Comprehensive Analysis of Seizure Outcomes, Recurrence Risks, and Prognostic Factors Following Partial Temporal Lobectomy and Amygdalohippocampectomy

Executive Abstract

The surgical resection of the anterior temporal lobe (ATL) and the amygdala, often involving the hippocampus, constitutes the primary therapeutic intervention for drug-resistant mesial temporal lobe epilepsy (mTLE) and a common procedure for temporal lobe neoplasms. The "chances of epilepsy" in this context refers to two distinct probabilistic outcomes: the likelihood of *continued* or *recurrent* seizures in patients with established epilepsy (surgical failure), and the risk of *de novo* epilepsy in patients undergoing resection for non-epileptic pathologies.

This report synthesizes data from extensive clinical cohorts to provide a definitive risk profile. In patients with drug-resistant epilepsy, short-term (1–5 year) seizure freedom rates (Engel Class I) range from 60% to 80%.[1] However, longitudinal surveillance reveals a progressive attrition in efficacy, with seizure-free probabilities declining to 41–56% by the 10-year mark and stabilizing between 30–65% at 20 years, depending on the definition of remission.[4] Key determinants of success include the extent of resection—specifically the inclusion of the piriform cortex—the presence of a discrete MRI lesion, and the duration of preoperative epilepsy. Conversely, for patients undergoing resection for incidental tumors without prior seizures, the risk of developing postoperative epilepsy is approximately 13–20%, modulated by tumor grade and surgical approach.[7]

This document serves as an exhaustive reference for clinicians and researchers, dissecting the anatomical, pathological, and neuropsychological variables that define these outcomes.

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1. Introduction: The Clinical Imperative of Temporal Lobe Resection

The temporal lobe, particularly its mesial structures including the amygdala, hippocampus,

and parahippocampal gyrus, is the most common site of origin for focal epilepsies in adults. Temporal Lobe Epilepsy (TLE) is frequently resistant to pharmacotherapy; epidemiological data suggests that up to one-third of epilepsy patients become medically intractable, necessitating surgical intervention to mitigate the risks of cognitive decline, injury, and Sudden Unexpected Death in Epilepsy (SUDEP).[9]

The user's inquiry regarding the "chances of epilepsy" following the removal of these structures necessitates a bifurcated analysis. For the epilepsy patient, the question is one of efficacy: "What are the chances my seizures will stop?" For the neuro-oncology patient, the question is one of iatrogenic risk: "What are the chances this surgery will cause seizures?"

Surgical strategies have evolved from the standard en bloc Anterior Temporal Lobectomy (ATL), which removes a substantial portion of the lateral neocortex along with the mesial structures, to more refined techniques like Selective Amygdalohippocampectomy (SelAH), which aims to spare the neocortex to preserve neurocognitive function. Understanding the interplay between these techniques and the complex neural networks of the limbic system is essential for interpreting the statistical outcomes presented herein.

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2. The Surgical Landscape: Techniques and Anatomical Targets

To accurately assess the probability of postoperative epilepsy, one must first delineate the surgical variables, as the extent of resection is a primary independent variable influencing both seizure control and functional deficit.

2.1 Anterior Temporal Lobectomy (ATL)

The standard ATL is the most historically validated procedure for mTLE. It involves the resection of the anterior 3.5 to 4.5 cm of the temporal neocortex, the uncus, the amygdala, and the anterior 2.0 to 3.0 cm of the hippocampus.

- **Anatomical Scope:** By removing the lateral neocortex, ATL addresses potential epileptogenic foci in the temporal pole and lateral gyri that may act as propagation pathways or independent seizure generators.
- **Theoretical Advantage:** The comprehensive nature of ATL is hypothesized to provide a "safety margin" against recurrence by removing the maximum volume of potentially epileptogenic tissue. This is supported by meta-analyses suggesting an odds ratio (OR) of 0.38 favoring ATL over selective approaches for seizure freedom in certain cohorts.[11]

2.2 Selective Amygdalohippocampectomy (SelAH)

Developed to minimize the cognitive morbidity associated with neocortical resection, SelAH

targets the mesial structures through a transcortical or trans-sylvian corridor, leaving the lateral temporal lobe intact.

