peak of occurrence was April, whereas only 12% of TTC occurred in summer (Fig. 1). The morning and the afternoon were the most frequent periods of onset of TTC. However, in our series, the onset of the disease may differ according to the stressful event: all TTC after surgery (n = 6) occurred in the morning, whereas aggression or robbery (n = 9) were found whatever the time of day.

TTC is defined as transient left ventricular dysfunction triggered by stress, with left ventricular regional wall motion abnormalities extending beyond a single epicardial coronary distribution and without any coronary lesion (2,3). This new cardiomyopathy preferentially occurred after an emotional or stressful event, and catecholamine excess remains the main hypothesis (2,4–6). Different types of stress are involved in the process of TTC, leading to differing results in TTC studies.

REFERENCES


Reply

We thank Dr. Mansencal and colleagues for their comments on our study (1). They reviewed 51 cases of Tako-Tsubo cardiomyopathy (TTC) from 2008 to 2009 and did not replicate our findings. They found a peak onset of TTC in April and not in summer, and morning and afternoon were preferred periods. The main limitation of epidemiological studies on TTC derives from the limited size of populations, so that very few cases may cause significant changes. As for seasonal variation, a further study from our Network, conducted on an enlarged population of 112 patients, confirmed the summer preference of onset of TTC for subjects either age <65 or ≥65 years (2). Moreover, a summer preference has been reported previously in a German single-center study (31 cases) (3), and more recently also confirmed for 70 patients included in an American registry (4). The existence of a main morning peak of occurrence was observed in a Japanese cohort of 50 cases (5). We do not suggest that our results are definitely conclusive. However, our study was conducted on one of the largest populations of TTC patients available in the literature to date (6), and first used a validated chronobiological method of analysis, focused on searching for underlying rhythmic reproducible patterns of occurrence, not only peaks of higher frequencies. Although the role of stress hormones remains controversial (7), the exposure to catecholamines and beta-receptor agonists could precipitate the clinical scenario of TTC (8). We agree with Dr. Mansencal and colleagues that stress events may play a pivotal role. Thus, their hypothesis...
that different types of stress may explain different results is certainly interesting, and deserves further in-depth studies on larger populations.

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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 on 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.

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