The Wenckebach Phenomenon: A Salute and Comment on the Centennial of Its Original Description

Charles B. Upshaw Jr., MD, and Mark E. Silverman, MD

In 1899, Karel F. Wenckebach unraveled the complicated arrhythmia that bears his name—one of the most famous eponyms in medicine. He reported his findings before the benefit of clinical electrocardiography or the discovery of the sinoatrial and atrioventricular nodes. Born and educated in the Netherlands, Wenckebach first worked in Utrecht in the physiology laboratory of T.W. Engelmann, his respected mentor, where he became familiar with kymographic recordings and rhythm disturbances in frog experiments. He then entered country practice in 1891, where he gained great respect for practicing physicians as well as the importance of clinical experience. In 1896, he returned to Utrecht to work again in the laboratory with Engelmann. In 1898, a woman consulted Wenckebach about her irregular pulse. His investigation of her irregular heart action by using radial arterial pulse tracings and experimental atrial and ventricular pulse tracings from the heart of a frog enabled him to discover the mechanism of partial heart block. In later years, he continued to be a leader in academic medicine, chairing the departments of medicine in Groningen, Strasbourg, and Vienna. He achieved fame for investigating cardiac arrhythmias and other contributions and is considered to be one of the founders of modern cardiology. He is remembered for his insight into atrioventricular conduction, which is as valid today as it was a century ago.

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From Piedmont Hospital and Emory University School of Medicine, Atlanta, Georgia. For current author addresses, see end of text.

The Wenckebach phenomenon, described by Karel Frederik Wenckebach of Utrecht, the Netherlands, in his 1899 paper "On the analysis of irregular pulses" (1), is one of the most famous eponyms in medicine. Wenckebach discovered this arrhythmia, now classified as Mobitz type I second-degree atrioventricular block (2), before the introduction of electrocardiography (3) by careful mathematical analysis of the relation between atrial and ventricular pulses. Our purpose is to review Wenckebach's remarkable report and to honor him for his major contribution to cardiology on the occasion of the 100th birthday of its publication. In so doing, we discovered an additional conduction disturbance in his tracings not fully appreciated in the original report.

Early Life

Wenckebach (**Figure 1**) was born on 24 March 1864 in The Hague, the Netherlands (4, 5). His father was an engineer and his grandfather was a justice in the supreme court in The Hague. When Wenckebach was 10 years old, his father died, and his family moved to Utrecht, where he attended high school. In 1881, he entered the University of Utrecht Medical School and, as a student, published several papers on embryology. After his graduation in 1888, he began his career at a zoologic institute. Color blindness, however, limited his studies, and he turned to a career in physiology. He was fortunate to work in Utrecht in the laboratory of Theodur Wilhelm Engelmann, a pioneer of cardiac electrophysiology, who was interested in inducing rhythm disturbances in frogs and studying the relation of excitation to contraction (4). With Engelmann's guidance, Wenckebach became familiar with the latest techniques of kymographic recording and rhythm disturbances in frog experiments.

At the age of 27 in 1891, Wenckebach left Utrecht (partly for financial reasons) and entered a country practice in Heerlen, where he married in 1892. Wenckebach soon gained great respect for practicing physicians and the importance of clinical experience (6). Fascinated by irregular pulses, which were common in his elderly patients, he enjoyed telling the story in which he listened to a patient's heart for so long that she thought he had fallen asleep (5). His interest in pulse irregularities prompted a return to Utrecht in 1896, where he could continue to practice medicine and resume studies in the physiologic laboratory. In 1898, Wenckebach and, independently, Arthur Cushny first reported that the ventricular extrasystoles with compensatory pauses observed in animal experiments also occurred in humans (5). Wenckebach also demonstrated that atrial extrasystoles were not accompanied by a compensatory pause.

Case Report

On 14 May 1898, Wenckebach saw the patient whose pattern of pulse irregularity would stimulate him to decipher the rhythm disturbance that became known as *Wenckebach periodicity* (1).

"A forty-year-old lady with nervous temperament" consulted Wenckebach about an irregular pulse. "She had noticed for a long time that her pulse was irregular. Her only heart trouble was a mild dilatation. Nevertheless, she suffered from deficient circulation and had the appearance of a weak and anemic person. The pulse was small, soft and intermittent after 3 to 6 contractions. Her heart sounds were weak but clear. Striking was that there was no extrasystole at all, as usually goes along with intermittence of the pulse." The patient also did not have symptoms that occur with extrasystoles, such as "a thud in the cardiac region" or being conscious of the pause or "the large blood wave of the postcompensatory systole."

