjavik Study, RBBB increased in prevalence from 0% in persons 30–39 years to 4.1% of men and 1.6% of women 75–79 years old. In men but not women, RBBB was associated with cardiomegaly, ischemic heart disease, and arrhythmias on resting ECG. However, neither total nor cardiovascular death were associated with RBBB on multivariate analysis. [32]

Left bundle branch block—In contrast to RBBB, left bundle branch block (LBBB) is more specific for the presence of cardiovascular disease (e.g. antecedent hypertension, cardiac enlargement, cardiomyopathy, or coronary heart disease).[33, 34] The prognosis of patients with LBBB, therefore, is closely tied to that of the underlying heart disease.

Both incidence and prevalence of LBBB increase with age. In the Framingham Heart Study, 55 new cases (31 men, 24 women) of LBBB were detected during the 18-year follow-up period, representing 1.1% of the total study population.[33] Mean age at onset of LBBB was 62 years (range 36 – 78 years). Only 15 (27%) of the 55 new cases of LBBB were free from all preceding cardiovascular abnormalities, although 5 (33%) of the 15 eventually developed coronary heart disease – a rate more than three times higher than control subjects without LBBB.

The Irish Heart Foundation screened a large general population ($n=110,000$) from 1968 to 1993 using a single resting ECG, revealing 112 subjects (0.1%) with LBBB and no prior history of hypertension or heart disease.[34] Follow-up of all cases and controls matched for age and sex was performed in 1993 via postal questionnaire, completed in 98% of cases and 97% of controls. Individuals with LBBB had a mean age of 51 ± 13 years and were predominantly male (73%). The prevalence of LBBB increased with age in both men and women (Figure 1). Cardiovascular disease developed in more patients with LBBB than in controls (21% vs 11%; $P=0.04$), although the incidence of cardiac death was not significantly increased. Two (1.9%) of the 112 subjects with new LBBB eventually required permanent pacemaker implantation.

TACHYARRHYTHMIAS

Tachyarrhythmias form a large, heterogeneous group of disorders in older adults. Below, we discuss the epidemiology of supraventricular and ventricular tachyarrhythmias in the elderly population.

Supraventricular tachyarrhythmias

Sinus tachycardia—Sinus tachycardia (ST) is common, may present without symptoms, and is usually a result of anxiety, fever, thyrotoxicosis, acute hypovolemia/anemia, or other acute illness. Because sinus tachycardia generally results from increased sympathetic and/or reduced parasympathetic tone, many texts classify this rhythm as a physiologic sign rather than a free-standing arrhythmia – just as ‘fever’ is generally considered a symptom rather than a diagnosis. As such, its overall incidence and prevalence are difficult to ascertain, as there are a myriad of etiologies, and the condition is usually transient in nature.

Supraventricular premature beats—Supraventricular premature beats, including atrial premature beats (APB) and those of junctional origin, occur on resting ECG in 5–10% of all individuals older than 60 years and are commonly seen during ambulatory 24-hour monitoring. Among 1372 predominantly healthy individuals aged 65 and older in the Cardiovascular Health Study (CHS), isolated APB were seen in 97% in a 24-hour period. [35] In healthy BLSA volunteers older than 60 years, resting ECGs demonstrated APB in 6%, exercise stress testing provoked APB in 39%, and 24-hour ambulatory monitoring captured APB in 88%. [36] Even when frequent, supraventricular premature beats on ambulatory monitoring were not predictive of increased adverse cardiac outcomes over a 10-year mean follow-up period. [37]

Atrial fibrillation—Atrial fibrillation (AF) is the most prevalent clinically significant rhythm disorder in the elderly, affecting approximately 2.3 million Americans, comprising around 6% of those over 65 years and 12% of individuals above 85 years [38,]. Furthermore, the prevalence of AF is projected to increase 2.5-fold over the next 50 years. [38] In 1995, Feinberg and colleagues [39] reported the combined age-specific prevalence of AF from four large studies: the Framingham, [40] Western Australia, [41] Rochester, [42] and Cardiovascular Health Study (CHS) (Figure 2). [43] The authors found that the overall prevalence of AF was higher in men than in women for all age groups, but because there were almost twice the number of women than men >75 years in the general population, the absolute numbers of women and men with AF were similar. A recent meta-analysis [44] of 10 studies involving 1,031,351 subjects found a markedly lower prevalence of AF in African Americans compared to Caucasians (OR 0.51, 95%CI 0.44 – 0.59, $P<0.001$) in both the general population and in those over 60 years of age.

