Typical versus atypical Wenckebach periodicity

Atrioventricular blocks classification criteria

Concept: dromotropic disorders located at any level of the sino-ventricular conduction system by conduction "slowing" in the atria (PA interval), AV node (AH interval), His bundle and its branches (HV interval) or association of the former.

Classification criteria

I) **By degree**

- 1st degree AV block;
- 2nd degree AV block:
  - Mobitz type I or Wenckebach
  - Mobitz type II
- Fixed 2:1 AV block;
- High-degree or advanced AV block;
- 3rd degree, complete or total AV block.

II) **By topography related to the His bundle**

- **Supra-Hisian or pre-Hisian**: may extend PA and/or AH intervals: conduction slowing in the atria (PA) and/or AV node (AH).
- **Hisian and infra-Hisian**.
  - Hisian: His bundle.
  - Infra-hisian, fascicular or divisional: branches and divisions.
- **Mixed**: they affect the PA, AH and HV intervals.

Etiologies of the atrioventricular blocks

They may be:

1) **Physiological**: vagotony: functional block, e.g.: post-left carotid sinus massage, valsalva maneuver, vomiting, etc. **Athletic training:** Well-trained athletes can demonstrate first-degree (and occasionally higher degree) AV block owing to an increase in vagal tone. First-degree AV block is observed in 5% to 30% of cases in athletes (in non-athletes, 0.65%) (**McClaskey D1, Lee D, Buch E.Outcomes among athletes with arrhythmias and electrocardiographic abnormalities: implications for ECG interpretation.Sports Med. 2013 Oct;43(10):979-91. doi: 10.1007/s40279-013-0074-5**).

2) **Pathological**.

- **Congenital**

- **Atrial Septal Defect (ASD)** of the familial ostium secundum type (ASD-OS) and Holt-Oran syndrome (it extends A-H interval). Mutations in the TBX5 gene cause Holt-Oram syndrome. This gene provides instructions for making a protein that plays a role in the development of the heart and upper limbs before birth. In particular, this gene appears to be important for the process of cardiac septation. The TBX5 gene also appears to play a critical role in regulating the development of bones in the arm and hand. Holt-Oram syndrome is estimated to affect 1 in 100,000 individuals.
- **Endocardial cushion defects:** partial and total.
- **Ebstein's anomaly:** PR interval prolongation in 20% of the cases by Neuropraxis phenomenon on the AV node, right ventricular hypertension and inferior implantation of septal fascicle of tricuspid valve. Electrophysiological studies proved the possible prolongation of the HV interval, which indicates that PR interval prolongation may be secondary to intraventricular dromotropic disorder.
- **Corrected Transposition of the Great Vessels of the Base:** in 75% of patients we find different degrees of AV block, from the 1st degree to total AV block.

- **Acquired**
  - By drugs (especially those drugs that increase the refractory time of the AVN, thereby slowing conduction): Digoxin or other cardiac glycosides, vagotonia, prostigmin (they extend the AH interval), antihypertensive agents (alpha-methyldopa), magnesium.
  - By antiarrhythmic use:
    - Of Class IA (quinidine, procainamide and disopyramide).
    - Of Class IC (propafenone, flecainide, encainide): they may cause significant increase of PR interval as a consequence of AH interval and HV interval prolongation.
    - Of Class III (amiodarone, sotalol, dofetilide, ibutilide): they extend HV interval.
    - Of Class II (β-blockers).
  - By coronary artery disease:

- Acute myocardial infarction: present in 8.5% of the cases of acute coronary insufficiency, independent from the site involved:
  - ✓ Acute inferior myocardial infarction: more frequently consequence of RCA obstruction.
  - ✓ Acute anterior myocardial infarction: involvement of anterior descending coronary artery. It usually causes PR interval shortening; nevertheless, it may extend the PR interval.
- Chronic phase of myocardial infarction.

➢ **Idiopathic degenerative diseases of the conduction system or Lev disease** is due to progressive degenerative fibrosis and calcification of the neighboring cardiac structures, or "sclerosis of the left side of cardiac skeleton" (including the mitral annulus, central fibrous body, membranous septum, base of the aorta, and crest of the ventricular septum). Lev disease starts in about the fourth decade of life and is believed to be secondary to wear and tear on these structures caused by the pull of the left ventricular musculature. It affects the proximal bundle branches and is manifested by bradycardia and varying degrees of AV block.

➢ **Lenègre disease.** This is a progressive impairment of cardiac conduction system observed in atrioventricular block in young or middle-aged adults with genetic background allelic with Brugada syndrome. The most common phenotype of gene carriers of a BrS-type SCN5A mutation is progressive cardiac conduction defects similar to the Lenègre disease phenotype. In consequence, carriers of a SCN5A mutation need a clinical and ECG follow-up because of the risk associated with severe conduction defects **(Probst V1, Allouis M, Sacher F, Pattier S, Babuty D, Mabo P, Mansourati J, Victor J, Nguyen JM, Schott JJ, Boisseau P, Escande D, Le Marec H.Progressive cardiac conduction defect is the prevailing phenotype in carriers of a Brugada syndrome SCN5A mutation.J Cardiovasc Electrophysiol. 2006 Mar;17(3):270-5. PMID: 16643399 DOI: 10.1111/j.1540-8167.2006.00349.x).**

➢ **Mitral or aortic valve annulus calcification.** The main penetrating bundle of His is located near the base of the anterior leaflet of the mitral valve and the noncoronary cusp of the aortic valve. Heavy calcium

deposits in patients with aortic or mitral annular calcification is associated with increased risk of AV block. Other valve diseases, especially tricuspid stenosis by compression of AV node and calcified aortic valve stenosis.

- **By myocarditis:**
  - **Bacterial:** rheumatic fever (it constitutes a minor sign of Jones), diphtheria (acute diphtheric carditis), lues, Lyme disease, tuberculosis.
  - **Viral:** Coxsackies A and B (especially Serotype B4), Echo, Cytomegalovirus, etc.
  - **Parasitic:** the paradigm is chronic chagasic cardiomyopathy (it may extend the PR interval by increase of PA, AH, and HV intervals of Hisian electrogram (HE)), trichinosis. Infective endocarditis, diphtheria, rheumatic fever, Chagas disease, Lyme disease, and tuberculosis all may be associated with first-degree AV block. Extension of the infection to the adjacent myocardium in native or prosthetic valve infective endocarditis (i.e., ring abscess) can cause AV block. Acute myocarditis caused by diphtheria, rheumatic fever, or Chagas disease can result in AV block.
- **Electrolyte disturbances (eg, hypokalemia, hypomagnesemia)**

Second-degree atrioventricular (AV) block, or second-degree heart block,

It is characterized by disturbance, delay, or interruption of atrial impulse conduction through the AV node to the ventricles. The term is applied when one or more (but not all) atrial impulses that should be conducted fail to reach the ventricles. Although patients with second-degree AV block may be asymptomatic, Mobitz type I (Wenckebach) AV block can cause significant symptoms, and Mobitz type II block may progress to complete heart block, with an associated increased risk of mortality.

Etiologies

- Cardioactive drugs are an important cause of AV block **(Cho 2010; Antoniou 2005; Neumar 2010).** They may exert negative (i.e., dromotropic) effects on the AVN directly, indirectly via the autonomic nervous system, or both. Digoxin,

beta-blockers, calcium channel blockers, and certain antiarrhythmic drugs have been implicated in second-degree AV block.

- Of the antiarrhythmic medications that may cause second-degree AV block, sodium channel blockers, such as procainamide, cause more distal block in the His-Purkinje system. Persistent second-degree AV block following adenosine infusion for nuclear stress testing has been reported **(Makaryus 2008).** The AV block may not resolve in many of the patients who take cardioactive medications. This suggests an underlying conduction disturbance in addition to the medications as the etiology of the AV block. At toxic levels, other pharmacologic agents, such as lithium, may be associated with AV block. Benzathine penicillin has been associated with second-degree AV block (**Belém 2009**). Presynaptic alpha agonists (eg, clonidine) may rarely be associated with, or exacerbate, AV block.
- Various inflammatory, infiltrative, metabolic, endocrine, and collagen vascular disorders have been associated with AVN block, as follows.Inflammatory diseases - endocarditis, myocarditis, Lyme disease (Haddad 2003), acute rheumatic fever.
- Infiltrative diseases - amyloidosis, hemochromatosis, sarcoidosis (AV conduction abnormalities can be the first sign of sarcoidosis) (**Van Herendael 2007**).
- Infiltrative malignancies, such as Hodgkin's lymphoma and other lymphomas, and multiple myeloma (**Lev 1964**).
- Metabolic and endocrine disorders – hyperkalemia, hypermagnesemia, Addison's disease, hyperthyroidism, myxedema, thyrotoxic periodic paralysis (**Hsu 2003**).
- Collagen vascular diseases - ankylosing spondylitis, dermatomyositis, rheumatoid arthritis, scleroderma, lupus erythematosus, Reiter syndrome, mixed connective tissue disease (**Vinsonneau 2005**).
- Other conditions or procedures associated with AV block are as follows.
- Cardiac tumors.
- Trauma (including catheter-related, especially in the setting of preexisting left bundle-branch block).
- Following transcatheter valve replacement.
- Myocardial bridging (**den Dulk 1983**).
- Ethanol septal reduction (also called transcoronary ablation of septal hypertrophy for the treatment of obstructive hypertrophic cardiomyopathy).
- Transcatheter closure of atrial and ventricular septal defects (**Lin 2007**; **Thanopoulos 2005**).

- Corrective congenital heart surgery, especially those near the septum.
- Progressive (age-related) idiopathic fibrosis of the cardiac skeleton
- Valvular heart disease complications, especially aortic stenosis and aortic valve replacement surgery
- Following some catheter ablation procedures
- Obstructive sleep apnea (**Arias 2007**)
- Muscular dystrophies
- Acute ethanol poisoning
- Acute myocardial infarction (MI)
- Any cardiac tumor has the potential for affecting the AVN if it is in close anatomic relation with the node. Myxoma is the most common primary cardiac tumor, but a variety of secondary tumors may also be found in the heart. Cho et al reported a patient with primary cardiac lymphoma who presented with unexplained dyspnea and a progressive AV block (**Cho 2010**).
- Erkapic and colleagues studied the incidence of AV block after transcatheter aortic valve replacement and found that up to 34% of patients (mean age, 80 ± 6 years) experienced second- and third-degree AV block, mainly within the first 24 hours of the procedure (**Erkapic 2010**). They did not observe any improvement in the AV block within the next 14 days, and most of these patients required permanent pacemaker implantation. In this report, preoperative right bundle-branch block and CoreValve prosthesis were associated with higher rate of AV block and subsequent pacemaker implantation (**Erkapic 2010**). On the basis of this report, the rate of postoperative AV block seems significantly higher in transcatheter valve replacement than a traditional surgical approach. Nardi and colleagues reported pacemaker implantation in only 3% of patients undergoing isolated aortic valve replacement (**Nardi 2010**). Nevertheless, patients who undergo transcatheter valve replacement are much sicker and older than those who undergo a traditional surgical valve replacement (80 ± 6 years in the Erkapic study compared with 69 ± 12 years in the Nardi study).
- **Catheter ablation of any structure close to the AVN** can be associated with AV block as an adverse effect of this procedure. In particular, AV block may be seen following ablation for AV nodal reentrant tachycardia (AVNRT) and some accessory pathways. Bastani and colleagues suggest that cryoablation of

superoparaseptal and septal accessory pathways may be a safer alternative to radiofrequency ablation in this regard (**Bastani 2010**).

- The conduction defects in patients with muscular dystrophy are progressive; therefore, these patients should undergo careful workup and follow-up, even if they present with a benign conduction defect such as first-degree AV block (**Sovari 2007**).
- Acute ethanol poisoning has been reported to be associated with transient first-degree AV block; however, a few case reports have shown occasional association with Mobitz I AV block and high-degree AV block (**Brvar 2009**).
- **Genetic factors**: In some patients, AV block may be an autosomal dominant trait and a familial disease. Several mutations in the *SCN5A* gene have been linked to familial AV block. Different mutations in the same gene have been reported in other dysrhythmias such as long QT syndrome (LQTS) and Brugada syndrome. In LQTS, a pseudo 2:1 AV block may be seen as a result of a very prolonged ventricular refractory period. Nevertheless, a true 2:1 AV block with possible primary pathology in the AVN and conduction system has also been reported in LQTS (**Cevik 2010**). Lenegre–Lev disease or Lev syndrome was initially described as an acquired complete atrial-ventricular (AV) block with RBBB or LBBB (**Carius BM1, Long B2, Schauer S2.Lev's Syndrome: A rare case of progressive cardiac conduction disorder presenting to the emergency department.Am J Emerg Med. 2019 May;37(5):1006.e1-1006.e4. doi: 10.1016/j.ajem.2019.01.0**) The disease is secondary to idiopathic fibrosis of the heart electrical conduction system and may cause syncope and sudden death. Schott et al. reported the first mutation in the SCN5A gene that segregated with progressive conduction defect (PCCD) in an autosomal-dominant manner in a large French family and a second SCN5A mutation that co-segregated in a smaller Dutch family with familial nonprogressive conduction defect.(Schott, J.-J., Alshinawi, C., Kyndt, F., Probst, V., Hoorntje, T. M., Hulsbeek, M., … Le Marec, H. (1999). *Cardiac conduction defects associate with mutations in SCN5A. Nature Genetics, 23(1), 20–21.* doi:10.1038/12618) Fifteen patients from the French family were clinically and electrocardiographically affected (the mean QRS duration was 135 ± 7 ms). RBBB was present in five patients, LBBB in two, LAFB or LPFB in three, and long PR interval (>210ms) in eight. None of the patients had structural heart disease. Four patients received a pacemaker implantation because