- **Functional Preservation:** The primary rationale is the preservation of the superior temporal gyrus (associated with auditory processing and language) and the inferolateral cortex (associated with visual memory).
- **Controversy:** The debate persists regarding whether sparing the neocortex compromises seizure control. While some studies show comparable outcomes [12], others indicate a trend toward earlier recurrence in SelAH patients due to residual epileptogenic tissue in the temporal stem or piriform cortex.[11]

2.3 The Amygdala as a Specific Target

The amygdala is central to the generation of the affective auras (fear, panic, epigastric rising) characteristic of mTLE. Its complete resection is anatomically challenging due to its superior extension into the globus pallidus and optic tract.

- **Resection Extent:** The correlation between the volume of amygdala resected and seizure outcome remains complex. Retrospective analyses have yielded conflicting results; some cohorts show that complete amygdala resection confers a slightly higher, though not statistically significant, chance of seizure freedom compared to partial resection.[14] Others suggest that in cases of anterotemporal foci, limited resection does not necessarily predict failure if the primary epileptogenic zone is hippocampal.[16]
- **Recent Volumetric Data:** More modern imaging studies using voxel-based morphometry suggest that while the extent of amygdala resection *per se* may not be the sole predictor, the resection of the adjacent **piriform cortex** is critical, a finding that will be explored in depth in Section 7.[17]

2.4 Laser Interstitial Thermal Therapy (LITT)

A newer, minimally invasive modality, LITT uses MRI-guided laser ablation to destroy the amygdala and hippocampus. While less invasive, initial meta-analyses suggest its efficacy (approx. 59% seizure freedom) may be slightly lower than open resection (approx. 70–72%), potentially due to the difficulty in achieving complete ablation of the amygdalohippocampal complex and the inability to resect the piriform cortex or entorhinal cortex fully.[19]

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3. Seizure Freedom in Drug-Resistant Epilepsy: The Statistical Reality

The "chance of epilepsy" remaining after surgery is the inverse of the "seizure freedom rate." The gold standard for reporting these outcomes is the Engel Classification system, where Class I represents freedom from disabling seizures.

3.1 Short-Term Outcomes (1–5 Years)

In the initial years following resection, the probability of seizure freedom is robust, reflecting the immediate disruption of the epileptogenic network.

- **Aggregate Efficacy:** A systematic review of outcomes indicates that approximately **82%** of patients achieve a satisfactory outcome (Engel Class I or II) after a mean follow-up of 64 months.[1]
- **Complete Freedom (Engel Ia):** When the definition is tightened to Engel Class Ia (completely seizure-free, no auras, no withdrawal seizures), the rate is approximately **51.18%** at 5 years.[1] This distinction is vital for patient counseling; while nearly 80% experience a life-changing reduction in seizures, only half are "cured" in the strictest sense.
- **Comparative Efficacy:** In a meta-analysis of 218 patients, pooled results showed a statistically significant reduction in seizure recurrence for ATL compared to SelAH (OR 0.38; p=0.008).[11] However, a separate systematic review of 983 patients found no significant difference (OR 1.12; p=0.53), suggesting that surgeon experience and patient selection may eclipse the technical differences between the two procedures.[12]

3.2 Pathology-Specific Probabilities

The underlying histology is the strongest predictor of the "chance of epilepsy" persisting.

- **Hippocampal Sclerosis (HS):** The classic indication for TLE surgery. Seizure freedom rates are consistently high, reported at **77.9%** in meta-analyses.[20]
- **Tumors (LEATs):** Long-term Epilepsy Associated Tumors (e.g., gangliogliomas, DNETs) have the most favorable prognosis. When gross total resection is achieved, seizure freedom rates range from **79% to 93%**.[20] The presence of a tumor allows for a clearly defined surgical margin, unlike the diffuse gliosis often seen in HS or dysplasia.
- **Vascular Malformations:** For cavernous malformations and AVMs, seizure control is excellent. One study cited a **93%** freedom from disabling seizures after resection.[21]
- **Non-Lesional (MRI-Negative) TLE:** This group presents the highest risk of failure. Seizure freedom rates are significantly lower, often ranging from **35% to 50%**.[23] The inability to visualize the focus increases the probability of incomplete resection of the epileptogenic zone.

