HEMODYNAMICS IN MYOCARDIAL INFARCTION

and therapeutic value of bedside monitoring of left ventricular pressure. Amer J Cardiol 23:107, 1969


25 Years Ago
Christening of a Carotid Vibration

In nine patients in whom subsequent clinical, cardiographic, and cardioscopic examination, established the presence of aortic stenosis and incompetence, we observed a characteristic sign, which by itself permitted a diagnosis of the combined aortic lesion from inspection of the neck. At the height of the carotid pulse, large on account of aortic incompetence, we noticed a quiver or vibration which lasted for a moment only. This effect we have named the carotid shudder for it best describes the visual clinical sign.

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Circulation, Volume XLII, October 1970
50 Years Ago—A Classic Description

Postero-inferior Myocardial Infarction in a 38-Year-Old Man
with Electrocardiograms
Initial Transient Heart Block—Early Atrial Fibrillation

REPORT OF CASE

. . . . Two hours before admission he had had an attack which was typical of the symptom complex due to an obstruction of one of the branches of a coronary artery.

He was 38 years of age . . . . He had always been in the best of health until August, 1916, when he had a slight attack of 'indigestion,' with epigastric distress and eructations of gas . . . . About December, 1916, he began to notice that his 'wind' was not so good as it had been, . . . and he also noticed occasional slight aching precordial pain.

March 4, 1917, suddenly, while he was in bed, asleep, he felt a sharp stabbing pain beneath the sternum. This radiated about the left chest and down the left arm and was very severe. He felt very weak and prostrated and thought he was going to die. His heart was beating very heavily, he said, and very slowly . . . .

On admission to the hospital two hours later the heart rate was 44 per minute and the rhythm was noted to be regular . . . .

During the next two days the cyanosis, increased breathing and precordial pain gradually decreased . . . . The heart rate varied from 60 to 80 per minute and continually showed the irregularity of auricular fibrillation . . . .

He returned to his work as a chauffeur about April 1 . . . continued to drive his car and often drove very fast.

. . . . He continued at work for most of the time though there were periods when he was prevented by dyspnea, which was noted as improved after taking digitalis for a time . . . . In June, 1919, he suddenly fell dead with an attack of severe precordial pain, just after having returned from a day of driving his automobile.

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Figure 1

(A) March 4. (B) March 13. (C) Probable region of ventricles affected.

Minuscule Review


In the story of atherosclerosis, Dr. Thomson’s report of the arterial disease in a transplanted human heart which had circulated the blood in the host recipient for 19½ months will be a classic reference. He describes an extensive deposition of lipid in the intimal zones of the coronary arteries of the young grafted heart, functioning in the milieu of hyperlipidemia, and ravaged by tissue rejection as modified by immuno-suppressive agents. The recipient, 58 years of age at the time of transplantation (January 2, 1968), had a blood cholesterol of 315 mg/100 ml, and later values were never below 300 mg. Marked lipid deposition was present in all the coronary arteries from the main trunks to the smallest epicardial branch. No lipid was demonstrated in the intramyocardial coronary branches. Calcification, which was a marked feature of the coronary arteries of the recipient’s original heart, was absent. Marked lipid deposition was present, also, in the donor aortic segment and in the anterior mitral leaflet. Plasma cell infiltration was present in this aortic segment and was believed to indicate a rejection of this tissue. Dr. Thomson discusses in detail the role which may have been played by the rejection phenomenon in the vessels resulting in the cellular matrix responsible for collection of the lipid and as a background for the accelerated “atherosclerotic” process. He suggests that patients with ischemic heart disease (with hypercholesteremia) are not likely to be suitable subjects for heart transplantation.

It is expected that lively discussion will continue concerning the pathogenesis of the described arterial lesions. Some pathologists, studying the lesions from the narrow viewpoint of human spontaneous atherosclerosis, will question whether the lesions should be labeled with the same term. Dr. Thomson’s description of the lesions is clear, and his report will become required reading for those interested in experimental arteriosclerosis. Each entrant to the jousts for a nomenclature for arterial disease will need to be well versed in the details of Dr. Thomson’s report. Possibly many will have been able to study “at first hand” the histologic preparations.

H.B.B.


