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No stress – no whiplash?

Prevalence of "whiplash" symptoms following exposure to a placebo rear-end collision

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Abstract 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

All data in this study were acquired within the scope of a thesis and were also presented at the annual meeting of the Spine Society of Europe, Munich, 1999

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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-

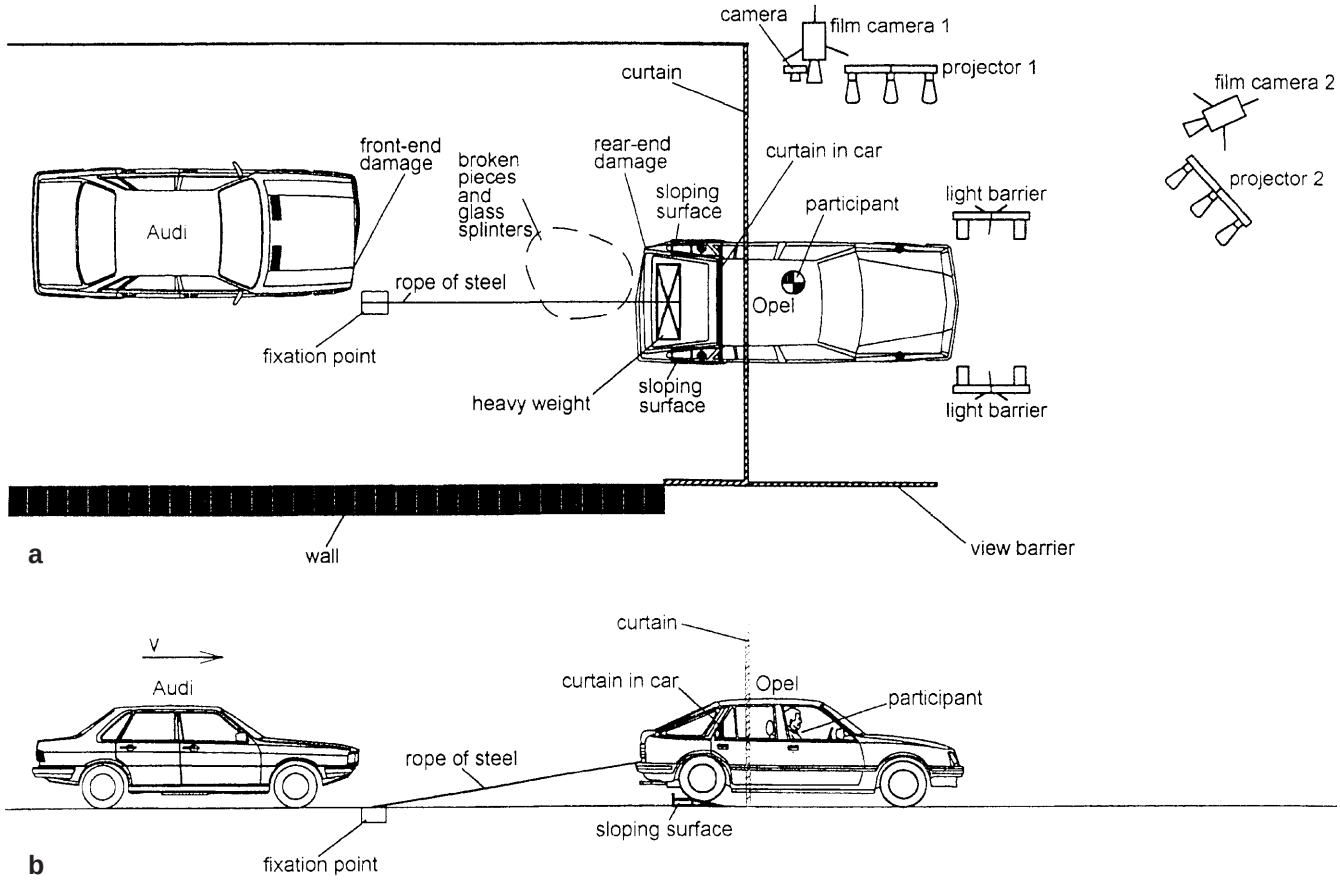

Fig. 1 a, b View of the experimental set-up: **a** from above; **b** side view

cle would move forward and the driver would brake suddenly (in a manner that could be heard), with no contact made with the test subject's vehicle. To add a further acoustic cue, the well for the spare wheel of the Opel ("struck" car) was removed from the boot (trunk) of the Opel and replaced with an iron plate. On this plate, there was a 110 kg iron weight which was raised by approximately 15 cm on one side with a bottle underneath it. At the same time as the driver of the Audi forcefully applied the brakes, by external activation the 110 kg weight would fall down on the bottle and the sound of breaking glass would be heard. To create a forward motion of the Opel, the car was placed with its rear wheels on a sloping surface (height 6 cm). The car was fixed in this position by a steel rope. When the weight fell on the glass, the rope also released the struck car, thus adding a visual and perhaps vestibular cue to simulate a collision. After the struck vehicle came to a stop, the back curtain was removed and the test subjects were shown the vehicle damage and debris. Each placebo collision was recorded by two video cameras.

