European Journal of Trauma and Emergency Surgery (2020) 46:463–472
https://doi.org/10.1007/s00068-019-01231-0

ORIGINAL ARTICLE

# The influence of alcohol on the outcome of trauma patients: a matched-pair analysis of the TraumaRegister DGU®

**Nils Wagner[1] · Borna Relja[1] · Thomas Lustenberger[1] · Max Leiblein[1] · Sebastian Wutzler[2] · Rolf Lefering[3] · Ingo Marzi[1] · the TraumaRegister DGU3**

Received: 26 May 2019 / Accepted: 12 September 2019 / Published online: 25 September 2019


## Abstract

**Background and purpose** In the diagnosis and treatment of trauma patients, numerous individual and trauma-related factors must be considered, all of which may influence the outcome. Although alcohol exposure is a major risk factor for an accident, its influence on the outcome is unclear. This matched-pair analysis investigates the hypothesis that alcohol has no negative impact on the outcome of trauma patients.

**Materials and methods** In a retrospective matched-pair analysis of the multi-centre database of the TraumaRegister DGU® patients with a maximum Abbreviated Injury Scale (MAIS) of 3 or greater from the years 2015 and 2016 with an alcohol level ≥ 0.5‰ were compared to patients with a measured alcohol level of 0.0‰. The patients were matched according to age, gender, AIS body regions (head, thorax, abdomen, pelvis/extremities) and survival presumption (Revised Injury Severity Classification Score (RISC) II intervals).

**Results** After matching, a total of 834 patients were enrolled, with 417 patients in group with positive blood alcohol levels (BAL+) with a median alcohol level of 1.82‰ and 417 patients in the negative-alcohol group (BAL −). As a mechanism of injury, the BAL+ group showed more often penetrating injuries, pedestrian accidents and low energy falls compared to car and motorcycle accidents in the BAL − group. BAL+ patients were significantly less sedated (BAL −: 66.7% vs. BAL+: 56.2%, $p = 0.002$), less frequently transported by rescue helicopter, were more frequently hypotensive (BAL −: 42 patients (10.3%) vs. BAL+: 61 patients (15.2%), $p = 0.045$, Table 2) and exhibited lower base excess levels associated with an acidotic metabolic status compared to sober patients (acidosis: BAL −: 24 patients (6.1%) vs. BAL+: 61 patients (17.2%), $p < 0.001$). There was no difference regarding in-hospital complications, length of stay or mortality rate.

**Conclusions and implications** Our data demonstrate that alcohol exposure in trauma patients has no impact on complication or mortality rates. On the other hand, there are initially clear differences in the mechanism of injury, sedation, mode of transport and the acid–base balance.

**Keywords** Alcohol · Outcome · Trauma patients · Base excess · Acidose

## Introduction

The treatment of trauma patients is a daily challenge. In 2016, more than 40,000 patients were documented in the hospitals of the TraumaRegister DGU® (TR-DGU), of which more than 27,000 patients had a maximum Abbreviated Injury Scale of 3 or higher (MAIS3 +), while 3554 patients died in the hospital [1, 2].

If the injured patients exhibited positive blood alcohol level (BAL), an additional variable which influences the diagnosis and treatment is added. On the one hand, the anamnesis and clinical assessments are difficult. Is the reduced Glasgow Coma Scale (GCS) conditioned by

TraumaRegister DGU: Committee on Emergency Medicine, Intensive Care and Trauma Management (Sektion NIS) of the German Trauma Society (DGU).

✉ Nils Wagner
nils.wagner@kgu.de

[1] Department of Trauma, Hand and Reconstructive Surgery, University Hospital Frankfurt, Goethe-University, Frankfurt, Germany

[2] Department of Trauma, Hand and Orthopaedic Surgery, Helios Dr. Horst Schmidt Hospital, Wiesbaden, Germany

[3] IFOM, Institute for Research in Operative Medicine, Faculty of Health, University Witten/Herdecke, Ostmerheimer Straße 200, 51109 Cologne, Germany

a traumatic brain injury or alcohol intoxication, or is the decreased oxygen saturation due to a chest trauma or a negative respiratory influence from the alcohol consumption? On the other hand, it is still unclear if alcohol has a positive or negative impact on the outcome of trauma patients. In numerous clinical, in vivo and in vitro studies, the effects of alcohol were investigated with very different results [3–12]. Studies with increased mortality, increased complication rates and worse outcomes [8, 13, 14] are opposed to studies with decreased mortality, reduced organ damage, and improved outcome [3, 5, 15]. As underlying mechanisms, amongst others, the influence of alcohol on the immune system, the post-traumatic inflammation, the coagulation, and the acid–base balance are discussed [8, 15, 16].

In general, the study comparability is often difficult or even not possible, because the inclusion criteria were chosen differently, the alcohol subgroups were defined differently in terms of alcohol concentration, or the outcome was measured at different times with different endpoints.

We hypothesized that alcohol exposure does not have a negative impact on outcomes following injury and we set out to test the hypothesis by matched-pair analysis on the TraumaRegister DGU® data.

## Materials and methods

The TraumaRegister DGU® of the German Trauma Society (Deutsche Gesellschaft für Unfallchirurgie, DGU) was founded in 1993. The aim of this multi-centre database is a pseudonymised and standardised documentation of severely injured patients.

Data are collected prospectively in four consecutive time phases from the site of the accident until discharge from hospital: (a) pre-hospital phase, (b) emergency room and initial surgery, (c) intensive care unit and (d) discharge. The documentation includes detailed information on demographics, injury pattern, comorbidities, pre- and in-hospital management, course on intensive care unit, relevant laboratory findings including data on transfusion and outcome of each individual. The inclusion criterion is admission to hospital via an emergency room with subsequent ICU/ICM care or reach the hospital with vital signs and die before admission to ICU.

The infrastructure for documentation, data management, and data analysis is provided by AUC—Academy for Trauma Surgery (AUC—Akademie der Unfallchirurgie GmbH), a company affiliated to the German Trauma Society. The scientific leadership is provided by the Committee on Emergency Medicine, Intensive Care and Trauma Management (Sektion NIS) of the German Trauma Society. The participating hospitals submit their data pseudonymised into a central database via a web-based application. Scientific data analysis is approved according to a peer-review procedure laid down in the publication guideline of TraumaRegister DGU®.

