Simultaneous Sinoatrial and Atrioventricular Wenckebach Phenomena*

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The electrocardiograms of a patient who showed atrial flutter, atrial fibrillation, and most interestingly, simultaneous Wenckebach phenomenon in the atria and atrioventricular node, are presented.

CASE REPORT

The patient was a 45-year-old man who had onset of palpitations, shortness of breath, and dull anterior chest pain on the morning of March 5, 1971. The man consulted his local physician who made the diagnosis of atrial flutter with 2:1 AV conduction and gave the patient 1.2 mg deslanoside (Cedilanid-D) and additional doses of 0.4 mg at 20-minute intervals for a total dose of 2 mg. Since the above therapy produced only transient 3:1 AV block, the patient was admitted to the Cardiology Service, Forest Hills Division, VA Hospital, Augusta, Georgia, two hours after his last dose of digitalis.

The patient had mild anterior chest discomfort on admission and received 50 mg of meperidine intramuscularly on two different occasions following admission. The patient's physical examination gave normal findings except for tachycardia of 150 per minute. His electrocardiograms never revealed evidence of myocardial ischemia or infarction. However, his SGOT rose from 29 units on the second day of admission to 80 units on the fourth day and was back to 21 units on the sixth day of hospitalization.

The electrocardiographic strips shown in Figure 1 were taken in the coronary care unit about five hours after the patient had received 2 mg deslanoside (Cedilanid-D). They

![Figure 1a. Atrial flutter with an atrial rate of 300/min and 2:1 AV block, recorded at 7:15 pm, five hours after administration of deslanoside. 1b. Rhythm has now converted to atrial fibrillation with widely varying ventricular response. 1c. The patient has now converted to sinus rhythm at a rate of 120/min.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21529/ on 06/27/2017)
SINOATRIAL AND ATRIOVENTRICULAR WENCKEBACH PHENOMENA

FIGURE 2. Three Wenckebach periods are shown. A ladder diagram is used to illustrate this arrhythmia. The time intervals are in milliseconds. The sinus node (S) rate of firing was estimated by dividing the time interval from the P wave of one Wenckebach period to the P wave of the next Wenckebach period by the four sinus cycles present. SA represents the sinoatrial conduction time which progressively lengthens until a P wave is dropped. A represents the atrial cycle. AV represents the atrioventricular conduction time which progressively lengthens. It so happens that the dropped beat is due to nonconduction of the sinus node impulse, allowing the AV node time to recover and preventing nonconduction through the AV node. V represents the ventricular cycle.

(An alternative explanation for this arrhythmia might be multiple premature atrial beats that occur with increasing prematurity. However, it seems unlikely that this condition would establish a repetitive pattern.)

are aliquots from continuous strips. Figure 1a shows atrial flutter with an atrial rate of 300/min and 2:1 AV conduction. The tracing in Figure 1b reveals that the patient has converted to atrial fibrillation with a ventricular response of between 60 and 150 per minute. Figure 1c shows that the patient has converted to sinus rhythm at a rate of 120 per minute.

Figure 2 is lead II of the electrocardiogram, taken six hours after the administration of 2 mg Cedilanid. This tracing reveals the characteristic Wenckebach phenomena involving atrial and atrioventricular nodal conduction (Fig 2, caption.)

The patient's rhythm and conduction subsequently returned to normal and remained so. Efforts to substantiate by independent means a diagnosis of organic heart disease failed and he was classified according to New York Heart Association as "no heart disease found: undiagnosed manifestation."1

The Wenckebach phenomenon is characterized by two features that result in grouping of the beats:2 (1) the conduction time progressively lengthens until one beat fails to conduct, producing a pause that is less than the length of two short cycles; (2) the increment of conduction lengthening is greater between the first and second beat and becomes progressively smaller thereafter. A seemingly paradoxical situation thus develops that results in acceleration of the heartbeat while the degree of conduction delay is actually increasing.

The Wenckebach phenomenon may occur in any of the myocardial conduction tissue.3 Winton3 also defined criteria for establishment of the Wenckebach phenomenon in the sinoatrial node that correspond to the previously listed characteristics.

A common manifestation of impaired conduction is the Wenckebach phenomenon.2 The impaired conduction may be related to organic disease of the conduction tissue, to the effect of drugs, or to vagal stimulation. The mechanism of the Wenckebach phenomenon is dependent upon the fact that the conducting tissue receives the impulse before it has fully recovered from the previous one. Therefore, each impulse occurs earlier in its relative refractory period and is conducted more slowly until the impulse arrives in the absolute refractory period and results in a dropped beat. After the pause, the conduction tissue recovers and the Wenckebach period starts over.