of syncope or complete AV block, and in a number of affected patients, the conduction defect increased in severity with age. On the other hand, in the Dutch family, the proband presented after birth with an asymptomatic first-degree AV block associated with RBBB. Three brothers were asymptomatic, one of whom had RBBB, and the asymptomatic mother had a nonspecific conduction defect with a QRS duration of 120 ms. By use of markers flanking SCN5A in the French family, these investigators demonstrated segregation of the disease with marker D3S1260 in every affected individual, and analyses with flanking markers of the region confirmed a linkage to the 3p21 locus. Sequencing the entire SCN5A coding region in this family identified a T→C substitution in the highly conserved +2 donor-splicing site of intron 22. This abnormal transcript predicts an in-frame skipping of exon 22 and an impaired gene product lacking the voltage-sensitive domain III S4 segment. This mutation was found in all affected members, but not in 100 control chromosomes. In the Dutch family, sequence analysis of the SNC5A gene revealed a deletion of a single nucleotide (G) at position 5280, resulting in a frameshift and a premature stop codon. This mutation co-segregated with the phenotype in all affected family members. These findings also indicated that with aging there is a progressive increase in cardiac fibrosis, which, in association with the SNC5A gene mutation, can slow the impulse along the electrical conduction system. In the Dutch family, the mutation conferring a premature stop codon and the presentation of PCCD at birth suggests that as a consequence of the sodium channel mutation a congenital phenotype can arise that may be either progressive or immediate. It is worth noting that none of the affected individuals had LQTS or BrS, although heterozygous mutations in the cardiac SCN5A gene have been associated with LQT3, BrS, and PCD. The same mutation in SCN5A can lead either to BrS or to an isolated cardiac conduction defect.**(Kyndt F1, Probst V, Potet F, Demolombe S, Chevallier JC, Baro I, Moisan JP, Boisseau P, Schott JJ, Escande D, Le Marec H.Novel SCN5A mutation leading either to isolated cardiac conduction defect or Brugada syndrome in a large French family.Circulation. 2001 Dec 18;104(25):3081-6**.) In a large family with both BrS and isolated cardiac conduction defects, a G-to-T mutation at position 4372 was found in 13 affected members and was predicted to change a glycine for an arginine (G1406R) between the S5 and S6 segments of domain III of the Na+ channel protein. Four individuals showed typical BrS phenotypes, including ST-

segment elevation in the right precordial leads and RBBB, and seven individuals had isolated cardiac conduction defects but no BrS phenotype; one patient with an isolated cardiac conduction defect (CDD) had an episode of syncope and required pacemaker implantation. These findings suggest that modifier gene(s) may influence the phenotypic consequences of a SCN5A mutation.Often a mutant cardiac sodium channel may be associated with multiple biophysical defects and concomitant clinical features of BrS and CCD. For example, LQT3, which is caused by mutations in the human cardiac SCN5A gene, may present, in addition to LQT, with bradycardia and sinus pauses. Veldkamp et al. reported the effect of the 1795insD Na+ channel mutation (previously characterized by the presence of a persistent inward current (Ipst) at −20 mV and a negative shift in voltage dependence of inactivation) on sinoatrial (SA) pacemaking.131 By use of functional studies, Ipst was characterized over the complete voltage range of the SA node AP by measuring whole-cell Na+ currents (INa) in HEK-293 cells expressing either wild-type or 1795insD channels. I for 1795insD channels varied between 0.8 ± 0.2% and 1.9 ± 0.8% of peak INa, and the activity of 1795insD channels during SA node pacemaking was confirmed by AP clamp experiments. When implemented into SA node AP models, the negative shift decreased sinus rate by decreasing diastolic depolarization rate, whereas Ipst decreased sinus rate by AP prolongation(**Veldkamp MW1, Wilders R, Baartscheer A, Zegers JG, Bezzina CR, Wilde AA.Contribution of sodium channel mutations to bradycardia and sinus node dysfunction in LQT3 families.Circ Res. 2003 May 16;92(9):976-83**.) A novel Na+ channel mutation in SCN5A, E161K, has been identified in individuals of two nonrelated families with symptoms of bradycardia, SND, generalized conduction disease, and BrS, or combinations thereof.(**Smits JP1, Koopmann TT, Wilders R, Veldkamp MW, Opthof T, Bhuiyan ZA, Mannens MM, Balser JR, Tan HL, Bezzina CR, Wilde AA.A mutation in the human cardiac sodium channel (E161K) contributes to sick sinus syndrome, conduction disease and Brugada syndrome in two families. J Mol Cell Cardiol. 2005 Jun;38(6):969-81. DOI: 10.1016/j.yjmcc.2005.02.024**) Functional studies of mutant Na+ channels were performed with wild-type or E161K Na+ channel α-subunit and β-subunit co-transfected into tsA201 cells. Whole-cell sodium current (INa) gauged using whole cell patch-clamp technique from cells containing the E161K mutation exhibited an almost threefold reduction in current

density and an 11.9-mV positive shift of the voltage-dependence of activation, whereas the inactivation properties of wild-type and mutant Na+ channels were similar. These data suggested an overall reduction of INa in E161K mutants. Computational models demonstrated a marked atrial and ventricular conduction slowing, as well as a reduction in sinus rate stemming from slowing of the diastolic depolarization rate and upstroke velocity of the sinus node AP. This reduction in sinus rate was further aggravated by application of acetylcholine, simulating the dominant vagal tone during the night.

Gene defects in Cardiac Conduction Defects (CCD)

Disease	**Chromos , locus**	**Gene**	**Protein**	**Mutation**	**References**
Progressive cardiac conduction defect (PCCD)/ Isolated cardiac conduction defect.	3p21	**SCN5A**			**Schott JJ 1999**
CCD	3p21–24	*SCN5A*	hNavl.5	E161K	**Smits JP1 2005**
CCD/Brugada	3p21–24	*SCN5A*	hNavl.5	G1406R	**Kyndt F 2001**
CCD/AV block	3p21–24	*SCN5A*	hNavl.5	G298S	**Wang DW 2002**
CCD/AV block	3p21–24	*SCN5A*	hNavl.5	D1595N	**Wang DW 2002**
Sick sinus syndrome	15q24–23	*HCN4*	HCN4–573X	1631delC	
	p21–24	*SCN5A*	hNavl.5	E161K	
	3p21–24	*SCN5A*	hNavl.5	Multiple mutant alleles	
	19q13.2-q13.3				**Brink PA 1995**

Familial sinus node disfunction (SND) and atrioventricular conduction dysfunction	7q21.1-q31.1	*GNB2*	the G-protein-activated K^+ channel (GIRK; Kir3.1/Kir3.4)		**Stallmeyer B 2017**

- Schott, J.-J., Alshinawi, C., Kyndt, F., Probst, V., Hoorntje, T. M., Hulsbeek, M., … Le Marec, H. (1999). *Cardiac conduction defects associate with mutations in SCN5A. Nature Genetics, 23(1), 20–21.* doi:10.1038/12618
- **Smits JP1, Koopmann TT, Wilders R, Veldkamp MW, Opthof T, Bhuiyan ZA, Mannens MM, Balser JR, Tan HL, Bezzina CR, Wilde AA.A mutation in the human cardiac sodium channel (E161K) contributes to sick sinus syndrome, conduction disease and Brugada syndrome in two families. J Mol Cell Cardiol. 2005 Jun;38(6):969-81. DOI: 10.1016/j.yjmcc.2005.02.024**
- **Kyndt F1, Probst V, Potet F, Demolombe S, Chevallier JC, Baro I, Moisan JP, Boisseau P, Schott JJ, Escande D, Le Marec H.Novel SCN5A mutation leading either to isolated cardiac conduction defect or Brugada syndrome in a large French family.Circulation. 2001 Dec 18;104(25):3081-6.**
- **Wang DW1, Viswanathan PC, Balser JR, George AL Jr, Benson DW. Clinical, genetic, and biophysical characterization of SCN5A mutations associated with atrioventricular conduction block. Circulation. 2002 Jan 22;105(3):341-6. DOI: 10.1161/hc0302.102592**
- **Wang DW1, Viswanathan PC, Balser JR, George AL Jr, Benson DW. Clinical, genetic, and biophysical characterization of SCN5A mutations associated with atrioventricular conduction block. Circulation. 2002 Jan 22;105(3):341-6. DOI: 10.1161/hc0302.102592**
- **Brink PA, Ferreira A, Moolman JC, et al. Gene for progressive familial heart block type I maps to chromosome 19q13. Circulation. 1995; *39*: 1633–1640**
- **Stallmeyer B1, Kuß J1, Kotthoff S1, Zumhagen S1, Vowinkel K1, Rinné S1, Matschke LA1, Friedrich C1, Schulze-Bahr E1, Rust S1, Seebohm G1,**

Decher N1, Schulze-Bahr E2.A Mutation in the G-Protein Gene GNB2 Causes Familial Sinus Node and Atrioventricular Conduction Dysfunction.Circ Res. 2017 May 12;120(10):e33-e44. doi: 10.1161/CIRCRESAHA.116.310112

- **Epidemiology**

In the United States, the prevalence of second-degree AV block in young adults is reported to be 0.003%. However, the rate is significantly higher among trained athletes (**Zehender 1990**). Nearly 3% of patients with underlying structural heart disease develop some form of second-degree AV block. The male-to-female ratio of second-degree AV block is 1:1. **Mond HG1, Vohra J2.The Electrocardiographic Footprints of Wenckebach Block. Heart Lung Circ. 2017 Dec;26(12):1252-1266. doi: 10.1016/j.hlc.2017.06.718.**

Brief History of second degree AV block

In 1899, Karel Frederik Wenckebach described a cardiac arrhythmia with periodic dropped beats now referred to as a Wenckebach sequence. (**Wenckebach KF. Zur Analyse des unregelmassigne Pulses: II. [On the analysis of irregular pulses: II.]. Z Klin Med. 1899; 37:475-88**.) (**Peréz-Riera, Femenia F, McIntyre WF and Baranchuk A. Karel Frederick Wenckebach (1864- 1940): A giant in medicine. Cardiology J 2011; 18: 337-339**.) This was later shown to be due to a block in the AVN, but currently, we identify Wenckebach sequences throughout the heart with most being recognized on the surface ECG as characteristic footprints.

Signs and symptoms

Most people with Wenckebach (Type I Mobitz) do not show symptoms. However, those that do usually display one or more of the following: Second-degree atrioventricular block (AV block) is a disease of the electrical conduction system of the heart. It is a conduction block between the atria and ventricles. The presence of second-degree AV block is diagnosed when one or more (but not all) of the atrial impulses fail to conduct to the ventricles due to impaired conduction. It is classified as a block of the AV node and is categorized in between first-degree (slowed conduction) and third-degree blocks (complete block).

History and Physical

History taking for patients with concerns for AV block should include:

1. History of heart disease, both congenital and acquired
2. Full list of medications and dosing. Particular drugs of interest include beta-blockers, calcium channel blockers, antiarrhythmic drugs, digoxin
3. Recent cardiac procedure
4. Signs and symptoms associated with other systemic diseases associated with heart block (amyloidosis, sarcoidosis)
5. Baseline exercise capacity
6. Potential expose to tick bites

The following symptoms should raise concerns:

- Dyspnea
- Fatigue
- Chest pain
- Presyncope Light-headedness, Dizziness or syncope(fainting)
- Sudden cardiac arrest

Types

There are two non-distinct types of second-degree AV block, called Type 1 and Type 2. In both types, a P wave is blocked from initiating a QRS complex; but, in Type 1, there are increasing delays in each cycle before the omission, whereas, in Type 2, there is no such pattern. (**Silverman ME, Upshaw CB, Lange HW (August 2004). "Woldemar Mobitz and His 1924 classification of second-degree atrioventricular block". Circulation. 110 (9): 1162–7. doi:10.1161/01.CIR.0000140669.35049.34. PMID 15339865**.) (**Hay J (1906). "Bradycardia and cardiac arrhythmia produced by depression of certain of the functions of the heart". The Lancet. 1906 (1): 139–143. doi:10.1016/s0140-6736(01)44443-**6.) Type 1 second-degree heart block is considered a more benign entity than type 2 second-degree heart block (http://health.medicscientist.com/wp-content/uploads/2011/04/SeconddegreeAVblock2_thumb.jpg) with type 1 not having structural changes found on histology. Both types are named after Woldemar Mobitz.(http://www.whonamedit.com/synd.cfm/2824.html(**W. Mobitz. Über die unvollständige Störung der Erregungsüberleitung zwischen Vorhof und Kammer des menschlichen Herzens. Zeitschrift für die Gesamte Experimentelle Medizin, Berlin 1924, 41: 180–237**.) Type I is also named for Karel Frederik Wenckebach,(**K. F. Wenckebach. De Analyse van den onregelmatigen Pols. III. Over eenige Vormen van Allorythmie en Bradykardie. Nederlandsch Tijdschrift voor Geneeskunde, Amsterdam, 1898, 2: 1132**.) and type II is also

named for John Hay. (**Silverman ME, Upshaw CB, Lange HW (August 2004). "Woldemar Mobitz and His 1924 classification of second-degree atrioventricular block". Circulation. 110 (9): 1162–7. doi:10.1161/01.CIR.0000140669.35049.34. PMID 15339865**) (**Hay J (1906). "Bradycardia and cardiac arrhythmia produced by depression of certain of the functions of the heart". The Lancet. 1906 (1): 139–143. doi:10.1016/s0140-6736(01)44443-6**)

Type 1 (Mobitz I/Wenckebach)

Type 1 Second-degree AV block, also known as Mobitz I or Wenckebach periodicity, is almost always a disease of the AV node. Wenckebach published a paper in 1906 on progressively lengthening PR intervals [8] that was later classified as Type I in Mobitz's 1924 paper.(***Mobitz, W (1924). "Über die unvollständige Störung der Erregungsüberleitung zwischen Vorhof und Kammer des menschlichen Herzens [On the partial block of impulse conduction between atrium and ventricle of human hearts]". Z Gesamte Exp Med. 41: 180–237. doi:10.1007/bf02758773***) Thus, both "Mobitz type I" and "Wenckebach block" refer to the same pattern and pathophysiology. In Wenckebach's 1906 paper, his original observations were from increasing delay in contraction of the atria & ventricles that shortened after a brief pause and this was later observed on ECG after Einthoven's invention in 1901 that became the ECG. Today, Mobitz I heart block is characterized by progressive prolongation of the PR interval on consecutive beats followed by a blocked P wave (i.e., a dropped QRS complex). After the dropped QRS complex, the PR interval resets and the cycle repeats. This grouped beating was described as "Luciani periods" after Luigi Luciani's work in 1873. (**Silverman ME, Upshaw CB, Lange HW (August 2004). "Woldemar Mobitz and His 1924 classification of second-degree atrioventricular block". Circulation. 110 (9): 1162–7. doi:10.1161/01.CIR.0000140669.35049.34. PMID 15339865**) The result is a lengthening of the R-R intervals as each subsequent P-wave reaches an increasingly refractory AV node until the impulse fails to conduct, which ultimately results in a blocked QRS complex.One of the baseline assumptions when determining if an individual has Mobitz I heart block is that the atrial rhythm has to be regular. If the atrial rhythm is not regular, there could be alternative explanations as to why certain P waves do not conduct to the ventricles.This is almost always a benign condition for which no specific treatment is needed for the rhythm itself. It can be seen in myocardial ischemia, propranolol use, digitalis use, rheumatic fever, and chronically in ischemic heart disease and other structural diseases (amyloidosis, mitral valve prolapse, aortic valve disease, and

atrial septal defect). In symptomatic cases, intravenous atropine or isoproterenol may transiently improve conduction. (**Lilly, L. S., Pathophysiology of Heart Disease. Baltimore: Lippincott Williams & Wilkins; 2007**).

