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Response to the Letter by Dr. Hanne Christensen: Blood Pressure in Acute Stroke

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Dear Sir,

We thank Dr. Christensen [1] for her letter and the opportunity to clarify our findings. Dr. Christensen's main concerns appear to be how we handled data from patients in whom the time of stroke onset was not certain and how data from patients in whom there was a significant delay between stroke onset and admission might have affected our findings.

There were 44 patients (28%) in whom the time of stroke onset was not certain because symptoms of stroke were first noted on waking. In these patients the time of stroke was defined as the time when a neurological deficit was first noted. We accept that this might have some impact on the results if there is an important difference between the actual time of stroke onset and our defined time of onset. However, the known morning surge in stroke inci-

dence [2] makes it likely that many of these strokes actually occurred on waking and thus the difference between the actual time of stroke onset and our defined time may not be great. Indeed, this was one factor in our making this methodological choice.

We agree that information regarding the timing of the first available blood pressure (BP) recordings should have appeared in the paper and apologise for its omission. For the overall study population of 157 patients, the median time from stroke onset to the first recorded BP measurement was 2 h 35 min (range: 0 min to 47 h 40 min). For the 113 patients with known stroke onset time, it was 2 h 12 min (range: 0 min to 47 h 40 min), while for the 44 patients with defined stroke onset time, it was 4 h 42 min (range: 30 min to 37 h 40 min). While there was a significant difference in the median time between the known and defined stroke onset time groups (median 2-sample test, $p = 0.006$), their empirical distribution functions were not statistically different (Kuiper 2-sample test, $p = 0.08$). In addition, the time trends observed in the mixed-effect models using the entire cohort of 157 patients (SBP = 0.30 mm Hg/h, DBP = 0.13 mm Hg/h; both $p < 0.05$) were also seen in the subgroup of 113 patients with a known time of onset (SBP = 0.23 mm Hg/h, DBP = 0.13 mm Hg/h; both $p < 0.05$).

With regards to 'missing' BP values from the interval between stroke onset and admission, mixed-effects modelling does not require balanced or evenly spaced recordings and there is no need to impute values that are 'missing', for example because of differences between patients in times of stroke onset and admission. Instead, group trends over time within these models utilise all available data after accounting for the serial correlation between repeated measurements on individuals. Thus a patient with a time from stroke onset to the first recorded BP measurement of, say, 8 h will only contribute information to the group trends over an 8- to 48-hour interval.

Dr. Christensen wonders whether a drop in BP on admission in patients recruited with varying delays from stroke onset to ad-

mission might produce the linear decline we observed. We have not examined whether admission results in an acute fall in BP but are unaware of any data suggesting this. Indeed, 'stress' related to the process of hospital admission has been postulated as one reason for high admission BPs [3]. As most of our patients first/admission BP recordings were taken within 8 h of stroke onset, and only 14 patients whose initial BP was taken beyond 24 h after stroke were included, our observed linear decline to 48 h would appear to be due to a continued fall in BP in the patients admitted early who are providing most of the data.

We also apologise to Dr. Christensen and her colleagues for misrepresenting the statistical methods used in their study [4] as 'regression analysis'. However, our point stands that mixed-effect models make better use of the available data than traditional statistical methods, and we hope that our findings have provided useful additional information on this subject as a result.

References

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