The Wenckebach phenomenon is sometimes seen in atrial flutter. However, this phenomenon did not manifest itself in our patient until he had converted to sinus rhythm. Digitalis has a direct effect on the atria and AV node which increases the refractory period and decreases the conduction velocity.4 The vagal effect of digitalis produces an additive effect at the AV node. The Wenckebach phenomenon was present only transiently in this patient and it is likely that its etiology is due to the effect of digitalis on the conduction tissue. In addition, the patient had chest pain and a rise in his SGOT. Therefore, myocardial ischemic injury, though not established,
may have played an additional role.

In conclusion, an electrocardiogram has been presented which showed the simultaneous development of the Wenckebach phenomenon in the atria and the atrioventricular node. The characteristic grouping of beats should be eye-catching and a hint that the Wenckebach phenomenon is present.

REFERENCES

Subtle Biochemical Assaults of Cigarette Smoking

With due regard to the biologic and psychologic complexities of cigarette smoking, it seems reasonable to assume that subjective gratification derived from it may be attributable to its stimulating or to its tranquilizing effect. In experimental animals intravenously administered nicotine labeled with radioactive C$^{14}$--in amounts corresponding to cigarette smoking--is followed by accumulation of nicotine in the central nervous system, particularly in the gray matter of the brain. The nicotine disappears within 30-60 minutes. Nicotine releases norepinephrine and epinephrine from their widespread sources. Norepinephrine stimulates the cortical areas of the brain. In animal experiments, increased activity was recorded in encephalograms following nicotine given intravenously. Also, the latter results in substantial increase in the oxygen consumption of the brain. Augmented release of catecholamines results in a sense of enhanced physical strength, increased cardiac output, dilatation of blood vessels in the voluntary muscles, and transient hyperglycemia. The tranquilizing influence of cigarette smoking (sense of ease and relaxation) may be due to increased production of corticosteroids by catecholamines and possibly by abnormally high levels of histamine. Pleasant lassitude or languor may result from hyperventilation due to stimulation by nicotine of the carotid and aortic bodies. Smoking-induced elevated carbon monoxide levels of the blood may be conducive to cerebral hypoxia with consequent pleasurable sensation. Over 1200 chemicals are inhaled with cigarette smoke. Some of them are known to have serious pathogenicity. Nicotine causes tachycardia and temporary elevation of the blood pressure. Hass, G et al (Circulation 38, Supplement 6, 8, 1968) observed that in rabbits injections of nicotine induced peripheral atheromatous, calcifying arterial changes and thromboarteritis in the myocardial vessels. Others recorded elevated blood cholesterol, thickening of the walls of arteries and arterioles in smokers. Godber, G E (Am J Publ Health 60:235, 1970) says that in men of 40-49 years of age who are heavy cigarette smokers the mortality from coronary disease is almost fourfold of that in nonsmokers. According to Pilgeram, L O et al (J Atheroscler Res 8:155, 1968) cigarette smoking causes accelerated fibrinogen synthesis and clotting. Augmented release of epinephrine may increase histamine in the blood. In addition, histamine is liberated in situ from disintegration of leukocytes in patients with chronic bronchitis, with consequent bronchospasm. Ladd, B et al (Dis Chest 43:151, 1963) reported that 90 per cent of patients with Tokyo-Yokohama asthma were smokers. Cigarette smoke contains 3.2 percent carbon monoxide. It raises carboxyhemoglobin levels of the blood to 4-6 percent in moderate smokers and up to 12 percent in heavy smokers. Experimental investigations reveal that such high concentrations of carbon monoxide may cause degenerative changes in the myocardium. Harkavy, J (Ann Allergy 26:447, 1968) states that tobacco extract is both antigenic and allergenic. In smokers in whom allergy to tobacco played either a primary or secondary role, he found the following: various arrhythmias, extrasystoles, auricular fibrillation, tachycardia, tobacco angina, coronary artery disease, migrating phlebitis, thromboangiitis obliterans, intermittent claudication and peripheral arteriosclerosis. From a random series of 200 atopic patients, Zussman, B M (Ann Allergy 28:371, 1970) found 16 percent to be clinically sensitive to tobacco smoke, manifesting nasal, ocular symptoms and attacks of asthma. Hydrogen cyanide, a component of tobacco smoke is strongly cytotoxic in relation to respiratory enzymes. Nitrogen dioxide and other irritants in tobacco smoke are likely causes of bronchospasm. Also, cigarette smoke contains several ciliotoxic agents. Interference with bronchocatharsis by inhibited ciliary function, and by bronchospasm contribute to the retention of carcinogens of cigarette smoke.

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