Type 2 (Mobitz II/Hay)

Type 2 Second-degree AV block, also known as Mobitz II, is almost always a disease of the distal conduction system (His-Purkinje System).

Mobitz II heart block is characterized on a surface ECG by intermittently nonconducted P waves not preceded by PR prolongation and not followed by PR shortening. There is usually a fixed number of non-conducted P waves for every successfully conducted QRS complex, and this ratio is often specified in describing Mobitz II blocks. For example, Mobitz II block in which there are two P waves for every one QRS complex may be referred to as 2:1 Mobitz II block. (**Dubin, Dale (2000). Rapid interpretation of EKG's : an interactive course (6. ed.). Tampa, Fla.: Cover Publ. ISBN 978-0912912066):181)**

The medical significance of this type of AV block is that it may progress rapidly to complete heart block, in which no escape rhythm may emerge. In this case, the person may experience a Stokes-Adams attack, cardiac arrest, or sudden cardiac death. The definitive treatment for this form of AV Block is an implanted pacemaker.

The impairment is usually below the AV node. (**Wogan JM, Lowenstein SR, Gordon GS (1993). "Second-degree atrioventricular block: Mobitz type II". J Emerg Med. 11 (1): 47–54. doi:10.1016/0736-4679(93)90009-V. PMID 8445186**.) Although the terms infranodal block or infrahisian block are often applied to this disorder, they refer to the anatomic location of the block, whereas Mobitz II refers to an electrocardiographic pattern.

P:QRS ratios

Because type I Mobitz block occurs in regular cycles, there is always a fixed ratio between the number of P waves and the number of QRS complexes per cycle. This ratio is often specified when describing the block. For example, a Mobitz type I block which has 4 P waves and 3 QRS complexes per cycle may be referred to as 4:3 Mobitz Type I block.[(**Dubin, Dale (2000). Rapid interpretation of EKG's : an interactive course (6. ed.). Tampa, Fla.: Cover Publ. ISBN 978-0912912066):181)**:179

Type II Mobitz block also usually occurs with a fixed P:QRS ratio, with a set number of P waves for every successfully elicited QRS.[12]:179 This ratio is also frequently

specified in referring to 3:1, 4:1, 5:1, or higher Mobitz type II block. Higher numbers of P waves for every QRS indicate more severe block.[12]:181 Of course, because type II Mobitz block is unstable by nature, it is common for the P:QRS ratio in Mobitz type II block to change over time.[citation needed]

The P:QRS ratio is always of the form X:(X − 1) in type I Mobitz block and of the form X:1 in type 2 Mobitz block because of the nature of the pattern of each. Thus one may leave out the type and refer to 3:1 Mobitz block or 4:3 Mobitz block, for example, without creating ambiguity, except in the case of 2:1 block.

Main differences between Type I and Type II second degree AV block

	Mobitz type I	Mobitz type II or Hay
Clinical	Usually acute inferior MI Rheumatic fever Digitalis Propranolol	Usually chronic anteroseptal MI Lenegre's disease Lev's disease Cardiomyopathy
Anatomical	Usually AV nodal, sometimes His bundle	Always subnodal, usually bundle branches. Is almost always a disease of the distal conduction system located in the ventricular portion of the myocardium.
Electrophysiological	Relative refractory period Decremental conduction	No relative refractory period All-or-none conduction
Electrocardiographic	RP/PR reciprocity Prolonged PR Normal QRS duration	Stable PR Normal PR Wide QRS
Atropine	Improves	Worsens
Exercise and catecholamines	Improves	Worsens
Carotid sinus massage	Worsens	Improves

2:1 AV block

In the case of 2:1 block (2 P waves for every QRS complex) it is impossible to differentiate type I from type II Mobitz block based solely on the P:QRS ratio or on a pattern of lengthening PR intervals.(**Barold SS1.2:1 AV block : The orphan of organizational guidelines for cardiac pacing.Herzschrittmacherther Elektrophysiol.**

2016 Jun;27(2):154-5. doi: 10.1007/s00399-016-0424-8.PMID: 27193770): In this case, a lengthened PR interval with a normal QRS width is most likely indicative of a type I-like pathology, and a normal PR interval with a widened QRS is most likely indicative of a type II-like pathology. It is strange that 2:1 AV block has not achieved better prominence in the major organizational guidelines for cardiac pacing. The reasons are unclear. 2:1 AV block is an important entity and the limitations regarding its importance involve both the ACCF/AHA/HRS and the ESC guidelines [1, 2]. 2012 ACCF/AHA/HRS focused update incorporated into the ACCF/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities The 2012 ACCF/AHA/HRS guidelines define advanced second-degree AV block as the blocking of two or more consecutive P waves and therefore do not include 2:1 AV block in this classification [1]. Referring to 2:1 AV block, the guidelines state that "when AV conduction occurs in a 2:1 pattern, block cannot be classified unequivocally as type I or type II, although the width of the QRS can be suggestive, as just described." This is illogical because 2:1 AV block is neither type I nor type II AV block. The suggested description in the guidelines is as follows, but it does not clarify the related statement about 2:1 AV block: "Type II second-degree AV block is characterized by fixed PR intervals before and after blocked beats and is usually associated with a wide QRS complex" (**Epstein AE, DiMarco JP, Ellenbogen KA, Estes NA 3rd, Freedman RA, Gettes LS, Gillinov AM, Gregoratos G, Hammill SC, Hayes DL, Hlatky MA, Newby LK, Page RL, Schoenfeld MH, Silka MJ, Stevenson LW, Sweeney MO, Tracy CM, Epstein AE, Darbar D, DiMarco JP, Dunbar SB, Estes NA 3rd, Ferguson TB Jr, Hammill SC, Karasik PE, Link MS, Marine JE, Schoenfeld MH, Shanker AJ, Silka MJ, Stevenson LW, Stevenson WG, Varosy PD, American College of Cardiology Foundation, American Heart Association Task Force on Practice Guidelines, Heart Rhythm Society (2013) 2012 ACCF/AHA/HRS**) focused update incorporated into the ACCF/AHA/HRS 2008 guidelines for device based therapy of cardiac rhythm abnormalities: a report of the American College of Cardiology Foundation/American Heart Association Task ForceonPracticeGuidelines and theHeart Rhythm Society. JAm Coll Cardiol 61:e6–75). The 2012 ACC/AHA/HRS guidelines indicate that "it is not always possible to determine the site of AV block without electrophysiological evaluation, because type I second-degree AV block can be infranodal even when the QRS is narrow. If type I second-degree AV block with a narrow or wide QRS is found to be intra- or infra-Hisian at electrophysiological study, pacing should be considered." There

is no mention of asymptomatic 2:1 AV block in this statement where it would be appropriate. However, it appears in the form of "second-degree AV block" in the formal recommendations: Class IIa. "Permanent pacemaker implantation is reasonable for asymptomatic second-degree AV block at intra or infra-His levels found at electrophysiological study." These guidelines could be interpreted to mean that all patients with asymptomatic second-degree AV block including 2:1AV block (except those with type II block), regardless of QRS duration, should undergo electrophysiological testing to determine the site of block. Further details to guide clinical practice would be beneficial. Finally, the ACC/AHA guidelines provide recommendations for pacing based on the rate during asymptomatic complete AV block, but no such recommendations exist for asymptomatic 2:1 AV block, especially when the QRS is narrow and conservative treatment is being evaluated. 2013 ESC guidelines on cardiac pacing and cardiac resynchronization therapy There is no mention at all of symptomatic or asymptomatic 2:1 AV block in the sections on acquired AV block (which is very brief) and in that on syncope with bundle branch block.(**European Society of Cardiology (ESC), European Heart Rhythm Association (EHRA), Brignole M, Auricchio A, Baron-Esquivias G, Bordachar P, Boriani G, Breithardt OA, Cleland J, Deharo JC, DelgadoV,ElliottPM,GorenekB,IsraelCW,Leclercq C, Linde C, Mont L, Padeletti L, Sutton R, Vardas PE (2013) 2013 ESC guidelines on cardiac pacing and cardiac resynchronization therapy: the task force on cardiac pacing and resynchronization therapy of the European Society of Cardiology (ESC). Developed in collaboration with the European Heart Rhythm Association (EHRA). Europace 15:1070–1118**) There are only two pertinent entries.

Class I: Pacing is indicated in patients with third- or second-degree type 2 AV block, irrespective of symptoms.

Class IIa: Pacing should be considered in patients with second-degree type 1 AV block which causes symptoms or is found to be located at intra- or infra-His levels at an electrophysiological study. The second recommendation would be more complete with the addition of asymptomatic 2:1 AV block with bundle branch block in which infranodal block is found.

Conclusion The organizational guidelines have served us well. Most of the missing points about 2:1 AV block presented here are implied in the guidelines and generally do not affect clinical practice. It could be argued that no minor changes regarding 2:1 AV block

are necessary. However, their implementation may improve clarity of the guidelines and promote their better understanding and improved clinical practice.

The typical **Wenckebach Periodicity** only occur in about 15% of cases, and atypical in 85%. Earlier reports regarded Wenckebach AV sequences as rare as they are only occasionally seen on the surface ECG. With the use of Holter monitoring, Wenckebach AV sequences occur in 4–6% of all traces and are particularly common at night in the young athletes. Most, but not all cases are benign. Atypical Wenckebach AV sequences are a complex group. Outside the atrioventricular conducting system, such as in the SAN, Wenckebach sequences may not be obvious as they are partially hidden from the ECG tracing. However, by understanding the sequence footprints, clues are available in interpreting tracing with periodic pauses. Dual chamber paced rhythms may show Wenckebach sequences due to electronic control of the AV delay. Rarely exit blocks at the cellular level in the atrium, ventricle or at the pacing electrode-tissue interface can demonstrate Wenckebach sequences recognized on the surface ECG.

Atypical type I sequence mistaken for type II block Many type I sequences are atypical and do not conform to the traditional mathematical structure of the Wenckebach phenomenon with progressive prolongation of the PR intervals. (**El-Sherif N, Aranda J, Befeler B, et al. Atypical wenckebach periodicity simulating Mobitz II AV block. Br Heart J 1978;40:1376–83. 30.)(Friedman HS, Gomes JA, Haft JI. An analysis of Wenckebach periodicity. J Electrocardiol 1975;8:307–15. 31.)(Denes P, Levy L, Pick A, et al. The incidence of typical and atypical A-V Wenckebach periodicity. Am Heart J 1975;89:26–31.**)(**Barold SS, Friedberg HD. Second degree atrioventricular block. A matter of defi nition. Am J Cardiol 1974;33:311–15**.) A frequent erroneous definition of type II block found in most textbooks describes it as an ‘electrocardiographic pattern characterized by failure of a single impulse to conduct to the ventricles in the absence of antecedent lengthening of the PR interval (normal or prolonged).’ This definition of type II block in fact also describes type I block when the terminal portion of a long atypical type I sequence contains PR intervals with no discernible or measurable change before the blocked impulse (figure x).(**El-Sherif N, Aranda J, Befeler B, et al. Atypical wenckebach periodicity simulating Mobitz II AV block. Br Heart J 1978; 40:1376–83**.)

Figure 1

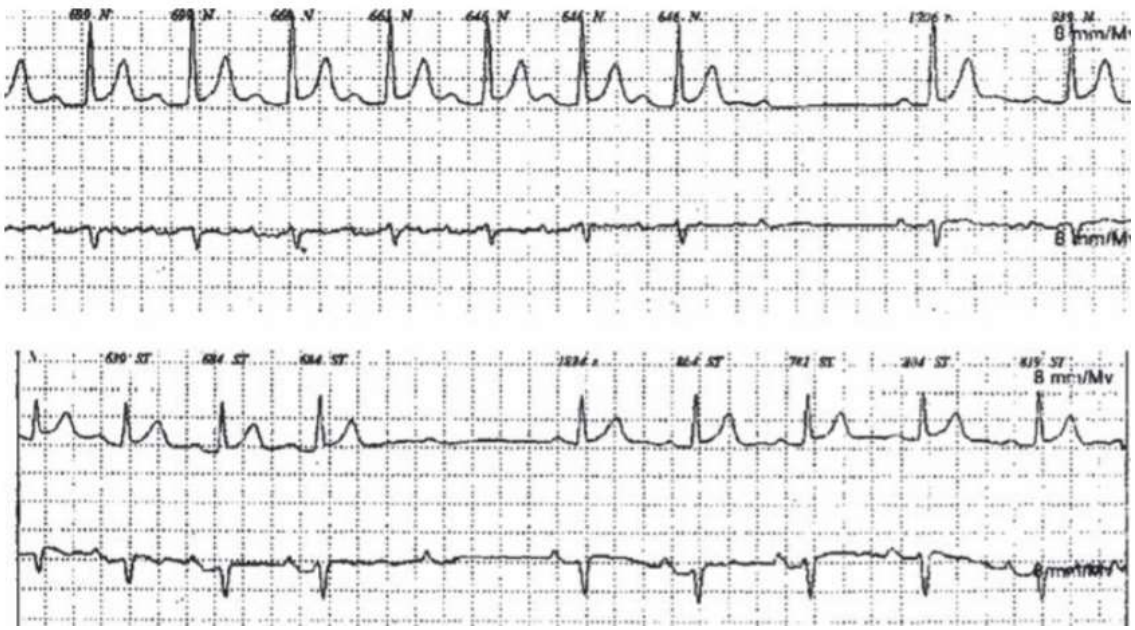


Figure 1 Rhythm strips from Holter recordings in a young man misdiagnosed as having type II block. There is vagally induced type I block in the setting of sinus slowing. Sinus slowing is evident at the time of AV block. Note the constant PR intervals before the non-conducted P wave and shortening of the PR interval after the blocked beat. The misdiagnosis of type II block was based on applying the incomplete and incorrect definition of type II block as the sudden occurrence of a blocked beat or P wave without preceding prolongation of the PR interval.

