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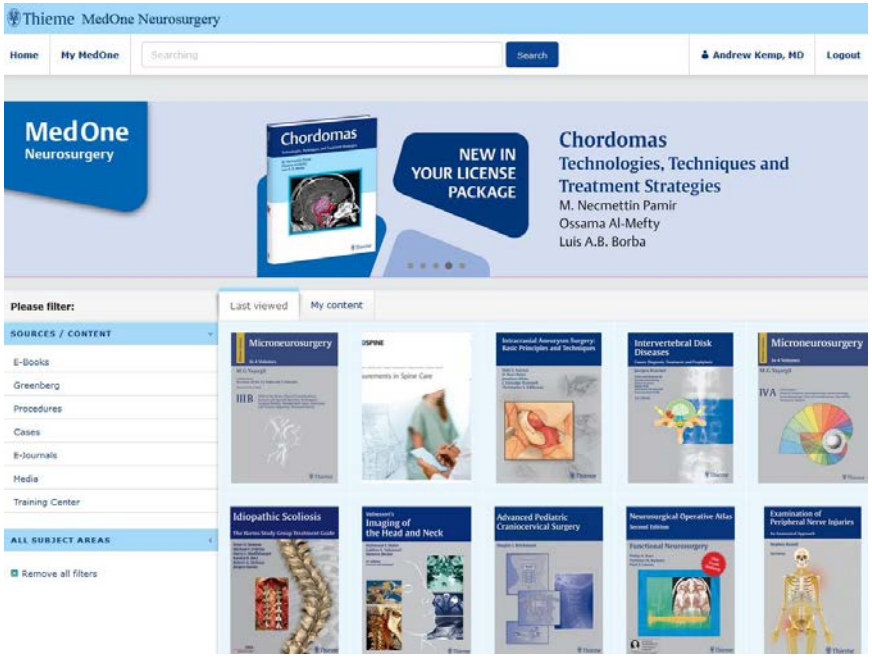
What's New

- Handbook of Neurosurgery, 8e
- Training Center with Q&A
- Full-Text E-Journals
- Refined E-Book View
- Personalization and Note Taking
- Responsive Design



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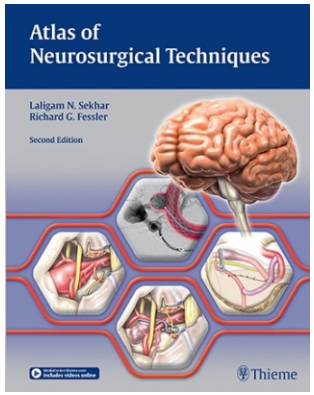