The participating hospitals are primarily located in Germany (90%), but a rising number of hospitals of other countries contribute data as well (at the moment from Austria, Belgium, China, Finland, Luxembourg, Slovenia, Switzerland, The Netherlands, and the United Arab Emirates). Participation in TraumaRegister DGU® is voluntary. For hospitals associated with TraumaNetzwerk DGU®, however, the entry of at least a basic data set is obligatory for reasons of quality assurance [17].

Patients were defined as sedated if they received analgesic, sedative or narcotic medication in the pre-hospital phase. Based on the SOFA scores a potential organ failure was determined [18]. If an organ failure of 2 organs occurred for at least 2 days, multiple organ failure was assessed. Sepsis was diagnosed according to SIRS criteria and the existence of a positive blood culture [20]. Acidosis was defined at a base excess (BE) < -6. Coagulopathy was determined based on partial thromboplastin time (PTT) or INR value, PTT ≥ 40 s or INR ≥ 1.4 [23]. Outcome of the patients was graduated into 5 categories: good recovery, minor disability (patient independent), severe disability (patient awake but needs support), persistent vegetative state and dead.

The present work is in accordance with the publication guideline of the TraumaRegister DGU® and is registered under the TR-DGU Project-ID 2017-001. The review process of the TraumaRegister DGU® was accomplished. The parameter "alcohol" was included in the data set in 2015. Depending on age, driving abnormalities and for novice drivers there are different alcohol limits for motorists in Germany. However, for most German motorists the 0.5‰ limit is valid [19]. This threshold was also set in our study as the lower limit for the group of patients with BAL + in the matched-pair analysis.

The standard data set of the TR-DGU was used for a retrospective analysis. All patients of the German hospitals of the TR-DGU from the years 2015 and 2016 were included with a MAIS 3 + and documented alcohol values [21]. Individual alcohol levels > 5‰ were excluded due to the implausibility of the measurements. Relocated patients (initial shock-room data not available) as well as patients relocated within 48 h (no outcome available) were also excluded.

The matched-pair analysis was performed between patients with an alcohol level ≥ 0.5‰ and patients with a measured alcohol level of 0‰. Matching was performed according to gender, age (10-year intervals), relevant injuries (Abbreviated Injury Scale (AIS) ≥ 3) in four body regions (head, thorax, abdomen, pelvis/extremities) and a similar survival probability: 5% probability intervals according to the Revised Injury Severity Classification Score, version II (RISC II) [22].

## Statistical analysis

Statistical analysis was performed using SPSS (version 23, IBM, Armonk, USA). The matching was used as a method for the production of comparable sub-collectives; the evaluation was done classically with independent test procedures, because of the comparison of different persons in two groups. The associated slight restriction of power is more than balanced by the high number of cases. Differences of about 5–6% or one-fifth of a standard deviation can, thus, be statistically secured. Fisher's exact test was used for categorical variables, the Mann–Whitney *U*-test was used for continuous variable. The level of significance was assumed to be 5% ($p < 0.05$).

## Results

In total, 27,997 patients were documented in 2015 and 2016 in German clinics with a MAIS score of 3 or greater. A total of 14,529 patients accounted for the quality management data set, which contains no alcohol value. Of the remaining 13,468 patients, 2225 were excluded because these patients were attached or relocated. There were no data on alcohol levels in 6304 patients, so 4939 patients were available for final evaluation with documented alcohol values. A total of 3592 patients exhibited no BAL, 856 patients had alcohol levels between 0.1 and 0.4‰, and 491 patients showed a BAL ≥ 0.5‰ (Fig. 1). After matching, 834 patients were enrolled, with 417 patients in the BAL − group and in the BAL + group (alcohol value ≥ 0.5‰).

An overview of the basic data of the groups after the matched pair analysis is shown in Table 1. Per group, 350 patients were male with a mean age of 45 years. The BAL − group had a mean ISS of 20.2 and was sober (0‰). The BAL + group showed a mean ISS of 19.2 and a median alcohol level of 1.82‰ (interquartile range: 1.29–2.52, Table 1).

With regard to the mechanism of injury, there were significant differences between the two groups; in the BAL + group pedestrians were much more frequently involved in a traffic accident or suffered under low energy falls (< 3 m) compared with the BAL − group (Table 2, Fig. 2). Furthermore, there were more penetrating injuries in the BAL + group (BAL − group: 17 (4.2%) vs. BAL + group: 32 (7.9%), $p = 0.025$, Table 2). In contrast, sober patients were more likely to be involved in car and motorcycle accidents (Table 2).

Preclinically, there was no significant difference between the groups in terms of intubation rate, catecholamine demand and number of unconscious patients (Glasgow Come Scale (GCS) ≤ 8). BAL + patients were significantly less sedated (BAL − group: 273 (66.7%) vs. BAL + group: 232 (56.2%), $p = 0.002$, Table 2), significantly less frequently transported by rescue helicopter (BAL − group: 138 (33.7%) vs. BAL + group: 69 (17.0%), $p < 0.001$, Table 2) and significantly more frequently admitted to a clinic during the night (BAL − group: 76 (18.4%) vs. BAL + group: 247 (60.2%), $p < 0.001$, Table 2) compared to BAL − patients.

