Anterior clinoidal meningiomas: Report of a series of 33 patients operated on through the pterional approach

Fabrizio Puzzilli,¹ Andrea Ruggeri, Luciano Mastronardi, Antonino Agrillo, and Luigi Ferrante

University "La Sapienza," Department of Neurological Sciences, 2nd chair of Neurosurgery, Rome, Italy

Between 1985 and 1995, 33 cases of clinoidal meningioma were surgically treated by pterional approach. In 6 cases, according to the grading scale of Al-Mefty, the lesions were group I, having originated from the lower part of the clinoid; in 22 cases, the lesions were group II, having originated from the upper or lateral part of the clinoid process; and in 5 cases, the lesions were group III since they arose from the optic foramen. Postoperatively, 17 patients showed an improvement, 4 were unchanged, and 6 presented further deficits. Five patients died after surgery: two from pulmonary thromboembolism, one from myocardial infarction, one from hematoma of the operative field, and one from cerebral ischemia after severe vasospasm of the internal carotid artery (unresponsive to treatment). The mean follow-up was 53.7 months (range 12-108 months) and included 19 patients. During this period, there were five recurrences, and three patients underwent resection again and showed no signs of tumor regrowth 1 year later; one patient who did not undergo resection again due to his age and poor general conditions died 3 years after onset of the recurrence; the last patient has so far refused a second operation. The clinical, diagnostic, and therapeutic aspects of this not infrequent pathology are discussed in the light of our experience and the pertinent literature. *Neuro-Oncology* 1, 188-195, 1999 (Posted to *Neuro-Oncology* [serial online], Doc. 98-21, June 16, 1999. URL <neuro-oncology.mc.duke.edu>)

The incidence of sphenoidal ridge meningiomas ranges from 14 to 23.7% of all intracranial meningiomas (Adegbite et al., 1983; Cook, 1971; Fohanno and Bitar, 1986; MacCarthy, 1973; Simpson, 1957). In 1938, Cushing (Cushing and Eisenhardt, 1938) proposed a classification that divided these tumors into outer or pterional, middle or alar, and deep inner or clinoidal.

In the past, the latter group, also known as sphenocavernous meningiomas, were frequently grouped with suprasellar meningiomas and those of the middle and external thirds, even though their relationship with the internal carotid artery, middle cerebral artery, optic nerve, and cavernous sinus often compromises their chance of radical removal and determines very different rates of morbidity, surgical mortality, and recurrences.

The incidence of clinoidal meningiomas in other studies ranges from 34 to 43.9% of all intracranial meningiomas (Al-Mefty, 1991; Al-Mefty and Ayoubi, 1991; Cophignon et al., 1979). They may develop toward the midline-involving the dura of the jugum sphenoidale, the hypophysis, and the clivus (Bonnal et al., 1980)-and extend to the cavernous sinus, or they may develop in a lateral direction (Risi et al., 1994), invading the orbital cavity (Cophignon et al., 1979; Fohanno and Bitar, 1986) or infiltrating the bone, causing hyperostosis (Pompili et al., 1982).

The grading system proposed in 1994 by Risi (Risi, 1994), based on the direction of tumor development, divided these lesions into pure clinoidal, clinoidal with lateral extension, or clinoidal with cavernous sinus extension. For the purposes of our study, however, the grading system used was the one proposed by Al-Mefty in 1990, which takes into consideration not only the point of origin of the meningioma but also its relationship with the internal carotid artery. In fact, Al-Mefty divided clinoidal meningiomas into three groups: Group I was all tumors

Received 25 September 1998, accepted 29 March 1999.

¹Address correspondence and reprint requests to Fabrizio Puzzilli, MD, Via V. Rotellini, no. 113 sc. B., Rome 00128, Italy.

