Volunteer studies of experimental, low-velocity rear-end collisions have shown a percentage of subjects to report short-lived symptoms, but the cause of these symptoms remains unknown. It is unclear whether the symptoms arise from biomechanical stress causing injury or from psychological stress causing symptom expectation and anxiety. Similarly, the cause of symptoms remains obscure in virtually all “whiplash” patients because it is impossible to identify acute pathology in many cases. In this study subjects were exposed to placebo collisions that almost completely lacked biomechanical stress. It was highly probable that if the symptoms reported following low-velocity collisions were not due to injury but to other factors (including misattribution of symptoms from other sources), then the proportion of subjects reporting symptoms would be similar to that reported for volunteers in true (experimental) low-velocity, rear-end collisions. A total of 51 volunteers (33 males and 18 females, mean age 32.4 years) were recruited through local newspaper advertisements. An experimental set-up for a placebo collision was constructed using two standard European cars. At time T0, prior to the placebo collision, a history and physical examination was performed, including a psychological analysis (Freiburger Personality Inventory). A symptom history and physical examination were also performed at time T1, immediately after the placebo collision, and the subjects completed symptom questionnaires 3 days (time T2) and 4 weeks (time T3) after the placebo collision. Data analysis included a determination of the predictive value of psychological data for the presence of symptoms following exposure to a placebo collision. At time T1, 9 out of 51 participants (17.6%) indicated symptoms. Within 3 days (time T2) after the placebo collision, 10 (19.6%) of the subjects had symptoms, and within 4 weeks (time T3) 5 subjects (9.8%) had symptoms. Of the last group, two of the five did not relate these symptoms to the “collision”. Subjects who endorsed symptoms at time T1 had significantly higher scores on the psychological scale of psychosomatic disorders (measured at time T0). Subjects endorsing symptoms at time T2 had significantly higher scores on emotional instability. There was also a tendency to higher scores on this sub-scale for subjects with whiplash-associated disorders (WAD) at time T3. A discriminant analysis using all four psychological scales from time T0 had a power of 87%, 83% and 92% for correct classification of subjects as symptomatic or asymptomatic at times T1, T2 and T3, respectively. Approximately 20% of subjects exposed to placebo, low-velocity rear-end collisions will thus indicate WAD, even though no biomechanical potential for injury exists. Certain psychological profiles place an individual at higher risk for this phenomenon.

Keywords Whiplash injury · Neck sprain · Placebo · Rear-end collision · Psychological examination
Introduction

The medico-legal expert often needs to correlate certain injury types and patterns with the mode and intensity of the energy transfer [17]. The forensic evaluation must always consider possible extremes, for instance the occurrence of severe disease after minimal trauma. Also simulations (faking) must be considered, but self-inflicted injuries are usually superficial and the history of foreign infliction is much more severe [15]. However, extremes of self-aggression may end in self-destruction [15, 16].

The evaluation of injury patterns and types can be further impaired if vascular reactions are primarily involved and if such disturbances lead to tissue damage only secondarily [12, 13].

All of the aforementioned types of correlation can exist in whiplash injuries after car accidents, rendering their evaluation potentially extremely difficult.

It is estimated that in the United States whiplash injuries cost $4.5 billion annually [42], and yet no definite conclusions can be made regarding effective treatment for this problem [6, 8]. One of the main reasons for this controversy is the current inability to identify an objective pathology that explains the acute or chronic symptoms in any consistent manner: the door to speculative medicine is wide open. Given decades of speculation, it is not surprising that in 1995 the Quebec Task Force [35] found the research reported in the literature “seriously deficient”.

Low-velocity collisions are a particular aspect of the controversy, given questions raised about the injury potential in these collisions. In Germany, 65% of rear-end collisions leading to injury claims involve striking velocities in the order of 30 km/h or less [3]. Understanding the mechanism of symptoms in these lower velocity collisions, although the results may not be generalisable to higher-velocity collisions, remains relevant to a sizeable proportion of whiplash patients.

