everybody knows, with a few exceptions perhaps, the results of our experience. Cappato commented on the occurrence of left atrial flutter as being a rather common adverse event after CPVA and thus a limiting factor of this technique. This is not now scientifically accurate. Unfortunately and surprisingly enough, Cappato has not reported in his editorial the aforementioned randomized study addressing this important and specific issue.2 The CPVA approach using additional lines in the posterior wall and mitral isthmus, which is from 2002 the current technique, has an incidence of left atrial tachycardia of only 3.9% when compared with 10% with CPVA alone.

We strongly believe that the two initial different strategies of the two pioneering groups on AF catheter ablation have now quite similar success rates and go towards a unified strategy, that is the CPVA approach.

References


Carlo Pappone
Department of Cardiology
Electrophysiology and Cardiac Pacing Unit
San Raffaele University Hospital
Milan 20132
Italy
E-mail address: carlo.pappone@hsr.it

Vincenzo Santinelli
Department of Cardiology
Electrophysiology and Cardiac Pacing Unit
San Raffaele University Hospital
Milan 20132
Italy

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Towards a unified strategy for atrial fibrillation ablation?: reply

I thank Drs Pappone and Santinelli for their interest in my editorial titled “Towards more effective techniques for catheter ablation of atrial fibrillation: to aim for electrical disconnection of pulmonary veins or not?” The authors observe that adding ablation lines at the left posterior wall and the mitral isthmus to the anatomical circumferential ablation (ACA) catheter technique for the treatment of paroxysmal or persistent atrial fibrillation is associated with a significantly lower incidence (from 10.0 to 3.9%) of iatrogenic left atrial flutter during long-term follow-up2 and that this lower incidence may justify an extended use of their technique. I have to admit that the manuscript quoted by Pappone and Santinelli had not been published yet at the time of final submission of my editorial to the European Heart Journal. Nevertheless, the arguments raised by the authors offer an interesting opportunity for debate.

Although the technique proposed by Pappone and Santinelli to limit the incidence of left atrial flutter in these patients is of interest and accurately investigated, the following observations should be carefully considered before it can be proposed in clinical practice. First, a 4% incidence of left atrial flutter in response to ACA is not a poor figure, particularly if one considers the drug refractoriness of this arrhythmia and the deterioration in quality of life that may produce when compared with pre-ablation. Secondly, this figure is obtained at the expense of additional pulses deployed in the left atrium, outside of the area delimited by the ACA design. Of interest, recent findings indicate that post-ablation left atrial flutter originate and perpetuate within the territory delimited by the ACA design, and that re-isolation of the pulmonary vein (PV) antrum effectively prevents left atrial flutter recurrence.3 As a result of this observation, PV electrical disconnection rather than empirically designed ACA would appear to have a better rationale for prevention of late left atrial flutter; also, PV electrical disconnection would not require adding ablation lines at the left posterior wall and the mitral annulus. Finally, the data from Pappone and Santinelli reflect the experience of a single centre and are not considered NT-proBNP as a suitable biomarker for the evaluation and monitoring of patients with aortic valve disease. According to their results on a large cohort, the authors considered NT-proBNP as a suitable biomarker for the evaluation and monitoring of patients with aortic valve disease. On the basis of our own experience in paediatric patients, we would like to add a note of caution regarding the diagnostic accuracy of natriuretic peptides in aortic stenosis.

In the last 4 years, we have measured BNP in almost 200 healthy children and more than 400 patients with congenital heart disease using the Triage BNP test (Biosite Inc., San Diego, California, USA).2 Within these examinations, we analysed 25 infants, children, and adolescents (aged 6 weeks to 27 years, median age 9.9 years, 18 males, and seven females) with aortic valve stenosis, with or without mild to moderate aortic insufficiency, but without additional

Plasma levels of B-type natriuretic peptide in children and adolescents with aortic valve stenosis

We read with interest the recent article of Weber et al.,1 which investigated the relation of N-terminal (NT) pro-B-type natriuretic peptide (BNP) to progression of aortic valve disease. According to their results on a large cohort, the authors considered NT-proBNP as a suitable biomarker for the evaluation and monitoring of patients with aortic valve disease. On the basis of our own experience in paediatric patients, we would like to add a note of caution regarding the diagnostic accuracy of natriuretic peptides in aortic stenosis.

