CC/NUMBER 49
DECEMBER 3, 1990

This Week's Citation Classic ®

Goodwin J F, Hollman A, Cleland W P & Teare D. Obstructive cardiomyopathy simulating aortic stenosis. *Brit. Heart J.* 22:403-14, 1960.
[Depts. Medicine and Surgery, Postgraduate Medical Sch. London, and Dept. Forensic Medicine, St. George's Hosp., London, England]

Eight patients are described in whom hypertrophy of the left ventricle of unknown cause produced a pressure gradient proximal to the aortic valve apparently due to obstruction of the left ventricular outflow and, in one case, the right ventricular outflow. It was considered that the condition was some form of heart muscle disease rather than a localised disorder of the subaortic valve region: hence the term *obstructive cardiomyopathy*. [The *SCI*® indicates that this paper has been cited in over 250 publications.]

Thirty Years On—A Window Opens on Myocardial Disease

J.F. Goodwin
18 Augustus Road
London SW19 6LN
England

July 13, 1990

Study of this paper reveals three main features: first, the radical change in investigative technique since 1960; second, the beginning of a new era when the heart muscle disorders—the cardiomyopathies—were logically defined and classified;[1] third, a continuing transatlantic debate as to whether gradients meant true obstruction to outflow or not.

It is ironic that we, having used the term *obstructive cardiomyopathy* (which fitted well with other concepts of obstruction in the disease at that time), should have subsequently reversed our opinion and denied the importance of obstruction! It is with some wry self-deprecation that I now regard myself as a refugee from obstruction!

In 1960 left ventricular angiography was not considered by our group to be feasible or safe, and it is amazing to recall that left ventricular/aortic gradients were often measured by the hair-raising (to us now) practice of puncturing the left ventricle through the chest wall and recording the pressure simultaneously with a recording from a peripheral artery! Angiography of the left heart at that time was indirect via the right side of the heart. The angiographic features, together with the autopsy appearances and haemodynamic and clinical features, convinced us that the condition was a diffuse left ventricular disorder characterised by massive hypertrophy, notably of the ventricular septum.

The signs and differences between valvar, subvalvar aortic stenosis, and obstructive cardiomyopathy were clearly described and were the keystones of diagnosis that have stood the test of time. The physical signs we described are accurate today with regard to the characteristic arterial pulse and the late-onset systolic murmur. We did not, at that time, appreciate the reason for the timing of the murmur nor did we appreciate the powerful left atrial contraction (atrial beat), which is such an important sign.

As investigation into the disease progressed, many descriptive terms were used. We subsequently modified the term *obstructive cardiomyopathy* to *hypertrophic obstructive cardiomyopathy* because we felt that hypertrophy was a much more important feature than "obstruction."[2] Thus the disease became hypertrophic obstructive cardiomyopathy or HOCM for short. Later still, believing obstruction to be relatively unimportant, we called the disease hypertrophic cardiomyopathy. This term is not widely used, though other titles such as muscular subaortic stenosis and idiopathic hypertrophic subaortic stenosis have been used. But these titles miss the point that hypertrophic cardiomyopathy is a generalised (though patchy) form of hypertrophic heart muscle disease and not a localised outflow tract problem of the left ventricle.

There is still widespread belief that obstruction is important.[3] But, in fact, it occurs only in the minority of cases; in the majority, the gradients are due to flow and turbulence rather than to true obstruction. This was first suggested many years ago[4] and supported by more recent work.[5]

But, what causes the hypertrophy? Is hypertrophic cardiomyopathy one disease or many? Can it be prevented? It seems certain that it is a genetically determined disorder,[6] probably due to a disturbance of heart muscle growth *in utero*. It seems that the way ahead lies with the techniques of molecular biology.[6] Clinical cardiologists and molecular biologists must combine their skills to elucidate these questions.

While by no means the first description of what we now know as hypertrophic cardiomyopathy, this paper has value because it firmly puts the disease into the category of the cardiomyopathies and emphasises the importance of clinical assessment in diagnosis and differential diagnosis.

1. **Goodwin J F.** Congestive and hypertrophic cardiomyopathies—a decade of study. *Lancet* 1:731, 1970. (Cited 310 times.)
2. **Cohen J, Effat H, Goodwin J F, Oakley C M & Steiner R E.** Hypertrophic obstructive cardiomyopathy. *Brit. Heart J.* 26:16-32, 1964. (Cited 200 times.)
3. **Wigle E D, Kimbale B P, Sassa Z & Rakowski H.** Haemodynamic profile. (Zipes D P & Rowlands D J, eds.) *Progress in cardiology.* Philadelphia: Lea & Febiger, 1989. Vol. 2/2. p. 73-86.
4. **Criley J M, Lewis K B, White R I, Jr. & Ross R S.** Pressure gradients without obstruction: a new concept of 'hypertrophic subaortic stenosis.' *Circulation* 32:881-8, 1965. (Cited 165 times.)
5. **Murgo J P, Alter B R, Dorethy J F, Altobelli S A & McGranahan G M J, Jr.** Dynamics of left ventricular ejection in obstructive and non-obstructive hypertrophic cardiomyopathy. *J. Clin. Invest.* 66:1369-82, 1980. (Cited 75 times.)
6. **Rourke J.** Genetics. (Zipes D P & Rowlands D J, eds.) *Progress in cardiology.* Philadelphia: Lea & Febiger, 1989. Vol. 2/2. p. 45-57.