Functional Deficits of Cranial Nerves in Patients with Jugular Foramen Lesions

Abstract—Lower cranial neuropathies are a leading presenting symptom in patients with tumors involving the jugular foramen. The purpose of this study is to assess acute and chronic functional deficits along with neurologic findings in 31 patients who underwent resection of a tumor involving the jugular foramen. Preoperative nerve dysfunction made intraoperative preservation unlikely, while postoperative lower cranial nerve dysfunction was found to be transient in many patients. Compensation of permanent dysfunction was usually excellent.

The anatomic topographic situation of the jugular foramen is characterized by a vascular and a neural compartment. Cranial nerves IX to XI and the inferior petrosal sinus run through the neural compartment and the sigmoid sinus runs through the vascular compartment. The internal carotid artery and facial nerve are usually found in close proximity.

Jugular foramen tumors involve a critical area of the skull base. Usually they are characterized by palsies of the cranial nerves IX, X, and XI. Depending on tumor extension, there are different approaches to the skull base and different surgical strategies aimed at preserving cranial nerves VII to XII.¹⁻⁵ The aim of this study is to present the acute and chronic functional deficits along with the neurologic findings in patients having undergone resection of a tumor involving the jugular foramen.

MATERIAL AND METHODS

This study is based on 31 patients (2 male, 29 female) who were operated on between 1982 and 1989 at the University ENT and Neurosurgery Departments in Freiburg, Germany. The age of the patients ranged from 14

to 69 years, with an average of 56 years. Eleven patients were operated on because of recurrent tumors. The tumor histologic findings of the lesions (which were all classified according to the Fisch classification as type C and D lesions) included 21 glomus jugulare tumors, five neuromas or schwannomas, two meningiomas, one hemangioma, and two metastatic tumors.⁶ The latter two cases consisted of a metastatic adenoid cystic carcinoma and a metastatic thyroid cancer (Table 1).

High resolution computed tomography (CT) scans with bone algorithms are essential to preoperative surgical planning and were obtained in all patients.⁷ Magnetic resonance imaging was obtained in only nine patients. Angiography was performed in 26 patients, followed by subsequent embolization of tumor in five patients. All patients underwent routine postoperative CT at 24 hours. All patients received neurologic examination before surgery, at discharge (which usually took place 3 to 4 weeks postoperative), and after a mean follow-up of 4 to 8 years. Their functional deficits and possible compensation were evaluated, and postoperative complications with necessary rehabilitation measures were assessed.

A subtotal petrosectomy was used for tumor resection in 29 patients and a transsigmoid-transbulbar ap-

Table 1. Tumor Histologic Findings of 31 Patients with Lesions of the Jugular Foramen

Glomus jugulare	21
Neurinomas, schwannomas	5
Meningiomas	2
Hemangioma	1
Metastases	2

proach was used in two. The tumor was resected using microsurgical techniques with preservation of single lower cranial nerves whenever surgically feasible. These tumors were removed in a one-stage procedure in all 31 patients. Petrosotomy including rerouting of the facial nerve was performed in 29 patients, whereas in two patients the facial nerve was left in its bony canal. Additionally, the facial nerve had to be severed in six patients. In five of these patients, a primary anastomosis was performed and in one, a sural graft was performed (Table 2). The lower cranial nerves IX to XI running through the jugular foramen had to be resected in seven patients. In three patients there was a resection of nerves X to XII. Cranial nerve IX alone was resected in two patients, and IX and XI had to be resected in one patient. It was necessary to resect at least one of the lower cranial nerves in 13 patients (Table 3).

RESULTS

Comparison of the preoperative facial nerve findings with those at discharge and long-term follow-up revealed that before surgery 21 patients had normal (I/I) function.⁸ The ten remaining patients had impaired facial nerve function preoperatively due to tumor involvement or previous surgery and were scored as II/VI to VI/VI palsies. At discharge, 22 patients showed facial nerve paralysis and were divided into two groups, ten with grade II/VI or III/VI, and 12 with grade V/VI or VI/VI palsies. Nine patients had normal facial nerve function. With follow-up, these observed impaired functions often improved. Long-term results yielded 16 patients with normal function, ten with grade II or III weakness, and three with grade V or VI paralysis. The patients with paralysis (grade V or VI) preoperatively remained so after surgery except for one patient (Fig. 1).

