Chapter 5

Endoscopic Third Ventriculostomy (ETV) Complications and Management, From Recent Literature

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Abstract

Endoscopic third ventriculostomy (ETV) is an established treatment for obstructive hydrocephalus in both children and adults and is increasingly used as an alternative to cerebrospinal fluid shunting. Contemporary series and systematic reviews suggest an overall complication rate of approximately 5–15%, with permanent morbidity around 2–3% and procedure-related mortality below 1% . Although these rates compare favorably with shunt surgery, the spectrum of possible complications includes rare but catastrophic events such as basilar artery rupture and late sudden deterioration due to stoma closure .

This chapter provides a contemporary, evidence-based overview of ETV complications across pre-operative, intra-operative, early post-operative, and late phases. We emphasize the interaction between patient selection, ventricular and vascular anatomy, intra-operative technique, and long-term follow-up in determining risk. Key topics include hemodynamic instability and bradycardia during floor perforation, hemorrhagic and vascular injury within the interpeduncular cistern, neuroendocrine and electrolyte disturbances, infection, neurocognitive sequelae from fornical or hypothalamic injury, and the mechanisms and clinical patterns of ETV failure. Particular focus is given to third ventricle floor and basilar artery morphometry, the concept of a vascular "safe zone," and emerging strategies to prevent catastrophic

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complications. Rather than viewing complications as idiosyncratic events, we frame them as largely predictable and often preventable outcomes of anatomical, technical, and organizational factors. Protocolized peri-operative care, meticulous anatomical planning, and structured long-term surveillance underpin a modern, systems-based approach to safe ETV.

1. INTRODUCTION

Endoscopic third ventriculostomy (ETV) is now a cornerstone procedure in the management of obstructive hydrocephalus. By creating a fenestration in the floor of the third ventricle, cerebrospinal fluid (CSF) is diverted from the ventricular system into the basal cisterns, bypassing the site of obstruction. In appropriately selected patients, long-term shunt independence can be achieved in roughly 60–90% of cases, with durable patency in many adult and pediatric cohorts [1–3].

Despite its minimally invasive nature, ETV is not a trivial operation. Contemporary large series and systematic reviews report overall complication rates around 8–10%, permanent morbidity of approximately 2–3%, and early procedure-related mortality well below 1% [1,2,5]. Complications include intra-operative hemorrhage, basilar artery rupture, neural structure injury, early post-operative hematomas, CSF leak, central nervous system infections, and procedure failure requiring further CSF diversion [1–5]. While the absolute rates are low, the potential consequences are often devastating, ranging from major neurological deficits to fatal hemorrhage or late sudden deterioration due to stoma occlusion [1,4,5].

The nature and frequency of complications depend heavily on patient age, etiology of hydrocephalus, ventricular size, third ventricle floor anatomy, and the experience of the surgical team [1–5]. Infants and complex hydrocephalus subtypes (e.g., post-infectious, post-hemorrhagic, myelomeningocele) carry higher failure rates and a different complication profile compared with older children and adults [1–3,5]. These nuances underscore the need for individualized risk assessment rather than a one-size-fits-all approach to indications.

In this chapter, we synthesize contemporary evidence on complications of ETV and organize them along a temporal continuum: pre-operative risk stratification and anatomical planning; intra-operative vascular, neural, and technical events; early post-operative and physiological complications; and late complications related to ETV failure and stoma closure. For each category we highlight mechanisms, incidence, clinical presentation, and evidence-based prevention and management strategies, integrating data from

large series, systematic reviews, and recent anatomical and morphometric studies [1–8].

2. PRE-OPERATIVE RISK STRATIFICATION AND PLANNING

2.1. Patient Selection and Predictors of Success

The first determinant of complication risk is whether ETV is the appropriate procedure for the individual patient. Several models, including the Endoscopic Third Ventriculostomy Success Score (ETVSS), incorporate age, hydrocephalus etiology, and shunt history to estimate the probability of shunt-free survival after ETV [2,4]. Success rates are highest in older children and adults with primary obstructive etiologies such as aqueductal stenosis or tectal gliomas, and lower in infants, post-infectious, and post-hemorrhagic hydrocephalus [1–3,5].