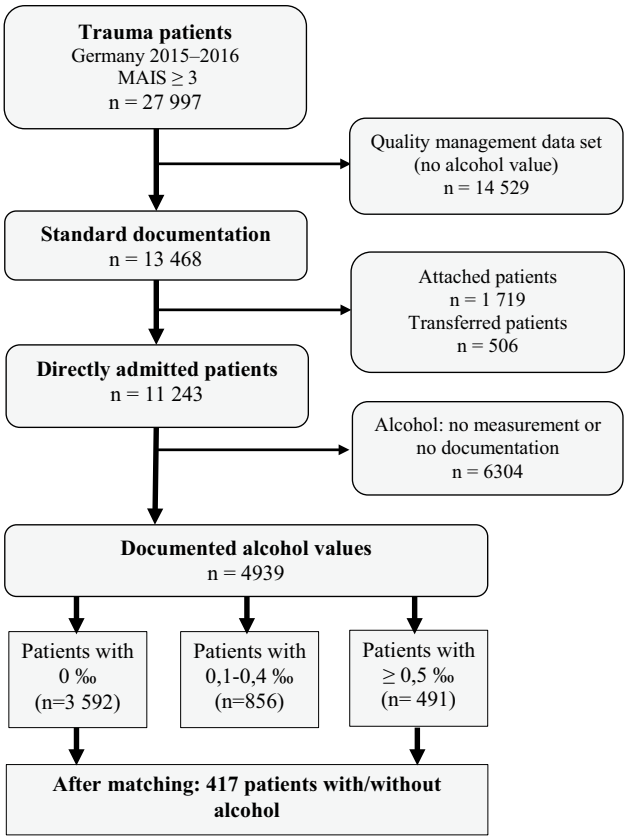


**Fig. 1** diagram about included and excluded patients. *MAIS* Maximum Abbreviated Injury Scale

There was no significant difference between the groups in terms of performing a whole-body computed tomography (CT) scan, the need for haemostasis therapy, or haemoglobin values in the emergency care and diagnosis (Table 3). BAL + patients were significantly more likely to be hypotensive pre-clinical or in the emergency department (blood pressure ≤ 90 mmHg) (BAL − group: 42 (10.3%) vs. BAL + group: 61 (15.2%), $p = 0.045$, Table 2).

During the in-hospital treatment, BAL + patients manifested significantly lower base excess (BE) values compared to BAL − patients (BAL − group: − 1.12 ± 3.52 mmol/l vs. BAL + group: − 3.13 ± 3, 67 mmol/l, $p < 0.001$, Table 3). In addition, BAL + patients were significantly more frequently acidic (BAL − group: 24 (6.1%) vs. BAL + group: 61 (17.2%), $p < 0.001$, Table 4). There was no correlation between blood alcohol values and BE in the BAL group (correlation coefficient 0.04, Fig. 3).

**Table 1** Summary of patient characteristics

| Patient characteristics | BAL − | BAL+ | All patients ($n=834$) | $p$ value (BAL − vs. BAL+) |
|---|---|---|---|---|
| Sex male ($n$, %) | 350 (83.9%) | 350 (83.9%) | 700 (83.9%) | 1.000 |
| Sex female ($n$, %) | 67 (16.1%) | 67 (16.1%) | 134 (16.1%) | 1.000 |
| Age (years, SD) | 45.0 ± 18.5 | 45.1 ± 18.0 | 45.05 ± 18.2 | 0.86 |
| BAC (‰, median, IQR) | 0 (0 – 0) | 1.82 (1.29 – 2.52) | – | – |
| ISS (SD) | 20.2 ± 9.6 | 19.2 ± 9.3 | 19.7 ± 9.5 | 0.096 |
| AIS ≥ 3 | | | | |
| Head ($n$, %) | 235 (56.4%) | 235 (56.4%) | 470 (56.4%) | 1.000 |
| Chest ($n$, %) | 162 (38.8%) | 162 (38.8%) | 324 (38.8%) | 1.000 |
| Abdomen ($n$, %) | 37 (8.9%) | 37 (8.9%) | 74 (8.9%) | 1.000 |
| Extremity ($n$, %) | 84 (20.1%) | 84 (20.1%) | 168 (20.1%) | 1.000 |
| RISC II-Score (mean) | 6.8% | 6.8% | 6.8% | 0.161 |

Data are presented as mean ± standard deviation (SD) or n (%); Some data were not available in all cases

*BAL − group* negative blood alcohol level, *BAL + group* blood alcohol level ≥ 0.5‰, *AIS* Abbreviated Injury Scale, *ISS* Injury Severity Score, *IQR* interquartile range, *RISC II-Score* Revised Injury Severity Classification Scores, Version II

**Table 2** Summary preclinical parameters

| Parameter | BAL − | BAL+ | All patients ($n=834$) | $p$ value (BAL − vs. BAL+) |
|---|---|---|---|---|
| *Preclinical phase* | | | | |
| Blunt trauma ($n$, %) | 391 (95.8%) | 372 (92.6%) | 763 (94.0%) | $p=0.025$ |
| Penetrating trauma ($n$, %) | 17 (4.2%) | 32 (7.9%) | 49 (6.0%) | $p=0.025$ |
| Traffic accidents, car ($n$, %) | 99 (23.9%) | 54 (13.0%) | 153 (18.4%) | $p<0.001$ |
| Traffic accidents, motorcycle ($n$, %) | 56 (13.5%) | 30 (7.2%) | 86 (10.4%) | $p=0.003$ |
| Traffic accidents, bicycle ($n$, %) | 51 (12.3%) | 39 (9.4%) | 90 (10.8%) | $p=0.18$ |
| Traffic accidents, pedestrian ($n$, %) | 14 (3.4%) | 38 (9.2%) | 52 (6.3%) | $p=0.001$ |
| Fall > 3 m ($n$, %) | 74 (17.8%) | 62 (14.9%) | 136 (16.4%) | $p=0.26$ |
| Fall < 3 m ($n$, %) | 75 (18.1%) | 130 (31.3%) | 205 (24.7%) | $p<0.001$ |
| Intubation ($n$, %) | 134 (32.8%) | 128 (31.0%) | 262 (31.9%) | $p=0.59$ |
| Catecholamines ($n$, %) | 31 (7.6%) | 30 (7.3%) | 61 (7.4%) | $p=0.86$ |
| Sedation ($n$, %) | 273 (66,7%) | 232 (56,2%) | 505 (61.4%) | $p=0.002$ |
| Glasgow Coma Scale (SD) | 11.9 ± 4.3 | 11.2 ± 4.2 | 11.5 ± 4.3 | $p<0.001$ |
| Unconscious, GCS ≤ 8 ($n$, %) | 93 (23.2%) | 106 (27.1%) | 199 (25.1%) | $p=0.22$ |
| Transport by rescue helicopter ($n$, %) | 138 (33,7%) | 69 (17,0%) | 207 (25.4%) | $p<0.001$ |
| Clinic admission during the night | 76 (18,4%) | 247 (60,2%) | 323 (38.7%) | $p<0.001$ |

Data are presented as mean ± standard deviation (SD) or $n$ (%); $p$ value for BAL + vs. BAL − group; some data were not available in all cases

B*AL − group*: negative blood alcohol level, *BAL + group* blood alcohol level ≥ 0.5‰, *GCS* Glasgow Coma Scale

The BAL − group had an average hospital length of stay of 19.9 days, with patients spending 8.6 days in intensive care and intubation for 4.0 days. In comparison, the patients of the BAL + group stayed in the hospital for, on average, 18.9 days, in the intensive care unit for 7.8 days, and were intubated for 4.1 days (Table 4). There was no difference between the groups regarding the development of multiple organ failure, sepsis or coagulopathy, the need for haemostasis therapy on ICU or the administration of packed red blood cells (PRBC) and the outcome (Table 4). The calculated mortality prognosis according to RISC II was 6.8% for both groups. There was a total mortality of 8.2% in the BAL − group compared to a total mortality of 5.8% in the BAL + group ($p=0.48$, Table 4, Fig. 4).

