Paroxysmal, Glycerol-Trinitrate Induced Hypertension

To the Editor:

Nitrates are used intravenously for various indications. When given intravenously, they rapidly reduce BP, and since they have a short half-life they are excellent agents for the urgent treatment of excessive hypertension especially in patients with ischemic heart disease and congestive heart failure.1 We report a case in which intravenous (IV) glycerol-trinitrate ([GTN] Nitroge
cine, Sanol Schwartz, Monheim, Germany) paradoxically caused BP elevation.

A 72-year-old woman with ischemic heart disease, hypertension, and recurrent pulmonary edema treated with enalapril, furosemide, and a low sodium diet, had been admitted for pulmonary edema secondary to myocardial ischemia which necessitated prompt intubation and mechanical ventilation. On the day of admission after a high salt meal and excessive effort, she developed pain and dyspnea. On admission BP was 210/120 mm Hg. Neurologic examination was normal and eye examination revealed slight narrowing of the retinal arteries. She was admitted to the ICU and IV administration of GTN was started at doses of 0.05 to 0.1 mg/min. Blood pressure was measured every 3 min by an automatic DINAMAP sphygmomanometer (Critiolin, EL), and intra-arterially, and its values were reduced to 170/100 mm Hg and congestive heart failure improved. Twenty hours after admission, BP suddenly rose to values of 220/140 mm Hg, the IV GTN dose was gradually increased up to 0.4 mg min, but BP continued to rise up to 300/220 mm Hg. Actually, BP rose with every increment in the dose of the GTN, and indeed when reduced, BP went down to 240/170 mm Hg. The process was repeated and every time GTN dose was reduced, BP came down only to increase again when GTN dose was increased. As we could not explain this effect, we thought the medication might have been replaced by mistake, so a new GTN ampulla was started; nevertheless, as GTN was given BP rose again. We discontinued GTN. Nifedipin was given enterally and BP was controlled at level of 170/100 mm Hg. At that time, repeated neurologic and eye examination were unchanged. Five hours later, left hemiparesis had occurred. Over the next month, the patient gradually improved her strength, which returned to near normal, and BP was well controlled and remained so, over 1 year of follow-up. Urinary catecholamines were found to be normal. Two weeks after the stroke, IV GTN was given and this time, it caused reduction of BP.

This case shows a paradoxical effect of GTN on BP in a patient with excessive hypertension and pulmonary edema. This effect was transient, occurred 20 hours after beginning of treatment, and was not reproducible 2 weeks later. We suggest that the special circumstances that caused this paradoxical effect were the first sign of the cerebrovascular accident that followed. In the setting of stroke, cerebral autoregulation may be impaired or lost and this may induce hypertension.2 Nitrites increase the cerebral blood flow and may further increase the intracranial pressure,3 and when cerebral flow autoregulation was lost, GTN might have had aggravated the stroke induced hypertension. Alternative explanation may be that after 20 h of treatment, nitrate tolerance developed peripherally, while the increment in the cerebral blood flow was maintained and aggravated the stroke induced hypertension.

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Dosing Efficiency and Particle-Size Characteristics of Pressurized Metered-Dose Inhaler Aerosols in Narrow Catheters

To the Editor:

I read with interest in the March 1993 issue of CHEST the article by Taylor et al.1 in which they discussed the dosing efficiency and particle-size characteristics of aerosols delivered by pressurized metered-dose inhalers (MDIs) through narrow catheters. We all recognize the problem of administration of inhaled bronchodilators in intubated patients.

The results presented by Taylor et al are very impressive, increasing the delivery by almost 50-fold when compared with endotracheal tube delivery. However, both the authors and Anthony J. Hickey, in an accompanying editorial,2 failed to comment on an important aspect of this technique. I tried the system described by Taylor et al, placing the tip of the 19-standard wire gauge catheter 2 cm away from the index finger of 6 pulmonary fellows and 2 pulmonary attending physicians. They all complained of a freezing, painful sensation that lasted for around 15 min. This could be explained by three properties that were not mentioned in the study: (1) The velocity of the particles close to the extended nozzle may produce barotrauma. (2) The expansion of the compressed gas at the tip of the catheter absorbs a great amount of heat. (3) The vaporization of the freon and oleic acid also absorbs heat, contributing to the freezing of the tip of the catheter and the finger skin. If used in humans, such a device is likely to confer significant trauma to the nearby bronchial mucosa and may precipitate cold-induced bronchoconstriction.

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REFERENCES

2. Hickey AJ. Characteristics influencing the effective administration of drugs as inhalation aerosols [editorial]. Chest 1993;103:657-58

To the Editor:

Dr. Zabner has expressed several concerns regarding potential injury to the bronchial mucosa following direct delivery to the lung of an MDI aerosol using the narrow catheter described in our article. We shared his concerns and have begun to investigate the safety of this technique in intubated, ventilated rabbits following administration of up to 20 doses of salbutamol by MDI, matching placebo by MDI, norepinephrine placebo by MDI, salbutamol solution, or control treatment (mechanical ventilation only).2 In our studies, the tip of the catheter lies within the lumen of the

REFERENCES