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EDITORIAL COMMENT

The haemodynamic cascade

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In this issue of the Netherlands Heart Journal, Jansen et al. describe the influence stress echocardiography has on clinical decision-making in patients with mitral valve disease [1]. In 1935, Tenant and Wiggers described the sequelae of coronary occlusion [2]. From their description the ischaemic cascade has been derived. In this cascade, symptoms are a relatively late phenomenon and are preceded by electrocardiographic changes. This has led to the routine application of stress electrocardiography in the evaluation of patients with suspected coronary artery disease. In patients with valvular heart disease one could assume the existence of a haemodynamic cascade. In the haemodynamic cascade, which starts with seemly insignificant valve defects, the second phase would be haemodynamic changes, followed by cardiac adaptation and as the last phase, symptoms. Analogous to stress electrocardiography, stress echocardiography can then be applied to relate symptoms with echocardiographic findings.

Stress in patients with mitral valve regurgitation may provoke an increase of the right ventricular systolic pressure (RVSP) as the increase of heart rate leads to an increase in regurgitant volume per unit of time. In case of mitral valve stenosis, acceleration of the heart rhythm leads to a decreased atrial emptying time resulting in an intra-atrial pressure rise and hence a rise in RVSP. Nowadays, RVSP can be determined non-invasively using conventional Doppler echocardiography. The problem in the clinical management of patients with valvular heart disease is that their symptoms might be caused by physical conditions other than their valvular heart disease, such as obesity and diastolic dysfunction to mention a few. A discrepancy may exist between the presenting complaints and the echocardiographic findings. The application of stress allows one to correlate symptoms with the intensity of stress and the corresponding haemodynamic changes. According to the haemodynamic cascade, guidelines based on symptoms are obsolete. Guidelines based on dimensions are still used but in the era that valve function can be determined non-invasively by Doppler echocardiography, guidelines should better be based on the haemodynamic consequences of valvular heart disease.

Theoretically, it should be possible to derive a heart rate/RSVP index in which the denominator is determined by guidelines and the numerator by the heart rate. The value of this index easily determines whether an operation has to be performed or not. For instance: the denominator is now 60 mmHg, (guidelines). If this value has been reached during stress with a heart rate of 120 beats/min, the value of this index is 2. If an RVSP of 60 mmHg has been reached during exercise with a lower heart rate, more serious valve disease, the index value diminishes. A value below 2 indicates intervention. Less serious valve disease is indicated by an index value above 2, above which an intervention is not needed. The discerning index value is determined on one hand by the guidelines (the denominator, the value above which an intervention is recommended) and on the other hand by the experience of the intervention cardiologist/surgeon. Increase of the index value means that an intervention is recommended in a less severe state of valve disease.

The application of stress and the estimation of RSVP is advised in the routine follow-up and management of patients with valve disease, which, in our experience, is often omitted [3].

In their article, Jansen et al. demonstrate in an elegant way that stress echocardiography has additional value in more than two-thirds of their patients. They evaluate the change in RVSP by echocardiography while the patient is cycling in a semi-supine position. This position alone can degrade the quality of the echocardiogram. Braunwald suggests that the estimation of the pulmonary artery pressure is done in the rest phase after stress. The majority of our patients are not in an optimal physical

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condition in which exercise would result in a steady rise and fall of the heart rate; rather, the less then optimal physical condition of our patients results in the maximal heart rate being reached in a relatively early stage of stress and being maintained long after discontinuation of stress. One therefore has time enough to determine RSVP in the rest phase. In our routine daily practice, we use an exercise step. During stress, the electrocardiogram is controlled and, once the patient has reached his/her point of maximal exertion, the RVSP is determined. All that is needed is a continuous wave recording with the ultrasound beam through the tricuspid valve. The position of the patient and the probe producing an optimal flow signal are determined during the echo preceding the application of stress; in other words echo-stress-echo.

In their paper, Jansen et al. also evaluated the regurgitant volume and were able to demonstrate an increase in mitral regurgitation severity in 9/14 patients. As the authors indicate in their discussion, reliable results are difficult to acquire without experience. This makes it perhaps less suited in practice. The question is: if it is possible to reliably determine the total cross-sectional area of the regurgitant jets, would this information be valuable in clinical decision-making? The answer, in view of the haemodynamic cascade, is yes. It is to be expected that the regurgitant volume, determined by the cross-sectional area of the vena contracta of the regurgitant jet and the driving force, precedes the development of cardiac adaptation and therefore plays a pivotal role in this process. This might allow intervention in an earlier state which, in the long term, would benefit the patient.

In their evaluation of mitral valve stenosis, 7 patients had organic mitral valve stenosis and 7 had mitral valve stenosis after a previous mitral valve repair operation. This may explain why there was no consistency in the stress-induced change in RSVP. In this group, Jansen et al. determined, apart from the RVSP, also the mean gradient across the mitral valve. This seems to be a valuable measurement as it allows one to differentiate between RVSP increase due to the valve stenosis or due to increased pulmonic vascular resistance. They indicate that not every patient with an RSVP of more than 60 mmHg had severe mitral valve stenosis, but patients with a mean gradient of more than 15 mmHg during exercise did.

In their paper, Jansen et al. made a distinction between patients with mitral valve regurgitation and mitral valve stenosis. In daily practice, a combination of stenosis and regurgitation is often encountered. In this patient population, the determination of RSVP after stress is possible and may hence be of additional value in their management .

Demonstrating the use of stress echocardiography and the important role it can play in the clinical management of patients with valvular heart disease, is not what makes Jansen et al.'s paper noteworthy. It is to their credit that they aim to highlight the value of a conventional Doppler-based method in the care of patients with mitral valve disease.

References

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