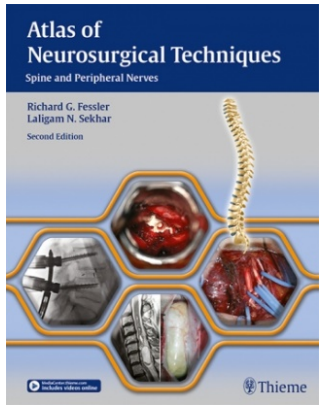

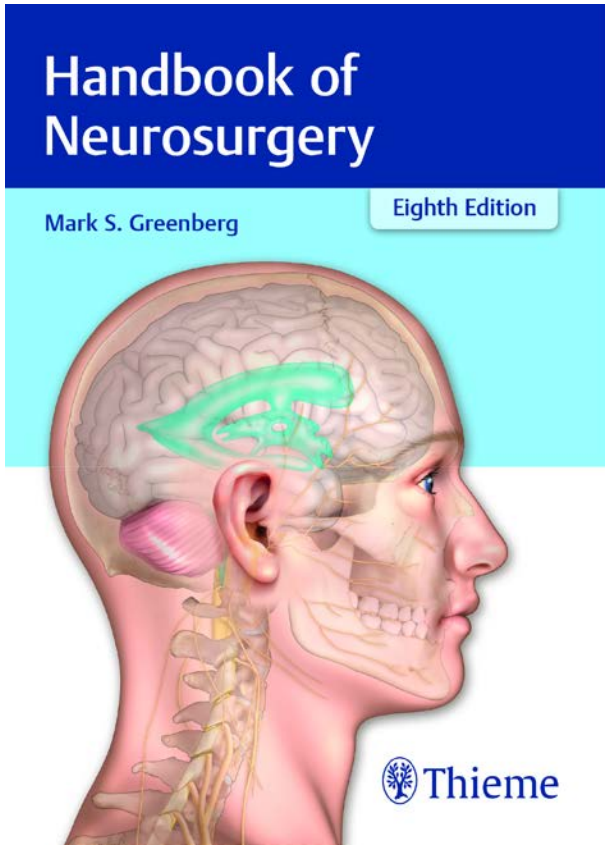

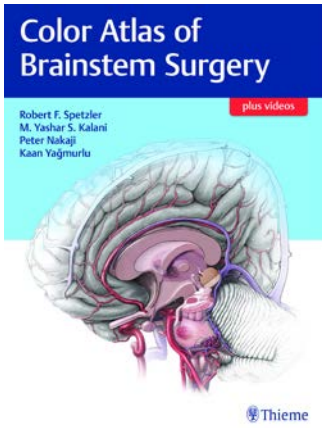

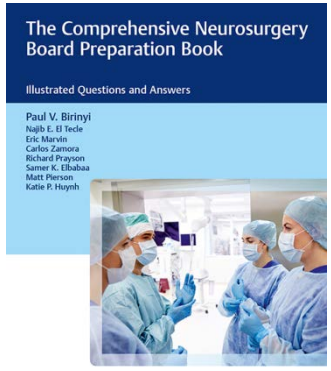

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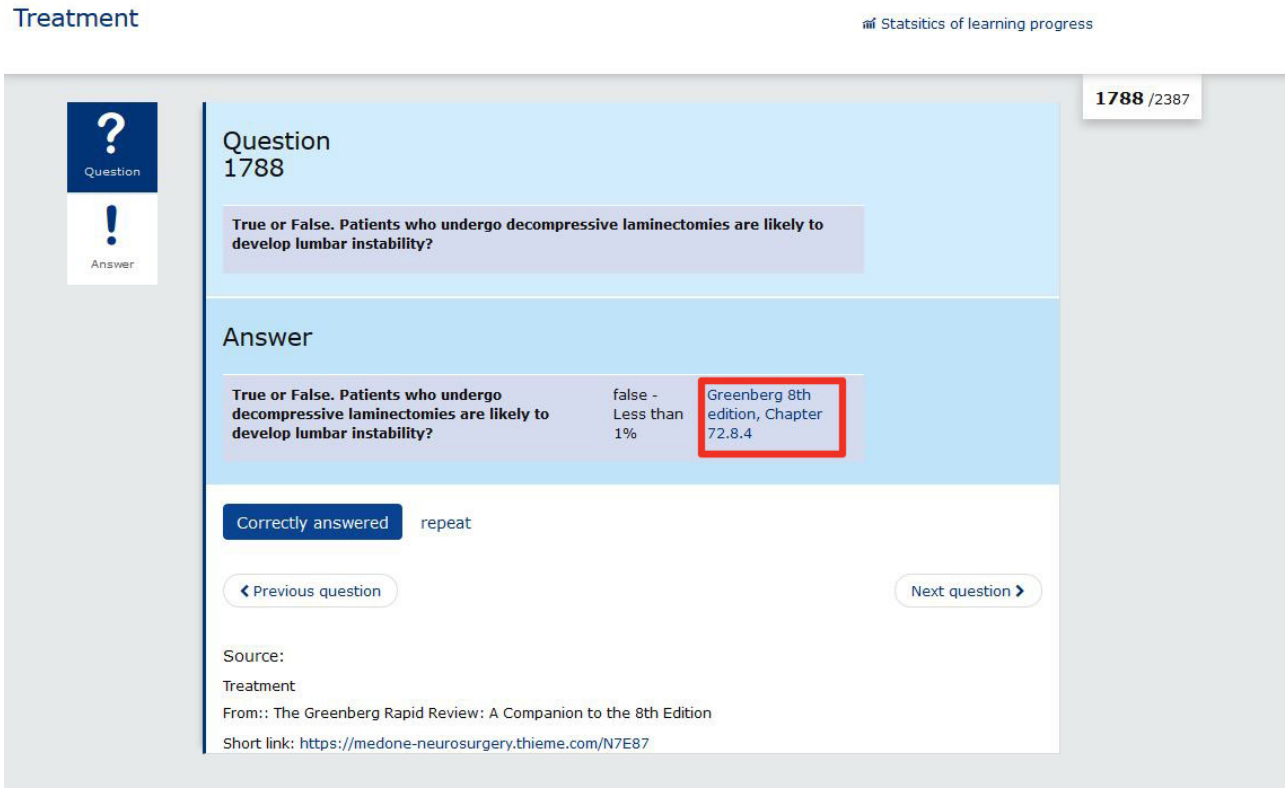
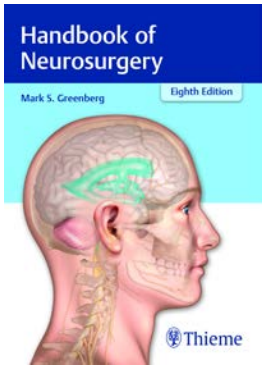
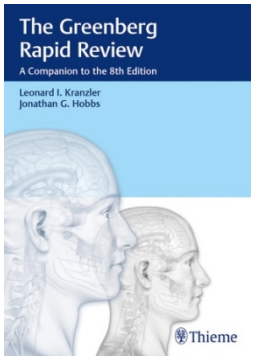
Training Center

More than 2,300 review questions linked to Greenberg for daily, need-to-know facts for all practicing neurosurgeons







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Step-by-step instructions for 418 surgical procedures

Endoscopic Approach to Craniopharyngioma

Nancy McLaughlin, Leo F. S. Ditzel Filho, Daniel M. Prevedello, Daniel F. Kelly, Ricardo Carrau, Amin Kassam

Quick access

Introduction and Background

Definition, Pathophysiology, Epidemiology, and Histology

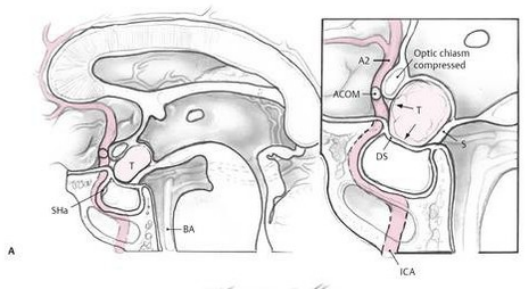
- Craniopharyngiomas are rare tumors of the central nervous system that occur at a rate of 1.3 per million person-years. Overall, they account for 2–5% of all primary intracranial neoplasms. Although they can be diagnosed at any age, craniopharyngiomas typically present a bimodal age distribution with a first peak in children 5–14 years old and a second peak in adults 50–74 years old.
- Craniopharyngiomas arise along the path of the craniopharyngeal duct, a canal connecting the stomodeal ectoderm with the evaginated Rathke pouch. Some authors have proposed that these tumors arise from neoplastic transformation of embryonic squamous cell rests of the involuted craniopharyngeal duct. Others have suggested that craniopharyngiomas result from metaplasia of adenohypophyseal cells in the pituitary stalk or gland.
- Craniopharyngioma are benign grade I tumors according to the World Health Organization classification. Histologically, two subtypes have been recognized, the adamantinomatous (most common, predominantly in young patients) and the papillary (almost exclusively in adults). Transitional or mixed forms have also been reported.
- Despite its benign histology, craniopharyngiomas tend to adhere and infiltrate surrounding structures. This characteristic accounts for their aggressive behavior and potentially significant morbidity and mortality. Rarely, craniopharyngiomas may present a malignant transformation, potentially and questionably induced by radiation therapy.

Clinical Presentation

- As craniopharyngiomas grow within the sellar/parasellar region, they may exert mass effect on critical structures of the nervous system including the optic apparatus, pituitary stalk and gland, floor of the third ventricle, hypothalamus, and cerebral vasculature of the circle of Willis.
- Headache, nausea/vomiting, visual disturbances, and symptoms related to hypothalamopituitary dysfunction are among the most commonly reported clinical manifestations. Less common presenting features include motor weakness, seizures, psychiatric symptoms, autonomic disturbances, and precocious puberty.
- Symptomatic elevated intracranial pressure may occur in any age population, resulting from obstruction of the foramen of Monro or of the aqueduct of sylvius by the tumor.