3.3 Table: Seizure Freedom Rates by Pathology (1–5 Years)

Pathology	Seizure Freedom Rate (Engel I)	Prognostic Notes	Source
Tumor (Low	**79% – 93%**	Best outcomes with	[9]

Grade)		Gross Total Resection (GTR).	
Hippocampal Sclerosis	**~78%**	Standard indication; favorable outcomes.	[20]
Vascular Malformation	**~93%**	Excellent control; focus is discrete.	[21]
Focal Cortical Dysplasia	**~50% – 60%**	Lower due to diffuse/microscopic margins.	[20]
Non-Lesional (MRI Neg)	**35% – 51%**	Highest risk of continued epilepsy.	[23]

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4. Longitudinal Trajectories: The Decay of Seizure Freedom Over Decades

A crucial and often under-discussed aspect of epilepsy surgery is the durability of the cure. The "chance of epilepsy" increases as the postoperative interval lengthens. Longitudinal studies reveal a phenomenon of "late recurrence" that challenges the notion of a permanent surgical cure for all patients.

4.1 The 10-Year Benchmark

As patients are followed into the second decade post-surgery, attrition in seizure control becomes evident.

- **Statistical Decline:** Studies tracking patients for ten years show seizure-free rates declining from the initial highs of ~70% down to **41% to 56%**.[4]
- **Relapse Characteristics:** Interestingly, among those who relapse, the epilepsy is often less severe than preoperative levels. One study noted that 51% of relapsers experienced one or fewer seizures per year.[4]
- **Survival Analysis:** Kaplan-Meier analyses estimate the probability of seizure freedom as **51%** at 10 years in mixed cohorts.[5] This suggests that for a patient walking out of the hospital seizure-free, there is roughly a coin-flip chance they will remain so a decade later.

4.2 The 20-Year Benchmark and Beyond

Data extending beyond two decades is rarer but illuminating.

- **Continuous vs. Terminal Freedom:** The definition of success matters immensely here. If "seizure freedom" is defined as *never having a single seizure since surgery*, rates drop to **30–37%** at 20 years.[4] However, if defined as "seizure-free at the last follow-up visit" (allowing for a period of relapse that was subsequently controlled), rates are higher, around **65%**.[2]
- **Predictors of Late Recurrence:** Patients with a longer duration of epilepsy prior to surgery (>20 years) are significantly more likely to experience late failure. In one study, only **37%** of patients with >20 years of preoperative epilepsy remained continuously seizure-free at 10 years, compared to **69%** of those with a shorter history.[4] This underscores the pathogenicity of the epileptic network itself—chronic seizures may "kindle" secondary foci that eventually become independent generators.

4.3 Table: Longitudinal Decay of Seizure Freedom

Time Post-Surgery	Seizure Freedom Probability	Key Insight	Source
2 Years	**55% – 75%**	Period of highest risk for early failure.	[5]
5 Years	**47% – 67%**	Stabilization of the "cured" cohort.	[1]
10 Years	**41% – 56%**	Significant attrition; late recurrences emerge.	[4]
20 Years	**30% – 65%**	Outcomes diverge based on continuous vs. terminal definitions.	[2]

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5. The "Running Down" Phenomenon and Late Remission

While the general trend is outcome attrition, a subset of patients defies this trajectory through the "running down" phenomenon. This refers to a scenario where patients continue to have seizures immediately after surgery, but these seizures progressively diminish in frequency and intensity over time, eventually leading to remission.

5.1 Prevalence and Mechanism

- **Incidence:** Approximately **12%** of patients may exhibit this phenomenon.[29] It is distinct from immediate surgical failure.
- **Pathophysiology:** The running down phenomenon is thought to represent the gradual dying out of "kindled" excitability in neural networks distant from the resection site. Once the primary driver (the amygdala/hippocampus) is removed, the secondary networks may initially sustain seizures but lack the drive to maintain them indefinitely, leading to gradual extinction.[30]

5.2 Clinical Implications

This phenomenon complicates the assessment of "chances of epilepsy." A patient who has seizures at 6 months post-op might still become seizure-free at 2 years.

- **Focal Cortical Dysplasia (FCD):** This phenomenon is notably observed in children with FCD, where the seizure-free rate was 49% in the first year but the "running down" mechanism contributed to seizure alleviation in long-term follow-up.[25]
- **Prognostic Factors:** Patients exhibiting this phenomenon often have intermediate-sized epileptogenic areas. Those with very large, diffuse epileptogenic zones tend not to run down but rather continue to have frequent seizures, while those with discrete, small zones are cured immediately.[32]

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6. De Novo Epilepsy: Seizure Risks in Tumor Resection and Non-Epileptic Conditions

For patients undergoing temporal lobe resection for indications *other* than epilepsy—such as the removal of an incidental glioma or a vascular malformation—the surgical risk profile is inverted. The concern is not curing epilepsy, but avoiding its induction (de novo epilepsy).