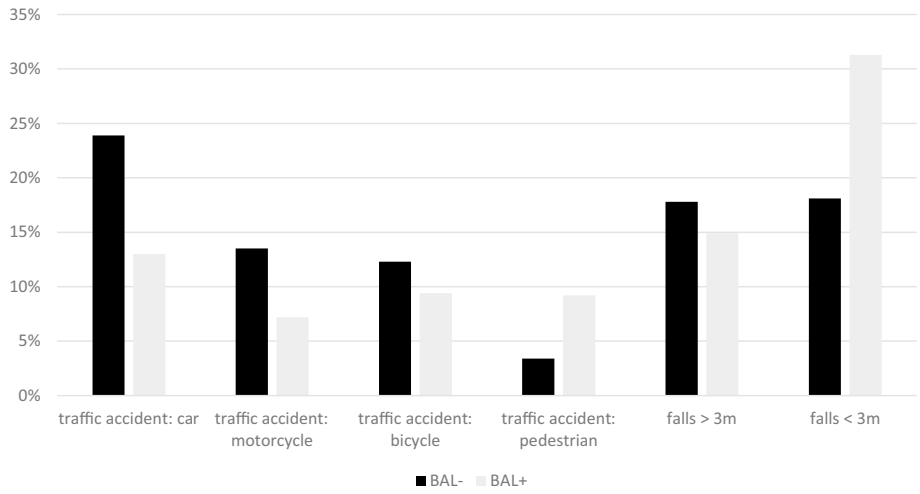


**Fig. 2** mechanism of injury. *BAL − group* negative blood alcohol level, *BAL + group* blood alcohol level ≥ 0.5‰

**Table 3** Summary emergency room parameters

| Parameter | BAL − | BAL+ | All patients ($n=834$) | $p$ value (BAL − vs. BAL+) |
|---|---|---|---|---|
| *Emergency room* | | | | |
| Whole-body CT scan ($n$, %) | 366 (88.0%) | 363 (87.7%) | 729 (87.8%) | $p=0.90$ |
| Hypotension (SBP ≤ 90 mmHg, preclinical or emergency room) ($n$, %) | 42 (10.3%) | 61 (15.2%) | 103 (12.7%) | $p=0.045$ |
| Haemostasis therapy ($n$, %) | 69 (17.8%) | 63 (15.9%) | 132 (16.8%) | $p=0.47$ |
| Quick`s value (TPT) (%, SD) | 87.9 ± 18.4 | 92.9 ± 18.8 | 90.4 ± 18.8 | $p<0.001$ |
| INR (SD) | 1.13 ± 0.36 | 1.09 ± 0.57 | 1.11 ± 0.48 | $p<0.001$ |
| Thrombocytes (cell count/ul, SD) | 222,541 ± 80,018 | 230,657 ± 79,568 | 226,609 ± 79,847 | $p=0.029$ |
| Hemoglobin (g/dl, SD) | 13.6 ± 1.8 | 13.5 ± 1.9 | 13.5 ± 1.9 | $p=0.68$ |
| Base excess (mmol/l, SD) | − 1.12 ± 3.52 | − 3.13 ± 3.67 | − 2.08 ± 3.73 | $p<0.001$ |

Data are presented as mean ± standard deviation (SD) or $n$ (%); $p$-value for BAL + vs. BAL − group; Some data were not available in all cases

*BAL − group* negative blood alcohol level, *BAL + group* blood alcohol level ≥ 0.5‰, *INR* international normalized ratio, *SBP* systolic blood pressure, *TPT* thromboplastin time

## Discussion

In the present study, the hypothesis that alcohol has no negative effect on the outcome of trauma patients (MAIS 3 +) was investigated. The majority of the BAL + patients were male, and the BAL + group had a median alcohol level of 1.82‰. Significant differences were found between both groups in terms of the mechanism of injury, sedation rate, type of transport and the time of admission to a hospital. Furthermore, BAL + patients were significantly more often acidic. The established hypothesis has been confirmed, as no difference were found between both groups in terms of outcome.

The clear trend in our study of penetrating injuries on the one hand and injuries resulting from low energy falls (< 3 m) on the other in the BAL + group is also found in the literature. Afshar et al. showed that patients with a moderate blood alcohol level are at an increased risk for penetrating injuries, whereas with increasing blood levels and especially with very high blood alcohol levels, blunt injury from falls occurs [24, 25].

The increased admission of BAL + patients at night is explained by the increased social alcohol consumption in the evening and night hours. This also explains the significantly lower number of transports by rescue helicopter in the BAL + group, as rescue helicopters are usually used in the context of primary rescue operations from sunrise to sunset. To what extent the injuries, especially the penetrating, in the BAL + group occurred more in the urban environment, in which the use of a rescue helicopter is often not useful, cannot be found from the data and should be investigated separately.

