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Traumatic, Iatrogenic, and Spontaneous Cerebrospinal Fluid (CSF) leak: endoscopic repair

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Abstract. *Traumatic, Iatrogenic, and Spontaneous Cerebrospinal fluid (CSF) leak: endoscopic repair.* Over the past two decades, Cerebrospinal Fluid (CSF) leak repair has advanced from open invasive intracranial approaches to transnasal endoscopic ones that avoid the traditional morbidities of frontal craniotomy approaches – such as anosmia, intracranial haemorrhage or oedema, seizures, memory deficiencies, and behaviour disorders – reducing morbidity, reducing hospitalisation times and accelerating return to work, and therefore cutting indirect costs.

The diagnosis of CSF rhinorrhoea is both clinical and radiological. The presence of CSF in clear nasal drainage should be established by analysis for CSF markers. Localisation of the leak site involves radiological investigation, mainly Computerised Tomography (CT) and Magnetic Resonance Imaging (MRI).

In addition to suppressing symptoms, the main goal of the closure of CSF rhinorrhoea is to prevent ascending meningitis. The operative management of cerebrospinal fluid leak is advised in the following circumstances: persistent, post-traumatic CSF leaks after 4 to 6 weeks of conservative treatment; all cases of spontaneous CSF fistulae; cases with intermittent leaks; delayed posttraumatic leaks; cases of CSF leak with a history of meningitis; false CSF rhinorrhoea coming from the petrous bone via the Eustachian tube.

The graft material used depends mainly on the authors' experience and did not significantly influence the success rate.

The main steps in the surgical procedures do not differ as much from one author to the other: accurate localisation of the defect; creation of a raw surface around the defect to accept the graft and to help in the formation of synechiae to support the seal later; plugging of the defect with fat covered with fascia lata supported by absorbable gelatin and Merocel. The differences between the authors relate to the use of fluorescein to locate the defect, the importance of prophylactic antibiotherapy, the plugging materials, the technique of underlay or overlay grafting, the use of fibrin glue and the need for lumbar drainage.

The success rate for endoscopic repair of CSF rhinorrhoea is high: approximately 90% at the first attempt.

Recent reports in the literature highlight the group of patients with spontaneous idiopathic CSF leak as a group with specific attributes and treatment challenges.

Introduction

Over the past two decades, Cerebrospinal Fluid (CSF) leak repair has advanced from open invasive intracranial approaches to transnasal endoscopic ones that avoid the traditional morbidities of frontal craniotomy approaches – such as anosmia, intracranial haemorrhage or oedema, seizures, memory deficiencies, and behaviour disorders – reducing morbidity, reducing hospitalisation times and accelerating return to work, and therefore cutting indirect costs.

These procedures were made possible, safe and accurate thanks to the development of advanced radiological localisation techniques: tomodensitometry (TDM), magnetic resonance imaging (MRI), intraoperative radiological guidance and the experience of the practitioners acquired with the surgical management of inflammatory diseases of the paranasal sinuses. Moreover, the improvement and the diversification of the endoscopic surgical instruments and the establishment of teams bringing together ENT surgeons and neurosurgeons have boosted these minimally invasive approaches.

The endoscopic approach to the anterior skull is now considered to be the current standard of care for repairing most cerebrospinal fluid fistulae and skull base defects.[1-4]

Some physiological data

CSF is produced within the choroid plexus of the lateral, third and fourth ventricles, as well as by capillary ultrafiltrate and the intracranial metabolism of water. The total volume in adults is 90-150 ml, and this volume is turned over three to five times per day. CSF flows into arachnoid space and is absorbed along the cerebral convexities by arachnoid villi which act as one-way valves. The absorption of CSF requires a pressure gradient of 1.5-7 cmH_2O for antegrade flow. Normal CSF pressure is 5-15 cmH_2O recorded at the lumbar level in a patient in the lateral decubitus position. In the supine state, ICP (IntraCranial Pressure) ranges from 5 to 15 cm of water, rising up to 40 cm water with rapid head elevation, and returning to baseline levels after compensation.[1] The pressure of CSF fluctuates during the day and neurological symptoms generally begin to occur when CSF pressure reaches 15-20 cmH_2O.[5] CSF is formed at a rate of 0.35 ml/min.

Clinical features

In cerebrospinal fluid leak, the patient reports a history of intermittent, generally unilateral, aqueous rhinorrhoea, head trauma, endoscopic or endocranial sinus, or skull base surgery. The main symptoms patients present with are anosmia, headache, vomiting, and recurrent episodes of meningitis.[6]

CSF leak may be classified on the basis of various characteristics, such as anatomic site, aetiology and intracranial pressure (ICP). The most frequent causes of CSF leak requiring repair are accidental trauma (15 to 51%) causing localised or extended fractures of the skull base, or surgical trauma (8 to 58%).[2,7-15] Spontaneous and congenital causes are also found. Although some authors use the term "spontaneous" for CSF leaks associated with multiple aetiologies like tumours, delayed CSF leak from trauma or associated with congenital malformation of the skull base,[16,17] this term should be limited to CSF leaks which start without any causal event and are perhaps better described as "spontaneous idiopathic CSF leaks"[18] (Table 1).

Most commonly, the leak is found mainly in a cribriform plate, sphenoid sinus or anterior ethmoid location, being located less often in the frontal sinus, posterior ethmoid or inferior clivus. The cribriform plate is the thinnest and weakest area of the anterior skull base and it is the area most likely to be fractured. The cribriform plate is also particularly vulnerable to the development of spontaneous leaks because of the pressure of maldevelopments with extension of the subarachnoid space through the foramina of the cribriform plate[6-22] (Table 2).

