tachycardia at hospital admission. Five individuals (26%) had a previous ablation, 4 of which were associated with successful resolution of the WPW pattern on the ECG. In the majority of cases (n = 16; 84%), SCD occurred at rest. The mean heart weight was 408 ± 105 g. In 10 patients (53%), the autopsy revealed a normal heart, 5 cases showed definitive cardiac pathology (4 cases of hypertrophic cardiomyopathy [HCM] and 1 case of cardiac sarcoid), and 4 cases demonstrated autopsy findings of uncertain significance (2 cases of idiopathic left ventricular hypertrophy, 1 with idiopathic fibrosis, and 1 with an enlarged left ventricle). Of the 5 asymptomatic patients, the postmortem revealed HCM in 3 and a normal heart in 2 cases. Two of the 4 patients with HCM showed myocardial fibrosis in the interventricular septum. All deaths attributed to HCM were characterized by left ventricular hypertrophy associated with myocyte disarray. None showed intracellular vacuolization characteristic of glycogen storage diseases associated with pre-excitation. None of the cases in this cohort showed significant valvular abnormalities. Among patients who underwent ablation, 3 showed a normal heart and 2 showed idiopathic left ventricular hypertrophy. Based on our pathologic series, the proportion of WPW cases with structural abnormalities at autopsy was similar to cases without the reported WPW ECG pattern (n = 1,870; 51%).

In conclusion, our findings suggest the following: 1) a proportion of cases with the WPW ECG pattern may die suddenly in the absence of symptoms; 2) many die at rest; 3) deaths may occur after the fourth decade; and 4) a substantial proportion of individuals have concomitant pathology that may contribute to atrial fibrillation. Previous prospective studies have showed that the presence of symptoms is not useful in the risk stratification of WPW patients (2,4,5). In addition, accessory pathway ablation did not seem to eliminate the risk of SCD, because 5 of the SCD cases were subjected to an ablation procedure. This is possibly due to the presence of multiple pathways or of other coexisting substrates for fatal arrhythmias. Finally, pre-excitation was associated with additional structural abnormalities in almost 50% of cases, underscoring the importance of performing baseline echocardiography and possibly cardiovascular magnetic resonance in all individuals with WPW and suggesting that the combination of pre-excitation with additional cardiac pathology may render individuals at higher risk of SCD.

Our study has some limitations. Although all the clinical information relating to the deceased was gathered in a meticulous fashion, only a small percentage of the entire cohort in our SCD registry was investigated with an ECG, therefore the true prevalence of SCD from WPW cannot be ascertained from this study. Our data suggest that WPW causes death in asymptomatic individuals and deaths may occur following ablation; however, this study cannot ascertain the prevalence of fatal events in these circumstances because our information relied on secondary reports and we did not have adequate details about the electrophysiological studies, including the refractory period or the number of pathways, respectively. Finally, the cardiac autopsy did not include a standardized demonstration of accessory pathways at the histological assessment.

Gherardo Finocchiaro, MD
Michael Papadakis, MBBS, MD
Elijah R. Behr, MBBS
***Sanjay Sharma, BSc, MBChB, MD**
Mary Sheppard, MBBCH, BAO, BSc
*Cardiology Clinical and Academic Group
St. George's, University of London
Cranmer Terrace
London SW17 0RE
United Kingdom
E-mail: sasharma@sgul.ac.uk

http://dx.doi.org/10.1016/j.jacc.2017.01.023

Please note: The authors have reported that they have no relationships relevant to the contents of this paper to disclose. Drs. Sharma and Sheppard contributed equally to this work.

REFERENCES

1. Obeyesekere MN, Leong-Sit P, Massel D, et al. Risk of arrhythmia and sudden death in patients with asymptomatic preexcitation: a meta-analysis. Circulation 2012;125:2308–15.

2. Pappone C, Vicedomini G, Manguso F, et al. Wolff-Parkinson-White syndrome in the era of catheter ablation: insights from a registry study of 2169 patients. Circulation 2014;130:811–9.

3. Basso C, Corrado D, Rossi L, Thiene G. Ventricular preexcitation in children and young adults: atrial myocarditis as a possible trigger of sudden death. Circulation 2001;103:269–75.

4. Pappone C, Manguso F, Santinelli R, et al. Radiofrequency ablation in children with asymptomatic Wolff-Parkinson-White syndrome. N Engl J Med 2004;351:1197–205.

5. Santinelli V, Radinovic A, Manguso F, et al. The natural history of asymptomatic ventricular pre-excitation: a long-term prospective follow-up study of 184 asymptomatic children. J Am Coll Cardiol 2009;53:275–80.

