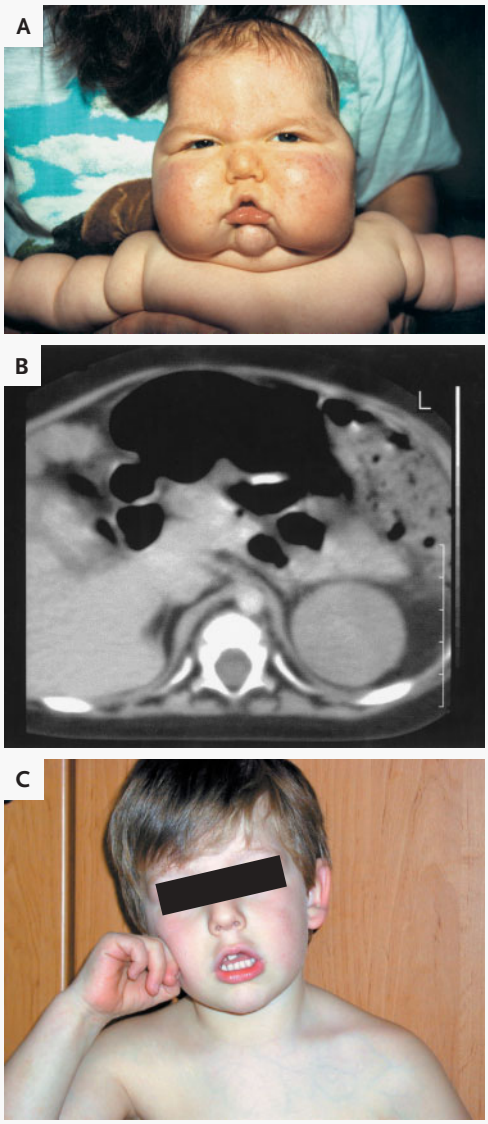

Figure 1. A Male Infant with Unusual Weight Gain and a Rash.

Panel A shows the child at seven months of age, and Panel B the solid mass in the region of the left adrenal gland. After excision of the mass, the child was healthy, as pictured at four and a half years of age (Panel C).

adrenal adenoma. At four and a half years of age, the child was healthy, and his psychomotor development corresponded to his age (Fig. 1C).

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Editor's note: There were 1188 responses submitted from 72 countries. Sixty-seven percent of the respondents suggested abnormalities consistent with Cushing's syndrome; of this group, 11 percent specifically diagnosed Cushing's disease, which this boy did not have, and 8 percent suggested a variety of exogenous sources of cortisol (such as topical cream used for the rash or from maternal use passed to the baby in breast milk). Cushing's syndrome refers to the clinical consequences of excess cortisol, whereas Cushing's disease — a specific cause of excess cortisol — is due to a pituitary adenoma and is named in recognition of the neurosurgeon, Dr. Harvey Cushing, who first described the syndrome and related it to a pituitary lesion. These conditions are discussed in the Case Records that appear elsewhere in this issue of the *Journal.*[2]

Many alternative diagnoses were suggested. Eight percent of the respondents suggested other endocrinopathies (such as the hypothyroidism, leptin deficiency, and excess growth hormone), 14 percent suggested a variety of congenital syndromes (such as the Prader–Willi syndrome), and 9 percent suggested various other conditions (such as the nephrotic syndrome and overfeeding). Two percent of the respondents suggested maternal illness as the diagnosis, such as gestational diabetes. Many insightful comments were received from readers, including this one from Alisa McQueen: "This infant's history of weight gain, moon facies, acneiform eruption, and hypertension suggests the presence of excess cortisol."