There is heated scientific controversy as to whether WAD (whiplash associated disorders, including emotional and cognitive impairment) in these cases are triggered by initial injuries or are in part or completely fabricated, i.e. simulated or otherwise independent of the trauma [8, 9, 10, 11, 18, 19, 22, 25, 26, 27, 28, 31, 32, 33, 34, 36, 37, 38, 39, 40].

In 1997, Castro et al. [2] published the results of an experimental study with 19 volunteers in which low-velocity, rear-end car and bumper-car impacts were analysed. According to Meyer et al. [23, 24] they used the velocity change due to collision (delta-v) as a surrogate measurement for the biomechanical stress acting on the affected person due to a collision. If the delta-v did not exceed 11 km/h, no symptoms were reported, neither were impact-related changes found on physical examination or MRI. Brault et al. [1] published the results of an experimental study with 42 volunteers. In contrast to the findings of Castro et al. [2], they reported that following exposure to a collision with a delta-v of 4 km/h, 29% of the subjects had WAD. Since the authors of that study did not provide a control group or placebo exposure, the origin of the symptoms in such cases remains unclear. Ferrari [5] and Kwan [21] indicated that symptoms reported may arise from other factors, including the possibility that coincidental symptoms that would otherwise have gone unnoticed or been ignored in daily life were amplified and registered in the experimental environment. Another possibility is that psychological consequences of the exposure to a collision (such as anxiety) may lower the pain threshold so that normal sensations are regarded as abnormal and “painful”, or that such symptoms may be the somatic component of the anxiety itself. In experiments to date, no attempt has been made to determine whether the symptoms are truly a result (consequence) of exposure or merely a temporal association (subsequence). Therefore, to determine this relationship one should also expose subjects to a simulated collision. We thus undertook a study to expose subjects to a placebo collision to see how often “whiplash” symptoms arose in the absence of injurious exposures.

Material and methods

Test subjects

Subjects were recruited by advertisements in local newspapers. All subjects gave consent to participate in the study after they were informed in detail about the characteristics of the study. With regard to the physical stress they would be exposed to during the “rear-end collision”, they were told that this would definitely not exceed the effects of rear-end collisions in bumper-cars at the funfair. They signed a form waiving liability for injury. Exclusion criteria were age below 18 years or above 65 years, abnormal findings at the physical examination prior to the placebo collision, past surgery of the cervical spine, known recent injury and sports activity or excessive alcohol consumption during the 2 days before the study.

Of the first 60 consecutive subjects responding to the advertisement and agreeing to be interviewed for eligibility, 9 were ruled out on to the criteria cited above. The remaining 51 subjects comprised 33 males and 18 females with a mean age of 32.4 years (minimum 18 years, maximum 58 years), a mean height of 177.8 cm (minimum 155 cm, maximum 198 cm) and a mean body weight of 75.0 kg (minimum 52 kg, maximum 110 kg).

Experimental collision set-up

The experimental set-up at the test track was designed to provide sufficient sensory cues to the subjects sitting in the struck vehicle so that they would believe they had been in a rear-end collision between two standard European cars. Yet, there would be no collision between the two vehicles, merely the perception for the subject that a collision had occurred: a placebo collision. For this purpose, prior to the study, we struck an Audi 200 against an Opel Ascona (rear-end collision, with an impact speed of almost 20 km/h, the vehicle coming from behind being parallel but 30% offset from a direct line of impact). This produced debris and glass splinters which were then collected and used “for show” after the placebo collisions. The procedure was as follows: first, the test subject would be brought to the vehicle they would occupy. A curtain blocked their full view of the vehicles involved so they could not see the damage already produced. (The subjects would be later shown the vehicle damage and splinters on the track as a visual cue to confirm their perceived collision experience.) Figure 1a, b illustrates the positioning of the vehicles as the subjects saw them. In order to produce the acoustic impression of a collision, the “striking” vehi-