Chapter

Benefits of Early Tracheostomy in TBI Patients

Sabrina Araujo de França, Wagner M. Tavares, Wellingson S. Paiva and Manoel J. Teixeira

Abstract

Severe traumatic brain injury (TBI) patients are constantly submitted to interventions to cope secondary injury and insults. Oxygen therapy is mostly initiated by endotracheal intubation at the scene of the accident. Due to the severity of the trauma, prolonged mechanical ventilation is expected and tracheostomy (TQT) is often indicated. TQT became one of the most common bedside surgical procedure performed in an Intensive Care Unit (ICU). However, discussion regarding the optimal time for TQT placement to improve outcomes of severe TBI patients remains under discussion. This chapter aims to review TBI's physiopathology and enlighten early tracheostomy's role in severe TBI management.

Keywords: benefits, early medical intervention, outcome, severe brain injury, tracheostomy, traumatic brain injury

1. Introduction

The major focus on traumatic brain injury (TBI) management is to avoid and restrain ongoing brain damage and to increase brain recovery chances by reducing brain edema and intracranial pressure (ICP). Optimizing oxygenation, perfusion, nutrition, glycaemia and temperature homeostasis are paramount [1]. In this chapter, we will discuss the role of oxygenation in TBI management with a special focus on early indication of tracheostomy (TQT) as a support to oxygen therapy.

2. Incidence and prevalence of TBI

TBI is a critical public health concern with large socioeconomic repercussions. The main causes of TBI include violence, falls and road traffic accidents [2]. In 2010, the global burden of disease (GBD) reported 89% of trauma-related deaths occurring in low- and middle-income countries (LMICs) [3]. In 2030, the worldwide estimated incidence of TBI places this type of trauma as a 4th leading cause of lost disability adjusted life years (DAYLS) and 7th cause of death [4].

The Centers for Disease Control and Prevention (CDC) estimated 2.53 million emergency department (ED) visits, 288.000 hospitalizations and 56.800 deaths related to TBI, in 2014 [5]. The TBI's lifetime economic costs (direct and indirect medical costs) was estimated at $76.5 billion (2010) and fatal TBIs can account for up to 90% of total medical costs. Since TBI is a growing health burden, it is an utmost importance the optimization of hospital resources and staff [2, 6].

TBI can be classified following its severity: Mild, Moderate and Severe [7]. This classification is based on the Glasgow Coma Scale (GCS), with Mild - GCS Score 13–15; Moderate - GCS Score 9–12; and Severe - GCS Score 8–3. Subsequent TBI management will rely on the first evaluation and the prevention of secondary injuries.

3. TBI physiopathology and oxygen importance

TBI presents two main classifications for intracranial lesions: focal or diffused [7–9]. Focal brain damage is the consequence of cortical lacerations, compression, or concussion forces, compromising blood supply and culminating in neuronal and glial necrosis. The structural injury is resulted from the brain collision to rigid structures, depressed skull fractures, vascular injuries or penetrating trauma [8–11].

Diffuse brain damage is caused by acceleration/deceleration forces, that shears and stretches brain tissue, causing functional disturbance, culminating in brain swelling or diffuse axonal injury [8–11]. The co-existence of both types of injuries are frequently present, as a result from the mechanical distortion of the head that leads to a combination of neural and vascular events [10–13].

Additionally, the TBI process can be breakdown in two successive, intertwined, pathophysiological moments, labeled as primary and secondary injury.

3.1 Primary injury

The primary injury arises from the mechanical damage occurring at the time of the impact, being exclusively responsive to preventive measures [8, 14]. On the macroscopic level, damage can be recognized by shearing of white-matter tracts, diffuse swelling, focal contusions, and intracerebral and extracerebral hematomas [15–17].

On the cellular level, mechanoporation of axolemma (caused by the traumatic axonal injury) results in sodium channelopaty [18] and unregulated influx of Ca^{2+}, which initiates calpain activation and mitochondrial swelling [19–22]. Calpain activation and cytochrome *c* accumulation increases axonal injury, detachment and apoptosis [23]. This cascade of events occurs 24 to 72 hours after the trauma and is denominated as secondary axotomy [11]. Injured axons are also susceptible to demyelination [17].

The microvasculature suffers from injury changes, such as swelling of perivascular astrocytic end-feet, increased adherence of intravascular leukocyte, perivascular hemorrhage, transvascular erythrocytes diapedesis, and increased activity of endothelial microvacuolation and micropseudopodia [24, 25].