Numerous factors likely contribute to the increase in AF prevalence with age, including an increased prevalence of comorbid conditions, such as hypertension, diabetes, thyrotoxicosis, and mitral valve disorders. [45] Increased left atrial size, which provides an anatomical substrate for wavelets of micro-re-entry, is common among elderly individuals, and is an independent predictor of AF. [46] An age-related increase in left ventricular stiffness, with resulting diastolic dysfunction and elevated left atrial pressure, may also serve as an important contributor. [47] AF may arise without obvious comorbidities (“lone AF”), accounting for 17% of men and 6% of women with this arrhythmia (mean ages 70.6 and 68.1 years, respectively) in the Framingham study. [48] Lone AF was associated with a 4-fold higher rate of strokes but no increase in coronary events or heart failure over long-term follow-up. AF accompanying structural heart disease is associated with increased rates of heart failure, stroke and cardiovascular death. AF accounts for nearly one quarter of strokes

occurring among octogenarians compared with only 1.5% in persons in their 50s. [49] AF is also a major predisposing factor to heart failure with preserved EF, the predominant form of heart failure in the elderly. [50]

Post-operative AF occurs in up to 40% of patients after cardiac surgery and is a major source of increased morbidity, hospital stay and costs in these patients. [51] Advanced age is a potent risk factor for post-operative AF. [51,52]

Despite advances in blood pressure control and risk factor management over the past decade, the high incidence of AF in older adults continues unabated. Recent research indicates that approximately 10% of patients over the age of 65 who have pacemakers or ICDs had subclinical and previously undetected AF, discovered when their devices were routinely interrogated three months following implantation.[53]

Atrial flutter—Atrial flutter (AFL) is a relatively uncommon arrhythmia in adults, caused by a macro-reentrant loop in either the right or left atrium. In a recent population study of over 54,000 individuals, the 4 year incidence of AFL was 0.14%, AF was 0.73%, and a combination of AFL and AF was 0.19%. The mean age of AFL and AF cohorts were 70 and 72 years, respectively (P=NS).[54] Although many predisposing factors are shared between AFL and AF (with many patients affected by both arrhythmias), subjects with AFL without concurrent AF were more likely to have had a history of obstructive lung disease (25% vs 12%, P=0.006) and heart failure (28% vs 17%, P=0.05), while hypertension was more common (63% vs 47%, P=0.01) in the cohort with AF without concurrent AFL. [54]

Paroxysmal supraventricular tachycardia—Supraventricular tachycardia (SVT) is a general term, encompassing the diagnoses of atrial tachycardia (AT), atrioventricular nodal re-entrant tachycardia (AVNRT), and atrioventricular reciprocating tachycardia (AVRT). Short, largely asymptomatic bursts of SVT have been observed in up to 50% of the normal elderly population in prior studies utilizing 24-hour monitoring,[35, 36] with increasing incidence associated with advanced age. Prevalence of clinically-evident paroxysmal SVT also increases with age, occurring in an estimated 6.16 cases per 1,000 population in patients > 65 years.[55] In the BLSA, short PSVT bursts detected during exercise testing also became more prevalent with age, but did not presage increased risk of subsequent coronary events in clinically healthy older adults.[56] The prevalence of exercise-associated PSVT episodes steadily increased in men, but reached a plateau after the age of 60 years in women (Figure 3).

Ventricular tachyarrhythmias

Ventricular premature beats and non-sustained ventricular tachycardia—

Ventricular premature beats (VPB) increase in prevalence with advancing age, in both unselected patient populations and those clinically free of heart disease. An evaluation of approximately 2,500 ECGs from elderly hospitalized patients revealed VPB in 8%.[57] while resting ECGs in apparently healthy BLSA volunteers with a normal ST-segment response to treadmill exercise showed isolated VPB in 8.6% of men over the age of 60, compared to only 0.5% in those in the 20–40 year age range.[58] Interestingly, there was no correlation of VPB prevalence with age in women.

Ambulatory 24-hour ECG recordings in multiple studies have shown a significantly higher prevalence of VPB, ranging from 69% to 96%, in the asymptomatic elderly compared to younger age groups.[35,36,59,60] In the CHS, VPB were found in 82% of 1372 elderly subjects, including 3- to 5-beat runs of non-sustained ventricular tachycardia (VT). [35] VPB were seen in 96% of individuals older than 80 years without clinical heart disease (n=50).[59] In 98 asymptomatic BLSA participants older than 60 years, 35% had multiform

VPB, 11% VPB couplets, and 4% brief bursts of nonsustained VT.[36] After a mean 10-year follow-up period, 14 (14.3%) of the 98 participants developed coronary events, with a virtually identical prevalence and complexity of VPB in those with and without events. [37] Thus, despite the presence of an increased prevalence and complexity of VPB in asymptomatic elderly, their prognostic significance appears minimal in the absence of clinical heart disease.

