Chapter 190: Surgery of the Anterior and Middle Cranial Base

Daniel W. Nuss, Ivo P. Janecka

Throughout most of modern medical history, the cranial base was regarded as surgically unapproachable because of the anatomic complexity and vital importance of the structures within its boundaries. Over the past several decades, however, many incremental advances have been made in oncologic and reconstructive surgery, anesthesiology, radiology, and other related fields, which have helped to overcome these obstacles, making surgery of the cranial base both technically feasible and therapeutically effective. As a result, surgical resection can now be considered as the primary treatment modality for many cranial base lesions that would have previously been considered inoperable.

The cranial base is often conceptually divided into three anatomic regions, named for their intracranial relations to the overlying cranial fossae: anterior, middle, and posterior (Fig. 190-1). From a diagnostic as well as a therapeutic point of view, it is useful to consider the anterior and middle cranial base regions together for a number of reasons. They both contribute to what is commonly referred to as the craniofacial junction, an important area in which the neurocranium and the viscerocranium meet (Fig. 190-2). Both share anatomic relationships with the orbits, the nasal airway, and the paranasal sinuses and are therefore affected by a number of similar pathological processes. Both the anterior and middle cranial base regions are commonly approached using craniofacial disassembly techniques. On the other hand, the posterior cranial base is usually regarded as a separate region; it has little connection to the viscerocranium, and it is affected by different kinds of pathologic lesions. It is most often approcahed surgically using neurosurgical and neurotologic methods.

This chapter focuses on the surgical management of lesions affecting the anterior and middle cranial base.

Surgical Anatomy

Anterior cranial base

The anterior cranial base can be defined as that portion of the skull base adjacent to the anteriro cranial fossa. It is bounded anteriorly by the *frontal bone,* which contains two surgically important structures: the frontal sinus and the supraorbital foramina (Fig. 190-3). The *frontal sinus* is extremely variable in size and extent but must be dealt with, if present, in most anterior cranial base operations. The *supraorbital foramina,* which in some individuals may be incomplete (and therefore referred to as supraorbital notches), transmit the supraorbital nerves and vessels. These vessels contribute a major portion of the blood supply to the galea and the pericranium of the frontal region. They must be preserved if the galea and pericranium are to be used in the reconstruction of anterior cranial base defects.

Superiorly, the anterior cranial base is formed by the frontal, ethmoid, and sphenoid bones (Fig. 190-4). From an intracranial perspective several important landmarks are visible. The most anterior of these is the *foramen cecum,* the site of a communication between veins of the nasal cavity and the origin of the superior sagittal sinus. The next anatomic landmark, the *crista galli,* protrudes upward from the midline to provide attachment for the falx cerebri.

On either side of the crista are the many openings in the *cribriform plate,* which transmit the olfactory nerves inferiorly. Just posterior to the last of these olfactory foramina is a smooth-surfaced area known as the *planum sphenoidale.* It forms the roof of the sphenoid sinus (when the sinus is well pneumatized). The *anterior clinoid processes* and *lesser sphenoid wings* delineate the most posterior limit of the anterior cranial base. Here the middle cranial base begins. Between and slightly below the clinoids are the *optic canals* and the *internal carotid arteries* (ICAs); although technically these structures are not part of the anterior cranial base, they are sufficiently close that they must be protected during anterior cranial base resections.

Extracranially, the anterior cranial base is topographically related to the nasal cavity, the ethmoid and sphenoid sinuses, and the orbits (Fig. 190-5). Several anatomic points are worthy of emphasis within the context of cranial base surgery. First, the floor of the anterior cranial fossa is usually quite uneven. The relatively flat orbital roofs slope downward medially. This places the ethmoid roof even lower. The cribriform area (the roof of the nasal cavity) may be even lower still. In some cases the cribriform area may be more than 1 cm below the level of the rest of the anterior cranial fossa floor. This nonplanar arrangement is important during transethmoid extracranial approaches to lesions of the cribriform area. For example, an axial plane of dissection that is safe along the roof of the ethmoid may risk injury to the dura and frontal lobes if extended medially to encompass the cribriform plate territory.

Second, the *orbits* contain several landmarks that can help surgical oritentation during cranial base operations (Fig. 190-6). The *superior orbital fissure* transmits the oculomotor, trochlear, ophthalmic, and abducens cranial nerves (III, IV, V-1, and VI) as well as the ophthalmic vein, and communicates with the middle cranial fossa. The *inferior orbital fissure* contains the maxillary nerve (V-2) and communicates with the pterygopalatine fossa; the lateral end of this fissure is an important landmark for placement of lateral orbital osteotomies. The *optic canal* transmits the optic nerve and ophthalmic artery. The *anterior* and *posterior ethmoid foramina* encompass the anterior and posterior ethmoid arteries. Ligation of these vessels can reduce intraoperative bleeding in the nasal vault region. More importantly, these ethmoid foramina mark the position of the frontoethmoid suture line, a valuable and constant guide to the level of the ethmoid roof and anterior fossa floor in this region. The posterior ethmoid foramen is of additional significance because of its consistent relationship to the optic canal. In the majority of individuals the optic nerve will be found 4 to 7 mm posterior to the posterior ethmoid foramen.

Middle cranial base

The middle cranial base forms the floor of the middle cranial fossa. From the intracranial perspective (Fig. 190-7), the middle cranial base begins anteriorly at the posterior edge of the lesser sphenoid wing; posteriorly it ends at the posterosuperior edge of the petrous part of the temporal bone. The intracranial surface of the middle cranial base is formed by the greater wing and body of the sphenoid bone as well as the petrous and squamous portions of the temporal bone. As such, it forms the roof of the infratemporal fossa, middle ear, mastoid, and condylar fossa as well as the lateral wall of the sphenoid sinus.

The floor of the middle fossa is distinguished by a series of important foramina. Beginning anteriorly, the *superior orbital fissure, optic canal,* and *foramen rotundum* (representing the intracranial end of the inferior orbital fissure) permit communication with the orbit. Next, the *foramen ovale* delivers the mandibular nerve (V-3) to the infratemporal fossa below, and the *foramen spinosum* transmits the middle meningeal artery (a branch of the internal maxillary artery) in the opposite direction.

Along the superomedial surface of the petrous part of the temporal bone, the roof of the carotid canal is frequently, dehiscent, revealing the horizontal *petrous ICA* and the *foramen lacerum.* This foramen receives the *greater superficial petrosal nerve* (GSPN; parasympathetic fibers from the facial nerve to the lacrimal gland) - after its exit from the facial hiatus a few millimeters posterolaterally - and conducts it to the pterygopalatine fossa inferiorly. This relationship between the GSPN, the foramen lacerum, and the carotid canal is often helpful at surgery, because the GSPN, which is readily identifiable, can be followed medially to lead the surgeon to the distal petrous ICA.

Other surgically important structures along the intracranial surface of the middle cranial base include, from lateral to medial, the *temporal lobe,* the *gasserian (trigeminal) ganglion,* and the *optic chiasma.* The *pituitary gland* is flanked on both sides by the cavernous portions of the carotid arteries and the contents of the cavernous sinuses.

From an extracranial viewpoint, the middle cranial base is one of the most anatomically complex and formidable areas of the body. It extends from the posterolateral walls of the maxillary sinuses anteriorly to the petrooccipital sutures posteriorly (Fig. 190-8). It is formed by the greater wing and body of the sphenoid bone and by the temporal bone, including the condylar fossa. Like its intracranial surface, this region also contains numerous openings for major nerves and blood vessels, including the previously mentioned foramina ovale, spinosum, and lacerum, as well as the *stylomastoid foramen* (cranial nerve VII), the *jugular foramen* (internal jugular vein, cranial nerves IX, X, XI), and the *carotid canal* (entrance of internal carotid artery into the temporal bone).

Many operative approaches to this region traverse the *infratemporal fossa,* where knowledge of several important anatomic relationships will facilitate surgical exposure and safety. First, the *muscles of mastication,* including the temporalis, masseter, and medial and lateral pterygoids, are encountered. These muscles all receive their blood supply from branches of the *internal maxillary artery,* which must be preserved if, for example, the versatile temporalis muscle is to be used as a flap for reconstruction. Second, both of the pterygoid muscles originates in part from the *lateral pterygoid plate* (of the sphenoid bone), which serves as an excellent landmark because (1) it is a palpable guide for surgical orientation; (2) it can be easily exposed by dissection medially along the greater sphenoid wing; and (3) it is easily identified on radiographic imaging studies, especially CT scans. The root of this lateral pterygoid plate is situated immediately posterior to the foramen rotundum and anterior to the foramen ovale. It can therefore be used as an index to the positions of the maxillary (V-2) and mandibular (V-3) divisions of the trigeminal nerve (Fig. 190-9). Once the foramen ovale is identified, the foramen spinosum (transmitting the middle meningeal artery) will be found just posterior to it; this leads to the next critical landmark, the *spine of the sphenoid.*

The sphenoid spine, situated just medial to the *condylar fossa,* also serves as a palpable, radiographically identifiable landmark that is important because of its location immediately lateral to the carotid canal (see Fig. 190-8). Thus it helps the surgeon to locate the highest portion of the cervical internal carotid artery (ICA), which can be followed distally to expose and mobilize the petrous ICA. Because the sphenoid spine is just medial to the condylar fossa, the mandibular condyle is often displaced anteroinferiorly or resected to enhance exosure of the ICA as it enters the cranial base.

A thorough and detailed understanding of the regional anatomy of the cranial base is best achieved through correlative study of dry skulls, cadaveric dissections, and radiographic images.

Preoperative Considerations

Symptoms

Symptoms caused by cranial base lesions may range from minor headache to major disturbance of central nervous system functions. Unfortunately, many cranial base disorders are insidious in that they cause few symptoms until they have reached advanced stages or have begun to impinge on cranial nerves, major vessels, or brain. This paucity of early symptoms makes early diagnosis difficult. In addition, many of the *nonneurologic* symptoms caused by cranial base lesions, such as nasal obstruction, hearing loss (from middle ear effusion), and sinusitis, are often mistakenly attributed to more commonplace causes, delaying diagnosis even further.