From the standpoint of complications, a low predicted success equates to a higher likelihood of early failure, repeat procedures, and unscheduled rescue interventions. In infants, ETV failure is often driven by impaired CSF absorption and an immature subarachnoid compartment, making these patients more vulnerable to both early deterioration and late, unexpected stoma closure [1–3,5]. When the ETVSS is low and there are additional risk factors such as severe ventriculomegaly, myelomeningocele-associated deformities, or prior infections, the threshold for primary shunt insertion or combined ETV–choroid plexus cauterization should be lower [2,4,5].

The pre-operative consultation must explicitly address the small but non-negligible risk of catastrophic late failure and sudden deterioration, particularly in children who appear well for years after an initially successful procedure [1,4,5]. Families and caregivers should be educated on early warning signs of ETV failure (re-emergence of headache, vomiting, irritability, gait disturbance, or visual decline) and the need for urgent neuroimaging should these occur. This risk counselling is integral to ethical patient selection and shared decision-making [2,4,5].

2.2. Ventricular and Vascular Anatomy

The third ventricle floor (3VF) and interpeduncular cistern contain structures critical to consciousness, memory, endocrine regulation, and brainstem function. Successful and safe ETV requires an accurate understanding of the patient-specific anatomy of the 3VF, basilar artery bifurcation, perforating vessels, mammillary bodies, and infundibular recess

[5–8]. Pre-operative MRI should be carefully reviewed in multiple planes, supplemented by MR angiography or CT angiography in complex cases or when anatomy is distorted by tumors or congenital malformations [5–8].

Recent endoscopic and morphometric studies have shown that 3VF anatomy is highly variable. Microanatomic work has described multiple floor configurations—thinned, thickened, partially erased, globular or herniated, and narrowed—each with different implications for the technical difficulty and safety of the fenestration [7,8]. The thickness of the floor, the relationship to the mammillary bodies and infundibular recess, and the configuration of the Liliequist membrane all influence both the perceived resistance during perforation and the likelihood of a durable stoma [6–8].

Radiomorphometric analysis has refined the concept of a vascular "safe zone" for third ventricle floor perforation. Rincón-Arias and colleagues analyzed the spatial relationships between the 3VF, basilar artery, and its branches, demonstrating how the size and location of a triangular region bounded anteriorly by the infundibular recess and posteriorly by the mammillary bodies varies with patient age and ventricular size [6]. Understanding whether the basilar apex lies high or low relative to the floor and how far posteriorly it projects is crucial for selecting a perforation site that minimizes the risk of vascular injury [6–8].

Where anatomy is significantly distorted—such as in posterior fossa tumors, complex cysts, or severe myelomeningocele-associated deformities—frameless neuronavigation and intra-operative ultrasound can be invaluable. These tools help define the optimal burr-hole position and trajectory through the foramen of Monro, maintain midline orientation, and confirm the expected relationship between the floor and the basilar artery in real time [5–8]. In high-risk anatomy, some authors advocate abandoning ETV in favor of shunt placement rather than accepting a markedly increased risk of vascular or hypothalamic injury [1,2,5].

2.3. Risk Communication and Long-Term Surveillance

Late failure of ETV is uncommon but uniquely hazardous because it may occur after a prolonged period of clinical stability, during which both families and healthcare providers may become less vigilant [1,4,5]. Several series and narrative reviews describe late rapid deterioration or "sudden death" due to acute hydrocephalus from stoma closure years after the initial procedure [1,4,5]. The reported incidence is low, but the consequences are invariably catastrophic.

For this reason, pre-operative counselling should include a clear explanation that ETV is not a once-and-for-all cure and that lifelong risk of failure persists, even if the probability diminishes over time [1,4,5]. Many centers implement structured surveillance protocols that combine periodic clinical review with MRI including phase-contrast CSF flow imaging, particularly in children and in patients with complex etiologies [1–3,5]. Establishing this expectation from the outset supports rapid recognition of failure and may prevent missed opportunities for timely intervention [2–5].

3. INTRA-OPERATIVE COMPLICATIONS

3.1. Hemodynamic Disturbances and Autonomic Events

Transient bradycardia and blood pressure fluctuations are among the most frequent intra-operative events during ETV. Prospective pediatric series and contemporary reviews document significant bradycardia during perforation and balloon dilation of the third ventricle floor in a substantial proportion of procedures [1,5]. These changes are thought to reflect a Cushing-like response mediated by transient brainstem compression or traction on hypothalamic and diencephalic structures [1,5].