There are no differences in the location of CSF leaks depending on whether the aetiology is traumatic or iatrogenic.[15-24] The frequency of spontaneous leaks is between 15 to 23%[13,14] with the most common locations being at the level of the cribriform plate, and mainly the sphenoid sinus.[15] Recent data suggest that intracranial pressure is involved in the origin of leaks and in the increased failure rate associated with spontaneous leaks. Benign intracranial hypertension is now considered to be a major contributor to spontaneous leak physiology alongside meningoencephalocele.[25] Spontaneous CSF leaks, which accounted historically for only 3 to 5%[2,3,26,27] of CSF leaks, were recently reported at higher rates ranging from 14 to 46%.[6,15,19-21,24,28] This suggests that this clinical entity is being increasingly recognised.

Diagnosis

The diagnosis of CSF rhinorrhoea is both clinical and radiological. The presence of CSF in clear nasal drainage should be established by analysis for CSF markers. The most commonly used approach is the assay for *beta-2 transferrin*, which has been established as a sensitive (97%) and a specific (93%) assay for the presence of CSF.[29,30] Cerebral neuramidase produces beta-2 transferrin from its native beta-1 transferrin by desialisation.[31] Beta-2 transferrin is found only in the CSF, vitreous humour of the eye and perilymph. Analysis for beta-2 transferrin requires as little as 0.17 mml of fluid with results in less than 3 hours using immunofixation electrophoresis laboratory techniques.[32] Quite rarely, false positives may occur involving patients with chronic leaver disease, genetic variations of the transferring gene and inborn error of glycoprotein metabolism.[33,34]

Another marker, the *beta trace*, can also be used for detecting CSF leak with a slightly improved sensitivity (100%) and specificity (100%).[34,35] Beta-trace protein is secreted into the CSF after being produced in the leptomeninges and the choroid plexus. This protein has been identified as prostaglandin D synthase[36] and is second only to albumin in its abundance in the CSF. Beta trace protein is present in other fluids throughout the body, but its concentration elsewhere is significantly lower than that found in CSF.[37]

Localisation of the leak site involves radiological investigation.

Table 1

Aetiology of skull base defect

Authors	1[2]	2[7]	3[8]	4[10]	5[11]	6[12]	7[13]	8[14]	9[15]	10[19]	11[6]	12[20]	13[21]
Year	1996	1999	1999	2004	2004	2004	2004	2004	2005	2005	2005	2005	2006
N of Patients	**36**	**27**	**12**	**52**	**20**	**92**	**39**	**53****	**24**	**97**	**267**	**20**	**24**
Spontaneous	%33	7.4	8.3	44	25	22	15	23	20.8	%30	46	20	25
Head Trauma	%11	14.8	24.9	25	20	20	51	28	20.8	%70	45	30	33
Surgical Traumas	%56	74	58.1	31	50	45	33	49	58.4		9	30	21
EES	%33	29.6	16.6	21		26			41.7				
Transphenoidal hypophysectomy		29.6	33.3	10		19			16.7*				
Other	0	3.7	8.3	0	5	12.5				0		20***	21

Percentage of the different aetiologies of cerebrospinal fluid leaks. Surgical traumas are predominant. The percentages listed under "EES" (endoscopic endonasal surgery) and "Transphenoidal Hypophysectomy" are covered by surgical traumas and have already taken into account in this row.

*Skull base procedures other than transphenoidal hypophysectomy are included.
**53 patients but 57 procedures.
***Encephaloceles.

Table 2

Location of the CSF leak

Authors	123	213	3 1,14	412	515	56	5*24	5**24
Year	2003	2004	2004	2004	2005	2005	2006	2006
N of Patients	**21**	**39**	**53**	**87**	**24*****	**267**	**135**	
Cribriform plate	%28.5		35	28	29	81	23.1	27
Sphenoid Sinus	%43		26	41	25	14		18.9
Anterior Ethmoid	%28.5	61.5	18	31	46	5	20.5	35.1
Posterior Ethmoid			9					
Frontal Sinus			10		21			
Inferior Clivus			2					

*Traumatic patients (skull base fracture) with, in these cases, 35.9% with multiple sites of injury.
**Iatrogenic patients.
***Some patients had more than one skull base defect.

As an initial procedure, numerous authors[12,24,25] have recommended 1-mm thickness axial and coronal *computed tomography scan* (CT) (Figures 1,2,3) with a bone algorithm. Coronal images are vital for the evaluation of the skull base along the roof of the ethmoid and sphenoid sinuses. The posterior walls of the sphenoid and frontal sinuses are best evaluated with axial images. Nevertheless, skull base defects do not necessarily imply CSF leaks because patients may have defects without active leaking.

CT cisternograms may provide more important information about the site of CSF leak. CT cisternography uses intrathecal water-soluble iodine contrast material rather than metrizamide, as was once the case. Ideally, the contrast will be detected in the nasal cavity or a specific sinus at the completion of the study. This sensitivity of this test is quite low (48 to 96%)[38] and this is likely due to the requirement that the patient must be actively leaking CSF at the time of the study. This procedure does involve a risk of lumbar puncture and intrathecal contrast.

Additional localisation techniques include *magnetic resonance (MR) imaging* (Figure 1). MR imaging using T2-weighted images and MR cisternography can also help with localisation. The magnetic resonance cisternography algorithm shows the CSF as black against adjacent tissues that are diminished in intensity. This technique involves a fast spin echo with fat suppression and image reversal. The sensitivity of this study is reported to be 85-92% with 100% specificity.[39]

Patients with spontaneous idiopathic leaks have some characteristic findings on CT scan or MRI that can help with diagnosis. These findings result from the increased intracranial pressure frequently observed in these patients, which induces benign intracranial hypertension (BIH). CT scans in these cases show broadly thinned and attenuated