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congenital heart disease. In 22/25 patients, BNP plasma level was normal according to age- and sex-matched controls (between 5 and 21 pg/mL), despite an invasive pressure gradient of up to 105 mmHg, a left ventricular systolic pressure of up to 220 mmHg, and a markedly increased left ventricular hypertrophy in several patients. Plasma BNP was slightly increased when compared with healthy controls in 2/25 patients (22 and 40 pg/mL, 14.5- and 13.8-year-old boys, peak-to-peak gradient 90 and 65 mmHg, respectively). Only in one case, we found a markedly increased plasma BNP level of 195 pg/mL (age- and sex-matched controls: 8.5 ± 7.5 pg/mL). This 7-week-old girl had aortic valve stenosis with a systolic invasive pressure gradient of 40 mmHg, without left ventricular hypertrophy, but with an increased left ventricular diastolic and systolic dimension (shortening fraction 30%).

We could not find any correlation between plasma BNP level and invasive peak-to-peak gradient or maximal aortic velocity assessed by continuous-wave Doppler echocardiography. To our experience, in children and adolescence, it would be harmful to wait for elevated BNP levels before initiating interventional or surgical treatment.

We estimate that elevated plasma levels of BNP do not correlate to the severity of aortic valve obstruction but reflect the occurrence and the degree of left ventricular dysfunction. Although we cannot exclude that plasma NT-proBNP and BNP show different patterns, the similar results for BNP and NT-proBNP in adults with aortic stenosis reported by Gerber et al. do not support this suspicion. More probably, the differences between paediatric and adult patients may be explained by the greater possibility for compensation of left ventricular function in younger patients than in elderly patients with aortic stenosis.

These differences according to age should be taken into account. We believe, the younger the patient, the less reliable is the measurement of natriuretic peptides for monitoring patients with aortic valve stenosis.

Impact of ventricular response irregularity in patients with atrial fibrillation and heart failure

We read the recent article by Melenovsky et al. concerning functional impact of ventricular response irregularity in patients with atrial fibrillation (AF) and heart failure and found it interesting. The authors found that irregular biventricular pacing produced an impairment in left ventricular (LV) function in comparison with regular pacing when the mean rate was 120 b.p.m., but not when the mean rate was 80 b.p.m. The results of the study highlight that rate control rather than regularization should be the primary aim in these patients.

We would like to point out an important aspect in the interpretation of these findings. A correct evaluation of the effect of heart rate on differences in LV performance between regular and irregular pacing requires algorithms of irregular stimulation to produce comparable degrees of RR interval variability and/or irregularity at different mean rates. This condition is a major issue for such an analysis, as it allows us to rule out the hypothesis that the detrimental changes in cardiac function observed during irregular vs. regular pacing at high rates, but not at normal rates, may derive from different RR variability or irregularity indices, and not from the increase in mean ventricular rate itself. Unfortunately, a comparison of such parameters between the ventricular cycle series used to stimulate irregularly at 80 and 120 b.p.m. was not reported in the text. The authors used computer-generated irregular sequences of RR intervals that followed the discrete Poisson distribution—a well-known probability function that accurately describes the probabilistic behaviour of the ventricular response to AF. However, a Poisson-distributed series of ventricular cycles may be characterized by consistently different degrees of RR interval variability, depending on the mean cycle length, the histogram bar width, and the minimal RR interval under which the probability reduces to zero, i.e. the absolute refractory period of the atrioventricular node. Beat-to-beat variability and RR irregularity indices may also vary significantly, even in distributions with similar mean values and standard deviations.

These considerations raise the problem of defining the concept of 'comparable' degrees of variability and/or irregularity at

References


Plasma levels of B-type natriuretic peptide in children and adolescents with aortic valve stenosis: reply

We thank Drs Koch and Singer for reporting their interesting findings on B-type natriuretic peptide (BNP) values in children and adolescents with aortic valve stenosis. These are very important data indicating a limitation of BNP and probably also NT-proBNP assessment to evaluate severity of aortic valve disease in children and young adults.

In our study, only adult patients with calcifying aortic valve disease were included. For these patients, an elevation of BNP and NT-proBNP related to severity of aortic valve disease or functional status has been consistently shown in several published studies including at least 750 patients. Thus, it has to be assumed that the different release pattern in children with aortic stenosis is related to differences in pathophysiology-like differences in left ventricular myocardial adaptation and compensation.

Therefore, our results cannot be extrapolated to a paediatric population. The diagnostic value of BNP and NT-proBNP in these patients has to be assessed in separate studies.

Dr. Michael W. Weber
Kerckhoff Heart Center Beneke Strasse 2-8 61321 Bad Nauheim Germany Tel: +49 603 299 60 Fax: +49 603 299 623 13 E-mail address: M.Weber@Kerckhoff-Klinik.de
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