Olfactory Function After Mild Head Injury in Children

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Abstract

Olfactory impairment has been shown to be linked to head injury. In addition, it is believed that measurement of olfactory function after head trauma represents a sensitive tool for measuring frontal brain damage. Aim of the study was to evaluate the effect of mild head trauma in children on olfactory function over a time period of up to 1 year after head trauma. The olfactory function of 114 children who suffered mild head trauma according to the Glasgow Coma Scale was assessed 3 times with an interval of 4 months. In addition, healthy, age-matched controls were tested for comparison of olfactory function. Patients scored significantly lower on the odor threshold test compared to the control group—but still within normal range. Between the 2 groups, no difference was found for suprathreshold testing. Neither olfactory threshold scores nor olfactory discrimination scores changed significantly over the study period of 1 year. This data prove an impact of mild head trauma on olfactory function of children. It seems unlikely that children who suffered mild head trauma will become hyposmic or anosmic.

Key words: children, head injury, olfaction

Introduction

Olfactory impairment has been studied as a sequel of head injury since the 19th century (Ogle 1870). It is one of the main causes of olfactory loss. Estimates are that about 5% to 17% of olfactory dysfunction is due to head trauma (Temmel et al. 2002; Damm et al. 2004). The frequency of olfactory impairment after head trauma varies greatly between studies. Percentages of 4–65 were reported (Costanzo and Zasler 1992; Callahan and Hinkebein 1999; De Kruijk et al. 2002; Haxel et al. 2005; Collet et al. 2009). This is due to different methods of evaluating olfactory function and differences in the studied populations (Zusho 1982; Callahan and Hinkebein 1999; Green et al. 2003; Reden et al. 2006). Only recently standardized olfactory tests became available to assess olfactory function in a clinical setting (Doty et al. 1984; Hummel et al. 1997).

There are 3 main pathologies, which are responsible for olfactory dysfunction after head trauma: 1) shearing and damage of the olfactory nerves, 2) obstruction within the nasal passage, and 3) damage or hemorrhage in olfactory eloquent areas in frontal brain regions (Costanzo and Zasler 1992; Delank and Fechner 1996; Green et al. 2003; Tomii 2012). The coup-contrecoup mechanism plays an important role in the damage of olfactory structures (Delank and Fechner 1996). Occipital injuries have been reported to be more likely to result in olfactory impairment than other forms of injuries (Zusho 1982; Costanzo and Miwa 2006; Swann et al. 2006). Other predictors that have been identified to be related to olfactory impairment are loss of consciousness, posttraumatic amnesia, and severity of the trauma (Sumner 1964; Levin et al. 1985; De Kruijk et al. 2002; Green et al. 2003; Swann et al. 2006). Varney et al. (1988) reported about a correlation between olfactory impairment after head trauma and vocational problems. These findings were confirmed by Correia and colleagues (2001). Therefore, it is
believed that olfactory function is a marker of frontal brain damage—making olfactory assessment not only interesting for ENT examination (Roberts and Simcox 1996; Correia et al. 2001). There are only a few studies that examined olfactory function in children after head trauma. Roberts and Simcox (1996) found olfactory impairment in children after head trauma, which was correlated to the severity of injury. Although it has been shown that mild head injury can cause olfactory dysfunction in adults (Sumner 1964; Callahan and Hinkebein 1999; De Kruijk et al. 2002), Sandford et al. (2006) concluded that it is unlikely to find olfactory impairment after mild head trauma in children.

The aim of the current study was to evaluate the impact of mild head injury on olfactory function in children in a large population. In addition, olfactory function was assessed longitudinally over a time period of approximately 1 year.

Material and methods

To observe the impact of mild head trauma on olfaction in children, patients were examined who matched the inclusion criteria for this study. In addition, an age-matched control group was formed to be able to compare olfactory function to a healthy population. Detailed information about the experiment was given to all participants, and written consent was obtained from participants and parents. All aspects of the study were performed in accordance with the Declaration of Helsinki. The study protocol was approved by the local Ethics Board of the Faculty of Medicine of the TU Dresden (EK 65022011).

Patients

Patients aged 4–17 were selected. Inclusion criteria were incidence of mild head trauma defined by the Glasgow Coma Scale (GCS) between 13 and 15, aged 4–17, and no known olfactory impairment prior to the trauma. Causes for mild head trauma varied greatly, from bicycle accidents to falling etc. Children included into the study did not exhibit nose or skull fractures. The side of trauma was not recorded. We were able to include 125 children. Due to several reasons occurring during the course of the study (chemotherapy, surgery, Kawasaki-syndrome etc.), 11 children were excluded. Therefore, 114 children (59 girls, 55 boys) between 4 and 17 years of age (mean = 8.7 years, SD = 3.5) were included in the final analysis.