Staging

- To date, authors have proposed various classification systems depending on their relation to the sella turcica, diaphragm sellae, optic chiasm, and third ventricle. The infundibulum is the key anatomic landmark that helps guide the modular exposure of endoscopic endonasal approaches (EEA) for craniopharyngioma resection (Fig. 60.1), as previously described by Kassam et al.
- *Type I craniopharyngiomas* are preinfundibular, located immediately anterior to the pituitary stalk (most accessible). They are located in the suprasellar space, guarded inferiorly by the diaphragm, superiorly by the displaced chiasm, posteriorly by the pituitary stalk, and laterally by the carotid arteries. Preinfundibular lesions are the most direct craniopharyngiomas to approach through an endonasal route.
- *Type II craniopharyngiomas* are transinfundibular lesions that grow within the long axis of the infundibulum, widening it circumferentially. Such lesions often create a component in the subchiasmatic space and extend rostrally through the tuber cinereum and into the third ventricle. In these cases, the stalk forms the capsule of the tumor.
- *Type III craniopharyngiomas* are retroinfundibular lesions, located posterior to the pituitary stalk (most challenging). They are bounded anteriorly by the pituitary stalk and posteriorly by the mamillary bodies and basilar apex. The tumor may extend rostrally (type 3a), through the membrane of Liliequist, to ultimately encroach or invade the third ventricle. It may also extend caudally (type 3b) to fill the interpeduncular fossa, potentially encroaching on the posterior circulation. Laterally, retroinfundibular craniopharyngiomas are bounded by the oculomotor nerves as they travel forward toward the cavernous sinus and the posterior communicating arteries as they travel between the posterior cerebral artery (P1) and the internal carotid artery (ICA).
- *Type IV craniopharyngiomas* are pure intraventricular tumors. These tumors may best be approached by a transcranial route as the endonasal corridors are often limited by the stalk and chiasm.



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Management and follow-up tips for 225 cases

Brain Abscess

Pedro M. Ramirez and Martina Stippler

Quick access

Case Presentation

A 52-year-old man with no previous medical history was brought to the emergency department with tonicoclonic seizure on the right side. The patient was afebrile, postictal at examination, but had no focal neurological deficit. He had a history of alcohol abuse (12-pack of beer a day). Laboratory workup showed: white blood cell (WBC) count 18.6 × 10^3 (neutrophil 91%); erythrocyte sedimentation rate (ESR) 10 (0–25); C-reactive protein (CRP) 1.4 (< 0.3), and blood glucose is 148 mg/dL. Noncontrast computed tomographic (CT) scan and enhanced magnetic resonance imaging (MRI) were obtained (Fig. 140.1). Blood cultures were negative and the patient is started on empirical antibiotic therapy. Two weeks after empirical therapy was the patient continued having headaches, the WBC count was 3.4 × 10^3, ESR was 15, and CRP was 0.5. A follow-up MRI scan was obtained (Fig. 140.2). The patient undergoes stereotactic needle aspiration, which showed a positive culture for *Nocardia*. Antibiotic therapy was titrated with ceftriaxone for 6 weeks and trimethoprim/sulfamethoxazole for 12 months.

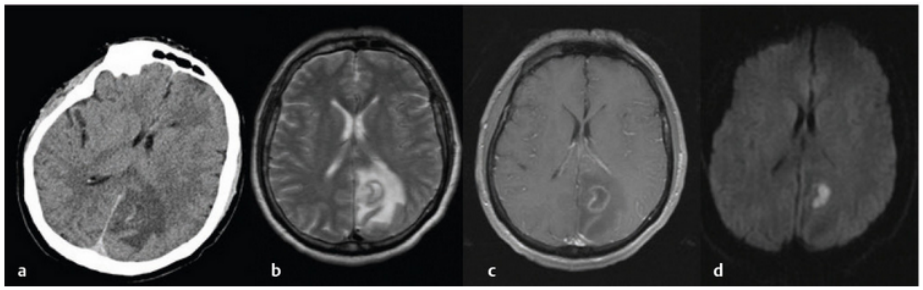

Fig. 140.1 Imaging studies at the time of admission. (**a**) Computed tomographic scan without contrast showing left parietal edema with a central rim. (**b**) T2-weighted magnetic resonance imaging (MRI) demonstrating the hyperintense lesion in the left parietal lobe with surrounding vasogenic edema. (**c**) Contrast-enhanced T1-weighted MRI demonstrating a left parietal ring-enhancing lesion. (**d**) Diffusion-weighted imaging revealing restricted diffusion.

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58,765 images with legends

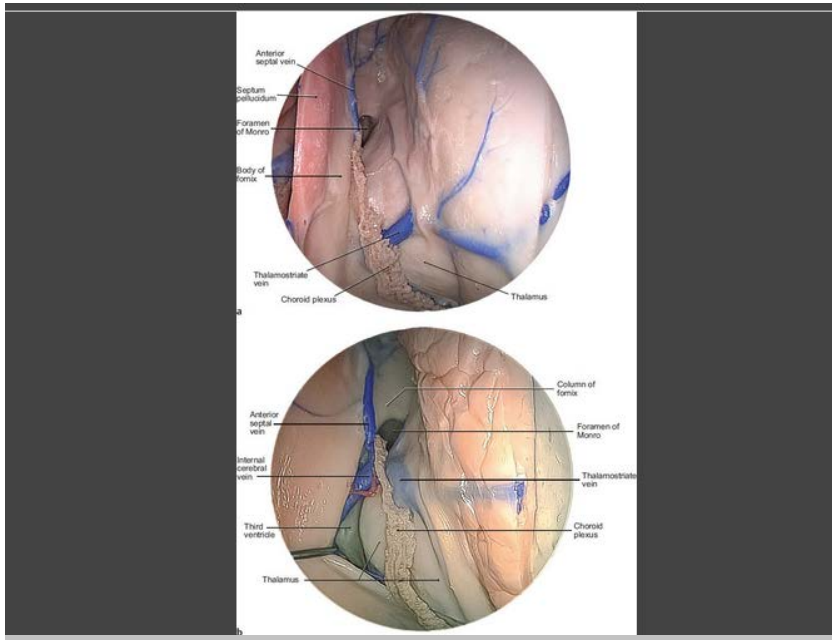
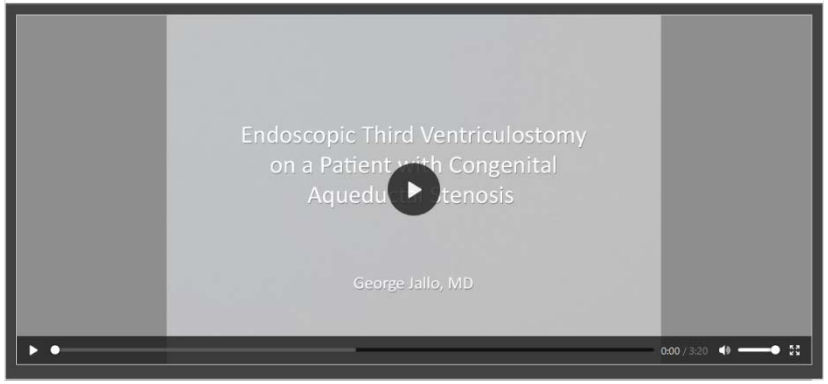


Figure 1.12. (a,b) Endoscopic views of the exposure of the third ventricle.