3.2 Secondary injury

The second injury emerges from a complex series of molecular and cellular interrelated events, resulted from the biochemical cascades triggered by the trauma [9, 11]. An essential goal in the critical care is to establish recognition and treatment for secondary injury, and prevent secondary insults, which worsen patient's outcomes [26, 27].

Post-traumatic edema likely occurs by the dysfunction of sodium-potassium pump, due to pH-induced conformational change or cellular energy failure, resulting in water and sodium accumulation within the cell [11, 25]. Other factors that contributes to intracranial edema are excitotoxicity (induces intracellular sodium accumulation) [28], and membrane disruption [29] and depolarization (induced by influx of chloride, due to sodium influx) [30].

Excitotoxicity is the result of the excess of excitatory amino acids (EAA) that are released in the extracellular space, such as glutamate and aspartate, which raises intracellular sodium, calcium, chloride and water [10, 15]. This accumulation results in organelle and plasma membrane swelling [31], apoptosis, activation of destructive enzymes (such as calpain, nitric oxide synthase) [32], positive feedback loop by voltage-gated calcium channels [33], and necrosis [34].

Besides other secondary brain injuries, such as calcium dysregulation (which leads to cytoskeletal degradation), patients experience superimposed secondary insults (with intracranial or systemic repercussions) [10, 14, 15, 27]. Systemic repercussions are hypotension, hypoxia, hyperthermia, and hypoxemia. **Figure 1** recapitulate TBI's sequence of events. **Figure 2** recapitulate TBI's neurometabolic cascade.

Intracranial insults include cerebral ischemia, elevated ICP (or intracranial hypertension), and cerebral fluid-mediated swelling. The Monro Kellie doctrine (**Figure 3**) demonstrates the constancy relationship in the sum of volumes of brain, intracranial blood and cerebrospinal fluid (CSF) [7, 36, 37]. Once the brain suffers from the intracranial insults and equal volumes of CSF and intracranial blood are compressed, ICP remains normal (compensated state). When the brain enters in a decompensate state (after exhaustion of compensate state), the balance is interrupted and the ICP raises exponentially [7, 16].

It is important to mention that secondary injury does not have the same meaning as a secondary insult [11, 14]. Secondary insult occurs at the organ system level, being considered as a second hit event, exacerbating the damage from the primary

