Case Reports

Unusually Marked Sinus Arrhythmia as a Complication of Acute Diaphragmatic Myocardial Infarction

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SUMMARY

An instance of markedly irregular sinus arrhythmia associated with acute diaphragmatic myocardial infarction has been described. It is quite unusual to observe an extreme sinus arrhythmia in an adult without the intervention of A-V nodal or ventricular escape rhythm.

The influence of digitalis, reserpine, guanethidine and morphine in relation to such cardiac arrhythmia has been discussed.

Sinus arrhythmia and/or sinus bradycardia are usually transient and require no treatment unless cardiac output is compromised. This arrhythmia may be mistaken for more serious arrhythmias such as A-V block, S-A block and sinus arrest because of marked irregularity with slowing of the heart rate.

Additional Indexing Words:

Sinus arrhythmia Arrhythmias in acute myocardial infarction
Diaphragmatic myocardial infarction

SINUS arrhythmia is one of the commonest forms of arrhythmia and is a normal concomitant of childhood and adolescence. It is rare during adult life and then occurs again in the aged as the heart rate becomes slower. Transitory sinus arrhythmia is occasionally observed in acute myocardial infarction and may be a result of the vagotonic effect of morphine.¹⁾ The incidence of sinus arrhythmia in acute myocardial infarction is between 8 and 11%^{2), 3)} and there is no relationship to other arrhythmias. The purpose of this paper is to report a case of acute diaphragmatic myocardial infarction complicated by unusually irregular sinus arrhythmia and to discuss some of its physiological and therapeutic implications.

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Fig. 1. Leads V₅-a and b and V₆-a and b are continuous in each given lead. This tracing shows a marked sinus arrhythmia with acute diaphragmatic subepicardial injury and an evidence of old antero-septal myocardial infarction. It is interesting to note that the heart rates vary from 40 to 105 per min.

CASE HISTORY

The patient H. L., a 46-year-old male, was admitted to the hospital because of acute diaphragmatic myocardial infarction on October 30, 1968. He had several episodes of severe angina pectoris in the past several years and an acute antero-septal myocardial infarction in 1962. He awoke on the day of admission with crushing substernal pain and diaphoresis. He was seen at the hospital 4 hours later where an electrocardiogram showed acute diaphragmatic subepicardial injury associated with marked sinus arrhythmia (Fig. 1). He was given 50 mg. Demerol and admitted with stable vital signs. The hematocrit was 51% and the white cell count 14,700 (83% neutrophils). On October 31, 1968 his SGOT had risen to 165 units and on November 1, 1968 it was 260 units. He also developed a dry cough, pleuritic chest pain, a temperature of 101°F, and a loud pericardial friction rub. By November 2, 1968 the patient was asymptomatic and his electrocardiogram showed normal sinus rhythm and evolution of acute diaphragmatic myocardial infarction (Fig. 2). Thereafter he recovered uneventfully.

Fig. 2. This tracing shows sinus tachycardia (rate: 110/min.) with acute diaphragmatic myocardial infarction and old anteroseptal myocardial infarction. Sinus arrhythmia is still present but much less pronounced than in Fig. 1.

COMMENTS

It is interesting that a lower subsidiary pacemaker did not initiate an escape rhythm despite the fact that the sino-atrial pacemaker was at times as slow as 40 per minute. This lack of escape mechanism suggests that there may have been transient injury of the A-V node. Fig. 1 shows that the longer R-R intervals are approximately 3 times the short R-R intervals, without the intervention of A-V nodal or ventricular escape mechanism. The diagnosis of sino-atrial block cannot be made because each of these long R-R intervals is a different length. However, the possibility of sinus arrest associated with marked sinus arrhythmia cannot be excluded.

Acute diaphragmatic myocardial infarction results from occlusion of the right coronary artery in about 90% of cases.^{4), 5)} Furthermore, slightly over half of the proximally located occlusions of the right coronary artery may produce ischemia and/or infarction of the sinus node, since this is supplied in 55% of humans by a branch originating about 2 cm. from the ostium of the artery. In 83% of cases the A-V node is supplied from the right coronary artery.⁶⁾ These facts explain why sinus bradycardia is more common in

diaphragmatic than in anterior myocardial infarction,⁷⁾ and why a very slow sinus rhythm is often an ominous sign.⁸⁾ However, the pacemaking cells of the sinus node are resistant to hypoxia⁹⁾ and survive longer than ordinary myocardial cells, although sufficient ischemia may slow their rate of impulse formation. This presumably is the reason why our patient's electrocardiogram on the 3rd day showed evolution of acute myocardial infarction while his rhythm had returned to normal sinus rhythm (Fig. 2). The sinus node lies less than a millimeter beneath the surface of the heart.¹⁰⁾ Therefore, pericarditis which is a frequent complication of acute myocardial infarction, as in this patient, may also affect sinus pacemaking.

Sinus arrhythmia is found in two principal forms: the *respiratory*, in which the variations in heart rate are cyclic and related to respiration, and the *non-respiratory*, in which this relation does not exist. The distinction is made by noting whether the cycle of sinus arrhythmia has the same periodicity as the respiratory cycle. In the *respiratory* form, the variations in the cardiac cycle are characterized by an increase of the rate during inspiration and decrease during expiration. The irregularity is produced by variations in the balance between vagal and sympathetic nervous activities, the vagus exerting the dominant influence. In dogs, sinus arrhythmia persists following both paralysis of respiratory movement with succinylcholine and beta adrenergic blockade with propranolol; however, vagotomy abolishes sinus arrhythmia.¹¹⁾ These variations of vagal tone result from fluctuations in the activity of the cardio-regulatory nerve centers located in the medulla and hypothalamus; these fluctuations in turn are due to the action of impulses ascending afferent paths from sensory end organs in the pulmonary and systemic circulations and perhaps also from a spread of the periodic discharge of the respiratory center itself. The stimuli exciting these peripheral end-organs may be pressure changes in the alveolar walls or in the vascular tree itself, or changes in the chemical composition of the blood. The mechanisms are thus principally of reflex origin. As the heart rate increases, any type of sinus arrhythmia tends to disappear, while with slower heart rates, it is more apt to become prominent. Exertion and breath holding tend to eliminate the respiratory form while increased depth of breathing tends to exaggerate it.¹⁾ In brief, respiratory sinus arrhythmia tends to occur in young people, is usually asymptomatic and needs no treatment.

