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Association between Paroxysmal Sympathetic Hyperactivity and Tracheostomy Weaning in Traumatic Brain Injury

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Abstract

Background

Rehabilitation following severe Traumatic Brain Injury (TBI) often involves the use of temporary tracheostomies. Tracheostomy weaning is influenced by physiological parameters, which are abnormal in the concomitant complication of Paroxysmal Sympathetic Hyperactivity (PSH).

Objective

To investigate the association between PSH and tracheostomy weaning in severe TBI.

Methods

This was a retrospective cohort study of consecutive patients with TBI and tracheostomy admitted to a Hyper-Acute Neurorehabilitation Unit over a 34-month period. Duration of tracheostomy wean and influencing characteristics were statistically compared between those with and without PSH.

Results

Fifty-one patients admitted with TBI required a tracheostomy. Of these, 10 patients were also diagnosed with PSH. The mean tracheostomy wean in the PSH group was longer compared to the non-PSH group (72.3, SD 61.0 versus 30.0 days, SD 16.2). This difference was statistically significant ($p = 0.007$, using Mann Whitney U test). The PSH group had more respiratory and oral secretions, but this was not statistically significant ($p = 0.16$ and 0.29).

Conclusions

This is the first study to demonstrate that PSH is associated with prolonged tracheostomy weaning in severe TBI. Awareness of this association should enable those planning rehabilitation to set realistic goals for a patient's tracheostomy weaning programme.

Key words

Paroxysmal Autonomic Instability, Dystonia, Intensive care, Neurorehabilitation

Introduction

In the initial period following a severe TBI, 14 – 18% patients require temporary tracheostomies to maintain a safe and effective airway (Gordon, 1995 & Richard, 2005). Once stable, the process of gradual reducing dependency on the tracheostomy (weaning) is initiated. This weaning is undertaken, as part of hyper-acute rehabilitation care, in neurorehabilitation units of some centres. The key weaning milestones include: cuff deflation for short periods; permanent cuff deflation; replacement with cuffless tube; use of speaking valve and finally decannulation (removal of the tracheostomy tube). Progression through weaning milestones is influenced by the patient's ability to maintain an airway and have satisfactory physiological parameters, which indicate clinical stability (Intensive Care Society, 2004). These physiological parameters are often abnormal during medical instability such as raised Intra Cranial Pressure (ICP), intercurrent illness and sepsis, when it would be appropriate for weaning to be delayed. However, sometimes these physiological parameters are abnormal for other reasons, notably PSH and autonomic dysreflexia.

PSH is not uncommon in severe brain injury and is characterised by episodes of sympathetic storm and clinical signs including fever, tachycardia, tachypnoea, hyperhidrosis and dystonic posturing. This phenomenon has been described in the literature with numerous names such as Paroxysmal Autonomic Instability with Dystonia (PAID), diencephalic seizures and midbrain dysregulatory syndrome. These episodes can occur either spontaneously or triggered by stimulus such as touch, pain or constipation (Perkes, 2010). The incidence of PSH is thought to be around 10 – 30% in severe brain injury (Laxe, 2013, Hughes, 2013 and Fernandez-Orlego, 2012). The diagnosis can be chal-

lenging; a recent consensus statement has attempted to clarify nomenclature and diagnostic criteria (Baguley, 2014). Since the diagnosis is one of exclusion, other differentials like infection and seizures need to be carefully excluded. This can subsequently delay appropriate treatment and it is known that this delayed recognition and treatment increases morbidity and long-term disability in these patients (Baguely, 1999).

Given that tracheostomy weaning is influenced by changes in the patient's physiological parameters, and that these parameters are characteristically abnormal in PSH, we hypothesise that the presence of PSH delays tracheostomy weaning. As far as we are aware, there are no studies in the current literature which examine this association of PSH and tracheostomy weaning. We hence designed a retrospective review study to investigate this association in more detail. Given the infrequent nature of this phenomenon, we felt that utilising an existing data source was the most efficient and effective way to analyse this relationship in order to identify key issues and implications for practice.