Table 1. Presenting symptoms grouped according to Al-Mefty's classification

Group	Total no. of cases	Headache	Epilepsy	Visual disturbances	Third cranial nerve deficit	Hemiparesis	Psychocognitive signs	Others
I	6	0	2	3	1	0	0	0
II	22	8	9	1	0	1	2	1
III	5	1	0	3	1	0	0	0

Classification according to Al-Mefty (1991).

that have an implant on the lower part of the clinoid and that develop in the carotid cistern and encase the artery, adhering directly to the adventitia in the absence of an arachnoidal membrane; Group II was lesions that originate from the superior or lateral aspect of the anterior clinoid process and that, as they grow, come into contact with the carotid artery, with interposition of an arachnoid membrane deriving from carotid and Sylvian cisterns; Group III was tumors that originate from the optic foramen in which the arachnoid membrane is present between vessels and tumor, but may be lacking between tumor and optic nerve.

The presence of the arachnoid membrane is essential for total removal because, by offering a plane of cleavage, it allows the vessels to be completely freed from the tumor. On the whole, it is safe to say that tumors belonging to Groups II and III can be totally removed, in contrast to those of Group I, which often demand subtotal resection to avoid severe postoperative neurological deficits.

In the past, total removal of these lesions was not always possible without exposing the patient to a substantial risk of severe neurologic deficits. Nowadays, advances in surgery of the skull base and cavernous sinus, together with the introduction of microsurgical techniques and new diagnostic investigations such as the Matas test, have made more radical surgery possible.

We describe here a series of 33 consecutive surgical cases of anterior clinoidal meningioma and review their clinical, diagnostic, and therapeutic features.

Materials and Methods

Between January 1985 and December 1995, 33 patients with clinoidal meningioma were consecutively treated: 19 (57.6%) were females from 36 to 83 years of age (average age, 57.7) and 14 (42.4%) were males between 13 and 75 years of age (average age, 56). On average, our patients spent 28.7 days in the hospital (range, from 15 to 69 days), while postoperative hospitalization averaged 18.4 days (range, from 8 to 58 days), excluding patients who died after surgery.

The length of clinical history averaged 39 months (range, from 5 to 25 years). The number of patients experiencing clinical signs and symptoms were as follows: visual disturbances, 15 (45.5%); seizures, 12 (36.4%); headache, 11 (33.3%); hemiparesis (varying in severity), 9 (27.3%); intracranial hypertension, 8 (24.2%), with just one case of Foster-Kennedy's syndrome; psychocognitive symptoms, predominantly of the frontal type, 7

(21.2%); exophthalmos, 4 (12.1%); language disorders, 2 (6%); and impairment of equilibrium, 1 (3%). Cranial nerve involvement consisted of a central deficit of the facial nerve in five patients (15%), common oculomotor nerve in five patients (15%), abducens in three patients (9%), first branch of the trigeminal nerve in one patient, and second and third branches in two (6%) patients. We also observed one case of cerebrospinal fluid leak, one of urinary incontinence, and one of Parkinson-type tremors.

The first sign of tumor (Table 1) and the number of patients exhibiting it were epilepsy in 11 (33%), headache in 9 (27.3%), visual disturbances in 7 (21%), third cranial nerve palsy in 2 (6%), frontal syndrome in 2 (6%), and hemiparesis in 1 (3%). In one asymptomatic patient, papilloedema was an incidental finding during ophthalmological examination.

All patients were investigated by CT (Figs. 1 and 2); 31 were investigated by angiography and 8 by MRI. In 11 cases there was edema; in 8 it was focal, and in 3 it was diffuse. The balloon occlusion test (Matas test) was done in nine patients.

The patients of our series were classified according to the criteria proposed by Al-Mefty based on the origin of tumor and its relationship with the neighboring vessels and nerves (Al-Mefty, 1990; 1991; Al-Mefty and Ayoubi, 1991).

