Ventricular septal defects (VSDs) are the commonest congenital heart defects (CHDs) and account for 20–25% of CHDs [1]. Since 1978, when the repair of four VSDs in the then General Hospital, Colombo was described [2], there has been no documentation of the situation regarding VSD repairs in Sri Lanka. The following study represents our recent experience in surgical closure of VSDs.

A retrospective descriptive study of 128 consecutive patients who underwent VSD repair in the Cardiothoracic Unit 1 of the National Hospital of Sri Lanka from February 1997 to February 2004 was done. The inclusion criterion was the presence of a VSD as the primary lesion. Acquired VSDs were excluded.

Information was obtained from the bed head tickets, operation notes, perfusion records and mortality records. As clinical details of 11 patients could not be retrieved, the remaining 117 records were analysed. The patients’ characteristics are shown in Table 1.

All patients underwent median sternotomy and cardiopulmonary bypass at 32ºC. Analysis of the VSD sizes showed that 80% of the VSDs were large, 11% medium and 9% small. Only one patient had multiple VSDs. All VSDs were closed with 0.6 mm Gore-tex patches. The early outcome is summarised in Table 2.

The optimal management of VSDs requires careful consideration of its natural history. A proportion of VSDs (25–40%) are known to close spontaneously, the probability of closure showing an inverse relationship to the age at diagnosis [3]. In those children in whom the VSDs persist, a multitude of complications can occur, as was evident in the patients in our series. When the shunt reverses, repair is contraindicated.

The ideal management of VSDs would be to intervene at the time of detection in infancy. Due to the limited facilities and the large number of patients in the National Hospital of Sri Lanka, the policy adopted in our unit is to follow up neonates and infants without pulmonary hypertension (PHT), until the body weight is adequate. Medical therapy to control heart failure and infective complications is given when indicated during this time. Patients with severe PHT detected by two-dimensional echocardiography undergo right heart cardiac catheterization (RHCC). If they are considered operable, pulmonary artery banding is done as the initial procedure in small infants and early repair in older ones. All other patients are operated on as they present via the waiting list.

When simple VSDs without concomitant cardiac lesions are repaired during infancy before the onset of complications in specialised paediatric cardiac centres in the world, the mortality rate is less than 1% [4]. In the presence of multiple defects and pulmonary hypertension, however, the mortality rate is higher [5]. As only one patient in our series had multiple defects, PHT could have been important in influencing our mortality rates.

The development of PHT is related to the size and the age of the patients, both of which appear to have played a role in this series. Delay in obtaining treatment could be an important contributing factor for the high occurrence

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**Table 1. Patients’ characteristics**

| Characteristic                  | No. of patients |
|--------------------------------|-----------------|
| Age                            | 8.2 (years)     |
| Range                          | 5 months–37 years |
| Weight                         | 8.7 (kg)        |
| Range                          | 3–60 (kg)       |
| Sex                            | Male 70 (54.68%) |
|                                | Female 58 (45.32%) |
| Clinical features              |                 |
| Asymptomatic                   | 13 (11.1%)      |
| Dyspnoea                       | 69 (59%)        |
| Chest pain                     | 9 (7.7%)        |
| Palpitations                   | 5 (4.3%)        |
| Systolic murmur                | 117 (100%)      |
| Complications                  |                 |
| Recurrent respiratory tract infection | 58 (49.5%)   |
| Pulmonary hypertension         | 58 (49.5%)      |
| Heart failure                  | 42 (35.9%)      |
| Failure to thrive              | 25 (21.4%)      |
| Cyanotic episodes              | 5 (4.2%)        |
| Right bundle branch block      | 1 (0.9%)        |

**Table 2. Outcome of the Study**

| Outcome                                      | No. of patients |
|----------------------------------------------|-----------------|
| Morbidity                                    |                 |
| Respiratory tract infection                  | 8 (6.8%)        |
| Reopening for bleeding                       | 4 (3.4%)        |
| Residual defect requiring redo closure       | 2 (11.9%)       |
| Pericardial effusion requiring pericardiostomy | 2 (1.5%)  |
| Pleural effusion requiring thoracostomy     | 2 (1.7%)        |
| Cerebral infarction                          | 2 (1.7%)        |
| Infective endocarditis                       | 1 (0.9%)        |
| Complete heart block                         | 1 (0.9%)        |
| Acute renal failure requiring dialysis       | 1 (0.9%)        |
| Wound infection                              | 1 (0.9%)        |
| Sinus formation                              | 1 (0.9%)        |
| **Total**                                    | **25 (21.4%)**  |
| Mortality                                    |                 |
| Acute heart failure                          | 4 (3.1%)        |
| Primary haemorrhage                          | 2 (1.6%)        |
| Cardiac tamponade and arrest                 | 1 (0.9%)        |
| **Total**                                    | **7 (5.5%)**    |
To the Editors:

First report of mucosal tissue localisation of leishmaniasis in Sri Lanka

In Sri Lanka, cutaneous leishmaniasis is an established disease with the causative organism identified as *Leishmania donovani* zymodeme MON-37 [1]. Since the first locally acquired case was detected in 1992 [2], no visceral or mucosal involvement has been reported until now. Two patients with oral mucosal lesions were identified recently.

**Case 1**

A 42-year old soldier stationed in Omanthai, presented with a 10 × 20 mm lesion on the inner surface of the upper lip (Figure 1). The lesion was non-tender with an indurated edge and a moist centre. It first appeared as a self-healing papule 5 months back, which recurred twice within 6 weeks. Then a small ulcer appeared which enlarged in size, with swelling of the lip. The lesion was excised surgically. A month later it had reappeared and enlarged to the present size in 2 months. There was no history of previous skin lesions or loss of weight or overseas travel. Microscopic examination of Giemsa stained lesion smears showed *Leishmania* amastigotes.

He was treated with 14 doses of sodium stibogluconate (20 mg/kg body weight) IM every other day. The lesion was completely healed with 40 days treatment.

**Case 2**

A 18-year old schoolboy from Embilipitiya had a lesion on the inner surface of the lower lip. It was a pink plaque 6 × 8 mm, with no ulceration or discharge. The lesion had initially appeared as a recurring papule, which did not respond to antibiotics. Histological appearance of a biopsy specimen of the lesion was suggestive of leishmaniasis. He was treated with 10 doses of sodium stibogluconate. The lesion resolved after 4 months.

Mucosal involvement in cutaneous leishmaniasis is known to occur in about 5% of infections [3]. This is a different clinical entity from the classical mucocutaneous leishmaniasis or ‘espundia’ due to *L. braziliensis*, which is a dreaded sequela of cutaneous leishmaniasis. It starts as a skin lesion with secondary involvement of the nasal mucosa, leading to mutilating deformities of the face. These lesions are frequently resistant to treatment.

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