When symptoms do develop, their nature depends on the location of the lesion. Anterior cranial base lesions are likely to present with anosmia, visual disturbance, or nasal obstruction. They may also cause diplopia and proptosis in cases of orbital wall extension. Middle cranial base lesions may also present with diplopia and proptosis, either by direct orbital extension through the orbital fissures or by cavernous sinus invasion. Lesions of the middle fossa floor can result in hemifacial or hemicranial pain or numbness caused by involvement of the trigeminal ganglion and its branches. Disease extension to the infratemporal fossa may lead to eustachian tube obstruction, serous otitis media, trismus, and difficulty in chewing. When the temporal bone is affected, the patient may complain of hearing loss, tinnitus, dysequilibrium, or facial weakness.

Far-advanced lesions of the anterior and middle cranial base can impinge on the frontal and temporal lobes, carotid artery, or midbrain and may therefore present with personality changes, memory loss, seizures, stroke, or other neurologic events. Disorders in or near the pituitary fossa and hypothalamus can cause a variety of endocrinopathies as well.

Thus the nature and severity of symptoms of cranial base lesions vary considerably. For this reason, patients who have persistent complaints referable to the cranial base, however subtle, should receive thorough diagnostic evaluation including imaging procedures.

Physical examination

Every patient being evaluated for cranial base disease should undergo a complete physical examination with special attention to the head and neck region. Because much of the cranial base is relatively inaccessible to direct inspection, many patients with cranial base disorders - even some with extensive pathologic conditions - will have no externally apparent sign of disease. For this reason the neurologic examination is particularly important as a means to elicit subtle signs of skull base involvement. It should include testing of cognitive functions (alertness, orientation, appropriateness), cerebellar function, all cranial nerves, spinal reflexes, and sensory and motor functions as well. Supplemental information obtained by ophthalmoscopy, indirect laryngoscopy, otomicroscopy, and flexible fiberoptic nasopharyngoscopy can also be quite useful. The combination of findings will often allow a reasonable estimate of the location and anatomic extent of the lesion. It should be emphasized, however, that the absence of objective physical signs does not exclude the possibility of cranial base disease, and any patient with persistent unexplained symptoms related to the skull base should be referred for precise diagnostic imaging of the skull base.

Diagnostic imaging

Diagnostic imaging of cranial base lesions has improved dramatically within the last decade because of refinements in computed tomography (CT) and magnetic resonance (MR) scanning techniques. These developments have made it possible to accurately map cranial base lesions, to define involvement of critical structures, and to plan optimal surgical approaches, all in a noninvasive way. All patients being evaluated for cranial base lesions should be studied with one of these two modalities. Frequently both modalities are employed. CT and MR images are complementary in that each technique has its own unique advantages and disadvantages. CT is especially useful in demonstrating changes in the bony architecture of the cranial base. MR, on the other hand, gives much better greater information about soft tissue differentiation. Combined data from CT (for example, bone destruction, calcification) and MRI (signal characteristics) allow a quite accurate preoperative diagnosis of tumor extent and often a reasonable probability of tumor type. This is especially important in deeply situated lesions, where preoperative biopsy may be unsafe or inadvisable.

Angiography

In many cases it is important to have information regarding the vascularity of a given cranial base lesion and the possible involvement of major neighboring blood vessels. For this reason, cerebral angiography is often part of the diagnostic imaging sequence. It permits an identification of the blood supply to the lesion and its own vascular pattern, and simultaneously it clarifies whether the ICA or other major vessels are compromised. Angiography also gives anatomic information about the integrity of the circle of Willis and existing collateral circulation to the cerebral hemispheres. It alone, however, does not give adequate *physiologic* information regarding the adequacy of the circle of Willis or other collaterals in supplying circulation to the brain (Janecka et al, 1991b). To obtain detailed physiologic information, cerebral blood flow studies are performed.

Cerebral blood flow evaluation

If a cranial base lesion involves or impinges on the internal carotid artery (ICA) or if it will be necessary to manipulate the carotid artery during surgery, then cerebral blood flow studies should be considered. These studies give a physiologic index of adequacy of circulation to the brain in a quantitative way and are useful for predicting whether an individual patient can tolerate occlusion of the ICA without major neurologic consequences (deVries et al, 1988; Erba et al, 1988; Sekhar et al, 1986, 1989).

The test begins with a temporary occlusion of the ICA, which is achieved by inflating a balloon-tipped catheter within the vessel. The catheter is placed percutaneously via the femoral artery and guided into the cervical portion of the ICA under fluoroscopic control, just as in routine cerebral angiography. This is performed with the patient awake, and serial neurologic assessments are done during the 15-minute ICA occlusion. If at any time during this clinical part of the test the patient develops any neurologic deficit, then the balloon is deflated immediately and further cerebral blood flow studies are not performed. Such a patient is considered to have failed the test and is presumed to be highly dependent on the flow in that ICA, placing him or her at high risk for stroke if the carotid is compromised at surgery. If the patient tolerates 15 minutes of ICA occlusion without developing neurologic deficit, then he or she is studied further using a quantitative test in which stable xenon gas is inhaled. The inhaled xenon is distributed throughout the circulation and into the brain where it is visible on CT scan, giving a picture of cerebral blood flow distribution (Fig. 190-10). This xenon-enhanced CT scan is performed both with the balloon inflated and deflated in the ICA. The uptake of xenon within both cerebral hemispheres is then quantitated using the digitized data from the CT scan.

The patient who has a significant ipsilateral decrease in hemispheric xenon uptake during ICA occlusion (despite the fact that he or she develops no clinically apparent neurologic deficit during the test) is considered to be at moderate risk for the development of neurologic sequelae should the carotid artery be occluded during surgery. Such patients would be candidates for extracranial-to-intracranial arterial bypass to enhance the intracranial circulation. Patients who have no drop in xenon uptake during balloon occlusion of the ICA are believed to be at low risk for postoperative stroke even if the ICA is resected or permanently occluded.

Preoperative embolization

Another advantage of preoperative angiography is the possibility for elective embolization of vascular tumor beds to help reduce intraoperative bleeding. In addition, the ICA itself may be electively embolized using detachable intra-arterial permanent balloons if the decision is made preoperatively that the carotid artery will have to be sacrificed. This is sometimes done to permit resection of malignant tumors.

Other evaluations

For lesions in the vicinity of the orbits, the visual acuity should be assessed quantitatively and measurements of the visual fields performed. For lesions in the vicinity of the temporal bone, audiologic testing as well as auditory brain-stem response (ABR), electronystagmography (ENG), and facial electromyography (EMG) should be done as indicated. The information obtained by these evaluations may help to uncover subtle cranial nerve dysfunction in some patients. In others it gives an objective measure of dysfunction that is already clinically apparent. It may also be helpful in predicting the degree of postoperative cranial nerve morbidity.

For any cranial base lesion in the proximity of the pituitary fossa, a complete endocrine evaluation should be performed. It is especially important to identify patients who preoperatively have syndrome of inappropriate antidiuretic hormone (SIADH), diabetes insipidus, or hypothyroidism, since these conditions can lead to major postoperative morbidity if not corrected. Also, abnormal levels of prolactin, growth hormone, or gonadotropins may be of diagnostic significance as indicators of tumors of the pituitary gland. The general medical status of the patient should be thoroughly investigated before undertaking cranial base surgery to identify any occult cardiac or respiratory problems as well as other systemic disorders that may negatively influence the postoperative outcome. Metastatic workup is conducted in patients with specific malignant tumors.

Pathology and treatment planning

A wide variety of disorders can affect the cranial base. Those disorders in which surgery has played a significant therapeutic role have included basicranial trauma, craniofacial anomalies, and congenital syndromes (for example, hypertelorism, Crouzon's syndrome), spontaneous cerebrospinal fluid fistulas, vascular problems (for example, petrous carotid artery aneurysms), infectious diseases (for example, petrositis, "malignant" external otitis), and neoplasms. Perhaps the widest acceptance and application of cranial base surgery have been in the treatment of neoplasms, for in many such cases the effectiveness of other modalities is limited.

Benign tumors and related lesions affecting the skull base have been reviewed by several authors (Austin and Mills, 1986; Dickins, 1981; Dickins and Graham, 1991) and are summarized in the box below. In general, benign cranial base neoplasms are treated surgically. Technically they are often managed by "piecemeal" removal, progressing from one surgical landmark to the next, to allow for maximal preservation of functionally important structures.

Box: Benign lesions of skull base

Extracranial

Inverted papilloma
Angiofibroma
Salivary gland tumors
Paraganglioma (glomus)
Mucoceles
Cholesteatoma

Intracranial

Pituitary adenoma
Craniopharyngioma
Meningioma
Schwannoma
Aneurysm
Arteriovenous malformation

Primary basicranial

Fibrous dysplasia
Osteoma
Osteoblastoma
Chondroma
Chordoma

Congenital

Cholesteatoma
Dermoid
Encephalocele.

Malignant skull base lesions are listed in the box on p. 3308. With some notable exceptions (leukemia, lymphoma, myeloma, metastases), malignant neoplasms are treated surgically, although in most cases surgery will not be used as the sole modality. Adjuvant treatments with radiation (by external beam, implantation, or brachytherapy) or chemotherapy are usually included in the therapeutic plan as well. Technically, malignant lesions are removed en bloc with margins of uninvolved tissue after broad circumferential exposure whenever possible (Sekhar and Janecka, 1991).