Believing that the pulse was "the mirror of the heart," Wenckebach used single-channel radial arterial pulse curves (sphygmograms) to study the irregular pulse of his patient, measuring time according to the intervals of a tuning fork at 1/10th of a second. On analyzing her radial arterial pulse curves (**Figure 2**), he noted regular pauses every 3 to 4 beats, but the small extra pulse waves seen during pauses after extrasystoles were absent. Furthermore, the length of the pauses, which were expected to be twice the length of the preceding pulse interval if the pauses were due to extrasystoles, was actually less than twice the preceding pulse interval. Wenckebach concluded that the regular and repeated pauses of the patient's pulse were not caused by extrasystoles. Further observations on the radial arterial pulse curve disclosed that the first interval after each pause was longer than the others. Subsequent intervals were shorter; however, the last interval before the pauses was sometimes lengthened slightly. The pattern of changing pulse intervals repeated again and again, displaying groups of beats that Wenckebach called "Luciani periods," after the Italian physiologist Luigi Luciani who, in 1873, observed group beating in a frog's heart while working in Carl Ludwig's laboratory in Leipzig, Germany (7).

In summary, from his analysis of the radial arterial pulse curves, Wenckebach made the following observations:

Figure 1. **Karel Frederik Wenckebach, MD.**

1. There were groups of beats.
2. The length of the pauses was less than twice the interval between the preceding pulses.
3. The interval between the first two pulses after the pause was longer than the other pulse intervals.
4. There was no evidence of extrasystole.

Wenckebach was at a loss to explain these data and turned to Engelmann for help. Engelmann made available polygraph tracings recorded on 5 December 1893 from a frog's heart by using the method of suspension (8). This technique of recording simultaneous atrial and ventricular contractions permitted measurement of the time between atrial and ventricular pulses; time was registered according to the intervals of a tuning fork at 1/25th of a second. In studying the frog data from Engelmann's

Figure 2. **Selected segment of a single-channel radial arterial pulse tracing from the patient.** Pauses are seen every three to four beats, each pause is less than twice the preceding pulse interval, and the first pulse interval after each pause is longer than the other intervals. The numbers below the pulse curve refer to the intervals between beats; the numbers above the pulse curve measure the pulse upstroke time from baseline to the greatest upward displacement of the pulse curve (measurements were made by Wenckebach). The pulse upstroke time is shortest after a pause, suggesting maximum rate of left ventricular ejection. All numbers are expressed in tenths of a second; the timeline beneath the pulse curve has a frequency of one notch every 1/10th of a second; and the paper speed, by our measurement, is 15 mm/s (range, 13 to 16 mm/s). Sinus and ventricular rates are 83 and 63 beats/min, respectively. This figure has been lightly retouched for clarity.

Figure 3. Polygraph tracing from the left atrium and ventricle of a frog with our measurements and labeling of the laddergram. The following phenomena are seen: beats occur in groups, the atrioventricular (*AV*) pulse intervals gradually increase until the pause, the pause is less than twice the preceding ventricular pulse interval; and the first AV interval is shortest after the pause. These findings are consistent with type I second-degree atrioventricular block. In addition, the intervals between the firing of the cardiac pacemaker and the atrial pulse waves (*SA*) gradually increase until the pause; the atrial pulse (*A*) pause is less than twice the preceding atrial pulse interval; the first SA interval is shortest after the pause; the atrial pulse intervals gradually shorten after the pause; and the atrial pulse wave is absent during the pause, indicating the additional presence of type I second-degree sinoatrial block. Numbers 1 through 14 above the top line of the laddergram refer to sequential sinus impulses; numbers under the atrial pulse waves and the numbered *V*s above the ventricular pulse waves refer to connected atrial and ventricular pulses, respectively. Arrows on the laddergram point in the direction of impulse propagation. Intervals are expressed in 1/25th of a second, and diminutive notches on the timeline above the laddergram have a frequency of one notch every 1/25th of a second; paper speed by our measurement is 12 mm/s (range, 11 to 13 mm/s). Sinus, atrial, and ventricular rates are 44, 38, and 32 beats/min, respectively. This figure has been lightly retouched for clarity. V = ventricular pulse waves.

laboratory (**Figure 3**), Wenckebach noted that the intervals between atrial and ventricular pulses gradually increased until the ventricular pulse wave was absent, resulting in a pause. After the pause, the first atrioventricular pulse interval became the shortest. In our review of the tracing, we also note a paradoxical lengthening of the last interval between the atrial and ventricular pulse waves and the interval between ventricular pulse waves before the pause, now recognized as the most common deviation from the classic structure of the Wenckebach phenomenon. This deviation may be due to changing atrial pulse intervals and atrioventricular conduction (Marriott HJ. Personal communication; 9). In addition, the atrial pulse wave sometimes "jumps over the top" of the previous ventricular pulse wave (for example, the 5th and 12th atrial pulse waves), indicating that the atrioventricular pulse interval is longer than the ventricular pulse interval (Marriott HJ. Personal communication). This may be the first time that this phenomenon was documented, although Wenckebach did not comment on it.

