had been taking MPH daily for 4 yr because of ADHD. Anaesthesia was induced by mask with sevoflurane. Arterial pressure and heart rate remained stable and oxygen saturation was 100%. After i.v. access was established, propofol 2 mg kg$^{-1}$ and alfentanil 10 μg kg$^{-1}$ were administered. Immediately after, the child developed a severe bradycardia followed by asystole. I.V. atropine 0.5 mg was given twice and external chest massage was performed. Normal cardiac rhythm with correct haemodynamic measures was restored 30 s after the start of the cardiopulmonary resuscitication. The planned operation was continued. Operation and emergence were uneventful. The patient was taken to the post-anaesthesia care unit where his heart rate remained stable at 90 beats min$^{-1}$. He was discharged home on the same day.

Amphetamines are indirect sympathetic amines with powerful central nervous system stimulation activity and peripheral α and β actions. Chronic administration can result in depletion of norepinephrine and dopamine storage. This decreased reserve of endogenous catecholamine can contribute to a blunted sympathetic response which can lead to bradycardia and refractory hypotension during anaesthesia. In our case, the patient did not take his medication on the morning of surgery, but it has been shown that intraneuronal catecholamine levels may not return to normal for days to weeks after cessation of amphetamine use.$^2$

Perioperative cardiac arrest in children has multiple causations.$^3$ Propofol has been associated with bradycardia and asystole$^4$ and the decrease in heart rate is more pronounced when propofol is combined with alfentanil.$^5$

We found in the patient’s medical files several previous uneventful general anaesthetics for the same procedure, before the patient was on amphetamine therapy. However, a severe bradycardia responding to atropine was noted during induction of a general anaesthesia several months earlier when the child was on amphetamine treatment. We believe that a blunted sympathetic response due to a chronic amphetamine exposition associated to the cardiac effects of propofol and alfentanil may have transformed a trivial bradycardia in a life-threatening asystole.

Therefore, the management of children on chronic amphetamine therapy should include avoidance or careful titration of cardiac depressor anaesthetic drugs. Direct acting vasoressors such as epinephrine or phenylephrine are preferable because of possible cross-tolerance to other indirect vasoressors such as ephedrine.$^6$ Premedication or pre-treatment with atropine may also be useful.

In conclusion, we have observed a severe cardiovascular complication during induction of anaesthesia, possibly in relation to chronic amphetamine treatment. In view of the increasing number of children on such treatment, further studies on the anaesthetic implications of this are required to determine if a specialized anaesthetic approach is appropriate in this group.

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What can you do in 12 weeks as a house officer in anaesthesia at a District General Hospital?

Editor—A house officer in anaesthetics and intensive care? It must be a holiday! Do you feel like a medical student again? Are you allowed to do anything? These are some of the comments I repeatedly heard throughout my 3 month placement from doctors in other specialities outside anaesthesia and intensive care (ITU). However, contrary to common belief, doing house jobs in anaesthetics and ITU is not just being an observer. It is a unique opportunity for junior doctors, who are less exposed to practical clinical skills, especially with the pressures of the European Working Time Directive, to acquire competences in performing essential and advanced clinical skills and to build their confidence in managing critically ill patients. Here, I explain how much I was able to do as a house officer in just 12 weeks.

The first thing someone learns at medical school is A B C. Although this is a simple principle known to every junior doctor, it is often not followed properly in practice. My hands-on experience in the anaesthetic room under direct supervision, mostly by a consultant, taught me how to approach a patient who suddenly stops breathing, how to use simple manoeuvres to maintain the airways, and more interestingly how to obtain definitive airways. I was able to perform 35 intubations and 42 LMAs during my time in anaesthetics, which enhanced my confidence in
dealing with the unconscious patient and will serve me a great deal in the emergency settings.

Breathing is the second most important aspect after establishing an airway. As soon as I became familiar with the anaesthetic machines, I was able to recognize when a patient needs mechanical ventilation and when they can breathe spontaneously. I also learnt when and how to change the volumes, the rates, and the pressures in relation to different situations.

My competency in managing circulation greatly improved throughout my placement, from using inotropes to fluid management, placing arterial lines and central lines. One of the most basic procedures is obtaining i.v. access, and cannulating nearly every patient who comes for surgery (83 cannulations performed) gave me great confidence. The effect was soon apparent when I was often called to cannulate patients that other junior doctors failed, which was satisfying.

**From anaesthetics to ITU**

Many people look horrified when entering the intensive care unit, and I certainly did when I first started. The patients are in critical states, and complicated machines are beeping all the time. On the ITU side, my days started with a consultant ward round spending considerable time with each patient, discussing the medical condition, the possible treatments, and at times the prognosis and hence ethical considerations. The ward rounds were certainly a valuable teaching opportunity of physiology, pathology, pain management, data interpretation, and communication skills. After finishing the ward round, I used to join the registrar and get on with the jobs from the round. In ITU, I carried out various procedures after first observing a number of them, and having a proactive approach I was allowed to perform procedures without supervision, such as arterial lines and chest drains.

All in all, my anaesthetic experience as house officer in a District General Hospital (DGH) was a corner stone in my training. With all the opportunities that it presents to acquire core competences and advanced skills, I would recommend it to everyone and I hope more DGHs would be able to offer FY1 posts.

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**Orthotopic liver transplantation without bypass in a patient with mitral valve prolapse**

Editor—Orthotopic liver transplantations (OLT) without bypass are associated with the potential for significant blood loss, sharply decreasing intravascular volume during anhepatic stage, aggressive overloaded volume on reperfusion, and developed reperfusion syndrome around the time of reperfusion. A feature of mitral valve prolapse (MVP) is mitral regurgitation (MR) which may be exacerbated by increased systemic vascular resistance (SVR), bradycardia or excessive tachycardia, and volume overload. We present a case of a successful OLT without bypass in a patient with anatomical MVP associated with moderate MR with a history of end-stage liver disease. We present the anaesthetic problems and management for this complicated case.

The patient was a 53-yr-old male with end-stage liver disease. He had hypertension with severe ascites, oesophageal varices, and moderate coagulopathy (international normalized ratio, INR > 2.0). He had a history of MVP for 5 yr. He had preoperative diuretic and digoxin therapy. Preoperative echocardiography showed that there was posterior systolic motion of the continuously juxtaposed MV leaflets behind the line that connects the valve’s closure and opening points (the C–D line) and billowing of thickened mitral leaflets superior into the left atrium (LA) during systole (Fig. 1). In addition to left ventricular (LV) dilation, moderate MR and a small amount of pericardial fluid were noted.

Anaesthesia was induced with fentanyl 6 mg kg\(^{-1}\) and etomidate 0.3 mg kg\(^{-1}\) and maintained with fentanyl 3 μg kg\(^{-1}\) h\(^{-1}\) and midazolam 0.04 mg kg\(^{-1}\) h\(^{-1}\). A pulmonary artery (PA) catheter was used to calculate haemodynamic variables, including cardiac output (CO) and systemic vascular resistant index (SVRI). During liver resection, a