Exercise stress testing is also associated with a marked increase in the prevalence and complexity of VPB occurrence with age. In a comparison of apparently healthy BLSA volunteers between the third and ninth decade, isolated VPB during or after maximal treadmill stress testing increased in prevalence from 11% to 57% from the third through the ninth decade.[58] Asymptomatic, exercise-associated runs of nonsustained VT (all 6 beats), were seen in 4% of BLSA volunteers aged 65 or older, a rate 25-fold higher than that of younger persons.[61] Over a two-year mean follow-up period, no study participant with nonsustained VT during exercise developed angina, myocardial infarction, syncope, or sudden death. In this same BLSA population, frequent or repetitive VPB during or after exercise testing were seen predominantly in older individuals, but were similarly unassociated with cardiovascular prognosis over 5.6 years of mean follow-up. [62] However, in a younger French cohort of 6101 men (aged 48 ± 2 years) initially free of clinical cardiovascular disease, those who had frequent VPB during exercise had a 2.5-fold increased risk of cardiovascular death over 23 years of follow-up, independent of standard coronary risk factors.[63]

Although the occasional VPB on resting ECG or during exercise stress testing does not portend a poorer prognosis in apparently healthy individuals, in older patients with coronary disease, the presence of complex VPB is an adverse prognostic indicator. In 467 patients aged 62 – 102 years in a long-term care facility, complex VPB occurred in 21%.[64] In those without a history of coronary disease, the future coronary event rate (4%) was similar to that of the general nursing facility population, while in those with coronary disease and complex VPB, the future event rate was over 11-fold higher (46%). Thus, it is important to ascertain the presence or absence of cardiovascular comorbidities in older persons with VPB.

Sustained ventricular tachycardia and sudden arrhythmic death—Sustained VT is a potentially life-threatening arrhythmia which occurs in two major forms: monomorphic (usually “macro re-entrant” and scar-related) and polymorphic (usually ischemia-related). Sudden arrhythmic death refers to hemodynamically-unstable VT or ventricular fibrillation (VF), and accounts for the majority of all episodes of sudden cardiac death (SCD), a diagnosis which includes hemodynamically-unstable VT, VF, asystole, and non-arrhythmic cardiac causes. Sudden cardiac death is responsible for an estimated 184,000 – 462,000 American deaths yearly, and is a major cause of mortality in the elderly.[65]

The Framingham Heart Study compared the incidence of SCD in women and men across all age groups (n=5209), aged 30 – 62 years at study entry.[66] Women comprised just over half of the study population (n=2873, 55%), with a peak incidence of SCD which lagged that of men by > 10 years. Congestive heart failure increased the risk of SCD by 5-fold in women and 16-fold in men. The incidence of SCD progressively increased with age, especially after age 74 years, reflecting the high prevalence of structural heart disease in the elderly (Figure 4).

SUMMARY

Aging is associated with a myriad of changes in the cardiac conduction system, some of which manifest in association with cardiovascular disease, and others develop as part of normal aging. These changes include sinus node dysfunction, slowing of AV nodal conduction, left axis deviation, bundle branch blocks, and an increased prevalence of both supraventricular and ventricular premature beats and arrhythmias. LBBB, AF, and sustained VT are particularly predictive of future adverse cardiac events, and frequently herald the presence of underlying cardiovascular disease. The prognostic significance of any given conduction abnormality or rhythm disturbance is dependent primarily on the presence and severity of any accompanying cardiac disease. Gaining an appreciation of the epidemiology of cardiac conduction disorders and arrhythmias in the elderly will assist the practitioner in differentiating ECG findings that represent normal aging from those suggesting a disease process requiring further evaluation.

