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Influence of alcohol consumption on blood coagulation in ROTEM - an in-vivo study

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Influence of alcohol consumption on blood coagulation in ROTEM - an in-vivo study

Running title: Influence of alcohol on coagulation
Abstract

Background: Twenty-five to 85% of trauma patients are under alcohol influence in addition to experiencing injury-related coagulation impairment. The viscoelastic point-of-care test (TEG, ROTEM) has become popular for a rapid assessment of haemostasis and therapeutic decision-making, not only in this setting. Alcohol affects these tests in-vitro, the specific in-vivo effect is unclear. Therefore, we evaluated effects of alcohol ingestion on ROTEM parameters at our department’s Christmas party.

Methods: Twenty volunteers gave informed consent to drinking red wine, whisky or vodka to a target blood alcohol concentration of 1 ‰ within one hour, calculated with the Widmark formula. Blood samples were collected before drinking, at a breath alcohol concentration of 0.5 ‰, and 1.0 ‰, but no later than one hour. After each blood collection, ExTEM and FibTEM tests were performed directly "at the bedside".

Results: All of our participants had a blood alcohol concentration (BAC) of 0.00 ‰ at the beginning. The mean BAC at the second and third collection was 0.48 ‰ and 0.76 ‰, respectively. There were no significant differences in the ExTEM parameters. FibTEM measurements showed a significant difference at the A10 value (13.0 vs. 14.0 mm, p = 0.014) and a trend at the maximum amplitude (MCF 13.7 vs. 16.2 mm, p = 0.075). We saw no significant differences in fibrinolysis parameter and also no hyperfibrinolysis in our ROTEM measurements.

Conclusions: Ethanol ingestion can impair early fibrin polymerization. Results might be of special relevance in trauma and support routine application of ROTEM/TEG in such cases.

Keywords: Blood coagulation test; Ethanol toxicity; Point-of-care testing.
Introduction

Trauma is notoriously associated with coagulopathy. Primarily, haemostatic impairment can be caused by trauma itself as described by Hess and Brohi under the term "trauma-induced coagulopathy" (TIC) [1]. However, 25–85% of patients admitted to trauma centres are additionally intoxicated. In particular, vehicle accidents are often associated with alcohol consumption [2-5]. Since alcohol has been shown to be able to alter coagulation, this might have far-reaching implications for trauma patients [6-8].

Previous studies showed multiple alterations in platelet function and prolonged bleeding time [9], inhibition of platelet adhesion to fibrinogen [10], impairment of fibrinolysis [11-13], slower rates of fibrin formation and fibrin cross-linking [3], as well as reduced fibrinogen and fibrin functionality [10, 13]. Other studies found no effect on standard coagulation measures and no haemorrhagic complications [14]. Nonetheless, exact in-vivo effects of ethanol on haemostasis and temporal correlations are still poorly understood.

While the prognostic relevance of ethanol levels in trauma is still unclear [15, 16], the strong correlation between compromised blood coagulation and a worse outcome after trauma indicate possible effects of ethanol consumption on clinical outcome [17, 18]. In fact, trauma mortality increases with the level of blood ethanol [19, 20] and is associated with an increased blood loss resulting in a need for volume resuscitation and red blood cell transfusion [21]. Consequently, precise knowledge and specific treatment of haemostatic impairment caused by ethanol seems to be crucial.

Conventional coagulation tests are restricted to certain stages and components of the clotting process and neglect clot characteristics and possible hyperfibrinolysis. Hence, many trauma centres routinely apply viscoelastic point of care tests such as rotational thrombelastometry (ROTEM) for a
more detailed haemostatic evaluation, as it reliably differentiates platelet or fibrinogen dysfunctions and moreover detects hyperfibrinolysis.

In a ROTEM-based *in-vitro* study, Engström et. al. demonstrated a prolonged clot formation time (CFT) of 34% at an ethanol concentration of 1 ‰. At an ethanol concentration of 4 ‰, they found a prolonged clot formation time of about 118%, and the fibrinolysis was significantly reduced [11].