6.1 The Incidence of Post-Craniotomy Seizures

Any neurosurgical intervention involving the cortex carries a risk of epileptogenesis due to the formation of a glial scar, hemosiderin deposition from micro-bleeds, and local inflammation.

- **Statistical Risk:** Large-scale population studies indicate that the cumulative risk of de novo epilepsy after craniotomy is **13.9%** at 1 year and rises to **20.4%** at 5 years.[7] This "1 in 5" risk is a significant consideration for patients with incidental findings.
- **Comparison to Background Risk:** This rate is markedly higher than the general

population and varies significantly by the indication for surgery.

6.2 Tumor-Specific Risks

The "chance of epilepsy" is heavily modulated by the nature of the lesion being removed.

- **Preoperative Seizures:** The strongest predictor of postoperative epilepsy is the presence of preoperative seizures. Patients who have even a single seizure before surgery have a much higher likelihood of requiring long-term anti-seizure medication (ASM).[33]
- **Tumor Grade:** Paradoxically, patients with *low-grade* tumors (WHO Grade I and II) often have higher rates of epilepsy than those with high-grade tumors (glioblastoma). This is likely because low-grade tumors grow slowly, allowing time for extensive peritumoral network reorganization and epileptogenesis, whereas high-grade tumors destroy tissue too rapidly to establish stable epileptic networks.[8]
- **Surgical Nuance:** In a study of meningioma and glioma resections, **12.5%** of glioma patients and **15.0%** of meningioma patients developed post-craniotomy seizures.[33]

6.3 Prophylaxis and Long-Term Management

- **Medication Withdrawal:** For patients who present with a tumor-related seizure, undergo resection, and remain seizure-free, the question of when to stop medication is critical. Data suggests that about **two-thirds** of such patients can eventually discontinue ASMs. However, **one-third** will require indefinite treatment.[22]
- **Acute Post-Op Seizures:** Seizures occurring in the first week after tumor resection are common and often provoked by acute factors (edema, blood). While they are a risk factor for future epilepsy, they do not guarantee it. Current guidelines often recommend a short course of ASMs (1–2 weeks) post-surgery, but long-term prophylaxis in seizure-naïve patients is generally not supported by Class I evidence.[22]

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7. Predictors of Outcome: The Role of the Piriform Cortex and Extent of Resection

Why do some surgeries fail while others succeed? Conventional wisdom focused on the volume of the hippocampus removed. However, cutting-edge research has shifted focus to the **piriform cortex**.

7.1 The Piriform Cortex: The Critical Node

The piriform cortex, located in the anterior temporal lobe adjacent to the amygdala, is chemically highly excitable and serves as a central hub for limbic seizures.

- **Validation Studies:** Recent multicenter validation studies have demonstrated that the extent of hippocampal or amygdala resection *alone* is often not significantly different

between cured and non-cured patients. Instead, the volume of piriform cortex resected separates the groups.[17]

- **The 50% Threshold:** Removing at least **50% of the piriform cortex** has been shown to increase the odds of seizure freedom by a factor of **16**.[18]
- **Data Divergence:** In patients with favorable outcomes, the median resected proportional volume of the piriform cortex was **51%** (IQR 42–61%), compared to only **13%** (IQR 11–18%) in those who continued to have seizures.[35] This suggests that many "failures" of SelAH or ATL may technically be failures to address the piriform cortex.

7.2 Incomplete Resection and Residual Tissue

The concept of "Gross Total Resection" (GTR) is vital, particularly in lesional epilepsy.

- **Tumor Surgery:** Subtotal resection is a major predictor of continued epilepsy. In glioneuronal tumors, GTR resulted in **79%** seizure freedom vs. **43%** for subtotal resection.[21]
- **Mesial Structures:** While some older studies suggested sparing the tail of the hippocampus or parts of the amygdala was acceptable [15], the aggregation of modern data implies that "more is better" regarding the epileptogenic zone, provided functional cortex is spared. The "temporal plus" epilepsies, where the network extends to the temporal pole or insula, often fail standard resections because the surgery is too conservative.[38]

7.3 MRI Status: The "Lesional" Advantage

The presence of a lesion on MRI is arguably the most robust conventional predictor of success.

- **The Disparity:** Patients with MRI-visible lesions (HS, tumors) have seizure freedom rates of **~70–80%**. In stark contrast, MRI-negative patients struggle to surpass **50%** freedom rates.[23]
- **Mechanism:** In MRI-negative cases, the surgeon is relying on electrophysiological data (EEG, SEEG) to define boundaries, which is inherently less precise than anatomical margins. This increases the probability of leaving epileptogenic tissue behind.