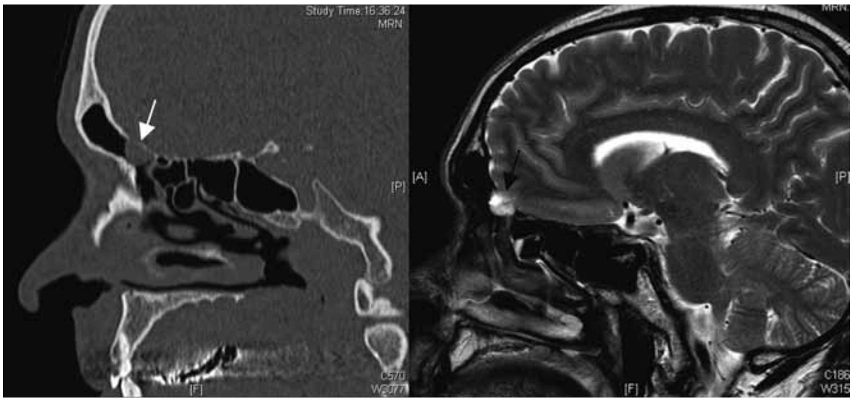

Figure 1
Small meningocele with CSF leak five years after a nasal trauma

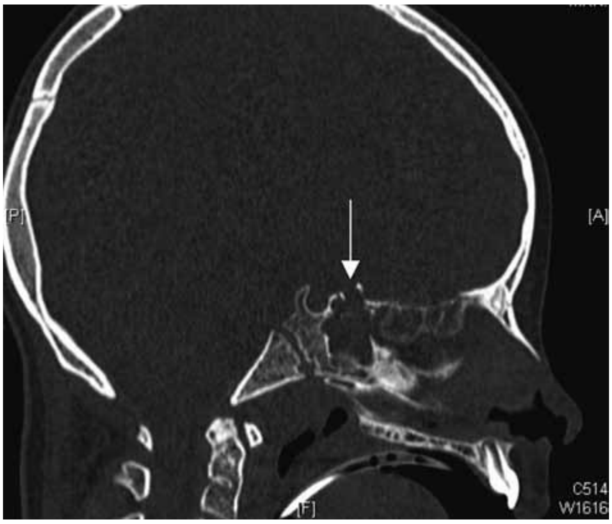

Figure 2
Fractures of the skull base with CSF leak rhinorrhoea

skullbase,[25] dehiscence of the ethmoid roof,[40] arachnoid pits due to bony impressions from arachnoid villi,[41] pneumatisation of the lateral sphenoid recess (Figures 4,5) (in 91% of the patients with spontaneous idiopathic CSF leak) by comparison with similar findings in controls (only 23-43%).[42]

In spontaneous idiopathic CSF leak, MRI is effective in assessing small meningoencephaloceles and determines their composition. Indeed, these patients have the highest rate of meningoencephaloceles formation[17,29,43] and may present with multiple simultaneous meningoencephaloceles.[29] Another benefit of MRI is the identification of empty sella resulting from an increase of the intracranial pressure with herniation of meninges and CSF through the sellar diaphragm, the point of least resistance.[29,44,45]

Radionuclide cysternography entails the intrathecal injection of a tracer (Technetium 99m) and a number of hours later, the measurement of radioactivity on pledgets placed in the nasal cavity at specific sites: the cribriform plate, the middle meatus and the sphenoethmoidal recess.[25] This technique is useful for intermittent low-volume CSF leak. Sensitivity is comparatively low (62-76%) with a 33% false positive rate.[16,38,46] Precise localisation of the site of CSF leak is not possible with this technique and it requires uncomfortable procedures like intrathecal injection and pledgets placement.

The intrathecal injection of fluorescein (Figure 6) may help to localise the CSF leak in the nose and viewing is enhanced with a blue light filter when the skull base is exposed.[47] The use of intrathecal fluorescein is not approved by the United States Food and Drug authority because of reported complications (seizures, lower extremity weakness and opistotonos).[48,49] These complications seem to be related to high fluorescein concentration, rapid injections or sub-occipital injection.[47,48] A range of dilution protocols can be found in the literature.[3,15,21,28,50] One of them[51] recommends diluting 0.1 ml of 10% sodium fluorescein in 10 ml of CSF, followed by the slow intrathecal injection of this mixture over 10 min. Once the fluorescein is injected the lumbar drain is clamped and the patient is placed in the Trendelenburg position.

Some authors advise the topical application of 5% fluorescein intranasally using cotton pledgets or directly with a syringe. The change from yellow to green fluorescence indicates the presence of CSF.[52]

Image guidance systems (Figure 7) may also provide additional help for the localisation of skull

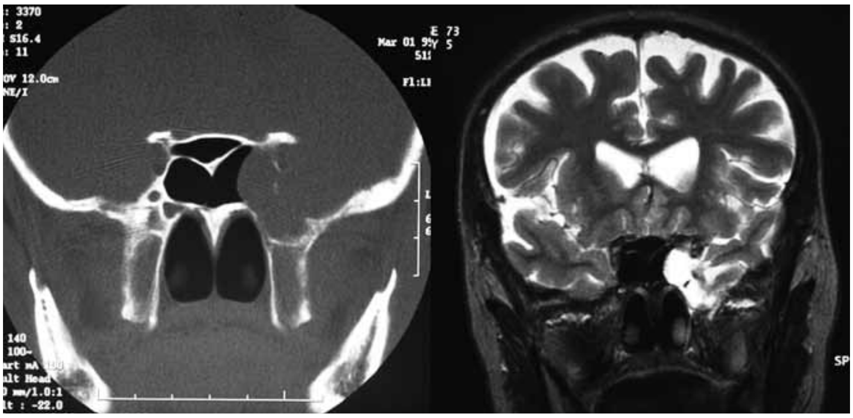

Figure 3
Meningocele protruding into the sphenoid sinus with CSF fluid rhinorrhoea

base defects but no statistically significant benefit for the successful closure of nasal CSF leaks can be found with computer-assisted surgery.[15] Nevertheless, some authors report increased surgeon confidence when using image guidance systems that can lead to more complete skeletonisation of the skull base.[53,54]

Treatment

Why and when should the skull base defect be closed?