References

1. Anversa P, Palackal T, Sonnenblick EH, et al. Myocyte cell loss and myocyte cellular hyperplasia in the hypertrophied aging rat heart. *Circ Res.* 1990; 67(4):871–85. [PubMed: 2145091]
2. Hiss RG, Lamb LE. Electrocardiographic findings in 122,043 individuals. *Circulation.* 1962; 25:947–61. [PubMed: 13907778]
3. Schwartz J, Gibb WJ, Tran T. Aging effects on heart rate variation. *J Gerontol Med Sci.* 1991; 46(3):M99–106.
4. Byrne EA, Fleg JL, Vaitkevicius PV, et al. Role of aerobic capacity and body mass index in the age-associated decline in heart rate variability. *J Appl Physiol.* 1996; 81(2):743–50. [PubMed: 8872642]
5. Shub C. Stable angina pectoris: 2. Cardiac evaluation and diagnostic testing. *Mayo Clin Proc.* 1990; 65(2):243–55. [PubMed: 2406523]
6. Robergs RA, Landwehr R. The surprising history of the ‘HRmax=220-age’ equation. *JEPonline.* 2002; 5(2):1–10.
7. Tanaka H, Monahan KD, Seals DR. Age-predicted maximal heart rate revisited. *J Am Coll Cardiol.* 2001; 37(1):153–6. [PubMed: 11153730]
8. Nes BM, Janszky I, Wisloff U, et al. Age-predicted maximal heart rate in healthy subjects: the HUNT fitness study. *Scand J Med Sci Sports.* 2012 Feb 29. Epub ahead of print.
9. Adan V, Owen LA. Diagnosis and treatment of sick sinus syndrome. *Am Fam Phys.* 2003; 67:1725–32.
10. Faddis MN, Rich MW. Pacing interventions for falls and syncope in the elderly. *Clin Geriatr Med.* 2002; 18(2):279–94. [PubMed: 12180248]
11. Lamas GA, Prosser AP, Edery TP, et al. Age and sex bias in pacemaker selection. *Circulation.* 1992; 86(Suppl I):I-449.
12. Gregoratos G. Permanent pacemakers in older persons. *J Am Geriatr Soc.* 1999; 47(9):1125–35. [PubMed: 10484258]
13. Tresch DD, Fleg JL. Unexplained sinus bradycardia: clinical significance and long-term prognosis in apparently healthy persons older than 40 years. *Am J Cardiol.* 1986; 58(10):1009–13. [PubMed: 3490781]
14. Kannel WB, Kannel C, Paffenbarger RS Jr, et al. Heart rate and cardiovascular mortality: The Framingham Study. *Am Heart J.* 1987; 113(6):1489–94. [PubMed: 3591616]
15. Mason JW, Ramseth DJ, Chanter DO, et al. Electrocardiographic reference ranges derived from 79,743 ambulatory subjects. *J Electrocardiol.* 2007; 40(3):228–34. [PubMed: 17276451]
16. Fleg JL, Das DN, Wright J, et al. Age-associated changes in the components of atrioventricular conduction in apparently healthy volunteers. *J Gerontol.* 1990; 45(3):M95–100. [PubMed: 2335725]
17. Golden GS, Golden LH. The “nona” electrocardiogram: findings in 100 patients of the 90 age group. *J Am Geriatr Soc.* 1974; 22(7):329–31. [PubMed: 4135242]

