170e Réunion de la Société Suisse de Neurologie
170. Tagung der Schweizerischen Neurologischen Gesellschaft

Abstracts
Lausanne, 21–23 novembre 2002

Neurologie générale

Syndrome de Horner associé à hémiparésie ataxique contralatérale: un nouveau syndrome thalamique

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Introduction: Un syndrome de Horner associé à une hémiparésie contralatérale est habituellement rencontrée lors de dissection carotidienne ou d'ictus hémisphérique étendu (syndrome télodiencéphalique). Cette présentation peut aussi témoigner une atteinte sousthalamique.

Méthodes: Nous avons considéré les patients du «Lausanne Stroke Registry» avec atteinte thalamique isolée, dont le diagnostic à été confirmé par CT/IRM, incluant les sujets avec syndrome de Horner central.

Résultats: Nous avons retenu 9 patients. La lésion intéressait dans 8 sujets le thalamus antérieur ou paramédian, s'étendant à la région sousthalamique, un patient avait une atteinte du thalamus latéral touchant le thalamus antérieur. Tous montraient une hémiparésie ataxique controlatérale. Les autres signes étaient dysphasie, somnolence, limitation du regard vertical, astérixis, et hémihypesthésie.

Conclusion: L'atteinte principale était représentée par le thalamus antérieur/paramédian, s'étendant dans la région sousthalamique. Le fait que les artères thalamopolaire et thalamo-paramédiane irriguent la partie postérieure de l'hypothalamus peut expliquer l'atteinte des fibres sympatico-exitatrices. Le syndrome caractérisé par l'association alterné d'une triade de Horner avec une hémiparésie ataxique peut donc représenter une manifestation d'une atteinte thalamo-sousthalamique. A notre connaissance, ceci est la première étude soulignant cette entité clinique, dont le diagnostic différentiel inclut la dissection de la carotide interne et le syndrome télo-diencéphalique.

Sidérose superficielle cérébrale

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Nous présentons une patiente de 60 ans souffrant d'une instabilité en station debout et de difficultés à la marche lentement progressives depuis 2 ans. La patiente rapporte également une hypoacousie bilatérale précédée par des acouphènes. Par ailleurs, elle décrit des douleurs abdominales basses avec une irradiation dans les membres inférieurs. Malgré une intervention pour utérus myomateux 15 mois avant l'hospitalisation, les douleurs persistent et se sont accompagnées de troubles sphinctériens par la suite. L'examen neurologique a principalement mis en évidence une surdité bilatérale et une ataxie statique ainsi que des hyperesthésies sévères au niveau du ventre dessous de D10 et au niveau des membres inférieurs.

L'IRM cérébrale en T_2 a montré un hyposignal linéaire entourant le cervelet, certains nerfs crâniens et les sillons corticaux. La zone hypodense est compatible avec des dépôts d'hémosidérine comme on le voit dans une sidérose superficielle cérébrale. La ponction lombaire était hémorragique et par la suite nous avons découvert une tumeur du cône médullaire. Un épendymome est suspecté. Après résection complète de la tumeur par le neurochirurgien, l'histologie confirme ce diagnostique.

Dans notre cas, la sidérose superficielle cérébrale était liée à une hémorragie sous-arachnoïdiennne chronique liée à une épendymome du cône médullaire, tel que cela a déjà été décrit dans quelques cas jusqu'à présent.

The clinical spectrum in restless legs syndrome: a video-presentation

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Restless legs syndrome (RLS) is easily diagnosed when a patient is complaining about unpleasant sensory sensations in the lower extremities appearing mainly in the evening, particularly when lying down, and with great relief while walking around. However, the spectrum of the clinical presentation is not only broad with respect to the type of sensory complaints ranging from tingling or burning to internal pressure, cramps or even pain, but also with respect to the time of day and the involved part of the body. It happens that patients can easily fall asleep but wake up two hours later with severe RLS. An involvement of the arms is reported in up to 30% whereas inclusion of the face was never described and a reason for considering another aetiology. Diagnostic difficulties may primarily arise in the pure motor form of the disease. Less than 10% of RLS patients do not complain about unpleasant sensory symptoms but rather about involuntary movements of the legs or other parts of the body. Periodic leg movements (PLM) in wakefulness and in sleep are often found but not a prerequisite for the diagnosis.