In addition to suppressing symptoms, the main goal of the closure of CSF rhinorrhoea is to prevent ascending meningitis. Indeed, this is consistent with reports in the literature that state that post-traumatic CSF fistulae develop into meningitis in 9% to 50% of cases.[55] The reported associated mortality rate is between 20% to 70%.[55] In acute post-traumatic non-iatrogenic dural fistulae, the risk of meningitis following dural repair was estimated at 4% and operative mortality at 1.3%. Closure of cerebrospinal fluid leaks can prevent ascending bacterial meningitis, whatever the cause of the CSF leak (traumatic-iatrogenic or spontaneous).[56] In some respects, endoscopic endonasal repair seems more effective than transcranial approaches.[57] Conservative collateral measures, such as avoiding peaks in intra-abdominal pressure, reducing intracranial pressure with the orthostatic position, prescribing relaxant medication and inhibitors of carbonic anhydrase, prohibiting nose blowing or sneezing with closed mouth, as

Figure 4
Spontaneous idiopathic CSF fluid leak in the lateral recess of the left sphenoid sinus. MRI Fast Spin Echo.

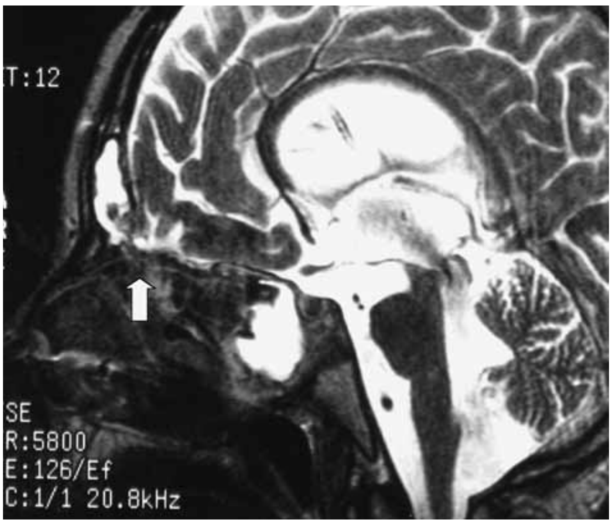

Figure 5
MRI Fast Spin Echo.
Posttraumatic defect of the posterior wall of the frontal sinus (white arrow).
CSF fluid frontal leak/CSF fluid sphenoid leak.
Small encephaloceles – Hydrocephalus (dilatation of the third ventricule).