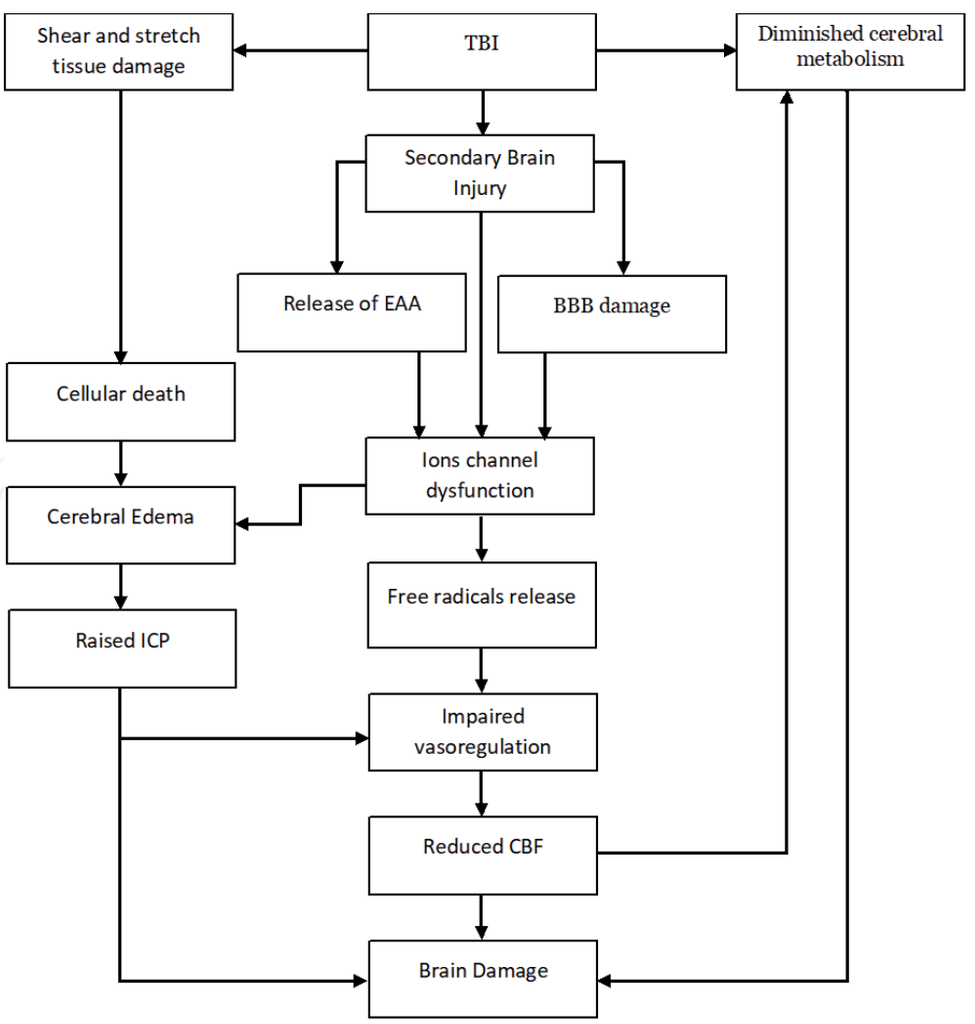

Figure 1.
TBI's sequence of events.

