into a branch while the tip remains in the main stream (figs. 2 and 3, *D*).

While such malfunctioning of the balloon may occur from the time of the initial placement of the catheter, we believe that aberrant function may be initiated following the introduction of a high positive end-expiratory pressure, high-tidal-volume ventilation, positional change of the patient, coughing, or even movement of the catheter tip with contraction of the heart. Distortion of surrounding lung tissue due to existing disease might also be a causative factor.

Since the initiation of this study, Lozman *et al.* have raised the question of the accuracy in PWP measurements in patients being ventilated with high positive end-expiratory pressure. They reported a single dog experiment in which they demonstrated uneven inflation of the balloon causing impinging of the catheter tip on the wall of the pulmonary artery. This report confirms that such phenomena occur in man, necessitating stringent interpretation of values obtained.

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Anaphylactic Reaction to Cephalothin during Anesthesia

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Allergic reactions to cephalothin (Keflin) are reported to occur in 3–5 per cent of patients.[1,2] There are no data concerning frequency of anaphylactic reactions to cephalothin, but its widespread use since introduction in 1964 has been associated with few reports of anaphylaxis during anesthesia and operation.[3] The following, representing the third known anaphylactic reaction to cephalothin during anesthesia, is presented because: 1) the patient survived; 2) the intra-arterial oxygen tension was monitored continuously.

Report of a Case

A 48-year-old man who had chronic back pain and sciatica was scheduled for lumbar laminectomy with fusion. Past history was noncontributory except for chronic osteoarthritis and penicillin allergy. Nine months previously he had had an appendectomy followed by several orthopedic procedures, all with general anesthesia and no complication. Twenty years previously the patient had had a reaction to an intramuscular injection of penicillin. He remembered that six hours after injection his tongue had "shed" and he had expectorated blood. A diffuse erythematous, urticarial rash had followed, accompanied by lower-extremity edema persisting for three days.

Physical examination and laboratory data disclosed no abnormality. After premedication with pentobarbital, 100 mg, morphine, 5 mg, and atropine, 0.4 mg, im, the patient arrived in the operating room, where the usual monitors were placed. With the patient's informed consent, a left radial arterial cannula was inserted. Through this an indwelling continuous P_{O_2} electrode‡ was threaded.

Anesthesia was induced with thiopental (200 mg) and tracheal intubation was facilitated by succinylcholine (100 mg). The patient was placed in the prone-flex position and anesthesia maintained with enflurane and N_2O/O_2. Pancuronium, 4 mg, was administered, and respiration was controlled with a mechanical ventilator. Two hours and 45 minutes after induction, 1 g cephalo-

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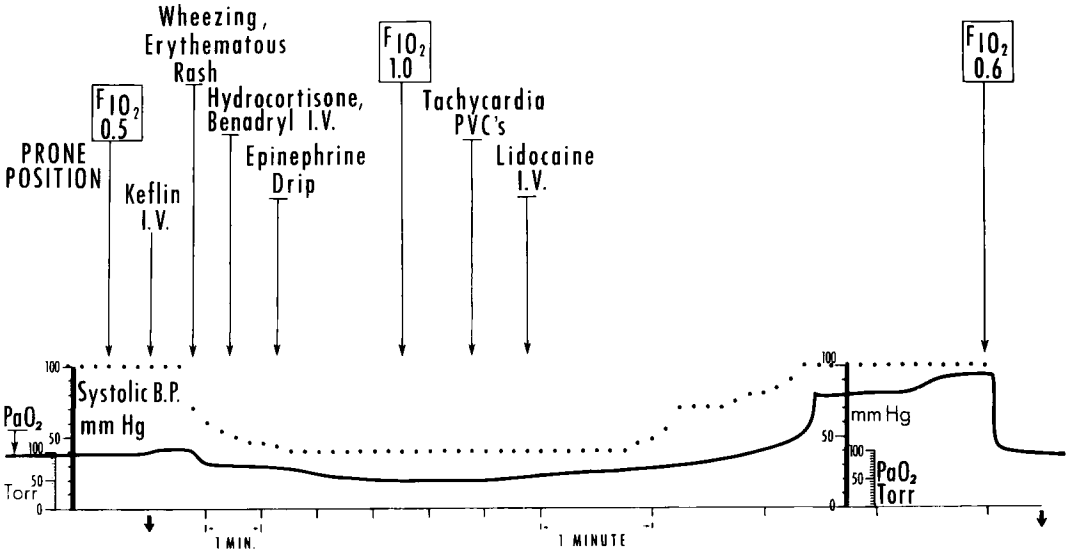