**Table 4** Summary hospital stay parameters

| Parameter | BAL − | BAL + | All patients ($n=834$) | $p$ value (BAL − vs. BAL+) |
|---|---|---|---|---|
| *Hospital stay* | | | | |
| Length of hospital stay (days, SD) | 19.9 ± 23.4 | 18.9 ± 18.2 | 19.4 ± 21.0 | $p=0.74$ |
| ICU (days, SD) | 8.6 ± 12.2 | 7.8 ± 10.6 | 8.18 ± 11.4 | $p=0.63$ |
| Mechanical ventilation (days, SD) | 4.0 ± 8.3 | 4.1 ± 8.8 | 4.0 ± 8.6 | $p=0.52$ |
| Mortality (*n*, %) | 34 (8.2%) | 24 (5.8%) | 58 (7.0%) | $p=0.22$ |
| 24 h mortality (*n*, %) | 11 (2.6%) | 7 (1.7%) | 18 (2.2%) | $p=0.48$ |
| Multi-organ failure (*n*, %) | 76 (19.6%) | 79 (20.3%) | 155 (19.9%) | $p=0.83$ |
| Sepsis (*n*, %) | 20 (5.3%) | 29 (7.7%) | 49 (6.5%) | $p=0.19$ |
| Acidosis (*n*, %) | 24 (6.1%) | 61 (17.2%) | 85 (11.4%) | $p<0.001$ |
| PRBC transfusion (*n*, %) | 29 (7.0%) | 26 (6.3%) | 55 (6.6%) | $p=0.68$ |
| Haemostasis therapy on ICU (*n*, %) | 55 (14.7%) | 45 (12.0%) | 100 (13.4%) | $p=0.28$ |
| Coagulopathy (*n*, %) | 34 (8.2%) | 36 (8.7%) | 70 (8.4%) | $p=0.80$ |
| *Outcome (survivor only)* | | | | |
| Good recovery (*n*, %) | 239 (62.7%) | 252 (64.5%) | 491 (63.6%) | $p = 0.81$ |
| Minor disability (*n*, %) | 92 (24.1%) | 89 (22.2%) | 181 (23.4%) | $p = 0.81$ |
| Severe disability (*n*, %) | 41 (10.8%) | 44 (11.3%) | 85 (11.0%) | $p = 0.81$ |
| Persistent vegetative state (*n*, %) | 9 (2.4%) | 6 (1.5%) | 15 (1.9%) | $p = 0.81$ |

Data are presented as mean ± standard deviation (SD) or *n* (%); *p*-value for BAL + vs. BAL − group; Some data were not available in all cases

*BAL − group* negative blood alcohol level, *BAL + group* blood alcohol level ≥ 0.5‰, *PRBC* Packed Red Blood Cells

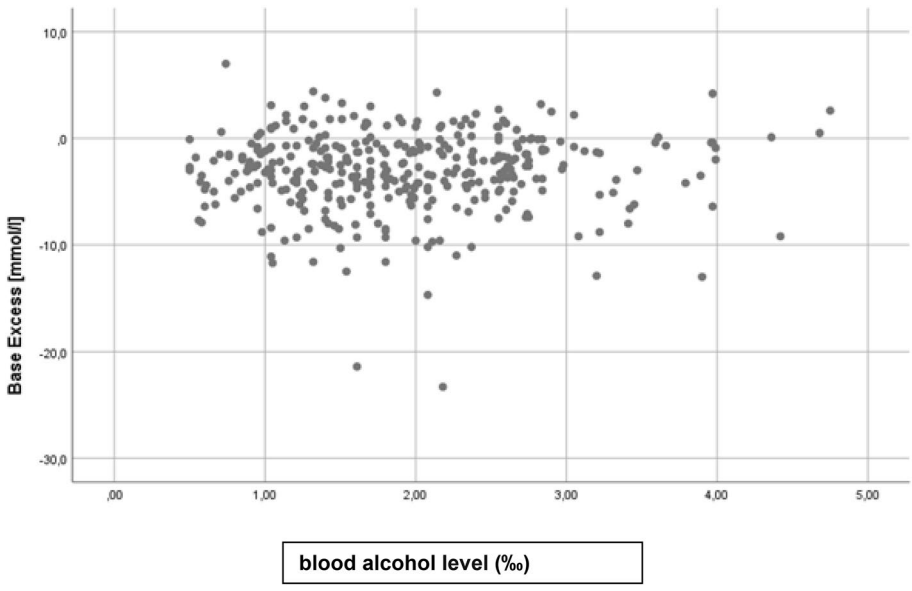


**Fig. 3** correlation between BAL and BE values. Analysis in the BAL + group; time point: emergency room; correlation coefficient: 0.04. BAL + : blood alcohol level ≥ 0.5‰

The low blood pressure in the BAL + group is consistent with clinical and in vivo studies and is explained by the haemodynamic influence of alcohol [7, 8, 26–28]. Since preclinical blood loss is not registered, the higher number of penetrating lesions in the BAL + group may also be a factor in the development of hypotension. However, no difference in the preclinical use of catecholamines has been shown, suggesting an alcohol-related hypotension.

Noticeable is the significantly lower number of sedated patients in the BAL + group with comparable preclinical GCS value, comparable injury severity and comparable preclinical intubation rate between both groups. Here we can only speculate about the reasons. On the one hand, a preclinical underestimation of the severity of injury in BAL + patients is much easier than in BAL − patients;

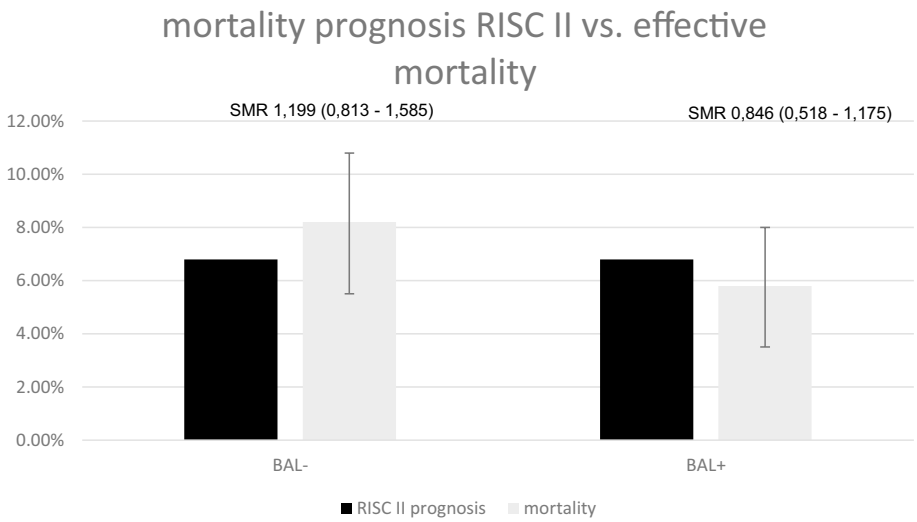


**Fig. 4** mortality prognosis RISC II vs. effective mortality. BAL − group: negative blood alcohol level; BAL + group: blood alcohol level ≥ 0.5‰; RISC II BAL − group 6.8% vs. effective mortality 8.2% (95% CI 5.5–10.8); RISC II BAL + group 6.8% vs. effective mortality 5.8% (95% CI 3.5–8.0); CI: confidence interval; SMR, standardized mortality ratio; RISC II Score, Revised Injury Severity Classification Scores, version II

on the other hand, concern about drug interactions with alcohol may contribute to the lower sedation rate.

