treatment. Similarly, it is unclear whether there is a hazard that accompanies discontinuation of prasugrel at any time point. Necessity therefore dictates it be treated as akin to clopidogrel until further data emerge.

**Conclusion**

Although there is a considerable amount of evidence supporting an increased risk of adverse cardiac events with clopidogrel discontinuation, the quality of this evidence is often questionable. It remains unclear whether there is a time threshold for discontinuation, after which event rates are negligible. Current guidelines for the duration of clopidogrel are understandably cautious, given the present uncertainty and catastrophic nature of stent thrombosis. Nevertheless, quantification of the actual risk of ‘premature’ clopidogrel discontinuation is elusive, and both financial and bleeding costs are often ignored. Ongoing clinical trials will provide insight, however, and newer antiplatelet agents and second-generation DESs may attenuate fears of stent thrombosis.

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**References**

The list of references is available in the online version of this paper.

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**CARDIOVASCULAR FLASHLIGHT**

**Sudden cardiac death in Ebstein’s malformation due to a cardiac haemangioma**

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A previously well 22-year-old man suddenly collapsed during a football game. Prolonged resuscitation of 50 min was unsuccessful. Pathological examination demonstrated Ebstein’s malformation with apical displacement by 30 mm of the septal and posterior tricuspid valve leaflets (Panel A), and rotation of the septal leaflet into the right ventricular outflow tract (RVOT). The anterior leaflet was partially fused with the septal leaflet, and tethered to the inter-ventricular septum. The right atrium incorporating the atrialized right ventricle was dilated. There was a 4 mm foramen ovale. The functional right ventricle was not significantly dilated and the RVOT was normal in size. Below the pulmonary valve and near the attachment of the septal leaflet to the inter-ventricular septum was the unexpected finding of a firm, lobulated dark haemorrhagic mass measuring 10 × 20 × 30 mm (Panels A–C). The location and thickness of the moderator band was unusual. It was below the pulmonary valve in the RVOT and immediately anterior to the mass (Panels B and C). It was hypertrophied to 20 mm in diameter. Both the mass and the moderator band were partially obstructing the RVOT.

Haematoxylin and eosin (magnification ×200, Panel D) and diaminobenzidine immunostained (dark brown staining of the endothelium, magnification ×350, Panel E) sections of the mass showed dilated endothelial lined channels filled with red blood cells diagnostic of a cystic haemangioma.

Sudden cardiac death may result from increased obstruction in the RVOT and the development of malignant arrhythmia during vigorous exercise. A medical examination in athletes with appropriate follow-up investigation, for example, by echocardiography may be helpful in detecting silent but potentially lethal abnormalities and preventing possible adverse outcomes.