Editor’s Dilettantism

Cardiac Glycosides, Blue Jays, and Butterflies

Monarch butterflies Danaus plexippus, when reared on tropical asclepiads and apocynads, contain cardiac glycosides which make them unpalatable to avian predators... Glycosides were extracted from seeds [of the plants] ... and insect tissue ... purified on a Florisil column ... and their identity was verified by thin-layer chromatography on silica gel G, by spectrophotometry, and by biological assay (inotropic response in rat heart, and antagonism of this by aldosterone)... The blue jay Cyanocitta cristata bromia is the major avian predator employed in studies of palatability. ... We do not know whether Danaus feeding on Asclepias simply accumulates glycosides according to the type and concentration in the plant, or if it has specific selective or concentration mechanisms.

-DUFFEY SS: Cardiac glycosides and distastefulness: Some observations on the palatability spectrum of butterflies. Science 169: 78, 1970. (Courtesy the American Association for the Advancement of Science: Copyright 1970).
Minuscule Review

Hanazono N, Shimoyama S, Ando Y: A consideration on the mechanism of development of the attack of the variant form of angina pectoris.


There has long been an interest in the electrocardiographic change during episodes of angina in the lead which faces the epicardial shell. This change, consisting of ST-segment elevation, has been observed both in spontaneous angina and in coronary insufficiency induced by various types of stress. Dr. Hanazono and associates report on three patients who had electrocardiograms showing ST-segment elevations. The group is not homogeneous: the first case showing ST-segment elevation with the maintenance of the QRS, the second, ST-segment elevation and high voltage T waves in the mid-precordial lead, and the third case a monophasic type of potential. In two of the cases the electrocardiograms simulated the changes of acute inferior myocardial infarction. One case showed second degree heart block with Wenckebach's phenomenon during the attack. The patients do not conform to the syndrome of "bradycardic angina." However, each had a decrease in the heart rate at the time of pain, and the authors found a decrease in the catecholamine content of the blood at the onset of the attack. They possibly have overexpanded the significance of their observations in support of their hypothesis, that a temporary state of obstruction of a large branch of the coronary artery occurred following the weakening of the dilating effects of the blood epinephrine. In two patients the response to isoproterenol infusion was studied, and in one no pain was experienced during the period of palpitation but a typical attack of pain occurred 3 min after the end of the infusion when the heart rate suddenly decreased. In the second patient an attack of conventional angina was provoked. In patients 1 and 2, propranolol produced no distress, to the apparent disappointment of the investigators.

In the cases that the authors report, it would have been of interest to know what coronary arteriography might have revealed. One would have expected obstructive coronary disease to have been demonstrated. However, the cases need to be viewed within the framework of possible maldistribution of coronary flow, with or without coronary disease. The report may be studied in relation to the case of variant angina pectoris with heart block reported by Whiting and associates (New Eng J Med 282: 709, 1970), and that of myocardial infarction in the inferior wall of the heart, with normal coronary arteries, by Sidd and co-workers (New Eng J Med 282: 1306, 1970).

H.B.B.


Style, Rhetoric, and Social Intercourse

In the very act of addressing someone we acknowledge a wish to push him around, and in our zeal to push a little harder, it is no wonder our voices begin to sound strident. It is with style that we try to behave like a decent person, one who ruefully concedes his drive for power while remaining aware of his reader's well-chosen resistance. Thus style is our way of becoming a person worth listening to, worth knowing.

A moral justification for the study of rhetoric lies right here. We improve ourselves by improving the words we write. We make our performance less monstrous, by acting like human beings. Just what comprises a satisfactory human performance is every man's complicated decision. But at least, by looking at rhetoric, we may begin to know more about who it is we are making believe we are. And then, perhaps, we can do something about it.—From Gibson W: Tough, Sweet & Stuffy: An Essay on Modern American Prose Styles. Bloomington, Indiana, Indiana University Press, 1966, p 110.
Minuscule Review


This comprehensive article emphasizes the heterogeneity of lesions associated with ventricular inversion and transposition (congenital corrected transposition of the great arteries), particularly the high incidence of single ventricle and left A-V valve anomalies.

The relatively long follow-up data on the natural history in these cases make it mandatory for physicians dealing with all age groups to be familiar with this group of entities. This report establishes that if an individual is alive at 1 year, he probably will be alive at 25 years, and that when pulmonic stenosis is present, the clinical course is more benign.