We therefore examined possible *in-vivo* haemostatic effects of ethanol through rotational thrombelastometry (ROTEM) in non-traumatized healthy volunteers.
Materials and Methods

This study was reviewed and approved by the local ethics committee at Hannover Medical (No. 2066-2013).

Participants

We used the results from Engström et. al. to calculate the required sample size. Based on the change in clot formation time (CFT) of about 34% at an ethanol concentration of 1 ‰, a power of 80% and a significance level of 0.05, 19 patients were required for sufficient evaluation.

All participants were employees of the “Department of Anaesthesiology and Intensive Care Medicine” at Hannover Medical School, volunteers and gave informed consent at least one day prior to our study. The participants were physically and mentally healthy at the time of the study and had no alcohol for seven days and no other oral ingestion for a minimum of six hours. Furthermore, all participants had no intake of any long-term medication or any other kind of medication for seven days. The study took place at the Christmas party of the department; our point of care setup is shown in figure 1. The study outline is depicted in figure 2.

Calculated alcohol intake

Target blood alcohol concentration was 1 ‰. Individually required alcohol amounts were calculated according to the Widmark formula [22]. For a study period of one hour, we calculated alcohol depletion in g: "1 hour × body weight in kg / 10". The formulas "alcohol in g = (body weight in kg × 0.6 × 1.0 ‰) – 1 hour * body weight in kg / 10" and "alcohol in g = (body weight in kg × 0.7 × 1.0 ‰) - 1 hour × body weight in kg / 10" were used for female or male volunteers, respectively.
The participants had to drink calculated amounts of alcohol within one hour. Alcoholic drinks (whisk(e)y, vodka, red wine) were selected according to individual preferences; amounts were adjusted to the respective ethanol concentration.

**Blood sampling**

Three blood samples were taken from a cubital vein. Butterfly cannulas (Safety-Multifly-Set, Sarstedt, Germany) and several test tubes (S-Monovette and Citrat-Monovette, Sarstedt, Germany) were used. The first millilitre of blood drawn was discarded before each sampling.

The first sample was taken as baseline before initial ingestion. Second sampling was conducted when breath alcohol concentration was about 0.5 ‰, Third blood collection was performed when breath alcohol concentration was about 1.0 ‰, but not more than one hour after initial ingestion. We assumed a maximum blood alcohol concentration one hour after ingestion with no further increase in blood alcohol concentration over time. Finding by Mitchell et al. support our assumption [23]. Each volunteer served as his/her own control.

**Breath alcohol concentration**

The measurement of breath alcohol concentration in this study was performed with the breath alcohol testing device AlcoMed 3011 (Envitec-Wismar GmbH, Germany). The device was calibrated and serviced 14 days before research by authorized personnel. For taking a sample, the volunteer blew into the device with sufficient pressure, evenly and without interruption until a continuous tone was sounded by the AlcoMed 3011. The result was presented in ‰ BrAC (breath alcohol concentration).
Samples were taken after the volunteer had abstained from smoking for at least five minutes and rinsed his/her mouth with water. The volunteers were instructed to breathe normally for at least two minutes before sampling. Mouthpieces were replaced for each measurement.

**ROTEM**

ROTEM (TEM International GmbH, Munich, Germany) (we used the ROTEM delta system) is a viscoelastic coagulation-testing device and has been described in detail [24]. ROTEM measurements "ExTem" and "FibTem" were initiated without delay after blood withdrawals.

**Blood alcohol concentration**

The department of forensic medicine at the Hannover Medical School performed the measurement of blood alcohol concentration.

Headspace gas chromatography was used for the serum ethanol concentration analysis. Chromatographic separation was performed on a CBK-B 60/80, 1 m x 2.0 mm (Restek GmbH, Bad Homburg, Germany), at a Clarus 480 gas chromatograph and a Turbo matrix 110 headspace sampler (Perkin Elmer Inc., Waltham, MA, USA).