Vagally mediated AV block Vagally mediated AV block shows obvious irregular PP intervals and is bradycardia-associated (not bradycardia-dependent). The stability of the sinus rate is an important criterion of type II block because a vagal surge (generally a benign condition) that occurs invariably at the level of the AV node usually causes simultaneous sinus bradycardia (sinus node) and depression of AV conduction (AV node), a situation that can superficially resemble type II second-degree AV block.19–21 Sinus slowing with AV block essentially rules out type II block. The large variety of ECG patterns seen with vagal AV block depends on the interplay of several factors infl uencing the net vagal effect in: (1) the moment in the cycle when the vagal effect occurs, (2) the intensity (and speed) of the vagal surge, (3) the atrial rate, (4) the sensitivity of the AV node and (5) the background sympathetic activity. Vagally induced AV block may be followed by either a shorter or longer PR interval and rarely by an unchanged PR interval. An unchanged PR interval after the blocked P wave may be due to a nonconducted P

wave occurring fortuitously with an escape AV junctional beat so that the P–QRS relationship or PR interval is equal to that seen before the blocked P waves. Alternatively, a residual vagal effect on the AV node may prevent the expected PR shortening. These mechanisms simulate type II second-degree AV nodal block. The clue to the diagnosis of AV nodal block is the presence of sinus slowing, no matter how slight, in the setting of a narrow QRS complex. Vagally mediated AV block occurs commonly during sleep when parasympathetic activity predominates. In this respect, a recent statement that 'episodes of Mobitz type II second-degree block may be present in some high-level athletes at night (24 h Holter) but if present in baseline ECGs during daytime, structural disease or subjacent conditions should be ruled out' implies erroneously that type II block at night is benign.(**Brugada J, Benito B. Electrocardiographic Findings in Athletes. E-Journal of Cardiology Practice 4. http:// www.escardio.org/knowledge/cardiology_practice/ ejournal_vol4/vol4n31.htm**)
Thus, according to this incorrect definition, the diagnosis of type II block can also be entertained with only two or three constant PR intervals before block of a single P wave (and ignoring the first post block PR interval).

Type I and type II in the same individual

The presence of both narrow-QRS type I and II second-degree AV block patterns in a Holter recording essentially excludes the diagnosis of type II second-degree AV block in this situation. (**Barold SS, Hayes DL. Second-degree atrioventricular block: a reappraisal. Mayo Clin Proc 2001; 76:44–57.**) The occurrence of both wide QRS type I and II block in the same recoding is unusual. (**Barold SS, Jaïs P, Shah DC, et al. Exercise-induced second-degree AV block: is it type I or type II? J Cardiovasc Electrophysiol 1997;8:1084–6**.) Thus, this combination with a narrow QRS is so rare that one can confidently label the tracing as showing only type I block in virtually all the cases. On this basis, probably, the reported cases of both type I and II in athletes represent only type I blocks.

Shortage of PR interval: no truly conducted first post block P wave

The literature of type II block is replete with errors because the diagnostic importance of an unchanged PR interval after a single blocked impulse is often ignored. A constant PR after the blocked beat is a sine qua non of type II block. The diagnosis of type II cannot be made if there is no P wave after a blocked impulse or the P wave is not conducted with

the same PR interval as all the other conducted P waves. In such a situation, the pattern is either type I or unclassifiable. The shorter PR interval after the blocked P wave may be due to either improved conduction (type I block) or AV dissociation due to an escape AV junctional beat that bears no relationship to the preceding P wave. (**Barold SS, Jaïs P, Shah DC, et al. Exercise-induced second-degree AV block: is it type I or type II? J Cardiovasc Electrophysiol 1997; 8:1084–6**.) (**Barold SS, Hayes DL. Second-degree atrioventricular block: a reappraisal. Mayo Clin Proc 2001; 76:44–57)**

2:1 AV block

Although 2:1 AV block can occur in either the AV node or the His–Purkinje system, this form of AV block cannot be classified into type I or type II second-degree AV block. 2:1 AV block is best considered as advanced second-degree AV block, as are higher degrees of block such as 3:1, 4:1, etc, according to the definitions promulgated by the WHO and ACC. (**Barold SS. 2:1 Atrioventricular block: order from chaos. Am J Emerg Med 2001; 19:214–17**.)

Other characteristics of type II block versus vagally induced block

True narrow QRS type II block is relatively rare and occurs without sinus slowing. It is typically associated with AV conduction ratios >2 (3:1, 4:1 which are rare in vagal block) and without associated type I structures. (**Lange HW, Ameisen O, Mack R, et al. Prevalence and clinical correlates of non-Wenckebach, narrow-complex second-degree atrioventricular block detected by ambulatory ECG. Am Heart J 1988;115:114–20**.) Sustained advanced second-degree AV block is far more common in association with true type II block than type I block or its variant. Although intense vagal tone can be associated with block of multiple consecutive P waves, vagally mediated AV block rarely involves more than block of two consecutive P waves. (**Lange HW, Ameisen O, Mack R, et al. Prevalence and clinical correlates of non-Wenckebach, narrow-complex second-degree atrioventricular block detected by ambulatory ECG. Am Heart J 1988; 115:114–20**.) The diagnosis of type II can only be made with confidence if the same pattern occurs repeatedly without sinus slowing and in the absence of any sequence suggesting type I second-degree AV block. Obviously, the PR intervals before and after the blocked impulse must remain constant.

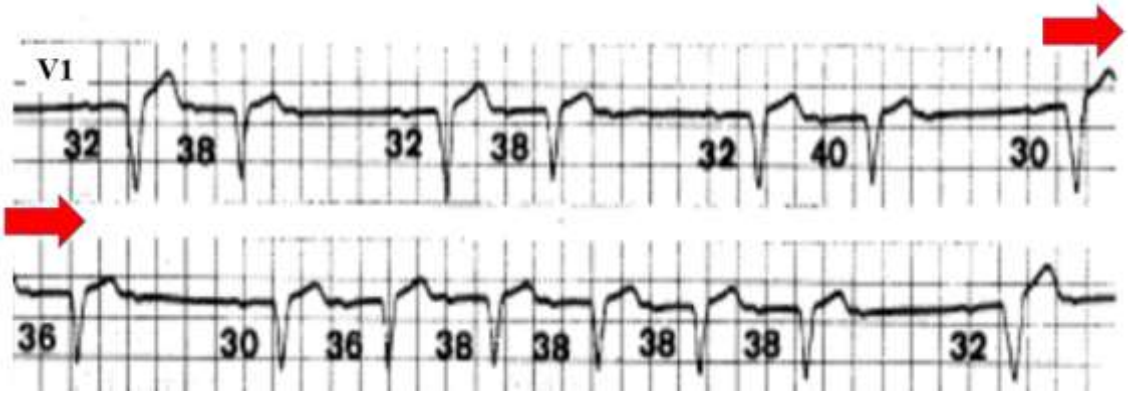
Impact of exercise

Exercise induces an increase in sympathetic tone with improvement of AV nodal conduction. Consequently, type I block commonly improves or disappears with exercise. In contrast, enhanced sympathetic activity exerts no effect below the AV node so that type II block commonly deteriorates on exercise. The purported cases of type II block in athletes appeared to behave like type I blocks on exercise, and no aggravation of the block occurred, providing further proof against the diagnosis of type II block.2–7
(**Viitasalo MT, Kala R, Eisalo A. Ambulatory electrocardiographic recording in endurance athletes. Br Heart J 1982; 47:213–20**.) (**Viitasalo MT, Kala R, Eisalo A. Ambulatory electrocardiographic fi ndings in young athletes between 14 and 16 years of age. Eur Heart J 1984; 5:2–6**.) (**DiNardo-Ekery D, Abedin Z. High degree atrioventricular block in a marathoner with 5-year follow-up. Am Heart J 1987; 113:834–7**.) (**Northcote RJ, Canning GP, Ballantyne D. Electrocardiographic fi ndings in male veteran endurance athletes. Br Heart J 1989;61:155–60**.) (**Bjørnstad H, Storstein L, Meen HD, et al. Ambulatory electrocardiographic fi ndings in top athletes, athletic students and control subjects. Cardiology 1994; 84:42–50.**) (**Jensen-Urstad K, Saltin B, Ericson M, et al. Pronounced resting bradycardia in male elite runners is associated with high heart rate variability. Scand J Med Sci Sports 1997; 7:274–8**.)

Typical Wenckebach periodicity is considered to be present when the record show:

1) The first PR interval of a cycle (AH interval in AV nodal block and HV interval in HP block) is the shortest.
2) Progressive lengthering of the PR interval with the increment between the first and second conducted beats being the largest;
3) Progressive decrease in the RR(VV) intervals;
4) A pause produced by the non-conducted P wave equal to the difference between the last PR before the pause and the first PR (after the pause) subtracted form twice the PP interval.

Atypical Wenckebach period failed to meet one or more criteria for typical Wenckebach peridicity. A pseudo-Mobitz II arrangement in the His bundle electrogram is defined as follows: A long Wenckebanc cycle in which the last three beats of the cycle show relatively constant PR interval (variation in the AH or HV intervals of> 10ms) and in which the first PR interval following the blocked beat is shorter than the last PR interval of the preceding cycle by ≥ 40ms. Figure 2



***Fifure 2 A Typical example of pseudo-MobitzII block in the AV node. Recording were obtained from a 55-year-old man two days after the onset of and acute inferior MI. The ECG swohs and old anteroseptal MI and intraventricular conduction defect of the incomplete LBBB pattern. Newly developed Q waves in the inferior leads are compatible with and acute inferior MI. The continuous rhythm strip at the bottom shows 4 successibe 3:2 Wenckebach cycles followed by a long 7:6 cycle. PR intervals are listed in hudredths of a second. During the long Wenckebach cycle the main increment of AV conduction delay occurre between the first and second beats of the cycles. The PR intervals remained almost constant(variation of ≤0.02s) for 5 sucessive beats before the blocked P wave. This was followed by significant shortening of the PR interval in the beat than immediately followed the blocked impulse. Rhis represents a pseudo-Mobitz II arrangement.Also note the presence of varying degrees of bradycadia-dependent LBBB pattern in the openign beats of the Wenckebach cycles that followed the loger pauses.* (El-Sherif N, Aranda J, Befeler B, Lazzara R. Atypical Wenckebach periodicity simulating Mobitz II AV block.Br Heart J. 1978 Dec;40(12):1376-83. PMCID: PMC483582 DOI: 10.1136/hrt.40.12.1376)**

Atypical Wenckebach AV block

Wenckebach AV block as seen on Holter, frequently does not meet strict mathematical sequence criteria. These cases are referred to as atypical Wenckebach AV block and are clinically much more frequent (85%) than typical (15%) sequences. (**Friedman HS, Gomes JAC and Haft JI. An analysis of Wenckebach periodicity. Journal of Electrocardiology. 1975; 8: 307-315**.) Clinically, the most common causes of atypical Wenckebach AV block during Holter monitoring are the nocturnal episodes in young patients with concomitant sinus bradycardia during the sequences (Figure 3).

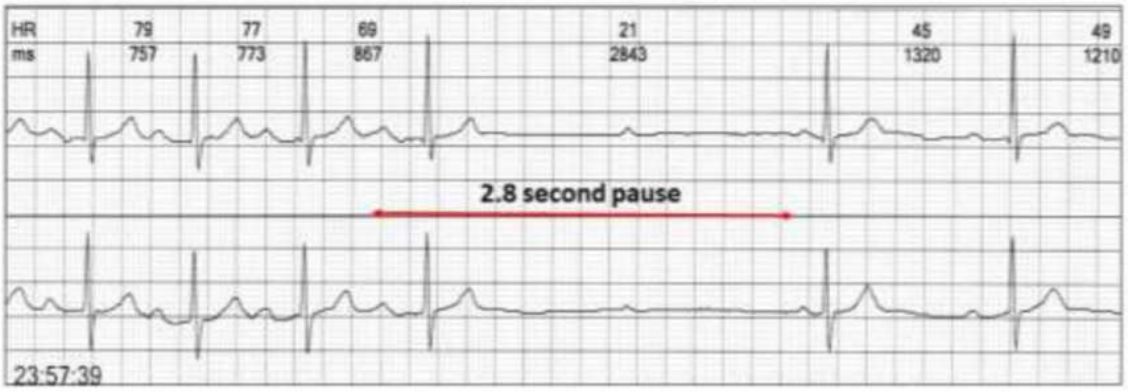


Figure 3 Two channel Holter monitor recordings of atypical Wenckebach AV sequences due to abrupt nocturnal slowing: Wenckebach AV sequence with a 2.8 second pause coinciding with the dropped beat.

Both nocturnal (**Kusumoto FM, Goldschlager N. Cardiac pacing. New England Journal of Medicine 1996; 334: 89- 97**.) sinus bradycardia and Wenckebach AV block are likely vagal induced and when they occur together result in an atypical Wenckebach AV sequence. There are a variety of other atypical Wenckebach AV sequences seen including varying PR intervals, failure to drop a beat particularly during long sequences, sinus arrhythmia and sinus arrest instead of a dropped beat (Figure 2B). With a very long PR interval, the next P wave may be buried in the QRS resulting in no apparent pause (Figure 3).

Wenckebach AV sequences may occur during low atrial rhythm particularly if it is also present during sinus rhythm (Figure 4).

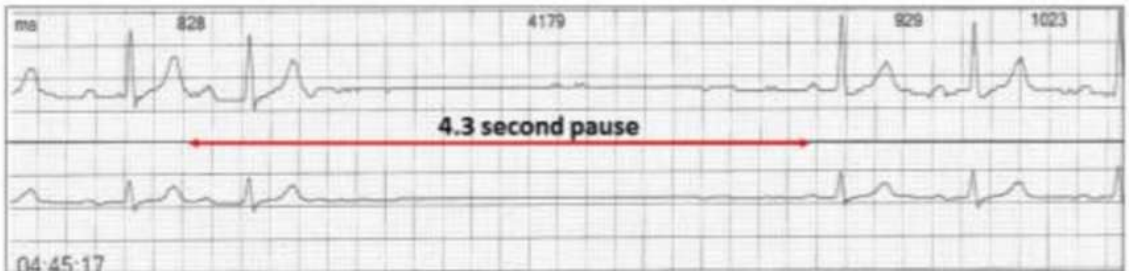


Figure 4***: Wenckebach sequence interrupted by a 4.3 second sinus pause and hence no dropped beat***.

There are a number of ways of terminating a Wenckebach AV sequence. These include non-conducted atrial rhythms including ectopic, couplets, triplets and focal atrial tachycardia (Figure 5).