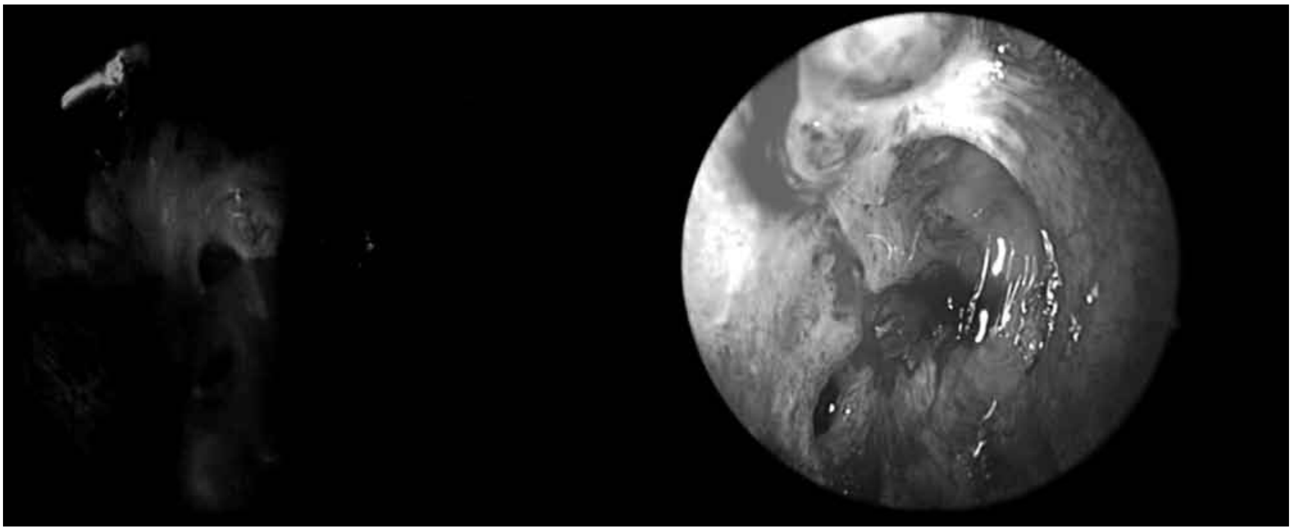

Figure 6
CSF fluid leaks identified with fluorescein

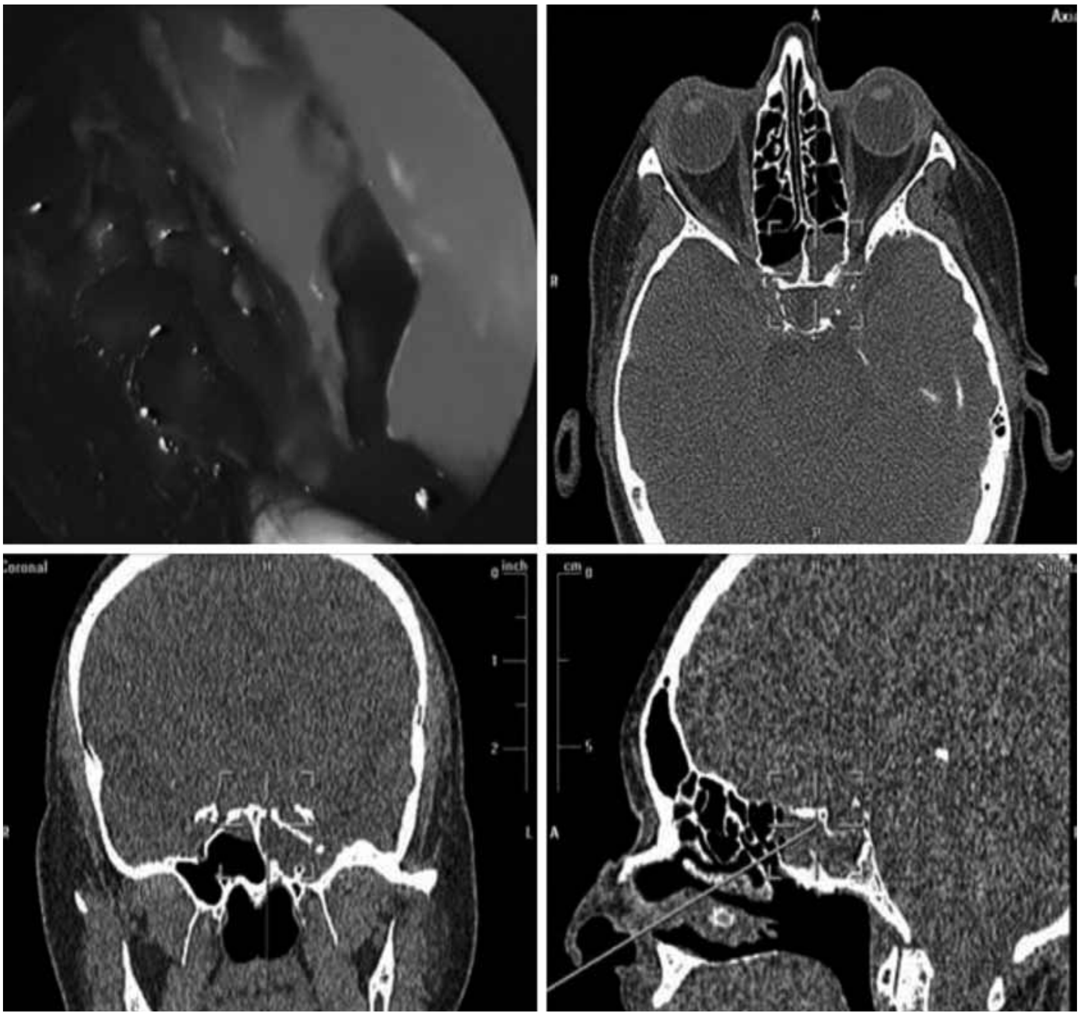

Figure 7
Spontaneous CSF fluid leak of the lateral recess of the left sphenoid sinus identified with CAS (Computer-Assisted Surgery)

well as avoiding physical exercise for a certain period of time, can help a tight closure of the skull base defect. Nevertheless, conservative management of dural defects has had a success rate of only 50%.[58] Moreover, after conservative management, the rate of ascending meningitis may rise to 18.5%.[57] In the light of these findings and the excellent results and low complication rate reported after minimally invasive endonasal surgery, the literature recommends endoscopic skull base revision for patients with a CSF leak, especially in those with a prior ascending bacterial meningitis even if the CSF leak is not active.[58] Conservative leak management, even when the active leaks stop, seems risky and so potential sites of leakage of the anterior skull base should be closed as soon as they are recognised.[2]

Conservative management (bed rest, acetazolamide 250 mg 8 hourly, broad-spectrum antibiotic prophylaxes, avoidance of straining, CSF drainage by repeated lumbar puncture rather than leaving a lumbar drain if the leak is found to be brisk[6]) is advised for a period of 4-10 days by some authors, or a period of 2 to 6 weeks by others, in acute traumatic CSF leaks because no dura is missing, while there is often a loss of dura in iatrogenic cases.

The use of prophylactic antibiotics is controversial.[59,60] Some authors claim that it definitively reduces the risk of meningitis[6] while others[59,60] disagree and believe that routine use of antibiotics actually exacerbates the risk of gram-negative infections by altering the nasopharyngeal microflora.[59]

The operative management of cerebrospinal fluid leak is advised in the following circumstances: persistent, posttraumatic CSF leaks after 4 to 6 weeks of conservative treatment; all cases of spontaneous CSF fistulae; cases with intermittent leaks; delayed posttraumatic leaks; cases of CSF leak with a history of meningitis; false CSF rhinorrhoea coming from the petrous bone via the Eustachian tube.[6]

With advances in endoscopic sinus surgery, the majority of closures of cerebrospinal rhinorrhoea are now carried out using an endonasal endoscopic approach. Nevertheless, a few cases still involve transcranial or external extracranial approaches: extensive and multiple fractures of the skull base, concomitant intracranial pathology, or frontal sinus leak.[6]

The approach to leak closure (Figures 8,9)

The majority of authors list the same key factors for the successful repair of cerebrospinal rhinorrhoea:[6,56] a skilled endoscopic surgeon who is thoroughly familiar with the endoscopic anatomy of the nose and the paranasal sinuses; proper patient selection; pre-operative localisation of the defect.[6] The main steps in the surgical procedures do not differ as much from one author to the other: accurate localisation of the defect; creation of a raw surface around the defect to accept the graft and to help in the formation of synechiae to support the seal later; plugging of the defect with fat covered with fascia lata supported by absorbable gelatin and Merocel. The differences between the authors relate to the use of fluorescein to locate the defect, the importance of prophylactic antibiotherapy, the plugging materials, the technique of underlay or overlay grafting, the use of fibrin glue and the need for lumbar drainage (Table 3).

If the CSF leak is closed and no relevant bulging of the dura towards the nasal lumen is present, as in most of the small or middle-size defects, additional bony or cartilaginous grafts are not needed. When needed, the grafts are easily harvested from the septum and placed between the dura and the bony skull base for supporting the cranial content[61] (underlay or inlay technique).