Acceleration of the vehicles and of the head and chest of the subjects was measured with one-dimensional accelerometers to document that no biomechanical stress had been produced. The acceleration was measured according to previously published methods [2].

Clinical data collection and analysis

Prior to participating in the placebo collision, at time T0, each subject gave a medical history and underwent a physical orthopaedic examination of the upper part of the body. Because the subjects would not be exposed to any relevant biomechanical stress, a radiological examination of the cervical spine was deemed an unnecessary exposure. Also at time T0, each subject completed four subscales of the Freiburger Personality Inventory (FPI) [4]. This instrument was selected because it is valid for a non-clinical sample, has high retest reliability, and has been used previously in studies concerning the "whiplash problem" [28]. The four sub-scales of the FPI used for analysis include life satisfaction (including job satisfaction and comfort with private life), tendency to psychosomatic disorders, health concern (fear of illness), and emotional instability. Each scale results from answers to 12–14 questions per scale. The results of the four sub-scales of the FPI at time T0 of the study subjects were compared with results from samples of the general German population [4]. Immediately after each placebo collision, on the same day (time T1), a history was taken once again and a physical examination performed. At 3 days (time T2) and at 4 weeks (time T3) after the placebo collision, each subject completed a questionnaire asking whether they had any symptoms, focusing on symptoms such as headache, neck pain, arm complaints, etc. After returning the final T3-results, each subject received a payment of 50 DM.

The T1–T3 symptom data of each subject were related to the FPI results at time T0 by the psychologist (C.N.). The participants were divided into two groups: one with and one without symptoms after the placebo collision. For the statistical analysis, the Mann-Whitney U-test was used and the level of significance was 5%.

Results

The 51 subjects each underwent a placebo collision. The mean vehicle acceleration was approximately 0.3 m/s^2 i.e. almost 0.03 *g*. After being released from the steel rope, the "struck" car rolled off the sloping surface and moved forward approximately 1.70 m.

Table 1 WAD reported by nine subjects at time T1 (immediately after the placebo collision)

Subject	Gender	Age (years)	Symptom
1	Male	25	Fright, neck stiffness
2	Male	33	Fright, neck stiffness
3	Male	34	Fright, dizziness[a]
4	Male	40	Fright
5	Female	23	Fright
6	Female	28	Palpitations
7	Female	28	Low back burning[a]
8	Female	37	Fright
9	Female	49	Fright

[a] Complaints lasted only a few seconds

Symptoms

At time T0, none of the volunteers recalled any symptoms such as neck or back pain or headache. Eight volunteers, however, recalled periods of neck pain or pain in the shoulder region in the year before participation. None of the subjects voluntarily recalled having headaches, 1 volunteer recalled previous upper (thoracic) back pain and 12 recalled low back pain. Physical examinations revealed no abnormalities of range of motion of the cervical spine, and 18 volunteers had muscular tenseness without pain, especially of the trapezius muscle.

At time T1 immediately following the collision, 9 out of 51 subjects (17.6%) had symptoms, which in 4 cases were limited to clear vegetative fright reactions such as tachycardia, palpitations and "trembling knees" (Table 1). All subjects attributed their symptoms to the collision they thought they had experienced. The physical examinations revealed no differences with the ones before the placebo collisions. Ten subjects (19.6%) indicated symptoms at 3 days after the placebo collisions (time T2), of whom 3 already had symptoms at time T1 (numbers 1–3 in Table 2). At time T3, only five subjects indicated symptoms, of whom four had also reported symptoms at time T2. However, two of these five no longer related their symptoms at time T3 to the "collision" (Table 3).