18. Sox HC Jr, Garber AM, Littenberg B. The resting electrocardiogram as a screening test. A clinical analysis. *Annals Intern Med.* 1989; 111:489–502.
19. Gorodeski EZ, Ishwaran H, Blackstone EH, Lauer MS. Quantitative electrocardiographic measures and long-term mortality in exercise test patients with clinically normal electrocardiograms. *Am Heart J.* 2009; 158:61–70.e1. [PubMed: 19540393]
20. Simonson E. The effect of age on the electrocardiogram. *Am J Cardiol.* 1972; 29(1):64–73.
21. Mymin D, Mathewson FA, Tate RB, et al. The natural history of primary first-degree atrioventricular heart block. *N Engl J Med.* 1986; 315(19):1183–7. [PubMed: 3762641]
22. Rodstein M, Brown M, Wolloch L. First-degree atrioventricular heart block in the aged. *Geriatrics.* 1968; 23(10):159–65. [PubMed: 5676498]
23. Cheng S, Keyes MJ, Larson MG, et al. Long-term outcomes in individuals with prolonged PR interval or first-degree atrioventricular block. *JAMA.* 2009; 301(24):2571–7. [PubMed: 19549974]
24. Crisel RK, Farzaneh-Far R, Na B, et al. First-degree atrioventricular block is associated with heart failure and death in persons with stable coronary artery disease: data from the Heart and Soul Study. *Eur Heart J.* 2011; 32(15):1875–80. [PubMed: 21606074]
25. DePaula RS, Antelmi I, Vincenzi MA, et al. Cardiac arrhythmias and atrioventricular block in a cohort of asymptomatic individuals without heart disease. *Cardiology.* 2007; 108(2):111–6. [PubMed: 17008799]
26. Shaw DB, Gowers JI, Kekwick CA, et al. Is Mobitz type I atrioventricular block benign in adults? *Heart.* 2004; 90(2):169–74. [PubMed: 14729789]
27. Parsonnet V, Bernstein AD, Galasso D. Cardiac pacing practices in the United States in 1985. *Am J Cardiol.* 1988; 62(1):71–7. [PubMed: 3132848]
28. Kojic EM, Hardarson T, Sigfusson N, et al. The prevalence and prognosis of third-degree atrioventricular conduction block: the Reykjavik study. *J Intern Med.* 1999; 246(1):81–6. [PubMed: 10447229]
29. Mond HG, Proclemer A. The 11th world survey of cardiac pacing and implantable cardioverter-defibrillators: calendar year 2009 – a World Society of Arrhythmia's project. *Pacing Clin Electrophysiol.* 2011; 34(8):1013–27.
30. Schneider JF, Thomas HE, Kreger BE, et al. Newly acquired right bundle-branch block: The Framingham Study. *Ann Intern Med.* 1980; 92(1):37–44. [PubMed: 7350871]
31. Fleg JL, Das DN, Lakatta EG. Right bundle branch block: long-term prognosis in apparently healthy men. *J Am Coll Cardiol.* 1983; 1(3):887–92. [PubMed: 6826977]
32. Thrainsdottir IS, Hardarson T, Thorgeirsson G, Sigvaldason H, Sigfusson N. The epidemiology of right bundle branch block and its association with cardiovascular morbidity-The Reykjavik Study. *Eur Heart J.* 1993; 14:1590–96. [PubMed: 8131755]
33. Schneider JF, Thomas HE Jr, Kreger BE, et al. Newly acquired left bundle-branch block: the Framingham study. *Ann Intern Med.* 1979; 90(3):303–10. [PubMed: 154870]
34. Fahy GJ, Pinski SL, Miller DP, et al. Natural history of isolated bundle branch block. *Am J Cardiol.* 1996; 77(14):1185–90. [PubMed: 8651093]
35. Manolio TA, Furberg CD, Rautaharju PM, et al. Cardiac arrhythmias on 24-hour ambulatory electrocardiography in older women and men: The Cardiovascular Health Study. *J Am Coll Cardiol.* 1994; 23(4):916–25. [PubMed: 8106697]
36. Fleg JL, Kennedy HL. Cardiac arrhythmias in a healthy elderly population: detection by 24-hour ambulatory electrocardiography. *Chest.* 1982; 81(3):302–7. [PubMed: 7056104]
37. Fleg JL, Kennedy HL. Long-term prognostic significance of ambulatory electrocardiographic findings in apparently healthy subjects 60 years of age. *Am J Cardiol.* 1992; 70(7):748–51. [PubMed: 1381549]
38. Lakshminarayan K, Solid CA, Collins AJ, Anderson DC, Herzog CA. Atrial fibrillation and stroke in the general Medicare population. A 10-year perspective (1992 to 2002). *Stroke.* 2006; 37:1969–74. [PubMed: 16809573]
39. Feinberg WM, Blackshear JL, Laupacis A, et al. Prevalence, age distribution, and gender of patients with atrial fibrillation. Analysis and implications. *Arch Intern Med.* 1995; 155(3):469–73. [PubMed: 7864703]

