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Letters to the Editors

Pressure injection demonstrates points of weakness in the posterior nasal arteries

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Dear Sirs

Recently, Chiu *et al.* published the above original article, which we read with great interest.[1] The authors suggested that there may be pre-existing points of weakness in the posterior nasal arteries, which could lead to epistaxis in cases of non-controlled hypertension. They tested this hypothesis by attempting to produce a vessel rupture in unfixed cadavers, via pressure injection of the maxillary arteries. In our opinion, this hypothesis is probably sound, but the study results remain inconclusive. Chiu and colleagues' findings could be of importance, but we have a number of concerns regarding their interpretation.

Firstly, the authors did not describe completely the data concerning their sample cadavers: i.e. their average age, gender, pre-mortem general pathology and length of time since death. For example, vascular risk factors such as atherosclerosis, arteriosclerosis and diabetes mellitus are already associated with vessel weakness, and could have influenced vessel disruption.[2–4] Moreover, post-mortem changes occur within the first hours of death and could have been significant at the point of study, especially in unfixed cadavers.[5] All these factors may have influenced the cadaveric vessel state, and could have biased the final study outcome.

Secondly, while Chiu *et al.* chose the optimal site of injection, they did not report the median volume (and method of estimation thereof) of the injected embalming fluid and latex in experimental cadavers and controls. This is very important, and not easy to quantify considering the possibility of vascular anastomoses.[6] We believe that this may have resulted in overestimation of the injection volume required to cause vessel disruption.

Unfortunately, the exact pathogenesis of spontaneous epistaxis remains unknown. However, we believe that there is always at least one cause triggering any episode of epistaxis, and that the term 'idiopathic epistaxis' should thus not be used.

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Authors' reply

Dear Sirs

I would like to thank Drs Asanau and Timoshenko for their comments on our paper; constructive criticism is always welcome.

I would like to point out that the paper did not state that pre-existing points of weakness lead to epistaxis in 'non-controlled hypertension'. As stated in the discussion, the relationship between epistaxis and hypertension is rather controversial at the best of times! I certainly agree that more information on the premorbid history of the cadavers would have potentially been useful to determine the representativeness of the sample; however, more often than not, cadavers do not come with complete medical notes. In addition, it is important to bear in mind that, whilst 'vascular risk factors' will undoubtedly influence vascular function and structure in the long term, they have never been directly implicated in the immediate pathogenesis of epistaxis specifically.

I agree that post-mortem changes mean that results derived from studies using cadaveric tissues and vessels cannot be directly correlated to the *in vivo* situation. However, one would have to assume that such changes would be more or less uniform, rather than altering the posterior nasal arteries differently compared with other vessels. Thus, I think it would be fair to assume that post-mortem weaknesses reflect pre-mortem weaknesses, and are not entirely due to subsequent experimental procedures.

I assume that by 'overestimation of the injection volume' the authors mean that injection of an excessive amount could result in vessel disruption. This is in essence the whole point of the study: to 'stress test' the system in an attempt to 'reveal' potential points of weakness.

I certainly agree that there are many unanswered questions in our basic understanding of the mechanism of epistaxis, but I hope that our paper at least provides a starting point for further research. As Asanau and colleagues allude, the vast majority of 'idiopathic' epistaxis is probably due to a combination of systemic and local factors that we are far from understanding at present.

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