Testing

Olfactory testing took place 4, 8, and 12 months after head trauma. For the evaluation of olfactory function, parts of the “Sniffin’ sticks” test battery were used and administered birhinally (Kobal et al. 1996; Hummel et al. 1997; Kobal et al. 2000). The children’s suprathreshold olfactory function was tested using the odor discrimination test. The odor threshold test for phenyl ethyl alcohol was adapted for children. Instead of the usual 7 reversals of the staircase (Kobal et al. 2000), the test was terminated after 5 reversals. The average of the last 4 reversals was used as a threshold estimate.

Participants

An age-matched control group was recruited to be able to compare olfactory function between patients and healthy children. Fifty-six children (28 girls, 28 boys) aged 4–17 (mean = 9.5 years, SD = 3.3) without any signs of olfactory dysfunction or illness associated with olfactory impairment volunteered. All children in the control group underwent olfactory testing one time using the modified version of the “Sniffin’ Sticks” as described above.

Statistical analyses

Data were analyzed using SPSS 19.0 (SPSS Inc.). T-tests and Spearman correlations were applied wherever appropriate. The level of significance was set at 0.05.

Results

All together, results from 171 children (114 patients, 56 controls) were included in the final analysis. The 2 groups did not differ in terms of age (t = 1.51, P = 0.13) and gender distribution (χ² = 0.04, P = 0.87). Children from the control group underwent olfactory testing one time. On average, they achieved 11.38 (SD = 2.09) points on the discrimination task, and their odor threshold was 7.33 (SD = 1.82) points (Table 1). Age did not correlate with olfactory performance (threshold: r = 0.12, P = 0.39; discrimination: r = 0.14, P = 0.29), and no gender differences could be observed (threshold: t = 0.51, P = 0.61; discrimination: t = 0.83, P = 0.41). The 10th percentile of the normal distribution of olfactory scores in healthy controls was used to define normal olfactory function. Therefore, normosmia was reached above a discrimination score of 8.00 and a threshold score of 4.50 points.

Children, who had suffered mild head trauma (n = 114) underwent olfactory testing 3 times within 1 year after trauma (after 4, 8, and 12 months). By far not all children were able to attend all 3 appointments. Therefore, 48 children were tested 4 months, 73 children were tested 8 months, and 91 children were tested 12 months after mild head trauma. Twenty-six children attended all 3 appointments.

Children with mild head trauma scored significantly lower on the odor threshold test. This was consistent for all time points of the study period of 1 year (Table 1): For 4 months (t = 2.32, P = 0.02), 8 months (t = 2.08, P = 0.04), and 12 months (t = 2.50, P = 0.01). Although a difference in odor threshold between the 2 groups was found, patients still scored within the normal range as determined by the
control group. No difference in odor discrimination performance was observed. Olfactory test scores did not differ significantly between the 3 test sessions within the patient group (threshold: $F = 0.93, P = 0.40$; discrimination: $F = 0.32, P = 0.73$).

The subgroup of patients ($n = 26$) tested at all 3 appointments did not differ from the control group in olfactory threshold performance at 4 ($t = 1.43, P = 0.16$) and 8 months ($t = 1.30, P = 0.19$) after trauma. Threshold scores in the patient group declined slightly but not significant within the year; nonetheless, a significant difference in odor threshold scores between the groups was observed at 12 months after mild head trauma ($t = 2.50, P = 0.02$). Again, no difference was found in odor discrimination performance ($t = 0.20, P = 0.84$).

### Discussion

Aim of the study was to evaluate the impact of mild head trauma on olfactory function in children. Although children with mild head trauma scored within the normal range on olfactory tests, they were significantly less sensitive (using odor threshold measures) than children from the control group.

The control group was included to establish normative data for the modified olfactory test using the “Sniffin’ Sticks.” The modified test is more fitting for children due to their shorter attention span. Previous studies reported difficulties in administering the “Sniffin’ Sticks” test battery to children, especially under the age of 6 (Hummel et al. 2007). The results of this study show no age differences in the test score. Therefore, we conclude that the modification is suitable for children 4 years and older.