Methods

This was a retrospective cohort study of patients admitted to a specialist hyper-acute neurorehabilitation unit with a diagnosis of TBI and also required a tracheostomy during their hospital stay. The 20-bedded hyper-acute neurorehabilitation unit is situated within the acute hospital, which is a tertiary Neurosciences and Major Trauma Centre serving a population of approximately 3.3 million. The hyper-acute neurorehabilitation unit provides rehabilitation for patients with highly complex rehabilitation needs; patients are admitted at a very early stage in their rehabilitation when they still have needs requiring continued active support from acute trauma, neurosciences or acute medical teams (British Society of Rehabilitation, 2015). In this unit decisions around tracheostomy weaning are made by a specialist multi-disciplinary team (including a Consultant in Rehabilitation Medicine, Rehabilitation Specialist Nurse, Physiotherapist and Speech and Language Therapist) who conduct weekly tracheostomy ward round and complete an electronic specialist tracheostomy ward round proforma that includes ongoing issues and actions related to weaning. All treatment case notes and reports are saved in the electronic health records of the hospital.

The unit care records covering a period of 34 months were reviewed, following which the patients who had a diagnosis of TBI and also required a tracheostomy at any time during their inpatient hospital stay were identified for inclusion into the study. A standard study proforma was created to collect relevant data. This data was then collected by authors HR and MB. The data collectors were not blinded to the study objectives. Data collected included demographic characteristics such as age, sex, mechanism of injury, admission Glasgow Coma Scale (GCS) and the duration of stay in intensive care unit. As this was

a retrospective service evaluation study and data was collected from pre-existing clinical data without influencing clinical treatments, formal ethical approval was deemed not necessary.

The severity of TBI was determined by initial GCS score, period of Post-Traumatic Amnesia PTA) and length of admission in critical care unit (Friedland 2013). Patients with PSH were identified by the presence of the terms Paroxysmal Sympathetic Hyperactivity (or any other well-known alternatives) documented in the medical notes. This diagnosis was interrogated further by retrospectively applying the recently developed PSH-Assessment Measure (PSH-AM) (Baguely, 2014). This measure includes an assessment of the severity of PSH (mild, moderate or severe) and a tool predicting the certainty of diagnosis (not PSH, possible and definite PSH). Treatment strategies utilised for PSH management were also recorded. The duration of tracheostomy weaning was determined from the date of tracheostomy insertion and date of decannulation / removal. Factors influencing the tracheostomy weaning duration including oral and respiratory secretions, tracheostomy problems and infections were identified from the tracheostomy ward round reports and the patient's physiotherapy treatment notes. Patients were excluded from analysis if they did not complete their tracheostomy wean during the study period.

The patients with a probable or possible diagnosis of PSH were assigned to the PSH-group and those without PSH were categorised as the non-PSH group. The duration of tracheostomy weaning and other factors influencing weaning were compared between the two groups. Statistical analysis was carried out using The Mann Whitney U test for continuous and Fisher Exact Test for categorical data. A statistical significance with a value of less than 0.05 was deemed as significant.

Results

During the 34-month study period, 59 patients with TBI and had had tracheostomies inserted at some point during their management pathway, were admitted to the hyperacute neurorehabilitation unit. Eight patients were excluded from analysis either due to death prior to decannulation (n = 3), discharge from the unit with a permanent tracheostomy (n = 2) or failure to decannulate by the end of the study period (n = 3). The data from the remaining 51 patients were included for analysis in the study.

Out of the 51, 10 patients (19.6%) were also diagnosed with PSH at some point during their management pathway. Clinical features of PSH were classified as mild (range 4-6) in 5 patients (50.0%) and moderate (range 7-11) in 5 patients (50%) based on the PSH-AM tool. The diagnostic likelihood of PSH was probable in 4 patients (40%, range 17-21) and possible in 6 patients (60%, range 13-16).

The demographic characteristics of those patients with and without PSH are compared in Table 1. Sex, initial GCS and duration of stay in the intensive care unit were comparable between the two groups. The two groups however significantly differed in their mean age, with the patients in the PSH group being younger. Also, more patients with PSH received their injury from a road traffic collision, whereas those without were more likely to have fallen from a height. The type of traumatic brain injury was heterogeneous in both groups, however overall the commonest finding on initial CT was multiple findings that included subdural haematoma, multiple contusions and/or traumatic subarachnoid haemorrhage (15 patients, 29.4%).