FIG. 1. Pa_{O_2} and blood pressure during anaphylactic reaction and anesthesia.

thin was administered by slow intravenous push. Shortly thereafter, a dramatic decrease in Pa_{O_2} from 100 to 50 torr (FI_{O_2} = 0.4) was recorded on the oxygen electrode (fig. 1). Systolic blood pressure fell from 100 to 50 torr, and wheezing was audible through the esophageal stethoscope. A diffuse erythematous, macular rash developed over the exposed arms, face and neck. The enflurane was stopped (FI_{O_2} = 1.0) and diphenhydramine, 50 mg, and hydrocortisone, 100 mg, were administered iv, with little improvement. An epinephrine drip (4 μg/ml) was added to the iv infusion, with subsequent improvement in blood pressure, alleviation of wheezing and rash, and return of Pa_{O_2} to normal. The next complication was junctional tachycardia with multifocal premature ventricular contractions; the epinephrine was discontinued and lidocaine, 100 mg, followed by sodium bicarbonate, 100 mEq, iv, were administered, with reversion to normal sinus rhythm. With gradual stabilization the same anesthesia was continued without further incident 30 minutes after the anaphylactic reaction. Total anesthesia time was 4½ hours. The trachea was extubated while the patient was fully awake in the recovery room. There was no ill effect postoperatively, and he was discharged a week later and followed in the Orthopedic Clinic.

DISCUSSION

This patient demonstrates classic anaphylaxis in man,[4,5] including cutaneous, respiratory, and cardiovascular signs (erythematous rash, bronchospasm, hypoxemia, and hypotension). The mechanism for hypoxemia includes bronchospasm, accompanied by laryngeal edema leading to pulmonary hyperinflation.[4,6] The hypotension is due to a decrease in cardiac output. During anaphylaxis anaerobic metabolism develops and the lactate/pyruvate ratio increases.[6]

When cephalothin was introduced in 1964, it was heralded as an answer to penicillin sensitivity, having a broad spectrum of activity and being effective in infectious states where penicillin was indicated but could not be given.[1,7] The cross-allergenicity problem between penicillin and cephalothin was thought to have been solved.[2,8] However, severe allergic and anaphylactic reactions to cephalothin in penicillin-sensitive patients have occurred.[3,8–10] These reports are relatively rare when one considers the widespread use of cephalothin intraoperatively.

Anaphylactic shock during anesthesia is rare, but has occurred with many drugs, including penicillin,[11–13] and is associated with high mortality.[3] The immune response during anesthesia is altered, and the anesthetic state may protect man from such reactions.[14] This patient may have recovered, in part, because anesthesia was deep, in contrast to lighter anesthesia in the cases of two other patients who died at the termination of

anesthesia.[3] More important, the diagnosis of anaphylactic shock was made rapidly on the basis of clinical signs and measurements from the arterial line, with immediate institution of intravenous therapy.

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A Simple Pressure-relief Valve to Prevent Increases in Endotracheal Tube Cuff Pressure and Volume in Intubated Patients

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Recent experiments *in vitro*[1,2] and in patients with endotracheal tubes in place[3,4] have demonstrated that oxygen and anesthetic gases diffuse into endotracheal-tube cuffs more rapidly than the nitrogen of the air-filled cuffs diffuses out. The result is that cuff volumes and pressures may markedly increase with time. An overinflated cuff can compress the wall of an endotracheal tube or cover its orifice and produce upper airway obstruction.[5–7] Overinflated cuffs also result in high cuff pressures.[8] The latter may be associated with an increased incidence of tracheal trauma.[5,8] In order to avoid these problems, we have suggested that cuffs be frequently deflated or be filled with a sample of the inspired mixture of gases rather than room air.[3,4] Another alternative is a pressure-relief valve which, when attached to the cuff catheter, will bleed off excess cuff gases but maintain cuff volume and pressure required for seal. We have designed a simple variable-pressure-relief valve, which has prevented increases in cuff volume and pressure and maintained cuff seal in 39 anesthetized patients during operations lasting as long as six hours.

The valve (fig. 1) is made of polycarbonate in the shape of a "T." One end of the cross portion of the "T" (fig. 2) is molded in the form of a standard male Luer fitting and is designed to be attached to the tip of an endotracheal-tube-cuff inflating catheter. This end of the "T" communicates with the other end through a hollow *body*. The second end of the cross of the "T" contains a spring-loaded check valve, which remains closed

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