Tagesschläfrigkeit und Fahrtauglichkeit

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An einer exzessiven Tagesschläfrigkeit leiden bis zu 5% einer gesunden Population. Nebst dem banalen Schlafmanko und unregelmässigen Schlafzeiten sind Schichtarbeit, Insomnien, das Schlaf-Apnoe-Syndrom oder funktionelle Hypersomnien die Ursache. Die Folgen sind Leistungsverminderung und Unfälle am Arbeitsplatz und im Strassenverkehr. Der Anteil der Verkehrsunfälle, die auf ein Einschlafen am Steuer zurückgeführt werden, dürfte ebenso hoch sein wie beim Alkohol. Eine Studie in Deutschland hat gezeigt, dass auf den deutschen Autobahnen 24% der tödlichen Unfälle auf ein Einschlafen am Steuer zurückgeführt werden können. Die typischen Merkmale von Verkehrsunfällen beim Einschlafen am Steuer sind zwar bekannt. Diese werden in der Schweiz aber noch viel zu wenig konsequent benützt, was dazu führt, das Problem zu unterschätzen und prophylaktische Massnahmen zu verzögern.

In der Schweiz und auch in anderen europäischen Staaten existieren z.Z. noch keine Richtlinien zur Beurteilung der Fahrtauglichkeit bei exzessiver Tagesschläfrigkeit vergleichbar mit den Richtlinien bei Epilepsie oder Alkoholabusus. Zur Prävention solcher Unfälle im Strassenverkehr gilt es primär, jeden einzelnen Verkehrsteilnehmer besser über die Gefahren der Schläfrigkeit zu informieren; Polizeikorps, Justiz und Behörden zu sensibilisieren, damit konkrete Präventivmassnahmen erfolgen, wie Aufstellen von Warnschildern an gefährlichen Strassenabschnitten, z.B. vor Tunnelbauten, oder Anbringen von gerippten Seitenstreifen. Bei Verdacht auf eine krankhafte Tagesschläfrigkeit soll der Fahrzeuglenker ärztlich weiter abgeklärt werden.

Dramatic recovery from long-lasting Wernicke-Korsakoff's syndrome

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A 66-year-old alcohol-addicted woman was admitted to our clinic for ataxia rehabilitation, three months after total hip replacement. Clinical examination showed, in addition to a severe limb and trunk ataxia, oculomotor abnormalities, a deep confabulatory amnesia, marked apathy, dysarthria and dysphagia. Indeed, history revealed that this clinical picture appeared immediately after surgery, then was complicated by respiratory failure due to repeated aspiration pneumonia and remained unchanged within the following three months. Brain MRI had been normal. Given this history and our findings, we made the diagnosis of Wernicke-Korsakoff's syndrome (WKS) and started treatment with vitamin B_1, multivitamin prescriptions and magnesium, along with adequate diet, and cognitive and physical rehabilitation. Within six months, oculomotor abnormalities, cerebellar signs, dysphagia and apathy completely recovered and, at home discharge, neuropsychological assessment showed only mildly impaired verbal learning with intact recognition. This case demonstrates that a "dramatic recovery" from WKS is still possible even when vitamin therapy is introduced after a great delay. Adequate rehabilitation and medical treatment are warranted even in such an apparently "desperate" case. It is unclear

whether the patient had the potential to recover because of an individually decreased susceptibility to WKS or a not complete consumption of vitamin reserves at a cellular level.

Cerebrospinal *tau* and *Aß42* levels in healthy and demented subjects

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The limitations and many conflicting results have cast serious doubts on the validity of *tau* and *Aß42* levels in cerebrospinal fluid on the biological diagnosis of Alzheimer's disease. We addressed the issue of defining reference limits for cerebrospinal fluid *tau* and *Aß42* in healthy individuals. 105 neurologically intact subjects were enrolled according to strict inclusion criteria, 10 of whom with autopsy confirmation. For *tau,* we found that an upper cut-off value of 616 pg/ml allowed 98% of the subjects to be correctly classified as normal. For *Aß42,* a lower cut-off value of 118 pg/ml allowed a correct classification of 88% of the subjects. The combination of both markers yielded a 95% sensitivity.

To further define reference limits for both *tau* and *Aß42,* we performed a second retrospective study on *tau* and *Aß42* in cerebrospinal fluid of autopsy-proven individuals with various causes of dementia. The potential of this study to correlate levels of *tau* and *Aß42* values with a diagnosis of Alzheimer's disease will be presented.

