Management of intraoperative pulmonary oedema in a child following systemic absorption of phenylephrine eyedrops

Editor—We read with interest the case report on intraoperative pulmonary oedema in a child following topical phenylephrine eyedrops. The authors must be congratulated on achieving a successful outcome in the child. This case report highlights a well-documented, but not a well-recognized, adverse event after topical phenylephrine.

However, we feel an opportunity was missed to highlight the correct management of the problem. This was discussed in detail by the New York State Phenylephrine Advisory Committee in its report, ‘New York State Guidelines on the Topical Use of Phenylephrine in the Operating Room’.

An important observation by this group was the role of β-blockers in the causation of pulmonary oedema and cardiac arrest after phenylephrine-induced hypertension (PIH). They looked at nine cases of PIH; five of the eight patients who developed pulmonary oedema had received β-blockers, either labetalol or esmolol. Of these five, the three patients who had fatal cardiac arrests had received labetalol. The committee commented on the fact that only labetalol was associated with death. Because of the shorter duration of β-blocking activity of esmolol, pulmonary oedema may not have progressed to cardiac arrest and death after this drug. Another review of 12 cases of cardiopulmonary compromise after topical phenylephrine, showed three cardiac arrests, all in patients who had received β-blockers as the first line of treatment for PIH.

The recommendation was to avoid the use of β-blockers and Ca-channel blockers in PIH.

When PIH occurs, a wait-and-watch policy (for 10–15 min) is recommended for mild to moderate hypertension, while for severe hypertension they recommend the use of vasodilators and α-blockers, which are safer than a β-blocker. Where a β-blocker has been used, the report stressed the theoretical benefits of glucagon in high doses (5–10 mg), as probably the safest therapeutic option to reverse β-blocker-induced cardiac depression.

The report recommends a 0.25% solution of phenylephrine as the one of choice, and doses not exceeding 20 μg kg⁻¹ in children. The role of alternative vasoconstrictors like oxymetazoline, as safer alternatives, is also mentioned.

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Editor—We thank Drs Krovvidi and Kulkarni for their comments. We described the rationale behind the use of labetalol in our patient with PIH in our original case report. Following phenylephrine administration, tachycardia and multifocal ventricular ectopics were observed, suggesting a β-agonist effect as well as α-mediated hypertension. The ectopics appeared to represent the most immediate threat to the patient’s life, and we were reluctant to use an α-blocker because of the risk of exacerbating the tachycardia.

The New York State Guidelines were drawn up following several instances of PIH during ear, nose and throat (ENT) surgery. In the index case, involving a 4-yr-old boy, phenylephrine absorption from the site of adenoidectomy produced a degree of hypertension and tachycardia similar to that seen in our patient. Labetalol 0.12 mg kg⁻¹ was administered, after which pulmonary oedema, asystole and death ensued. In our own patient, labetalol 0.66 mg kg⁻¹ was given. It appears that we were most fortunate not to encounter more serious complications. Given our experience, the guidelines’ recommendation that β-blockers
should not be used for treatment of PIH would seem to apply equally in ENT and ophthalmic theatres.

However, those recommendations relating to phenylephrine concentration and dose appear to be specifically relevant to ENT surgery. The weakest commercially available ocular phenylephrine preparation is 2.5%. It is doubtful whether a 10-fold reduction in phenylephrine concentration would result in clinically useful pupillary dilatation. Had our surgeons used 2.5% rather than 10% phenylephrine, in keeping with the British National Formulary (BNF) recommendation we cited in our case report, the dose administered to our patient would still have been more than five times greater than the initial dose of 20 μg kg⁻¹ suggested in the New York State Guidelines.

Presumably ocular doses of this magnitude are widely deemed acceptable because phenylephrine absorption from the intact conjunctiva and nasolacrimal duct is much less than would be expected from a bleeding adenoidectomy site. In our view, the most significant factor leading to excessive phenylephrine absorption in our patient was the short interval between application of the eyedrops and the conjunctival incision. Had the eyedrops been administered as prescribed, preoperatively on the ward, the complication may not have occurred. Other general preventative strategies specifically applicable to ophthalmic surgery were discussed in our article.

As regards a possible role for oxymetazoline, ocular phenylephrine is used to effect mydriasis rather than vasoconstriction. It is the only sympathomimetic listed for this purpose in the BNF and we are unaware of an appropriate alternative.

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