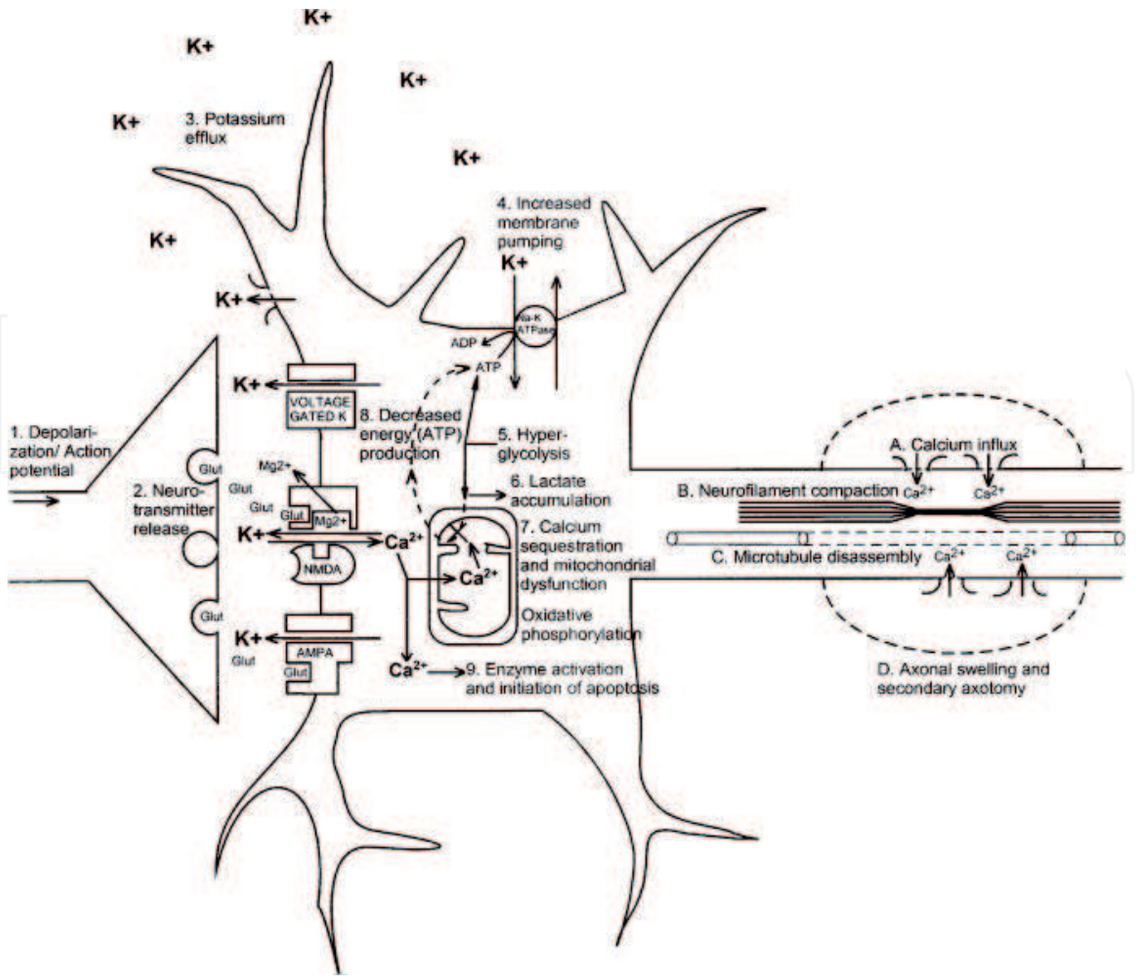

Figure 2.
[35] TBI's neurometabolic cascade. (1) nonspecific depolarization; (2) neurotransmitter release - excitatory neurotransmitters (EAAs); (3) increase potassium efflux; (4) increased membrane pumping to restore homeostasis; (5) Hyperglycolysis to increase adenosine triphosphate (ATP) availability; (6) lactate accumulation; (7) calcium sequestration and mitochondria dysfunction resulting in oxidative metabolism; (8) decreased ATP production; (9) Calpain activation and apoptosis initiation. A - Axolemma and calcium influx. B - Neurofilament compaction. C - microtubule disassembly. D - axonal swelling and secondary axotomy. K^+: potassium; Na^+: sodium; Glut: glutamate; Mg^{2+}: magnesium; Ca^{2+}: calcium; NMDA: N-methyl-D-aspartate; AMPA: d-amino-3-hydroxy-5-methyl-4-isoxazole-propionic acid.

TBI. The same reasoning is applied to the primary insult, which alters the cerebral metabolism and blood flow, resulting in cellular dysfunction and predisposition to cognitive impairment, seizures, hypotension and hypoxia [8].

3.3 Oxygen and TBI

The brain requires an uninterrupted supply of glucose and oxygen to maintain cellular viability and metabolism, consuming up to 20% of individual's total oxygen, with an average of cerebral metabolic rate of oxygen ($CMRO_2$) between 3 and 3.8 ml/100 g/min [38–41]. Brain metabolism represents the largest source of energy consumption in the human body, since neuronal activity is supported through the production of adenosine triphosphate (ATP), which consumes nearly 60% of oxygen [42, 43]. When cerebral oxygenation is maintained, minimization of secondary insult can be achieved [44].

Brain's energy consumption fluctuates following neuronal activity on localized regions, and in order to provide adequate energy supply, neurovascular and neurometabolic coupling mechanisms are involved [42]. However, within hypoxia or low oxygen conditions, prolyl hydroxylase domain-containing enzymes (PHDs) are