Psychological analysis

Subjects with symptoms immediately after the placebo collision (T1) showed higher scores in the sub-scale psychosomatic disorders at time T0 ($P < 0.05$) (Table 4). Subjects with symptoms at time T2 (3 days) scored significantly ($P < 0.05$) higher on the sub-scale emotional instability at time T0 (Table 5). Subjects with symptoms at time T3 (4 weeks after the placebo collision) scored lower on life satisfaction (less satisfaction) and higher on emotional instability (more instability) and health concern (more concerned for health) at time T0. However the difference between the subjects with and without symptoms on this sub-scales at time T0 did not reach statistical significance (Table 6). A discriminant analysis using all four psychological scales from time T0 had 87%, 83% and 92% powers for a correct classification of subjects as symptomatic or asymptomatic, at times T1, T2, T3, respectively. Compared to the average German population [4], the average values of all participants of the study were higher for life satisfaction and lower for the other three sub-

Table 2 WAD reported by 10 subjects at time T2 (days 1–3 after the placebo collision)

Subject	Gender	Age (years)	Symptom	Symptom onset (h after placebo collision)	Symptom duration (h)
1	Male	25	Neck pain, nausea, vomiting	0	20
2	Male	33	Neck/shoulder pain	0	5
3	Male	34	Neck pain, dizziness, tinnitus	2	12
10	Male	22	Poor concentration	0.5	1
11	Male	25	Headache, nausea	0.5	2
12	Male	28	Neck pain	3	24
13	Male	39	Neck pain	4.5	12
14	Male	39	Neck pain	2	24
15	Female	35	Neck pain	36	36[a]
16	Female	44	Headache, fatigue	2	70[a]

[a] Complaints lasted longer than 3 days after the placebo collision

Table 3 WAD reported by five subjects at time T3 (days 4–28 after the placebo collision)

Subject	Gender	Age (years)	Symptom	Symptom onset (days after placebo collision)	Symptom duration (days)
2[a]	Male	33	Paresthesia of arms and feet, numbness of the lip, both arms powerless	14	14[b]
3	Male	34	Neck pain	5	2
15	Female	35	Neck pain	2	2
16	Female	44	Neck pain, headache, dizziness, paresthesia of the right arm	0	28[b, c]
17[a]	Male	54	Low back pain	27	1[b]

[a] These participants did not relate complaints to the placebo collision
[b] Complaints lasted longer than 4 weeks after the placebo collision
[c] Intermittent complaints

Table 4 Results of psychological assessment (at time T0) of subjects with WAD at time T1 immediately after the placebo collision (*SD* standard deviation, *n.s.* not significant)

Scale	Complaints		No complaints		Mann-Whitney U-test
	Mean	SD	Mean	SD	
Life satisfaction	7.33	3.57	8.28	2.29	n.s.
Psychosomatic disorders	2.50	2.20	1.10	1.36	$P < 0.05$
Health concern	3.00	2.45	3.31	2.20	n.s.
Emotional instability	3.89	2.15	3.57	2.46	n.s.

Table 5 Results of psychological assessment (at time T0) of subjects with WAD at time T2, days 1–3 after the placebo collision (*SD* standard deviation, *n.s.* not significant)

Scale	Complaints		No complaints		Mann-Whitney U-test
	Mean	SD	Mean	SD	
Life satisfaction	7.20	3.88	8.34	2.10	n.s.
Psychosomatic disorders	1.78	1.72	1.22	1.56	n.s.
Health concern	3.80	2.47	3.12	2.17	n.s.
Emotional instability	5.30	3.06	3.22	2.04	$P < 0.05$

Table 6 Results of psychological assessment (at time T0) of subjects with WAD at time T3, 4–28 days after the placebo collision (*SD* standard deviation, *n.s.* not significant)

Scale	Complaints		No complaints		Mann-Whitney U-test
	Mean	SD	Mean	SD	
Life satisfaction	6.00	3.74	8.34	2.33	n.s.
Psychosomatic disorders	1.60	2.30	1.29	1.52	n.s.
Health concern	4.80	2.68	3.08	2.14	n.s.
Emotional instability	6.00	4.06	3.37	2.05	n.s.

Table 7 Differences in psychological scales between the participants of the study and the average German population (*SD* standard deviation)

Scale	Study participants (T0)		Average German population	
	Mean	SD	Mean	SD
Life satisfaction	8.10	2.56	7.03	1.13
Psychosomatic disorders	1.23	1.58	4.04	3.04
Health concern	3.25	2.23	5.92	3.16
Emotional instability	3.63	2.39	6.18	3.55

scales (i.e. the subjects, compared to the general population were more satisfied with life and less likely to be emotionally unstable, with less tendency towards psychosomatic disorders and fewer health concerns) (Table 7).

Discussion

In the present study, almost 20% of the subjects indicated "whiplash-like" symptoms within 3 days after the placebo collision. None of the test subjects raised any doubts about having been in a real collision. The acceleration the subjects experienced in the placebo collision (i.e. 0.03 *g*) was negligible, 4–5 times less than a person taking their first step when they start walking. Yet, symptoms were reported and attributed to this exposure. How can this be explained?