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8. Histopathology as a Determinant of Risk

The "chance of epilepsy" is biologically encoded in the tissue being removed.

8.1 Hippocampal Sclerosis (HS)

HS is characterized by neuronal loss and gliosis in the CA1, CA3, and hilar regions. It is a progressive pathology often linked to a history of febrile seizures.

- **Outcomes:** HS responds exceptionally well to surgery, with long-term seizure freedom often reported in the **70–85%** range.[20] The pathology is typically confined to the mesial structures, making it an ideal target for ATL/SelAH.

8.2 Focal Cortical Dysplasia (FCD)

FCD involves abnormal neuronal migration and organization. It is often the culprit in MRI-negative cases (Type I) or subtle lesional cases (Type II).

- **Risk Profile:** FCD has a higher rate of recurrence than HS or tumors, often due to the difficulty in visualizing the full extent of the dysplastic network. Seizure freedom rates hover around **50–60%.**[25] The "running down" phenomenon is more common here, but so is late recurrence if the dysplasia extends beyond the resection margin.

8.3 Long-Term Epilepsy Associated Tumors (LEATs)

- **Prognosis:** These benign tumors (gangliogliomas, DNETs) are highly epileptogenic but surgically curable. They offer the best "chances" for the patient, with cure rates often exceeding **80–90%.**[20] The surgery is often a lesionectomy plus corticectomy, and the distinct borders of the tumor facilitate complete removal.

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9. Pediatric vs. Adult Outcomes: Age-Related Plasticity and Prognosis

The age at which the temporal lobe and amygdala are removed significantly influences the "chances of epilepsy" resolving.

9.1 The Pediatric Advantage

Children often exhibit better surgical outcomes than adults, a finding attributed to greater neural plasticity and a shorter duration of the epileptic network's existence.

- **Statistical Superiority:** Some studies report pediatric seizure freedom rates as high as **92.1%** following temporal lobe surgery, significantly outperforming adult cohorts in similar settings.[3] Other large meta-analyses show a trend toward better outcomes in children (Engel I rates of **74%** vs **69%** in adults), though this does not always reach statistical significance across all studies.[42]
- **Histopathology Influence:** In children, the type of surgery and underlying pathology (often dysplasia or tumor rather than sclerosis) play a larger role in determining outcome than in adults.[42]

9.2 The "Time is Brain" Hypothesis

The duration of epilepsy before surgery is a critical variable that intersects with age.

- **Early Intervention:** Surgery performed earlier in the disease course (<20 years duration) yields significantly better results. The probability of being seizure-free at 10 years is nearly double for those with a short history compared to those with a chronic history (>20 years).[4]
- **Cognitive Rescue:** In children, early surgery not only stops seizures but prevents the "epileptic encephalopathy" that degrades cognitive potential. This makes the risk/benefit calculation heavily weighted toward early, aggressive resection.[43]

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10. Neuropsychological Sequelae: Memory, Language, and the Amygdala's Role in Emotion

The removal of the temporal lobe and amygdala is an exchange: seizure freedom is bought at the cost of neural tissue involved in memory and emotion.

10.1 Memory Decline

The hippocampus and parahippocampal gyrus are the engines of episodic memory. Their resection carries known risks.

- **Verbal Memory (Dominant Hemisphere):** Resection of the language-dominant (usually left) temporal lobe is the highest-risk procedure for memory. Significant verbal memory decline occurs in up to **47%** of ATL patients.[9] Selective procedures (SelAH) reduce this risk to approximately **14%**, although they may carry slightly higher seizure recurrence risks in some datasets.[1]
- **Visual Memory (Non-Dominant Hemisphere):** Resection of the right temporal lobe is associated with deficits in visual memory (e.g., spatial navigation, face recognition), though these deficits are often less functionally debilitating than verbal memory loss.[9]

10.2 The Amygdala and Emotional Processing

The amygdala is famous as the brain's "fear center." What happens when it is removed?

- **Emotion Recognition:** Patients often exhibit deficits in recognizing negative emotional expressions, particularly fearful faces. This is a specific, localized deficit directly related to amygdala loss.[45]
- **Emotional Flattening?** Contrary to fears of emotional blunting, many patients experience *improved* emotional regulation. This is likely because the epileptic amygdala was not functioning normally but was instead a source of pathologic affective storms.