Box: Malignant lesions of skull base

Extracranial

Squamous cell carcinoma
Adenoid cystic carcinoma
Mucoepidermoid carcinoma
Malignant mixed tumor
Leukemia
Lymphoma
Rhabdomyosarcoma
Neurogenic sarcoma
Undifferentiated carcinoma
Hemangiopericytoma
Synovial sarcoma
Adenocarcinoma
Basal cell carcinoma

Intracranial

Esthesioneuroblastoma
Malignant schwannoma

Primary basicranial

Chondrosarcoma
Osteogenic sarcoma
Multiple myeloma
Histiocytosis X
Chordoma

Metastatic

Breast
Lung
Kidney
Prostate
Melanoma.

When considering surgery for tumors and tumorlike disorders of the cranial base, the first question that must be answered concerns the biologic behavior of the lesion. As mentioned previously, an accurate clinical diagnosis can often be made on the basis of information obtained from the history, physical examination, and imaging studies. This should be supplemented by a histologic diagnosis before definitive treatment plans are carried out.

When tumor is present within the nose, paranasal sinuses, middle ear, mastoid, oral cavity, pharynx, or neck, direct biopsy can be performed using standard techniques. When direct biopsy is not feasible, CT-guided needle biopsy may be done in selected cases, such

as tumors of the infratemporal fossa. Occasionally a tumor may be inaccessible by either of these routes, or biopsy without adequate skull base exposure may be judged unsafe because of concerns over injury to nearby critical structures or because of vascularity of the lesion. In these situations the surgeon must proceed with an operative approach to the skull base that is designed to provide access to the tumor for safe biopsy before any irreversible ablative steps are taken. Then, if the frozen-section biopsy result contraindicates resection or is questionable, an alternative treatment plan may be made. On histologic grounds alone, extirpative surgery is usually *not* performed when (1) a malignant lesion is metastatic from a distant source or (2) a malignancy is of a type that responds well to other treatment modalities (for example, lymphoma).

In addition to histologic criteria, the decision for or against surgical treatment is influenced by clinical considerations. The *expected morbidity of resection* can usually be reasonably predicted by the location and extent of the lesion together with the physiologic age and general medical status of the patient. This morbidity must be compared with the anticipated *natural course of the untreated lesion* and also with the *likely result of nonsurgical treatments* when applicable. The surgeon must also try to realistically assess the *likelihood of surgical cure or significant palliation.* All of these factors must be carefully weighe4d and discussed with the patient and family. Only then can an appropriate therapeutic plan be designed.

Anesthesia

Anesthetic management is a crucial factor in determining the outcome of cranial base operations. Techniques of neuroanesthesia are used with the primary goals of maximal neuronal preservation and simultaneous facilitation of a controlled surgical environment (Gonzalez and Khalouf, 1991). Maintenance of *hemodynamic factors* is a key element in this scheme because cerebral blood flow cannot be allowed to drop below critical levels for any significant length of time. Thus close monitoring of arterial pressure, central venous pressure, cardiac function, and urine output are all of paramount importance.

Electrophysiologic monitoring, including somatosensory evoked potentials (SSEP) to assess cortical function and electromyography (EMG) to assess motor cranial nerve function, is another key element in achieving neuronal preservation. Appropriate selection of anesthetic agengts and limited use of neuromuscular blocking drugs enhance the reliability of such monitoring.

Cerebral edema, a common problem in intracranial surgery, can be minimized by intraoperative use of colloids (albumin, plasma) rather than crystalloid fluids. It can also be minimized by controlled hyperventilation, which, by virtue of decreasing arterial PCO_2, causes mild cerebral vasoconstriction and a corresponding reduction in intracranial volume. Generally, PCO_2 between 25 and 30 mm Hg is desirable for this purpose. Lower PCO_2 levels begin to significantly reduce cerebral perfusion and are not recommended. Another technique used by the anesthesiologisgt that is helpful in reducing brain swelling is the preoperative placement by an indwelling lumbar drain, which decompresses the subarachnoid space by removing cerebrospinal fluid (CSF). Lumbar drains are also important postoperatively in selected cases because continuous, short-term CSF decompression can decrease the possibility

of CSF fistula. (These drains are not used in patients who have major intracranial space-occupying lesions because of the risk of brain stem herniation.) Cerebral edema may also be lessened by administration of steroids, which are especially helpful if ecdema is present preoperatively as a result of tumor or other mass lesions. Finally, diuretics such as furosemide or mannitol may be used to decrease brain swelling, but these are less desirable in view of their concomitant reduction of systemic intravascular volume.

The anesthesia team is also responsible for infusion of blood products to replenish surgical blood loss, which can be considerable in some cranial base procedures. Dilutional thrombocytopenia and other coagulopathies can occur after multiple transfusions of stored blood. These problems can be successfully treated by replacement of clotting factors (in the form of fresh frozen plasma) and platelets in proportion to red cell transfusion. Related issues with which the anesthesiologist is often concerned include the advance donation of autologous blood for transfusion and the employment of "cell saver" devices to collect blood intraoperatively and reinfuse it. With concerns over blood transfusion-related infectious diseases, these techniques are enjoying increasing popularity. One limitation of the cell saver is that it should not be used trhere is risk of reinfusing tumor cells from the operative field.

The anesthesiologist plays an essential role in successful surgery for cranial base lesions. Optimal anesthetic management depends on close communication between anesthesiologist and surgeons before, during, and after the operation.

Operative Techniques

General considerations

Overview of approaches

A wide variety of approaches have been developed for exposure of the anterior and middle cranial base regions, ranging from purely intracranial to purely extracranial. Most of the current approaches for dealing with lesions of the skull base employ combined intracranial and extracranial methods. Many of these techniques are still evolving. For anterior cranial base lesions, the most commonly used approaches combine frontal craniotomy with some form of transfacial (transnasal, transmaxillary, or transorbital) exposure. For middle cranial base lesions, access is most often provided by combining temporal or frontotemporal craniotomy with infratemporal fossa dissection, transfacial exposure, or transtemporal techniques. In both anterior and middle cranial base approaches, craniofacial disassembly techniques are being increasingly applied.

Implicit in the term *craniofacial disassembly* is the concept of systematic, stepwise dissection of cranial and facial soft tissues based on knowledge of regional vascular territories and functional anatomy, followed by osteotomies and dismantling of the craniofacial skeleton. Some of these techniques, developed originally by plastic and reconstructive surgeons for correction of congenital craniofacial deformities, have assumed a position of major importance in cranial base surgery because they allow wide exposure of the skull base through temporary displacement of the viscerocranium (Jackson et al, 1986; Tessier, 1967). The enhanced exposure of the skull base from below the neuraxis significantly reduces the need for brain retraction and therefore helps to minimize postoperative neurologic dysfunction. It also allows

the surgeon greater oncologic precision during the extirpative phase, with preservation of the functional and aesthetic units of the face for reconstruction (Nuss et al, 1991a).

Planning the operative approach

Whether the surgery is being performed for benign tumor, for malignancy, or for other indications (such as inflammatory or vascular lesions or CSF fistula), the approach must be planned (and executed) so as to accomplish four specific goals applicable to all cranial base operations.

Box: Goals of cranial base surgery

1. Extirpation of disease
2. Protection of vital structures
3. Restoration of critical anatomic barriers
4. Functional and aesthetic reconstruction.

First, the approach must provide adequate exposure to allow extirpation of the disease process. To a large extent the degree of needed exposure will be based on information obtained from the physical examination and imaging studies.

Second, the approach must be designed to protect critical structures in the vicinity of the lesion. This often means extending the exposure beyond the boundaries of the lesion itself to allow, for example, identification/mobilization of nearby cranial nerves, the ICA, orbital contents, or brain. Although obtaining this added exposure may increase the operative time, it is a major factor in reducing postoperative morbidity. Attainment of this goal is also enhanced by the appropriate use of electrophysiologic monitoring, including SSEP, ABR, and EMG (Sekhar and Janecka, 1991).

Third, the approach must be designed so that, at the completion of the extirpative phase, critical barriers between the neurocranium and viscerocranium can be readily and reliably restored. These barriers, particularly the dura and subjacent soft tissues, normally serve to effectively insulate the intracranial contents and the ICA from exposure to the aerodigestive tract below, including the nasal cavity, sinuses, eustachian tube, and pneumatic spaces within the temporal bone. Once disturbed, the barriers must be restored in order to reduce the potential for such consequences as CSF fistula, meningitis, and carotid artery rupture. Also, an approach should respect the vascular territories of local tissues such as the temporalis muscle, galea, and pericranium, which can then be used for the reconstruction. Such vascularized local flaps are in most cases preferable to free flaps or nonvascularized grafts (Janecka and Sekhar, 1991; Jones et al, 1987).

Fourth, the choice of operative approach should also reflect consideration for functional and aesthetic reconstruction, including placement of incisions within natural skin lines that respect "aesthetic units" of the face. The soft tissue closure should follow plastic surgery principles. Excellent skull base exposure can usually be achieved by elective osteotomies and temporary removal of craniofacial bone segments, which are subsequently replaced, preserving facial contour (Janecka et al, 1990, 1991a; Lauritzen et al, 1986; Nuss et al, 1991a). Even in cases where aesthetically important segments must be removed from

oncologic reasons, acceptable cosmesis can usually be accomplished through judicious use of bone grafts and soft tissue flaps or through surgical closures designed to support alloplastic or prosthetic materials.

Anterior cranial base approaches

Surgical approaches to the anterior cranial base include methods that are purely extracranial and those that utilize combined extracranial and intracranial exposures. The extracranial techniques - external ethmoidectomy, frontal sinusotomy, and intranasal ethmoidectomy - are suitable only for management of discrete, well localized lesions such as CSF fistulas and some very limited benign anterior cranial base tumors. These procedures have been well described elsewhere (Calcaterra, 1980; Montgomery, 1984).

The remaining majority of anterior cranial base lesions are best managed using the combined intracranial-extracranial techniques, of which there are basically two types: the *anterior craniofacial resection* and the *basal subfrontal approach.* Both of these approaches require bifrontal craniotomy for obtaining intracranial exposure. Except in special circumstances (for example, prior surgery, trauma), the most utilitarian incision for surgery of the anterior cranial base is the bicoronal incision.

