closely monitoring this variable during the first postoperative year.

An important implication of this study suggests that inflammation is an important mediator of homograft stenosis and dysfunction. The authors defend this view on the grounds that stenosis happens at all levels of the graft, including the proximal suture line, mid conduit, and distal suture line, with nearly 60% of their stenoses occurring midway along the conduit. In conjunction with the finding that all peak pulmonary gradients upon leaving the operating room were <10 , these morphologic patterns of stenosis suggest that dysfunction is unlikely to be related to surgical technique. Based on data from their series, they suggest that the decellularized homograft may perform better in the long run than the standard homograft because it may be less proinflammatory. Although the low rate of homograft dysfunction in their series is impressive and despite the fact that we routinely utilize a decellularized homograft

for pulmonary valve pathology, it may be early to say that decellularized homografts ought to become the standard homograft. These data do not yet constitute long-term results because the median follow-up was only 2.2 years, and the type of homograft employed was not significantly associated with the cumulative incidence of homograft dysfunction in their study. Despite these limitations, this study provides invaluable, granular data that improves our understanding of homograft dysfunction, and may lead to an incremental reduction in homograft dysfunction after the Ross procedure, which continues to be the Achilles' heel of this operation.

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Commentary: I want to believe it... so it must be true

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Chauvette and colleagues¹ present a retrospective review of a large cohort of patients undergoing the Ross procedure using data collected from the Canadian Ross Registry. An important objective was to evaluate the outcomes of decellularized pulmonary homografts used to reconstruct the right ventricular outflow tract. Patients were reviewed with the aim to assess the incidence, predictors, progression, and morphology of homograft dysfunction. The

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CENTRAL MESSAGE

Decellularized homografts have greater durability than standard grafts. This is a finding that many, if not all of us, can easily get behind. But do the methods used in this study justify the conclusions?

data set included nearly 500 patients, with a mean follow-up of just more than 2 years. Homograft dysfunction was defined as a peak gradient >30 mm Hg, pulmonary valve regurgitation $>2+$, or the need for homograft reoperation. Thirty patients (6%) met at least 1 criterion for

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dysfunction: 28 with stenosis and 2 with regurgitation. By multivariable analysis, only younger age was predictive of dysfunction. Decellularized homografts had a lower rate of failure.

Although the authors' conclusion regarding improved longevity for decellularized grafts is easy to accept and very likely to be true, readers should view these findings with some uncertainty. First of all, this was a retrospective study with very short follow-up. Although the finding of greater dysfunction among standard grafts is evident, longer follow-up is necessary to be sure this remains the case. Second, the primary mode of failure was stenosis, with most failed grafts demonstrating diffuse stenosis. However, fully 25% of the failed grafts did so because of proximal or distal suture line stenosis, a finding that possibly could be due to technical factors. Would removing these grafts from the analysis change the authors' findings? Lastly, but perhaps most importantly, any comparison of decellularized versus standard homografts in this specific patient cohort must be interpreted in light of differences between the groups. The authors provide a propensity-matched comparison of the standard versus decellularized grafts but these results should be taken with a grain of salt because of the small number of standard homografts and the confounding factor of age. The authors already concluded that younger age was

the only predictor of failure, so is age the issue or is it that failure is caused by the homograft preparation itself? Other findings include a clinically trivial increase in gradient during the first postoperative year (6 vs 11 mm Hg), a finding that could be at least partially due to changes in right ventricular function or suture line scarring, and the inability to utilize transcatheter techniques because of coronary artery compromise, which is already well established.

It is always easy to accept those things we already believe to be true. Indeed, our own group demonstrated better duration in decellularized grafts² so this article fits well with generally accepted dogma. The findings are certainly important and this study represents a valuable addition to the literature. It is equally important to critically review conclusions and ask fair questions about its scientific validity before accepting the findings simply because we already knew that. After all... I want to believe it, so it must be true.

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