Source: Color Atlas of Brainstem Surgery > Internal Anatomy of the Brainstem

1,102 videos



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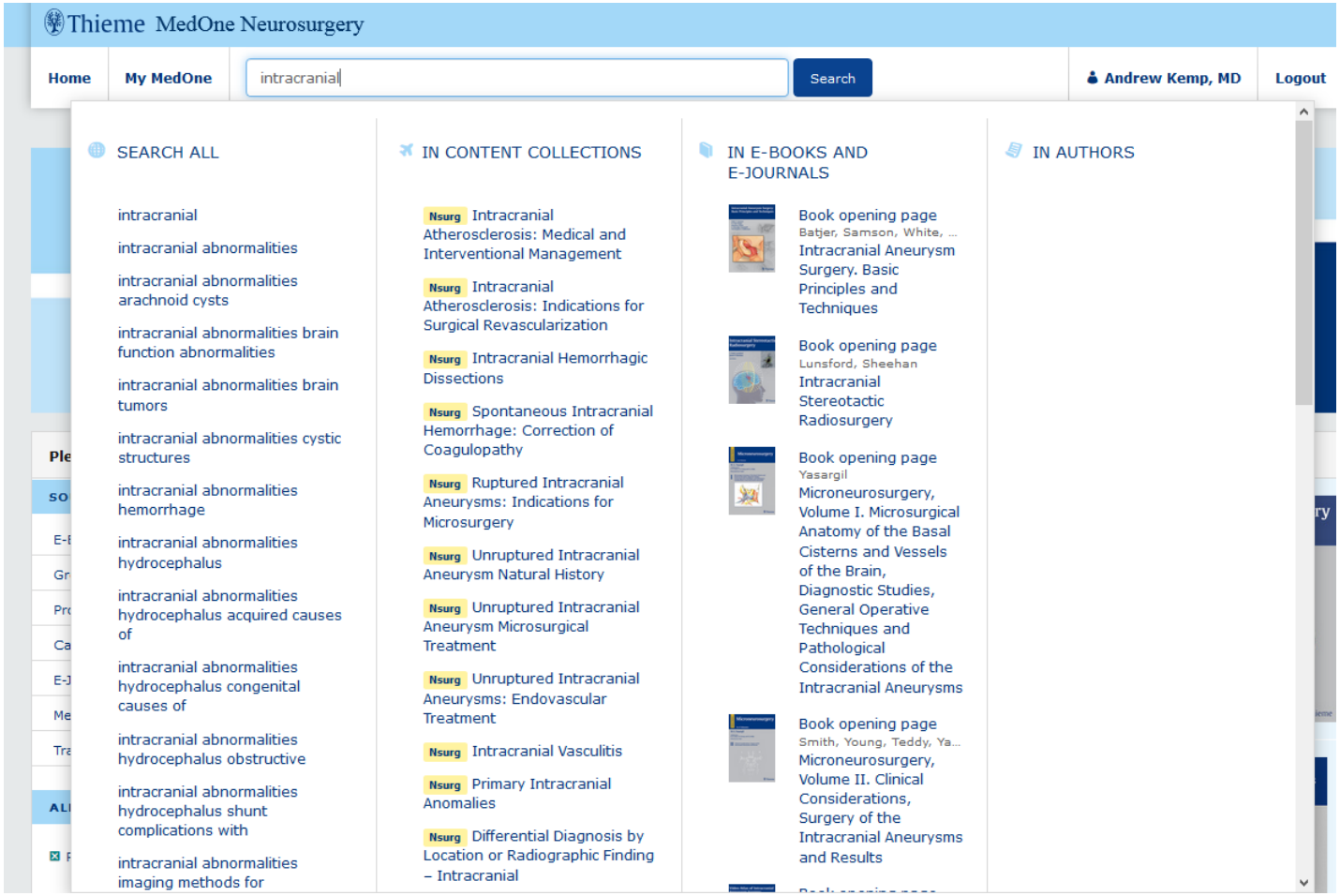