Technical Note: The Bicoronal Incision

The bicoronal incision should be in the true coronal plane at the level of the top of the helix of the ear or slightly anterior to it (Fig. 190-11). A short, forward directed, preauricular extension can then be made on both sides to enhance scalp flap rotation. This coronal placement of the scalp incision is chosen for several reasons. First, it preserves the anterior branches of the superficial temporal artery, which in turn adds to the vascularity of the skin and enhances viability of the scalp flap. Second, it substantially increases the length of vascularized galea and pericranium available for reconstruction as compared with incisions made along the anterior hairline, midforehead crease, or brow.

The central portion of the anterior scalp flap (that is, the portion between the two superior temporal lines) is elevated in the subperiosteal (subpericranial) plane. Lateral to the two superior temporal lines, it is elevated in the plane just above the deep temporal fascia. Therefore, at the temporal lines, the pericranium must be sharply incised to separate it from the origin of the deep temporal fascia (see Fig. 190-14).

This deep temporal fascia (DTF) begins to split into superficial and deep layers beginning at approximately the level of the superior orbital rim (Abul-Hassan et al, 1986). These fascial layers then diverge to envelop the lateral and medial surface of the zygomatic arches inferiorly; between the layers is the temporal fat pad. The frontal (temporal) branches of the facial nerve course just superficial to the zygoma along the superficial temporal fascia (temporoparietal fascia) and are prone to injury if the dissection is done at that level (Fig. 190-12, A). Often these injuries can be avoided by maintaining the plane of dissection at the level of the deep layer of the DTF (that is, at the surface of the temporalis muscle itself). This deep plane of exposure essentially elevates the fat pad along with the superficial fascia and the superficial layer of the DTF, protecting the facial nerve branches (Stuzin et al, 1989) (see Fig. 190-12, B). Once the dissection reaches the level of the zygomatic arch, the arch is

palpated and its superior surface is directly exposed by sharply incising the fat pad and periosteum.

Further medially, flap elevation proceeds down the frontal area toward the supraorbital rims. Here, care must be taken to preserve the supraorbital vascular pedicles, which give blood supply to the galea and pericranium. To avoid injury to this pedicle, the elevation of the supraorbital rim periosteum begins laterally and proceeds medially. A fine elevator is used first to palpate the supraorbital foramen (or notch) and then to expose its margins. If the pedicle is completely surrounded by bone, a 3 mm osteotome is used to fracture the inferior margin of the foramen, liberating the pedicle and allowing it to be elevated intact along with the remaining pericranium and underlying periorbita (Fig. 190-13). This is done bilaterally to preserve maximum vascularity. If the entire skin flap is difficult to rotate inferiorly, the preauricular parts of the incision can be extended to as low as the tragus (without danger to the facial nerve trunk) or anteriorly (as far as the temporal hairline) to further enhance flap rotation.

Anterior craniofacial resection

Anterior craniofacial resection (Johns and Kaplan, 1986; Schramm, 1984; Schramm et al, 1979) (Fig. 190-14) combines bifrontal craniotomy with transfacial exposure of the nasal cavity, ethmoid, maxillary, and orbital areas, usually by modifications of lateral rhinotomy, midfacial degloving, or other transfacial approaches. Anterior craniofacial resection is most often used for management of neoplasms that originate in the sinonasal tract and invade the anterior cranial fossa floor, such as squamous cell carcinoma, esthesioneuroblastoma, and adenocarcinoma. In cases where tumor invades the soft tissues of the orbit, the approach is extended to include orbital exenteration.

The operation begins with transfacial exposure, followed by bifrontal craniotomy performed by the neurosurgeon. If the patient has a large frontal sinus, an osteplastic flap may be elevated to avoid burr holes in the forehead area, which could later become aesthetically unsatisfactory. In such cases the thin posterior table of the frontal sinus is opened with a diamond burr to expose the dura and then removed. If the frontal sinus is small, then a frontal bone flap is developed using a guarded craniotome introduced via burr holes placed above the hairline or in the temporal areas. Near the midline, care is taken to separate the dural fold containing the sagitall sinus away from the bone to protect it before the craniotome is allowed to cross the sagittal plane. The lower horizontal bone cut should be kept low (within 1 cm of the superior orbital rims) to lessen the need for subsequent brain retraction. Withdrawing 25 to 50 mL of CSF from the lumbar subarachnoid catheter, lowering PCO_2 through controlled hyperventilation, and occasionally administering mannitol or steroids further reduce the need for mechanical frontal lobe retraction.

Next, the frontal lobes are elevated from the anterior cranial fossa floor by incising the dura and severing the olfactory nerves at the cribriform plate. This always results in some leakage of CSF, which is controlled by direct dural suture or a dural patch (from temporal fascia, fascia lata, or pericranium). The dural closure should be as close to watertight as possible. Dura is further elevated to expose the orbital roofs and planum sphenoidale and finally the base of the anterior clinoid processes.

If disease extirpation mandates removal of the planum, the intracranial portions of the optic nerves should be exposed through unroofing of the optic canal in order to protect them from injury at the time of sphenoid osteotomy (Sekhar and Janecka, 1991). If the disease is confined to the cribriform area, however, the planum may be entered with a cutting burr or osteotome (Schramm, 1984) to establish the posterior bony marging without optic nerve decompression. This completes the intracranial portion of the exposure for anterior craniofacial resection.

The transfacial exposure often utilizes modifications of a lateral rhinotomy incision, which may or may not transect the upper lip. This depends on whether a total maxillectomy will be done in conjunction with the resection. The periosteum is elevated from the nasal bone as well as from the medial and inferior surfaces of the orbit. The nasolacrimal duct is identified and transected distally. The anterior and posterior ethmoid arteries are then identified and cauterized or clipped. In most cases it is necessary to perform a complete en bloc ethmoidectomy. For this purpose a contralateral Lynch incision is made to elevate the contralateral periorbita, cauterize the anterior and posterior ethmoid vessels, and make the appropriate osteotomies using a sagittal saw. If preoperative imaging studies or intraoperative observations confirm the presence of tumor within the soft tissues of the orbit, then orbital exenteration may be facilitated by extending the skin incision laterally to include a portion of the eyelids. Depending on the nature and extent of the tumor, osteotomies may also be made to include part or all of the maxilla as well.

At this point the sagittal saw is used to create the osteotomies of the cranial floor. The frontal lobes are retracted or protected from the saw by inserting an appropriate malleable retractor. The reciprocating saw is introduced through the nasal and ethmoid exposures, and under direct vision osteotomies are created from the planum sphenoidale, along the ethmoid roof, forward to the front of the cribriform plate. These osteotomies may or may not include the "supraorbital bar" of bone, which is a portion of the frontal bone between the supraorbital rims (see Fig. 190-14, D). If the tumor extends anteriorly to a significant degree, this supraorbital bar is removed with it as a single specimen. After removal the specimen can then be closely inspected. If the supraorbital bar is not actually involved by tumor, it may be detached from the specimen and replaced for reconstruction at the completion of the operation.

Once the resection margins have been verified by frozen section and are negative, reconstruction begins. As previously mentioned, it is critical that the dura be closed in a watertight fashion. This usually involves placement of a patch of pericranium, temporal fascia, or fascia lata, followed by placement of a vascularized pericranial flap over the defect in the floor of the anterior cranial fossa. The pericranial flap is developed by sharp dissection from the previously reflected scalp flap (Snyderman et al, 1990) (see Fig. 190-14, F). Elevation of this flap of pericranium is continued down to the level of the glabella, taking care not to injured its vascular pedicles. It is then rotated intracranially and positioned to form a bridge of soft tissue across the orbital roofs and back to the planum sphenoidale. If necessary, the distal end of the pericranial flap may be sutured to the dura over the planum for security. This provides a vascularized tissue barrier between the dura above and the nasal cavity below (see Fig. 190-14, G and H). Unless a large amount of anterior cranial fossa bone has been resected and concern for brain herniation exists, it is usually not necessary to place a bone graft across the bony defect. Also, it is usually not necessary to place a skin graft on the undersurface of

the pericranium (facing the nasal cavity), since this tissue has been shown to "mucosalize" readily on its nasal surface. Once the pericranial flap is in place, the spinal drain is clamped so that no further intraoperative CSF decompression will take place. This will allow gradual reexpansion of the brain to make contact with the pericranial flap, obliterating any residual dead space. Because the pericranial flap traverses the frontal sinus, it is necessary to obliterate the frontonasal ducts and to remove all mucosa from within the sinus. A small amount of fat or free muscle may be packed into the ducts in order to then obliterate any dead space inferiorly. The flap then covers the frontonasal duct orifices and sinus floor. If the sinus is small, it may be obliterated by packing with abdominal fat. If the sinus is quite large, however, it may be advisable to remove the posterior table completely and allow the brain and dura to expand and fill the space (cranialization of the frontal sinus).

In closing the facial incision, the medial canthal ligament is identified and secured to the remaining medial orbital wall. The upper and lower lid canaliculi may be intubated with a canalicular stent in order to prevent dacryostenosis. Before further closure, several tacking sutures are placed to secure the frontal dura to the margins of the craniotomy site. This will help to prevent postoperative epidural fluid and blood collections. The bifrontal craniotomy bone flap is then replaced and secured according to the surgeon's preference. This may be done with wires, plates, or sutures. (If the patient will subsequently require radiation or repeated imaging studies, it is desirable to avoid the placement of radio-dense materials that will lead to radiation scatter or imaging voids.)