In most cases, the bradycardia is self-limited and resolves promptly when endoscope manipulation is paused and irrigation pressure reduced. However, severe bradycardia, asystole, or hypertensive surges can occur and may be exacerbated by excessive irrigation, rapid increases in intracranial pressure, or inadvertent mechanical distortion of midbrain structures [1,5]. Continuous anesthetic–surgical communication, early recognition of adverse trends on the monitor, and a shared plan for temporarily aborting the maneuver are essential components of safe ETV practice [1,5].

Prevention strategies include maintaining normocapnia, avoiding unnecessary head rotation or jugular compression, and using the lowest irrigation pressure that provides adequate visualization. Some teams prefer volume-controlled rather than pressure-driven irrigation systems to minimize sudden changes in intracranial pressure. In high-risk patients (e.g., infants, severe cardiopulmonary comorbidity), the threshold for abandoning the procedure in the face of recurrent hemodynamic instability should be low [1,2,5].

3.2. Hemorrhagic and Vascular Complications

Hemorrhagic complications range from minor ependymal oozing to catastrophic rupture of the basilar artery. Large clinical series and systematic

reviews report intra-operative hemorrhage in a single-digit percentage of ETV procedures, with severe hemorrhage and basilar artery rupture remaining rare but life-threatening events [1,2,5]. Minor venous or ependymal bleeding is usually manageable with irrigation and brief tamponade, but arterial hemorrhage can rapidly obscure the field, cause abrupt increases in intracranial pressure, and lead to irreversible brain injury or death [1,5].

Basilar artery perforation remains the most feared vascular complication. Precipitating factors include perforation too posterior or too close to the mammillary bodies, loss of midline orientation, or an unrecognized high-riding basilar apex [5–8]. Deep advancement of instruments in a narrow interpeduncular cistern and uncontrolled balloon dilation in a rigid, thickened floor also increase risk [5–8]. Once major arterial bleeding occurs, options are limited: rapid irrigation, controlled hypotension, tamponade with the endoscope sheath, and, in some cases, emergent external ventricular drainage and decompressive craniectomy [5]. Survival with good outcome after frank basilar rupture is exceptional [1,5].

Preventive measures are therefore paramount. These include detailed pre-operative evaluation of basilar artery position and third ventricle floor thickness, neuronavigation-guided planning of the entry point and trajectory, conservative use of the balloon (small initial dilation, gradual expansion), and strict adherence to the concept of a vascular safe zone as defined by recent morphometric work [5–8]. Doppler microprobes have been proposed in select cases to verify the position of the basilar artery beneath the floor before perforation, although their routine use is not yet established [5,6].

3.3. Injury to Neural and Hypothalamic Structures

Neural injury during ETV primarily involves the fornix, hypothalamus, thalamus, and midbrain. Contemporary series estimate permanent neurological morbidity (hemiparesis, gaze palsy, memory impairment, altered consciousness) at around 1–2%, and permanent hormonal morbidity (diabetes insipidus, weight gain, precocious puberty) at just under 1% [1,2,5].

The fornix is particularly vulnerable during endoscope advancement through the foramen of Monro. Even subtle contusion or stretching of the fornical columns can result in anterograde amnesia and executive dysfunction. A systematic review of ventricular neuroendoscopy procedures identified neurocognitive complications in approximately 3% of cases, with persistent memory impairment in about 1% [9]. These deficits are

often under-recognized unless formal neuropsychological assessment is performed [9].

Hypothalamic and pituitary stalk injury can produce a spectrum of endocrine disturbances, including diabetes insipidus, syndrome of inappropriate antidiuretic hormone secretion (SIADH), hyperphagia, weight gain, and pubertal disturbances [1,5,12]. Although permanent hypothalamic obesity after ETV is rare, the hypothalamus is a known driver of energy balance and puberty, and experimental and clinical data link hypothalamic inflammation with obesity and precocious puberty [12]. Endocrine complications may be immediate or delayed, underscoring the need for post-operative surveillance of fluid balance, electrolytes, and growth trajectories, particularly in children [1,5,12].

