was the goal of our retrospective study which showed that the use of HES 130/0.4 resulted in similar level of DGF, when compared with HES 200/0.6. However, the serum creatinine levels were lower in the group treated with HES 130/0.4 than in the group treated with HES 200/0.6. This result is still valid at 1 yr. Although only the donors were matched, analysis of recipient characteristics did not show significant differences. Of note, this study was not supported by industry. The limitations of the study design are extensively reported in the discussion section. Use of a randomized design is not feasible since in France HES 200/0.6 was withdrawn from the market many years ago. We did not make a conclusion on the superiority of any given HES but on the need to clarify the safety of third generation HES preparations with low degree of substitution in the field of renal transplantation. One can acknowledge that this conclusion was careful. The results of retrospective studies should be better considered because they reflect the real-life conditions. Regarding the literature, it is not so clear that renal function was affected by the use of a low dosage of third generation colloid if the guidelines for its use are carefully respected, that is, contraindication in patients with renal failure, respect of maximal dosages, and limited duration of use. In our opinion, the multicentre randomized trial entitled ‘Efficacy of Volume Substitution and Insulin Therapy in Severe Sepsis (VISEP)’ cannot be used in all conditions. First, we did not investigate patients with severe sepsis. Secondly, the renal function of our patients was by definition normal. In VISEP, it seems that 25% of the patients had an initial level of serum creatinine above 180 μmol litre⁻¹. Use of HES in these patients can be deleterious. Thirdly, the characteristics of the colloid that was used in VISEP are close to our ‘retrospective’ control, that is, the HES 200/0.6. As indicated above, this solution has not been available in France for many years. Fourthly, we infused 34 ml h⁻¹ of colloid to our brain-dead patients. With respect to the mean duration of brain death (18 h), our brain-dead patients received approximately 10 ml kg⁻¹ of HES, that is, <612 ml. Just as reminder, the negative effects in VISEP appeared at a dosage >22 ml kg⁻¹ with a mean dosage of 136 ml kg⁻¹, that is, approximately 8 litre of colloids during the stay.

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**Unexpected awakening from anaesthesia after hyperstimulation of the medial thalamus in the rat**

Editor—We would like to attract the attention of anaesthetists and neurophysiologists to a case in which rats unexpectedly and transiently awoke from anaesthesia during a stereotaxic procedure performed as a part of an experimental lesional study. In our opinion, this important observation, described below, further contributes to the current knowledge on the functional mechanisms underlying the induction and maintenance of anaesthetic-induced unconsciousness.

The stereotaxic procedure served as the initial intervention for the study into the role of thalamic activity in EEG scalp-recordings and incorporated ibotenic acid...
(IBO)-induced lesions of different thalamic regions. The protocol was performed in accordance with high standards of animal care as approved by the Institutional Animal Experimentation Committee (Utrecht University, The Netherlands). Adult male Wistar rats (n=9) were anaesthetized with i.p. fentanyl 0.3 mg kg\(^{-1}\) and medetomidine 0.3 mg kg\(^{-1}\). Fentanyl/medetomidine (from the same batch for all animals in the experimental group) is the anaesthetic combination of choice for adequate surgical anaesthesia of rats.\(^1\) Subsequently, (i) the rats’ heads were fixed in a stereotaxic apparatus; (ii) their skulls exposed and cleaned, following a caudal to rostral skin incision over the midline and removal of the periosteum after a local lidocaine block; and finally, (iii) small holes were drilled in their skulls to allow access of a syringe for bilateral IBO (5 μg μl\(^{-1}\) PBS 0.1 M; 0.3 μl per injection site) or sham (0.3 μl of PBS 0.1 M per injection site) injections over 2 min in the medial intralaminar (IBO: n=4; sham: n=1) or lateral thalamus (IBO: n=2; sham: n=2) (stereotaxic coordinates according to Paxinos and Watson).\(^2\) respectively. IBO, by way of hyperstimulating glutamatergic receptors, induces an intracellular calcium overload which subsequentially results in neuronal cell death by apoptosis or necrosis,\(^3,4\) and is commonly applied in lesional studies.

Following the preparations described, and approximately 40 min after induction of anaesthesia, the first injection (IBO or PBS) was made in all rats. A few seconds after the first IBO injection in the medial intralaminar thalamic region, but not after sham injection, the IBO-injected rats demonstrated signs of transient awakening from anaesthesia during a 10–30 s period, that is, (i) increased behavioural activity (purposeful, intense movement of paws and hind body, vocalization) and (ii) increased physiological activity (fast and superficial respiration comparable with the awake state and unlike the reduced but stable respiration during anaesthesia). These phenomena were followed by respiratory depression ultimately leading to 100% loss of these animals. In contrast, IBO and sham injections in the lateral thalamic region did not result in any change in behavioural nor physiological activity during the procedure.