The amount of surgical removal was classified using the scale proposed by Simpson (Cook, 1971): grade I, macroscopically complete removal of tumor, with excision of its dural attachment and of any abnormal bone; grade II, complete removal with coagulation of dural attachment; grade III, complete removal without resection or coagulation of its dural attachment or its extradural extension (for example, an invaded sinus or hyperostotic bone); grade IV, partial removal, leaving the intradural tumor in situ; and grade V, simple decompression.

Results

In accordance with the Al-Mefty scale, our clinoidal meningiomas belonged to group I in 6 cases, group II in 22 cases, and group III in 5 cases (Tables 2, 3, and 4). In five cases, it was not possible to identify the exact point of origin of the tumor on the basis of the written account of the operation alone. In these cases, tumors that certainly infiltrated the carotid artery wall were classified as group I, while those that involved the carotid artery without a sure adhesion or with an interposed arachnoid membrane were classified as group II.

Removal was total (Simpson grades I or II) in 18 cases (54.5%) and subtotal in 15 (45.5%).

Fig. 1. Cerebral CT scans of three patients in this series.

Histologically, 16 meningiomas were of transitional type (2 with atypias), 11 were endothelial (2 with atypias), 2 syncytial, 2 psammomatous, 1 vascular, and 1 fibrous. The tumor involved the carotid artery in 18 cases (54.5%), middle cerebral artery in 18, cavernous sinus in 13 (39.4%), optic nerve in 12 (36.4%), middle cranial fossa in 10 (33.3%), A1 segment in 9 (27.3%), middle and external thirds of the sphenoid wing in 7 (21.2%), orbit and/or optic canal or foramen in 6 (18%), tuberculum sellae in 5 (15%), and anterior cranial fossa in 2 (6%).

Neurological status improved in 17 patients after surgery and remained the same in 4. Seven patients presented permanent deficits: ischemia-related hypostenia on the side opposite the lesion in two and on the same side in one, third cranial nerve palsy in four, ophthalmoplegia in one, fourth cranial nerve palsy in one, and seventh cranial nerve palsy in one patient.

Five patients died in the postoperative period: two from pulmonary thromboembolism; one from acute myocardial infarction that occurred unexpectedly during postoperative recovery, during which all patient's neurological symptoms had improved; one from untreatable vasospasm of the carotid artery, which caused an extensive ischemic lesion; and one from cardiorespiratory failure 11 days after reoperation for the removal of a hematoma of the operative field. The last two patients had a group I meningioma, the remaining three had a group II lesion.

Complications included: one case of brain edema, successfully treated with osmotics; one case of sepsis of surgical wound, which required surgical revision; two cases of cerebral hematoma, which also required operation; and two cases of hydrocephalus, which appeared 10 days and 5 months after surgery, respectively, both resolved by application of a ventricular-peritoneal shunt.

Fig. 2. (A) Preoperative and (B) postoperative CT-scan of one patient in this series.

Follow-up

Clinical, CT, and MRI follow-up ranged from 12 to 108 months (average, 53.7 months) and was performed in each patient at intervals of 1 year. It was possible in 19 cases; 5 patients died and 9 were lost during follow-up. The last patients were followed from 4 months to 1 year prior to their being lost, without clinical or neuroradiological recurrence of tumor.

Tumor removal was total in 7 and subtotal in 12 patients; the tumor was group II in 11, group III in 4, and group II in 4 patients. There were five clinical recurrences in patients who had been treated by subtotal removal; recurrences were 1 year after operation in two (Al-Mefty groups I and II), 2 years after operation in two

(group II), and 7 years after operation in one patient (group III). Of these, the tumors of three were resected again and did not present signs of tumor regrowth 1 year after the second operation; one patient (No. 13) did not undergo resection again in view of his age and poor general conditions and died 3 years after the onset of recurrence; and the remaining patient (No. 32) has so far refused another operation.