Testing for the presence and subsequent quantification of ethanol in blood was carried out in static headspace mode. All samples were prepared and injected in duplicate. Linear regression analysis was used to generate calibration curves with duplicate measurements of aqueous calibration standards (0.5, 1.0, 2.0, 3.0, 4.0 and 5.0 mg/L). The limit of quantification was 0.1 mg/L. The intra-day precision (expressed as percent relative standard deviation (%RSD)) and the accuracy (expressed as% bias) of the method were 5% and 7%, respectively. Serum ethanol concentrations were converted into blood ethanol concentration as usual in forensic blood ethanol analysis [25, 26].

**Statistics**
Demographic data included age, gender, weight and height. Event data were start and end times of drinking, amounts and types of drinks, and blood alcohol levels. Ethanol intake was calculated from ingested beverages as described above.

Data were exported to SPSS 22 (IBM Corp., Armonk, NY, USA) for further descriptive and explorative data analysis. We compared the ROTEM data with the Wilcoxon test. A p-value < 0.05 was considered significant. For multiple comparisons, p-Values were calculated using the Holm-Bonferroni method.
Results

We examined eleven males (55%) and nine females (45%) for a total of 20 healthy volunteers. Mean age was 29.4 ± 6.0 years, average height was 177.5 ± 8.6 cm and weight 77.7 ± 16.5 kg. Resulting mean body mass index (BMI) was 24.6 ± 4.2 kg/m². The basic participant data are summarized in table 1. Each participant could choose between three types of alcohol: 10% of our participants chose red wine, 30% whisk(e)y and 60% vodka. The volunteers consumed a mean amount of 51.29 ± 13.51 g ethanol each. The first blood withdrawal before the consumption of ethanol showed a blood alcohol concentration of 0.00 ± 0.0 ‰ for all of our participants. The mean blood alcohol concentration was 0.48 ± 0.3 ‰ and 0.76 ± 0.2 ‰ for the second and third blood sample, respectively. Detailed results of our ROTEM (ExTEM and FibTEM) analysis of the blood samples are shown in table 2. We focused on the ExTEM and FibTEM parameters clotting time (eCT), clot amplitude after ten minutes (eA10 / fA10), maximum clot firmness (eMCF / fMCF) and maximum lysis (eML / fML). Mean baseline values were 61.4 ± 7.9 s for eCT. After the one-hour blood collection, we observed an eCT of 60.20 ± 9.48 s. The A10 value shows the amplitude of the ROTEM trace 10 minutes after the clotting started. The eA10 test showed 56.40 ± 6.24 mm, while the fA10 was 14.00 ± 4.76 mm. After the consumption of the whole amount of ethanol, we found an eA10 value of 55.70 ± 6.46 mm and an fA10 value of 12.95 ± 4.199 mm. Mean clot firmness (MCF) in ExTEM was an eMCF of 62.90 ± 5.60 mm in the first blood sample and an eMCF of 62.10 ± 5.95 mm in the last one. In FibTEM, we found an fMCF of 16.20 ± 8.67 mm and an fMCF of 13.65 ± 4.86 mm, respectively. We saw no hyperfibrinolysis in the 60 ROTEM measurements performed, which we assumed to indicate a maximum lysis (ML) of > 15%. Furthermore, we saw no significant difference in any parameter of fibrinolysis at all. Comparisons regarding the ROTEM parameters revealed no significant differences.
Gender aspects