Figure 5

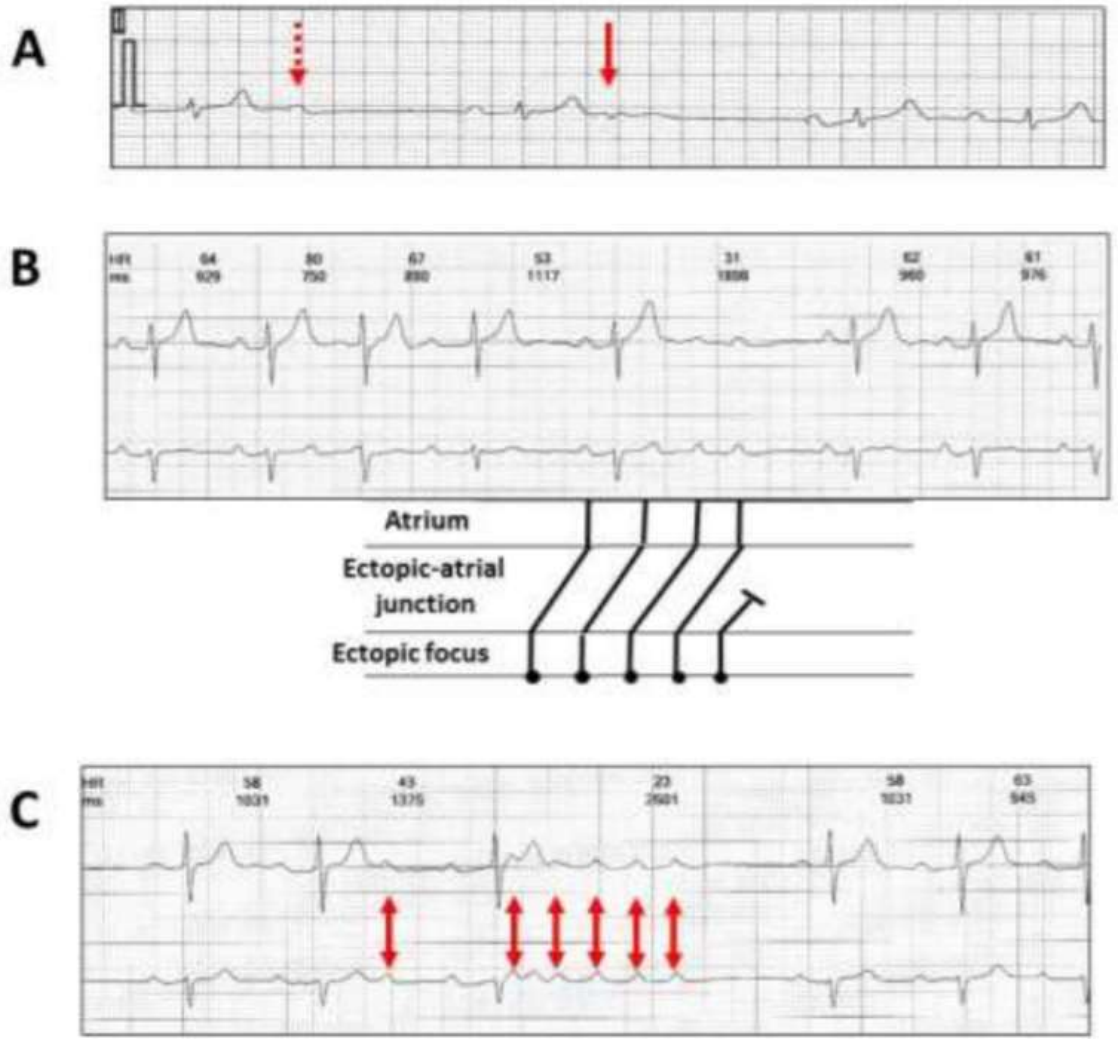


Figure 5: A: ECG lead II of Wenckebach AV sequences, the first of which is terminated by a P wave (stippled vertical arrow) and the second interrupted by a non-conducted atrial ectopic (solid vertical arrow). B, C: Two channel Holter monitor recordings of a Wenckebach AV sequence terminated by a run of non-conducted focal atrial tachycardia. This ectopic focus interrupts a Wenckebach AV block. The focal atrial tachycardia also demonstrates a Wenckebach block at the ectopic-myocardial junction in the atrium. The ladder diagram shows this Wenckebach sequence with a gradual increase in the tachycardia rate before termination.

Conducted atrial rhythms may also demonstrate Wenckebach AV sequences (Figure 6A, B). Figure 6C demonstrates a typical Wenckebach sequence, where the pause is extended by a non-conducted atrial ectopic.

Figure 6

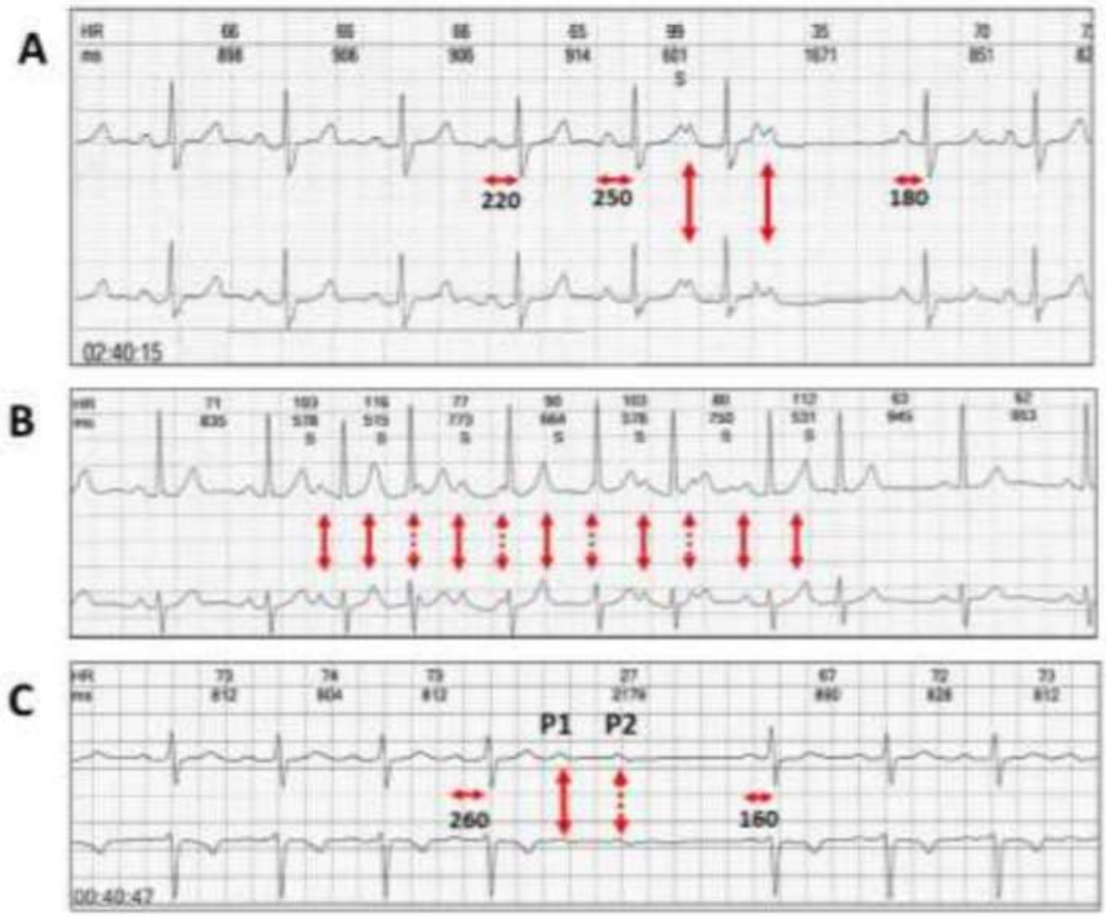


Figure 6: Atrial Ectopy A: Two channel Holter monitor recording demonstrating a Wenckebach AV sequence interrupted by an atrial couplet, which also demonstrates a Wenckebach sequence (vertical arrows). 25
B: Two channel Holter monitor recording demonstrating a short run of atrial tachycardia 170 bpm with 3:2 and 2:1 Wenckebach AV sequences. The tachycardia terminates prior to a non-conducted P wave. Solid vertical arrows are conducted and stippled vertical arrows are non-conducted P waves.
C: Two channel Holter monitor recording demonstrating a Wenckebach AV sequence which appears to be terminated by an atrial couplet. However, the first P wave (P1 solid vertical arrow) is not premature and identical to other sinus P waves. The second P wave (P2 stippled vertical arrow) is an ectopic which originates from either the sinus node or adjacent to it. The combination of the two P waves terminates the sequence with an extended block.

Wenckebach sequences can be interrupted by a number of mechanisms including ventricular ectopics, couplets or triplets (Figure 7).

Figure 7

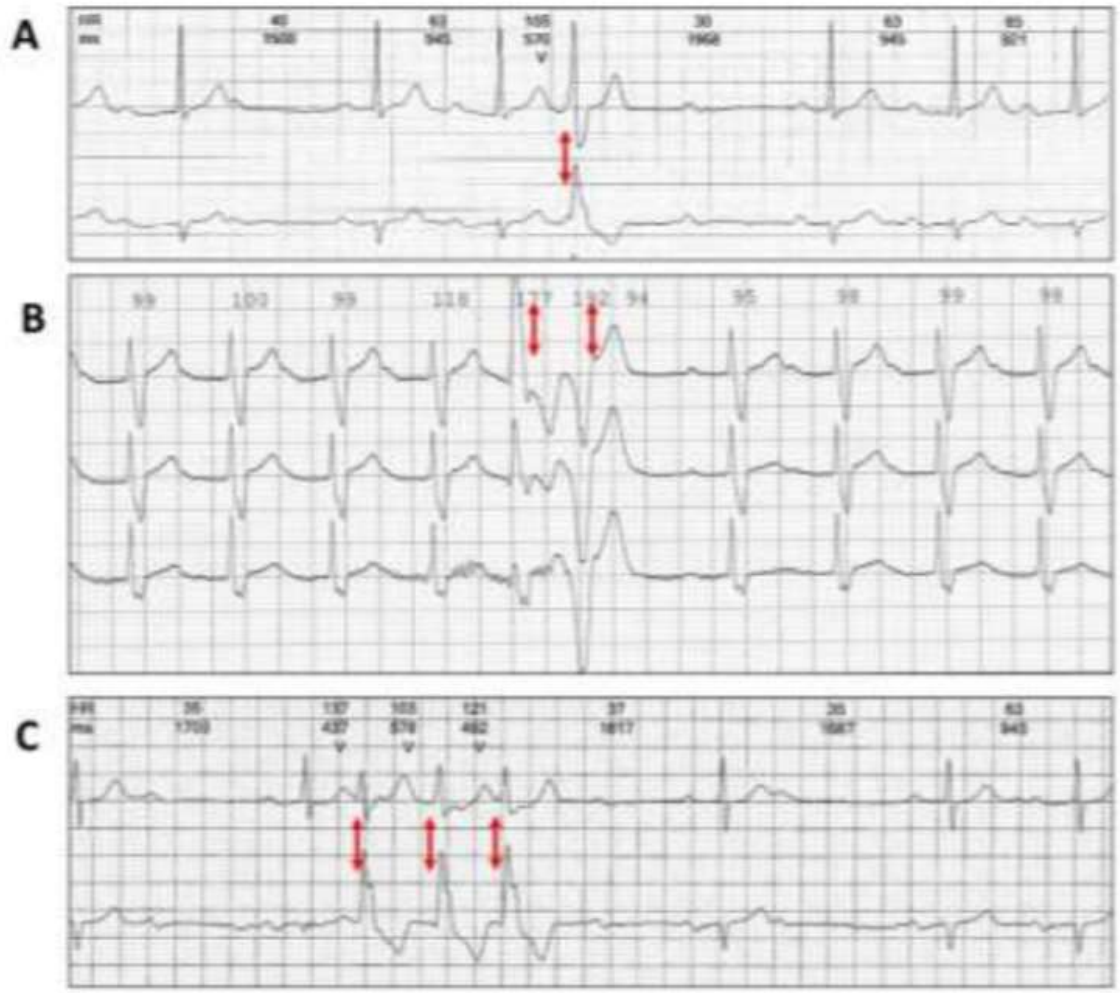


Figure 7: Ventricular ectopy
A: Two channel Holter monitor recording demonstrating a Wenckebach AV sequence interrupted by a single ventricular ectopic (vertical arrow). The next P wave is concealed within the ectopic and the following P wave also fails to conduct terminating the sequence.
B: Three channel Holter monitor recording demonstrating a Wenckebach AV sequence interrupted by a ventricular couplet (vertical arrows). The concealed P wave within the couplet fails to conduct terminating the sequence.
C: Two channel Holter monitor recording demonstrating a Wenckebach AV sequence interrupted by a ventricular triplet (vertical arrows). There are concealed P waves within the triplet and the next P wave fails to conduct terminating the sequence. (***Strasberg B, Amat-Y-Leon F, Dhingra RC, Palileo E, Swiryn S, Bauernfeind R, Wyndham C and Rosen KM. Natural history of chronic second-degree atrioventricular nodal block. Circulation 1981; 63: 1043-1049***.)

Infra-nodal Wenckebach AV sequences can occur in the bundle branches and are generally associated with other features of high degree AV block requiring cardiac pacing. (***Tandon A, Simpson L and Assar MD. Unusual origin of type 1 atrioventricular block with comments on Wenckebach contribution. Baylor University Medical Center***

Proceedings. 2011; 24: 9- 12.). The surface ECG features may present as typical Wenckebach sequences in the presence of a bundle branch block. In rare instances, Wenckebach AV sequences can occur from within the left or right bundle branches and recognized on the surface ECG. (***Narula OS and Samet P. Wenckebach and Mobitz type II A-V block due to block within the His bundle and bundle branches. Circulation 1970; 41: 947-965***.) (B***renes JC, Brenes-Pereira C and Castellanos A. Wenckebach-phenomenon at the left bundle branch. Clinical Cardiology 2006; 29: 226***.) (***Huang W, Lim PCY and Ching C-K. Wenckebach pattern in right bundle branch- benign or not? Journal of Electrocardiology 2017; 50: 223-226***.)(***Peter T, Harper RW, Vohra JK and Hunt D. The electrocardiographic recognition of the Wenckebach phenomenon in sites other than the atrioventricular junction. Heart and Lung 1976; 5: 747-754***.),

The QRS width widens for one or more beats without a 10 change in the PR interval (Figure 8).