Discussion

In reported series of sphenoidal meningiomas, incidence of the clinoidal variety ranges from 34% (Al-Mefty, 1991) to 43.9% (Cophignon et al., 1979). The clinical

Table 2. Patients in Group I (Al-Mefty classification system)

Patient No.	First symptom	Other symptoms	Postoperative deficits		Tumor extension	Removal ^a	Recurrence
			Transient	Permanent			
9	cranial nerve II (3 yr)	Foster-Kennedy's, hemiparesis		death	orbital roof, MCF, MCA, ICA, cranial nerve II	subtotal-Simpson IV	N/A
10	eyelid ptosis (10 yr)	cranial nerves II, VI, VII, exophthalmos	hemiparesis, aphasia	cranial nerves II, III, VI, VII	ICA, MCA, cranial nerve II, cavernous sinus, free edge of the tentorium	subtotal-Simpson IV	No-9 yr
17	cranial nerve II (5 yr)	eyelid ptosis	eyelid ptosis	cranial nerve II	ICA, ACA, MCA, cranial nerve II, cavernous sinus	subtotal-Simpson IV	No-5 yr
20	epilepsy (4 mo)	amnesia		death	ICA, MCA, tuberculum sellae, cavernous sinus, free edge of the tentorium	total-Simpson II	N/A
21	epilepsy (2 yr)		cranial nerve III		both ICAs, ACA, MCA, cranial nerve III, sella	subtotal-Simpson IV	No-6 yr
32	cranial nerve II (5 mo)	headache, exophthalmos		cranial nerve VII, hemiparesis	ICA, MCA, cranial nerve II, MCF, tentorium	subtotal-Simpson IV	Yes-1 yr

ACA, anterior communicating artery; ICA, internal carotid artery; MCA, middle cerebral artery; MCF, middle cranial fossa.

Classification was according to the Al-Mefty system (1991), except for Removal, where the Simpson classification system was used (Simpson, 1957). All tumors were Grade 1 of the American Scale of Anesthesiological Risk.

signs observed in these studies and in our own are summarized in Table 5.

Postoperatively, clinical status either improved or remained the same in 50-78% of reported cases (Al-Mefty, 1991; Fohanno and Bitar, 1986; Risi et al., 1994) and in 63.6% of our own series. In reported series (Al-Mefty, 1991; Fohanno and Bitar, 1986; Risi et al., 1994), between 10.7 and 29% of patients worsened, while this was true in 21.2% in our series.

In the series described by Bonnal et al. (Bonnal et al., 1980), out of seven patients (classified by the author as group A), three died after operation (42.8%). In Fohanno's series of patients operated on for meningiomas of the internal third of the sphenoid wing (Fohanno and Bitar, 1986), mortality was 27%. Two of the 28 patients of Al-Mefty's series died (7.2%), one from pulmonary embolism and one from cerebral ischemia secondary to vasospasm (Al-Mefty, 1991). In his series of 34 patients, Risi reported 2 deaths (5.9%) due to severe brain ischemia (Risi, et al., 1994). In our series, five patients died in the postoperative period: two from pulmonary thromboembolism, one from acute myocardial infarction, one from cerebral ischemia secondary to unresponsive vasospasm of the carotid artery, and one from cardiorespiratory failure 11 days after reoperation for cerebral hematoma.

The most frequent operative complications described in the literature (Al-Mefty, 1990, 1991; Al-Mefty and Ayoubi, 1991; Fohanno and Bitar, 1986; Risi et al., 1994) were cerebrospinal fluid leakage, hematoma, cerebral infarction, hydrocephalus, third cranial nerve palsy, diabetes mellitus, aggravation of seizures, and hemiplegia. In Risi's series, there were three cases of cerebrospinal fluid leak, two epidural hematomas, one hydrocephalus, and

one brain infarct. Al-Mefty reported four cases of diabetes insipidus (transient in three, permanent in one) and one of vasospasm. Our complications included one case of cerebral edema, one case of wound infection, two cases of intracerebral hematoma, and two cases of tardive hydrocephalus.