Basal subfrontal approach

The basal subfrontal apprach or transbasal approach (Fig. 190-15) (Derome, 1977; Sekhar and Janecka, 1991) is in many ways similar to the anterior craniofacial resection operation except that the transfacial exposure is less extensive. Because the target area in this approach is more posterior (sphenoid and clivus) than in the anterior craniofacial resection (ethmoids and cribriform), the craniotomy bone flap is generally somewhat larger, and the orbital bone cuts are broader. This approach is used for lesions that primarily or secondarily involve the bony cranial base in the regions of the sphenoid body and upper clivus, such as meningiomas, fibrous dysplasia, chordomas, chondrosarcomas, and ossifying fibromas (Derome and Visot, 1987).

This approach also begins with a bicoronal incision. After exposing the orbital rims, periorbita is elevated from beneath the orbital roofs and medial walls in preparation for osteotomy. The anterior and posterior ethmoid arteries are identified bilaterally as landmarks but need not be divided since the axial ethmoid osteotomy can be made just above this level. Bifrontal craniotomy is then performed, and dura is elevated from above the orbital roofs and cribriform areas as previously described. Using malleable retractors to protect the orbital contents and brain, the reciprocating saw is used to create osteotomies that result in temporary removal of both orbital roofs and the supraorbital bar (see Fig. 190-15, A).

The coronal osteotomies along the posterior orbital roof should be made as far posteriorly as possible to simplify reconstruction, by conserving orbital contour, and to prevent postoperative pulsatile exophthalmos. These orbital cuts can be made as far back as the posterior ethmoid foramen but must be made only under direct vision, from both intracranial and extracranial perspectives. With the use of these osteotomies, a very broad and

basal exposure of the entire anterior cranial base is achieved (see Fig. 190-15, B). The neurosurgeon, working with the aid of the microscope, completes the approach by rongeuring or drilling a small amount of bone remaining posteriorly to unroof the optic nerves, superior orbital fissures, and sphenoid sinus. Extirpation then proceeds as required by the tumor, followed by reconstruction, which is similar to that done for anterior craniofacial resection.

Middle cranial base approaches

In contrast to the anterior cranial base, surgical approaches to the middle cranial base are quite numerous and varied. This reflects the anatomic complexity of the region as well as the diversity of lesions found there. The following discussion reviews a number of approaches and presents examples of those that we have found most useful. Approaches that have been well described elsewhere will have only specal major points highlighted.

Division into central and lateral compartments

When considering surgical approaches to the middle cranial bone, it is helpful to subdivide the region into a single *central compartment* and paired *lateral compartments* (Fig. 190-16) (Krespi and Sisson, 1984). Viewing the skull from inferiorly, the *central compartment* may be defined as that area between two parasagittal lines drawn from the medial pterygoid plate to the occipital condyle on each side. These lines correspond approximately to the pathways of the ICAs through the skull base. Thus the central compartment consists of the pituitary fossa, the sphenoid rostrum and lower sphenoid sinus, the nasopharynx, the pterygopalatine fossa, and the lower portion of the clivus. (The lower clivus is actually part of the occipital bone and not technically part of the middle cranial base. However, the surgical approaches to this area and also to the craniovertebral junction and upper cervical spine are essentially extensions of the approaches to the central compartment, and these areas will be addressed here as well.)

The *lateral compartment,* then, includes the entire infratemporal fossa, the parapharyngeal space, and the petrous portion of the temporal bone. As such, it constitutes an anatomic space of singular importance because of the high density of neural and vascular structures within, including (extratemporally) the ICA, internal jugular vein, maxillary nerve (V-2), mandibular nerve (V-3), facial nerve (VII), cranial nerves IX, X, XI, and XII, sympathetic nerves, and major branches of the external carotid artery, as well as (intratemporally) the cochlea, vestibule, and cranial nerve VIII.

These distinctions between central and lateral compartments are valid in the sense that limited lesions within their respective boundaries will be clearly more amenable to resection using one type of approach or the other. However, it is not unusual for advanced lesions to extend beyond the line separating central from lateral compartment, and therefore the surgical approach will have to be tailored to suit each specific clinical situation.

Approaches to central compartment

The major surgical approaches to the central compartment of the middle cranial base are shown in Table 190-1, along with the anatomic areas where they provide useful exposure. Excluding pituitary surgery, all of these approaches are used primarily for management of

extradural lesions along the skull base.

Although most of the central compartment approaches described in literature can give access to the sphenoid sinus, not all are suitable for pituitary or parasellar surgery because of indirect exposure or oblique angle of entry into the sinus, which increases the risk of injury to adjacent neurovascular structures. When lesions are limited to the sphenoid sinus or pituitary fossa, the *transethmoidal-sphenoidotomy* (Fig. 190-17) (Kirchner, 1984) and the *transseptal-sphenoidotomy* (Fig. 190-18) (Hardy, 1971; Sasaki, 1984) approaches are usually satisfactory, safe, and quite effective. In addition, these approaches are useful for obtaining biopsy specimens of more extensive lesions involving that region. For larger lesions involving adjacent areas, however, the exposure is inadequate because the operative field is deep and narrow, requiring use of the operating microscope in most cases.

The *lateral rhinotomy* (Fig. 190-19) (Schramm and Myers, 1978) and *transantral* (Fig. 190-20) (Hamberger et al, 1961) procedures afford wider access to the anterior sphenoid and adjacent nasopharynx, pterygopalatine fossa, maxilla, and ethmoid regions. However, they are generally not satisfactory for extirpation of any but the smallest lesions situated in the sphenoidal-clival area.

The *midfacial degloving approach* (Fig. 190-21) (Price, 1986) is more suitable for dealing with larger central compartment lesions because it allows improved midline access through the nose and both maxillary sinuses. Medial maxillectomy and resection of the ascending process of the palatine bone are included if necessary, giving the surgeon good visualization of the nasopharynx and adjacent skull base. The *LeFort I osteotomy* (Fig. 190-22) (Bell, 1975; Brown, 1989) can be an alternative method for reaching these areas (from a slightly more inferior angle), or it may be used as an adjunct to midfacial degloving, offering additional access to the oronasopharynx and clivus by displacing the hard and soft palate inferiorly.

For lesions of the clivus that also involve the upper cervical vertebrae (craniovertebral junction (CVJ)), *mandibulotomy* (Fig. 190-23) (Biller and Lawson, 1986; Biller et al, 1981; Krespi and Sisson, 1984) will afford a paramedian route to reach the lower areas of extension. The *transpalatal* (Fig. 190-24) (Jenkins and Canalis, 1984; Kennedy et al, 1986) and *transoral* (Fig. 190-25) (Crockard, 1985; Fang and Ong, 1962) approaches are also used for management of lesions at the CVJ, and these may be used concomitantly with mandibulotomy if necessary. A significant benefit of such a combined approach is that, with mandibulotomy, it is also possible to expose the parapharyngeal space and therefore to safely dissect along the carotid artery from the neck up to its entrance into the temporal bone (Biller and Lawson, 1986; Biller et al, 1981; Krespi and Sisson, 1984). This advantage is particularly important when removing tumors that arise primarily in the central compartment but have extended laterally. (Conversely, for lesions originating in the lateral compartment and extending centrally, the infratemporal fossa approach (Fisch, 1977) or one of its modifications can be used. These approaches, along with the facial translocation approach (Janecka et al, 1990), which is suitable for lesions in both compartments, will be discussed in the following section.)

An alternative approach recently developed for removal of extensive lesions of the clivus and CVJ is the *extended maxillotomy* operation or its modification, the *subtotal maxillectomy* (Fig. 190-26) (Cocke et al, 1990). In general, these innovative techniques combine the advantages of many of the above approaches. They afford wide exposure of the central compartment by unilaterally displacing or resecting the hemimaxilla.

For very extensive clival and CVJ tumors, when bilateral transmaxillary access is needed, the *midfacial split approach* (Fig. 190-27) can be employed (Janecka et al, 1991a). This approach provides access to the entire central skull base in a unified surgical field that can extend vertically from the anterior fossa floor to the level of the second cervical vertebra (and lower, if combined with mandibulotomy) and horizontally from jugular fossa to jugular fossa. This exposure is achieved using craniofacial disassembly to completely displace the midfacial skeleton, including both maxillae, orbital floors, and palate. It is especially useful for management of large tumors originating in the central compartment that have also involved the adjacent anterior cranial base, CVJ, or lateral compartment.

Approaches to lateral compartment

Numerous approaches have been developed and advocated for obtaining surgical access to the lateral compartment of the cranial base. Despite this apparent diversity of techniques and the sometimes confusing terminology by which they are described, considerable overlap exists between them, and most of the currently used approaches arrive at the cranial base via one (or more) of four main avenues: transtemporal, infratemporal, transfacial, and intracranial. These "avenues" refer to the primary direction or orientation from which the surgical exposure is ultimately achieved. In most cases this orientation will influence to some extent the utility and morbidity of the operation. The cranial base surgeon must be familiar with the advantages and limitations of each of these four routes. For extensive lesions it often becomes necessary to combine approaches.

Transtemporal approaches. The *transtemporal routes* include the *transcochlear* (House and Hitselberger, 1976), *translabyrinthine* (Glasscock et al, 1978; House, 1964), and combined (Jackson et al, 1991) approaches. These are lateral, primarily extradural techniques that traverse the mastoid and petrous portions of the temporal bone to provide exposure of lesions of the petrous apex, clivus, and cerebellopontine angle. The anterior limit of purely transtemporal approaches is the intrapetrous course of the ICA, and they therefore give only limited access to the middle cranial base. As such, they are more often used in the management of tumors of the posterior cranial base (such as acoustic neuromas, petroclival meningiomas, and aggressive cholesteatomas) and will therefore be discussed in more detail in Chapter 191. With respect to the middle cranial base, the transtemporal approaches are useful mainly as adjunctive techniques in combination with craniotomy, infratemporal, or transfacial approaches. They can be used to enhance exposure of advanced lesions of the middle cranial base that have secondarily extended into or beyond the petroclival region and posterior to the course of the ICA. Postoperative consequences include permanent unilateral deafness with or without dysequilibrium caused by functional loss of the middle ear structures and facial paralysis of variable degree and duration caused by seventh nerve decompression or transposition.