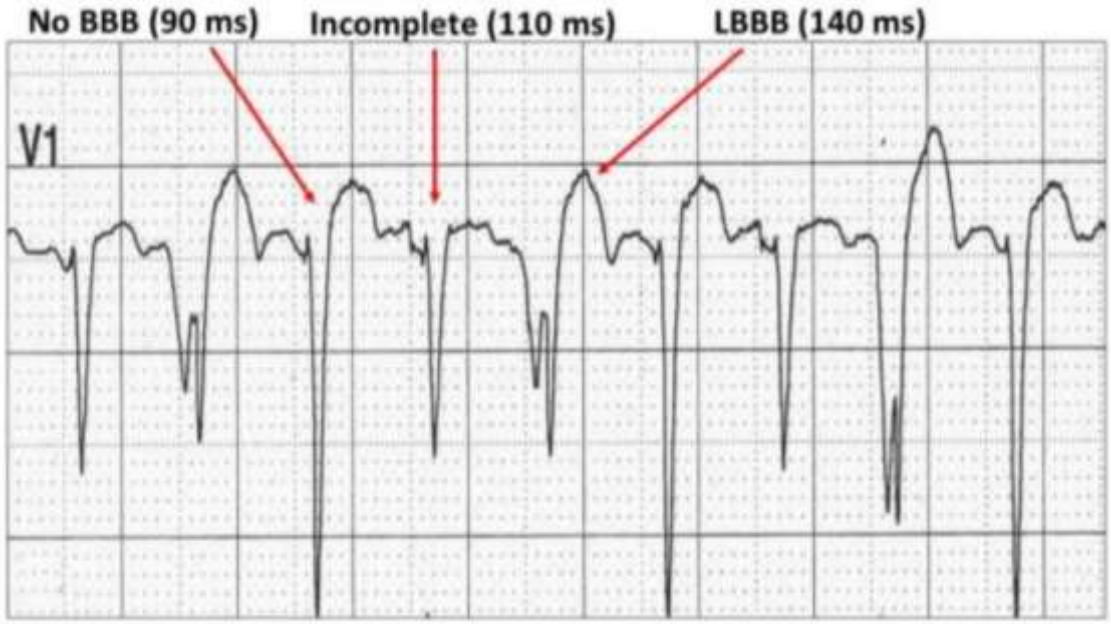


Figure 8: ECG, lead V1, sinus tachycardia. There is a Wenckebach sequence within the left bundle branch. The first labelled complex has a narrow QRS (90 ms), the second an incomplete bundle branch (110 ms) and the third, a left bundle branch block (140 ms). The sequence is then repeated.

Although the 12-lead surface ECG appearances allow differentiation into left or right bundle Wenckebach AV block***, (****Peter T, Harper RW, Vohra JK and Hunt D. The electrocardiographic recognition of the Wenckebach phenomenon in sites other than the atrioventricular junction. Heart and Lung 1976; 5: 747-754*) most cases recognized

today will be from Holter monitor recordings, where the differentiation will not be as apparent. Alternating Wenckebach is an atypical sequence which is often interpreted as Mobitz type II or even complete heart block. In this situation, each second P wave of the sequence is conducted and shows a progressive Wenckebach prolongation of the PR interval until one is dropped and the sequence commences again (Figure 8A). Coupled with this are alternate P waves, which are all dropped. The result is that the sequence terminates with two or maybe three dropped beats. (***Halpern MS, Nau GJ, Levi RJ, Elizara MV and Rosenbaum MB. Wenckebach periods of alternate beats. Clinical and experimental observations. Circulation 1973; 48: 41-49***.) The appearance may also occur with a bundle branch block. (***Medeiros CM, Rossi MB and Medeiros N. Alternate Wenckebach conduction through the right bundle branch. International Journal of Cardiology 1989; 23: 400-402***.) There are a number of explanations for alternating Wenckebach sequences which include block in the AV node and His bundle either in conjunction with each other or separately, (***Amat Y Leon F, Chuquimia R, Wu D, Denes P, Dhingra RC, Wyndham C and Rosen KM. Alternating Wenckebach periodicity: A common electrophysiologic response. The American Journal of Cardiology 1975; 36: 757-764***.) and is referred to as multilevel AV block. (***Kosowsky BD, Latif P and Radoff AM. Multilevel atrioventricular block. Circulation 1976; 54: 914- 921***.) Patients may present with syncope and sudden death, particularly if a bundle branch block is also present. (***Halpern MS, Nau GJ, Levi RJ, Elizara MV and Rosenbaum MB. Wenckebach periods of alternate beats. Clinical and experimental observations. Circulation 1973; 48: 41-49***.) A variant of alternating Wenckebach is shown in Figure 8B. Here the Wenckebach sequence occurs with alternate beats, which are bigeminal atrial ectopics

Another atypical Wenckebach sequence is so-called, reverse AV Wenckebach. In this rare ECG appearance, the PR intervals shorten in a reverse 3:2 Wenckebach sequence, so that the first PR interval is longer than the subsequent conducted beats (Figure 9).

Figure 9

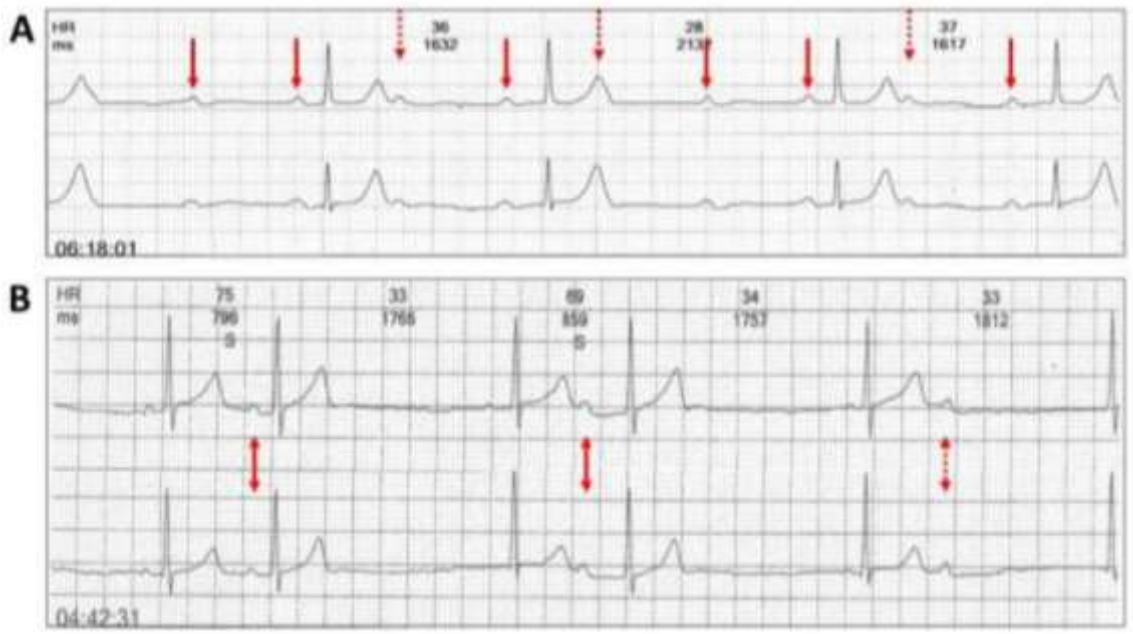


Figure 9: Alternating Wenckebach" AV sequences. A: Two channel Holter monitoring recording. Every second P wave of the sequence is conducted and shows a progressive Wenckebach prolongation of the PR interval until one is dropped and the sequence commences again (solid vertical arrows). Coupled with this are alternate P waves which are all dropped (stippled vertical arrows). The sequence terminates with two dropped beats. B: Two channel Holter monitor recording. The underlying sinus rhythm shows a varying PR interval with the last complex, a junctional escape beat. There is atrial bigeminy present (solid vertical arrows) with a Wenckebach AV block and a dropped beat (stippled vertical arrow).

(***Zahid M and Arora S. Reverse Wenckebach "pseudo-supernormal" conduction or paroxysmal atrioventricular block. Journal of Cardiovascular Disease Research. 2013; 3: 225-227***.)

Because dropped beats may not occur, it may be incorrect to refer to this as a Wenckebach sequence. (***Castellanos A, Interian Jr. A, Cox MM and Myerburg RJ. Alternating Wenckebach periods and allied arrhythmias. Pacing Clin Electrophysiol 1993; 16: 2285-2300***.). This bizarre arrhythmia may present with syncope and is an indication for permanent cardiac pacing. Wenckebach block can also occur with ventriculo-atrial (VA) conduction and to prevent confusion would be best referred to as Wenckebach VA or retrograde block. It has been reported with the combined toxic effects of potassium and digitalis. Atrio-ventricular conduction becomes depressed resulting in complete AV dissociation with intermittent sequences of retrograde VA conduction in a Wenckebach fashion. (***Fisch C and Ridolfo AS. Reverse Wenckebach block and complete***

atrioventricular dissociation due to potassium and digitalis. Circulation 1958; 18: 852-855.)

The ECG Footprints of Sino-Atrial Wenckebach Block Sino-atrial Wenckebach block is a very poorly understood cardiac arrhythmia, the features of which, unlike Wenckebach AV block, are not readily apparent on the surface ECG because the sinus impulse can only be recorded indirectly once it leaves the sinus node and activates the atrium creating a "sinus" P wave. By examining the rhythmic sequences involving the P-P intervals, it does fulfil the footprints for Wenckebach block. Like Mobitz AV block, sino-atrial block can also be categorized as types I and II. A sino-atrial Wenckebach block sequence can be summarized as follows and illustrated in Figure 10:

Figure 10

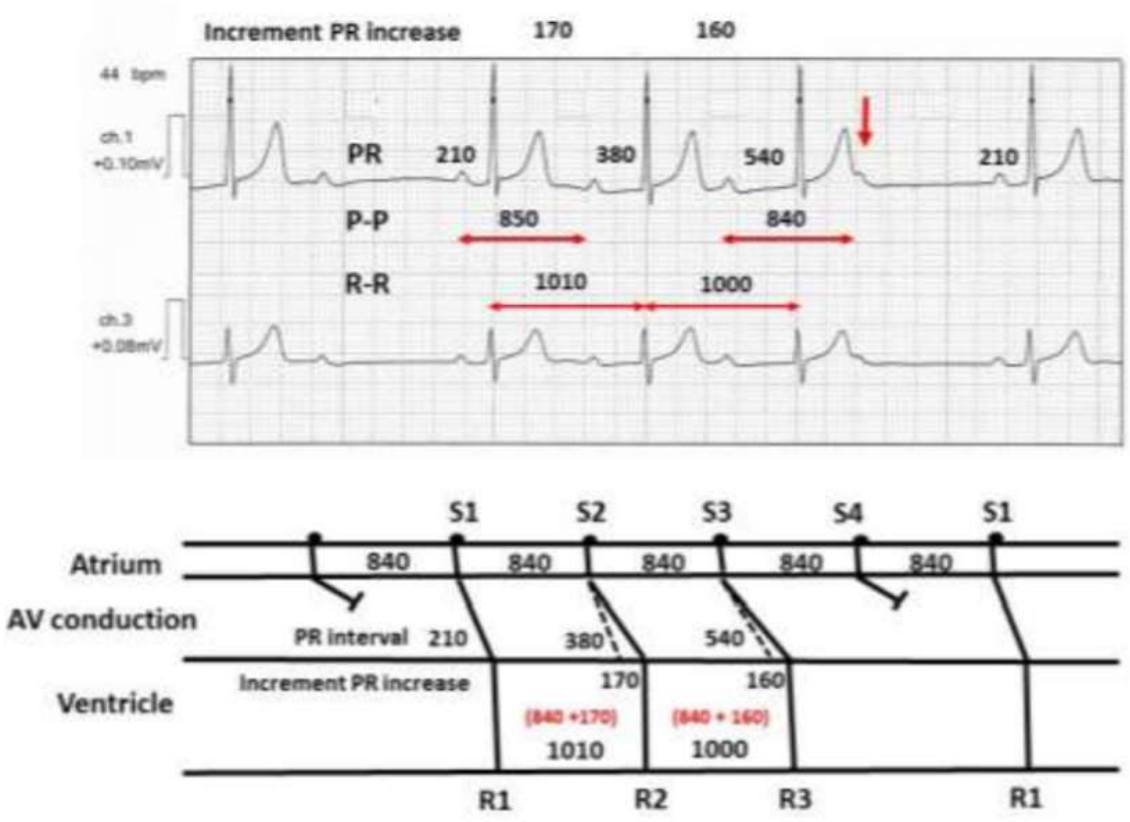


Figure 10: Above: Typical Wenckebach AV sequence. The P-P interval is steady at 840-850 ms. The PR interval (PR) increases from 210 ms to 540 ms over three complexes. The increment PR increase falls from 170 ms to 160 ms. There is then failure of P wave conduction to the ventricle (vertical arrow) and the sequence starts again. The PR interval is longest before the dropped beat and shortest after the pause. Below: Ladder diagram to explain why the R-R intervals shorten before the dropped beat. Assume no sinus arrhythmia. With each PR interval, there is a reduction in the increment increase of the PR interval from 170 ms to 160 ms. These values are added to the sinus rate of 840 ms and hence there is a small reduction in the R-R interval (increase in ventricular

rate). Abbreviations: PR = PR interval; S = Sinus; R = QRS; P-P = Sinus rate; R-R = Ventricular rate.

Figure 10

- The Wenckebach block lies within the sinus node and is thus "pre-P wave".
- Progressive fatigue of sino-atrial conduction culminates in sinus block to the atrium. The node then rests and conduction returns after one dropped beat.
- The greatest increase in sino-atrial conduction is typically between the first and second beat with the "increment increase" of each conducted beat becoming shorter. (***Graybiel A, McFarland RA, Gates DC and Webster FA. Analysis of the electrocardiograms obtained from 1,000 young healthy aviators. American Heart Journal 1944; 27: 524-549***)
- As a result, the P-P interval progressively shortens with each beat of the cycle. Because the PR intervals are constant, there is also sequential shortening of the R-R intervals. The ladder diagram in Figure 10 illustrates an almost identical sequence to AV Wenckebach with the ventricular level transposed higher to the atrial level. In the absence of sinus arrhythmia, the pause on the ECG resultant by failure of sino-atrial conduction to the atrium is generally twice the interval of the base sinus rate, which is difficult to determine as the P-P intervals are constantly shortening. 42 What is clear is that the P-P interval before the dropped beat is significantly shorter than the first P-P interval after the pause. The differential diagnosis of sino-atrial Wenckebach is a sinus ectopic beat arising from close to or within the sinus node, having an identical P wave configuration to the sinus P wave and resulting in a pause. (***Thery C, Gosselin B Lekieffre J and Warembourg H. Pathology of the sinoatrial node. Correlations with electrocardiographic findings in 111 patients. American Heart Journal. 1977; 93: 735-740***.)
- Similar pauses may occur with nonconducted atrial ectopics (**Figure 11A**). Sinus pauses can occur within the node as a result of a transient failure of sinus impulse generation although the pause may not be a multiple of the base P-P interval. Marked sinus arrhythmia can also mimic sinoatrial block, but generally the P-P and R-R intervals will gradually lengthen immediately prior to a pause in response to respiration and venous filling of the heart (**Figure 11B**).

