that different types of stress may explain different results is certainly interesting, and deserves further in-depth studies on larger populations.

*Rodolfo Citro, MD
Mario Previtali, MD
Eduardo Bosrone, MD
Roberto Manfredini, MD

*Cardiology Department
A.O.U. San Giovanni di Dio e Ruggi d’Aragona
Salerno 84131
Italy
E-mail: rodolfo.citro@tele2.it


REFERENCES


Pierre Soulié, a “Pre-Echocardiographic” Pioneer of Hypertrophic Cardiomyopathy

Maron et al. (1) recently issued an in-depth review of evolving concepts for hypertrophic cardiomyopathy. These prestigious investigators report the concordant or controversial issues discussed over 50 years. They finally present an understanding of the disease consistent with the early descriptions.

I understand that the report is clearly dedicated to the evolving opinions and debates generated by the disease in the last 50 years. I also am well aware of the decisive contribution of echocardiography for understanding its mechanism since 1969 and of the value of genetics for the study of the transmission of the disease and its heterogeneous clinical spectra. Nevertheless, grouping important contributions of researchers who deciphered its early characteristic features from 1957 to about 1967 under the nonspecific “pre-echocardiographic” era, in the way historians refer to the B.C. period, is at risk to overlook some pioneers.

In the late 1950s, namely from 1957 to 1959, all the cards were not in the same hands to diagnose hypertrophic cardiomyopathy; on the one hand, when looking at ventricular walls, it was an arrested heart for surgeons (2) or at necropsy for anatomists. This enabled the discovery of asymmetrical septal hypertrophy (3), but the dynamic features lacked the ability necessary to recognize the disease; the global hypertrophy found at surgery or necropsy was interpreted as residual in the outflow tract after reduction of a valvular or subvalvular membranous stenosis or secondary to systemic hypertension, or of unknown origin (2.4–5). On the other hand, those studying the beating heart with their mere ears, fingers, and stethoscopes did not “see” the underlying cardiac structures’ abnormalities and attributed the murmurs they heard to rheumatic aortic stenoses, which were the rule. Titles of papers reflect their astonishment (2.4–6). In 1958, Pierre Soulié was the first to link asymmetrical septal hypertrophy, subvalvular pressure gradient, and the typical arterial pattern in the new concept of a “true subvalvular muscular stenosis” in a paper presented at the French Society of Cardiology (October 19, 1958). Detailed findings were printed later in September 1959 (6). Typical features of the arterial pressure tracings were shown and detailed: initial rapid upstroke followed by a lower prolonged tidal wave accounting for the hindrance to flow due to the “nonpermanent systolic muscular obstruction.” He emphasized its diagnostic value versus valvular stenosis. Although Brock (2) had vaguely noted such a transitory pattern on some contractions without drawing any diagnostic information (Fig. 11 in Brock [2]), confirmation of the arterial pattern came from Brachfeld and Gorlin’s later review (7).

Reading Soulié’s paper shows an obvious observational advance over previous references (2,4,5). Finally, Soulié’s hypothesis about the septal subvalvular site of the murmur was later substantiated by intracardiac phonocardiography (8). Similar features were found on flow velocity traces in the aorta (9) and later transcutaneously (10).

Rapidly improving invasive procedures added complementary information at the end of this “pre-echocardiographic” era. The stage was, thus, ready for echocardiographic and genetic studies to complement the whole spectrum of the disease.

*Colette Veyrat, MD

*Institut Mutualiste de Montsouris
Department of Cardiovascular Medicine
42, Boulevard Jourdan
Paris, Ile de France 75674
France
E-mail: colette.veyrat.resedal@noos.fr


REFERENCES

Periprocedural Myocardial Injury: Not a Benign Entity

Prasad et al. (1) present valuable insights into the prognostic influence of spontaneous myocardial infarction (SMI) and periprocedural myocardial infarction (PMI) following stent insertion in the ACUITY (Acute Catheterization and Urgent Intervention Triage Strategy) trial. They concluded that PMI is of limited prognostic significance as it is predominantly a marker for procedural and baseline risk factors, and that, in contrast to SMI, it does not have independent prognostic significance. Given the differences in the pathophysiology, it is unsurprising that PMI and SMI have differential influence on prognosis at 1 year, but we wish to question some of the investigators’ interpretation of their data, as we feel that the significance of PMI has been understated.

Their principal conclusion about the lack of prognostic influence is based on the 1-year mortality hazard ratio for PMI being found to be not significant after adjustment for procedural and baseline characteristics. In this study, both PMI (3.2% vs. 0.8%, p < 0.0001) and SMI (5.0% vs. 0.8%, p < 0.0001) are associated with higher 30-day mortality without any significant difference between the 2 types of myocardial infarction (p = 0.27). No hazard ratio for 30-day mortality after the time-updated covariate-adjusted multivariate analysis is quoted, but the Kaplan-Meier graphs show similar curves for SMI and PMI in the early post-procedural period, suggesting that both types of myocardial infarction predict an adverse short-term outcome. Interestingly, stent thrombosis in patients that sustained PMI appears to have a worse prognosis than SMI without stent thrombosis.

The relevance of periprocedural enzyme elevation has been the topic of considerable debate, but it is increasingly clear that even small periprocedural troponin rises reflect new areas of myocardial necrosis on magnetic resonance imaging (2,3). Debate persists about the influence on prognosis of these small/moderate enzyme rises, but there is little doubt about the influence of large periprocedural enzyme rises—even in this study, the 1-year mortality for Q-wave PMI is extremely high at 27%, versus 17.3% for Q-wave SMI (p = 0.22).

Previous studies from patients undergoing nonemergency percutaneous coronary intervention, whether using creatine kinase-myocardial band or troponin definitions of PMI, have shown that PMI is an adverse long-term prognosticator (4–6). It is notable in this study of patients with acute coronary syndromes that a definition combining new elevation of >3× creatine phosphokinase or creatine kinase-myocardial band and electrocardiography criteria has been used for PMI. Troponin, a more sensitive and specific biomarker, is not used for the diagnosis of PMI but is used for the diagnosis of SMI.

We agree that PMI and SMI are different entities, and inevitably SMI would portend a worse prognosis. However, we feel that PMI with imaging evidence of myocardial necrosis represents an adverse event with long-term outcome implications. The long-term consequences of minor periprocedural enzyme rises are more uncertain and need more investigation, especially with the advent of the new universal definition of PMI using a troponin cutoff of >3× the 99th percentile (7).

*Chris C. S. Lim, MBBS
William J. Van Gaal, MBBS
Adrian P. Banning, MD

Cardiology Department
John Radcliffe Hospital
Headley Way
Oxford OX3 9DU
United Kingdom
E-mail: chingsung@yahoo.com

doi:10.1016/j.jacc.2009.09.037

REFERENCES


Reply

We thank Dr. Lim and colleagues for their interest in our paper (1). While agreeing with the principal finding of our study (1) that spontaneous myocardial infarction (SMI) portends a worse prognosis than periprocedural myocardial infarction (PMI), Lim and