Supraventricular Tachycardia in a Child Undergoing Strabismus Surgery

To the Editors:

Supraventricular tachycardia (SVT) encompasses all types of tachycardia that start from above the bifurcation of the bundle of His with a heart rate ranging from 150 to 250 beats/min in children.1,2 Paroxysmal SVT occurs suddenly and can end rapidly.1 We describe a healthy 3-year-old boy who developed primary SVT during intraoperative extraocular muscle manipulation.

Our patient underwent surgery to correct inferior oblique overaction. Under laryngeal mask airway anesthesia (propofol, fentanyl, ketorolac, isoflurane, and sevoflurane), the inferior oblique muscle was isolated using muscle hooks through a fornix incision. A 6-0 polyglactin suture was then placed through the muscle near its insertion. The patient then went into SVT. Contact with the muscle was stopped, but SVT continued at 227 beats/min. Carotid massage, ice placed on the forehead, and adenosine 1.5 mg 3 doses had no effect. A 12-lead electrocardiogram demonstrated features of reentry SVT (Figure). An additional 3 mg of adenosine restored sinus rhythm at 142 beats/min. One more incidence of SVT occurred, which was treated with 3 mg of adenosine. The inferior oblique muscle was re-isolated, and there was an episode of bradycardia treated with glycopyrrolate. The cardiac rhythm stabilized and surgery continued without complication. Subsequent transthoracic echocardiography was normal and the patient started propranolol 10 mg orally three times daily. In retrospect, a family history was positive for Wolff–Parkinson–White (WPW) syndrome in a first cousin.

SVT is the most common form of arrhythmia in children and 90% is mediated through a reentry mechanism. This is facilitated by the presence of an accessory pathway in addition to the normal atrioventricular node and Purkinje system.3 Jacobson et al. detailed paroxysmal SVT during strabismus surgery in a 17-year-old with WPW syndrome.4 Paroxysmal reentrant SVT occurs in WPW syndrome using an accessory retrograde pathway that is manifest on electrocardiogram with a short PR interval and Delta waves.5 When these features are absent in reentrant SVT, presence of a concealed pathway is presumed.3 Our patient's electrocardiogram did not have the characteristics of WPW and thus the reentrant SVT was mediated by a concealed pathway. It is speculated that in our case and in the patient with WPW, hypotension induced by anesthesia may have increased sympathetic tone, favoring conditions for a reentry circuit via the preexisting accessory pathway. The SVT may have been initiated by an ectopic escape beat in the setting of bradycardia from vagal stimulation induced by manipulation of the muscle.

Acute treatment of SVT includes vagal maneuvers such as Valsalva maneuver and cold water immersion of the face. If these treatments fail, intravenous adenosine should be used; it works by inducing transient AV block that terminates SVT by interrupting the reentry pathway and allowing restoration of sinus rhythm.1 Final treatment options include elective cardioversion or procainamide. Chronic SVT treatment can involve the use of propranolol or other antiarrhythmic agents and catheter ablation.1

REFERENCES
Congenital Unilateral Brown Syndrome in Non-Twin Siblings

To the Editors:

Brown syndrome is mainly a sporadic condition affecting either one or both eyes. We report a case of two siblings with unilateral congenital Brown syndrome.

Brown syndrome is characterized by a restrictive deficient elevation of the eye in adduction. It is frequently a sporadic, unilateral condition. Scant reports have presented familial cases among non-twin siblings or monozygotic twins.

Two brothers presented to our medical center with an inability to elevate the right eye in adduction. The older, a 4-year-old, had a slight head turn to the left and chin up since the age of 6 months. Visual acuity was normal in both eyes. In the primary position, he had right hypotropia with vertical diplopia. An alternate prism cover test revealed right eye hypotropia of 9 prism diopters. Ocular movement examination revealed -3/4 of right eye elevation in adduction with normal elevation in abduction and no ptosis (Fig. A). No overaction of the superior oblique muscle was noted. Stereopsis was measured as normal using the Lang stereo test (400”). Fundus examination was normal. A forced-duction test, performed under general anesthesia, demonstrated restricted elevation of the right eye in adduction. A tenotomy of the superior oblique muscle was performed with preservation of the intermuscular septum. On his last visit, he had no abnormal head posture or hypotropia in primary position.

His brother, a 10-month-old boy, presented with similar restriction in adduction of his right eye (a deviation of -3/4, Fig. B). At primary gaze, both eyes were straight. For this child, the parents chose to continue with conservative regular follow-ups.

Clinical identification of Brown syndrome is based on eye movements and validated by forced-duction testing demonstrating restricted passive elevation in adduction. These may differentiate Brown syndrome from other adduction elevation entities such as inferior oblique muscle palsy or fibrosis. Acquired Brown syndrome is usually related to conditions as following trauma, inflammatory conditions, or orbital surgery. The cause of congenital cases is less clear, although the accepted view is of a tight superior oblique tendon. Other speculations include an anomalous tendon–trochlea telescoping mechanism or retention of embryonic tissue at the junction.

Although congenital cases can improve with conservative treatment, advanced cases require surgical intervention. Unless treatment is sought, little improvement is expected in such cases. Treating Brown syndrome is determined according to the severity of the condition. Mild and moderate cases, which manifest mainly in adduction and are straight in primary gaze, are observed. More severe cases, as in our older patient, when patients suffer from deviation in primary position, are offered surgical correction. Most surgeons advocate ipsilateral superior oblique tenotomy. However, this may result in superior oblique palsy and a hyper-deviation in adduction. To address this potential complication, authors have proposed to preserve the intermuscu-
lar septum or perform an inferior oblique recession. Other options include using nonabsorbable sutures and sewing inert spacers to the cut ends.²

Due to the rarity of such cases, further reports will be needed before a better understanding of the pathophysiological basis is reached.

REFERENCES


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