Auscultation, ECG-VCG, and chest x-rays, alone or in combination, did suggest the diagnosis of ventricular inversion in 88% of the cases.

The authors emphasize the importance of (1) a single S₂ in the second left intercostal space only when coupled with an inaudible S₂ in the second right intercostal space; (2) conduction anomalies, often progressive; and (3) the distortion of the initial QRS vectors, directed to the left and posterior, producing an absence of a Q wave in V₆ and usually a qR pattern in V₄R or V₁ (68% of cases).

An important contribution is the documentation of significant subaortic obstruction, which may be progressive, within the cavity of the diminutive anatomic right ventricle. An angiogram depicting this subvalvar obstruction would have been helpful.

Also important is the authors' admonition that the heterogeneity of lesions associated with ventricular inversion is so great that the exact pathologic anatomy and physiology must be available prior to consideration of any surgical procedure. Selective angiocardiography plays a key role in acquiring the data on which to base such decisions and whether or not to advise corrective surgery.

A greater cohesiveness of the clinical profile might have been achieved if the cases had been divided into two groups: (a) those with ventricular inversion and two ventricles, and (b) those with a single ventricle and ventricular inversion. In the former group, the ECG-VCG pattern usually is that of "left ventricular hypertrophy" or perhaps "biventricular hypertrophy"; whereas in the latter, the ECG-VCG pattern is a "right ventricular hypertrophy" pattern often with a qR pattern in V₄R or V₁.

Also, the article contains several areas of concern: (1) the use of the height of the c wave as the sole criterion for left A-V valve incompetence; (2) use of the term "infundibular stenosis" when relating to right heart obstruction ("subvalvar" would be a better term, as with the ventricles inverted, the "venous ventricle" has no infundibulum); and (3) use of the term "increased left ventricular voltage" when applied to cases with increasing subaortic obstruction and hypertrophy of the diminutive anatomic right ventricle.

Gerold L. Schiebler, M.D.
Minuscule Review

Friedman NJ: Echocardiographic studies of mitral valve motion: Genesis of the opening snap in mitral stenosis.
Amer Heart J 80: 177, 1970.

This study is an excellent example of the type of clinical investigation that can be done with echocardiography. The ability to record continuously the motion of intracardiac structures, such as the anterior leaflet of the mitral valve, provides a new method of seeking answers to old clinical questions.

The observation that the opening snap in patients with mitral stenosis corresponds very closely with the completion of the rapid opening of the mitral valve lends considerable support to the theory that valve motion is responsible for this abnormal sound. It should be pointed out that the author only studied the anterior leaflet of the mitral valve. The posterior mitral leaflet can also be recorded with ultrasound but not with the analog system used in this paper. A study of the motion of the posterior leaflet may add further information as to the origin of the opening snap. Although the author gives an excellent theoretical discussion as to why an opening snap occurs with mitral stenosis but not normally, a personal observation that the motion of the posterior leaflet is markedly altered in mitral stenosis introduces another possible factor. In any case, one must remember that the motion of the posterior leaflet may be somewhat different from that of the anterior leaflet in some situations.

The author's finding that the echocardiographic mitral valve opening corresponds more closely to the opening snap than did the apexcardiographic 0 point is not surprising. Other investigators have noted the error in using the 0 point for timing the opening of the mitral valve. However, this observation together with the relationship between the echocardiographic mitral opening and the apexcardiographic 0 point prompted an interesting review of the origin of the 0 point. This part of the study again illustrates how a direct recording of intracardiac structures with ultrasound can lend some insight into other diagnostic tools, such as apexcardiography.

Harvey Feigenbaum, M.D.
Minuscule Review


Based on a study of nine patients over a period of 3½ years, the authors urge prompt surgical therapy utilizing conditions of cardiac bypass for patients with mitral stenosis who have systemic emboli. The nine patients were found to have severe mitral stenosis at operation. It is of interest and a little perplexing that six patients were stated to be without cardiac symptoms preoperatively and that mitral valve disease was not even suspected initially in four of them. Two of the patients had had saddle emboli, and the valve orifices were estimated by the surgeon at 6 mm and 8 to 10 mm. These observations raise the old question of where the aortic embolus comes from when it is large enough to block the terminal aorta yet the mitral valve orifice is minute; these circumstances obviously suggest that a thrombus possibly was propagated through the mitral orifice and enlarged in the ventricle. The age range of the patients was from 19 to 60 years; all except the two youngest had atrial fibrillation.

