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Olfactory function in acute traumatic brain injury

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ABSTRACT

Objective: Traumatic brain injury (TBI) represents a significant public health problem and is associated with a high rate of mortality and morbidity. Although TBI is amongst the most common causes of olfactory dysfunction the relationship between injury severity and olfactory problems has not yet been investigated with validated and standardized methods in the first days following the TBI.
Methods: We measured olfactory function in 63 patients admitted with TBI within the first 12 days following the trauma by means of the Sniffin' Sticks identification test (quantitative assessment) and a parosmia questionnaire (qualitative assessment). TBI severity was determined by means of the Glasgow Coma Scale (GCS) and by duration of post-traumatic amnesia (PTA) as measured by the Galveston Orientation and Amnesia Test.
Results: Poor olfactory scores correlated with a longer amnesia period, but not with GCS scores. Further, we observed higher parosmia scores in assault victims than in victims of falls or motor vehicle collisions.
Conclusions: We show that PTA is intimately related to olfactory problems following a TBI. Thus, a thorough evaluation of olfaction is essential in order to detect posttraumatic olfactory dysfunction and to take appropriate actions early on to help the individual deal with this impairment.

1. Introduction

Traumatic brain injury (TBI) represents an significant public health problem and is associated with a high rate of mortality and morbidity [1] as well as several physical and sensory disturbances. Sensory deficits such as auditory and visual disorders are well known and documented but olfactory problems are much less discussed. This is despite the fact that TBI are amongst the most common causes of olfactory dysfunction [2]. Potential pathomechanisms of posttraumatic olfactory dysfunction include stretching or contortion of the olfactory nerves as well as a direct injury to the brain and the central nervous system [3,4]. Fortunately, the olfactory system shows a very high degree of regeneration, typically within 2-4 months after injury [5]. Most studies on olfactory deficits in TBI patients were carried out several months after the trauma (e.g., [6]); hence many cases of olfactory deficits in acute TBI may go undetected, and olfactory dysfunction after acute TBI may be much more widespread than commonly thought.

Most studies on the effect of TBI on the sense of smell investigated quantitative olfactory dysfunction (anosmia: total loss of olfactory function; hyposmia: partial loss of olfactory function). Older studies based on notoriously unreliable auto-evaluation [7] reported 2-40% of patients with TBI to exhibit anosmia or hyposmia (see [8] for an overview). In fact, most TBI patients with an olfactory deficit do not know that they have a problem with their sense of smell [9,10]. When patients' olfactory abilities were directly assessed, TBI related olfactory deficit was found between 13% and

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65% [6,9–11]. Qualitative alterations such as parosmia (perception of qualitatively altered smells in the presence of an odor source [12]) may be found in up to 40% of patients with TBI related smell complaints [13], but only recently a parosmia test based on four standardized statements has been introduced [14].

Interestingly, the link between acute parameters of TBI severity such as the Glasgow Coma Scale (GCS) and the extent of quantitative olfactory dysfunction is poorly established [6,9–11]. Only one study did in fact observe a better identification scores for patients with a relatively high GCS, but no difference was detected between low and intermediate GCS scores [15]. This may be due to the fact that GCS considers only a limited time after the trauma [16] and may be affected by preclinical interventions. An alternative measure to determine TBI severity is post-traumatic amnesia (PTA) [17,18], which is relatively independent from the GCS score. In fact, duration of PTA as a measure of initial injury severity, is the best predictor of long term outcome after a TBI [19–23] over a wide range of degree of impairment [24]; it may therefore be better suited to investigate the link between TBI severity and olfactory dysfunction [15]. In this context it is extremely important to point out that PTA assessment should rely on objective testing such as the Galveston Orientation Assessment Test (GOAT) rather than on unreliable subjective reports [25].

In summary, only a few studies have examined posttraumatic olfactory deficits; most reports rely on data from several weeks, months or years post-TBI and on non-validated tools when assessing olfactory function or TBI severity. Better comprehension of how the sense of smell is affected shortly after a brain injury will allow us to improve our understanding of the impact of a TBI on the brain and subsequent recovery. In this study we therefore assessed quantitative and qualitative olfactory dysfunction in a group of patients with a TBI in the early days and weeks after the trauma. We specifically assessed the link between characteristics of the trauma, such a TBI severity or cause, and olfactory function. For all measures we used validated and standardized measures to reduce observer effects. We hypothesized that (a) injury severity is correlated with degree of olfactory dysfunction and that (b) injury mechanism is correlated with degree of olfactory dysfunction.

