Dear Editor

**William’s syndrome: was intubation rather than anaesthetic drug choice a cause of cardiac arrest?**

I was interested to read the recent case report describing sudden death of a child with William’s syndrome during anaesthesia¹ – but surprised there appeared to be no anaesthetist amongst the authors.

The authors describe cardiac arrest (with unsuccessful resuscitation) immediately after tracheal intubation. Anaesthesia was with sevoflurane 4% and intubation was preceded by intravenous alfentanil (75 µg/kg) and propofol (0.75 mg/kg). At post mortem examination the child had severe biventricular hypertrophy and near occlusion of the right coronary ostia. The authors infer that vasodilation due sevoflurane and propofol led to a fall in afterload and ‘worsen the effects on myocardial blood supply’ leading to acute myocardial infarction. The authors state that sevoflurane and propofol were not the optimal choice of agents, but offer no alternative agents.

In this case cardiac arrest occurred not immediately after propofol administration but after intubation. It is well recognised that laryngoscopy and tracheal intubation induces physiological “stress”. This is most commonly observed as hypertension (and tachycardia) with an increase in rate pressure product which is particularly undesirable in the presence of ventricular hypertrophy. There are associated rises in catecholamine levels. Such adverse haemodynamic and hormonal effects may lead to arrhythmias. It would seem at least feasible that haemodynamic and catecholamine responses to laryngoscopy and intubation led to poor cardiac perfusion and arrhythmia. No evidence of acute myocardial ischaemia was found at post mortem.

This child was undergoing dental surgery and for me this raises several additional/different questions.

- Was intubation – with its associated stress response necessary?
- Might cardiac arrest have been avoided if a suitable supraglottic airway device had been used instead of intubation?
- If intubation was chosen, might the stress response to intubation have been mitigated by administration of a larger dose of opioid or other agents (e.g. lidocaine, magnesium, clonidine etc)?
- As muscle relaxation facilitates rapid intubation might this also have been beneficial in reducing haemodynamic changes?

I agree that drugs associated with less vasodilatation (e.g. ketamine) may have been a wise choice for induction but I think anaesthetic technique and co-agents should also be considered in the causation of cardiac arrest and in considering alternative management of this challenging case.

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**Reference**


**Conflicts of interest**

None