Letter to the Editor


Metoprolol reduces the coronary collateral flow index: alternative explanations

It was with great interest that I read the elegant work of Billinger et al. [1] which attempted to investigate the haemodynamic effects of metoprolol on the coronary collateral flow index (CFI) immediately after percutaneous transluminal coronary angioplasty (PTCA) in the setting of chronically developed collateral pathways. The authors suggested the hypothesis that the reduction in CFI secondary to metoprolol administration is mostly due to its direct vasoconstrictive effect on collateral vessels. In addition to the authors’ conclusions, I would like to point out two supplementary mechanisms leading to post-procedural reduction in collateral flow.

First, the intramyocardial collateral fraction accounts for one major component of the coronary collateral flow [2]. Furthermore, the collateral flow displays a predominant systolic profile which highlights the importance of systolic function as a crucial provider of total coronary collateral flow [3,4]. As the metoprolol reduces the myocardial contractility and as none of the patients had previous contractility anomalies in the area of PTCA, it is therefore not surprising to observe a reduction of CFI following administration of metoprolol.

Second, independent of pharmacologic interventions, a subsequent reduction in collateral flow following the instauration of antegrade coronary flow has been advanced as “functional downregulation of collateral flow” [5]. As the flow measurement subsequent to metoprolol administration is carried out after the third balloon inflation, the participation of this speculated mechanism in CFI reduction cannot be formally excluded.

Finally I like to congratulate the authors once more for their very well structured clinical trial and their consequent contribution to this ongoing field.

Sincerely yours
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References


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doi:10.1016/j.ehj.2004.05.032

Relationship between reduced elasticity of extracardiac vessels and left main stem coronary artery disease

To the Editor,

With great interest we read the study on reduced vessel elasticity in left main stem (LMS) coronary artery disease (CAD) by Hadjinikolaou et al., published recently in the Eur Heart J [1]. The authors elegantly showed, by careful ex vivo experiments, that the elasticity of extracardiac arteries and veins is impaired in patients with LMS-CAD compared to non-LMS CAD.

These findings are well in line with our observation that increased arterial stiffness and wave reflections, as assessed by non-invasive pulse waveform analysis (PWA), are independent risk markers for CAD. Moreover, we found statistically significant correlations between the extent of CAD and the Augmentation Index (AIx), a measure of increased wave reflections, as well as time-to-return of the reflected wave (Tr), a surrogate for pulse wave velocity and thus arterial stiffness. Both measures provide information about the mechanical properties of the complete arterial system. Extended analysis of our study population revealed even more support for Hadjinikolaou’s study: We analysed 394 patients (259 men, mean age 66.4 years), undergoing coronary angiography as well as non-invasive PWA at our institution, with a final diagnosis of CAD, excluding those with valvular heart disease or impaired systolic function, as the AIx is sensitive to these. 23 were found to have LMS-CAD, the others had distal CAD without significant involvement of the LMS. Patients with LMS-CAD had increased wave reflections, as indicated by a higher AIx, as well as higher arterial stiffness, manifested by a shorter Tr (Table 1).

In conclusion, patients with LMS-CAD are characterised by impaired mechanical properties of their circulatory system. This can be shown likewise by non-invasive PWA and by ex vivo methods and might contribute to their worse prognosis, even after revascularisation.3

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Table 1 Increased wave reflections (Augmentation Index — AIx) and shorter time-to-return of the reflected wave (Tr — indicating higher pulse wave velocity and arterial stiffness) in patients with left main stem (LMS) coronary artery disease (CAD) compared to patients with non-LMS CAD

<table>
<thead>
<tr>
<th></th>
<th>LMS-CAD</th>
<th>Non-LMS CAD</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIx</td>
<td>32.4 ± 12.3</td>
<td>25.1 ± 12.3</td>
<td>0.006</td>
</tr>
<tr>
<td>Tr (ms)</td>
<td>130.3 ± 14.4</td>
<td>136.2 ± 13.1</td>
<td>0.04</td>
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</tbody>
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Letter to the Editor


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doi:10.1016/j.ehj.2004.05.030

Prognosis of heart failure in idiopathic dilated cardiomyopathy: has it improved in tertiary referral centres over the last 10 years?

We read with great interest the recent article of Schaufelberger et al. about improvement of the prognosis of heart failure between 1988 and 2000 in Sweden. Current medical and non-medical therapies (such as the use of ACE-inhibitors and beta-blockers, more frequent use of implantable cardioverter defibrillators, and bi-ventricular pacing have improved the overall prognosis in heart failure whatever the aetiology.

Treatment of myocardial infarction has had major changes during this period. In contrast, it is unclear whether prognosis was really improved in the particular setting of patients with idiopathic dilated cardiomyopathy (IDC) seen in the tertiary referral centres. Despite the improvement of the medical strategy in heart failure and its recognised efficacy, the prognosis of patients with well characterised IDC seemed unchanged in centres similar to our institution in the recent years. The recruitment of more severe patients (larger left ventricular diameters, lower left ventricular ejection fraction and/or higher filling pressures) that might be candidate to some non-medical therapies may thus induce a selection bias in these centres.

Reference


Atrial (ANP) and B-type natriuretic peptide are peptide hormones released in response to myocyte stretch. Novel biomarkers have been evaluated for four clinical purposes: prognostic screening in asymptomatic individuals, risk stratification in patients with clinical disease, diagnosis of clinical disease in patients with symptoms of uncertain cause and guidance in the selection or titration of drugs in patients with known disease. A dramatic rise in the number of studies exploring the potential clinical use of measurement of the natriuretic peptides for all the above purposes has been observed after the development of commercially available assays for both B-type natriuretic peptide and its inactive N-terminal fragment.

Left ventricular hypertrophy (LVH) is a strong risk factor for future cardiovascular events independent of blood pressure levels in patients with essential hypertension. Electrocardiographic criteria for LVH have proven to be insensitive. The most widely used method for estimating LVH is echocardiography, but it seems to be uneconomical. Therefore, a simple screening test might be highly useful for early identification of patients who might benefit from intensive therapy. In the literature, there are conflicting reports about the use of BNP as a marker for LVH in hypertensives. We have shown that in patients with essential hypertension, plasma BNP levels were positively correlated with left ventricular mass index, while plasma ANP levels did not have a similar correlation. Plasma concentrations of BNP and ANP were slightly higher in hypertensive patients in comparison to normotensive controls, but there was no statistically significant difference between the two groups. Plasma BNP and ANP levels were not correlated with systolic, diastolic and pulse pressure.

The above results could be explained from the secretion of BNP primarily by ventricular myocytes. Elevated plasma BNP levels are considered to reflect ventricular structural and functional alterations. Increased cardiac chamber wall stretch is the major stimuli for the release of natriuretic peptides. ANP secretion is immediately released in response to atrial stretch, while BNP secretion usually requires a long-term stimulus. However, it is well-known that plasma BNP levels are affected by age, gender renal failure and drugs, such as β-blockers and diuretics. It has also been reported that plasma BNP levels may be elevated before the establishment of left ventricular hypertrophy in hypertensive patients. In conclusion, although it seems that plasma B-type natriuretic peptide is related to left ventricular mass in patients with essential hypertension, the above mentioned multi-factorial aetiology of elevated plasma BNP levels shows that they must not be used as a replacement for echocardiography in the clinical assessment of left ventricular hypertrophy.

References


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