Figure 11

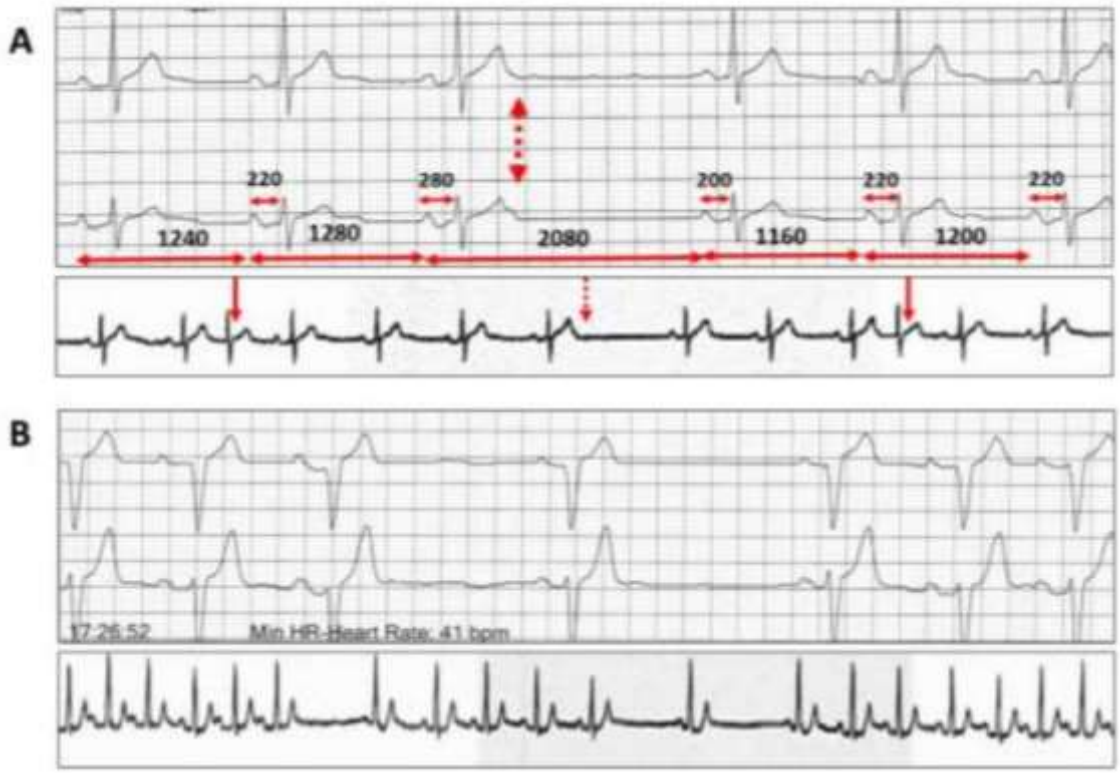


Figure 11: Differential diagnoses of sino-atrial Wenckebach. A: Non-conducted atrial ectopic. Two channel Holter monitor recording with a condensed single channel tracing below. There is a subtle Wenckebach AV sequence, but no failure of AV conduction. A non-conducted atrial ectopic (stippled vertical arrow) is followed by a compensatory pause. The condensed tracing below demonstrates conducted atrial ectopics (solid vertical arrows) as well as the nonconducted atrial ectopic (stippled vertical arrow). B: Sinus arrhythmia. Two channel Holter monitor recording with condensed single channel tracing below demonstrating first degree AV block. There are two consecutive pauses and the condensed ECG shows cyclical sinus arrhythmia. (*Friedman HS, Gomes JAC and Haft JI. An analysis of Wenckebach periodicity. Journal of Electrocardiology. 1975; 8: 307-315*.)

- A Wenckebach sequence from the sino-atrial node similar to that of the AV node was first postulated by Wenckebach and others as early as 1906. (***Thery C, Gosselin B Lekieffre J and Warembourg H. Pathology of the sinoatrial node. Correlations with electrocardiographic findings in 111 patients. American Heart Journal. 1977; 93: 735-740***.) It was rarely recognised on the standard surface 12-lead ECG, but became more apparent with Holter monitoring. As with AV conduction disturbances, it has been categorised into three degrees of block.
- First degree sinoatrial block represents an inappropriate time lapse between the generation of the sino-atrial impulse and atrial depolarization. The delay cannot be seen on the surface ECG. • Second degree sinoatrial block can also be divided

into a Wenckebach block and a type II block where there is regular sinus rhythm punctuated by one or more sinus pauses of a multiple of the P-P interval. • Third degree sinoatrial block is represented as periods of sinus arrest as a result of failure of conduction of the sinus impulse to the atrium. Although sino-atrial Wenckebach is generally regarded as benign, there is however, a high probability that patients will progress to higher levels of symptomatic sinoatrial block. (***Dąbrowski A, Piotrowicz R, Kramarz E and Kubik L. Sinoatrial Wenckebach periodicity as an independent marker for the development of high-degree sinoatrial exit block. Cardiology Journal 2007; 14: 391-395***.) It may also be a marker of sinus node dysfunction in patients presenting with syncope. The etiological factors resulting in sino-atrial Wenckebach block may be degenerative with loss of node cells particularly with age, (***Thery C, Gosselin B Lekieffre J and Warembourg H. Pathology of the sinoatrial node. Correlations with electrocardiographic findings in 111 patients. American Heart Journal. 1977; 93: 735-740***.) infiltrative with fibrous tissue and amyloid deposition (***Thery C, Gosselin B Lekieffre J and Warembourg H. Pathology of the sinoatrial node. Correlations with electrocardiographic findings in 111 patients. American Heart Journal. 1977; 93: 735-740***.), (***Evans R and Shaw DB. Pathological studies in sinoatrial disorder (sick sinus syndrome). British Heart Journal 1977; 39: 778-786***.) or post cardiac surgery such as for congenital heart disease. The sinus node may also be detached from the surrounding atrial myocardium by fibrous tissue or amyloid deposition in the peri-nodal tissue. (***Evans R and Shaw DB. Pathological studies in sinoatrial disorder (sick sinus syndrome). British Heart Journal 1977; 39: 778-786***.)

Sinoatrial block may be transient as a result of the influence of the autonomic nervous system, drugs or inflammatory processes. In the elderly, it may be a combination of both degenerative and transient factors, such as digitalis toxicity, beta blockade or 14 calcium antagonists. In sinus node dysfunction, both atrial tachyarrhythmias and rapid atrial pacing commonly result in significant pauses shown predominantly to be due to sino-atrial block, rather than overdrive suppression.(***Asseman P, Berzin GB, Desry D, Vilarem D, Durand P, Delmotte C, et al. Persistent sinus node electrograms during abnormally prolonged postpacing atrial pauses in sick sinus syndrome in humans: sinoatrial block vs overdrive suppression.***)(***Wu D, Yeh S-J, Lin F-C, Wang C-C and Cherng W-J. Sinus automaticity and sino-atrial conduction in severe symptomatic sick sinus syndrome.***

Journal of the American College of Cardiology 1992; 19: 355-364.) The simultaneous findings of sino-atrial and AV block in the same patient has been rarely reported in humans. (***Aronson RJ. Idiopathic atrial flutter, high grade atrioventricular block and sino-atrial dysrhythmia in a young man. Effects of exercise testing. Chest 1977; 72: 526-529***.)(***Bhargava K, Kler TS and Wellens HJ. An example of combined sinoatrial and atrioventricular block. Journal of Electrocardiology 2008; 41: 355-356.***)(***Kaushik JS, Gupta P, Rajpal S and Bhatt S. Spontaneous resolution of sinoatrial exit block and atrioventricular dissociation in a child with dengue fever. Singapore Medical Journal 2010; 51: e146- e148***.).

The combination may occur without symptoms or seen as the ECG manifestations of digitalis toxicity and myocarditis. In Figure 12A, the combination of sinoatrial and AV Wenckebach block is seen in the same ECG tracing. This suggests extensive disease in the conducting system. Figure 12B demonstrates a more subtle, combined sino-atrial and AV Wenckebach AV block with the absent P wave terminating a Wenckebach sequence.

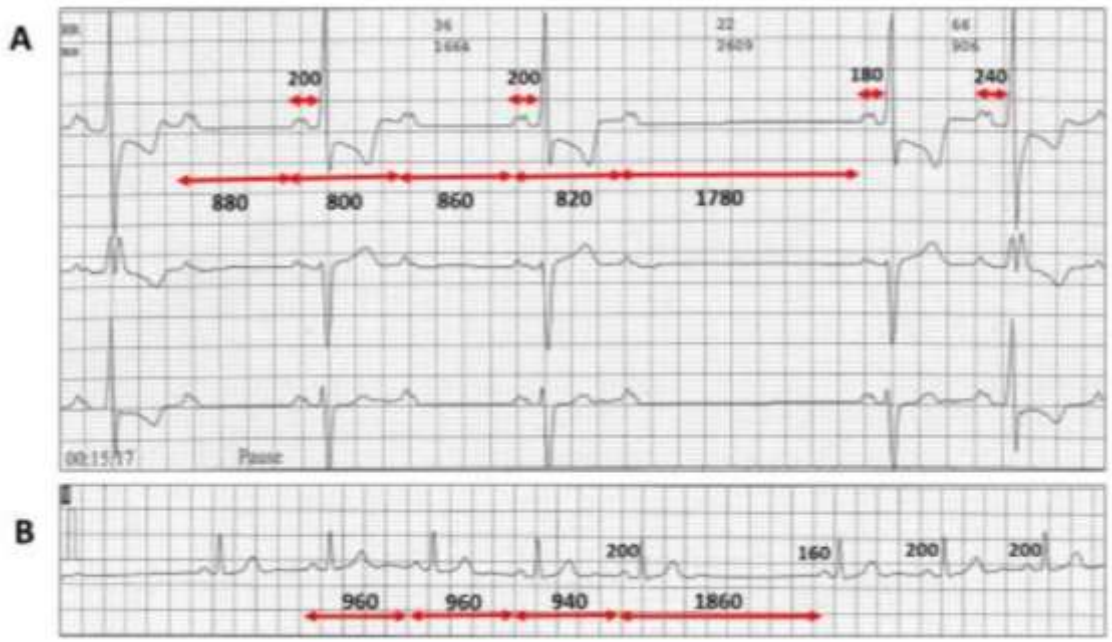


Figure 12. Sino-atrial Wenckebach and Wenckebach AV block. A: Three channel Holter monitor recording demonstrating 2:1 AV block culminating in a Wenckebach AV sequence. There is a rate dependent bundle branch block. The P-P intervals are almost regular with a shortened P-P interval before the pause which is almost equal to twice the base P-P interval and thus sino-atrial Wenckebach block. The combination of the three blocks constitutes a pan conduction defect. B: ECG demonstrating Wenckebach AV and sino-atrial Wenckebach blocks with the sequence interrupted by the sino-atrial block.

This finding is not unusual in Holter monitoring recordings. The ECG Footprints of Wenckebach The ECG Footprints of Wenckebach Block at the Ectopic Myocardial Junction Although rarely recognized and reported, Wenckebach sequences can occur at the myocardial cellular level, where ectopic tachyarrhythmias are generated and must pass an electrical barrier to depolarise surrounding muscle. This can be seen in both the atrium and ventricles.(***Schamroth L. The theory and mechanism of the Wenckebach phenomenon. South African Medical Journal 1967; 41: 827-832***.) Although the obvious features of Wenckebach are hidden, footprints can be recognized which allow identification. (**Sobotka PA, Mayer JH, Bauemfeind RA and Kanakis C. Arrhythmias documented by 24-hour continuous ambulatory electrocardiographic monitoring in young women without apparent heart disease. American Heart Journal 1981; 101: 753-759**.).

Non-Conducted Atrial Tachycardia

A focal atrial tachycardia may occur as a run of atrial depolarizations without conduction to the ventricles. In Figures 5B and 5C, a conventional Wenckebach AV sequence is interrupted by a short run of rapid non-conducted atrial tachycardia which results from a Wenckebach ectopic-atrial block at its point of generation. (***Schamroth L. The theory and mechanism of the Wenckebach phenomenon. South African Medical Journal 1967; 41: 827-832***.).

Once again, the Wenckebach sequence is not visible on the ECG and must be inferred by a sequential acceleration in the atrial tachycardia rate. Ventricular Tachycardia Ventriculo-atrial conduction occurs in approximately 50% of patients with VT. (***Wellens HJJ, Bär FW and Lie KI. The value of the electrocardiogram in differential diagnosis of a tachycardia with a wide QRS complex. American Journal of Medicine 1978:64: 27-33***.), This may manifest as a Wenckebach sequence at the ectopic ventricular junction which acts as an electrical barrier to the surrounding myocardium. The non-sustained Wenckebach sequence is not visible on the ECG and once again must be inferred by acceleration in the ventricular tachycardia rate prior to reversion or exit block. (***Oreto G, Luzza F, Satullo G and Arrigo F. Non-sustained ventricular tachycardia with Wenckebach exit block. Journal of Electrocardiology 1987; 20: 51-54***.) This is demonstrated in Figure 13 with a four-beat run of rapid non-sustained VT (187–196 bpm) with gradual shortening of the R-R interval until the tachycardia is terminated by a block at the ectopic-ventricular junction.