The mean time for tracheostomy weaning for patients in the non-PSH group was 30.0 days (SD 16.2). The mean time in the PSH group was longer at 72.3 days (SD 61.0). This was a statistically significant relationship using the Mann Whitney U test ($p = 0.007$).

Clinicians regularly recorded the factors they felt were the cause for delayed wean on the tracheostomy ward round proforma or in the physiotherapy notes. Common documented factors delaying weaning included excess respiratory secretions (51.0% patients) and oral secretions (47.1% patients). A higher proportion in the PSH group had excess respiratory or oral secretions when compared to the non-PSH group (70.0% versus 41.4%, and 70.0% versus 46.3%)) however this difference was not statistically significant ($p = 0.16$ and 0.29). PSH was a documented as a factor in delayed tracheostomy weaning in 1 of the 10 affected patients. The other documented factors delaying weaning are compared in Table 2.

The treatment approach for PSH was heterogeneous. In the majority of cases Gabapentin or Pregabalin was used as the first-line medication (n = 7, 70%). Propanolol was added when symptoms persisted despite this (n = 4, 40%). One patient was treated with Clonidine, and 2 patients received no pharmacological treatment, instead the treating medical team aimed to reduce any potential triggering factors such as excessive manual handling, bowel/bladder problems or pain.

Discussion

This study is first of its kind in the current literature to suggest that presence of PSH is associated with prolonged tracheostomy weaning in individuals with TBI. Current literature suggests the factors that influence tracheostomy weaning in TBI are level of consciousness, site of brain injury, respiration, tracheal secretions, phonation, swallowing and cough (Zanata, 2014 and Mitton, 2017). Our study suggests presence of PSH also influences the weaning process. Understanding and recognising the factors that influence tracheostomy weaning is important for the multidisciplinary team managing these patients in an inpatient setting. It helps them to plan the rehabilitation program accordingly and implement treatment strategies, as well as ultimately plan the appropriate discharge destination (Aresani, 2013).

The etiology behind the association between PSH and prolonged tracheostomy weaning observed in this study is not clear. One hypothesis could be that those patients with PSH had a more severe and extensive brain injury and thus a more prolonged tracheostomy wean. The neuroanatomical basis for PSH is poorly understood, however it is thought that it could be due to disruption to components of the central autonomic network, many of which are deeper structures (Hinson, 2015). Hence those patients with brain injury involving the deeper structures, therefore could be more likely to have both PSH and ventilatory problems in the early stages. Assessing the extent of brain injury in this study would be challenging as not all patients had a MRI brain scan. All patients in this study had severe brain injury and there were no other characteristic difference between groups other than the presence of PSH (Table 1).

Although not statistically significant, the PSH-group had increased oral and respiratory secretions, the cause of which is uncertain, but may have accounted for the longer weaning times. Given that the control of secretions is predominantly parasympathetic driven (Rogers 2003), one would expect with heightened sympathetic activity, that secretions should be lessened. However, increased secretions can also drive PSH as a noxious stimuli, therefore their presence could worsen the severity of PSH. The association between oral and respiratory secretions and PSH is also not conclusive in current literature and needs to be explored in future research studies.

We feel that the difference in weaning times is due to the fact that the tracheostomy weaning protocol used in our hospital is based on physiological parameters, which are abnormal in PSH. This consequently explains the longer weaning times. The heightened sympathetic response, for example during suctioning, also may contribute to the reluctance to wean. Exclusion of infection and other differentials in the acute stages (i.e.in the intensive care setting), that would routinely occur in these patients, could not have been a factor for delay since this should only take only few days on an average. This would not explain the difference of more than 40 days between the two groups' weaning time.

Knowledge of this association between PSH and prolonged tracheostomy weaning should encourage those working in the acute rehabilitation setting to be more mindful for this condition. Baguely et al., have attempted to reach consensus regarding nomenclature and created a diagnostic tool (PSH-AM) to help with this early and timely diagnosis (Baguely, 2014). It is already acknowledged that delayed recognition and treatment of PSH increases morbidity and long-term disability in patients with traumatic brain injury. (Aresani, 2013). As we have preliminary data that could suggest a potential advantage

of early detection of PSH in patients with TBI and tracheostomy, it is important that this diagnostic criteria becomes integrated into standard practice. This will not only improve vigilance of the condition for those in the acute rehabilitation setting but it will also allow us to gain stronger data from medical records in future.