Non-respiratory sinus arrhythmia (also called idiopathic or pernicious sinus arrhythmia) as seen in the adult is usually drug-induced or the result of some diseases involving the S-A node.¹⁾ It has been known for a long time that digitalis may produce a marked sinus arrhythmia presumably by increasing vagal tone.¹²⁾⁻¹⁴⁾ More recently it has been shown experimentally and clini-

cally that reserpine (Serpasil) and guanethidine (Ismelin) not only potentiate the action of digitalis but also may induce unusually slow sinus bradycardia as well as A-V nodal escape rhythm.¹⁴⁾⁻¹⁸⁾ The mechanism by which digitalis disturbs sinus impulse formation and conduction is complex. Probably multiple factors are involved, including vagal stimulation, direct effect on automaticity of the sinus node due to prolongation of slow diastolic repolarization, and possibly an antiadrenergic effect and changing calcium levels.¹⁹⁾⁻²²⁾ As previously mentioned, acute diaphragmatic myocardial infarction may result in sinus arrhythmia and/or sinus bradycardia because of occlusion of the right coronary artery and consequent ischemia of the S-A node.

The clinical significance of sinus arrhythmia and/or sinus bradycardia as a consequence of acute myocardial infarction is related to the symptoms that it may cause: apprehension, palpitations, lightheadedness, and the possible further decrease of cardiac output. The normal heart maintains cardiac output during slow rates by increased diastolic filling and augmentation of the resultant stroke volume. The heart with an infarct is not only incapable of increasing stroke volume adequately, but during the phase of aneurysmal bulging a portion of each stroke is trapped and never ejected.⁵⁾ A decrease in heart rate from 105 to 40, as occurred cyclically in this patient, must be associated with a transient reduction of at least 50% in cardiac output; certainly a serious deficit if persistent.

It has long been recognized that slow heart rates are associated with a variety of escape rhythms, particularly in acute myocardial infarction.^{5), 23)} This is another potential hazard of marked sinus arrhythmia and/or sinus bradycardia; the ischemic ventricle is an additional factor favoring the onset of arrhythmias.⁵⁾

During intense ischemia in the region of the sinus node, there may be A-V block due to vagal reflex. This is a dangerous self perpetuating phenomenon in which the vagal reflex producing bradycardia and heart block is compounded by the results of bradycardia. As a consequence of slow irregular heart rate hypotension with weakness and severe pain during acute myocardial infarction, patients understandably become apprehensive. Fright itself enhances vagal activity, particularly if there is associated hopelessness or despair, and may be associated with arrhythmias and sudden death.²⁴⁾

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REFERENCES

1. Katz, L. N. and Pick, H.: Clinical Electrocardiography: The Arrhythmias. Lea & Febiger, pp. 68-71, 1956.
2. Metzler, L. E. and Kitchell, J. B.: Prog. Cardiovas. Dis. **9**: 50, 1966.
3. Robinson, J. S., Sloman, G., and McRae, C.: Med. J. Aust. **1**: 427, 1964.
4. James, T. N.: Circulation **24**: 761, 1961.
5. James, T. N.: Prog. Cardiovas. Dis. **10**: 410, 1968.
6. James, T. N. and Burch, G. E.: Circulation **17**: 90, 1958.
7. George, M. and Greenwood, T. W.: Lancet **ii**: 739, 1967.
8. James, T. N.: Am. Heart J. **64**: 416, 1962.
9. Hoffman, B. F. and Cranefield, P. F.: Electrophysiology of the heart. McGraw Hill Book Company, 1960.
10. James, T. N.: Anat. Rec. **141**: 109, 1961.
11. Hamlin, R. L.: Am. J. Physiol. **210**: 321, 1966.
12. Kisch, B.: Strophanthin. Brooklin Medical Press, 1944.
13. Gold, H., Kurt, N. T., Otto, H., and Fox, T.: J. Clin. Invest. **18**: 429, 1939.
14. Chung, E. K.: Digitalis Intoxication. Excerpta Medica, The Netherlands, 1968 (in press).
15. Dick, H. L. H., McCawley, E. L., and Fisher, W. A.: Arch. Int. Med. **109**: 501, 1962.
16. Lown, B., Ehrlich, L., Lipschultz, B., and Blanke, J.: Circulation **24**: 185, 1961.
17. Albano, T. and Motta, R.: Minerva Med. **49**: 3904, 1958.
18. Griffiths, H. J. L.: Am. Heart J. **75**: 371, 1968.
19. Braunwald, E. and Kahler, R. L.: Physiology for Physicians **2**: No. 2, 1964.
20. Hoffman, B. and Singer, D. H.: Prog. Cardiovas. Dis. **7**: 226, 1964.
21. Mendez, R. and Mendez, C.: J. Pharmacol. Exper. Ther. **24**: 107, 1953.
22. Thomas, L. L., Jr.: Am. J. Physiol. **199**: 146, 1960.
23. Han, J., DeTraglia, J., Millet, D., and Moe, G. K.: Am. Heart J. **72**: 632, 1966.
24. Richter, C. P.: Psychosom. Med. **19**: 191, 1957.