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Author's reply

Sir—We reported the use of MRI along with diffusion-weighted imaging and apparent diffusion coefficient mapping, not, as Gerda Zeeman and colleagues state, to show residual areas of infarction, but to differentiate vasogenic oedema from cytotoxic oedema caused by infarction. Results suggested vasogenic oedema and prompted us to lower blood pressure. Conversely, we would have avoided antihypertensive treatment if cytotoxic oedema had been present.

Zeeman and colleagues express their concern about this rationale. They remark that there was no evidence that empirically treating blood pressure of more than 160/100 mm Hg in eclampsia worsens central nervous system changes from ischaemic lesions. Furthermore, they mention that empirical treatment for diastolic blood pressures of more than 105–110 mm Hg was recommended irrespective of vasogenic or cytotoxic oedema. In support, they quote a study that uses apparent diffusion coefficient mapping in only one patient.

We do not subscribe to this point of view. Ischaemic stroke is accepted as being a disorder with many causes that complicates some cases of eclampsia and haemolysis, raised liver enzymes, and low platelet count syndrome. We believe that once valuable diagnostic information can be obtained to distinguish vasogenic from cytotoxic oedema, it should be used to tailor antihypertensive treatment in accordance with evidence-based guidelines. If stroke is present in an eclamptic patient, accepted treatment for stroke should be applied.

There are no data that define what levels of raised blood pressure mandate emergent treatment after stroke. The most recent guidelines for the management of patients with acute ischaemic stroke, published by the American Heart Association, recommend—with the exception of stroke associated with myocardial infarction, renal failure, intracerebral haemorrhage, or aortic dissection—withholding antihypertensive agents unless systolic blood pressure exceeds 220 mm Hg, or mean arterial pressure exceeds 130 mm Hg.[1] Despite these guidelines, data suggest that antihypertensive treatment may be vastly overused in acute stroke with potentially harmful consequences for the penumbra after reduction of cerebral perfusion pressure.[2,3]

Finally, we share the concerns about time and money that has to be spent for diffusion-weighted imaging. Nevertheless, given the potential effect on therapeutic decisions, experts in the specialty of neuroradiology postulate that this technique should be used in all eclamptic patients[4] and should even be implemented as the principal imaging method for initial assessment of acute stroke.[5]

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Loss of taste is loss of weight

Sir—Morbid obesity is an important health issue in more-developed countries. Therapeutic attempts by pharmaceutical agents, psychotherapy, and abdominal surgery are of only limited success.[1] A further possibility for treatment might be modulation of taste.

A man aged 53 years underwent resection of a vocal-cord polyp. During surgery, emergency intubation was necessary because of sudden apnoea. Intubation was complicated, lengthy, and induced massive bleeding. Immediately after surgery, the patient noticed loss of taste. He was taking no drug known to alter taste perception.[2] Smelling was slightly impaired. Gustatory tests revealed that taste was greatly impaired on the tip of his tongue, and that taste perception was absent for sour, salt, bitter, and severely reduced for sweet. 20 months later, the patient started tasting again, beginning on the tip of the tongue and with sweet taste. After 36 months, ageusia had completely disappeared. Immediately after surgery, the patient was slightly obese with a weight of 74 kg. He had repeatedly tried to lose weight, but none of his attempts was successful. Surprisingly, during the period of ageusia he lost 20 kg. He simply noticed decreased appetite and ate less.

Decline in smell and taste can decrease appetite and lead to anorexia.[3–5] There are several possibilities for the pathogenesis of ageusia in our patient. First, ageusia occurred because of bilateral pressure palsy of the afferent sensory pathways, especially the chorda tympani, by lengthy laryngoscopic manipulation. Second, the central gustatory pathways might have been affected by an anaesthetic, any other medication given during surgery, or by cerebral hypoxia during emergency intubation. Third, gustatory receptors on the tongue were directly disturbed by difficult intubation. Most probably, the enforced manipulations with the blade of the laryngoscope led—directly or indirectly by haematoma—to bilateral local pressure of peripheral nervous structures, resulting in pressure palsy of the gustatory pathways and, consequently, to ageusia.

Iatrogenic ageusia, like in the presented case, could be a therapeutic option for patients with morbid obesity. Possible interventions to induce ageusia could be drugs with ageusia as a side-effect,[2] thalamic stereotactic interventions, and interruptions of the centripetal gustatory projections on the peripheral or the central side.

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