CSF orexin-A (hypocretin-1) levels in Zurich: the results of the first 100 patients

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Introduction: Recent studies showed a link between low (<200 pg/ml) or undetectable (<10 pg/ml) CSF orexin-A levels and narcolepsy.

Objective: To examine orexin-A levels in narcolepsy and other neurological disorders.

Methods: CSF orexin-A levels of 100 patients with a variety of neurological disorders (22 of whom with narcolepsy) were determined by a highly sensitive radioimmunoassay.

Results: The range of orexin-A levels was <10–1333 pg/ml. The overall mean was 464 (median 383) pg/ml. In 12 narcoleptics, orexin-A levels were undetectable (<10 pg/ml), in 10 other narcoleptics levels were low (<200 pg/ml). In 76 of the remaining 78 patients orexin-A levels were >200 pg/ml. Low CSF orexin-A levels (123 and 168 pg/ml, respectively) were measured in 2 of 3 patients with normal pressure hydrocephalus. High orexin-A levels were observed in patients with Guillain-Barré syndrome (mean 1025 pg/ml, n = 3), Lewy-Body disease (mean 988, n = 4) and acute headache (993 pg/ml, n = 3).

Conclusions: We confirm that low/undetectable CSF orexin-A levels have a high sensitivity and specificity for narcolepsy. The upper limit and the clinical significance of high orexin-A levels remain unknown.

Maladies extrapyramidales

Objective quantification of bradykinesia in Parkinson's disease

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Objective: To objectively quantify the bradykinesia in PD patients with STN-DBS switched ON and OFF and compare them to healthy subjects.

Background: Bradykinesia is a major sign of PD that limits daily life activities. While dyskinesia and tremor have been quantified, no objective quantification of bradykinesia is yet available.

Design/methods: 10 age-matched normal subjects and 10 PD patients, 20 ± 3 months after STN-DBS implantation, were recorded during activities of daily living. PD patients were recorded, with STN-DBS ON and OFF. Portable recording system with 3D gyroscopes measured angular velocity (Mh: degree/sec) and the range of hand rotations (Rh: degree). UPDRS motor subscores were obtained at the same time. Comparisons were performed using correlation analysis and t-test.

Results: Mobility parameters were measured separately for each axis. Roll axis had the best correlation with UPDRS score (Mh: corr = –0.837; p <0.0001; Rh median: corr = –0.827; p <0.0001;

UPDRS subscore 2). For the three axes there was a significant difference between mobility of the hands between healthy subjects and PD patients with STN-DBS switched OFF ($p < 0.01$), with STN-DBS switched ON there was a significant difference only for the roll ($p = 0.05$) and yaw ($p = 0.0025$).

Conclusion: Bradykinesia can be accurately quantified using gyroscopic sensors and portable recording systems using parameters like angular velocity and range of hand rotation.

Objective effects of STN-DBS on gait parameters in Parkinson's disease

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Objective: To quantify different gait parameters using the Physilog®, an ambulatory gait analysis system based on gyroscopes and compare them in PD patients with STN-DBS switched ON and OFF and to healthy subjects.

Introduction: Gait is impaired in PD. Temporal and spatial parameters of gait have already been analysed by accelerometres but gyroscopic quantification has not been reported for PD.

Methods: 10 PD patients, 20 ± 3 months after STN-DBS implantation, were asked to walk 20 metres while carrying the Physilog® twice, with STN-DBS switched ON and OFF. Same protocol was used to measure 10 normal, age-matched subjects. Spatial and temporal parameters for each gait cycle were estimated using six sensors attached to four limbs. Time of the whole gait cycle (GS) (sec), range of movement of the shank (MS), angle of hand movement (HM) in 3D, angular velocity of the shank (VS) (degree/sec), stance (St), swing (Sw), and double support (DS) were analysed.

Results: Parkinsonians were different from controls for all gait parameters: GS, DS and St increase, MS, HM and VS decrease (all $p < 0.01$). Turning STN-DBS ON, significantly improved all parameters but not up to normal values.

Conclusion: Physilog® system provided a simple way of gait analysis in PD subjects. STN-DBS significantly improves the gait of PD patients. However, independently of their state, PD patients keep significant differences in comparison to controls.

