not be an indicator alone for aortic valve replace-
ment. As a general rule, the indication for valve
replacement is given when there is: severe valve
obstruction (pressure gradient $\geq 50$ mmHg and aortic
valve area $\leq 0.8$ cm$^2$); clinical symptoms (angina
pectoris, pre-syncpe or syncope, congestive heart
failure); inadequate pressure rise or pressure fall
during exercise.

Conclusions

In the paper of Lund et al.$^{[1]}$ several risk factors have
been identified which are associated with an adverse
outcome after valve replacement, mainly the presence
of systolic and diastolic dysfunction. These function
parameters seem to be useful prognostic indicators
for postoperative outcome, especially in patients with
concomitant coronary artery disease. However, previous
studies have shown that the occurrence of
isolated diastolic dysfunction is no indication for
earlier operation when valve stenosis is not severe.

L. MANDINO
P. KAUFMANN
W. MAIER
O. M. HESS
Inselspital, Bern,
Switzerland

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Pulmonary arterial morphology and intravascular pressure
revisited

See page 1988 for the article to which this Editorial
refers

The relationship between pulmonary arterial
morphology and haemodynamics is important, but
still unresolved.

Borges et al.$^{[1]}$ have attempted to contribute to
this question, using the correlation between intra-
vascular ultrasound imaging and pulmonary haemo-
dynamic data obtained by routine right heart
catheterization.

Intravascular ultrasound is a relatively new
method and this article is a pioneer work. Intravas-
cular ultrasound is an invasive method and its
clinical application is fully justified only when it can
obtain substantially more information than other
methods.

Borges et al.$^{[1]}$ conclude that intravascular
ultrasound is capable of assessing vascular muscular
hypertrophy and intimal proliferation. Nevertheless,
no significant correlation was found between these
changes and pulmonary arterial hypertension. This
finding, which corroborates previous observations, is
not surprising: histological changes within the pulmo-
nary arteries are at the same time the consequences of
existing pulmonary hypertension and the cause of its
further evolution so that the correlation between
these two variables will always be only marginal with
a significant overlap. Moreover, the authors demon-
strate that intravascular ultrasound cannot predict
the functional vasodilatory reserve of the resistance vessels.

There is, however, one important contribution of intravascular ultrasound to the physiology of pulmonary circulation: intravascular ultrasound focused our attention on the fact (well known but often neglected) that the pulmonary blood flow is pulsatile and that, consequently, a simple calculation of pulmonary vascular resistance does not exactly express the right ventricular afterload\(^1\). A correlation between morphology and haemodynamics could be particularly interesting in this specialized field.

**The clinical use of intravascular ultrasound**

There is no doubt that this method could yield useful information in patients with thromboembolic pulmonary hypertension\(^3\): it could discover the small endothelial abnormalities which can escape us in angiography but which are important to our decision making.

Further diagnostic contributions by intravascular ultrasound seem to be limited: it is known that the morphological changes within the pulmonary vessels are always the same regardless of the origin of the pulmonary hypertension (except perhaps its so-called 'primary' form)\(^4\).

The authors emphasize the possible importance of intravascular ultrasound in the assessment of patients before heart transplantation in order to assess the reversibility of secondary pulmonary hypertension in these cases. There is no doubt about the significance of data concerning the reversibility of pulmonary hypertension, but it is debatable whether intravascular ultrasound is the most reliable method to obtain these data.

As for the exactness: it is understood that the 'gold standard' for this classification is the lung biopsy. It has been pointed out\(^4\) that there is a clear cutting point indicating the start of irreversibility of pulmonary hypertension: the presence of plexiform lesions. Such a detailed description of pulmonary vasculature could hardly be achieved by intravascular ultrasound.

Borges *et al.*\(^1\) emphasize that the lung biopsy is not free of complication and therefore could be of risk in cardiac patients, but acceptable for open lung biopsy performed by thoracotomy (although I do not know the complication rate, especially in cardiac patients). The complication rate, however, for a biopsy performed by, for example, the transbronchial route should be lower and the diagnostic gain sufficient\(^5\), as judged from diagnostic biopsies in chronic lung diseases.

Another invasive method for the pre-operative examination of patients undergoing heart transplantation is a direct measurement of the vasodilatory response in the pulmonary vascular bed, i.e. the measurement of pulmonary arterial pressure and cardiac output and the calculation of pulmonary vascular resistance following either prostacycline or nitric oxide\(^6\).

Intravascular ultrasound and haemodynamic measurements can be performed simultaneously via right heart catheterization. Therefore, similar studies should be performed particularly for two drugs mentioned above. It is probable that the results of both methods could be complementary and that their combination may contribute to our better understanding of the problem.

In conclusion, intravascular ultrasound as a new diagnostic and research method is ready to be compared with more widely used approaches and further studies are mandatory.

V. JEZEK

Clinic of Pneumology and Thoracic Surgery, Hospital Bulovka, Prague, Czech Republic

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