Patients of the BAL + group were significantly more likely to be in an acidotic metabolic state compared to the BAL − group. However, when interpreting the data, it must also be considered that there were more penetrating injuries in the BAL + group. So, the preclinical rescue strategy, e.g. permissive hypotension, may have contributed to the development of acidosis. It is known that alcohol exposure can cause metabolic and respiratory acidosis, even without a trauma [29–31]. In a previous in vivo work, we were able to show the development of a combined, respiratory and metabolic acidosis by alcohol, with a further aggravating of the acidosis through a subsequent trauma [8]. Furthermore, in our current study the BAL + group showed slightly, but significantly, more negative BE values compared to the BAL − group. In addition, in the work of Dunham et al. and Davis et al, lower BE values were found for BAL + patients [32, 33]. In the current study, there was no difference between both groups regarding mortality, outcome or in-hospital complication rates. This is interesting, as the BE parameter is used as an indicator of trauma severity, mortality, shock severity and volume requirement [34–38]. Among others, through the study of Abt et al, the relation between the BE value as a predictor of mortality in the context of trauma patients was demonstrated [34]. In the current literature, to what extent the relationship between more negative BE and higher mortality for BAL − patients also applies to BAL + patients is unclear. In the study of Gustafson et al. the established relationship between BE and outcome in BAL − patients was not confirmed for BAL + patients [39]. In contrast, Zehtabchi et al., Dunne et al. and Ibrahim et al. conclude that the use of BE is not negatively affected by alcohol [38, 40, 41]. In our study, we found no difference regarding in-hospital complications or mortality rates between both groups. The mortality presumption calculated according to the RISC II score is slightly underrun through the BAL + group and slightly overrun by the BAL − group. However, there was no statistical difference; in any case, the BAL + group does not show a worse outcome. When interpreting our results, the relatively small difference of the BE value between groups should also be considered. In the work of Gustafson et al, the BE threshold for prediction between survivors and non-survivors was − 6.95 mmol/L [39], while, in the work of Davis et al, BE was a significant indicator of serious injury from a BE threshold ≤ -6 mmol/L [33]. In contrast to the studies above, our study was not designed to confirm the value of BE as a predictor of mortality. However, our data clearly demonstrate that alcohol exposure in trauma patients can be a major factor in the development of acidotic metabolic disorder without impacting on the outcome.

Consistent with our findings, in the study by Zeckey et al. no difference in the clinical course was found between patients with ISS ≥ 16 with and without alcohol [42]. In the BAL + group with an ISS of 29.7, there was no significant difference regarding sepsis, multiple organ failure or mortality compared to the BAL − group with an ISS of 28.7 [42].

Friedman showed a reduction in overall complications in the BAL + group, cardiac complications were decreased by 23.5% and renal complications were decreased by 30.0%, but there was an increased risk of aspiration pneumonia and pancreatitis [43]. In contrast, in a matched-pair analysis according to age, gender, trauma mechanism, ISS, and AIS between BAL + and BAL − patients, increased incidence of in-hospital cardiac arrest and increased mortality for BAL + patients were found [44]. In addition, in the study of Stübig et al. an increased preclinical mortality was shown for BAL + patients [45].

The present studies regarding the BE and the outcome differ clearly in the study design: retrospective/

prospective, hospital care level, blood alcohol level, blunt/penetrating injuries, severity of injury, so that the comparability between the studies is difficult. Numerous other studies on positive and negative alcohol effects can also be found and prove the overall need for further studies. A main focus should be placed on the underlying mechanism. Amongst others, possible explanation for the effect of alcohol exposure is the influence of alcohol on the inflammation system, the coagulation or the blunting of the adrenergic response [15, 16, 46]. Regarding traumatic brain injuries, the alcohol caused attenuation of the catecholamine surge may be a possible explanation for the different study results of BAL + and BAL − patients [47, 48].

The limitations of retrospective evaluations are also present in this work. In the study planning, we decided for a matched-pair analysis to detect the influence of alcohol under otherwise comparable conditions. However, it should be noted that the BAL + group has a wide range of alcohol values and, thus, subgroups with positive and negative effects are possible. The advantage of register evaluations is, among other things, the high number of cases and the routine recording of all patients. Regarding the relatively new parameter "alcohol", it has been shown that no value was measured or documented for 6304 data records. Experience has shown that it always takes a certain time period for new parameters until they are inserted in a high percentage. Finally, it remains unclear whether BAL was only determined when there was a suspicion of intoxication, and most of the unfilled or unmeasured patients were sober. As well, a combined exposure of alcohol and other drugs were not investigated. In this respect, a bias effect cannot be excluded. A routine measurement of BAL is recommended for all patients with severe trauma and is carried out in our clinic. Here, there is often a clear discrepancy between the clinical assessment and the measured value, so that a measured BAL is helpful in the assessment of patients, i.a. the subsumption of the vigilance or pathological blood results. As a limitation of the study, it should be mentioned that the evaluation of the recorded BAL cannot show a dynamic effect. In addition, a distinction between acute and chronic alcohol consumption based on the data is not possible and preclinically deceased patients are omitted from the statistics, as these are not covered by the TraumaRegister DGU®. Furthermore, it is not possible in register evaluations to respond to individual cases in more detail. In addition, limited by the inclusion criteria, admission to ICU, it is possible that some individual patients could not be included in the study.

In summary, our data demonstrate that the presence of alcohol in trauma patients can cause an acidotic metabolic state. However, this has no influence on the complication or mortality rate in the present study. The different study results of the literature underline the need for further studies.

## Compliance with ethical standards

**Conflict of interest** Prof. Lefering is a member and advisor of Emergency Medicine, Intensive Care and Trauma Management (Sektion NIS) of the German Trauma Society (DGU). His institute receives financial support from the AUC—Academy of Trauma Surgery GmbH, the operator of the TR-DGU, as part of a cooperation agreement which also includes the statistical support of scientific publications. The other authors declare that there is no conflict of interest.