Comparing the function of cranial nerves IX to XII

Table 3. Handling of the Cranial Nerves (Excluding VII) in Patients with Lesions of the Jugular Foramen

	<i>Sacrifice Without Repair</i>			
	<i>IX-XI</i>	<i>IX</i>	<i>IX, XI</i>	<i>X-XII</i>
Glomus jugulare	4		1	1
Schwannoma	1	1		
Meningioma	1			1
Hemangioma		1		
Metastasis	1			1

Figure 1. House classification of facial nerve function on admission, at discharge, and at follow-up.

preoperatively and postoperatively revealed no change. In 11 patients preoperatively a paresis of nerve IX was evident, and following tumor resection this number increased to 23 patients (75%). Patients with deficits of nerve X were in the same range. Preoperatively, 12 patients had a paresis of nerve X but postoperatively this increased to 19 patients, whereas in the long-term only 16 patients had dysfunction. Paralysis of nerve XI increased from nine patients preoperatively to 17 patients postoperatively and subsequently decreased to 14 patients in the long term. Ten patients had paralysis of nerve XII preoperative and postoperative these deficits remained in 14 patients (Fig. 2).

COMPLICATIONS

In one patient there was a postoperative hemorrhage in the cerebellopontine angle (CPA), which had to be

Table 2. Handling of the Facial Nerve in Patients with Lesions of the Jugular Foramen

	<i>Mobilization</i>	<i>Nonmobilization</i>	<i>Sacrifice with Repair</i>	<i>Sacrifice with Nerve Interposition</i>
Glomus jugulare	25		2	1
Schwannoma		2		
Meningioma	1		1	
Hemangioma	1			
Metastasis	2		2

Figure 2. Function of cranial nerves IX to XII on admission, at discharge, and at follow-up.

evacuated. In one other patient an internal carotid artery thrombectomy with excellent clinical outcome was performed. Two patients required closure of a cerebrospinal fluid (CSF) fistula. Three patients required a temporary tracheostomy and one professional speaker required a vocal cord medialization. Two patients died of intercurrent diseases.

DISCUSSION

Cranial neuropathies are a leading presenting symptom in patients with tumors involving the jugular foramen. The vagus was affected the most, whereas nerve XI was seldom involved. There was a tendency for neurologic deficits in one or multiple lower cranial nerves in patients with neuromas and schwannomas due to their specific growth pattern, which often involves the intra- and extradural compartments. Statistical analysis was not possible in this study due to the small number of patients. Preservation and identification of the cranial nerves is particularly difficult for the following reasons:

1. The cranial nerves lie very close to each other in the CPA and within the jugular foramen.
2. The distance between the large vessels and the petrosal sinuses is small.
3. The tumors may have an infiltrative pattern of growth.
4. The anatomic situation is modified by expansive tumors.

The situation in the loose connective tissue of the upper neck makes preservation of neural structures more likely in this region. With tumor removal, the surrounding neural structures may be damaged in several ways. Some nerves must be resected if they are involved in the tumor mass. Nerves may also be damaged by drilling, coagulation, stretching, or loss of vascular supply (such as mobilization of VII). In cases where preservation of neural structures is attempted, these functional deficits are not

always permanent. Depending on the grade, these deficits may be temporary (I–III) but usually remain if they are severe (V–VI). In some cases a functional recovery with preoperative neural impairment was seen but this was a rare occurrence. At discharge, a partial or complete dysfunction of the lower cranial nerves was pronounced, but in some patients an intact function was observed.

The deficits were prominent for nerve IX due to the surgical approach used to prepare the intratemporal segment of the internal carotid artery. This was closely followed by deficits of nerves X, XI, and XII. Due to the resection of one or multiple lower cranial nerves, the majority of patients had severe and moderate deficits at discharge. These deficits improved during follow-up, but when the dysfunction was severe preoperatively it was found to persist with time. All patients had their tracheostomy and feeding tubes removed. They were able to breathe normally and maintained their weight by adequate oral intake. Intensive voice rehabilitation was mandatory because of a breathy voice and in professional speakers a medialization procedure of the vocal cord may be indicated, but in our patient population all were able to compensate with time.

CONCLUSIONS

1. Due to the anatomic situation, preservation of a single lower cranial nerve is difficult.
2. Preoperative nerve dysfunction made intraoperative preservation unlikely.
3. Postoperative lower cranial nerve dysfunction was often transient.
4. Compensation of permanent dysfunction was usually excellent.
5. Voice training in all patients was mandatory.
6. Medialization of the vocal cord for professionals may be indicated.
7. Surgery for dysphagia was unnecessary.

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