40. Wolf PA, Benjamin EJ, Belanger AJ, et al. Secular trends in the prevalence of atrial fibrillation: the Framingham Study. *Am Heart J*. 1996; 131(4):790–5. [PubMed: 8721656]
41. Lake RR, Cullen KJ, deKlerk NH, et al. Atrial fibrillation in an elderly population. *Aust N Z J Med*. 1989; 19(4):321–6. [PubMed: 2789508]
42. Phillips SJ, Whisnant J, O'Fallon WM, et al. Prevalence of cardiovascular disease and diabetes in residents of Rochester, Minnesota. *Mayo Clin Proc*. 1990; 65(3):344–59. [PubMed: 2248634]
43. Furberg CD, Psaty BM, Manolio TA, et al. Prevalence of atrial fibrillation in elderly subjects: the Cardiovascular Health Study. *Am J Cardiol*. 1994; 74(3):238–41.
44. Hernandez MB, Asher CR, Hernandez AV, et al. African American race and prevalence of atrial fibrillation: a meta-analysis. *Cardiol Res Pract*. 2012; 2012:275624. [PubMed: 22548197]
45. Schnabel RB. Can we predict the occurrence of atrial fibrillation? *Clin Cardiol*. 2012; 35 (Suppl 1): 5–9. [PubMed: 22246951]
46. Psaty BM, Manolio TA, Kuller LH, et al. Incidence of and risk factors for atrial fibrillation in older adults. *Circulation*. 1997; 96:2455–61. [PubMed: 9337224]
47. Tsang TS, Gersh BJ, Appleton CP, et al. Left ventricular diastolic dysfunction as a predictor of the first diagnosed nonvalvular atrial fibrillation in 840 elderly men and women. *J Am Coll Cardiol*. 2002; 40(9):1636–44. [PubMed: 12427417]
48. Brand FN, Abbott RD, Kannel WB, et al. Characteristics and prognosis of lone atrial fibrillation 30-Year follow-up in the Framingham Study. *JAMA*. 1985; 254(24):3449–49. [PubMed: 4068186]
49. Wolf PA, Abbott RD, Kannel WB. Atrial fibrillation as an independent risk factor for stroke: the Framingham Study. *Stroke*. 1991; 22(8):983–8. [PubMed: 1866765]
50. Kitzman DW, Gardin JM, Gottdiener JS, et al. Importance of heart failure with preserved systolic function in patients 65 years of age. *Am J Cardiol*. 2001; 87:413–9. [PubMed: 11179524]
51. Mathew JP, Fontes ML, Tudor JC, et al. A multcenter risk index for atrial fibrillation after cardiac surgery. *JAMA*. 2004; 291:1720–9. [PubMed: 15082699]
52. Amar D, Zhang H, Leung DH, et al. Older age is the strongest predictor of postoperative atrial fibrillation. *Anesthesiology*. 2002; 96(2):352–6.
53. Healey JS, Connolly SJ, Gold MR, et al. Subclinical atrial fibrillation and the risk of stroke. *New Engl J Med*. 2012; 366(2):120–9. [PubMed: 22236222]
54. Mareedu RK, Abdalrahman IB, Dharmashankar KC, et al. Atrial flutter versus atrial fibrillation in a general population: differences in comorbidities associated with their respective onset. *Clin Med Res*. 2010; 8(1):1–6. [PubMed: 19920163]
55. Orejarena LA, Vidaillet H Jr, DeStefano F, et al. Paroxysmal supraventricular tachycardia in the general population. *J Am Coll Cardiol*. 1998; 31(1):150–7. [PubMed: 9426034]
56. Maurer MS, Shefrin EA, Fleg JL. Prevalence and prognostic significance of exercise-induced supraventricular tachycardia in apparently healthy volunteers. *Am J Cardiol*. 1995; 75(12):788–92. [PubMed: 7717280]
57. Fisch C. Electrocardiogram in the aged. An independent marker of heart disease? *Am J Med*. 1981; 70(1):4–6. [PubMed: 7457490]
58. Fleg, JL. Epidemiology of ventricular arrhythmias in the elderly. In: Paciaroni, E., editor. *Proceedings of the 13th National Congress of Cardiology (Aging and Cardiac Arrhythmias)* I.N.R.C.A.; Ancona: Istituto a Carattere Scientifico; 1991. p. 26-30.
59. Kantelip JP, Sage E, Duchene-Marullaz P. Findings on ambulatory electrocardiologic monitoring in subjects older than 80 years. *Am J Cardiol*. 1986; 57(6):398–401. [PubMed: 3946253]
60. Camm AJ, Evans KE, Ward DE, et al. The rhythm of the heart in active elderly subjects. *Am Heart J*. 1981; 99(5):598–603. [PubMed: 7369099]
61. Fleg JL, Lakatta EG. Prevalence and prognosis of exercise-induced nonsustained ventricular tachycardia in apparently healthy volunteers. *Am J Cardiol*. 1984; 54(7):762–4. [PubMed: 6486025]
62. Busby MJ, Shefrin E, Fleg JL. Prevalence and long-term prognostic significance of exercise-induced frequent or repetitive ventricular ectopic beats in apparently healthy volunteers. *J Am Coll Cardiol*. 1989; 14:1659–65. [PubMed: 2479667]

63. Jouven X, Zureik M, Desnos M, et al. Long-term outcome in asymptomatic men with exercise-induced premature ventricular depolarizations. *N Engl J Med.* 2000; 343(12):826–33. [PubMed: 10995861]
64. Aronow WS, Epstein S, Koenigsberg M, et al. Usefulness of echocardiographic abnormal left ventricular ejection fraction, paroxysmal ventricular tachycardia, and complex ventricular arrhythmias in predicting new coronary events in patients over 62 years of age. *Am J Cardiol.* 1988; 61(15):1349–51. [PubMed: 3376895]
65. Goldberger J, Cain M, Hohnloser S, et al. American Heart Association/American College of Cardiology Foundation/Heart Rhythm Society scientific statement on noninvasive risk stratification techniques for identifying patients at risk for sudden cardiac death: a scientific statement from the American Heart Association Council on Clinical Cardiology Committee on Electrocardiography and Arrhythmias and Council on Epidemiology and Prevention. *Circulation.* 2008; 118(14):1497–1518. [PubMed: 18833586]
66. Kannel WB, Wilson PW, D'Agostino RB, et al. Sudden coronary death in women. *Am Heart J.* 1998; 136(2):205–12. [PubMed: 9704680]

Key Points

1. Changes in the cardiac conduction system include sinus node dysfunction, slowing of AV nodal conduction, left axis deviation, bundle branch blocks, and an increased prevalence of both supraventricular and ventricular premature beats and arrhythmias. LBBB, AF, and sustained VT are particularly predictive of future adverse cardiac events, and frequently herald the presence of underlying cardiovascular disease.
2. The prognostic significance of any given conduction abnormality or rhythm disturbance is dependent primarily on the presence and severity of any accompanying cardiac disease.
3. Gaining an appreciation of the epidemiology of cardiac conduction disorders and arrhythmias in the elderly will assist the practitioner in differentiating ECG findings that represent normal aging from those suggesting a disease process requiring further evaluation.