It is long known that head injury can cause olfactory impairment (Sumner 1964; Schechter and Henkin 1974). Several studies reported a correlation of trauma severity and frequency of olfactory dysfunction (Costanzo and Zasler 1992; Roberts and Simcox 1996; Green et al. 2003). The criteria for severity of trauma varied between studies. Some studies used the GCS (Costanzo and Zasler 1992; Roberts and Simcox 1996; Callahan and Hinkebein 1999; Sandford et al. 2006), whereas others used loss of consciousness (Green et al. 2003; Roberts et al. 2010). A third criterion, which was often used to measure severity of head trauma, is the duration of posttraumatic amnesia (Levin et al. 1985; Swann et al. 2006). Therefore, it is difficult to make comparisons between these studies. In our study, mild head trauma was defined by a score between 13 and 15 on the GCS measured at the point of admittance to the hospital. Previous studies are contradictory whether mild head injury would affect the sense of smell. Some data show olfactory impairment after mild head trauma (Costanzo and Zasler 1992; Callahan and Hinkebein 1999; De Kruijk et al. 2002; Roberts et al. 2010), whereas others failed to find an effect (Levin et al. 1985; Green et al. 2003). In the current study, patients scored lower on the olfactory threshold test compared to the control group, but they still scored within normal range. In the literature, it is described that suprathreshold tests are more likely to be affected than olfactory threshold tests (Costanzo and Zasler 1992; Green et al. 2003). The reason for this is believed to be the impact of head injury, which leads to brain damage rather than damage to the olfactory periphery such as the olfactory mucosa (Green et al. 2003; Hedner et al. 2010). In the current study, we were not able to confirm these findings because the suprathreshold test did not exhibit any difference between patients and healthy controls. Differences in our study to previous reports might be attributed to the age of the studied population. The impact of head trauma on a developing olfactory system (as in children) might be different to its impact on the adult olfactory system. In addition, it has to be noticed that the side of damage is not known. There is a tendency that damage to frontal brain regions results in suprathreshold olfactory deficits, whereas damage to the olfactory nerve is more likely to be accompanied by lower olfactory threshold scores. This might account for some of the differences between previous studies and the present work.

Olfactory function was assessed up to 1 year after head trauma. The initial difference in olfactory threshold performance between the 2 groups was still present 1 year after head trauma. Patients had slightly lower but stable threshold scores. Olfactory recovery rates after head trauma are reported to be lower than in other etiologies of olfactory impairment (Reden et al. 2006). In addition, olfactory recovery is more likely to occur early after head injury. Costanzo and Zasler (1992) reported that recovery takes place within 6 months, and Sumner (1964) even observed the most frequent recovery rate within 10 weeks after the incident. It has been reported that olfactory impairment after head trauma is rather permanent when recovery does not happen early.
(Sumner 1964; Reden et al. 2006). Only a few cases are mentioned where olfactory recovery occurs after a longer interval (Welge-Lussen et al. 2012). In our study, olfactory function of patients was not assessed until 4 months after head injury. Therefore, we might have missed patients with initial olfactory impairment, who recovered fast after head trauma. It could also be that patients who initially exhibited olfactory dysfunction after head trauma recovered to normal olfactory function but still measurable below the olfactory function of healthy subjects. Previous studies have reported that the olfactory system is more likely to recover after damage in young compared with old age (Suzukawa et al. 2011). The turnover of olfactory receptor neurons making it possible to grow new axons from the olfactory periphery to the olfactory bulb is higher in young age and decreases with ageing (Loo et al. 1996). Therefore, future studies are necessary to observe olfactory function in children during the first weeks after head injury.

In our study, hyposmia in patients did not occur more often than in the control group. Nonetheless, it is important to assess the olfactory function after head trauma in children. It has been reported that olfactory deficits after head trauma in adults are correlated with vocational problems (Varney 1988; Correia et al. 2001). Therefore, olfactory dysfunction is thought to be a sensitive marker of frontal brain damage even when neuropsychological examinations fail to report abnormalities (Varney 1988; Correia et al. 2001; Green et al. 2003). In children, executive dysfunction after head trauma is more likely when olfactory dysfunction is present (Roberts and Simcox 1996; Roberts et al. 2010). Using olfactory testing, children with frontal brain damage could be identified and support could be provided to prevent vocational problems in the future.

The children included in this study did not undergo any further testing such as attentional or cognitive testing. The focus of the current study was to investigate the impact of mild head trauma on olfaction. Future research is needed to address the relationship of mild head trauma, olfaction, and cognitive measures.

Examinations revealed that olfactory impairment in children after head trauma occurs at a lower frequency than in adults (Jacobi et al. 1986; Sandford et al. 2006). Our study is in line with these findings, but it has to be considered that in our study only children with mild head trauma were examined. The rate of olfactory dysfunction is likely to increase with trauma severity.

**Conclusion**

Although children, who suffered mild head trauma, as defined by the GCS, scored lower on the olfactory threshold test, it is not likely that mild head trauma will result in hyp anosmia. The assessment of olfactory function still provides valuable information because it is believed to be a marker of frontal brain damage.

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**Conflict of interest**

None of the authors has any conflict of interest to disclose.

**References**


Ogle W. 1870. Anosmia, or cases illustrating the physiology and pathology of the sense of smell. Med Chir Trans. 53:263–290.