Figure 13

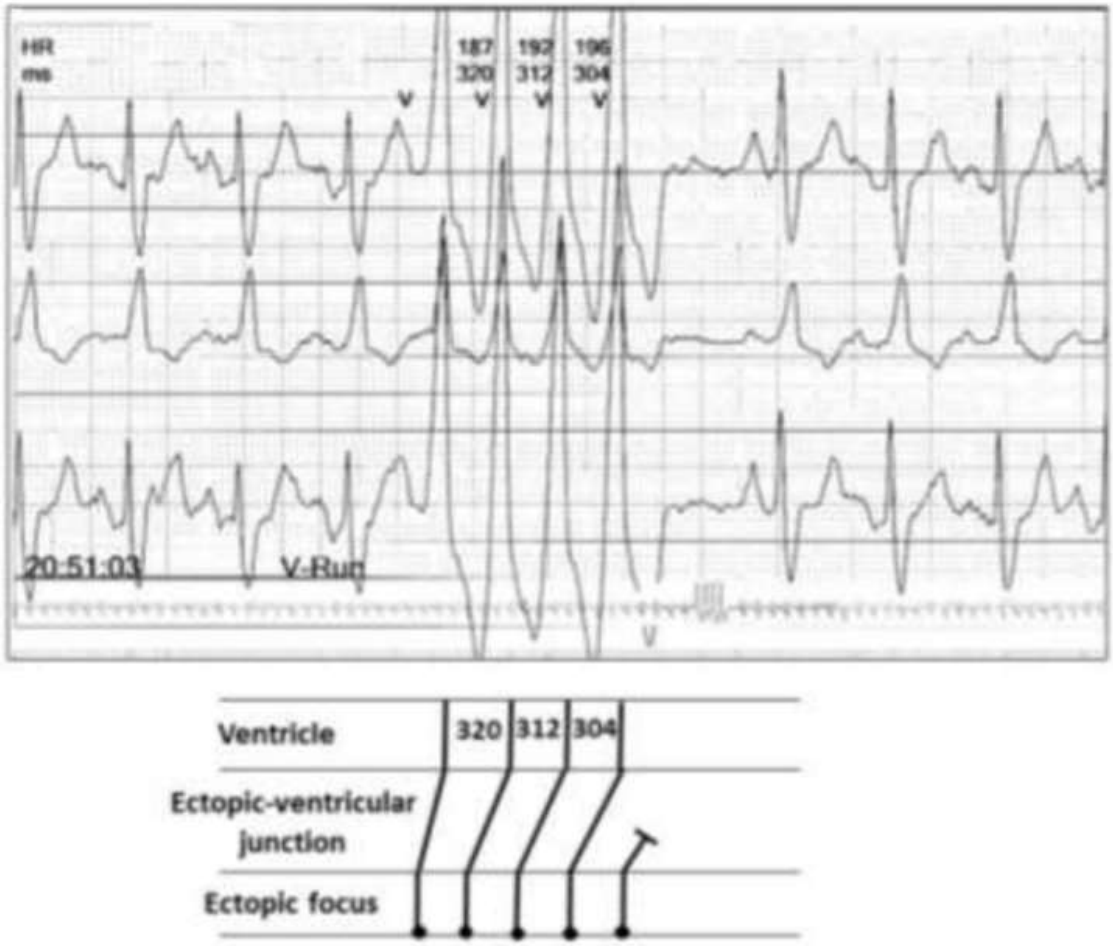


Figure 13: Above: Three channel Holter monitor recording of a four beat run of ventricular tachycardia demonstrating a Wenckebach sequence at the ectopic-ventricular junction. There is a gradual shortening of the R-R interval with the tachycardia terminated with the block. Below: Ladder diagram showing a rapidly firing ectopic focus with an increasing block

The ECG Footprints of Pacemaker Wenckebach Block Cardiac pacing can create a number of ECG patterns which can be regarded as atypical Wenckebach block. The most common of these are with implanted dual chamber systems and are specifically designed protective algorithms with electronic AV delays rather than PR intervals. Wenckebach "Upper Rate Limit" Block All dual chamber pacemakers have an upper rate limit to protect the ventricular paced response in the presence of sinus tachycardia or atrial tachyarrhythmias. This upper rate can be programmed, but is also limited electronically by the total atrial refractory period which consists of two-time intervals; the AV delay

and the period when the pacemaker is refractory to atrial sensing called the post ventricular atrial refractory period. Because these two programmable variables can significantly limit the upper rate, modern dual chamber pacemakers are also able to physiologically shorten these periods with increasing atrial rates in order to achieve respectable ventricular pacing rates with exertion. However, once the upper rate limit has been reached, the pacemaker can no longer increase the ventricular rate. If the atrial rate increases beyond this determined upper rate, the paced ventricular rate remains fixed, creating an atypical Wenckebach response with dropped ventricular paced beats as P waves fall within the refractory period. With the DDD pacing mode, which allows both atrial and ventricular pacing and sensing, an atrial paced beat will occur after a short delay. A more recognizable Wenckebach sequence will occur if there is only atrial sensing and no pacing allowed (VDD) (Figure 14).

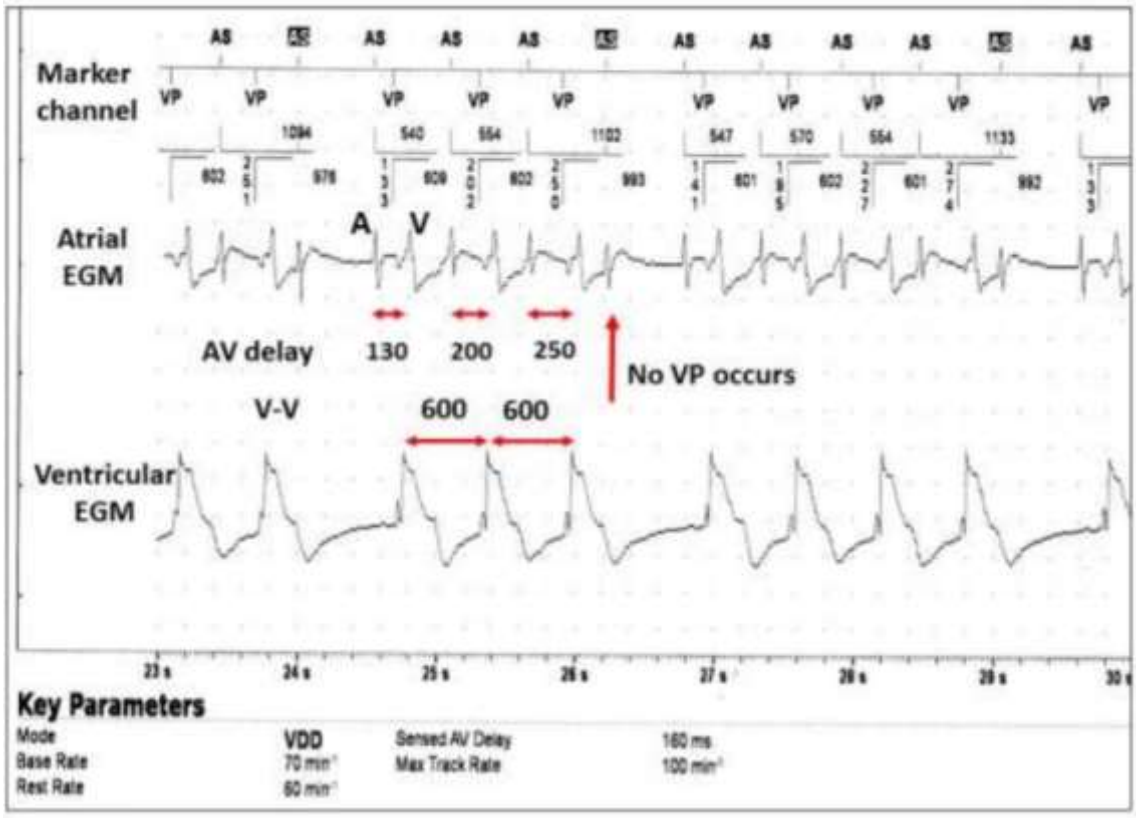


Figure 14: Pacemaker upper rate Wenckebach sequence. Dual chamber pulse generator; Identity ADx XL DR model 5386 (St Jude MedicalAbbott, St Paul, Minnesota). The patient has complete heart block and the pulse generator has been programmed VDD mode (no atrial pacing). The maximum 29 tracking rate (upper rate) has been programmed to 100 ppm. The marker channel shows atrial sensing (AS) at a rate faster than 100 bpm (570 increasing to 540 ms). The ventricular channel (VP) has a maximum tracking rate of 100 bpm (600 ms). Consequently the AV delay (electronic PR interval) gradually increases from 130 to 250 ms (horizontal arrows). The next P wave (vertical arrow), although sensed, falls within the post ventricular atrial

refractory period and thus there is no VP. The next complex has a normal programmed AV delay.

Wenckebach "Managed Ventricular Pacing" Block Managed Ventricular Pacing MVP© (Medtronic Inc, Minneapolis, MN) is a dual chamber pacing algorithm designed to minimise ventricular pacing in patients with AV conduction. When programmed ON, pacing is purely atrial or AAI with ongoing ventricular sensing in the background. If there is failure to sense a conducted ventricular native QRS, the next sensed or paced P wave is followed with a short 80 ms AV delay. Hence the footprints of a Wenckebach sequence, albeit atypical have been fulfilled in that the last AV delay before the pause is the longest, the first AV delay after the pause is the shortest and there is a dropped beat (Figure 15).

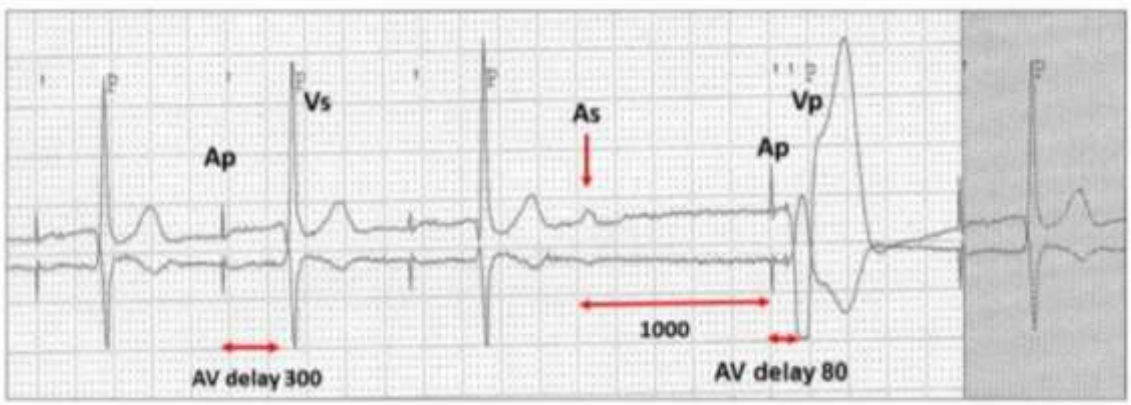


Figure 15: Two channel Holter monitor recording of Managed Ventricular Pacing (MVP©, Medtronic Inc.) demonstrating a Wenckebach sequence. There is atrial pacing (AP) followed by ventricular sensing (VS) with a long AV delay of 300 ms. A premature atrial sensed P wave (AS) occurs without AV conduction. After a pause of 1000 ms, the next AP occurs this time followed by ventricular pacing (VP) with a short AV delay of 80 ms. Although electronically created, this is nevertheless a Wenckebach sequence in that the last AV delay before the pause is the longest, the first AV delay after the pause the shortest and there is a dropped P wave. The soft "P" annotation at the top of the illustration indicates either AP or VP.

Wenckebach Pacemaker-Ventricular Block Pacemaker-ventricular block is a rarely recognized or reported block akin to the ectopic-atrial or ectopic-ventricular block discussed earlier. It is an altering temporal relationship between the stimulus artefact and the subsequent ventricular depolarization due to the toxic effects of antiarrhythmic drugs generally in the presence of profound electrolyte disturbances in terminally ill patients. (***Huang W, Lim PCY and Ching C-K. Wenckebach pattern in right bundle branch-benign or not? Journal of Electrocardiology 2017; 50: 223-226***.)(***Kistler P, Mond HG***

and Vohra J. Pacemaker ventricular block. Pacing and clinical Electrophysiology 2003; 26: 1997-1999.)(***Peter T, Harper R, Hunt D and Sloman. Wenckebach phenomenon in the exit area from a Transvenous pacing electrode. British Heart Journal 1976; 38: 201-203***.)(***Gay RJ and Brown DF. Pacemaker failure due to procainamide toxicity. The American Journal of Cardiology 1974; 34: 728-732***.)(***Moss AJ and Goldstein S. Clinical and pharmacological factors associated with pacemaker latency and incomplete pacemaker capture. British Heart Journal 1969; 31: 112-117***)

As with other forms of conduction block, there is first degree pacemaker-ventricular block or pacemaker latency, second degree pacemaker-ventricular block once again with types I and II- and third-degree pacemaker-ventricular block with loss of capture. Figure 16 demonstrates two examples of Wenckebach pacemaker-ventricular block. In both cases, there is ventricular pacing with a delay or latency between the pacemaker stimulus artefact and ventricular depolarization. Over three paced complexes, the latency proceeds as a Wenckebach sequence until there is failure to capture. (***Matta BF and Magee P. Wenckebach type heart block following spinal anaesthesia for caesarean section. Canadian Journal of Anaesthesia 1993; 39: 1067-1068***.).

Figure 16

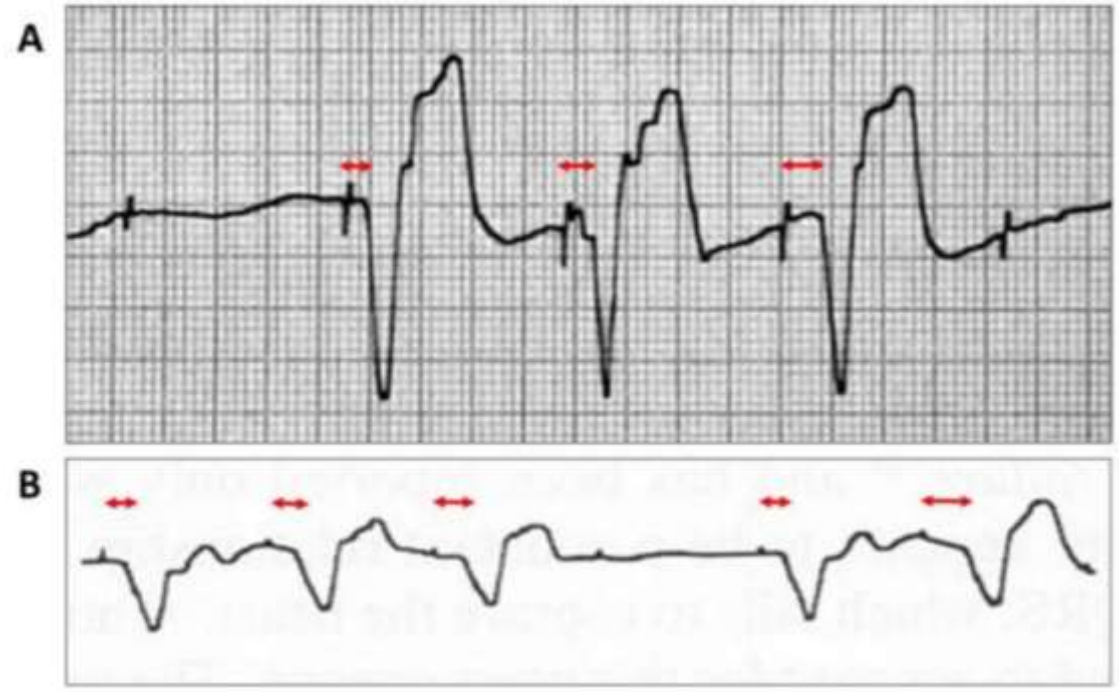


Figure 16: Wenckebach pacemaker ventricular block. In both Figures A and B there is an increasing latency (horizontal arrows) between the stimulus artefact and the paced QRS until there is loss of capture. In both cases, the ECG findings were preterminal.

Conclusion Although Wenckebach sequences are most commonly seen and recognised in the AV conducting system, similar sequences with pauses can occur in many areas of the heart and even though some appear “invisible” on the surface ECG, nevertheless they leave characteristic footprints which allow recognition.