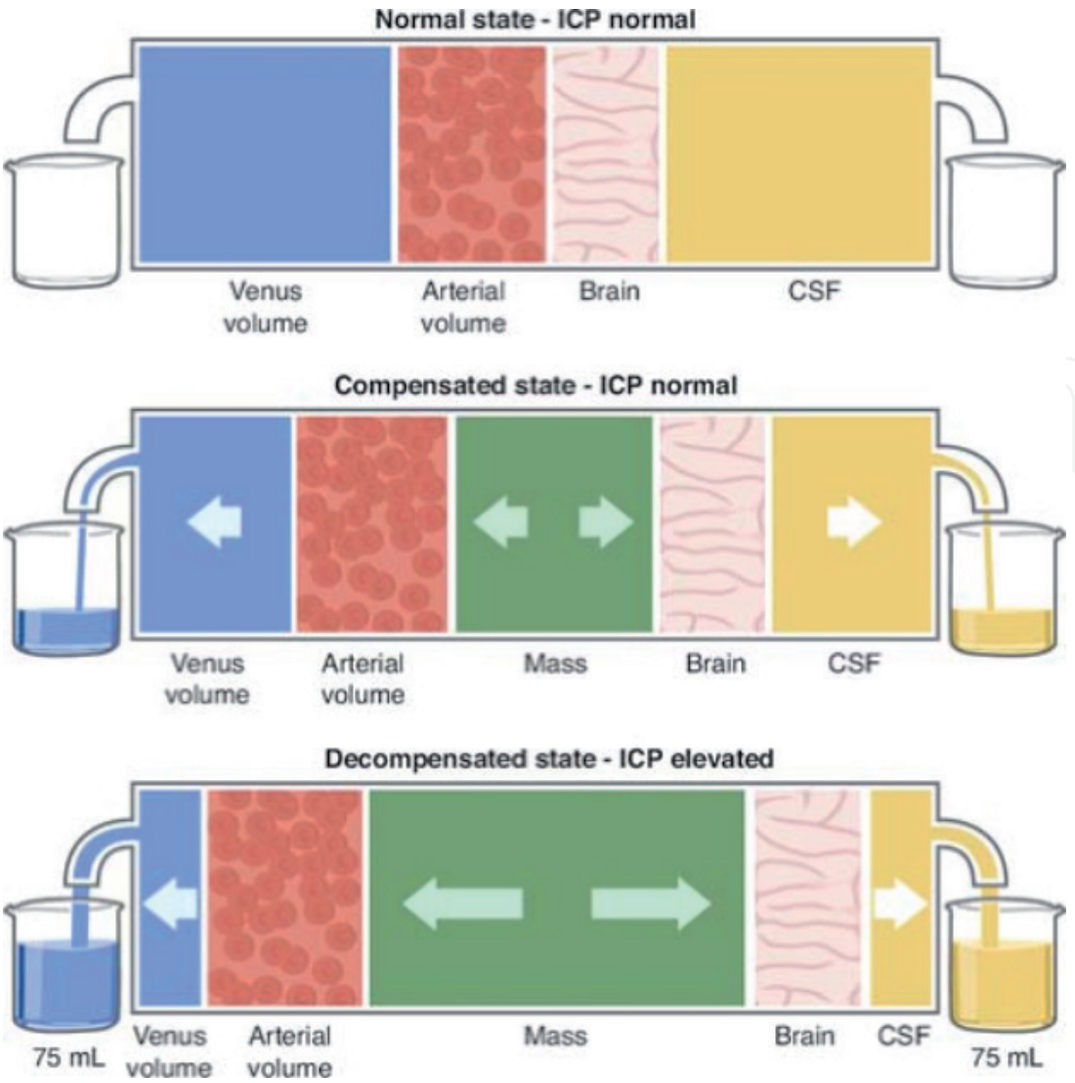

Figure 3.
The Monro Kellie doctrine.

inhibited, reducing inproline hydroxylation, and altering availability of hypoxia-inducible factor-1α (HIF-1α), which assist the metabolism adaptation and function during hypoxic conditions [42, 45–47]. Then, nuclear accumulation of HIF-1α enhance transcriptional activity within HIF-β, promoting gene expression that contains a hypoxia response element (HRE) [42, 46, 48]. Remarkably, HIF-2α is also induced in hypoxic brain, being expressed in astrocytes and endothelial cells [49]. This dysfunction is associated with poor neurological outcomes [50].

In order to cope hypoxia stress, this adaptive response converts cellular metabolism to anaerobic metabolism and inducts erythropoiesis, glycolysis, angiogenesis (by vascular endothelial growth factor), among other events [46, 48, 51]. Nevertheless, anaerobic glycolysis is unable to apport sufficient energy to sustain brain demands, depleting ATP stores, which results in failure of ATP dependent membrane ionic pumps [52]. Likewise, under chronic hypoxic conditions, there is an increase in oxidative stress, cell death, inflammation and the interruption of cerebral blood flow (CBF), directly affecting brain structure and function, leading to neuronal damage and death [51, 53, 54]. A normal average of CBF in adults is 44–45 ml/100 g/min. However, the CBF threshold for irreversible tissue damage (in TBI) occurs with the decrease to 15 ml/100 g/min [27, 41, 55] and cellular function is disrupted under 10 ml/100 g/min [56]. Neurons in the hippocampus, striatum and cortical regions die after 5, 10, and 15–20 min of ischemia [57, 58], respectively.

Considering that the brain is susceptible to ischemic injury, cerebral perfusion and oxygenation are vital to be maintained. In TBI setting, cerebral ischemia occurs due to different mechanisms: damage to blood vessels, hypotension, mechanical compression, and reduced perfusion (impaired autoregulation, which leads to greater propensity to hypoperfusion) [16, 59]. Hypoxemia can represent a relative risk of mortality of 75%, when associated with hypotension [7].

According to the Brain Trauma Foundation [60], patients with severe TBI present pulmonary aspiration risks or compromised airway function, and initial treatment goals include early airway protection, adequate supplemental oxygen, and circulation support, ensuring that adequate oxygen and blood flow are delivered to the brain [61].

