and

$$\cos \alpha = (R_1 - R_2)/(R_1 + R_2).$$

Using this formula, the d_{eff} of various combinations of commonly used balloons is shown in Table 1. The area method consistently results in an underestimation of d_{eff} by approximately 1 to 3 mm.

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Reply

We have considered Yeager's suggestion about calculation of effective dilating diameter carefully and have concluded that he is, in fact, correct. The assumption should be made of constant perimeter rather than constant area. Indeed, we have incorporated his suggestions into our own use of the double balloon technique.

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Quantitative Morphology of the Aortic Arch

Morrow et al. (1) recently presented quantitative echocardiographic data concerning the morphology of the aortic arch in neonatal coarctation. They misquote the fundamental segmental measurements of the thoracic aorta introduced by Sinha et al. (2) in 1969. They also neglect to refer to a recent morphometric study of the aortic arch (3) that is similar in design and results and is based on extensive echocardiographic (n = 70) and angiographic (n = 91) measurements.

Sinha et al. (2) present both in the text (p. 386) and in Figure 3 (p. 387) a quantitative classification and discriminant function analysis of aortic arch pathology based on measurements of the transverse arch and descending aorta, and *not* the "pulmonary valve dimension" as stated by Morrow et al. (1) (p. 619).

Lappen et al. (3) presented echocardiographic and angiographic data indicating specifically that hypoplasia of the transverse aortic arch (TAA) is almost universally present in coarctation of the aorta, and importantly, persists long after the neonatal period (body surface area 0.2 to 1.4 m^2). Lappen et al. (3) further introduced the diameter of the left common carotid artery (LCCA) as a norm against which the diameter of the transverse aortic arch can be quantitated. There is indeed no appreciable difference between the quantitative data of Lappen et al. (3) for the LCCA/TAA measurements (0.84 ± 0.10 for coarctation versus 0.55 ± 0.03 for normal controls by echocardiography, and 0.83 ± 0.14 for coarctation versus 0.45 ± 0.12 for normal controls by cineangiography) and the same measurements of Morrow et al. (1) (LCCA/TAA of 0.96 ± 0.18 for coarctation versus 0.48 ± 0.08 for normal controls). Furthermore, Lappen et al. (3) found the same LCCA/TAA ratio in patients who had coarctation and a large ventricular septal defect. Transverse aortic arch hypoplasia could not be demonstrated in patients with a large left to right shunt or other significant defects who had no associated coarctation of the aorta.

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Reply

The hypothesis in our study was that measurements of the great arteries were a reflection of intrauterine blood flow relations and that coarctation was associated with redistribution of blood flow in utero. This hypothesis was supported by the finding that transverse arch hypoplasia with dilation of the pulmonary artery was present with coarctation. Once significant systemic hypertension results from coarctation, the presumption that structure size is a reflection of blood flow is not valid and therefore we excluded all patients older than 1 month of age. Our study clarifies the cause for the right ventricular preponderance and right ventricular hypertrophy on the electrocardiogram with coarctation of the aorta despite relatively low pulmonary artery pressures, which were described as a "misleading reflection of the physiologic status" by Sinha et al. Right ventricular hypertrophy is present because of the marked increase in right ventricular stroke work, which is secondary to aortic arch obstruction in utero. We appreciate the information of Lappen et al., but it was not included in our discussion because of differences in methodology—namely the use of the left carotid artery to normalize the measurements by angiography and echocardiography. Perhaps the most important finding of our study was the increase in the size of the left carotid artery in neonates with coarctation compared with normal subjects, suggesting increased carotid blood flow in utero and immediately after birth. We speculate that cerebral complications of coarctation late in life may be due, in part, to increased cerebral blood flow in utero. Hypoplasia should not be viewed as a separate diagnosis and almost never requires surgical treatment. It is simply a reflection of altered blood flow relations in the developing great arteries in fetuses with coarctation of the aorta.

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