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11. Psychiatric Outcomes: Anxiety, Depression, and

the Burden of Normality

The "chance of epilepsy" is often entwined with psychiatric comorbidities. Surgery alters this landscape.

11.1 De Novo Anxiety and Depression

Postoperative psychiatric symptoms are common, affecting up to **50%** of patients in the acute phase.[45]

- **Mechanism:** This can be biological (forced normalization of networks, withdrawal of psychotropic ASMs) and psychosocial.
- **The Burden of Normality:** Patients who have lived with epilepsy for decades may struggle to adjust to a seizure-free life—a phenomenon known as the "burden of normality." The loss of the "sick role" and the new expectations of independence can trigger anxiety and depression.

11.2 Mood Improvement

Despite these risks, successful surgery is frequently associated with long-term mood improvement.

- **Right Amygdala Effect:** Fascinatingly, resection of the **right amygdala** has been statistically associated with improvements in mood and reductions in depression scores ($r = 0.5$, $p = 0.008$).[46] This suggests a lateralization of emotional processing where the right amygdala may mediate negative affect, and its removal alleviates this burden.
- **Contrast with Hippocampus:** In the same studies, resection of the right hippocampus was associated with *worsened* mood, highlighting the intricate balance of the limbic system.[46]

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12. Mortality and SUDEP: The Survival Benefit of Surgery

Ultimately, the risks of surgery must be weighed against the risks of *uncontrolled* epilepsy. The most severe risk is Sudden Unexpected Death in Epilepsy (SUDEP).

12.1 The Mortality Gap

Uncontrolled epilepsy confers a mortality rate approximately 3 times higher than the general population. Surgery narrows this gap.

- **Data:** In a large cohort, surgically treated patients had a mortality rate of **8.6 per 1,000 person-years** compared to **25.3 per 1,000 person-years** in non-surgical candidates.[49]
- **Seizure Freedom as Protector:** The survival benefit is almost entirely driven by seizure

freedom. Seizure-free patients have a mortality rate (5.2/1000) that approaches the general population, whereas those with persistent seizures after surgery continue to face high risks (10.4/1000).[49]

12.2 SUDEP Reduction

Surgery is a proven prophylactic against SUDEP.

- **Statistics:** The incidence of SUDEP in operated, seizure-free patients is extremely low (**0.07 per 1,000 person-years**) compared to non-operated or failed-surgery patients (**2.0–6.1 per 1,000 person-years**).[50]
- **Time Course:** The protective effect is most pronounced in the first 10 years after surgery. Some data suggests the risk gap may narrow after a decade, possibly due to late recurrences or age-related comorbidities, but the initial survival advantage is substantial.[52]

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13. Conclusion

The "chances of epilepsy" following the removal of the amygdala and part of the temporal lobe are not a singular probability but a dynamic spectrum defined by patient history, anatomy, and pathology.

For the Patient with Drug-Resistant Epilepsy:
The surgery represents a high-probability opportunity for cure.

- **Success:** There is a **~70–80%** chance of becoming seizure-free in the short term, particularly if the patient has a discrete lesion like Hippocampal Sclerosis or a tumor.
- **Attrition:** Patients must be counseled that this probability decays over time. By 10 years, the chance of remaining continuously seizure-free is closer to **50%**. However, even "failed" surgeries often result in significant palliation (fewer seizures).
- **Optimization:** The emerging focus on the **piriform cortex** suggests that future surgeries targeting this area may push success rates higher, potentially breaking the historical 70% ceiling.

For the Patient with a Non-Epileptic Tumor:
The surgery carries an iatrogenic risk.

- **Risk:** There is an approximate **14–20%** chance of developing de novo epilepsy in the years following a craniotomy. This risk is higher for those with preoperative seizures or low-grade gliomas.
- **Management:** While this risk is non-trivial, it is generally manageable with medication, and the primary goal of oncological control typically outweighs the secondary risk of epilepsy.

Final Summary:
Partial temporal lobectomy and amygdalohippocampectomy remain the most effective

interventions for temporal lobe epilepsy. While not a guaranteed permanent cure for every patient, they offer a dramatic reduction in seizure burden, a significant survival benefit via SUDEP prevention, and, for the majority, a life free from the devastating unpredictability of seizures. The integration of advanced outcomes data, particularly regarding the piriform cortex and long-term recurrence, allows for more precise, individualized risk stratification than ever before.

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