Diastolic Blood Pressure and Myocardial Damage

What About Coronary Perfusion Time?

In a recent issue, McEvoy et al. (1) examined the relationship between diastolic blood pressure (DBP) with subclinical myocardial damage (using

high-sensitivity cardiac troponin T) and with coronary heart disease (CHD), stroke, or death (1). They concluded that particularly among subjects with systolic blood pressure ≥120 mm Hg, and thus elevated pulse pressure, low DBP was associated with subclinical myocardial damage and coronary events.

In this insightful analysis, a very low DBP coupled with a high pulse pressure seemed to be very deleterious and these results put new perspective on studies that support lower targets for blood pressure lowering therapy (2).

It has been shown that overaggressive antihypertensive treatment, that leads to low DBP and thus hypoperfusion of the coronary arteries, results in cardiac ischemic events (3). McEvoy et al. (1) focused their attention on DBP as the predominant determinant of coronary perfusion. However, it should be remarked that several factors in addition to DBP, such as perfusion time, vessel wall diameter, and vasomotor tone, may importantly affect coronary blood flow. It has been reported that any increase in heart rate (HR) impinges on diastolic time more than on systolic time, reduces the perfusion time, and leads to subendocardial ischemia, especially in patients with coronary artery disease (4). Furthermore, a large body of evidence indicates that high HR can be considered a strong predictor of cardiovascular morbidity and mortality in different clinical settings (4). In their report, the authors, unfortunately, did not address the association of HR with subclinical myocardial damage and events across the various DBP categories. Considering that HR is a simple parameter that strongly correlates with diastolic coronary flow it should be routinely taken into account for refining risk stratification of CHD in treated hypertensive patients.

*Gian Battista Danzi, MD
Cesare Cuspidi, MD
*Division of Cardiology
Ospedale Santa Corona
Via XXV April, 38
Pietra Ligure, Savona 17027
Italy
E-mail: gbdanzi@tin.it

http://dx.doi.org/10.1016/j.jacc.2016.11.086

Please note: Both authors have reported that they have no relationships relevant to the contents of this paper to disclose.

REFERENCES

1. McEvoy JW, Chen Y, Rawlings A, et al. Diastolic blood pressure, subclinical myocardial damage, and cardiac events: implications for blood pressure control. J Am Coll Cardiol 2016;68:1713–22.

2. The SPRINT Research Group, Wright JT Jr., Williamson JD, et al. A randomized trial of intensive versus standard blood-pressure control. N Engl J Med 2015;373:2103–16.

3. Selvaraj S, Steg PG, Elbez Y, et al. Pulse pressure and risk for cardiovascular events in patients with atherothrombosis: from the REACH Registry. J Am Coll Cardiol 2016;67:392–403.

4. Palatini P, Rosei EA, Casiglia E, et al. Management of the hypertensive patient with elevated heart rate: Statement of the Second Consensus Conference endorsed by the European Society of Hypertension. J Hypertens 2016;34:813–21.

Diastolic Hypotension and Myocardial Ischemia

A Reason to Remember Cuff Artifact in Blood Pressure Measurement

In their important paper showing that diastolic hypotension increased the risk of myocardial ischemia, McEvoy et al. (1) nicely explained the mechanism: similar to cerebral perfusion, most of myocardial perfusion occurs during diastole. They found that the risk was largely driven by a wide pulse pressure (>60 mm Hg), meaning that the persons at risk had stiff arteries. This supports the hypothesis (2,3) that the J curve may be due to diastolic pressures that are actually much lower than measured by a cuff.

In 1978, colleagues and I reported (4) that among patients >60 years of age with diastolic pressures >100 mm Hg but no end-organ disease, one-half had a cuff diastolic pressure that was 30 mm Hg higher than the intra-arterial pressure. At the time I called this “pseudohypertension” because I was focusing on the cutoff of 90 mm Hg then used to define the need for antihypertensive therapy. A better name would be cuff artifact. We found that mean arterial pressure calculated from cuff pressures more closely approximated intra-arterial pressures (5). I have estimated that this problem occurs in ~4% of patients attending hypertension clinics, but it is likely that lesser degrees of cuff artifact are much more common.

In patients with stiff arteries, bradycardia widens pulse pressure because a larger stroke volume is being pushed into a stiff aorta. Thus beta-blockers or diltiazem may aggravate the problem of unrecognized diastolic hypotension. In elderly patients who complain of hypotensive symptoms, but whose cuff pressures do not seem to be hypotensive, it is important to measure intra-arterial pressure. The findings of McEvoy et al. (1) serve as a reminder to consider that the blood pressure measured by a cuff may actually be much higher than the true blood pressure.

*J. David Spence, MD