between atrial flutter with 2 to 1 block, which is unresponsive to adenosine, and ‘true’ SVT (atrioventricular nodal re-entrant tachycardia and atrioventricular re-entrant tachycardia) which is usually terminated by adenosine administration.

N. J. Morgan-Hughes
Sheffield, UK

Editor—I read with interest the case report by Kannan and Sherwood who stated that administration of propofol was associated with termination of a supraventricular tachycardia (SVT). They were surprised that this effect in adults had not been reported previously in the literature. I would like to add to the discussion by reporting a case of propofol reverting a case of ventricular tachycardia (VT) to sinus rhythm.

A 76-yr-old man was admitted with shortness of breath and palpitations. He was known to have ischaemic heart disease and chronic renal impairment with secondary anaemia. He had had a DDD pacemaker inserted some years previously for first-degree heart block and left bundle branch block. His medications included amiodarone, lisinopril, isosorbide mononitrate, frusemide, bisoprolol, aspirin, and warfarin. Three days later he was transferred to the coronary care unit after an episode of VT (pacemaker not capturing), which was successfully direct current (DC) cardioverted. The following day, he again went into VT (no pacemaker capture) with associated hypotension. Preparation was made for DC cardioversion. The patient had not eaten or drunk in the preceding 8 h, which avoided the need for a rapid sequence induction. After preoxygenation, anaesthesia was induced by slow titration of propofol and after approximately 150 mg, it was noticed that the rhythm had converted to sinus (pacemaker capturing) with improvement in the arterial pressure. He remained in paced sinus rhythm for over 24 h but subsequently continued to have runs of VT that were unresponsive to medical therapy. He was therefore referred for insertion of an automated implantable cardioversion device.

Kannan and Sherwood have highlighted several proposed mechanisms, direct and indirect, for the action of propofol in terminating SVT. Pires and colleagues examined the effects of propofol in pig hearts and concluded that propofol caused dose-related depression of the sinus node and the His–Purkinje system, but had no effect on the atrioventricular node. The exact mechanism of cardioversion in my case is unclear. Other antiarrhythmic functions of propofol are noted in a report by Miro and colleagues, who described three patients with fast atrial fibrillation (AF) needing DC cardioversion. All were given propofol for sedation pre-shock and all converted to sinus rhythm after its administration.

R. M. Heames
Basingstoke, UK

Editor—Drs Kannan and Sherwood describe an interesting case of the possible conversion of a supraventricular tachycardia (SVT) to sinus rhythm by propofol. They state that the 12-lead ECG on presentation (unfortunately not published in their article) was ‘suggestive’ of an SVT. However, the published rhythm strip (lead 11) would appear to support the alternative diagnosis of atrial flutter with 2:1 block. The failure of adenosine to convert this arrhythmia to sinus rhythm (a rare occurrence with SVT), and its apparent transient conversion to atrial flutter with 4:1 block might also support this alternative, especially when considering the predictable effect of adenosine upon AV nodal conduction. Atrial flutter may convert to sinus rhythm spontaneously, or following manoeuvres that create a Valsalva effect. Perhaps good preoxygenation with a tight fitting mask followed by cricoid pressure...
created these conditions? Propofol may have had some effect in converting atrial flutter in this patient to sinus rhythm but I do not think that we can be certain from the information provided by Kannan and Sherwood.

H. Hack
Manchester, UK

Editor—We would like to thank Drs Morgan-Hughes, Heames and Hack for taking an interest in our case report. We agree, in retrospect, that the original rhythm in our patient was atrial flutter with an atrioventricular conduction block. A definitive diagnosis before the administration of adenosine was hindered by the varying ventricular rates. Hence, we had used the term ‘rhythm suggestive of supra ventricular tachycardia (SVT)’ in our case report description. Although adenosine unmasks atrial flutter by prolonging atrioventricular nodal conduction, it can rarely induce atrial flutter in patients with paroxysmal SVT and vice versa. Since it is less likely that a given patient would have two different types of supraventricular arrhythmias at different points in time, a more appropriate title would have been ‘Termination of atrial flutter by propofol’. Interestingly, atrial flutter has been classified as a form of SVT. In spite of what we thought was an extensive search of the literature, we failed to identify the reported cases of termination of atrial fibrillation with propofol. We would like to thank Dr Heames for bringing them to our attention. His report of termination of ventricular tachycardia with propofol is interesting. It is not clear which anaesthetic the patient received for the first cardioversion. If the patient had received propofol for the same, it obviously did not terminate the rhythm at that instance. As for its effect on atrioventricular node conduction, Alphin and colleagues have demonstrated that propofol slows atrioventricular conduction time in a concentration-dependent manner in guinea-pigs.

S. Kannan
N. Sherwood
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7 Alphin RS, Martens JR, Dennis DM. Frequency dependent effects of propofol on atrioventricular nodal conduction in guinea pig isolated heart. Mechanism and potential antidysrhythmic properties. Anesthesiology 1995; 83: 382–94