These effects after IBO injection in the medial intralaminar thalamic region, together with the fact that awakening during stereotaxic surgery is an extreme stressful event for the animal, led us to prematurely stop this experimental approach from an animal welfare point of view. Hence, a limited number of animals is described. In addition, the histological verification of the medial intralaminar thalamic injection site in these animals could not be reliably assessed, as IBO needs several hours to days, to exert its neuronal apoptotic or necrotic effects.\(^5\) Since these specific animals died shortly after IBO administration, the exact extent and location of the lesion in these animals could not be evaluated. However, prior pilot experiments, using guided cannula’s or electrolytic lesioning, did allow reliable verification of the coordinates used in this procedure for targeting the medial intralaminar thalamus, thus providing supporting evidence on the use of correct coordinates in this experimental approach. In contrast, the coordinates for the lateral thalamus location were histologically verified in this study, as these animals survived the surgery thereby allowing IBO to fully develop its cytotoxic effects.

We attribute the observation that IBO-induced hyperstimulation of the medial intralaminar thalamus, rather than of the lateral thalamus, is able to transiently reverse anaesthesia, to the neurofunctional role of the medial intralaminar thalamus,\(^5,6\) rather than to alternative explanations such as possible biological variation between rats in the response to anaesthetic drugs. Anaesthetic effects leading to unconsciousness have long been associated with effects on the brainstem ascending reticular activating system, rather than primary sensory pathways.\(^5\) Sensory input-induced activity in the brainstem reticular activating system induces and maintains arousal.\(^5,8\) The medial intralaminar thalamus, being an extension of the brainstem reticular activating system, is involved in arousal mechanisms as well. As such, the medial intralaminar thalamus is considered to be involved in processing sensory-input-induced activity from the brainstem reticular activating system to cortical regions involved in perceptual awareness of the different sensory modalities.\(^5,9\) In contrast, the lateral thalamus, as part of the primary somatosensory pathway, is functionally involved in somatosensory discrimination, rather than arousal.\(^10\) Combined, the ability of IBO-induced hyperstimulation of the excitatory medial intralaminar thalamic neurons, rather than of lateral thalamic neurons, to reverse anaesthesia, is completely in line with, and underscores, the pivotal neurofunctional role of the medial intralaminar thalamus in arousal and, alternatively, anaesthetic-induced unconsciousness.\(^5,6,8,11\)

In conclusion, in relation to animal welfare, the increased risk of unwanted side-effects and need for meticulous control of the experimental protocol must be considered when experiments involve the medial thalamus as a target of excitotoxic lesioning.

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Coanda effect as an explanation for unequal ventilation of the lungs in an intubated patient?

Editor—The Coanda effect is an established physical phenomenon of fluid flow which has practical applications in the medical field (Fig. 1).1 2 I would like to report an occurrence of unequal inflation of the lungs during artificial ventilation of a paralysed and intubated patient using a single-lumen tracheal tube, in which I propose that the observed ventilation pattern may have resulted from the Coanda effect occurring in the trachea.

A 58-yr-old ASA I female patient underwent left total knee replacement under general anaesthesia. After induction of anaesthesia with thiopental and fentanyl and muscle relaxation using atracurium, the trachea was intubated with a 7.5 mm single-use cuffed tracheal tube (Unomedical Sdn Bhd, Malaysia) with a grade I laryngeal view. On examination for correct tracheal tube position and ventilation, inspection showed initial right chest expansion followed later by left chest expansion. This was consistently observed during each breath. This asynchrony was also noted during auscultation of the lungs when the breath sounds in the left lung were heard only late in inspiration, compared with the right lung where breath sounds were audible throughout inspiration. The phenomenon persisted despite adjusting and testing the depth of insertion of the tracheal tube. Tube adjustments were repeatedly checked with direct laryngoscopy to avoid the risk of the tube exiting the trachea and were stopped when the tube’s cuff started to be seen below the vocal cords.

At this stage, the preoperative chest X-ray was re-checked, and this showed significant tracheal deviation including an abrupt rightwards angulation of the trachea at the T2 vertebral level to an angle of about 20° from the vertical, most likely caused by unfolding of the aortic arch. At this point, we suspected that due to this tracheal deviation, the distal end of the tracheal tube may have been eccentrically located in the tracheal lumen, close to the right tracheal wall. According to the Coanda effect principle, this would result in the jet of gas from the end of the tube adhering to the right tracheal wall, following its contours into the right main bronchus. To examine our assumption, with the cuff deflated we rotated the tube 90° to the left while maintaining the same tube depth. After this adjustment, both lungs became synchronously and equally ventilated by both manual and mechanical ventilation. The 90° rotation was chosen because we thought that if rotation per se did not correct the eccentricity of the tube’s distal end, the 90° would at least move the flow along the anterior wall of the trachea from where it would divide into both lungs at the carina. A pre-extubation chest X-ray was done and showed that the distal end of the tracheal tube was about 4 cm above the carina. Pre-intubation and post-extubation testing of the integrity of the tracheal tube cuff showed a leak-free and symmetrically inflating cuff, so the eccentric position of the tracheal tube tip that may have caused the Coanda effect to occur was assumed to be related to the internal geometry of the trachea in this patient.

In this case, the tip of the tracheal tube was envisaged as a nozzle providing a jet of gas flow close to one side of the wall of a wider diameter tube (the trachea), thus