## References


1. Greenspan L, McLellan BA, Greig H. Abbreviated injury scale and injury severity score: a scoring chart. J Trauma. 1985;25:60–4.
2. Sektion Notfall- & Intensivmedizin, Schwerverletztenversorgung (NIS) der Deutschen und AUC - Akademie der Unfallchirurgie GmbH. TR-DGU-Jahresbericht_2017.
3. Berry C, Ley EJ, Margulies DR, Mirocha J, Bukur M, Malinoski D, Salim A. Correlating the blood alcohol concentration with outcome after traumatic brain injury: too much is not a bad thing. Am Surg. 2011;77:1416–9.
4. Lin H-L, Lin T-Y, Soo K-M, Chen C-W, Kuo L-C, Lin Y-K, et al. The effect of alcohol intoxication on mortality of blunt head injury. Biomed Res Int. 2014;2014:619231. https://doi.org/10.1155/2014/619231.
5. Raj R, Skrifvars MB, Kivisaari R, Hernesniemi J, Lappalainen J, Siironen J. Acute alcohol intoxication and long-term outcome in patients with traumatic brain injury. J Neurotrauma. 2015;32:95–100. https://doi.org/10.1089/neu.2014.3488.
6. Albrecht JS, Afshar M, Stein DM, Smith GS. Alcohol is not protective against mortality following traumatic brain injury. Am J Epidemiol. 2017. https://doi.org/10.1093/aje/kwx254.
7. Wagner N, Akbarpour A, Mors K, Voth M, Stormann P, Auner B, et al. Alcohol intoxication reduces systemic interleukin-6 levels and leukocyte counts after severe TBI compared with not intoxicated TBI patients. Shock. 2016;46:261–9. https://doi.org/10.1097/SHK.0000000000000620.
8. Wagner N, Franz N, Dieteren S, Perl M, Mörs K, Marzi I, Relja B. Acute alcohol binge deteriorates metabolic and respiratory compensation capability after blunt chest trauma followed by hemorrhagic shock-A new research model. Alcohol Clin Exp Res. 2017;41:1559–67. https://doi.org/10.1111/acer.13446.
9. Relja B, Wilhelm K, Wang M, Henrich D, Marzi I, Lehnert M. Acute ethanol gavage attenuates hemorrhage/resuscitation-induced hepatic oxidative stress in rats. Oxid Med Cell Longev. 2012;2012:983427. https://doi.org/10.1155/2012/983427.
10. Relja B, Menke J, Wagner N, Auner B, Voth M, Nau C, Marzi I. Effects of positive blood alcohol concentration on outcome and systemic interleukin-6 in major trauma patients. Injury. 2016;47:640–5. https://doi.org/10.1016/j.injury.2016.01.016.
11. Mörs K, Hörauf J-A, Kany S, Wagner N, Sturm R, Woschek M, et al. Ethanol decreases inflammatory response in human lung epithelial cells by inhibiting the canonical NF-kB-pathway. Cell Physiol Biochem. 2017;43:17–30. https://doi.org/10.1159/000480313.
12. Nau C, Wutzler S, Dörr H, Lehnert M, Lefering R, Laurer H, et al. Liver cirrhosis but not alcohol abuse is associated with impaired outcome in trauma patients: a retrospective, multicentre study. Injury. 2013;44:661–6. https://doi.org/10.1016/j.injury.2012.06.013.
13. Pandit V, Patel N, Rhee P, Kulvatunyou N, Aziz H, Green DJ, et al. Effect of alcohol in traumatic brain injury: is it