Three of the patients reported by Risi sustained a recurrence 1, 2, and 3 years after their first operation, while Cophignon noted one recurrence after 1 year in a series of six patients. Al-Mefty observed two recurrences, one in a patient with a group I meningioma and one in a group II patient, both of whom had undergone subtotal resection of tumors. In our series, there were five recurrences (all after subtotal resection) 1 year after operation in two cases (groups I and II according to Al-Mefty's classification), 2 years after in two cases (group II), and 7 years after in one case (group III).

Adegbite et al. (1983) found no statistically significant difference between the results of Simpson Grade I and Grade II surgery; on the other hand, there was a considerable difference in comparison with Grade IV surgery. The high percentage of recurrences in clinoidal meningiomas described in published series is, in fact, linked to the difficulties frequently encountered in achieving radical surgery (Cook, 1971; Dolenc, 1979; Pompili et al., 1982). In many cases, the chances of radical removal are limited by involvement of the internal carotid artery and its branches, of the cavernous sinus, and of the optic nerve.

Surgical radicality in the management of tumors arising near the optic foramen is the object of controversy. Microsurgical technique has dramatically improved the chance of total removal, reducing the incidence of visual morbidity (Al-Mefty, 1990, 1991; Al-Mefty and Ayoubi,

Table 3. Patients in Group II (AI-Mefty classification system)

Patient No.	ASA grade	First symptom	Other symptoms	Postoperative deficits		Tumor extension	Removal	Recurrence
				Transient	Permanent			
1	1	epilepsy (3 yr)		disorientation, aphasia		middle 1/3 sph. wing, ICA, ACM	total-Simpson I	
3	1	headache (6 mo)	CSF leakage	hemiparesis, monolateral ophthalmoplegia, disorientation		middle and external 1/3 sph. wing, tuberculum sellae, free edge of the tentorium	total-Simpson II	
4	1	epilepsy (9 yr)	cranial nerve VII	cranial nerve VII		middle 1/3 sph. wing	total-Simpson I	
5	3	headache (10 yr)	hemiparesis, psychocognitive signs, urinary incontinence, cranial nerves VI, VII, papilloedema	hemiplegia, stupor	death	MCF	total-Simpson II	N/A
7	1	headache (1 yr)	hemiparesis, papilloedema	hypostenia		middle 1/3 sph. wing, frontal planum	total-Simpson II	
8	1	headache (3 yr)	epilepsy, papilloedema	cranial nerves III, IV		cavernous sinus, MCF, free edge of tentorium	total-Simpson II	No-8 yr
13	1	epilepsy (10 yr)	hemiparesis, aphasia, cranial nerve VII, psychocognitive signs	hemiparesis, aphasia		middle and external 1/3 sph. wing	subtotal-Simpson IV	Yes-4 yr
14	1	psycho-cognitive signs (2 yr)	headache, cranial nerve VII, hypostenia	neurologically intact		cavernous sinus	total-Simpson I	
15	1	headache (25 yr)	cranial nerve II	cranial nerve II		tuberculum sellae, cranial nerve II	total-Simpson I	
16	1	hemiparesis (3 mo)			hemiparesis, cranial nerve III	tuberculum sellae, cavernous sinus, Sylvian fissure	total-Simpson II	
18	1	headache (1 yr)	vomiting, papilloedema	aphasia, paresis right upper limb		MCA, Sylvian fissure	total-Simpson I	
19	1	papillo-edema		neurologically intact		cavernous sinus	subtotal-Simpson IV	Yes-6 yr
22	1	epilepsy (5 d)		aphasia, eyelid ptosis		ICA, ACA, MCA, cranial nerve II, MCF, cavernous sinus	total-Simpson II	No-2 yr
23	2	psycho-cognitive disturbances (1 yr)	cranial nerve II, papilloedema	psychocognitive signs		ICA, ACA, MCA, cranial nerve II	total-Simpson I	No-1 yr
24	1	epilepsy (4 yr)	eyelid ptosis, cranial nerves VI, II		homolateral ophthalmoplegia, hemiplegia	ICA, ACA, MCA, posterior comm. artery, cranial nerve II, optic foramen, cavernous sinus	total-Simpson II	No-5 yr
26	1	cranial nerve II (2 yr)	tremor of one hand, epilepsy, Babinsky+	homolateral ophthalmoplegia	tremor, cranial nerve II	ICA, ACA, MCA, posterior comm. artery, MCF	subtotal-Simpson IV	Yes-4 yr
27	2	epilepsy (6 mo)	cranial nerve II		cranial nerve II	ICA, cranial nerve II	subtotal-Simpson IV	No-1 yr
28	1	headache (2 mo)	cranial nerve V (2 nd and 3 rd branch)	hemiparesis		ICA, ACA (A2), MCA, cranial nerve III, cavernous sinus	subtotal-Simpson IV	No-2 yr
29	2	episode of aphasia and ataxia (4 yr earlier)			hemiparesis	ICA, ACA, MCA, MCF, cavernous sinus, free edge of tentorium	total-Simpson II	No-2 yr
30	2	epilepsy (15 d)		neurologically intact	death (myocardial infarction)	MCA, MCF, ACF, middle and external 1/3 sph. wing, cranial nerve IV, tentorium, interpeduncular cistern	total-Simpson II	
31	1	epilepsy (6 mo)	cranial nerve II, V, neuralgia	neurologically intact		ACA, MCA, MCF, cavernous sinus	subtotal-Simpson IV	No-1 yr
33	2	headache (2 mo)	psychocognitive signs, cranial nerve II	drowsiness	death		subtotal-Simpson IV	N/A