4. Prehospital care and oxygenation

TBI management begins at the prehospital care, assuring that the patient has no signs of upper airway obstruction, maintaining Oxygen saturation (spO_2) > 90% and considering intubation in patients that presents Glasgow Coma Scale (GCS) < 9, altered swallowing reflex or contributing to hypoventilation [60, 62–64]. However, it is of substantial value that the prehospital team is technically qualified (within technical skills, medical devices/medication, and protocols) to perform airway management and control possible detrimental effects of therapeutic interventions (such as worsening of cervical spine injury during endotracheal intubation [65]).

Studies examining the impact of prehospital intubation have conspicuously conflicting results. A Finnish [66] comparison between physician-staffed prehospital team and paramedics team (PM) demonstrated that the physician team performed 98% of advanced airway management against 16% in paramedics patient's group. Hypoxia was higher in the PM group at the emergency department arrival. Furthermore, one-year mortality rate was higher in the PM group. Singularly, anesthetics were available for physician teams only, while PM were limited to the sedatives. Patients that were submitted to emergence intubation during prehospital care presented an increased risk of morbidity and mortality (poor neurologic outcome [67, 68] and decreased survival rate [68–71]).

The French Society of Anesthesia and Intensive Care Medicine strongly recommends a prehospital medicalized team to assess patients, claiming higher survival rates [72]. Divergently, an Australian study [73] indicated that rapid sequence intubation performed by paramedics increased the 6 months rate of favorable neurologic outcome. The Trauma Research and Education Foundation of San Diego [74] also demonstrated improved survival in patients intubated in the field. Meanwhile, any benefit or harm of pre-hospital intubation could be stated [63].

Marehbian et al. [75] inferred that these inconsistencies may be attributed to multiple factors: GCS applied as a single scale to identify intubation candidates (can be misinterpreted by illegal substances or sedative effects), variability of protocols, and inadequate intubation or ventilation approaches, that can lead to hypo or hyperventilation.

Hyperventilation is commonly revealed as a higher incidence among prehospital intubated patients. Hemodynamically, hyperventilation rises the intrathoracic pressure, leading to a decrease in cardiac output [76–78]. Regarding the cerebral perfusion, hypocapnia decrease the cerebral blood volume (CBV), which directly decrease the cerebral blood flow (CBF), and lower ICP [79–82]. Studies had shown regional and local ischemia with tissue lactic acidosis immediately after hyperventilation, suggesting harmful effects in cerebral tissue due to cerebral vasoconstriction [7, 81, 82]. Currently, hyperventilation should be reserved to

refractory cases immediately before surgical intervention. It should not be used in ICU management of refractory ICP because of its detrimental hypoperfusion properties [7, 83–85].

The use of supplemental oxygen is required to correct hypoxemia and attempt to avoid secondary injury. Therefore, initial management for severe traumatic brain injury involves intubation and ventilation for airway management [7, 72]. However, the decision to perform invasive procedures on the trauma stage should be evaluated in a case to case basis since the experience of the team involved together with the conditions of the patient/surroundings (patient inside a car wreckage or in a war site) of the trauma can have a great influence in the decision making.

5. Tracheostomy and oxygenation

To prevent or reverse hypoxemia and provide oxygen to the tissues during acute respiratory failure, airway access is often provided by translaryngeal endotracheal tube. When mechanical ventilation is expected to be prolonged, TQT tube is frequently chosen as part of the airway management care plan [86–88].

Earlier TQT records were found in the Edwin Smith Papyrus (1600 BC), whereas an emergency airway was performed after a trauma [89]. The first surgical description of successful case of TQT was performed by Antonio Musa Brasavola (1546) [90] and a full book dedicated to this procedure, previously known as bronchotomy, was published in 1620 [91] by Nicolas Habicot, who pictured it as demonstrated in **Figure 4**.

Figure 4.
[92] Patient's tracheostomy by Nicolas Habitot. A: the patient; B: the larynx; C: bronchotomy insertion; D: bronchotomy's instrument; E: the cannula; F: cannula's strap; G: a band to apply over the cannula to control the air leakage; H: the needle to suture the wound when needed.

During the Second World War, TQT grown relevance in chest trauma patients [93] and since then, it is expanding its role in airway management, as well improvement of the surgical techniques, instruments and cannulas.