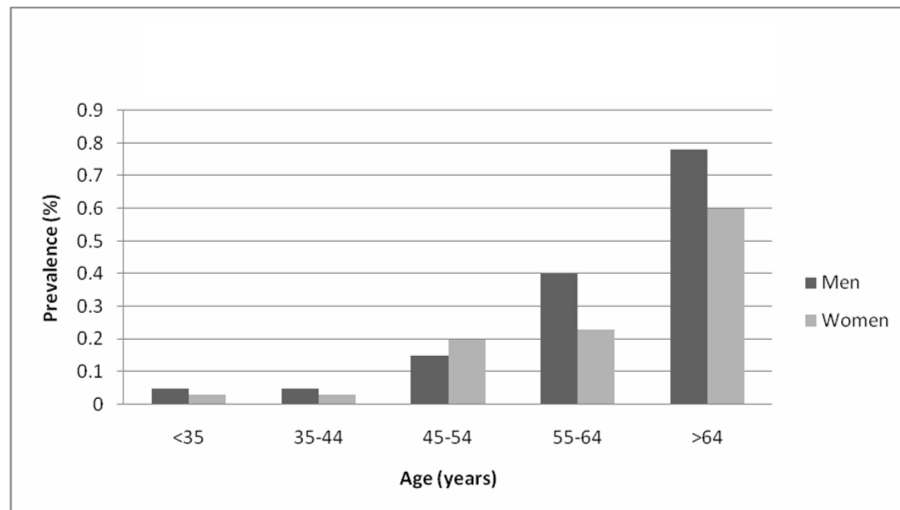


Figure 1. Prevalence of LBBB in the Irish Heart Foundation study, according to age. The incidence and prevalence of LBBB increases with age, with highest levels seen in the elderly cohort. LBBB, left bundle branch block. Adapted from Fahy et al., 1996 [34]

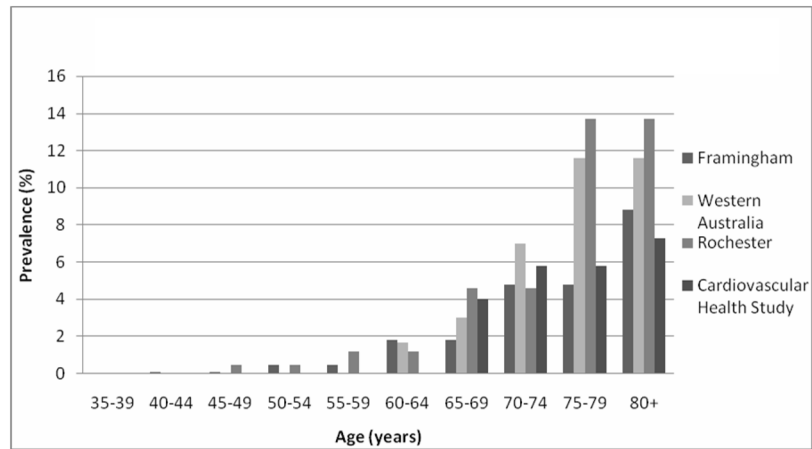


Figure 2. Prevalence of atrial fibrillation in four population-based surveys. The incidence and prevalence of atrial fibrillation increase with age, with highest rates seen in the elderly. Adapted from Feinberg et al., 1995 [39]

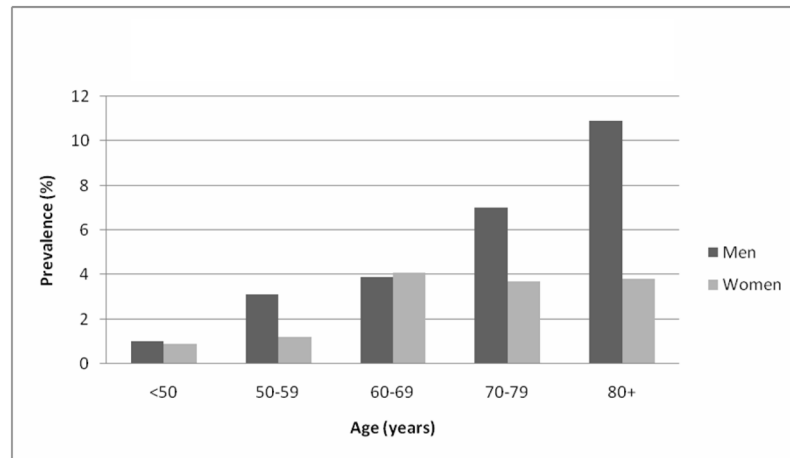


Figure 3.