(See Table 2 definition of abbreviations. ACF, anterior cranial fossa.)

Classification was according to the AI-Mefty system (1991), except for Removal, where the Simpson classification system was used (Simpson, 1957).

Table 4. Patients in Group III (Al-Mefty classification system)

Patient No.	ASA grade	First symptom	Other symptoms	Postoperative deficits		Tumor extension	Removal	Recurrence
				Transient	Permanent			
2	1	dylopia (17 mo)	cranial nerve II		cranial nerves II, III	orbit, cavernous sinus	total-Simpson II	
6	1	cranial nerve II (1 yr)	exophthalmos		cranial nerves II, III, IV	MCF, optic foramen, tentorium	subtotal-Simpson IV	Yes-3 yr
11	1	cranial nerve II (2 yr)	cranial nerve II contralateral	coma	cranial nerves II, III	cranial nerve II	subtotal-Simpson IV	No-8 yr
12	1	cranial nerve II (18 mo)	exophthalmos		cranial nerve II, midriasis	optic canal, ICA	total-Simpson I	No-8 yr
25	2	headache, vomiting (4 mo)	papilloedema	neurologically intact		ICA, cranial nerve II, optic foramen, tentorium	total-Simpson I	No-4 yr

(See Table 2 for definition of abbreviations.)

Classification was according to the Al-Mefty system (1991), except for Removal column, where the Simpson system was used (1957).

1991). However, when cleavage of the arachnoid membrane is not recognizable, dissection of tumor from the optic nerve and/or chiasm is impossible. In these cases, it seems better to perform only a subtotal removal, considering that recovery of visual deficits after clinoidal meningioma surgery is poor (Al-Mefty, 1990, 1991; Al-Mefty and Ayoubi, 1991).

Another problem relates to invasion of the cavernous sinus, which seems to be the main cause of recurrence (Bonnal et al., 1980). Fifteen of the patients in Risi's series presented with cavernous sinus invasion, and total resection of tumor was possible in only 4 of them. In our series, total excision of tumor was possible in 7 of 13 patients with cavernous sinus involvement, with one postoperative death, one case of ophthalmoplegia, two permanent and two transient deficits of the third cranial nerve.