Although advised by some authors for all defects larger than 1 cm[2,3], it has been found that, in the reconstruction of skull base defects with bone grafts, these grafts can result in dead space and an increased risk of infectious complications.[62] In our experience, it is often difficult to place a graft above the skull base without further trauma to the dura, which may exacerbate the risk of displacement after surgery and often have an answer effect on the probability of the mucosal flap healing. Shaping and placing underlay bone grafts in patients with spontaneous cerebrospinal fluid leak may easily lead to the fracture of the broadly attenuated skull base and make the defect even larger.[63] However, some authors recommend placing bone grafts in the epidural space in cases of spontaneous cerebrospinal fluid rhinorrhoea with elevated intra cranial pressure (ICP) in order to provide improved structural support.[47]

A well-vascularised flap with sufficient volume to include the perichondrium may provide an adequate support for the brain tissue without bony reconstruction. However, if necessary, different types of graft are used to support the skull base: cartilage, bone,

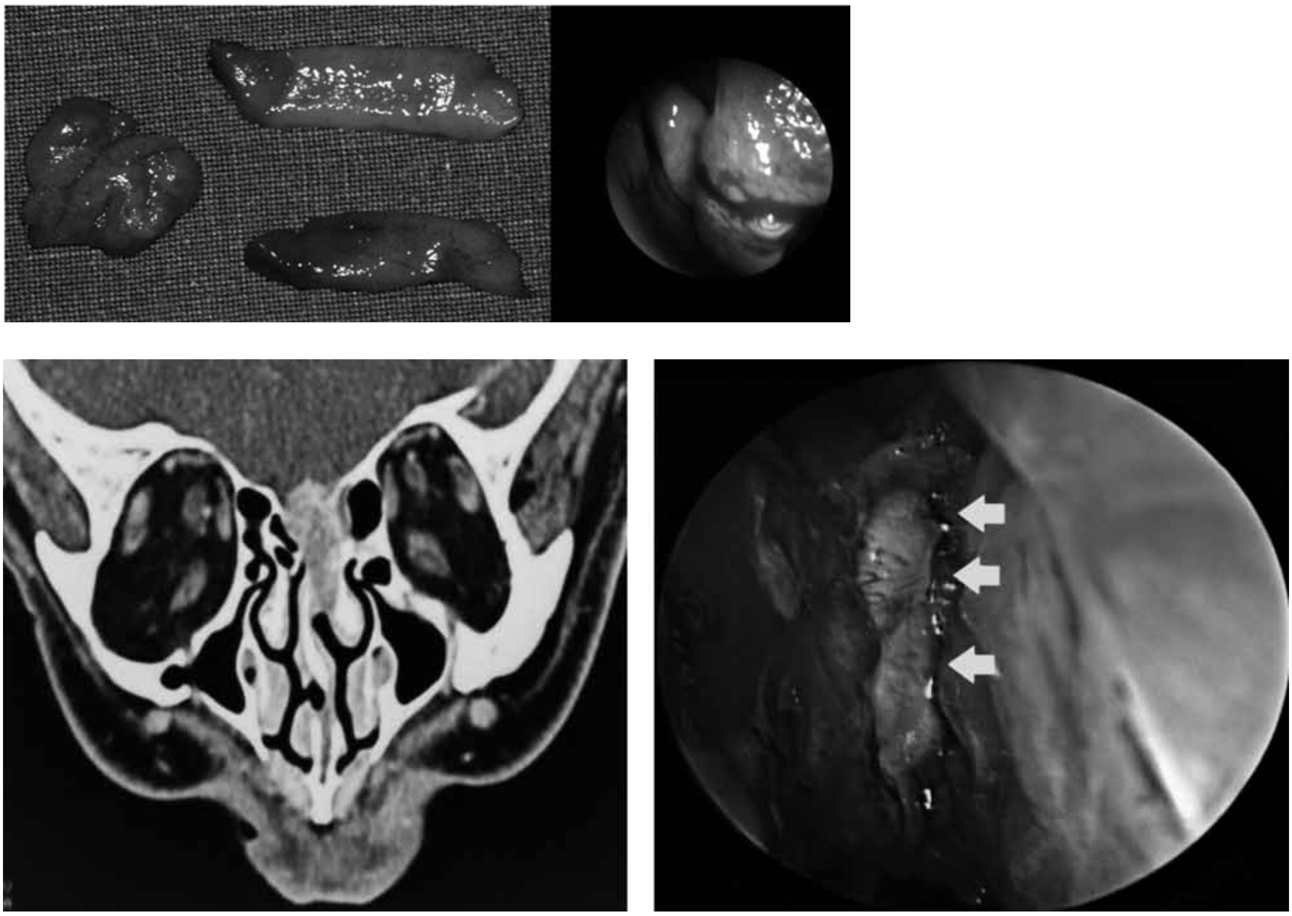

Figure 8
Repair of a defect of cribriform plate with a mucosal flap harvested from the inferior turbinate

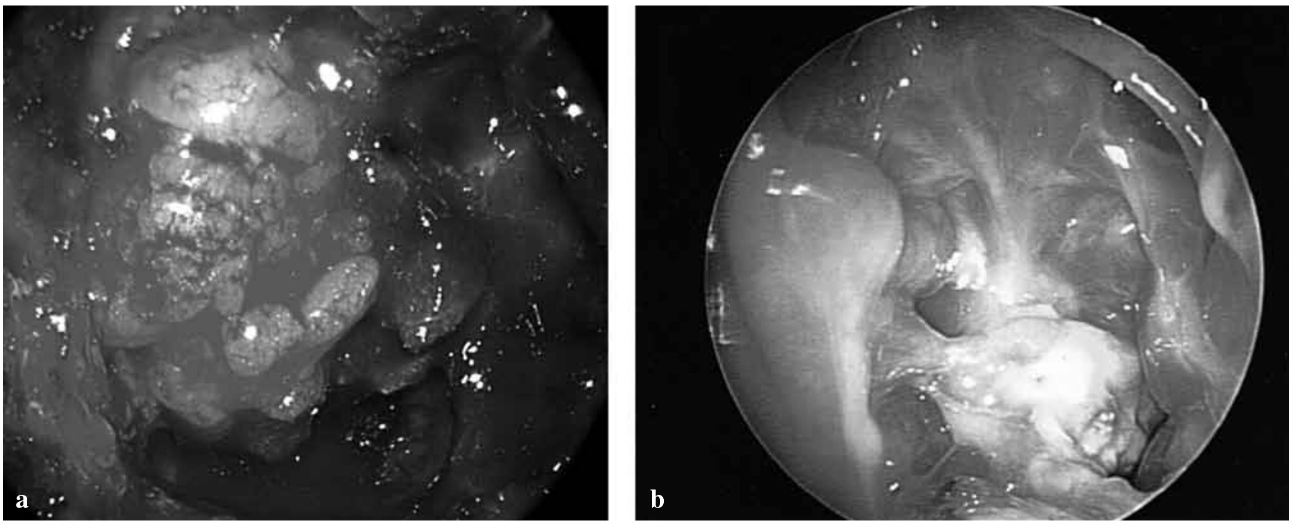

Figure 9
A fat plug repairing an ethmoidal iatrogenic defect of the skull base (A); fat and fascia temporalis (B)