Prolonged/impractical intubation, ventilation support for weaning, pulmonary hygiene management and airway protection are main indications for TQT placement [86–88, 94]. Patients can benefit from tracheostomy that is performed by open surgical (OST) or percutaneous dilatory (PDT) techniques.

Patient's individual aspects assist the medical team to decide whether to use PDT or OST. PDT is recommended for patients who can hyperextend the neck, tolerate hypercarbia and hypoxemia, and present at least 1-cm distance between the inferior cricoid cartilage and the suprasternal notch (in case of needed re-intubated after accidental extubation) [86–88]. PDT relative contraindications are emergency airway access, anatomical incompatibility, coagulopathies, higher levels for support oxygenation (e.g. positive end-expiratory pressure ≥ 10 mm Hg or fraction of inspired oxygen ≥0.7), and infection at insertion site surroundings [86–88].

Studies were carried out to establish advantages and preferences between techniques. A Cochrane review did not find statistical difference for mortality and serious life-threatening adverse events between techniques [95]. However, PDT presented significantly reduced rate for wound infections/stomatitis and unfavorable scaring. Other systematic reviews and meta-analysis confirmed the same result trend: no difference in mortality and life-threatening complications [96–100]. Significant positive outcomes for PDT was cited as less infection rate [97–100] and less procedure time [96, 100–102]. Besides these results, OST could also impact hospital expenditures, since the procedure can require an operatory room and staff [88, 103, 104].

6. The benefits of tracheostomy on TBI

A multidisciplinary team collaborates in patient's care for adequate communication, ventilation and oxygenation [104]. The presence of a TQT may promote greater airway security, assisting in patient's mobilization and engagement to physical therapies [88]. Likewise, TQT allows sedation reduction or cessation, reduction of laryngeal lesions, assist in weaning protocol and improve oral nutrition and communication [105–107]. Mentioned risks are tracheal stenosis, tracheomalacia and hemorrhage [108]. However, TQT benefits overcome procedures risks [94, 109, 110].

Over the past decade, extensive research has been done concerning TQT timing for optimal results in patient's care, and an oscillation of a cut out day to consider TQT as an early procedure (ET) is perceived. Literature reveals authors acceptation of TQT as an early procedure, as those ones performed between 2 and 12 days after admission [111–116].

A systematic review and meta-analysis [115] revealed that ET, in severe TBI patients, is associated with shorter length of mechanical ventilation and intensive care unit (ICU) and hospital stay. Likewise, decreased risk of ventilator associated pneumonia was found. Complementary literature comparing early and late tracheostomy (LT) populations demonstrated lower ICU stay [113, 117–120], lower hospital stay [117, 120], lower rates for pneumonia [113, 117, 119, 120] and lower costs [113, 117].

Healthcare cost management has increasing its role as part of patient's care plan. Given an aging population and rising medical comorbidities, expertise in resource allocation is crucial. Herrit and colleagues [121] demonstrated the average weighted cost of ET (≤4 days) patients in ICU is $4316 less when compared with

LT (≥11 days). A continous demand/imporance of resources was produced and exposed by the latest worldwide heath care crisis caused by Corona Virus 19 (Covid-19). Mattioli et al. [122] briefly exposed that ET (≥7 days <14 days) could promote expedited ICU beds availability. Nonetheless, studies are needed to assure TQT role for COVID-19 management [123].

Mostly of the presented mortality rates between LT and ET analysis do not demonstrate statistically significance [113, 114, 117, 119, 120, 124–128], which could be a response of ET placement in critical state patients [86]. Hence, no definitive conclusion could be drawn by the absence of mortality significance, as well, patients functional state at discharge could not be assured.

The variation of tracheostomy protocols can contribute to misleading results. A retrospective study [129] across 19 countries and 54 TBI centers in Europe demonstrated that the incidence of ET (≤7 days after admission) ranged from 0 to 17.6% and LT from 7.9 to 32%. A delayed procedure was more likely to happen than an earlier one. LT patients presented higher reintubation, VAP and respiratory failure rates than ET.

7. Conclusion

Overall, ET could contribute to lower exposure to secondary insults and nosocomial adverse events, rising patient's early rehabilitation and discharge rates, and improve hospital/staff resources management. Establishment of guidelines for further homogenous approaches to better assist severe TBI patients and improve second injury control is concerned.

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Conflict of interest

The authors declare no conflict of interest.

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