Many authors have expressed their opinion regarding the treatment of meningiomas involving the cavernous sinus (Al-Mefty and Smith, 1988; Kleinpeter and Bock, 1990; Sekhar et al., 1989; Sephernia et al., 1991). Recently, Suzuki et al. (1995) described a series of patients treated by chemical embolization of the feeding arteries to reduce intraoperative bleeding, followed by subtotal surgical removal. It does seem possible that in the near future combined treatment of this pathology will allow better results accompanied by lower rates of mortality, morbidity, and recurrence.

Radiation therapy is a viable adjuvant therapy in treating residual or recurring tumors (Al-Mefty, 1990). In the presence of small clinoidal meningiomas, so long as the tumor is not severely compressing the optic nerve or chiasm, radiosurgery can be performed and a high rate of tumor growth can be arrested (over 90%) (Chang and Adler, 1997; Kondziolka et al., 1991; Leber et al., 1998; Pendl et al., 1997). The risks to vision are low, but are dependant on the proximity of the optic apparatus (Leber et al., 1998).

Conclusions

The aim of our study was to evaluate the relationships existing between surgical outcome and the point of origin of tumor on the internal third of the sphenoid wing.

From the literature and from our experience, a total resection seems to be more feasible in clinoidal meningiomas belonging to groups II and III of Al-Mefty's classification. From Al-Mefty's scale and in our experience, meningiomas of the first group present the greatest difficulties in terms of both surgical treatment and functional recovery of preoperative deficits, due to their site of origin and their relationship with neurovascular structures.

Because of the risk of severe postoperative neurological deficits with total resection, the degree of removal in these meningiomas is still not satisfying (in group I patients, total resection in one of six, subtotal in five of six in our series). The degree of tumor removal is also influenced by the frequency of carotid artery infiltration (four of six infiltration, two of six encasement in our series) and involvement of cranial nerves II, III, and IV (three of six visual field impairment, one of six third nerve palsy).

The particular tendency of these tumors to infiltrate the cavernous sinus and rapidly involve the structures within it makes them a challenging pathology, even

Table 5. Comparison of preoperative symptoms in published series

Symptom ^a	Risi ^a (%)	Al-Mefty ^b (%)	Fohanno ^c (%)	Our series (%)
Epilepsy	44	10.7	33.3	36.4
Headache	23.5		39	33.3
Visual disturbances	44	86	55.5	45.5
Psychocognitive	29.4		33.3	21.2
Hemiparesis	20.6		22.2	27.3
Papilledema	23.5	25	50	21.2
Foster-Kennedy's		3.6	16.6	3
Exophthalmos	14.7		5.5	12.1
Cranial nerve III	17.6		10	15.2
Cranial nerves III + V		21.4		
Cranial nerve V			11.1	6
Cranial nerve VI	11.7			9

^aRisi et al., 1994.
^bAl-Mefty, 1991.
^cFohanno and Bitar, 1986.

though constant evolution of microsurgical techniques and greater diagnostic potential (of arteriography and of

preoperative embolization) will make them easier and less dangerous to treat in the future.