Table 3
The approach to closure

Author reference.	Year	Number of patients	Fluor-escein.	Closure material	Proph A.biotic.	Underlay Overlay	Fibrin glue	Lumbar Drain
2	1996	36	Yes	- Mucoperisteum (septum, turbinate) - Fat-temporalis - fascia- - myofascial graft - Bone-cartilage	Yes	Underlay	N	Y
72	1997	6	Yes	Fat				Y
61	2000	16		- Free mucosal flap from the inferior or middle turbinate. - - Lateral displacement of the M Turb - - - septal cartilage		Underlay	Y	N
73	2001	15		Fat-cartilage			Y	
23	2003	21		- Plastic material - Abdominal Fat-fascia lata - rotated middle turbinate flap			Y	
11	2004	20		Fat-fascia lata		Both	Y	
10	2004	52		Fat in the majority of the cases		Underlay		
13	2004	39		- Turbinate composite or mucosa - Dura substitute - Abdominal Fat				
6	2005	267	No	Fat-fascia lata	Yes		N	N
15	2005	24		Temporalis fascia - Conchal, septal, chondral, cartilage - middle turbinate - Hydroxyapatite			Y	
56-57	2005	39	Yes	Fascia lata*+ Mucosa	Yes	Underlay	Y/N	N
20	2005	20		- Fat – Bone - Chondral septum - Mucoperiosteum - Perichondral mucosa	No	Underlay		Y
74	2005	16	Yes	- Fascia lata - Middle turbinate. Mucosa - Nasal perichondrium - Ear cartilage - Abdominal. Fat				
47	2008	56**		Bone (septal- Turbinate-mastoid)		Underlay		Y

*In two or three pieces.
**All Spontaneous CSF leak.

fascia lata, temporalis muscle. Fat, which maintains its size and consistency, is generally preferred to muscle, which shrinks to a fibrous reticulum.[6] We recommend[61] the use of fibrin glue and further support with gelfoam or surgicel packing. When multilayer skull-base reconstruction is needed for larger defects, the intracranial surface is supported by fat or myofascial graft, followed by a bone- or cartilage-free graft placed on the intracranial side of the defect.[2] This multilayer skull base repair may be temporarily supported by an inflatable balloon.[28]

Authors have also reported the use of free, vascularised tissue transfer, including radial forearm fascia[64,65] and rectus abdominis[66] muscle for complex (usually multiple) non-localised, skull-base defects. Each CSF leak requires an individual approach, with larger cranial defects requiring close consideration of extranasal tissue sources.

The graft material used depends mainly on the authors' experience and did not significantly influence the success rate.[67]

In the presence of encephalo-celes, the mucosal capsule (if present) is incised and cauterised to achieve haemostasis. Resection is achieved by gradual fulguration with bipolar cautery. To allow any residual encephaloceles to recede intracranially, all intranasal connections with the encephaloceles are carefully severed.[2]

The defects located at the level of the *cribriform plate* are more difficult to close because an underlay technique medially is almost impossible due to the lack of bony support. In these cases, the fascia are positioned on the dura and bent towards the Crista galli.

CSF leaks located in the central position of the *sphenoid sinus* can usually be approached in a standard transethmoid endoscopic approach with possible conversion to transseptal assistance, depending on visualisation. Central leaks can be repaired with standard grafting techniques or by sinus obliteration with fat or muscle after complete removal of the mucosa. As complete removal of the mucosa is unlikely, precluding the possibility of mucocele later, the first technique is preferable.[1]

Lateral sphenoid leaks are more difficult to visualise and access safely in a transethmoid approach. In these cases, the transepterygoid approach may be an alternative.[68,69]

Similarly, *frontal sinus* techniques depend on the location of the leak. Lateral and superior leaks may require an extracranial approach with osteoplastic flap and obliteration. Inferiorly and posteriorly based defects at the level of the frontal recess and cribriform plate may benefit from a combined endoscopic frontal approach and frontal trephination. Leaks at the level of the frontal recess or at the origin of the frontal outflow tract may be closed using endoscopic approaches.[70]

The endoscopic repair of frontal sinus CSF leaks and encephaloceles can be effective if meticulous attention is paid to the preservation of the frontal sinus outflow tract, preventing an osteoplastic flap and obliteration. The major limiting factor for an endoscopic approach is extreme extension superiorly or laterally within the posterior table beyond the reach of current instrumentation.[69]

It is essential, especially with defects of the anterior ethmoidal roof, to keep the opening to the frontal sinus free of obstructing mucosa. In these cases, a large opening of the frontal ostium is required and, in some cases, a rain stent is required in the frontal infundibulum.[61]

The success rate of the endoscopic repair of the CSF rhinorrhoea is high (Table 4) at about 90% at the first attempt. The complication rate rises to approximately 2.5%[71], but to approximately 8.5% with encephaloceles repair. Reviews underscore the morbidity of endoscopic CSF leak repair compared with craniotomy-based approaches.[4,71-73]

Spontaneous idiopathic CSF leaks

Recent reports in the literature highlight the group of patients with spontaneous idiopathic CSF leak as a group with specific attributes and treatment challenges.

