# Perventricular closure of muscular ventricular septal defect in an infant

Bir bebekte müsküler ventriküler septal defektin perventriküler yolla kapatılması

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#### Introduction

Percutaneous closure of muscular ventricular septal defects (VSD) is encouraging (1-7). However, in case of low patient weight or poor vascular access percutaneous device closure of muscular VSD is not possible. Hybrid therapy in VSD closure combines advantages of surgical and interventional techniques (8, 9). This report presents successful device closure of a muscular VSD using Amplatzer muscular VSD occluder device in a child of 2.5 months old age and weight of 5 kg.

#### Case report

A 10 days old male baby was admitted with dyspnea; on physical examination there was II/VI systolic murmur, and weak femoral pulses. Transthoracic echocardiography revealed dilated left ventricle with aortic coarctation and midmuscular VSD. Urgent balloon angioplasty was performed because baby was very sick for surgery. At the age of two months, he presented with clinical signs of heart failure. On echocardiographic examination there was 4-5 mm midmuscular VSD. He underwent perventricular closure of the muscular VSD with an Amplatzer device (AD). Patient was discharged in five days on 4 mg/kg/day aspirin for 6 months. On echocardiographic examination which was done the day after procedure there was a minimal shunt inside the device. Holter control on first month of procedure was normal. At last control after 12 months from the procedure, there was no sign of shunt at the ventricular septum.

#### **Surgical Technique**

Child was intubated via transnasal approach and a median sternotomy was done. The procedure was applied under continuous transesophageal echocardiography (TEE) guidance. Diameter of the defect was measured 4-5 mm. For right ventricular (RV) puncture a 5-0 polypropylene purse-string suture was placed on beating heart at carefully chosen location away from papillary muscles, and the septum. An 18-gauge needle was introduced into RV cavity and a 0.035-in. glide wire was passed into left ventricular (LV) cavity. An 8 Fr short introducer sheath with a dilator was fed over the wire into the LV cavity. The dilator was removed and the sheath tip positioned in the LV cavity. The 6 mm device was screwed to the cable and pulled inside a 7Fr loader. Device was manipulated inside the delivery sheath and LV disc was deployed in the mid-LV cavity. The cable and the sheath were withdrawn until the LV disc was against the septum. When position was confirmed, sheath was again retracted to expand the RV disc. The device was released by counterclockwise rotation of the cable using pin vise (Fig. 1). During the procedure, a portable X-ray machine was used to reveal the position of the wire. After release of the device TEE was repeated and minimal residual shunt was seen inside the device.

### **Discussion**

Transcatheter closure of muscular VSDs is a well accepted therapeutic modality. Percutaneous closure of VSDs in small babies (weight <5 kg) can be challenging because of increased risk of residual shunts, procedure related complications and relatively large delivery sheaths which may result in rhythm disturbance and hemodynamic compromise (3, 4, 6, 7). After the use of Amplatzer muscular VSD closure device, success rate increased significantly compared to previous devices. This device needs relatively large sheaths and this limits its use in small babies (3, 4). Perventricular closure method had been developed by Amin et al (8) to reach the desired success rates without complications following results of successful studies on animals.

Although surgery is the preferred method, cardiopulmonary bypass, and right ventriculotomy to assess VSD may cause prolonged procedure time and subsequent complications. Perventricular VSD closure technique does not require cardiopulmonary bypass or full sternotomy (8, 9). The time needed to cross the VSD and position the device is less than 30 minutes in majority of cases. Other advantages include avoidance of transsection of the RV muscle bundles and any ventricular incisions and immediate confirmation of adequate closure (9). Irrespective of weight, these infants can undergo perventricular VSD closure if they are not candidates for percutaneous approach. This

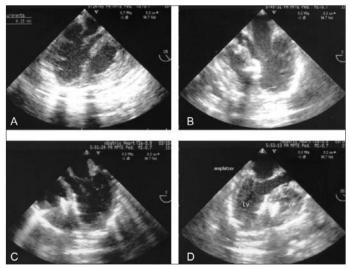


Figure 1. (A) Midmuscular ventricular septal defect is demonstrated by transesophageal echocardiography (B) The left ventricular disc deployed and pulled against the ventricular septum. (C) The device has both deployed but still attached to cable (D) The device is released from the cable

modality of treatment is also indicated for neonates with complex defects and large muscular VSDs, where a one-stage repair via sternotomy can be applied (10). Our case, first in Turkey, was closed successfully by perventricular closure using Amplatzer muscular VSD device.

#### Conclusion

We conclude that Amplatzer muscular VSD occluder seems to be a safe and effective device for closure of muscular VSDs. Further clinical trials with this device are underway. This hybrid technique involving both pediatric cardiologists and cardiothoracic surgeons can be utilized to close muscular VSDs even in small babies with ease.

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## Bilateral common peroneal nerve palsy following cardiac surgery

Kardiyak cerrahi sonrası bilateral komon peroneal sinir paralizi

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## Introduction

Although cardiac surgery improves the life expectancy in the patients with severe heart diseases, the post-operation period is associated with severe complications (1-4). Even though peripheral nervous system complications are less frequent and usually less severe than others, they have an importance due to a source of additional postoperative disability. In this report, we presented 3 cases with bilateral common peroneal nerve palsy (CPNP) following cardiac surgery.

#### Case 1

A 52-year-old man was referred to our clinic for bilateral foot droop with sensory loss on the lateral aspect of his legs and feet. The patient underwent a three-vessel coronary artery bypass graft surgery 3 weeks ago and his complaints started immediately after surgery. He had no concomitant disease except 11 years' history of diabetes. The

patient's examination revealed weakness of feet dorsiflexion/eversion (2-/5) and diminished sensation on the dorsum of the feet and anterolateral side of both calves. Nerve conduction studies showed prolonged latency and slow velocity around the fibula head as compared with distal segment for common peroneal nerve. Small compound muscle action potentials from the extensor digitorum brevis muscles were observed by both proximal and distal stimulation. Needle electromyography (EMG) of bilateral tibialis anterior and peroneus longus muscles revealed motor unit potentials of normal amplitude, duration, and phasicity; increased insertional activity, 2+ fibrillations, 2+ positive sharp waves and reduced recruitment. An isolated partial lesion of CPN bilaterally was diagnosed. The patient started in a physical therapy and rehabilitation program (PTRP) including active assistive ranges of motion exercises and electrical stimulation for 5 days per week for 1 month, and then home exercise program was prescribed. In addition to foot orthosis, correct positions of ankles were described. At the  $3^{rd}$  and  $6^{th}$  months, there was a significant improvement clinically. In addition, repeated needle EMG showed reinnervation of motor units via axonal regeneration.