Prevalence of exercise-associated PSVT in the BLSA, stratified by age group. The prevalence of PSVT increased steadily in men, whereas a plateau was seen in women after the age of 60 years. PSVT, paroxysmal supraventricular tachycardia; BLSA, Baltimore Longitudinal Study of Aging. Adapted from Maurer et al., 1995 [56]

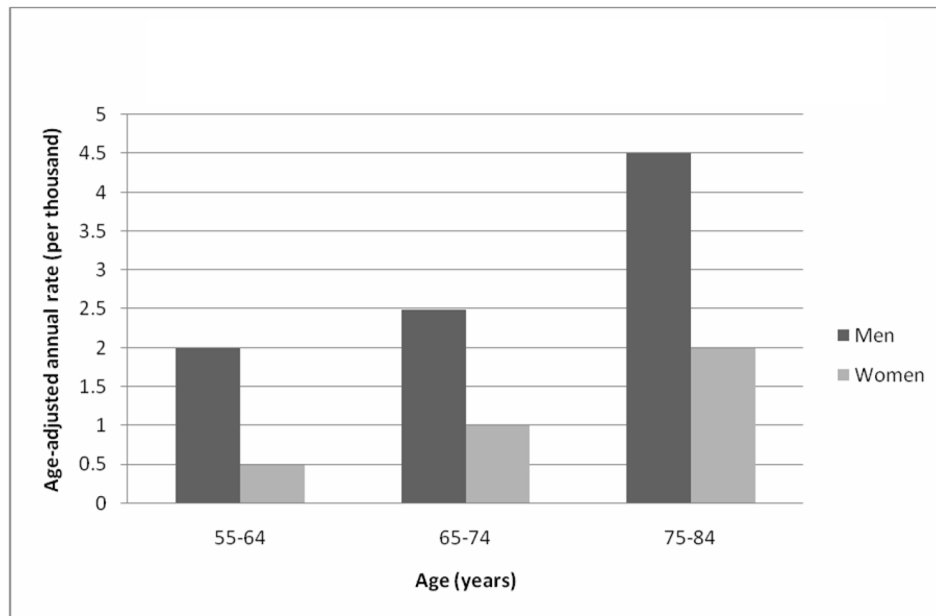


Figure 4. Incidence of sudden death in the Framingham study, grouped by age and sex. Incidence of sudden death progressively increased with increasing age, affecting men more frequently than women in each age group. Adapted from Kannel et al., 1998 [66]

Overall Framingham deaths by resting heart rate in the elderly, according to sex. A trend of increasing overall mortality was seen with stratification of the study population by resting heart rate. Higher heart rates were associated with a higher age-adjusted annual mortality rate in both men and women. All trends significant at $p<0.01$. Adapted from Kannel et al., 1987.[14]

Table 1

Resting heart rate (bpm)	Population at risk (person-years)		Age-adjusted annual mortality rate (per 1000)	
	Men	Women	Men	Women
30–67	3660	3560	35	22
68–75	3646	6068	43	28
76–83	2172	4208	46	25
84–91	1572	3270	61	30
>91	1408	3264	64	35

Survival of patients with Mobitz I second degree heart block by age group, with and without a pacemaker. Patients between 45 and 79 years who received a pacemaker for the indications of symptomatic Mobitz I or subsequent high-degree AV block had a significant survival advantage over those who did not.

Table 2

Variable	Age group (years)								All patients	
	20 – 44		45 – 64		65 – 79		80+		Paced	Unpaced
Subjects (n)	Paced	Unpaced	Paced	Unpaced	Paced	Unpaced	Paced	Unpaced	Paced	Unpaced
	5	10	18	7	52	18	15	22	90	57
Mean age (years)	34	32.6	57.1	58.1	72.9	72.2	85	85.9	69.6	68.8
5-year survival %, (SD)	100 (0)	100 (0)	94.4 (5.4)	57.1 (18.7)	76.6 (5.9)	50 (11.8)	45.9 (13)	33.4 (10.3)	76.3 (4.5)	53.5 (6.7)
p-value	1		0.001*		0.003*		0.76*		0.0014*	

* Log rank test (age imbalances were sufficiently small to be negligible). Adapted from Shaw et al., 2004.[26]