



Microvascular Decompression for Hemifacial Spasm: analysis of surgical failures and repeat surgery.

Mark G Bigder BA, HBK, MD; Anthony M. Kaufmann MD, BSc (Med), MSc  
Section of Neurosurgery, University of Manitoba



Introduction

Hemifacial spasm (HFS) is a condition of debilitating, involuntary contractions of facial muscles with an estimated prevalence of 10/100,000. The potentially curative microvascular decompression surgery (MVD) aims to alleviate compression upon the facial nerve root entry zone (fREZ) by mobilizing culprit vessels and maintaining them off the nerve with implant material. Surgery is undertaken in less than 10% of sufferers across North America, such that few centres have a concentrated volume of MVD experience. In cases where surgery fails to alleviate symptoms, patients and surgeons are faced with a potentially difficult management decision with regard to repeat surgery. Few studies have attempted to identify the source of failure in cases of MVD for HFS.

Object

The aim of this study is to describe clinical outcomes and neurovascular compression findings in patients undergoing repeat MVD procedures.

Methods

A database of over 700 MVDs performed by the senior author was reviewed to identify patients undergoing repeat surgery for HFS where the original surgery was performed elsewhere. Intraoperative findings were obtained from operative reports and diagrams. Outcomes were determined from hospital records and telephone questionnaires.

Results

Twelve HFS patients were identified and all had intra-operative evidence of neurovascular compression on the facial root exit zone documented at repeat surgery. In 8 of 12 cases, prior surgical implant material was discovered at the distal cisternal portion of the facial nerve. One of these patients had an intra-operative finding of a 5mm incidental PICA aneurysm in proximity to the facial root entry zone and in close proximity to the culprit neurovascular compression. In 3 of 12 patients, the previous surgeon implanted surgical material over the facial root exit zone, however, failed to identify a second, potentially more significant source of compression more proximally. In 1 patient, there was no evidence of any previously implanted surgical material. All patients reported clinical improvement in post-operative spasm status on last follow-up at 3 to 180 months. Ten patients reported complete resolution of spasms, of which 2 reported an occasional quiver. One patient reported mild spasm with an overall reduction of 75% while 1 patient reported moderate spasms with greater than 50% reduction post-op. Three patients experienced post-operative complications: 1 patient with permanent mild facial weakness (House-Brackmann (HB) 2), 1 patient with transient mild facial weakness (HB 1), and one patient with aseptic meningitis and subsequent complete resolution of symptoms.

Conclusions

Neurovascular compression of the fREZ was evident in all cases at repeat MVD surgery. Previous surgery most often erroneously targeted the distal cisternal portion of the facial nerve. In all cases, persisting culprit compression was observed more proximally at the facial root exit zone. The majority of failed MVD surgeries for HFS are a result of inadequate alleviation of neurovascular compression on the fREZ at prior surgery; such cases are amenable to repeat surgery with good results, provided the surgeon is proficient in performing this relatively rare operation.

Learning Objectives

- By the conclusion of this session, participants should be able to:
- 1) Describe the anatomical target for microvascular decompression for HFS.
  - 2) Identify common sources of treatment failure in MVD for HFS.
  - 3) Discuss treatment options for patients with failed MVD