connective tissue disorders as in the Broome article, and perhaps to confirm the presence of kidney disease in those with likely NSF [3]. As Dr Boehm rightly notes, no specific biomarker of NSF exists. As such, the diagnosis of NSF requires a high index of clinical suspicion and confirmatory histology, with laboratory evidence providing indirect additional information to revise the probability of disease. We agree that identification of a sensitive and specific serum biomarker would be useful in the diagnosis of this clinical entity.

Conflict of interest statement. None declared.

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Measuring lock solution spillage in-vivo versus in-vitro

Sir,
I have read with interest the paper ‘Measuring of the heparin leakage into the circulation from central venous catheters—an in vivo study’ [1] pre-published in NDT.

I agree with the authors that lock solution spillage might be affected by patient’s movement in vivo, but this is in best case an additional effect that increases spillage further after lock instillation. I disagree with the results and with the validity of the proposed method.

The events that take place when the catheter is flushed and locked are governed by physics, and there is no difference whether the lock is injected into a blood vessel or a beaker. In order to corroborate this statement, I have done measurements as described below.

Physics
As described in a previous paper [2], ~25% of the lock is spilled into the beaker or the patient when the lock is instilled. Within seconds, the lock between the tip and the most distal side hole is flushed out. Quantitatively, this contribution is small (1–2%). This process is followed by additional losses as documented previously [3] that might be influenced by patient movements.

Lock aspiration
When the lock is aspirated in vitro using Newtonian fluids (aqueous solutions), the reverse effect can be observed. When an amount equal to the lumen volume is aspirated, only 75% of the lock is recovered. If the lock is aspirated from blood, the simple theory does no longer work because blood is a non-Newtonian fluid.

Measurements
I have used the venous lumen of the Medcomp SplitCathIII 11F/28 cm catheter without side holes. The nominal volume is 1.8 mL (ven). The measured volume was 1.95 ± 0.0029 mL when measured in triplicate.

Instillation
Immediately after instillation, the catheter was removed, and the fluid in the lumen was collected. The concentration in the lumen was measured. Two measurements were done. The result was 77% and 73%, respectively. This means that ~25% was spilled which is in agreement with the theory and previous measurements.

Lock aspiration
Haematocrit of the fluid in the syringe, in the catheter lumen and in the beaker was measured. The three samples together with the volumes allow for an estimate of the measurement error. Two measurements were done. The result was 16% and 13%, respectively. The spectrophotometric measurement results were 15% and 12%, respectively. The accuracy checks resulted in errors of 5% and 7%, respectively.

Summary
The measurements corroborate the theoretical expectation that ~25% of lock is spilled. They also corroborated the prediction that instillation and withdrawal of lock solution are not ‘symmetric’ and that haematocrit cannot be used for lock spillage measurement.

Conflict of interest statement. None declared.

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Reply

Sir,

We thank Dr Polaschegg for his comments related to our work [1]. I will try to address the doubts by responding to them in accordance with their order in the aforementioned letter.

I disagree with the first assertion that catheter leak is exclusively determined by the physics of fluid and that there is no difference whether a catheter lock is injected in a blood vessel or a dispenser. However, I do not think it is also crucial. The author neglects the facts that a catheter under in vivo conditions usually has many irregularities:

- The shape of the tube that fluid flows through [measurements in vitro, usually with a vertically positioned catheter [2] with no curves, in contrast with catheters in vivo which are usually bent (bending of jugular catheters is approximately 160°)].
- Narrowing (deformities) of a tube that fluid flows through (everybody is probably familiar with deformity produced by the clamps of catheter branches), narrowing caused by possible clots, narrowing caused by ‘a knee’ of a catheter . . .
- Change of speed when injecting lock solution.

The aforementioned irregularities influence the physics of fluid and cause a change in movement from a predominantly laminar to a predominantly turbulent blood flow.

Since early catheter leak is a direct consequence of the laminar flow of fluid (this is influenced by the aforementioned and other numerous factors that cannot be mentioned here due to the brevity of the letter), the switch to the turbulent flow will create a certain barrier and reduce catheter leak (something similar to a small bubble of air on the barrier among fluids [3]).

The author of the letter claims that catheter leak under in vitro conditions amounts to ~25%!

The facts are as follows: the results of the study that the author refers to show that early catheter leak amounts to 20–25% [3]. It should be noted that the other study by the same author says that catheter leak amounts “up to 15%” [4]. The results of other studies on the size of early catheter leak show the following: 11% [2] and 18%–30%. It should be noted that all five kinds of catheters in this study were with side holes [5]. The aforementioned results refer to various kinds of catheters that I consider being the main reason for the obvious difference between the results (differences in catheter architecture, various localizations of lateral holes, various diameters and catheter length . . .).

Furthermore, the author claims that when we aspirate the volume, which corresponds to the catheter volume, only 75% of the lock solution is recovered, and then concludes that ‘instillation and withdrawal of lock solution is not symmetric. . .’. We could not agree more with the aforementioned assertions, and we were aware of them during the experiment.

If one carefully reads our work, one will see that we did not aspirate the volume that corresponds to the catheter volume but twice and three times a larger volume. By doing so, we wanted to extract the complete volume of the lock solution and avoid the lock solution remaining in the catheter. We included in the aforementioned equation the excess of the volume that we aspirated and it was compensated in this manner.

Conclusion

We think that the main reason for different results in various in vivo studies, as well as different results in our study (catheter leak 12% for cuffed and 31% for the uncuffed catheters [1]), is catheter architecture. However, other aforementioned and numerous not mentioned reasons that exist in vivo, compared to in vitro conditions, significantly change catheter leak.

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Letter to the editor regarding ‘Acute phase reaction to gadolinium-DTPA in dialysis patients’

Sir,

We read with great interest ‘Acute phase reaction to gadolinium-DTPA in dialysis patients’ by Steen et al. [1].