We had 9 female and 11 male participants in our study. In our subgroup analysis divided by gender, we found no significant difference in the measured ROTEM parameters in our female subgroup (table 3). In our male subgroup, we saw a significant difference in the fA10 FibTEM value (p = 0.04, figure 3). There were no significant differences in age, height, weight and BMI. The female volunteers had to drink significantly less ethanol to reach the target of 1 ‰ (p = 0.001) and had a significantly lower blood alcohol concentration after the one-hour period (p = 0.004). The mean blood alcohol concentration of the female participants after one hour was 0.62 ± 0.2 ‰ compared to the concentration of the male participants of 0.87 ± 0.2 ‰.
Discussion

In this study, we determined the *in-vivo* effect of alcohol consumption on coagulation measured by rotational thrombelastometry (ROTEM). In our healthy participants, we found a significant change in the early fibrin-clot (FibTEM) at the fA10 value. Other parameters were not significantly influenced by the alcohol intake.

The reduced FibTEM amplitude ten minutes after the clot formation starts (A10) combined with a lower FibTEM maximum clot firmness (MCF) suggests that a moderate level of about 0.8 ‰ blood alcohol can affect the polymerization of fibrinogen. This finding is in line with results of previous studies where an impairment of fibrinolysis [11-13], slower rates of fibrin formation and fibrin cross-linking [3] and reduced fibrinogen and fibrin functionality were described [10, 13]. Howard et al. showed in a ROTEM based study on more than 400 trauma patients a bidirectional effect on coagulation related to ethanol ingestion. They demonstrated an initially impaired clot formation and a subsequent inhibition of fibrinolysis. They assumed these balancing mechanisms made a supposed correlation of altered ROTEM measurements and outcome in ethanol intoxicated trauma patients very difficult. Interestingly, we cannot demonstrate an impaired fibrinolysis in our healthy volunteers. The underlying mechanisms appear to be specific for the condition of a severe trauma in combination with ethanol ingestion [27].

We found a significant difference in the A10 FibTEM value in our male subgroup, whereas in the female subgroup there was no difference. Spoerke et. al. demonstrated that gender as an independent factor impacted the coagulation system and the influence of ethanol on haemostasis. They found a significantly decreased clot formation speed and reduced cross-linking of fibrin after alcohol ingestion in healthy male volunteers [3]. Generally, the outcome after severe trauma seems
to differ between males and females [28, 29]. However, in our study the male participants had significantly higher BAC after one hour than female volunteers.

De Lange et al. reported an inhibition of platelet adhesion to fibrinogen after intake of alcohol in alcopops (Barcardi breezer) but not after red wine (Rioja) [10]. The variety of alcohol can have impact on coagulation alterations, in particular on fibrin polymerization. Unfortunately, in our study only two participants (10%) chose red wine. Therefore, a comparison of different kinds of alcohol to red wine was not eligible.

We were unable to fully reproduce Engström’s in-vitro results in our in-vivo approach, as they showed strong disturbances of overall clot formation in their study. Clot formation time (CFT), which indicates the dynamics of clot formation, was reduced by 34% at an ethanol concentration of 1 ‰, and by 118% at 4 ‰, respectively [11]. Moreover, the fibrinolysis activity was reduced in their investigation. In our study, neither the dynamic parameters nor the fibrinolysis was different after alcohol intake. All participants consumed a calculated amount of ethanol to reach 1 ‰ blood ethanol concentration (BAC). For obvious reasons, for the in vivo setting, we did not take the 2 or 4 ‰ condition into account. With a mean BAC of 0.76 ‰, we did not reach the calculated and targeted 1 ‰ within one hour. Engström and colleagues used 96% pure ethanol added directly to a test tube, obviously without any additives. This and different metabolites of ingested ethanol such as acetaldehyde may impair the assays differently [30]; we furthermore allowed different types of alcohol (red wine, whisk(e)y and vodka). Moreover, we used citrated blood sample tubes in our study. Naturally, Engtröms et al. established no correlation to the in-vivo situation due to the limitations of the in-vitro setting.