1. Hartmann H, Schumacher U. A medical mystery. N Engl J Med 2005;352:273.
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Peripartum Dissection of the Right Coronary Artery

TO THE EDITOR: Spontaneous coronary dissection is a rare but serious complication in the peripartum and postpartum periods.[1,2] However, we disagree with Frimerman and Meisel (Nov. 11 issue)[3] about the diagnosis in the Images in Clinical Medicine article.[3] First, the luminal structure that is described

as dissection extends to the outside of the vessel. Second, the injection of contrast medium does not show a possible connection between the lumen and the luminal space in question. Third, the luminal space remains visible after stent deployment. Given the normally thin appearance of coronary arteries in young persons[4] and the characteristic anatomical course of cardiac veins, we believe that what is seen is a cardiac vein, not arterial dissection. We think this is the correct interpretation of these particular intravascular images. At least, these video clips do not show signs of dissection. Although this patient's diagnosis remains a mystery, refractory coronary spasm, which can also reportedly be addressed with intracoronary stent placement,[5] might be considered.

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Dr. Fitzgerald reports having served as a consultant for Boston Scientific.

1. Koul AK, Hollander G, Moskovits N, Frankel R, Herrera L, Shani J. Coronary artery dissection during pregnancy and the postpartum period: two case reports and review of literature. Catheter Cardiovasc Interv 2001;52:88-94.
2. Lee FH, Yeung AC, Fowler MB, Fitzgerald PJ. Spontaneous postpartum coronary dissection. Circulation 1999;99:721.
3. Frimerman A, Meisel SR. Peripartum dissection of the right coronary artery. N Engl J Med 2004;351:e18 (Web only). (Available at http://www.nejm.org/cgi/content/full/351/20/e18.)
4. Fitzgerald PJ, St Goar FG, Connolly AJ, et al. Intravascular ultrasound imaging of coronary arteries: is three layers the norm? Circulation 1992;86:154-8.
5. Gaspardone A, Tomai F, Versaci F, et al. Coronary artery stent placement in patients with variant angina refractory to medical treatment. Am J Cardiol 1999;84:96-8, A8.

THE AUTHORS REPLY: This young patient had chest pain with ischemia, confirmed by ST-segment depression, echocardiographic stress testing, and myocardial-perfusion imaging. The angiogram showed haziness at the very proximal right coronary artery, and respective intravascular ultrasound images showed a wide, crescent-shaped, false lumen encircling more than half of the arterial circumference, with a free-moving dissection flap entering the true lumen. Neither type of imaging showed spasm. The injected contrast material did not enter the false lumen because of a small communication and free flow within the defect. Moreover, after deployment of the stent, the haziness disappeared, the false lumen seen on intravascular ultrasound images almost disappeared, and the patient was asymptomatic for more than a year. We do not think that the false lumen is a coronary vein because it is not of the proper size and shape. We are not aware of the presence of a large vein so close to the ostium of the right coronary artery. On the basis of these considerations, this case is not a "mystery" but, rather, a rare case of peripartum spontaneous coronary dissection, which was successfully sealed by a stent, with excellent long-term results.

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Imatinib and Regression of Type 2 Diabetes

TO THE EDITOR: We report the case of a nulliparous, 70-year-old woman with long-standing type 2 diabetes mellitus who had regression of the disease during treatment of chronic myeloid leukemia with imatinib, an antineoplastic agent. Type 2 diabetes mellitus was diagnosed when the patient was 62 years of age and weighed 60 kg (body-mass index [the weight in kilograms divided by the square of the height in meters], 24.2) She was treated with diet for one year, oral agents for four years, and insulin thereafter. After the detection of leukocytosis and immature myeloid cells in the blood, chronic myeloid leukemia was diagnosed (in March 2004) and treatment with imatinib (400 mg per day) was initiated. Hematologic remission was documented two months later. During treatment with imatinib, the patient's blood glucose level progressively declined, and insulin doses were titrated down. Insulin treatment was discontinued in June 2004. In July 2004, a standard oral glucose-tolerance test revealed the following plasma glucose values: 6.7 mmol per liter (121 mg per deciliter) while the patient was fasting, 10.7 mmol per liter (193 mg per deciliter) at one hour, and 8.2 mmol per liter (148 mg per deciliter) at two hours. The corresponding serum insulin values were 105, 336, and 315 pmol per liter. Therefore, the diagnosis of type 2 diabetes mellitus was no longer tenable.