There are a number of possibilities. These have been suggested elsewhere [5, 21] as a potential explanation for the similar findings in the study by Brault et al. [1] of symptoms reported after a low-velocity collision with a delta-v of 4 km/h. Such explanations seem necessary, as a delta-v of 4 km/h is more or less equivalent to a mean vehicle acceleration of 1 *g*. The cervical spine regularly experiences such stresses in daily life without symptomatic sequelae (e.g. when bending the trunk forward in a standing position). Thus, participating in a collision experiment may cause psychological responses such as psychosomatic sensitisation and anxiety. Anxiety can amplify bodily sensations, thereby lowering the pain threshold and causing trivial and normal bodily sensations to be perceived as more severe or abnormal. Furthermore, the general population has a background incidence of these symptoms coming and going in daily life, yet few can recall them. The failure to recall may be due not to an absence of such symptoms, but rather to the absence of a need to remember them: they have no significance, being viewed as minor and benign in most cases. In the setting of an experiment, symptoms that occur coincidentally as part of daily life will now have a special significance and on that basis be registered in a fashion that draws further attention to them. Additionally, anxiety and low life satisfaction are proven predictors of chronicity of back and neck pain [20]. Therefore, the interpretation of the collision accident as dangerous may open the development of chronicity for subjects with a high psychological risk profile.

It is clear from our study that accident victims may quite honestly believe they have been physically injured in a low-velocity collision even when they have not been injured. The subjects of this study were convinced they had been involved in a collision and attributed their initial symptoms and even later symptoms (in all but two cases) to a collision that from a biomechanical point of view never took place. Physical injury is clearly not necessary for the occupant to register symptoms and attribute them to the collision. It is important from both the patient's and the clinician's point of view to know whether symptoms following an accident arise from injury or not. The patient's perception of the origin of their symptoms may greatly influence their behaviour and be one factor on the road to chronic pain [6, 7]. This issue also has legal implications, as it is all too frequently assumed that the symptoms often reported after low-velocity collisions arise from injury [41].

It is apparent that certain psychological profiles increase the likelihood that symptoms will be reported. The psychological analysis showed that subjects with symptoms immediately after the placebo collision had a higher score on the psychosomatic disorder scale. Subjects who

were more emotionally unstable, less content with their life and more concerned about their health also had a higher risk of reporting symptoms after the placebo collision. These persons may focus more on minor (non-pathological) changes in bodily perceptions and by doing so amplify them. As a result a vicious circle starts. As these factors are seen to occur in almost 20% of the subjects in placebo collisions, it is likely that they have at least as much importance when symptoms are noted after a real traffic accident.

The influence of such factors is perhaps even underestimated in our study: the comparison of the FPI analysis of the participants in this study and the average German population shows that our participants have more life satisfaction, fewer psychosomatic disorders, fewer health concerns, and less emotional instability. Thus, to deal with the controversy of whiplash and low-velocity collisions, one should focus more on a general fact of traumatology: an injury occurs when the biomechanical stress acting on a particular part of the body (e.g. the spine) exceeds the maximum physiological level. Determining an exact injury threshold is difficult, especially for the different subject types and accident types that may occur, but one can at least define a reasonable minimum threshold below which injury is highly unlikely. Castro et al. [2] found that volunteers started to report symptoms when the velocity change exceeded 11 km/h: no impact-related objective findings were even registered up to a velocity change of 14.2 km/h (a psychological analysis of these volunteers is not reported). Another important result of the study of Castro et al. [2] was that from the biomechanical point of view, the stresses in most rear-end car collisions were the same as in bumper-car collisions: delta-v and acceleration signals over time in struck bumper-cars and struck normal cars were identical. In bumper-car collisions, on the one hand maximum biomechanical stresses were measured of up to 15 km/h [23, 24], and on the other hand no series of "whiplash injuries" after bumper-car driving can be found in the literature: as far as we know only one case of a "whiplash injury" after bumper-car use is reported [14].

Symptoms, however, may not always be the most useful surrogate for injury threshold. Although the aim of the study presented here was not to ascertain a threshold, it definitely affirms how important it is to consider psychological factors when defining such a threshold (even if there is no or little biomechanical stress there might be relevant psychological stress). Furthermore, it seems imperative that future low-velocity studies designed to evaluate symptom thresholds be conducted as randomised, placebo-controlled trials. Only by having a control group exposed to a placebo collision can one estimate the increased risk of symptoms from a given collision exposure. One may never be able to define a threshold injury for a given subject, but in both medicine and the courts, one is often faced with the problem of risk in a specific individual, and sample probabilities from studies should be relied upon.

In conclusion, a placebo collision was found to induce WAD in almost 20% of subjects. This may explain symptoms in those persons exposed to low-velocity rear-end collisions where it is likely that no physical injury exists. Certain psychological profiles place individuals at higher risk for this phenomenon. This study also reveals why the results of a study, e.g. that of Brault et al. [1], cannot be properly interpreted without a control group subjected to a placebo collision.

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