A polygraph tracing recorded "at a later stage of dying of the frog's heart" (**Figure 4**) showed changes similar to those in the atrioventricular pulse intervals relative to the pause (**Figure 3**), but now the greatest increase in the atrioventricular pulse interval occurred with the second beat after the pause. In addition to the absence of the ventricular pulse wave during the pause, the atrial pulse wave was also missing. Before the discovery of the sinoatrial node in 1907 (10), Wenckebach believed that the "rhythmic excitation [of the heart] arises from the root of the heart next to the mouth of the vena cava." He attributed the absence of the atrial pulse wave to injured conduction between the root of the heart and the atria. He postulated that the group beating, along with the changes in the atrioventricular pulse intervals, was due to "regular heart action disturbed by a regular constant influence": that is, decreased conduction of the heart muscle at the atrioventricular border. (The atrioventricular node would not be discovered until 1906 [11].) He explained (1) that

> A strong suppression of conductibility and its consequences is demonstrated best . . . with the A_s [atrial systole]-V_s [ventricular systole] interval of a dying frog's heart. As heart action gradually becomes worse, the A_s-V_s interval gets longer, caused by decreased conductibility. The interval A_s-V_s can widen so much that V_s only starts very shortly before the beginning of the following A_s. Anyone who did not follow the slow development of this phenomenon could get the impression that A_s follows V_s, and so a reversal of the usual sequence of contraction. With conductibility de-

Figure 4. Selected segment of polygraph tracing from the left atrium and ventricle of a frog with our laddergram added, showing simultaneous type I second-degree atrioventricular block and type I second-degree sinoatrial block. Numbers 1 through 15 above the top line of the laddergram refer to sequential sinus impulses; numbers above the atrial pulse waves and the numbered *V*s under the ventricular pulse waves refer to connected atrial and ventricular pulses, respectively. Numbers below the notched timeline and those above the ventricular waves are measurements made by Wenckebach of intervals between atrial and ventricular pulse waves and intervals between ventricular pulse waves, respectively. Intervals are expressed as 1/25th of a second; diminutive notches on the timeline above the laddergram have a frequency of one notch every 1/25th of a second. Paper speed by our measurement is 9 mm/s. Sinus, atrial, and ventricular rates are 34, 28, and 28 beats/min, respectively. This figure has been lightly retouched for clarity. A = atrial pulse waves; AV = intervals between atrial and ventricular pulse waves; SA = intervals between the firing of the cardiac pacemaker and the atrial pulse waves; V = ventricular pulse waves.

> creasing, there finally comes a time when the excitation is not conducted anymore, A_s-V_s equals infinity, and the contraction of the ventricle is absent. During this pause, caused by the absence of ventricular contraction, conductibility has time to recover, so the next excitation is conducted again, and the ventricle contracts.

Wenckebach next constructed "a model of heart action" (**Figure 5**) based on his analysis and understanding of the radial pulse and polygraph tracings. (The laddergram was devised by Chauveau in 1885 and Engelmann in 1896 [12].) The model illustrated the following points:

Figure 5. Schematic "model of heart action" drawn by Wenckebach showing type I second-degree atrioventricular block. The numbers above the lowest horizontal line represent the delay in conduction at the atrioventricular border; the shortest interval occurs after a pause; the greatest increment occurs with the next beat; the intervals gradually increase to the maximum; and the pause is less than twice the preceding ventricular pulse interval. Time is shown on the abscissa, and paper speed is not specified. Numbers 1 through 7 at the top and bottom of the laddergram refer to sequential heart beats; all other numbers are intervals in unspecified time units. This figure has been lightly retouched for clarity. ρ = cardiac pacemaker at the root of the heart; A = conduction through the atria (the middle horizontal line represents the atrioventricular border); TV = time between ventricular complexes; V = conduction through the ventricles.