Avoidance of neural injury depends on careful trajectory planning, strict midline orientation, gentle endoscope manipulation, and respect for visual cues such as the choroid plexus and thalamostriate vein within the lateral ventricle. Limiting the number of instrument passes, avoiding excessive lateral sweeping of the endoscope tip, and maintaining clear visualization at all times reduce the likelihood of inadvertent damage to critical structures [1,2,5,7].

3.4. Technical Failures and Aborted Procedures

Technical failures—such as inability to perforate a thickened third ventricle floor, inadequate fenestration size, poor visualization due to cloudy CSF, or loss of anatomical orientation—account for a significant proportion of intra-operative complications and aborted procedures [1,2,4,5]. In contemporary series, a small but important fraction of ETVs are abandoned because of hemorrhage, distorted anatomy, or anesthesia-related events [1,2].

When visualization is inadequate or anatomy appears substantially different from pre-operative imaging, it is safer to abort the ETV and convert to shunt placement rather than to persist with repeated attempts at perforation [4,5]. Similarly, if the floor cannot be safely dilated to a sufficient diameter to ensure durable CSF flow, some authors recommend placing an external ventricular drain or shunt rather than accepting a high risk of early closure and failure [2,4,5].

Structured training, simulation-based practice, and gradual progression of case complexity for trainees are critical to reducing technical errors. Many centers reserve complex anatomy and high-risk pediatric cases for surgeons with substantial ETV experience, while ensuring that less-experienced

surgeons perform ETV under direct supervision until they have mastered the basic steps [1,2,5].

4. EARLY POST-OPERATIVE AND PHYSIOLOGICAL COMPLICATIONS

4.1. CSF Leak, Pseudomeningocele and Subdural Collections

CSF leak from the burr-hole wound or along the tract can present as scalp swelling, persistent clear drainage, or pseudomeningocele. Large series report CSF leak in a small proportion of cases, while subdural hygromas are less frequent but clinically relevant when symptomatic [1,2,5]. These complications are usually related to inadequate dural closure, poor wound sealing, or rapid reduction of intracranial pressure in patients with chronically enlarged ventricles [1,2].

Most CSF leaks can be managed conservatively with head elevation, compressive dressings, and short-term lumbar drainage. Persistent leak or large pseudomeningoceles may require surgical revision of the wound and dural closure. Subdural hygromas are often asymptomatic and resolve spontaneously, but symptomatic collections or those associated with mass effect may necessitate burr-hole drainage or, rarely, subduroperitoneal shunting [1,2,5].

Preventive measures include meticulous multilayer wound closure, avoidance of oversized cortical openings, and post-operative positioning that minimizes hydrostatic pressure at the burr hole. In patients with marked macrocephaly and thin cortices, some surgeons reduce the amount of CSF removed intra-operatively and avoid aggressive over-drainage immediately after the procedure [1,2,5].

4.2. Electrolyte and Endocrine Disturbances

Sodium and water balance disturbances are important but often underappreciated early complications of ETV. While diabetes insipidus and hypernatremia are more widely recognized, hyponatremia and SIADH-related disturbances have also been reported in early post-operative series and reviews [5]. Symptoms such as headache, vomiting, and lethargy may mimic recurrent hydrocephalus and risk misinterpretation as ETV failure if serum sodium is not closely monitored [5].

Endocrine complications may result from transient or permanent dysfunction of the hypothalamus, pituitary stalk, or posterior pituitary [1,5]. Clinical manifestations include polyuria/polydipsia, hypernatremia,

hyponatremia, inappropriate weight gain, and pubertal disturbances [1,5,12]. While overt, permanent hormonal morbidity appears to be relatively uncommon, subtle hypothalamic dysfunction and long-term weight changes may be underreported, particularly in children [1,5,12].

Perioperative management should therefore include routine monitoring of fluid balance, daily serum sodium in the first postoperative days, and a low threshold for endocrinology consultation when abnormalities are detected. In pediatric patients, ongoing surveillance of growth, weight trajectory, and pubertal development is advisable, particularly after operations that involve extensive manipulation of the third ventricle floor or hypothalamic region [1,5,12]

4.3. Infection

Infectious complications of ETV include meningitis and ventriculitis. Pooled data and recent reviews suggest central nervous system infection in a small minority of cases, generally below the rates associated with shunt surgery [1,2,5]. Nevertheless, these infections remain clinically significant and can contribute to prolonged hospitalization, neurological sequelae, and procedure failure [1,2,5].