2. Material and methods

2.1. Participants

A total of 63 consecutive patients admitted to the Traumatic Brain Injury Program of the McGill University Health Center – Montreal General Hospital (MUHC-MGH) between April 2013 and October 2013 and diagnosed with a TBI were included in the study. Neuroimaging data are presented elsewhere (De Guise et al., in press). The diagnosis of TBI was made by the treating physician. A patient was diagnosed with a TBI when either (a) radiological imaging showed intracranial or cranial injury or (b) at least two of the three following indicators were present: (1) loss of consciousness, (2) altered state of consciousness (reduced GCS), and (3) amnesia for the event.

2.2. Injury severity

Glasgow Coma Scale (GCS): We collected GCS scores that were assessed upon admission to the emergency room. TBI severity was determined by GCS and documented according to the score (score of 13–15: mild TBI; 9–12: moderate TBI; 3–8: severe TBI).

Post-Traumatic Amnesia (PTA): We assessed the duration of PTA by means of the Galveston Orientation and Amnesia Test (GOAT [25]). This test is administered daily and consists of 10 items that involve the recall of events that occurred prior and after the injury, as well as questions about disorientation. A score of 75 or more (of 100) on this scale on two consecutive days indicates the end of the PTA episode [25]; we collected the length of PTA in days.

In addition to these measures we recorded trauma etiology, which fell into 4 categories (falls; motor vehicle collision; assaults; others (including sports accidents and suicides)). Patients who sustained facial fractures (facial fractures, nasal, maxillary sinus, orbital fractures), who were neither English nor French-speaking as well as patients with a pre-morbid dementia, psychiatric diagnosis, substance abuse problems or receiving narcotics were excluded from the study. Patients were seen once they were medically stable (outside of the intensive care unit). Approval for this study was granted by the research ethics board of the MUHC-MGH.

2.3. Procedure

Patients' olfactory function was tested by an experienced occupational therapist within the first 12 day following the trauma, or when patients were able to participate in formal testing following the PTA period evaluated with the GOAT [25]. We assessed quantitative and qualitative olfactory dysfunction. (1) We used the identification portion of the Sniffin' Sticks test to assess *quantitative* olfactory dysfunction [26,27]. Sixteen felt-tip pen-like odor dispensing device were presented to patients. Their task was to identify the odor by choosing from 4 different descriptors (forced choice). One point per correct answer was given for a maximum of 16 points (identification score). According to normative data, a score of less than 11 is indicative of hyposmia [26,28]. (2) We assessed *qualitative* olfactory dysfunction by means of four standardized statements described elsewhere [12,14]. These were: 1: "food tastes different than it used to before my accident"; 2: "sometimes I think I can smell something bad even when other people do not"; 3: "some of the smells that I find unpleasant, other people find pleasant", 4: "One of my biggest problem is that the smells used to smell different before my accident". For each statement,

Table 1
Overview over average values (SD: standard deviation) for key variables for patients within 4 categories of trauma etiology. GCS indicates initial score on the Glasgow Coma Scale; PTA indicates the duration of posttraumatic amnesia as assessed by the Galveston Orientation Amnesia Test (GOAT); Sniffin' Sticks indicate the number of correct odor identification (of a total of 16); parosmia indicates the presence and severity of parosmia symptoms.

Type of trauma	Variable dimension	Age years	Women/men n	GCS points	PTA days	Sniffin' Sticks points	Parosmia %
fall	Mean	58.2	10/23	13.3	9.0	8.6	17.7
	SD	19.2		2.6	6.4	3.9	30.8
motor vehicle collision	Mean	48.0	9/11	13.5	3.1	9.7	2.1
	SD	23.6		1.7	3.5	4.9	8.3
assault	Mean	39.5	1/5	13.7	6.8	7.7	60.0
	SD	16.0		1.5	5.2	5.2	41.8
other	Mean	51.5	3/1	8.3	24.0	3.3	33.3
	SD	17.2		5.7	18.9	5.9	44.1
Total	Mean	52.7	23/40	13.0	7.8	8.5	17.9
	SD	20.9		2.8	8.3	4.6	31.6