Infratemporal approaches. The *infratemporal* routes to the lateral compartment include the infratemporal fossa, lateral transparotid, extended rhytidectomy, lateral transtemporal-sphenoid, lateral facial, and subtemporal-infratemporal approaches.

The *infratemporal fossa approach* of Fisch (Fisch, 1977; Fisch and Pillsbury, 1979; Fisch et al, 1984) encompasses three distinct variations for use in specific clinical situations (Fig. 190-28). The type A approach provides exposure between the sigmoid sinus and the condylar fossa, designed to reach to the petrous apex and infralabyrinthine areas. It is most useful for management of cholesteatomas, meningiomas, and glomus tumors of those regions. The type B approach allows access from the sigmoid sinus to the petrous tip (including exposure of the horizontal ICA and foramen ovale) to reach lesions of the clivus, such as chordomas, meningiomas, and extensive apex cholesteatomas. The type C approach expands this access to include the parasellar region, the cavernous sinus, foramen rotundum, and foramen lacerum. Removal of the pterygoid plates in this approach also facilitates access to the nasopharynx. This type C approach is used in the resection of small nasopharyngeal carcinomas, adenoid cystic carcinomas, and angiofibromas. All three variations of the infratemporal fossa approach involve mastoidectomy, facial nerve dissection (and transposition), and obliteration of the eustachian tube, middle ear, and external auditory canal with resultant permanent conductive hearing deficit. Thus the types A, B, and C infratemporal fossa approaches share some features of the basic transtemporal approaches and yet extend the field of access anteriorly to reach the middle cranial base by virtue of exposing and controlling the petrous ICA (see Chapter 193).

The *transparotid approach* (Fig. 190-29) (Panje and Pitcock, 1991) is most useful for dealing with tumors of the parapharyngeal space and infratemporal fossa that originate in or secondarily involve the parotid gland. It begins by mobilizing the parotid gland off the sternocleidomastoid muscle to expose the posterior belly of the digastric muscle and the styloid complex (stylohyoid and stylopharyngeus muscles and stylohyoid ligament). The facial nerve is then identified extratemporally either at the stylomastoid foramen or by following one of its peripheral branches proximally, depending on the type and location of the tumor. Parotidectomy is then performed, preserving the facial nerve, and the infratemporal fossa is accessed by dividing the digastric muscle and styloid complex and retracting the mandible forward. For larger tumors it may be necessary to also perform mandibulotomy or dissection of the neck (for control of the ICA, internal jugular vein, and cranial nerves IX to XII). These maneuvers allow wider access and thereby facilitate safe dissection of tumor from nearby nerves and major vessels. An extension of the transparotid approach has also been described that essentially combines this exposure with a transtemporal approach (Panje and Pitcock, 1991). This extended approach provides better exposure for lesions that originate in the infratemporal fossa or pterygoid space and secondarily ivnolve the temporal bone, such as schwannomas of the lower cranial nerves or various mesenchymal tumors.

The transparotid approach and its extensions require dissection of the facial nerve, which may be time consuming and which carries the small but important risk of facial paralysis or paresis. The *extended rhytidectomy approach* (Fig. 190-30) (Panje and Pitcock, 1991) has been proposed as an alternative approach that avoids direct facial nerve dissection and instead exposes the infratemporal fossa by first elevating a skin flap based anteriorly (as in rhytidectomy) and then elevating the mimetic muscles, facial nerve, and parotid gland as a unit based posteriorly, uncovering the facial skeleton. This procedure has also been termed

the *facial biflap procedure.* The infratemporal region can then be entered by way of mandibulotomy and retromaxillary dissection.

The transparotid and extended rhytidectomy approaches may be used for dealing with tumors that, by origin or extension, involve the more inferior portions of the infratemporal fossa, the pterygoid space, nasopharynx, parapharyngeal space, and posterior maxilla. For lesions of the middle cranial base that do not involve these lower regions to a great extent, the *lateral facial approach* (Fig. 190-31) (Gates, 1988) and the *lateral transtemporal sphenoid approach* (Fig. 190-32) (Holliday, 1986) have been described. Both of these procedures reach the infratemporal fossa from a slightly more superior direction by displacing the zygomatic arch inferiorly and reflecting the temporalis muscle. The greater wing of the sphenoid is then followed to the lateral pterygoid plate, which is used as a surgical landmark as outlined earlier. Both of these approaches also incorporate partial removal of the greater wing of the sphenoid, that is, they include subtemporal craniectomy and extradural dissection. The advantages of these procedures are that they require no facial nerve dissection (the temporal branch of cranial nerve VII is retracted inferiorly with the skin flap), and they allow some access to the cranial base from both intracranial and extracranial perspectives.

The lateral facial approach is quite useful as a straight-forward approach to a limited area of the middle cranial base that is technically simple to perform (once the anatomy is well understood) and that incurs relatively little morbidity. It is applicable in the management of small- to medium-sized benign tumors with limited intracranial extension, such as occurs with some angiofibromas. The similarly oriented lateral transtemporal sphenoid approach can be extended so that it is significantly more complex than the lateral facial approach but can afford additional exposure as well, making it useful for management of lesions in the clivus, parasellar region, nasopharynx, petrous apex, and infratemporal fossa. As the name implies, the lateral transtemporal approach is really a combination of techniques, not unlike the infratemporal fossa approaches of Fisch except that it does not usually result in permanent conductive hearing loss and does not require rerouting of the facial nerve.

The *subtemporal-preauricular infratemporal approach* (Fig. 190-33) (Sekhar et al, 1987) is basically a combination of the transparotid and lateral transtemporal sphenoid procedures in addition to temporal craniotomy. As such, it provides excellent exposure of much of the middle cranial base and allows improved access for intracranial dissection when necessary. The orientation of the exposure begins laterally and progresses medially so that it is quite useful for the management of lesions of the greater sphenoid wing, petrous apex, and middle and lower clivus. Together with our neurosurgical colleagues, we have used this approach extensively for resection of tumors such as meningiomas, chondrosarcomas, chordomas, and petrous apex cholesteatomas. Because it illustrates many of the important techniques used in surgery of the middle cranial base, it will be described in detail.

A hemicoronal or curved temporal incision is extended into the preauricular and cervical regions to expose the temporalis muscle, zygomatic arch, parotid gland, and upper neck. (As discussed in the description of the bicoronal incision, the temporal fascia and fat pad are elevated with the skin flap, protecting the frontal branch of the facial nerve.) The cervical extension is used to gain access to the ICA, internal jugular vein, and cranial nerves IX to XII and also to help identify the facial nerve extratemporally. The parotid gland is mobilized away from the sternocleidomastoid muscle, and the underlying styloid complex is

divided, revealing the high cervical ICA, internal jugular vein, and cranial nerves IX to XII. Next, the zygomatic arch is osteotomized with a reciprocating saw and removed temporarily allowing the temporalis muscle to be reflected inferiorly to expose the greater wing of the sphenoid and the underlying infratemporal fossa. In cases where the lesion also involves the orbit (such as sphenoid wing meningiomas), the lateral orbital wall and rim may also be osteotomized and removed along with the zygoma. The condyle of the mandible may then be either retracted or resected to expose the spine of the sphenoid and its nearby structures, including the middle meningeal artery, cranial nerve V-3, and the ICA at its entrance into the carotid canal. More medially, cranial nerve V-2 can be identified just anterior to the lateral pterygoid plate.

A low temporal craniotomy is then performed, and by extradural dissection the temporal lobe is elevated off the middle fossa floor, revealing the intracranial landmarks: GSPN, middle meningeal artery, and cranial nerves V-2 and V-3.

By progressive removal of the greater wing of the sphenoid, cranial nerves V-3 and then V-2 may be completely unroofed. If necessary, the sphenoid sinus may be openmed by removing the bone between cranial nerves V-2 and V-3, as well as the base of the pterygoid plates. The superior orbital fissure may also be unroofed in the same way.

The intrapetrous course of the ICA may then be exposed beginning at the infratemporal opening of the carotid canal and extending to just above the foramen lacerum (where the ICA enters the cavernous sinus). This is accomplished with a high-speed drill and a diamond burr using a technique analogous to that used for facial nerve decompression. To facilitate this exposure, the middle meningeal artery and GSPN must first be divided, and the high cervical ICA with its fibrous periosteal sheath must be rendered accessible by removal of the styloid process and surrounding bone. The ICA may then be displaced forward out of its bony canal. This is useful whenever the lesion involves the clivus medial to the ICA or the petrous part of the temporal bone posterior to the ICA. The inferior aspect of the cavernous sinus may also be approached from this exposure (Sekhar et al, 1989).

Of course, the exact sequence and extent of exposure at the skull base depend on the nature of the lesion to be extirpated. Certain steps in the procedure may be eliminated or modified to suit the needs of the particular case. For example, it is only necessary to dissect the neck if the procedure will benefit from proximal control of the ICA or from low exposure of the internal jugular vein or cranial nerves IX to XII. Otherwise, the cervical extension may be avoided.

Once the lesion has been extirpated, the wound is irrigated with antibiotic solution and carefully inspected. Any dural openings are meticulously repaired either primarily or using grafts of pericranium, temporalis fascia, or fascia lata. In addition to repairing dura, every effort is made to reconstitute a functional barrier between the intracranial compartment and the visceral spaces of the head and neck. Usually this does not include any attempt to rebuild the bony basicranium. Instead, efforts are focused on the interposition of reconstructive soft tissues - vascularized tissues whenever possible - into the surgical defect.