References

- Adegbite, A.B., Khan, M.I., Paine, K.W.E., and Tan, L.K. (1983) The recurrence of intracranial meningiomas after surgical treatment. *J. Neurosurg.* **58**, 51-56.
- Al-Mefty, O. (1991) Clinoidal meningiomas. In: Al-Mefty, O. (Ed.), *Meningiomas*. New York: Raven Press, Ltd. pp. 427-443.
- Al-Mefty, O. (1990) Clinoidal meningiomas. *J. Neurosurg.* **73**, 840-849.
- Al-Mefty, O., and Ayoubi, S. (1991) Clinoidal meningiomas. *Acta Neurochir. Suppl.* (Wien) **53**, 92-97.
- Al-Mefty, O., and Smith, R.R. (1988) Surgery of tumors invading the cavernous sinus. *Surg. Neurol.* **30**, 370-381.
- Bonnal, J., Thibaut, A., Brotchi, J., and Bom, J. (1980) Invading meningiomas of the sphenoid ridge. *J. Neurosurg.* **53**, 587-599.
- Chang, S.D., and Adler, J.R., Jr. (1997) Treatment of cranial base meningiomas with linear accelerator radiosurgery. *Neurosurgery* **41**, 1019-1027.
- Cook, A.W. (1971) Total removal of large global meningiomas at the medial aspect of the sphenoid ridge. *J. Neurosurg.* **34**, 107-113.
- Cophignon, J., Lucena, J., Clay, C., and Marchac, D. (1979) Limits to radical treatment of sphenoid-orbital meningiomas. *Acta Neurochir. Suppl.* (Wien) **28**, 375-380.
- Cushing, H., and Eisenhardt, L. (1938) *Meningiomas*. Springfield, Ill.: Thomas.
- Dolenc, V. (1979) Microsurgical removal of large sphenoidal bone meningiomas. *Acta Neurochir. Suppl.* (Wien) **28**, 391-396.
- Fohanno, D., and Bitar, A. (1986) Sphenoidal ridge meningiomas. *Adv. Tech. Stand. Neurosurg.* **14**, 137-174.
- Kleinpeter, G., and Bock, F. (1990) Invasion of the cavernous sinus by medial sphenoid meningioma: "radical" surgery and recurrence. *Acta Neurochir.* (Wien) **103**, 87-91.
- Kondziolka, D., Lunsford, L.D., Coffey, R.J., and Flickinger, J.C. (1991) Gamma knife radiosurgery of meningiomas. *Stereotact. Funct. Neurosurg.* **57**, 11-21.
- Leber, K.A., Bergloff, J., and Pendl, G. (1998) Dose-response tolerance of the visual pathways and cranial nerves of the cavernous sinus to stereotactic radiosurgery. *J. Neurosurg.* **88**, 43-50.
- MacCarty, C.S. (1972) Meningiomas of the sphenoidal ridge. *J. Neurosurg.* **36**, 114-120.
- Pendl, G., Schrottner, O., Eustacchio, S., Feichtinger, K., and Ganz, M. (1997) Stereotactic radiosurgery of skull base meningiomas. *Minim. Invasive Neurosurg.* **40**, 87-90.
- Pertuiset, B., Farah, S., Claves, L., Goutorbe, J., Metzger, J., and Kujas, M. (1985) Operability of intracranial meningiomas. Personal series of 353 cases. *Acta. Neurochir.* (Wien) **76**, 2-11.
- Pompili, A., Derome, P.J., Visot, A., and Guiot, G. (1982) Hyperostosing meningiomas of the sphenoid ridge. Clinical features, surgical therapy, and long-term observations: Review of 49 cases. *Surg. Neurol.* **17**, 411-416.
- Risi, P., Uske, A., and de Tribolet, N. (1994) Meningiomas involving the anterior clinoid process. *Br. J. Neurosurg.* **8**, 295-305.
- Sekhar, L.N., Sen, C.N., Jho, H.D., and Janecka, I.P. (1989) Surgical treatment of intracavernous neoplasms: A four year experience. *Neurosurgery* **24**, 18-30.
- Sephenia, A., Samii, M., and Tatagiba, M. (1991) Management of intracavernous tumors: An 11-year experience. *Acta Neurochir. Suppl.* (Wien) **53**, 122-126.
- Simpson, D. (1957) The recurrence of intracranial meningiomas after surgical treatment. *J. Neurol. Neurosurg. Psychiat.* **20**, 22-39.
- Suzuki, M., Mizoi, K., and Yoshimoto, T. (1995) Should meningiomas involving the cavernous sinus be totally resected? *Surg. Neurol.* **44**, 3-13.