Vulvar Heart Disease in Pregnancy (letter)
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TO THE EDITOR: In their review of valvular heart disease in pregnancy, Reimold and Rutherford (July 3 issue) do not address the early puerperium. In our opinion, this period may be crucial. Many clinicians tend to believe that pregnancy in a patient at risk has been successfully completed after an uncomplicated delivery. Although the authors briefly discuss issues related to cardiovascular physiology immediately after delivery, the literature emphasizes the importance of the puerperium. Confidential inquiries into maternal deaths have revealed that care may be suboptimal during the postnatal period, since the intensity of monitoring is often decreased at this time, despite the fact that the majority of deaths occur after delivery.

The early puerperium may be a period associated with a risk of heart failure because of the physiological return of extravascular fluid from the limbs and lower body to the systemic circulation. This mobilization phase may take nearly a week. Clinicians should be aware of this risk and be advised to conduct continuous, close monitoring for a minimum of 72 hours after delivery, preferably in a multidisciplinary setting.

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Valvular Heart Disease in Pregnancy

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Cardiovascular Genomics

TO THE EDITOR: I believe that Table 2 of the article by Nabel (July 3 issue) gives misleading information concerning apparent mineralocorticoid excess: this disease is said to be due to mutations in the gene encoding 11β-hydroxylase, but in fact, mutations in this gene cause one form of congenital adrenal hyperplasia. Apparent mineralocorticoid excess is caused by inactivating mutations in the gene encoding 11β-hydroxysteroid dehydrogenase type 2, the microsomal enzyme that metabolizes cortisol into its receptor-inactive keto form, cortisone, in sodium-transporting epithelia, such as the kidney, and thus protects the nonselective mineralocorticoid receptor from occupation by cortisol itself. In the same table, apparent mineralocorticoid excess is said to be associated with an absence of circulating aldosterone and decreased plasma volume, but in fact, plasma volume is expanded, as in states involving true mineralocorticoid excess, because of sodium retention induced by the unopposed activation of the aldosterone receptor by cortisol.

As a very rare condition, apparent mineralocor-