Based on our results, the implications for daily clinical practice remains unclear. Although the absolute reduction in fibrin clot firmness was only 1 mm at fA10 and 3 mm at fMCF, in patients
suffering from an underlying coagulopathy (e.g. trauma patients) an additionally impaired coagulation due to alcohol might affect bleeding, clinical outcome and clinical therapeutic decisions. Alcohol is a socially accepted and commonly used drug. Moderate amounts are even considered to be protective for the cardiovascular system [31, 32]. The impact of acute ethanol intake on coagulation and parameters of coagulation still needs to be elucidated and is hotly debated. Some studies found no effect, in particular on standard coagulation measurements and haemorrhagic complications [14]. Others described multiple effects of ethanol on the coagulation system such as alterations in platelet function and prolonged bleeding time [9] or inhibition of platelet adhesion to fibrinogen [10]. Point of care coagulation assessment by ROTEM or TEG has become a routine option in the last decade as it is considered to be a better predictor of clinical bleeding tendency than other coagulation tests [33, 34]. Furthermore, it seems to be more sensitive and specific than routine coagulation tests in detecting impairments of the coagulation system [34-37]. The ROTEM/TEG analysis assesses haemostasis in a more holistic manner than the standard coagulation test. With these tests, the detection of the whole clot formation, fibrinogen-platelet interactions and hyperfibrinolysis is possible [24].

A trial investigated haemostasis in 264 trauma patients with elevated ethanol blood concentration and found impaired clot formation in thrombelastographic (TEG) assays, while the elevated blood concentration was not predictive for transfusion requirements or early or late mortality. The authors concluded that ethanol potentially directly affect the TEG measurements and that further studies were needed [38].

Nevertheless, a significant number of patients in an emergency department are under the influence of higher blood alcohol concentrations [2-5]. Trauma mortality increases with the level of blood ethanol [19, 20], and trauma combined with ethanol seems to be associated with increased blood loss requiring volume resuscitation and red blood cell (RBC) transfusion [21]. It is well known that,
massive haemorrhage is one of the leading causes of death in trauma patients and is thus responsible for approximately 40% of trauma deaths [39]. Furthermore, the trauma itself can cause an imbalance in haemostasis known as trauma-induced coagulopathy (TIC) [40], which may lead to increased bleeding if alcohol intoxication comes into play as an additional factor. TIC is a very complex mechanism which is triggered by hypoperfusion and activated protein C and eventually leads to hypofibrinogenemia, hyperfibrinolysis, impaired fibrinogen polymerization and subsequent inhibition of normal clot formation, especially in case of extensive tissue trauma [41, 42]. Extrapolating our results, it could be presumed that alcohol might aggravate haemostasis impairment at this point. One conceivable mechanism beside the potentially direct impairment of fibrinogen polymerization is that alcohol-positive trauma patients had elevated base excess levels compared to non-alcoholised trauma patients with a comparable severity of injury [38]. Hess and Brohi described the coagulopathy of trauma as follows: the acidaemia itself impairs the function of the plasma protease and the activity of coagulation factor complexes [1]. Ethanol and/or its metabolites possibly aggravate acidaemia and therefore contribute to the alteration of coagulation.

This study has some limitations: we don’t reach the average blood alcohol target of 1‰ in every volunteer, which limits the comparability of our data. Moreover, the type of the alcohol was freely selectable. Because of this, a discrimination of a specific type-dependent alcohol effect is complicated. Furthermore, the sample size was not powered to show specific gender effects. In addition, all participants were healthy.

