# EPIDURAL HEMATOMA

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# EPIDURAL HEMATOMA

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## 1. Core Definition

An **Epidural Hematoma** (EDH), often referred to interchangeably as an extradural hematoma, constitutes a severe and life-threatening form of traumatic brain injury characterized by the accumulation of blood within the potential space situated between the inner surface of the skull bones and the outer layer of the dura mater, known as the epidural space. This condition is distinct from a subdural hematoma, where blood collects beneath the dura mater. The defining feature of EDH is the source of the hemorrhage, which is overwhelmingly arterial, resulting from the rupture of a high-pressure blood vessel, most commonly the Middle Meningeal Artery (MMA) or one of its major branches. Because arterial bleeding is rapid and voluminous, EDH typically progresses acutely and rapidly, leading to significant mass effect and a subsequent swift rise in Intracranial Pressure (ICP). Untreated, the resulting compression and displacement of brain tissue (herniation) lead quickly to irreversible neurological damage or death, making EDH an acute neurosurgical emergency requiring immediate intervention. The initial definition, as exemplified by the simple scenario of a blow to the head causing rapid bleeding, underscores the necessity of fast diagnosis following high-impact trauma.

The distinction between the epidural space and the subdural space is critical for understanding the clinical trajectory and radiographic appearance of EDH. In the cranial cavity, the dura mater is normally closely adherent to the periosteum of the skull, meaning the epidural space is considered a potential space rather than a true anatomical cavity. The accumulation of blood in this space forcefully strips the dura away from the skull, a process requiring considerable pressure. This dynamic explains the characteristic biconvex or lens shape appearance observed on neuroimaging, as the collection is contained laterally by the firm attachments of the dura to the sutures of the skull. This containment mechanism limits the spread of the hematoma across suture lines, which helps distinguish EDH from subdural hematomas which typically cross these boundaries. The hematoma itself acts as an expanding mass lesion, displacing cerebrospinal fluid (CSF) and venous blood, and eventually causing fatal compression of vital brain structures, primarily within the brainstem.

From a pathophysiological perspective, the rapid expansion of the hematoma creates a positive feedback loop of cerebral insult. The increasing ICP compromises cerebral perfusion pressure (CPP), reducing the blood flow available to brain tissue (ischemia). Simultaneously, the direct mechanical compression causes local tissue damage and potentially shifts midline structures, leading to transtentorial or uncal herniation, which manifests as critical neurological deterioration, including fixed and dilated pupils due to oculomotor nerve compression. While EDH accounts for a

relatively small percentage (1%-3%) of all head injuries, its high mortality rate if management is delayed--often exceeding 30%--demands specialized expertise in both emergency medicine and neurosurgery. Early recognition of the mechanism of injury, paired with rapid assessment of the patient's neurological status using the Glasgow Coma Scale (GCS), is paramount to achieving a positive outcome.

## 2. Anatomical and Pathophysiological Basis

The anatomical location of the epidural hematoma dictates its clinical severity. The cranium comprises several layers of protection for the central nervous system, starting externally with the scalp and skull, followed internally by the three meningeal layers: the dura mater, the arachnoid mater, and the pia mater. The dura mater is a thick, tough, fibrous membrane consisting of two layers: an outer periosteal layer, which adheres to the inner surface of the skull, and an inner meningeal layer. The epidural space, where the hematoma forms, exists between the skull and the periosteal layer of the dura. The blood supply to the dura itself is crucial; the Middle Meningeal Artery (MMA), a major branch of the maxillary artery, runs in a groove on the inner surface of the temporal bone, often providing a weak point susceptible to trauma. The vulnerability of the MMA, particularly where it crosses the thin squamous part of the temporal bone (pterion), is the primary reason why trauma to the lateral head is the most frequent cause of EDH.

The mechanism of injury typically involves a severe lateral impact that results in a linear skull fracture spanning the course of the MMA. While the fracture itself may appear minor, the associated displacement or fracture edges can lacerate the high-pressure artery or adjacent venous sinuses, initiating rapid hemorrhage. The arterial nature of the bleed ensures that blood pressure continues to drive the hematoma's expansion, despite the increasing counter-pressure exerted by the developing hematoma and the stripping of the adherent dura. In contrast to subdural hematomas, which usually involve low-pressure bridging veins and develop more slowly, the high-pressure arterial bleed in EDH can accumulate 50 to 100 milliliters of blood within minutes to hours, sufficient to cause acute and irreversible brain compression. This rapid accumulation is the core reason for the often dramatic and sudden neurological decline witnessed in these patients.