Risk factors include prolonged operative time, CSF leak, repeated instrumentation, and pre-existing systemic or cranial infections [1,2,5]. Standard prophylactic measures consist of a single pre-operative dose of a cephalosporin (or an alternative agent in the case of allergy or resistant colonization), strict adherence to sterile technique, and minimization of operative time and device exchanges [1,2,5].

Post-operative fever, meningismus, wound breakdown, or neurological deterioration should prompt evaluation with laboratory tests and neuroimaging, followed by lumbar puncture or ventricular CSF sampling where appropriate. Treatment follows standard meningitis and ventriculitis protocols, typically combining intravenous broad-spectrum antibiotics with targeted therapy once organism susceptibilities are known. In some cases, temporary CSF diversion with an external ventricular drain may be required [1,2,5].

4.4. Neurological Worsening and Intracranial Hematomas

Early postoperative neurological deterioration after ETV may result from intracranial hematomas (subdural, intraventricular, intracerebral, or epidural), worsening hydrocephalus due to ineffective fenestration, metabolic disturbances, or seizures [1,2,5,10,11]. The overall incidence

of clinically significant intracranial hematomas after ETV is low but non-negligible, and small asymptomatic hemorrhages may be more frequent on routine postoperative imaging [1,2].

Any unexpected worsening in level of consciousness, focal neurological deficit, or seizure activity after ETV warrants urgent imaging—ideally CT for rapid assessment, followed by MRI where indicated—to differentiate between hemorrhage, hydrocephalus progression, and other causes. Management ranges from observation with serial imaging for small, asymptomatic hematomas to surgical evacuation or decompression for lesions causing mass effect or elevated intracranial pressure [1,2,5].

Post-operative seizures are an additional concern, particularly in pediatric patients. Recent pediatric series report early seizure rates of several percent after first-time ETV, with higher risk among children with pre-existing epilepsy, cortical injury, or intraventricular hemorrhage [10,11]. Optimization of antiepileptic therapy perioperatively and careful monitoring in the early post-operative period are important components of comprehensive care [10,11].

5. LATE COMPLICATIONS AND ETV FAILURE

5.1. Definitions and Timing of Failure

ETV failure can be categorized as early (within 30 days), delayed (1–24 months), or late (>2 years) based on the interval from surgery to clinical or radiological recurrence of hydrocephalus [1–4]. Most failures occur within the first 6–12 months, particularly in infants and complex etiologies, and are often related to inadequate initial fenestration, insufficient CSF absorption, or subarachnoid scarring [1–4].

Late failures, although less frequent, are of special concern because they may present abruptly after years of clinical stability. Mechanisms include progressive gliosis and scarring of the stoma, formation of arachnoid membranes in the interpeduncular cistern, or new obstruction related to tumor growth or vascular changes [1,4,5]. The risk of late failure appears higher in patients operated at a very young age, in post-infectious hydrocephalus, and in those with significant cisternal scarring [1–3,5].

5.2. Mechanisms of Stoma Closure and CSF Pathway Obstruction

Classical series analyzing mechanisms of ETV failure in infants and children highlight several patterns: inadequate primary fenestration, early closure from gliosis or arachnoid adhesions, and persistent impairment

of CSF absorption despite a patent stoma [2,4,5]. In some cases, the initial stoma is technically successful but cannot overcome an underlying communicating hydrocephalus component, particularly after hemorrhage or infection [1–3,5].

High-resolution MRI with heavily T2-weighted cisternographic sequences and phase-contrast CSF flow imaging has improved recognition of these mechanisms. Lack of flow signal across the stoma, thickening of the third ventricle floor, or the presence of arachnoid membranes in the prepontine cistern support the diagnosis of functional or anatomical failure [1–3,5]. In selected cases, repeat ETV can restore patency and long-term shunt independence, particularly when failure is due to focal stoma closure rather than diffuse cisternal scarring [2–4].

5.3. Late Rapid Deterioration and Sudden Death

One of the most feared late complications is abrupt neurological deterioration or sudden death due to rapid reaccumulation of CSF after stoma closure. Classical pediatric reports describe children who deteriorated rapidly years after apparently successful ETV, with imaging or autopsy demonstrating occluded stomas and acute obstructive hydrocephalus [4,5].