In conclusion, our results show a moderate impairment of thrombelastometric measurements, limited to early fibrin clot formation, in healthy volunteers. However, it is to be expected that the fibrin-clot, determined by thrombelastometric methods, diminishes after usual alcohol intake with the potential risk of falling below critical levels if other coagulation impairments supervene. Further
studies are required to evaluate the influence of ethanol itself on the thrombelastometry and to determine the effects of acute ethanol intoxication on haemostasis, particularly in trauma patients.
References


Table 1. Basic Patient Data and Ethanol Ingestion

<table>
<thead>
<tr>
<th></th>
<th>All (n = 20)</th>
<th>Male (n = 11)</th>
<th>Female (n = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean</td>
<td>SD</td>
<td>mean</td>
</tr>
<tr>
<td>Age / years</td>
<td>29.4</td>
<td>6.0</td>
<td>32.2</td>
</tr>
<tr>
<td>Height / cm</td>
<td>177.5</td>
<td>8.6</td>
<td>181.5</td>
</tr>
<tr>
<td>Weight / kg</td>
<td>77.7</td>
<td>16.5</td>
<td>84.8</td>
</tr>
<tr>
<td>BMI</td>
<td>24.6</td>
<td>4.2</td>
<td>25.71</td>
</tr>
<tr>
<td>Ethanol / g</td>
<td>51.3</td>
<td>13.5</td>
<td>59.37</td>
</tr>
<tr>
<td>BAC after 1 h / %</td>
<td>0.75</td>
<td>0.20</td>
<td>0.87</td>
</tr>
</tbody>
</table>

(SD: standard deviation; P: significance of intergender value differences, BAC: Blood Alcohol Concentration)
**Table 2.** Blood Alcohol Concentrations and ROTEM Parameters (n = 20)

<table>
<thead>
<tr>
<th></th>
<th>baseline (n = 20)</th>
<th>30 min (n = 20)</th>
<th>1 h (n = 20)</th>
<th>$P$</th>
<th>$P^*$</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>mean (SD)</td>
<td>mean (SD)</td>
<td>mean (SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BAC /‰</td>
<td>0 (0)</td>
<td>0.48 (0.26)</td>
<td>0.75 (0.21)</td>
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<td></td>
</tr>
<tr>
<td>BrAC /‰</td>
<td>0 (0)</td>
<td>0.63 (0.19)</td>
<td>0.63 (0.15)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>eCT /s</td>
<td>61.4 (7.9)</td>
<td>62 (12.2)</td>
<td>60.2 (9.5)</td>
<td>0.608</td>
<td>1.0</td>
</tr>
<tr>
<td>eA10 /mm</td>
<td>56.4 (6.2)</td>
<td>56.9 (6.2)</td>
<td>55.7 (6.5)</td>
<td>0.354</td>
<td>1.0</td>
</tr>
<tr>
<td>eMCF /mm</td>
<td>62.9 (5.6)</td>
<td>63 (5.2)</td>
<td>62.1 (5.9)</td>
<td>0.248</td>
<td>1.0</td>
</tr>
<tr>
<td>eCFT /s</td>
<td>86.5 (19.7)</td>
<td>83.5 (20.5)</td>
<td>85.6 (21.0)</td>
<td>0.768</td>
<td>1.0</td>
</tr>
<tr>
<td>eML /%</td>
<td>0.5 (1.1)</td>
<td>0.2 (0.5)</td>
<td>0.1 (0.4)</td>
<td>0.149</td>
<td>0.894</td>
</tr>
<tr>
<td>fA10 /mm</td>
<td>14 (4.8)</td>
<td>13.2 (4.7)</td>
<td>13 (4.2)</td>
<td>0.014</td>
<td>0.112</td>
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<tr>
<td>fMCF /mm</td>
<td>16.2 (8.7)</td>
<td>13.7 (4.6)</td>
<td>13.7 (4.9)</td>
<td>0.075</td>
<td>0.525</td>
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<td>fML /%</td>
<td>1.4 (2)</td>
<td>2.2 (2.5)</td>
<td>2.1 (2.3)</td>
<td>0.279</td>
<td>1.0</td>
</tr>
</tbody>
</table>