There are several limitations in this study. Firstly, retrospectively analysis of medical records has inherent weaknesses and potential for bias. Although we retrospectively interrogated the diagnosis of PSH using the recently published diagnostic tool PSH-AM, (Baguely, 2014) this tool is best implemented prospectively. Also, due to retrospective study design, we were unable to analyse the effect of prompt diagnosis and treatment of PSH on weaning time. Secondly, the sample size was small, which limits the generalisation of results. However, the condition is uncommon and it is extremely difficult to capture a reasonable sample of patients with TBI and tracheostomy from one centre. We however have a hyperacute neurorehabilitation unit that manages this patients from early on and the hospital has robust electronic records (including physiological parameter recording) for every patient encounter that has enabled us to capture the required data for this study. Finally, patients were excluded from analysis if they had died or not completed their wean; the influence of PSH on weaning in these patients therefore could not be undertaken due to lack of the end-point of successful decannulation.

In summary, PSH may be associated with prolonged tracheostomy weaning time in patients with TBI. As far as we are aware, this is the first study to report this association. Awareness of this should enable those working in the acute neurorehabilitation environment to set realistic goals and expectations for a patient's tracheostomy wean as part of planning their rehabilitation pathway. We would also recommend early diagnosis of the

condition using PSH-AM and initiating early treatment. Future prospective studies are needed to (a) confirm this observed association between PSH and tracheostomy weaning time and (b) explore whether changing the physiological parameter thresholds for weaning decisions is needed to optimise the weaning time in these patients and finally (c) whether early diagnosis and successful management of PSH optimises the tracheostomy weaning time.

Declaration of Interest

None declared

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None declared

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Table 1: A Comparison of the Characteristics of Patients with and without PSH

Characteristics	PSH group (n=10)	Non-PSH group (n=41)	Significance (p value)
Sex: Male Female	8 (80.0%) 2 (20.0%)	32 (78.0%) 9 (22.0%)	1.00
Age (years)	30.6 (SD 15.7)	51.1 (SD 15.4)	< 0.01
Mechanism of trauma: Road Traffic Collision Fall Gunshot Assault	 7 (70.0%) 2 (20.0%) 1 (10.0%) -	 12 (29.3%) 26 (63.4%) - 3 (7.3%)	 **0.028** **0.03** 0.20 1.00
CT findings: DAI Isolated SDH Contusions Traumatic SAH EDH Penetrating injury Multiple findings	 2 (20.0%) 3 (30.0%) 1 (10.0%) 1 (10.0%) 1 (10.0%) 1 (10.0%) 2 (20.0%)	 1 (2.4%) 11 (26.8%) 7 (17.1%) 6 (14.6%) 3 (7.3%) - 13 (31.7%)	 0.094 1.0 1.0 1.0 1.0 0.20 0.71
Initial Glasgow coma scale	5.3 (SD 3.4)	6.0 (SD 4.0) NB 1 exclusion as not documented	0.66
Duration in ICU (days)	23.0 (SD 15.9)	21.7 (SD 9.0)	0.55
Duration of Tracheostomy Wean (days)	72.3 (SD 61.0)	30.0 (SD 16.2)	**0.007**
Key: DAI – Diffuse Axonal Injury, SDH – Subdural Haematoma, Multiple – SDH plus contusions and/or traumatic SAH			

Table 2: A Comparison of Documented Factors Delaying Weaning in Patients with and without PSH

Factors delaying wean	PSH group (Mean weaning time 72.3 days)	Non-PSH group (Mean weaning time 30.0 days)	Significance
Rib fractures	-	5 (12.2%)	p = 0.57
Pneumothorax	-	1 (2.4%)	p = 1.00
Oral secretions	7 (70.0%)	19 (46.3%)	p = 0.29
Respiratory secretions	7 (70.0%)	17 (41.4%)	p = 0.16
Tracheostomy complications	2 (20.0%)	3 (7.3%)	p = 0.25
Respiratory Infections	2 (20.0%)	11 (26.8%)	p = 1.00
Other infections	-	2 (4.9%)	p = 1.00
Tone/posture problems	2 (10.0%)	-	**p = 0.035**
Agitation	1 (10.0%)	-	p = 0.20