Any communications with the aerodigestive tract are identified. These will most often include the sphenoid sinus, nasopharynx, and eustachian tube. If the sphenoid sinus has been opened, its mucosa is removed and it is obliterated with autogenous fat or by transfer of vascularized temporalis muscle medially. If the eustachian tube has been entered, it is denuded of mucosa, packed with fat or muscle, sutured closed, and covered with vascularized temporalis muscle. If the nasopharynx has been violated, the opening is filled with either a temporalis muscle flap or free microvascular flap (for example, rectus abdominis), depending on the size of the defect. Special care is taken to ensure that the ICA is covered with vascularized tissue throughout its course, particularly in cases where the nasopharynx has been entered. Failure to protect the ICA in this way may result in continued exposure of the artery to bacterial contamination from the upper aerodigestive tract, with subsequent carotid rupture.

Finally, the zygomatic and craniotomy bone segments, previously displaced by osteotomy, are returned to their anatomic positions and secured appropriately. (Again, because of the concerns over postoperative imaging quality and therapeutic radiation "scatter", radio-dense materials are avoided whenever possible. Stable fixation of craniotomy bone flaps and zygomatic bone segments can be achieved using multiple 2-0 braided monofilament sutures passed through holes drilled on either side of osteotomy sites. Alternatively, titanium miniplates may be used.) If the temporalis muscle has not been used as a reconstructive flap, it is returned to the temporal fossa. If a temporalis flap has been used, the temporal fossa may be filled with autologous fat from the abdomen or thigh to minimize the temporal fossa depression. Soft tissue closure then followes. Usually a subgaleal suction drain is placed; this is removed in 24 to 48 hours.

Transfacial approaches. Transfacial exposure of the cranial base, particularly of the lateral compartment - middle cranial base region, is not a new concept. Operative procedures for removal of neoplasms from this region were described by several authors in the 1960s (Fairbanks-Barbosa, 1961; Tertz et al, 1969). Unfortunately, these early procedures - used initially for radical resection of malignant tumors - resulted in unacceptably high morbidity and mortality as well as considerable deformity, largely ebcause methods to adequately reconstruct major cranial base defects were not yet available. With the subsequent introduction of craniofacial disassembly techniques and vascularized reconstructive flaps, it has become possible in recent years to dismantle the facial skeleton, to extirpate deep-seated lesions, and to perform functional reconstruction to an extent not previously possible. As a result, lesions of the lateral and central compartments are more amenable to surgical treatment.

A transfacial approach to this region is desirable because it eliminates the viscerocranial skeleton as an obstacle to exposure, thus "opening up" not only the entire infratemporal fossa but also the nasal cavity, nasopharynx, pterygopalatine fossa, and sphenoid region to direct access all at once. The *facial translocation approach* (Fig. 190-34) (Janecka et al, 1990) is such a procedure; we have used it extensively and will describe it in detail.

Incisions are made in the face and scalp as shown in Fig. 190-34, A. The horizontal incision connects a lateral rhinotomy with a hemicoronal incision to create superior and inferior soft tissue flaps. (At this stage the frontal branches of the facial nerve are identified using evoked EMG, tagged, and then divided, to be reconnected later in the procedure. This horizontal incision is a key element in the exposure because it allows a single, unified surgical field unhampered by the need to alternatively work from separate cranial and facial incisions

that limit the surgeon's ability to develop three-dimensional exposure at the cranial base.) The soft tissue flaps are then elevated in the subperiosteal plane from the zygoma and maxilla. Periorbita is likewise elevated from the lateral, inferior, and medial orbital rims and walls. A reciprocating saw is then used to create osteotomies, and the orbitozygomaticomaxillary skeletal segment is temporarily removed.

After inferiorly reflecting the temporalis muscle, the subtemporal skull base is fully exposed, and further dissection proceeds as dictated by the lesion at hand, using the same skull base landmarks as in other approaches. Intracranial exposure is readily obtained if needed by subtemporal craniectomy or by frontal and/or temporal craniotomy. Similarly, the pterygoid plates and muscles may be removed, revealing the nasopharynx, sphenoid sinus, and clivus. As mentioned previously, facial translocation may therefore also be used as an approach to the central compartment of the middle cranial base. Thus it provides a unified surgical field with access to both compartments.

At the completion of the extirpation, reconstruction follows the same principles discussed in the preceding section. After dural repair the temporalis muscle is used to fill the skull base defect. Because of its appreciable soft tissue bulk, the muscle is also useful for obliterating the maxillary sinus space, where it serves as a vascularized tissue bed for the orbitozygomaticomaxillary bone segment, which is then replaced and secured as a free bone graft. Bony stabilization is achieved with sutures or miniplates. If it has not been resected for oncologic reasons, the infraorbital nerve (which was transected during development of the lower skin flap) is rerouted through its maxillary canal and reconnected to its distal trunk by suture neurorrhaphy. The frontal branches of the facial nerve are also repaired. The nasolacrimal system is stented to prevent dacryostenosis, and the medial canthal ligament is secured to the lacrimal crest. Temporary tarsorrhaphy helps support this repair during the early postoperative period. Closure of skin incisions completes the procedure.

Postoperatively, tarsorrhaphy sutures may be removed within 1 week; nasolacrimal stents remain in place for 6 to 8 weeks. In the majority of patients, infraorbital sensation returns within 3 to 6 months, and frontalis muscle function resumes in 6 to 9 months.

The facial translocation technique, as described here in its fully developed form, is valuable for obtaining the broad exposure needed in managing large or advanced lesions of the lateral and central compartments of the middle cranial base, especially those in which much of the lesion is extracranial. Because of its modular nature, it can also be applied in a variety of scaled-down versions tailored for dealing with smaller cranial base lesions.

Intracranial approaches. *Temporal craniotomy* is the traditional, standard neurosurgical method for gaining access to the middle cranial fossa. For intradural lesions within the temporal lobe or for extradural lesions along the lateral convexity of the middle fossa, it is the most direct approach (Maxwell and Chou, 1988). For lesions at the cranial base, however, temporal craniotomy alone is often less than ideal because it requires (by virtue of its superolateral-to-inferomedial orientation) significant retraction or, in some cases, resection of brain in order to reach a target area. Such brain manipulation can result in intraoperative and postoperative cerebral edema; temporal encephalomalacia; and deficits of speech, memory, and cognition; as well as long-term risk of seizure disorder (see section on postoperative concernes). These risks increase as the location of the lesion becomes more

medial and more temporal lobe retraction is required.

Still, temporal craniotomy alone is useful for some limited lesions of the lateral compartment - middle cranial base region, such as meninigiomas of the sphenoid wings. Risks of intracranial complications can be minimized by designing the craniotomy bone flap as low as possible, thereby giving a more basal and direct access to the skull base with less need for brain retraction. Naturally, the limiting factors here are the petrous ridge of the temporal bone and the zygomatic arch. When preoperative imaging suggests that these structures will be impediments to adequate exposure, a wider approach should be chosen, such as the subtemporal-infratemporal, lateral facial, or transtemporal sphenoid approach.

The *middle fossa approach* of House (House, 1961) utilizes temporal craniotomy as a preliminary step to reach the internal auditory canal by removal of bone from the superior surface of the petrous portion of the temporal bone. Because most lesions in these areas originate in relation to the seventh and eighth cranial nerves, this approach will be considered in more detail in Chapters 183, 185, and 191.

When the skull base is involved only to a minimal extent and the lesion is mostly infracranial, a *subtemporal craniectomy* may be done (see Figs. 190-31 and 190-34). In this procedure only a limited area of cranial bone is removed, generally as a safe means of establishing a superior margin when resecting, for example, the base of the pterygoid plates and surrounding soft tissue. Subtemporal craniectomy may be done by inserting a Kerrison or similar rongeur extradurally inside the foramen ovale and gradually removing bone in the direction of the foramen rotundum. The resulting bone window allows protection of the middle fossa dura under direct vision while removing the subjacent lesion.

For many lesions of the lateral compartment of the middle cranial base, temporal craniotomy and subtemporal craniectomy are used adjunctively, in combination with transtemporal, infratemporal, or transfacial exposure. These intracranial approaches provide an added dimension of control over the neural and vascular structures in the region. As in all transcranial operations for cranial base lesions, close cooperation between otolaryngologist and the neurosurgeon is essential.

Postoperative Concerns

The postoperative course in cranial base surgical patients is critical in determining the ultimate treatment outcome. Because of the magnitude of the surgery and the frequent need for manipulation of major neural and vascular structures, a variety of problems ranging from the trivial to the potentially lethal must be anticipated. Postoperative management must therefore include a concerted effort from all members of the cranial base team directed at three interrelated goals: prevention of foreseeable complications, early detection of problems when they arise, and an aggressive approach to intervention. Obviously, some consequences of surgery are predictable based on preoperative symptoms, signs, and imaging studies. Accordingly, patients and their families should be carefully counseled preoperatively regarding expected deficits, deformities, or disabilities. Although some complications are not fully predictable, it is still important for the patient and family to be well aware of the potential for such problems and also to be emotionally prepared to deal with trouble when it arises.

Initial postoperative care

In view of the potential for complications in cranial base surgery, patients must be intensively monitored and cared for postoperatively. All patients are initially admitted to an intensive care unit staffed by nurses experienced in neurosurgical and head-and-neck-surgical care. Consultants in critical care medicine, internal medicine, and other specialties actively participate in management when appropriate. Typically, patients will have continuous recording of the electrocardiogram, oxygenation (via pulse oxymetry), and systemic blood pressure (via arterial catheter). In specific circumstances, Swan-Ganz catheters or central venous lines are also used to assess cardiovascular status and fluid balance. These parameters are crucial in maintaining optimal intravascular volume, cardiac output, and cerebral blood flow.

Many cranial base operations require extended periods of anesthesia and involve considerable fluid administration and blood component replacement. For these reasons, close monitoring of hematologic and chemical parameters is essential (hemoglobin, hematocrit, platelets, prothrombin and partial thromboplastin time; sodium, potassium, calcium, magnesium, phosphorus, and serum osmolality). Appropriate replacement of blood components maintains oxygen delivery (red cells) and prevents coagulopathy (platelets, plasma). Electrolyte balance is especially important since deviation from physiologic norms can lead to confusion, agitation, stupor, or seizure activity, all of which might be otherwise mistaken for surgery-related neurologic insults.

