able to compensate for the decrease in peripheral resistance by increasing cardiac output by approximately 0.8 l/min. These changes, however, were short-lived, and all hemodynamic parameters returned to baseline within a matter of several minutes.

We also performed radiolabeled microsphere studies and dose-response studies in rabbits,[2] examining hypertonic glucose and hypertonic mannitol. We found that rate and dose were important factors influencing change in systemic vascular resistance and in systemic arterial pressure, *i.e.*, the faster the rate of administration and the greater the osmotic load, the greater the hemodynamic effect. The vascular bed primarily responsive to this hypertonic load was in muscle tissue. One wonders how long the hypotension lasted in the patients studied by Goertz *et al.*, whether this was an effect that was sustained for more than a transient period (as we observed with 25% mannitol), and whether the phenomena might have been caused by vasodilation of the vascular supply to muscle tissue, resulting in a reflex rather than a direct cardiac effect.

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In Reply:—As expected, we found a decrease of arterial pressure in response to hypertonic saline/hetastarch that was caused by peripheral vasodilation. However, the primary aim of our study was to evaluate a possible positive inotropic effect of hypertonic saline. With changes in left-sided loading and possibly left ventricular contractility occurring at the same time, end-systolic pressure-volume (length, area, respectively) relationship (ESPVR) had to be used to assess left ventricular contractility. To obtain the ESPVR-curve, arterial pressure maneuvers must be performed (*e.g.*, phenylephrine bolus administration), which in turn interfere with alterations of blood pressure in response to hypertonic saline. Obviously, a comment on the time course of blood pressure changes after that maneuver is not possible. Furthermore, because we did not use different rates of administration or doses of hypertonic saline, we are not able to comment on the interesting questions addressed by Coté concerning the influence of both variables on the degree of hemodynamic changes.

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Defective Carbon Dioxide Absorber as a Cause for a Leak in a Breathing Circuit

To the Editor:—We would like to bring to attention an unusual cause of a leak in the breathing circuit of a circle anesthesia system involving the carbon dioxide absorber canister (Soda-Sorb NSN 6505-00-782-6484, WR Grace, Lexington, MA).

During a routine preuse machine check, we noted a leak within the breathing system. Visual inspection of the breathing circuit did not reveal the source of the leak, and all joints appeared to be intact. A draft could be felt near the carbon dioxide canisters, initially, we thought that they were misaligned and removed them and changed them from top to bottom. However, the leak persisted. Thinking that the absorbers were still out of alignment, we removed them from the housing. This caused some free granules to fall to the floor. On closer inspection of the canister, it was noted to be defective. The canister is made of a clear plastic cylinder, filled with absorbent

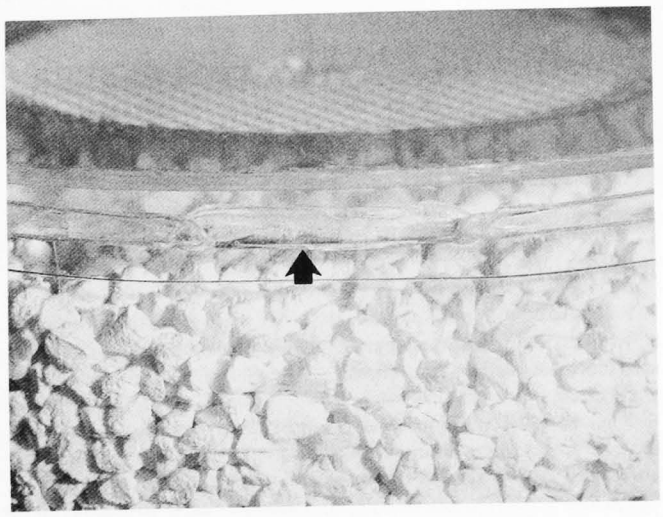

Fig. 1. Arrow showing gap between lid and canister.

granules and covered with a perforated lid, which is then heat-sealed to the top edge of the cylinder. This canister was defective because the edge of the lid had not been properly seated with the canister before heat-sealing. The height of a correctly sealed canister is 89 mm; our canister edge was 94 mm, leaving a 5-mm increase in canister height (fig. 1). This resulted in a gap between the lid and the side wall of the canister, which allowed gases to escape from the breathing circiut. The problem was easily solved by replacing the canister. However, this case reiterates the need to conduct a thorough pre-use check of the anesthesia machine.

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Intraoperative Latex Anaphylaxis Observed in a Farmer

To the Editor:—Contact urticaria to natural latex and its products, such as rubber gloves, balloons, and condoms, is a well known allergy type (type IV). Furthermore, latex hypersensitivity was recently recognized as one of the major IgE mediated intraoperative anaphylaxis (type I).[1] The risk group for latex hypersensitivity includes health-care workers, hospital employees, people wearing household rubber gloves, atopic persons, rubber industry workers, and patients, especially children who have multiple surgeries and catheterizations with latex catheters.[2,3]

We encountered an intraoperative latex anaphylaxis in a patient from the eastern part of Turkey who is an practicing farmer in his own land. This 55-yr-old male patient had undergone a lower lip epidermoid Ca operation. It was noted that the patient was undergoing a surgery for the first time in his life. Meperidine (60 mg) was used for sedation, and induction of anesthesia was achieved using 3 μg/kg fentanyl, 5 mg/kg thiopental, and 0.1 mg/kg vecuronium followed by intubation. Anesthesia was maintained by oxygen/nitrous oxide and 1% enflurane. Forty-five minutes after the start of surgery, blood pressure decreased from 110/80 to 60/30 mmHg, and pulse increased from 80 to 110 beats/min. Extensive urticaria had developed on the skin, and severe bronchospasm was heard. Oxyhemoglobin saturation decreased to 85%. All the anesthetic agents were discontinued, and the lungs were ventilated with 100% O_2. Despite rapid intravascular administration of crystalloid and colloid solutions, blood pressure decreased to 30/0 mmHg. Ten- and 20-μg doses of intravenous epinephrine were given, and 250 mg methyl prednisolone was administered; blood pressure increased to 60/30 mmHg. An epinephrine infusion at a dosage of 0.05 $\mu g \cdot kg \cdot^{-1} \cdot min^{-1}$ was started. It took about 15 min to stabilize the patient. Anesthesia was maintained with a low-dosage fentanyl infusion, nitrous oxide/oxygen, and the operation was completed within 20 min. Because the patient was not considered to be in the risk group, latex anaphylaxis was not suspected during the operation. An intravenous infusion of epinephrine was discontinued after the patient was taken to the post-operative intensive care unit, and the trachea was extubated uneventfully. Six weeks after discharge, skin test results with fentanyl, thiopental, and vecuronium were negative. Further history revealed that, in his daily farming activities, the patient often used latex gloves that were causing urticaria in his hands and arms. Results of analysis for specific IgE to latex using RAST method are shown in table 1. Scratch test was performed on the patient using his own rubber gloves, and the result was positive.[4,5]

Even though latex anaphylaxis generally is observed among certain risk groups (medical personnel, rubber industry workers), our case

Table 1. Immunoglobulin E Concentrations

Immunoglobulin E	Normal	Patient
Total	14–240 μg/lt	310 μg/lt
Latex-specific (RAST method)	<0.3 PRU/ml	2.8 PRU/ml (class 2)