really protective? J Surg Res. 2014;190:634–9. https://doi.org/10.1016/j.jss.2014.04.039.
14. Teng SX, Katz PS, Maxi JK, Mayeux JP, Gilpin NW, Molina PE. Alcohol exposure after mild focal traumatic brain injury impairs neurological recovery and exacerbates localized neuroinflammation. Brain Behav Immun. 2015;45:145–56. https://doi.org/10.1016/j.bbi.2014.11.006.
15. Relja B, Hohn C, Bormann F, Seyboth K, Henrich D, Marzi I, Lehnert M. Acute alcohol intoxication reduces mortality, inflammatory responses and hepatic injury after haemorrhage and resuscitation in vivo. Br J Pharmacol. 2012;165:1188–99. https://doi.org/10.1111/j.1476-5381.2011.01595.x.
16. Lustenberger T, Inaba K, Barmparas G, Talving P, Plurad D, Lam L, et al. Ethanol intoxication is associated with a lower incidence of admission coagulopathy in severe traumatic brain injury patients. J Neurotrauma. 2011;28:1699–706. https://doi.org/10.1089/neu.2011.1866.
17. Akademie der Unfallchirurgie GmbH. Manuskript—AUC. http://www.traumaregister-dgu.de/de/forschung/manuskript.html. Accessed 23 Mar 2018.
18. Vincent JL, Moreno R, Takala J, Willatts S, Mendonça A de, Bruining H, et al. The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. On behalf of the Working Group on Sepsis-Related Problems of the European Society of Intensive Care Medicine. Intensive Care Med. 1996;22:707–10. https://doi.org/10.1007/bf01709751.
19. § 24a StVG - Einzelnorm. 15.03.2018. https://www.gesetze-im-internet.de/stvg/__24a.html. Accessed 21 Mar 2018.
20. Calandra T, Cohen J. The international sepsis forum consensus conference on definitions of infection in the intensive care unit. Crit Care Med. 2005;33:1538–48. https://doi.org/10.1097/01.ccm.0000168253.91200.83.
21. Akademie der Unfallchirurgie GmbH. Teilnahme - AUC. http://www.traumaregister-dgu.de/de/registerstruktur/teilnahme.html. Accessed 23 Mar 2018.
22. Lefering R. Ein neuer Prognose-Score im TraumaRegister DGU® (RISC II). Orthopädie und Unfallchirurgie - Mitteilungen und Nachrichten. 2015;04:80–1. https://doi.org/10.1055/s-0041-100418.
23. Pape H-C, Lefering R, Butcher N, Peitzman A, Leenen L, Marzi I, et al. The definition of polytrauma revisited: an international consensus process and proposal of the new 'Berlin definition'. J Trauma Acute Care Surg. 2014;77:780–6. https://doi.org/10.1097/TA.0000000000000453.
24. Afshar M, Netzer G, Murthi S, Smith GS. Alcohol exposure, injury, and death in trauma patients. J Trauma Acute Care Surg. 2015;79:643–8. https://doi.org/10.1097/TA.0000000000000825.
25. Afshar M, Netzer G, Salisbury-Afshar E, Murthi S, Smith GS. Injured patients with very high blood alcohol concentrations. Injury. 2016;47:83–8. https://doi.org/10.1016/j.injury.2015.10.063.
26. Molina PE, Zambell KL, Norenberg K, Eason J, Phelan H, Zhang P, et al. Consequences of alcohol-induced early dysregulation of responses to trauma/hemorrhage. Alcohol. 2004;33:217–27. https://doi.org/10.1016/j.alcohol.2004.07.002.
27. Bilello J, McCray V, Davis J, Jackson L, Danos LA. Acute ethanol intoxication and the trauma patient: hemodynamic pitfalls. World J Surg. 2011;35:2149–53. https://doi.org/10.1007/s00268-011-1191-7.
28. Yoonhee C, Jung K, Eo E, Lee D, Kim J, Shin D, et al. The relationship between alcohol consumption and injury in ED trauma patients. Am J Emerg Med. 2009;27:956–60. https://doi.org/10.1016/j.ajem.2008.07.035.
29. Halperin ML, Hammeke M, Josse RG, Jungas RL. Metabolic acidosis in the alcoholic: a pathophysiologic approach. Metab Clin Exp. 1983;32:308–15.
30. Lamminpaa A, Vilska J. Acid-base balance in alcohol users seen in an emergency room. Vet Hum Toxicol. 1991;33:482–5.
31. Sahn SA, Lakshminarayan S, Pierson DJ, Weil JV. Effect of ethanol on the ventilatory responses to oxygen and carbon dioxide in man. Clin Sci Mol Med. 1975;49:33–8.
32. Dunham CM, Watson LA, Cooper C. Base deficit level indicating major injury is increased with ethanol. J Emerg Med. 2000;18:165–71.
33. Davis JW, Kaups KL, Parks SN. Effect of alcohol on the utility of base deficit in trauma. J Trauma. 1997;43:507–10.
34. Abt R, Lustenberger T, Stover JF, Benninger E, Lenzlinger PM, Stocker R, Keel M. Base excess determined within one hour of admission predicts mortality in patients with severe pelvic fractures and severe hemorrhagic shock. Eur J Trauma Emerg Surg. 2009;35:429. https://doi.org/10.1007/s00068-009-8245-7.
35. Mutschler M, Nienaber U, Brockamp T, Wafaisade A, Fabian T, Paffrath T, et al. Renaissance of base deficit for the initial assessment of trauma patients: a base deficit-based classification for hypovolemic shock developed on data from 16,305 patients derived from the TraumaRegister DGU®. Crit Care. 2013;17:R42. https://doi.org/10.1186/cc12555.
36. Rixen D, Raum M, Bouillon B, Lefering R, Neugebauer E. Base deficit development and its prognostic significance in posttrauma critical illness: an analysis by the trauma registry of the Deutsche Gesellschaft für unfallchirurgie. Shock. 2001;15:83–9.
37. Davis JW, Shackford SR, Mackersie RC, Hoyt DB. Base deficit as a guide to volume resuscitation. J Trauma. 1988;28:1464–7.
38. Ibrahim I, Chor WP, Chue KM, Tan CS, Tan HL, Siddiqui FJ, Hartman M. Is arterial base deficit still a useful prognostic marker in trauma? A systematic review. Am J Emerg Med. 2016;34:626–35. https://doi.org/10.1016/j.ajem.2015.12.012.
39. Gustafson ML, Hollosi S, Chumbe JT, Samanta D, Modak A, Bethea A. The effect of ethanol on lactate and base deficit as predictors of morbidity and mortality in trauma. Am J Emerg Med. 2015;33:607–13. https://doi.org/10.1016/j.ajem.2015.01.030.
40. Zehtabchi S, Baron BJ, Sinert R, Yadav K, Lucchesi M. Ethanol and illicit drugs do not affect the diagnostic utility of base deficit and lactate in differentiating minor from major injury in trauma patients. Acad Emerg Med. 2004;11:1014–20. https://doi.org/10.1197/j.aem.2004.06.008.
41. Dunne JR, Tracy JK, Scalea TM, Napolitano LM. Lactate and base deficit in trauma: does alcohol or drug use impair their predictive accuracy? J Trauma. 2005;58:959–66.
42. Zeckey C, Dannecker S, Hildebrand F, Mommsen P, Scherer R, Probst C, et al. Alcohol and multiple trauma: is there an influence on the outcome? Alcohol. 2011;45:245–51. https://doi.org/10.1016/j.alcohol.2010.08.004.
43. Friedman LS. Complications associated with blood alcohol concentration following injury. Alcohol. 2014;48:391–400. https://doi.org/10.1016/j.alcohol.2014.01.008.
44. Hadjizacharia P, O'Keeffe T, Plurad DS, Green DJ, Brown CVR, Chan LS, et al. Alcohol exposure and outcomes in trauma patients. Eur J Trauma Emerg Surg. 2011;37:169–75. https://doi.org/10.1007/s00068-010-0038-5.
45. Stübig T, Petri M, Zeckey C, Brand S, Müller C, Otte D, et al. Alcohol intoxication in road traffic accidents leads to higher impact speed difference, higher ISS and MAIS, and higher preclinical mortality. Alcohol. 2012;46:681–6. https://doi.org/10.1016/j.alcohol.2012.07.002.
46. Opreanu RC, Kuhn D, Basson MD. Influence of alcohol on mortality in traumatic brain injury. J Am Coll Surg. 2010;210:997–1007. https://doi.org/10.1016/j.jamcollsurg.2010.01.036.

47. Woolf PD, Cox C, McDonald JV, Kelly M, Nichols D, Hamill RW, Feliciano DV. Effects of intoxication on the catecholamine response to multisystem injury. J Trauma. 1991;31:1271–5. https://doi.org/10.1097/00005373-199109000-00012 (**discussion 1275-6**).

48. Woolf PD, Cox C, Kelly M, McDonald JV, Hamill RW. Alcohol intoxication blunts sympatho-adrenal activation following brain injury. Alcohol Clin Exp Res. 1990;14:205–9. https://doi.org/10.1111/j.1530-0277.1990.tb00473.x.