In this group, the failure rate can rise by 25 to 87% after a first attempt at endoscopic CSF leak closure[74-79] by comparison with other CSF leak aetiologies (Table 4). In these cases, subsequent CSF leaks can occur at a site other than the site repaired and so the failures can be attributed to a failure of the management of elevated intracranial pressure rather than to a failure of the operative repair.[58]

In studies attempting to identify common factors in this group, it has been shown that female sex, obesity and age played a key role in this condition.[80,81] The majority of patients with spontaneous idiopathic nasal CSF leaks have been shown to have elevated intracranial pressure (ICP). Additional precautions may therefore be warranted before, during and after the repair of these CSF leaks.[18]

Elevated ICP most commonly manifests itself in the syndrome of benign intracranial hypertension (BIH). Patients with BIH present clinically with headache, pulsatile tinnitus, balance problems, and visual disturbances. Studies have indicated that the spontaneous aetiology of CSF leak most often represents a variant of BIH[82]. CT scan or MRI shows that many of these patients have total or partial empty sella syndrome[83] due to dural herniation through the sellar diaphragm into the sella thurcica, abnormalities of the optic nerve sheath complex, encephaloceles, arachnoid pits or multiple dural ectasia,[84] broadly attenuated and thinned skull base,[63] and dehiscence of the ethmoidal roof.[40]

For all these reasons, the management of spontaneous cerebrospinal fluid leak may require procedures to supplement the surgical closure of the skull base defect: reducing significant weight loss,[80] lumbar drain pressure recording, acetazolamide 500 mg twice daily or, in severe cases, a ventriculoperitoneal shunt for intracranial hypertension.[47] The most important factor for successful repair in these patients is the reduction of their ICP by nutritional, medical, or surgical means.

Table 4

Success rate

Success rate %	**Authors**	**Year**	**Follow-up**	**Comments**	**N of patients**
83.3%-90*%	Burns *et al.*[27]	1996	5-68 months		42
94%	Lanza *et al.*[2]	1996	2-57 months Mean 24.6 months		51
100%	Wormald *et al.*[72]	1997	13 months		6
81.5%	Castillo *et al.*[7]	1999	24.7 months		27
83%	Nachtigal *et al.*[8]	1999	?		12
95%	Zweig *et al.*[75]	2000	?		53
90-97*%	Hegazy *et al.*[4]	2000	?	Meta-analysis Subdural intracranialabcess- Meningitis less than 1%	289
88%	Bachert *et al.*[61]	2000	?		17
87.1%	Castelnuovo *et al.*[67]	2001	1 year minimum		31
80%-93.3%	Guevara *et al.*[73]	2001	6 months-7 years Mean 2.9 years	Sphenoid CSF leak[4]*	15
94.9-100%[2]*	Schick *et al.*[76]	2001	?		126
89%-97%*	Chin *et al.*[77]	2003	?		36
95.2%	Lopatine *et al.*[23]	2003	9-42 months		21
90-100*%	Briggs *et al.*[10]	2004	27 months		52
100%	EL-Banhawy *et al.*[11]	2004	?		20
78-92*%	Mc Mains *et al.*[12]	2004	12-82 months		88
92.3%	Lee *et al.*[13]	2004	?		39
81-89*%	Lindstrom *et al.*[14]	2004	?		47
67-96*%	Tabaee *et al.*[15]	2005	1 m-10.6 y Mean 1.5 y	Computer-assisted[3]*	24
96.6-98.9%[2]*	Kirtane *et al.*[6]	2005	?		267
97%	Bernal-Sprekelsen *et al.*[56]	2005	22-120 months Mean 65 months		41
90-97*-99[2]*%	Mirza *et al.*[19]	2005	?		72
90%-95*%	Marton *et al.*[20]	2005	6 months-3y	2 late infections[6]*	20
100%	Carrau *et al.*[28]	2005	24-84 months	No complications	25
93.3%-98.9*%	Locatelli *et al.*[24]	2006	?		135
100%	Silva *et al.*[21]	2006	?	No complications	24
71.4%[5]*-87.5%	Gendeh *et al.*[74]	2005	?		16
91%-100%*	Meco *et al.*[79]	2007	?	Success analysed by absence of Beta Trace	29
95%	Woodworth *et al.*[47]	2008	34 months	All spontaneous CSF leak	56

*: success rate at a second endoscopic attempt
[2]* success rate at at a third-attempt
[3]* computer assisted surgery
[4]* all the patients with a sphenoid CSF leak
[5]* Success rate in spontaneous idiopathic CSF leak
[6]* 1 meningitis, 1 intranasal abcess.

Post-operative issues

In the post-operative phase, bed rest, avoidance of straining and Valsalva manoeuvres, especially vomiting, blowing the nose or avoiding uneasy defecation, can be targeted as ways of avoiding rapid changes in intracranial pressure. The slow resumption of normal activities is mandatory. A post-operative follow-up visit is arranged at 1-2 weeks, with conservative management of crusting. Debridement is undertaken only at six weeks postoperatively to minimise the possibility of dislodging the graft. Regular follow-up continues weekly until the repair leak site is completely mucosalised and ventilation of paranasal sinuses is ensured.[1]

Post-operative IntraCranial Complications (ICC), meningitis, cerebral abscess and pneumencephalus are rare (at less than 1%)[4] and, in any case, occur much more often during the pre-operative period (21.7%) than during the peri-operative (2.8 %) or post-operative period.[85] Risk factors for presenting with an ICC and meningitis (0.9%) are revision cases and leaking encephaloceles.[85]

Conclusion

The endoscopic approach to the anterior skull is now considered to be the current standard of care for repairing most cerebrospinal fluid fistulae and skull base defects. For optimising outcomes and ensuring proper treatment, the surgeon must have a comprehensive knowledge of the anatomy, diagnostic tools and surgical approaches. Mainly, these surgical approaches can only be safely carried out by a team bringing together ENT surgeons and neurosurgeons, radiologists and ophthalmologists, and also require an up-to-date technical environment. The most important component of the repair is the good visualisation provided by the endoscopes. The identification of the site of the cerebrospinal leak, the preparation of the bed for the graft, and the repair are therefore better safeguarded with the endoscopic approach. The high, and long-term, success rate over two decades demonstrates the superiority of the endoscopic approaches compared with the extracranial ones.

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