ardous. In our view, awareness of this phenomenon, together with advanced planning, is critical in successfully securing the airway in these patients.

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# Successful Use of Terlipressin in Post–Cardiac Arrest Resuscitation after an Epinephrine-resistant Anaphylactic Shock to Suxamethonium

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EPINEPHRINE is considered in most guidelines as the first-line treatment of anaphylactic shock occurring during anesthesia. In some cases, epinephrine is not effective, and alternative therapies have been proposed, such as norepinephrine or metaraminol[1] or, more recently, arginine vasopressin.[2–4] To our knowledge, we are the first to report the successful treatment of anaphylactic shock with terlipressin, a structural analog of arginine vasopressin.

## Case Report

A 47-yr-old male patient was scheduled to undergo elective inguinal hernia repair. This patient was a current smoker (30 pack-years) with a history of gastroesophageal reflux disease and of four uneventful general anesthetics. One hour before surgery, hydroxyzine (1.5 mg/kg) and effervescent cimetidine (400 mg) were administered orally for premedication.

General anesthesia was induced using a target-controlled infusion with propofol (Diprivan®; AstraZeneca, Rueil-Malmaison, France) and remifentanil (Ultiva®; GlaxoSmithKline, Marly-le-Roi, France), and 1 mg/kg succinylcholine (Celocurine®; Pfizer, Paris, France). After intubation with an 8-mm-ID endotracheal tube (Mallinckrodt Medical, Athlone, Ireland), the patient was ventilated using an anesthesia ventilator (Felix®; Taema, Antony, France).

Four minutes after succinylcholine injection, a severe bronchospasm was observed in the absence of any skin manifestation. Cardiac arrest followed 30 s later. Chest compressions, pure oxygen (fraction of inspired oxygen = 1) ventilation, discontinuation of sedation agents, and fluid resuscitation (1 l lactated Ringer's solution) were immediately started. A bolus of 1 mg intravenous epinephrine was administered. Recurrent ventricular fibrillations were treated by four electric shocks and amiodarone administration (300 mg over 15 min). Because of hemodynamic instability, continuous epinephrine infusion was rapidly started at increasing doses (1–15 mg/h). Although epinephrine was rapidly efficient on bronchospasm, it did not restore blood pressure (systolic blood pressure 60 mmHg). After 45 min of resuscitation, an echocardiogram showed a hypokinetic left ventricle. Then, a 2-mg bolus of terlipressin (Glypressine®; Ferring SAS, Gentilly, France) was administered, allowing for hemodynamic parameters stabilization (heart rate 80 beats/min, systolic blood pressure 90 mmHg) and a rapid epinephrine weaning (15 mg/h down to 0 mg/h in 30 min).

Blood samples taken less than an hour after the onset of symptoms showed elevated serum tryptase ($> 200$ μg/l, normal when $< 13.5$ μg/l) and plasma histamine ($> 100$ nM, normal when $< 10$ nM). Anaphylaxis to succinylcholine was confirmed using a specific immunoglobulin (Ig) E radioimmunoassay with an inhibition step[5] (quaternary ammonium ion IgE 19.93% [positivity $> 2.0$] with a 54.49% inhibition by succinylcholine).

## Discussion

Anaphylactic reactions occurring during anesthesia remain a potentially life-threatening event, with an estimated incidence ranging between 1 in 10,000 and 1 in 20,000 anesthesias.[6] Neuromuscular blocking agents are responsible for more than half of those events, and suxamethonium has been implicated in a majority of cases.[7] However, diagnosis of anaphylaxis could be difficult in the anesthesia setting because clinical signs are often misleading or nonspecific.[8]

Successful treatment of epinephrine- and/or norepinephrine-resistant anaphylaxis using arginine vasopressin has been recently reported.[2,3] However, unlike its long-acting analog terlipressin, arginine vasopressin is not available in France.

Terlipressin has been successfully used as a second-line vasopressor in various hypotensive situations where sym-

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pathetic agonists were ineffective (catecholamine-resistant septic shock,[9] hypotension during anesthesia in patients chronically treated with angiotensin-converting enzyme inhibitors[10]). However, in a recent study, Dewachter *et al.*[11] compared epinephrine, arginine vasopressin, and terlipressin in an ovalbumin-sensitized anesthetized anaphylactic shock rat model. Unexpectedly, terlipressin did not change mean arterial blood pressure, regardless of the dose used, whereas epinephrine and AVP increased mean arterial pressure in a dose-dependent fashion.[11] Interspecies variations could explain the differences observed between these experimental results and our clinical case. One should also note that in our case, terlipressin was administered after epinephrine administration, whereas it was administered as a single drug in the previously reported experimental study. Therefore, one could not exclude a beneficial effect of the combination of both drugs.

Our case report underlines the interest of immediate tryptase and specific IgE measurements in case of unexpected cardiac arrest after anesthesia induction. It suggests that adding terlipressin to standard therapy should be considered when epinephrine does not restore vascular tone.

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