A less common but important pathophysiological variant involves venous bleeding, typically originating from a torn dural sinus, such as the superior sagittal sinus. Venous EDHs tend to occur more frequently in the posterior fossa or vertex regions and generally progress much slower than arterial EDHs, sometimes taking days to become symptomatic. However, the vast majority of clinically significant epidural hematomas are arterial, particularly in adults. The characteristic appearance of the hematoma--the biconvex, lenticular shape--is due to the forceful stripping of the dura mater away from the skull. Since the dura is firmly fused at the cranial sutures, the hematoma cannot easily cross these boundaries, resulting in the localized, defined shape that is

pathognomonic for EDH on computed tomography (CT) imaging. Understanding the dynamics of this space--defined by high pressure and anatomical containment--is essential for interpreting imaging results and predicting the speed of deterioration.

## 3. Etiology and Common Causes

The etiology of epidural hematoma is overwhelmingly linked to severe physical trauma, accounting for over 90% of cases. The most common scenario involves blunt force trauma to the head, often sustained during motor vehicle accidents, pedestrian collisions, falls from significant heights, or assaults involving strikes to the lateral aspect of the skull. The specific point of vulnerability is often the pterion, a region of the skull where the frontal, parietal, temporal, and sphenoid bones meet in an H-shaped junction, making it structurally weak and lying directly over the path of the Middle Meningeal Artery. A forceful impact here frequently results in a skull fracture that lacerates the MMA, initiating the hemorrhage. The relationship between skull fracture and EDH is strong: approximately 75% to 95% of patients diagnosed with an EDH will also have an associated overlying skull fracture, often a linear fracture through the temporal or temporoparietal bone.

While trauma is the primary cause, the characteristics of the patient population affected by EDH are notable. EDH is most commonly observed in children, adolescents, and young adults (typically under the age of 30) who are involved in high-velocity trauma, primarily because their dura mater is less adherent to the inner skull compared to that of older adults. This weaker adherence makes the dura easier to strip away by arterial pressure. Conversely, infants and the elderly tend to have a lower incidence of EDH. In infants, the skull sutures are not yet fused, potentially allowing decompression through the fontanelles, while in the elderly, the dura is often more fibrously fused to the skull, requiring greater force to cause stripping and bleeding. These demographic differences highlight the crucial role of biomechanics and anatomical adherence in the development of the condition.

In rare instances, non-traumatic causes of epidural hematoma have been documented, although these are exceptions rather than the rule. Non-traumatic causes might include underlying vascular abnormalities such as arteriovenous malformations (AVMs), bleeding diatheses (coagulopathies), or, very rarely, complications following spontaneous rupture of a dural vessel or complications from central nervous system infections. However, when evaluating a patient presenting with classic EDH symptoms, particularly the presence of a lucid interval, the clinical presumption must always default to traumatic etiology, necessitating immediate trauma protocol activation and neuroimaging to rule out the arterial bleed. The rapid assessment of the mechanism of injury is often the most critical piece of diagnostic information available to the emergency medical team.

## 4. Clinical Presentation and Diagnosis

The clinical presentation of an epidural hematoma is classically described by the presence of a **lucid interval**. This phenomenon refers to a period immediately following the traumatic event during which the patient is concussed or briefly unconscious, but then regains consciousness and appears relatively normal or minimally symptomatic for a period ranging from minutes to hours. This deceptive period occurs because the brain has temporarily accommodated the initial injury. However, as the arterial hemorrhage rapidly expands, the ICP quickly reaches a critical threshold, leading to sudden, catastrophic neurological deterioration, including rapid onset of severe headache, persistent vomiting, altered mental status, hemiparesis (weakness on one side of the body), and eventually, ipsilateral pupil dilation due to uncal herniation (compression of the third cranial nerve). It is the potential for this rapid progression from apparent stability to deep coma that makes the lucid interval such a dangerous and defining feature of EDH.

Definitive diagnosis relies almost exclusively on immediate neuroimaging, specifically a non-contrast Computed Tomography (CT) scan of the head. The CT scan is essential because it can rapidly visualize the location, size, and shape of the hematoma, as well as identify associated injuries like skull fractures or underlying brain contusions. The pathognomonic radiographic finding for EDH is a hyperdense (bright white, signifying acute blood) collection that exhibits a **biconvex** or lenticular shape, tightly conforming to the inner curvature of the skull but strictly respecting the cranial suture lines. Measuring the volume of the hematoma and the degree of midline shift (how far the brain has been pushed across the midline) are critical parameters derived from the CT scan that inform immediate surgical decision-making. Significant midline shift (typically greater than 5 mm) or large hematoma volume necessitates immediate surgical decompression, regardless of the patient's GCS score.

Further diagnostic considerations involve continuous monitoring of the patient's neurological status and vital signs, particularly blood pressure and respiratory effort, as signs of Cushing's triad (hypertension, bradycardia, and irregular respiration) suggest impending or established brainstem compression. While Magnetic Resonance Imaging (MRI) offers higher detail of soft tissue, it is usually impractical in the acute trauma setting due to time constraints and the need for immediate intervention. The speed and accessibility of the CT scanner in a trauma center are indispensable tools for managing EDH. Laboratory tests, including a complete blood count and coagulation studies (e.g., INR, PTT), are essential to identify any underlying coagulopathy that might complicate bleeding control or surgical management, ensuring that necessary clotting factors or reversal agents are prepared immediately upon diagnosis.