1. There were small groups of heart beats.
2. Each ventricular pulse interval progressively shortened after a pause.
3. The longest ventricular pulse interval (pause) was less than twice the shortest ventricular pulse interval.
4. Atrioventricular conduction was best after a pause.
5. The greatest increase in atrioventricular conduction occurred in the second beat after the pause.

These findings comprise the typical features now known as Wenckebach periodicity or *type I second-degree atrioventricular block.*

In comparing the radial arterial pulse tracings of his patient with the polygraph tracings from the frog, Wenckebach was "led to the astonishing conclusion that in both cases the regularly repeated irregularity is exactly the same" and that the pattern of pauses and varying atrioventricular pulse intervals was "repeated with almost mathematical precision." He inferred that these changes were the result of damaged conduction of the heart muscle at the atrioventricular border. He also noted that the pause of his patient's pulse was least frequent when the heart rate was slowest. Moderate exercise by the patient led at first to fewer pauses, but more frequent pauses then occurred as a stage of fatigue followed. He provided a follow-up of his patient:

> And indeed it was possible to watch the improvement of heart action in our case . . . after a long rest, nourishing food and a stay in the country, the patient was examined again on June 19, 1898, and the pauses were seen less frequently. Later, October 23, 1898, when the patient was again available for sphygmographic examination, regular heart action was recovered and the regular pauses had disappeared . . . the cure was for the patient certainly very pleasant.

In the laddergrams that we drew below **Figures 3** and **4**, we noted an additional level of conduction disturbance not fully appreciated in the original report. All of the measurements in the laddergram for **Figure 3** are our own; Wenckebach made no measurement on this tracing. In drawing the laddergram for **Figure 4**, we faithfully used Wenckebach's original measurements, bearing in mind his admonition of the difficulty in measuring pulse tracings: "One has to consider that small errors can't be avoided, because it is not always possible to find the exact beginning of A_s and V_s." Although Wenckebach attributed the absence of atrial pulse waves to damaged conduction between the root of the heart and the atria, he failed to note that the pattern of sinoatrial conduction was also similar to the progressive conduction delay between the atria and the ventricles that he had described so skillfully. The laddergrams in **Figures 3** and **4** show that 1) the atrial pulse waves are absent during pauses, 2) sinoatrial conduction is best in the first beat after pauses, 3) the intervals between atrial pulse waves shorten after pauses, and 4) atrial pulse pauses are less than twice the preceding atrial pulse interval. These findings indicate the additional presence of type I second-degree sinoatrial block, now commonly referred to as *Wenckebach sinoatrial block.*

Because the sinus node impulse is "silent," type I second degree sinoatrial block can be inferred only indirectly by a dropped atrial pulse and through the effect of the lengthening sinoatrial conduction time on the atrial pulse intervals. The measurement and its graphic illustration are accomplished as follows:

1. The total interval of the group from the first atrial pulse after a pause to the first atrial pulse after the next pause is measured in millimeters.
2. This interval is divided by the number of atrial pulses in the group plus 1 added for the "dropped" atrial pulse to obtain the sinus node discharge rate in millimeters per beat.
3. The paper speed (in millimeters per second) is divided by the sinus node discharge rate and is multiplied by 60 seconds to obtain the sinus node discharge rate in beats per minute.
4. To demonstrate sinoatrial conduction, a dot is arbitrarily placed in the sinoatrial tier of the laddergram 3 mm before the first atrial pulse after a pause to represent sinus node discharge. By using the derived sinus node discharge rate (see step 2,

above), subsequent dots are placed at regular intervals. Each dot is connected to the beginning of the next atrial pulse in the atrial tier of the laddergram.

In **Figure 4**, the varying atrioventricular intervals indicate the gradual development of type 1 second-degree atrioventricular block, but total block of the sinus impulses does not occur at the atrioventricular border because the impulses never reach this level; instead, they are blocked at the more proximal sinoatrial level. Therefore, the pauses are due to sinoatrial block and not to atrioventricular block. In **Figure 3**, however, the pauses are due to simultaneous type I second-degree block at both the sinoatrial and atrioventricular levels.

Type I second-degree sinoatrial block is an uncommon cardiac arrhythmia (13, 14). The combination of type I second-degree sinoatrial and atrioventricular block is rare; we are aware of only five reports (other than the present one) of this simultaneous conduction system disturbance (15–19).

