tional or alternative thrombolytic therapy being given and so to early coronary artery reperfusion.

Sigwart *et al* recommended that streptokinase antibodies should be assayed routinely in patients being given thrombolytic therapy after myocardial infarction.[7] The commonly used radioimmunoassay, however, is time consuming and inconvenient and therefore not suitable as a guide to clinical strategies in critically ill patients. Some clinicians routinely measure serum fibrinogen immediately after administration of streptokinase to identify patients in whom delayed or failed reperfusion is likely because streptokinase antibodies are presented.[8] This method is still useful, but there is a quick and easy assay for streptokinase antibodies that can be used for screening and to guide clinical treatment.

J BRÜGEMANN
J VAN DER MEER
VJJ BOM
KI LIE
Department of Cardiology,
University Hospital Groningen,
The Netherlands

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Traumatic rupture of the thoracic aorta diagnosed by transoesophageal echocardiography

SIR,—The case reported by de Belder *et al* confirms the diagnostic value of transoesophageal echocardiography (TOE) in traumatic aortic rupture.[1] They say that the echocardiographic appearances were "previously undescribed". However, I and others first reported the role of TOE in traumatic aortic rupture in 1991,[2] with echocardiographic images similar to their case. Others subsequently confirmed our findings.[3 4] I hope that the interesting report of de Belder *et al* will further promote this rapid, accurate bedside technique to diagnose a frequently fatal condition.

IAN W BLACK
Cleveland Clinic Foundation,
Cardiology F-15,
9500 Euclid Avenue,
Cleveland OH 44195, USA

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Maldevelopment of conotruncal and aorto-pulmonary septum with absent left central pulmonary artery: anatomical and clinical implications.

SIR,—We were most interested in the excellent report by Schulze-Neick *et al* in which they described a case of atresia of the pulmonary valve associated with absence of the central component of the left pulmonary artery.[1] We would like to suggest an alternative interpretation of the underlying morphological malformation.

We are confused by their assertion that there was no arterial ligament or duct. As the left pulmonary artery had no central connection with the pulmonary trunk, from where did it receive its blood flow before the establishment of the modified Blalock-Taussig shunt? If, as we suspect, there was a connection with the concavity of the aortic arch, surely this vessel should be regarded as representing the arterial duct?

Failure of incorporation of the orifice of the right sixth aortic arch into the confluence of the pulmonary arteries, caused by deviation of this orifice or of the aorto-pulmonary septum, results in a single (right) anomalous pulmonary artery arising from the ascending aorta (a condition sometimes erroneously called a "hemitruncus"), in which case the anomalous pulmonary artery remains entirely separated from the pulmonary trunk and the left pulmonary artery. It is difficult to see how the right sixth arch could have two proximal ends with one opening into the aorta and the other joining the pulmonary trunk. The distal end of this arch cannot be involved because had it persisted, it would be connected with the right subclavian artery, and because the non-arcuate portion of the right pulmonary artery terminates in the right lung. We consider that this anomaly was more likely to

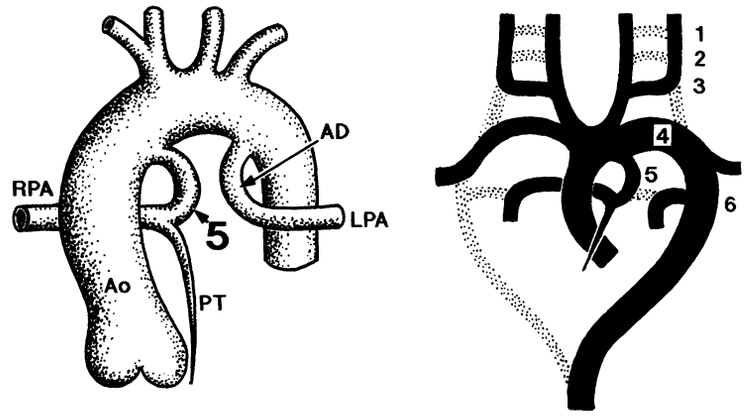

Figure 1 Diagrams of our interpretation of the state of the great arteries in the case described by Schulze Neick, et al[1] and in our case (fig 2), and of the underlying embryological arrangement. Ao, aorta; AD, arterial duct; LPA, left pulmonary artery; PT, central pulmonary trunk; RPA, right pulmonary artery; 1–6, embryological aortic arches.

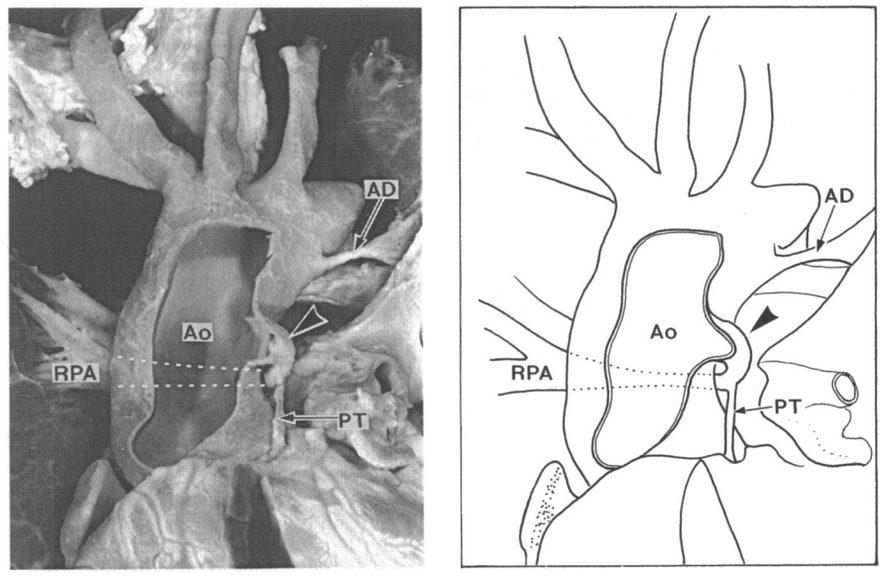

Figure 2 Photograph and key diagram of the specimen in the Brompton collection. The anomalous origin of the right pulmonary artery is indicated by the large arrow head. Ao, aorta; AD, arterial duct; LPA, left pulmonary artery; PT, central pulmonary trunk; RPA, right pulmonary artery; 1–6, embryological aortic arches.