## 5. Management and Treatment Protocols

The management of Epidural Hematoma is a critical race against time, centered on stabilizing the patient, controlling intracranial pressure, and surgically evacuating the hematoma. Initial treatment begins with standard trauma resuscitation protocols (ABCs: Airway, Breathing, Circulation).

Maintaining adequate oxygenation and ventilation is paramount, as hypoxia can severely exacerbate secondary brain injury. Fluid resuscitation must be managed carefully to maintain adequate cerebral perfusion pressure (CPP) without causing excessive hemodilution or hypertension that could worsen cerebral edema. In cases of significant neurological decline (GCS less than 8), endotracheal intubation and controlled hyperventilation may be initiated briefly to acutely decrease ICP while preparing for surgery, although prolonged hyperventilation is avoided due to the risk of cerebral vasoconstriction and ischemia.

The definitive treatment for a symptomatic or large EDH is immediate surgical evacuation. The procedure of choice is a **craniotomy**, where a section of the skull bone (bone flap) is removed directly over the hematoma, allowing the neurosurgeon to rapidly access and remove the clotted blood and identify the source of the bleeding, which is then cauterized (usually the Middle Meningeal Artery). The urgency of this procedure cannot be overstated; the time from diagnosis to decompression is a primary determinant of outcome. In extremely critical situations, where neurological deterioration is rapid and immediate transport to an operating room is delayed, a bedside placement of a burr hole (a small hole drilled into the skull) may be performed in the emergency department to provide temporary relief of pressure, especially if the location of the hematoma is clearly established as temporal. However, burr holes rarely suffice for definitive evacuation of a solid, clotted arterial hematoma.

Following surgical evacuation, careful postoperative management is required, focusing on preventing complications such as re-bleeding, infection, seizures, and cerebral edema. Patients are typically admitted to the neurosurgical intensive care unit (NICU) for continuous monitoring of ICP, blood pressure, and neurological status. The surgical success rate is high when performed promptly, but the patient's long-term functional recovery is highly dependent on their pre-operative neurological status. Patients who are awake or only moderately impaired prior to surgery (GCS 13-15) generally have an excellent prognosis, whereas those who are deeply comatose or presenting with fixed, dilated pupils have a significantly poorer outcome due to the severe, irreversible brain damage sustained during the period of high ICP.

## 6. Prognosis and Complications

The prognosis following an epidural hematoma is highly variable and directly correlates with the timing of surgical intervention and the patient's neurological state immediately prior to surgery. The "talk and die" syndrome, though rare, vividly illustrates the rapid progression of this injury: a patient who initially appears fine (the lucid interval) suffers a sudden, fatal deterioration due to delayed recognition and intervention. For patients who undergo rapid evacuation while maintaining a good GCS score (GCS 13-15), the expected outcome is generally favorable, with most individuals recovering fully or with only minor long-term deficits. However, if the patient presents with a GCS score below 8 or signs of fixed, dilated pupils--indicating established brain herniation--the mortality

rate can exceed 50%, and survivors often face significant long-term neurological disability.

Complications associated with EDH are numerous and include secondary brain injury mechanisms. The most immediate complication is uncal or transtentorial herniation, leading to brainstem compression and subsequent cardiorespiratory failure. Even if surgically managed, patients may suffer from post-traumatic epilepsy (seizures), focal neurological deficits (such as persistent weakness or sensory loss), cognitive impairment, and hydrocephalus (accumulation of CSF). The volume of the hematoma, the speed of its formation, and the degree of associated underlying brain injury (e.g., contusion) are all major predictive factors of long-term disability. Advances in trauma systems, rapid CT scanning access, and prompt neurosurgical response have collectively improved outcomes for EDH patients over the past few decades.

Long-term rehabilitation is often necessary for survivors, especially those who experienced prolonged periods of elevated ICP or significant neurological deficits. This rehabilitation may involve physical therapy, occupational therapy, and speech therapy, tailored to address cognitive deficits, motor impairments, and functional independence. Given that EDH primarily affects young, otherwise healthy individuals, the potential years of life lost and the long-term burden of disability emphasize the need for continued public health efforts focused on prevention of high-velocity trauma, such as promoting helmet use and improving road safety standards. The sustained effort in both acute surgical response and chronic rehabilitative care defines the full scope of managing the consequences of this critical injury.

## Further Reading

Epidural Hematoma (Wikipedia)
Middle Meningeal Artery
Craniotomy
Intracranial Pressure
Glasgow Coma Scale