Later Life

With his reputation greatly enhanced by his work on arrhythmias, Wenckebach was chosen to be chair of medicine at the University of Groningen in the Netherlands in 1901. In 1903, he published his classic book *Arrhythmia of the Heart*, dedicated to Engelmann, in which he discussed the myogenic action theory of the heartbeat and provided a detailed analysis of experimental and clinical rhythm disturbances along with case histories and clinical observations. The book, originally published in German, was translated into English in 1904. A second edition in 1914 received widespread attention and respect. Between 1902 and 1905, Wenckebach published articles on the treatment of septic endocarditis and the use of animal endocrine extracts (organotherapy).

Wenckebach developed a correspondence and close friendship with James Mackenzie of Burnley, England, a general practitioner who was one of the first to investigate cardiac rhythm disturbances in humans by using a polygraphic technique to analyze simultaneous jugular venous and arterial pulses. In 1902, Mackenzie published *The Study of the Pulse*, which received little attention in England but drew the admiration of Wenckebach. Together, Mackenzie and Wenckebach opposed the commonly held belief that extrasystoles always implied serious heart disease.

After Willem Einthoven introduced the string galvanometer electrocardiograph, Wenckebach was able to demonstrate in 1906 that the PR intervals lengthened before the dropped beat (16)—a confirmation of his previous observations on second-degree atrioventricular block in frogs. In the same article, he described a muscle band arising from the superior vena cava extending across the sulcus terminalis that he thought might transmit the sinus impulse (16). This became known as the *Wenckebach bundle.*

From 1911 to 1914, Wenckebach was professor and chair of medicine at the University of Strasbourg, then in Germany. During this period, he reported studies on the relation of respiration and circulation, on pericarditis, and on the first use of quinidine sulfate for converting auricular fibrillation (brought to his attention in 1912 by a merchant from the Dutch East Indies, who confided that quinine as a tonic also relieved palpitations). He became chair of medicine at the University of Vienna in 1914. During World War I, he investigated the important problem of soldier's heart. He later became involved with the prevalent problems of malnutrition, establishing a clinic to study rickets as well as directing relief work in Vienna to help malnourished children. After that war, Wenckebach was a highly sought-after consultant, and many students, celebrities, and eminent figures in medicine visited his Vienna clinic.

His interest in beri-beri heart disease led him to study the illness in the Dutch East Indies; in 1934, Wenckebach published a book that provided the basis for recognition of this disease. A two-volume work, *Die unregelmassige Herztatigkeit* [Irregular cardiac activity], was published in 1927 with Heinrich Winterberg as coauthor (20). This book brought together his extensive experimental and clinical experience and fully incorporated the electrocardiogram and pulse tracings into the interpretation of arrhythmias. In 1929, at 65 years of age, Wenckebach retired from his position at the University of Vienna. In 1940, at 76 years of age, he died in Vienna of sepsis related to kidney infection.

Wenckebach achieved fame as a pioneer in investigating cardiac arrhythmias, as a consultant who was a master of physical diagnosis, and as a founder of modern cardiology. However, he remained modest and unaffected, saying, "No, I am not a great man; I am a happy man" (21). His colleagues called him "Venky" and considered him a lovable person with unusual charm and a keen sense of humor. His unflagging optimism and enthusiasm were widely admired. His friends and colleagues were international; James Mackenzie, William Osler, and others considered him to be an effective link between European and British medicine (21, 22). We remember him primarily for his important insight into the atrioventricular conduction to which his name is firmly attached and which is as valid today as it was a century ago in 1899.

Summary

It is appropriate to honor Wenckebach on the centennial of his astute discovery and analysis of

type I second-degree atrioventricular block, commonly known as *Wenckebach atrioventricular block.* We salute him for his keen observation and understanding of this complicated arrhythmia by using radial arterial pulse curves obtained from his patient and using Engelmann's polygraph pulse curves obtained from the atrium and ventricle of a frog. We note the additional presence of type I second-degree sinoatrial block in the frog's tracings; this phenomenon was not noted by Wenckebach in his original paper. Wenckebach's observations and deductions were all the more remarkable considering that they were made before the introduction of clinical electrocardiography or the discovery of the sinoatrial and atrioventricular nodes. His insight into atrioventricular conduction was confirmed by electrocardiography and remains as true today as it was in 1899.

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Requests for Reprints: Charles B. Upshaw Jr., MD, 35 Collier Road, NW, Suite 425, Atlanta, GA 30309.

Current Author Addresses: Dr. Upshaw: 35 Collier Road, NW, Suite 425, Atlanta, GA 30309.
Dr. Silverman: 1968 Peachtree Road, NW, Atlanta, GA 30309.

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