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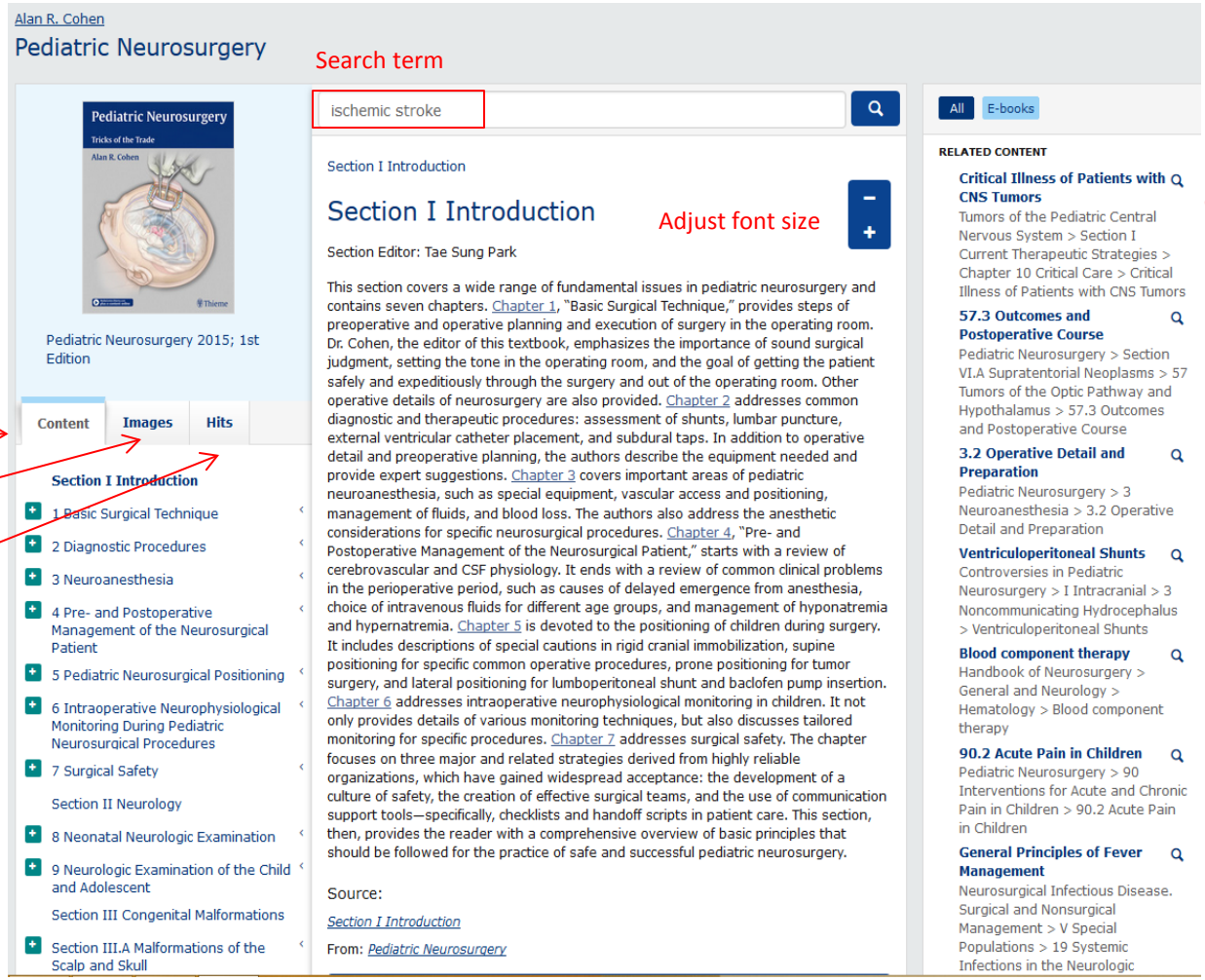

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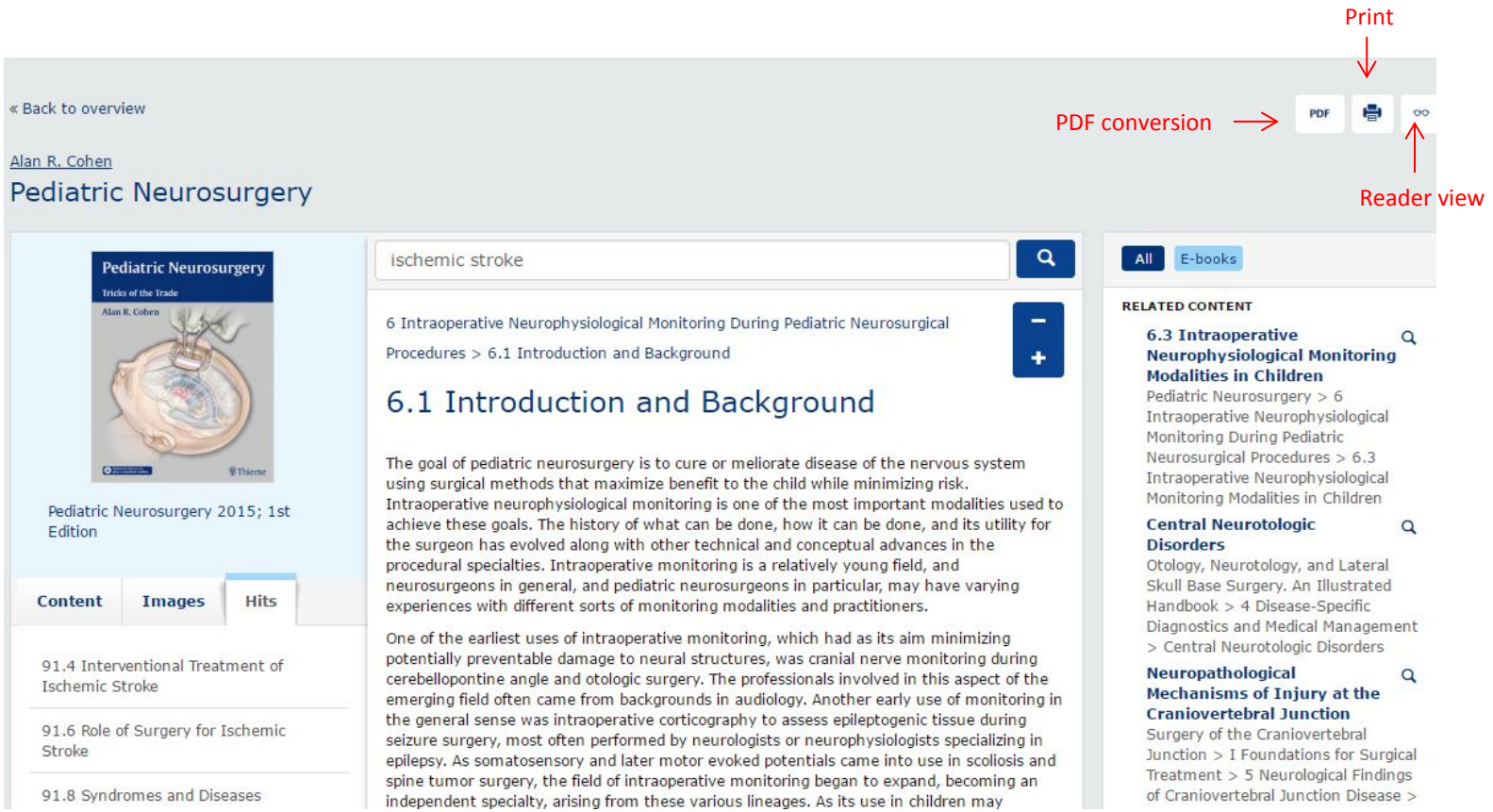