The pathophysiology of these events is incompletely understood but likely reflects the loss of compensatory mechanisms in patients who have adapted to a chronic steady-state CSF circulation after ETV [4,5]. Once the stoma closes, CSF accumulates rapidly, often faster than in de novo hydrocephalus, and clinical deterioration can evolve over hours. Because symptoms may initially be nonspecific (headache, vomiting, behavior change), early recognition depends on a high index of suspicion by families and frontline clinicians [4,5].

Strategies proposed to mitigate this risk include: rigorous long-term follow-up with periodic imaging, explicit education of families about the possibility of late failure and the need for emergent assessment of recurrent symptoms, and, in select cases, adjunctive measures such as placement of a ventricular access device to facilitate rapid CSF drainage and diagnosis [1–5]. However, no strategy completely eliminates the possibility of late sudden deterioration; thus, risk communication remains central [4,5].

5.4. Management of ETV Failure

Management options for proven or suspected ETV failure include repeat ETV, shunt insertion, or, in selected cases, combined approaches [2,4,5]. Repeat ETV is most successful when the original failure mechanism is focal

stoma closure with otherwise favorable anatomy, and when the interval from the first procedure is relatively long [2–4]. In contrast, diffuse cisternal scarring, post-infectious hydrocephalus, and very young age are associated with lower success rates for repeat ETV and often favor shunt placement [1–4].

In acute deterioration, initial management focuses on rapid stabilization, emergent imaging, and CSF diversion—typically with an external ventricular drain—followed by definitive treatment once the mechanism is clarified [4,5]. Given the potential for rapid decompensation, centers that perform ETV regularly should have clear protocols for emergency access, imaging, and neurosurgical intervention at all times [1–5].

6. PREVENTION AND RISK MITIGATION STRATEGIES

The overarching theme from contemporary literature is that most serious ETV complications are not random events but predictable outcomes of specific anatomical, technical, and organizational factors [1–5,7,8]. Effective prevention therefore rests on three pillars: rigorous patient selection, meticulous anatomical planning and technique, and structured peri-operative systems of care [1–5].

First, patient selection should be guided by ETV success scores, etiology-specific data, and a frank appraisal of the center's experience with high-risk subgroups such as infants and post-infectious hydrocephalus [1–4]. When the predicted success is low and the anatomy is unfavorable, shunt placement may offer a safer and more reliable initial strategy [2,4,5].

Second, meticulous anatomical planning is essential. This includes high-quality pre-operative MRI (and angiography in selected cases), systematic assessment of third ventricle floor morphology and basilar artery position, and, where appropriate, neuronavigation to define a safe burr-hole and trajectory [5–8]. Surgeons should be familiar with endoscopic and morphometric classifications of floor variants, the radiomorphometric vascular safe zone, and the characteristic appearance of the interpeduncular cistern [6–8].

Third, peri-operative systems of care should incorporate standardized checklists, clear intra-operative communication protocols between surgeon and anesthesiologist, explicit criteria for aborting the procedure in the face of poor visualization or hemodynamic instability, and structured postoperative monitoring of neurological status, wound integrity, and electrolytes [1,2,5,9–11]. Longitudinal follow-up pathways are essential to detect late failure and to address subtle neurocognitive or endocrine sequelae [1–5,9,12].

7. CONCLUSION

ETV has transformed the management of obstructive hydrocephalus and offers durable shunt-free outcomes for many patients [1–3]. At the same time, the procedure carries a distinct spectrum of complications that range from transient bradycardia and minor CSF leaks to devastating basilar artery rupture and late sudden deterioration from stoma closure [1,4,5]. Modern practice demands that neurosurgeons move beyond anecdotal experience and incorporate robust evidence on the incidence, mechanisms, and prevention of these events [1–5].

When patient selection is judicious, anatomical planning is meticulous, and perioperative care is protocolized, ETV can be performed with a high degree of safety and efficacy [1–5,7,8]. Ongoing research into third ventricle and vascular morphometry, neurocognitive and endocrine outcomes, and long-term failure mechanisms will further refine risk stratification and guide individualized management strategies for patients undergoing this powerful but unforgiving procedure [1–5,7–12].

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