Poidvin et al. [1] recently reported "a relationship between hemorrhagic stroke and GH treatment in childhood for isolated growth hormone deficiency or childhood short stature". This report was the result of reanalyzed problematic data, specifically the Carel et al. report on treatment with growth hormone (GH) in childhood contributing to increased mortality in young adults [2] However, a subsequent publication by Savendahl et al. reported very low mortality rates due to circulatory system disease. Furthermore, the US FDA identified a number of study design weaknesses in the Carel et al. report [3], including insufficient details of the completeness of outcome ascertainment, which relied primarily on death certificates. In contrast, Savendahl et al. used public health records and medical chart review to ascertain mortality and cause of death in 98% of GH recipients.

Poidvin et al. [1] did little to overcome shortcomings in data collection and analysis of the previous publication. Low response rate to questionnaires (45%) increased the likelihood of responder bias. Event rates were extrapolated based on the absence of data, accepting a lack of reports as evidence of such events. One hundred subjects between low- and high-risk groups were reclassified without describing the criteria for reclassification and its effect on the analysis. Both reports make inapt comparisons to population-based control groups. The implication of such comparisons attributed the outcomes to GH treatment, whereas it may be inherent in the subjects studied (short, slow-growing, predominantly male children with one of three diagnoses: GH deficiency, small for gestational age, and idiopathic short stature). A more suitable cohort for comparison (admittedly not easy to obtain) would be untreated adults of similar age, height, and diagnosis. Importantly, short stature [5] and untreated adult GH deficiency [6] themselves have been associated with adverse cardiovascular outcomes. The elevated mortality rate observed in the shortest subjects (at initiation of treatment) argues that the underlying condition rather than the treatment increases risk.

Finally, while most hemorrhagic strokes occurred in GH-deficient adults, the accompanying editorial [7] suggested that adults treated with GH in childhood should undertake surveillance and primary preventive treatment strategies. This study may cause unnecessary anxiety in previously-treated patients, and those contemplating treatment.