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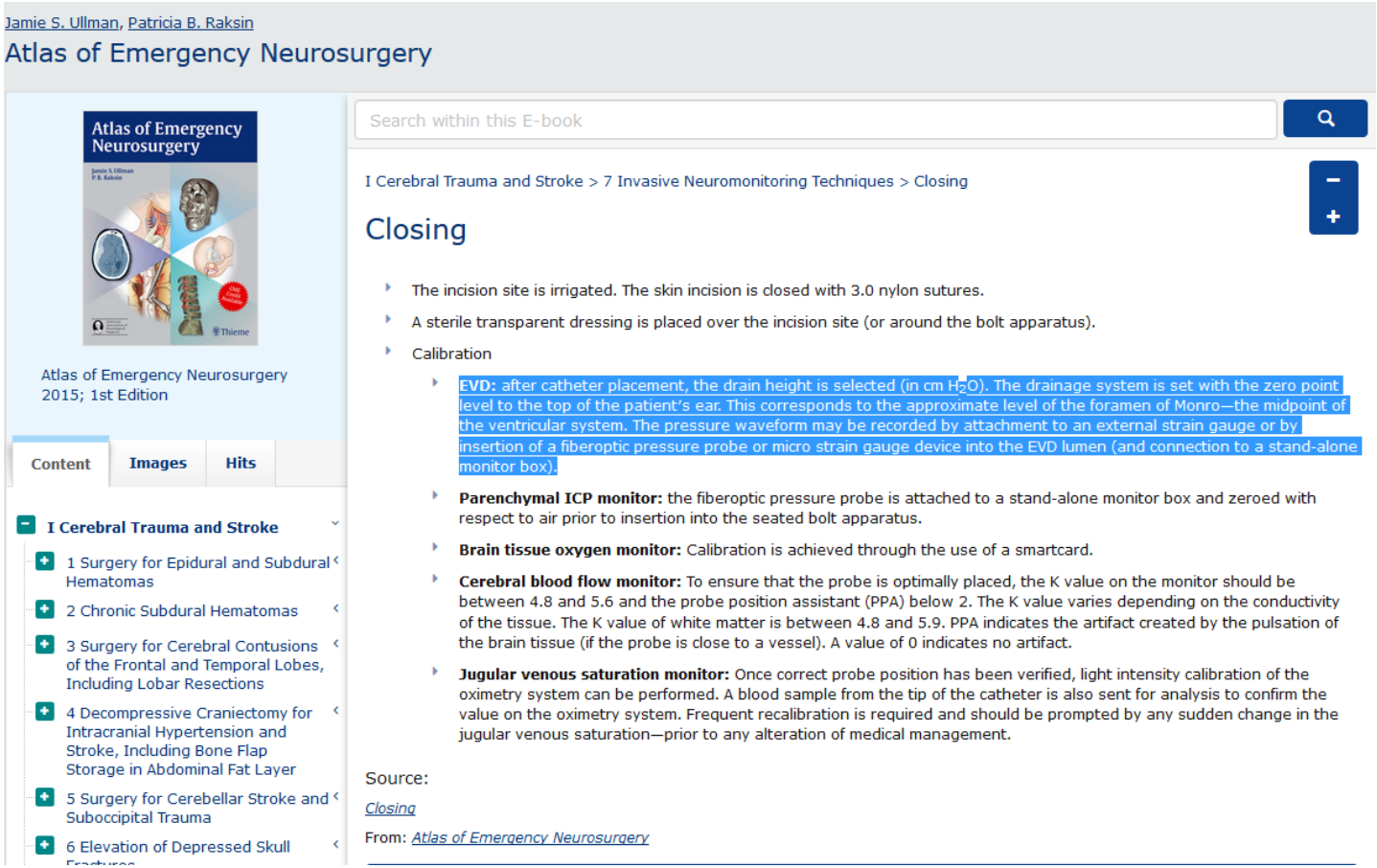
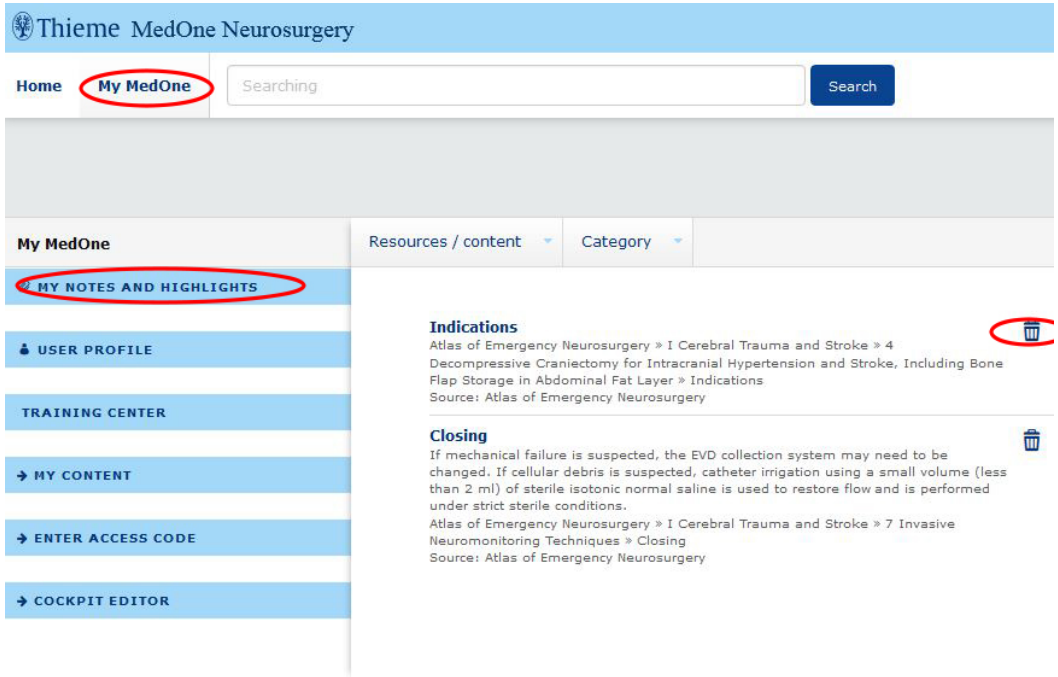
Note Taking