Pharmacologic therapy plays an important role in prevention of complications. Antibiotics are routinely given, beginning just before surgery and for a duration of 24 to 48 hours afterward. Inhibitors of gastric acid secretion are given postoperatively to reduce the chance of stress ulceration and gastrointestinal bleeding and are continued until the patient is tolerating adequate enteral nutrition. Anticonvulsants are used to prevent seizures whenever significant frontal or temporal lobe manipulation has been necessary. Once instituted, anticonvulsant therapy should be periodically monitored using serum drug levels and should be continued for at least 6 to 12 months after surgery. Analgesic therapy is limited in most cases to the judicious use of intramuscular codeine, which has a mild, predictable (and reversible) effect on mental status and respiratory drive. Stronger narcotics as well as benzodiazepines and related sedatives are generally avoided because of their more profound and less predictable influences on these functions, which would confound accurate neurologic assessment.

For most procedures in which the cranial vault has been opened, a CT scan is obtained on the first postoperative day. This scan serves as a baseline study for comparison with any subsequent studies should neurologic complications arise.

Prophylaxis against deep venous thrombosis and pulmonary embolism includes intraoperative and postoperative use of dynamic compression stockings. In addition, patients are mobilized as soon as feasible after surgery.

Complications

Postoperative problems may be brain-related or non-brain-related. Many of the most serious complications of cranial base surgery are brain-related (Sekhar and Janecka, 1991), and their prevention and management demand neurosurgical expertise. First, brain manipulation, especially by excessive retraction, can lead to *cerebral edema, subdural hematoma,* and *acute brain dysfunction* specific to the anatomic site involved. In the long term, it may cause *encephalomalacia* and *deficits of speech, memory,* and *cognitive and intellectual functions.* Brain injury (by contusion) may also predispose the patient to *seizures,* especially when the temporal lobe is affected. These problems are best prevented by obtaining adequate surgical exposure through basal bone removal and craniofacial disassembly (limiting the need for significant brain manipulation) and by intraoperative "brain relaxation" through use of subarachnoid CSF drainage, hyperventilation, and steroids.

Pneumocephalus may occur as a sudden event in the early postoperative period, after a patient attempts (against advice to the contrary) to blow his or her nose, inadvertently insufflating air through the dural closure. This rapid-onset pneumocephalus may cause intracranial mass effect (tension pneumocephalus) with confusion, obtundation, and progressive neurologic deterioration. Pneumocephalus may also develop more slowly as a result of overdrainage of CSF from a lumbar spinal drain, which can cause a siphon effect, drawing air upward from the nasal cavity. Preventive measures therefore include keeping patients intubated or tracheotomized until they are alert enough to follow instructions, and judicious use of spinal drainage at a conservative rate and for short duration. In most patients after cranial base surgery, the lumbar catheter is allowed to drain 25 to 50 mL every 8 hours and is removed after 24 to 72 hours. When pneumocephalus is suspected, prompt CT assessment is necessary. Small collections of air may simply be observed, whereas larger ones may require decompression, either by needle aspiration (through an osteotomy site or burr hole) or by reexploration and reinforcement of the reconstruction. In cases of acute-onset pneumocephalus, administration of 100% oxygen is a useful adjunctive measure to enhance resorption of the intracranial air. Lumbar subarachnoid CSF drainage should be discontinued in the presence of any significant pneumocephalus.

CSF fistula is another brain-related complication, important because of its association with meningitis. It usually presents as rhinorrhea but may also cause the patient to complain of a salty taste in the throat. If there is doubt as to the nature of a nasal discharge, the beta2-transferrin assay may be used to confirm or rule out the presence of CSF (Oberascher, 1988). For small-volume leaks, which are by far the most common, observation and continued spinal drainage (or seral lumbar spinal taps) usually suffice. For high-volume leaks (grossly apparent rhinorrhea) or those that are persistent, careful sinonasal endoscopy or CT cisternography will be necessary to attempt to localize the fistula site (frontal, ethmoid, sphenoid, eustachian tube, temporal bone), after which operative repair is frequently needed. A CSF fistula may also present as otorrhea or as wound discharge; the management principles are similar in either case. In some situations the original repair simply needs to be revised; in others, additional vascularized tissue may be needed to obliterate the fistula site. Such tissues may include temporalis muscle, galeal, or free microvascular flaps (for example, rectus abdominis or omentum). In selected cases the closure may be augmented by the judicious use of free autogenous fat grafts or fibrin glue. The risk of reconstructive failure is substantially decreased if the original repair is done using vascularized local tissues (Snyderman et al,

1990).

Central nervous system infections, including meningitis and brain abscess, may also occur. These complications can lead to extreme neurologic morbidity or death. Preventive measures include the use of perioperative antibiotics, strict adherence to sterile technique, minimization of dural exposure to the aerodigestive tract, and meticulous attention to reconstruction as outlined earlier. Treatment consists of antimicrobial agents (intravenously and sometimes intrathecally) and surgical exploration when necessary to drain abscesses or to obliterate sources of continued bacterial contamination.

Cerebrovascular complications are of major concern in the perioperative period. These events may have several causes. Carotid rupture may occur as a result of infection (and pseudoaneurysm) or excessive adventitial dissection. This complication is often sudden and fatal but may sometimes be preceded by a sentinel bleed, which, if recognized as such, can give time for prompt intervention (reexploration and bypass or permanent ICA occlusion). Thrombotic ICA occlusion or embolism into distal vessels can cause stroke and death. Strict maintenance of systemic blood pressure both intraoperatively and postoperatively avoids hypotension, which can precipitate ICA thrombosis. Careful surgical technique when working in the vicinity of the ICA and cautious postoperative anticoagulation in high-risk patients may decrease embolic phenomena. Although the incidence of stroke in cranial base surgery is low, it is a source of considerable morbidity when it does occur. This morbidity can usually be lessened when the cranial base team adopts a vigorous approach to intervention and rehabilitation. Intervention after stroke has occurred should include tight control of hemodynamic factors affecting cerebral blood flow to prevent extension of the infarct. It should also include measures to ensure optimal homeostasis, such as control of airway and oxygenation, maintenance of fluid and electrolyte balance, and nutritional support. Rehabilitation of stroke patients (and also of patients with neurologic deficits from other causes) is greatly facilitated by input from professionals in rehabilitation medicine, physical and occupational therapy, and speech therapy.

Cranial nerve deficits deserve special consideration because of their profound impact on postoperative recovery and quality of life. Sacrifice of olfactory nerves (I) is often regarded as a minor disability, but it can contribute to significant malnutrition in some patients who, because of anosmia, no longer enjoy eating. Deficits of cranial nerves II, III, IV, and VI lead to visual disability of variable degree, depending on the extent of surgical trauma and nerve involvement by the disease process itself. Dense palsies of the extraocular muscles seldom recover completely. Patients in whom such deficits are predictable should therefore be prepared for loss of binocular vision before surgery is undertaken. Extraocular muscle surgery by the ophthalmologist can sometimes help to compensate for such deficits. Loss of visual acuity is a potential complication of anterior cranial base surgery but is unusual unless the optic nerves are compromised preoperatively. In these patients, recovery of optic nerve function is difficult to predict (Derome and Visot, 1987).

Deficits of the fifth and seventh nerves are dangerous mainly because of loss of sensation (V-1) or protection (VII) of the cornea. Tarsorrhaphy and careful attention to ocular lubrication are important means of preventing keratitis and visual loss in these cases. Fifth nerve dysfunction, in addition to the obvious sensory deficits, may also cause significant problems with mastication (and ultimately with nutrition as well) because of loss of motor

28

innervation to pterygoid, temporalis, and masseter muscles (V-3) (Nuss et al, 1991b). Seventh nerve paralysis can be a tremendous social and emotional handicap and should be rehabilitated aggressively whenever possible. Eighth nerve deficits, although rarely reversible, can usually be compensated for (when unilateral) by means of audiologic and vestibular training.

Deficits of cranial nerves IX, X, and XII are potentially life threatening, especially when they occur together, because of aspiration and dysphagia with subsequent pneumonia and malnutrition. Management of patients in whom these deficits are present preoperatively or expected postoperatively should therefore include tracheostomy and gastrostomy. These procedures should be done either at the time of the cranial base surgery or very soon thereafter - before complications compromise the patient's recovery. Laryngoplasty or Teflon injection of the unilaterally paralyzed larynx are also beneficial. Patients who have multiple, *bilateral* deficits of IX, X, and XII are extremely difficult to rehabilitate. Management in these cases nearly always involves permanent tracheostomy and gastrostomy and sometimes also laryngotracheal separation.

Complications of cranial base surgery that are not brain related are many (Table 190-2). Most are not unique to cranial base surgery and are associated with major surgery of all kinds. These complications can affect any of the physiologic systems. Again, an aggressive approach to prevention, detection, and management of complications is critical to successful postoperative outcome.

Table 190-2. Nonneurologic complications in cranial base surgery

Type of complication	Examples
Cardiovascular	Myocardial infarction Hypotension Congestive heart failure
Respiratory	Pulmonary embolism Pneumonia Aspiration Respiratory distress syndrome Pulmonary edema
Hematologic	Posttransfusion coagulopathy Anemia Deep venous thrombosis
Renal	Acute tubular necrosis Drug-induced nephropathies
Infectious	Pneumonia Sepsis Thrombophlebitis Wound infections Viral hepatitis Urinary tract infections
Gastrointestinal	Malnutrition Stress ulcer Gastrointestinal bleeding
Metabolic	Derangements of serum sodium, potassium, calcium, phosphorus, magnesium, and other minerals
Endocrine	Diabetes insipidus Syndrome of inappropriate ADH Hypopituitarism
Hepatic	Drug-induced hepatitis Cholestasis.