The inference may be drawn that the authors recommend surgical therapy for all patients with mitral stenosis when arterial emboli occur. The experience reported, however, does not relate directly to the more difficult problem than that exemplified by the authors’ patients; namely, the individual with the moderate to mild mitral stenosis with (usually) atrial fibrillation. For the latter problem, judgment remains difficult and the final correct protocol is yet to be established.

The paper by Dr. Borman and associates has the attributes of conciseness and clarity. Only 10 “key” references are given, and thus a complete review of the literature is not given.

H.B.B.
examination, in 1821. Guy Hosp Rep 1: 53, 1836

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14. Willis FA, Keys TE: Cardiac Classics. St. Louis, C. V. Mosby, 1941, p 293

15. Wells CW: On rheumatism of the heart. Trans Soc Improv Med Chir Knowledge 3: 373, 1812

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100 Years Ago

Acme of Pathos (Hugo)

All that was in him of generosity, of enthusiasm, of eloquence, of heart, of soul, of fury, of anger, of love, of inexpressible grief, ended in—a burst of laughter! And he proved, as he had told the Lords, that this was not the exception, but that it was the normal, ordinary, universal, unlimited, sovereign fact, so amalgamated with the routine of life that they took no account of it. The hungry pauper laughs, the beggar laughs, the felon laughs, the prostitute laughs, the orphan laughs to gain his bread; the slave laughs, the soldier laughs, the people laugh. Society is so constituted that sin and want and each and every catastrophe, fever, ulcer, and pang, is resolved on the surface of the abyss into one frightful grin of joy. Now, he was the prototype of that universal grin; that grin was himself. The law of Heaven, the unknown power which governs, had willed that a spectre, visible and palpable, a spectre of flesh and bone, should be the synopsis of the monstrous parody which we call the world; and he was that spectre.


. . . . in all time, travesty has been the argument of oppression. . . .

Minuscule Review

MacGregor MI, Block AJ, Ball WC Jr: Serious complications and sudden death in the Pickwickian syndrome.

MacGregor, Block, and Ball report the clinical findings, laboratory data, and hospital course in 22 patients with obesity, alveolar hypoventilation, and symptoms of somnolence, periodic breathing, and dyspnea seen at The Johns Hopkins Hospital from 1959 through 1969. Fourteen of their patients developed severe complications and seven of these died. With one exception, all of the deaths and complications were related to respiratory failure (10 patients; four deaths) or pulmonary emboli (four patients; two deaths). The authors emphasized the danger of sudden unexpected death in these patients and recommended the use of assisted ventilation and respiratory stimulants at the first sign of deterioration of consciousness or increasing hypoxemia.

Although the incidence of sudden death is higher in the Johns Hopkins series than in other studies of this syndrome, this type of complication is not unexpected. The combination of mass loading of the respiratory muscles (Sharp, et al., J Appl Physiol 19:959, 1964) and marked ventilation-perfusion abnormalities (Holley, et al., J Clin Invest 46:475, 1967) found in the obese patient produce a precarious balance between barely adequate lung function and severe respiratory insufficiency. The patients in the Johns Hopkins study were in an older age group and had a high incidence of cardiac failure. These two factors may explain the unusually high mortality in this series.

The etiology of the hypoventilation associated with obesity is not clear. Lyons and Huang (Amer J Med 42:881, 1968) found that weight reduction is not always accompanied by improvement in alveolar ventilation. Lourenco (J Clin Invest 48:1609, 1969) using an EMG electrode to record diaphragm activity demonstrated that obese subjects with alveolar hypoventilation had significantly less integrated electrical activity in their diaphragms in response to increasing carbon dioxide tension than did a matched group of obese subjects with normal alveolar ventilation. These studies suggest that the ventilatory drive from the medullary chemoreceptor may be inadequate in some patients to overcome the load on the respiratory muscles imposed by obesity. They also provide us with a physiologic basis for the use of respiratory stimulation, both chemical and verbal, in patients with this syndrome.

Richard S. Kronenberg, M.D.