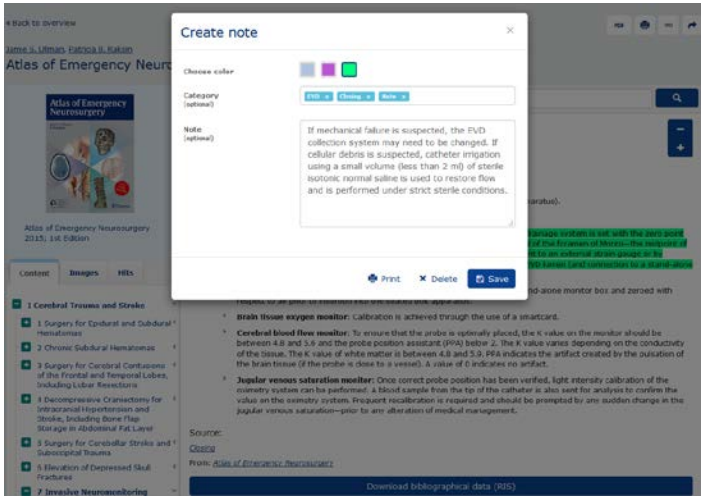
Select text by pressing mouse over desired text, a window opens. In the window, assign categories, write a note, and click the “save" button.



Note Taking

Click “my MedOne” tab to open and review all saved notes





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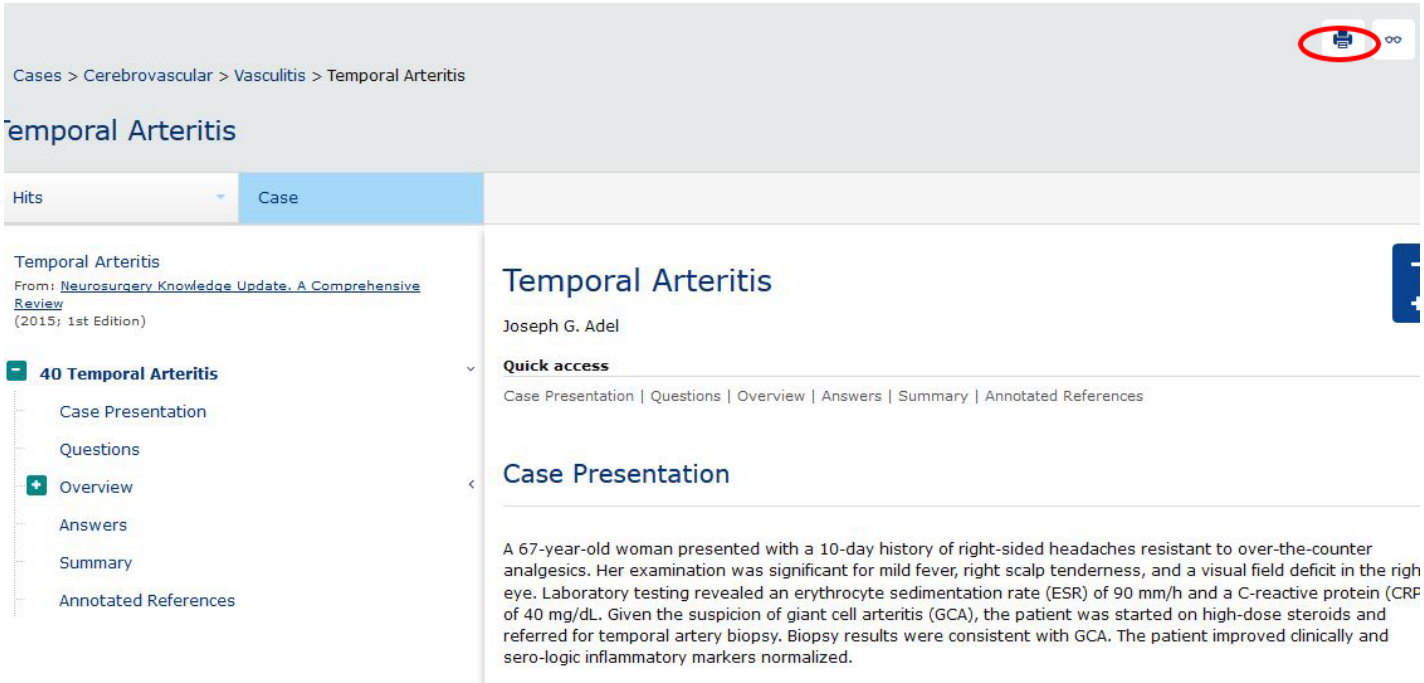