(SD: standard deviation; BAC: blood alcohol concentration; BrAC: breath alcohol concentration; eCT: ExTEM clotting time; eA10: ExTEM amplitude 10 minutes after clotting started; eMCF: ExTEM maximum clot firmness; eCFT: ExTEM clot formation time; eML: ExTEM maximum lysis; fCT: FibTEM clotting time; fA10: FibTEM amplitude 10 minutes after clotting started; fMCF: FibTEM maximum clot firmness; fML: FibTEM maximum lysis; $P$: significance of value differences between baseline and 1 h; $P^*$: significance of values after applying the Holm-Bonferroni method)
### Table 3. Gender Aspects

<table>
<thead>
<tr>
<th></th>
<th>Gender</th>
<th>baseline</th>
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<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>mean</td>
<td>SD</td>
<td>mean</td>
<td>SD</td>
<td>P</td>
<td>P*</td>
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</tr>
<tr>
<td></td>
<td>male (n = 11)</td>
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<td></td>
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</tr>
<tr>
<td>cCT</td>
<td>/ s</td>
<td>64.7</td>
<td>5.4</td>
<td>63.7</td>
<td>8.4</td>
<td>0.748</td>
<td>1.0</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>eA10</td>
<td>/ mm</td>
<td>55.2</td>
<td>5.4</td>
<td>53.6</td>
<td>6.0</td>
<td>0.190</td>
<td>1.0</td>
<td></td>
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</tr>
<tr>
<td>eMCF</td>
<td>/ mm</td>
<td>61.8</td>
<td>5.3</td>
<td>60.2</td>
<td>5.9</td>
<td>0.178</td>
<td>1.0</td>
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<tr>
<td>eCFT</td>
<td>/ s</td>
<td>90.2</td>
<td>17.4</td>
<td>92.1</td>
<td>20.2</td>
<td>0.688</td>
<td>1.0</td>
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<tr>
<td>eML</td>
<td>/%</td>
<td>0.8</td>
<td>1.3</td>
<td>0.2</td>
<td>0.6</td>
<td>0.208</td>
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<tr>
<td>fA10</td>
<td>/ mm</td>
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<td>/ mm</td>
<td>16.4</td>
<td>10.8</td>
<td>12.1</td>
<td>4.3</td>
<td>0.960</td>
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<tr>
<td>fML</td>
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<td>2.3</td>
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<td>2.3</td>
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<tr>
<td></td>
<td>female (n = 9)</td>
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<tr>
<td>cCT</td>
<td>/ s</td>
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<td>8.8</td>
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<tr>
<td>eA10</td>
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<tr>
<td>eMCF</td>
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<tr>
<td>eCFT</td>
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<tr>
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<tr>
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<tr>
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(SD: standard deviation; eCT: ExTEM clotting time; eA10: ExTEM amplitude 10 minutes after clotting started; eMCF: ExTEM maximum clot firmness; eCFT: ExTEM clot formation time; eML: ExTEM maximum lysis; fCT: FibTEM clotting time; fA10: FibTEM amplitude 10 minutes after clotting started; fMCF: FibTEM maximum clot firmness; fML: FibTEM maximum lysis; P:
significance of value differences between baseline and 1 h; P* significance of values after applying the Holm-Bonferroni method)
Fig. 1. Point of care setup for our study at the department’s Christmas party
Fig. 2. Flow-chart of the study design

- Enrollment: n=20
- Baseline

Every 10 min; breath alcohol concentration

- Blood alcohol concentration 0.5 promille OR after 30 min

Second blood withdrawal: n=20

Yes: ROTEM n=20
No: Breath alcohol concentration

Third blood withdrawal: n=20

Yes: ROTEM n=20
No: Blood alcohol concentration n=20
Fig. 3. ROTEM parameters (eCT: ExTEM clotting time; eA10: ExTEM amplitude 10 minutes after clotting started; eMCF: ExTEM maximum clot firmness; eCFT: ExTEM clot formation time; fCT: FibTEM clotting time; fA10: FibTEM amplitude 10 minutes after clotting started; fMCF: FibTEM maximum clot firmness; * p < 0.05, differences between baseline and 1 h)