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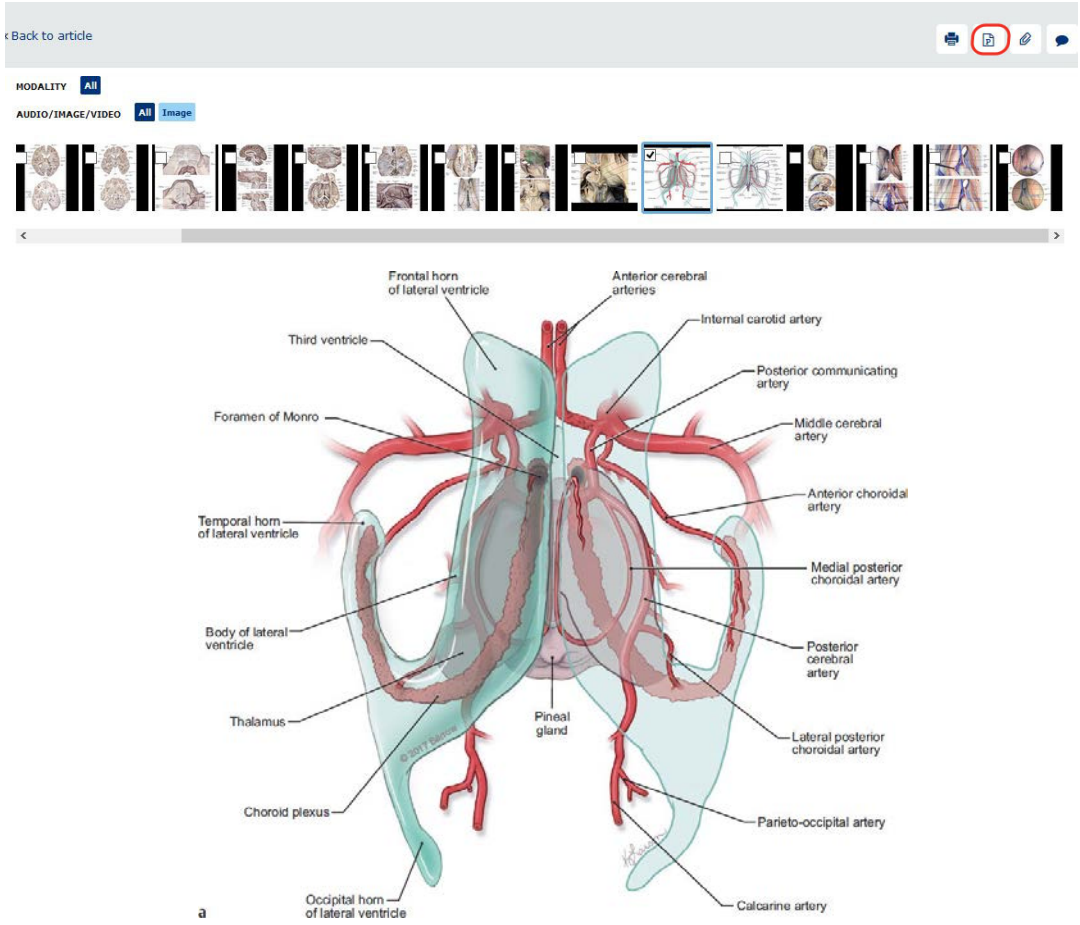
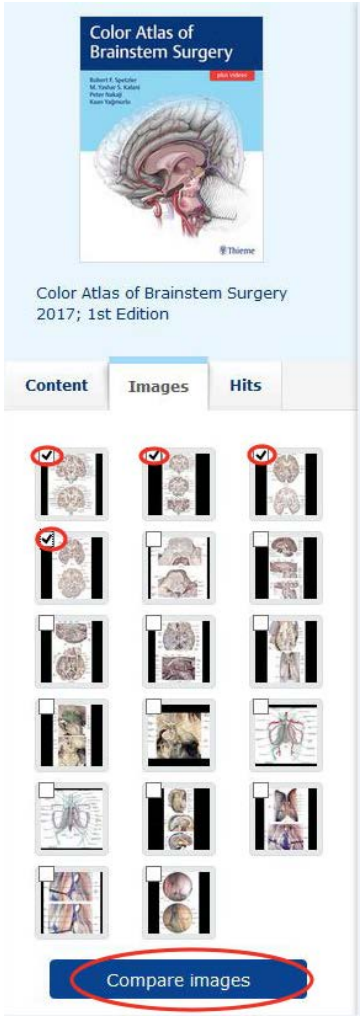
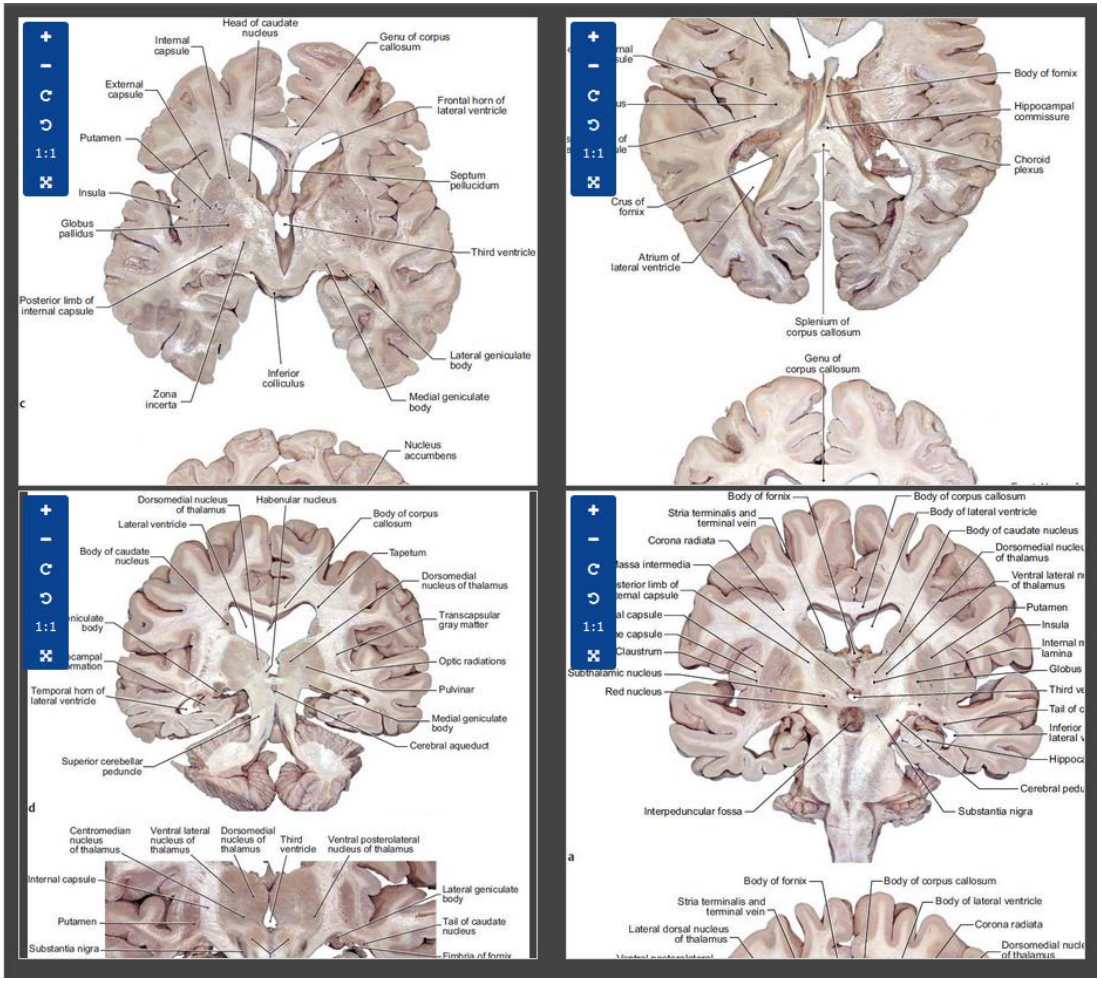


Figure 1.8. Superior view of the relationship of the cerebral **(a)** arteries and **(b)** veins to the lateral and third ventricles.
Source: Color Atlas of Brainstem Surgery > Internal Anatomy of the Brainstem

Compare images for enhanced functionality





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