Results and adverse events in 53 consecutive PD patients treated by STN-DBS

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Introduction: Most available studies on STN-DBS in PD are limited to one year or less and based on a restricted number of patients. We studied the effect of STN-DBS in 53 consecutive PD patients with follow-up up to 3 years.

Methods: 53 consecutive PD patients with motor fluctuations and dyskinesia underwent bilateral STN-DBS implantation under stereotactic guidance, microrecording, and clinical control. Medications were stopped 12 hours before implantation, and reintroduced only if needed. UPDRS I to IV were performed at baseline, 3, 6, 12, 24, 36 months; 34 patients were assessed with DBS switched off.

Results: 51 patients had regular follow-up. At last follow-up, 22 patients had no medication and 29 had it reduced by 56%. There was no difference ($p > 0.2$) between these groups for age, duration of disease or preoperative medication. The group improved activity of daily living (ADL) ($p < 0.01$) and fluctuations and dyskinesia almost disappeared ($p < 0.01$). UPDRS III "off" was reduced by 38% ($p < 0.001$). Switching DBS on/off modifies the UPDRS III "off medication" by 57% ($p < 0.001$). UPDRS III postoperative improvement correlated highly with preoperative results of 200/50 levodopa/carbidopa challenges. Complications comprised: one air embolus, one seizure, 7 transient confusional states, 4 leads repositioning, one connector wound dehiscence, one infection, neither haemorrhage nor any permanent neurological deficit.

Conclusions: STN DBS led to (a) almost complete disappearance of dyskinesia and fluctuations, (b) stabilisation of ADL and motor improvements similar to baseline "on", (c) 75% reduction of medication. Complications were limited without permanent neurological deficit. These results were sustained up to 3 years.

Value of routine Holter screening for detection of paroxysmal atrial fibrillation (PAF) in patients with cerebral ischaemic attacks (CIA)

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Between 01/00 and 06/02 308 patients had routine Holter post CIA (17.1% of Holter indications at our institution), age 35 to 92, median 68. PAF was diagnosed in 9 patients (2.9%): in 2 patients anticoagulation (AC) was refused/contraindicated; 2 patients were on AC and PAF was already documented on 12-lead-ECG; one patient had carotic stenosis >90% as additional risk factor for CIA; one patient had PAF, but was on AC for basilar thrombosis; one had PAF before and was on ASS; in only 2 patients, new diagnosis of PAF was established. These 3 patients were put on AC after Holter.

Thus in 3/308 patients (1%) with CIA, routine Holter resulted in DRM. Given the costs of 300 CHF per Holter, overall expenses were 92 400 CHF that is 10 200 CHF/PAF or 30 800 CHF/drug regimen modification (DRM). The "number-needed-to-investigate" is 103 Holter for one DRM.

Conclusions: Our data demonstrate that PAF in CIA patients: (a) has a low incidence, (b) if diagnosed, hardly leads to a drug modification and (c) has high per-case costs. Therefore routine Holter screening is not recommended in patients with CIA.

Aggravation of ischaemic injury by *tissue*-plasminogen activator (t-PA): experimental evidence and underlying mechanisms

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Aims: Thrombolysis is currently the only efficacious treatment in acute ischaemic stroke. Recently, concerns were raised that the thrombolytic *tissue*-plasminogen activator (t-PA) may have detrimental side effects. Since such effects may be of major clinical relevancy, we conducted the present studies.

Methods: Male C57BL/6j mice were submitted to middle cerebral artery thread occlusions. At defined time points before, during and after ischaemia, t-PA was *i.v.* administered at various doses (0.2–10 mg/kg). Brain perfusion was measured by laser Doppler flowmetry (LDF) and cerebral blood flow (CBF) autoradiography. Brain injury was assessed histochemically 24 hours after reperfusion.

Results and conclusions: Our results confirm previous findings that *i.v.* t-PA increases brain injury in non-embolic focal ischaemia. In addition, our data show that t-PA treatment is followed by a secondary hypoperfusion. Both the decrease in blood flow and increase in injury were efficaciously prevented by heparin (200 IU/kg). Heparin alone, on the other hand, neither influenced brain perfusion nor tissue damage. Besides, the NMDA receptor antagonist MK-801 (0.2 mg/kg) attenuated t-PA-mediated injury, but did not have an effect when given alone at this low dose. The effects of MK-801 were independent of haemodynamic changes. The present studies confirm that the beneficial effects of t-PA may be compromised by unfavourable changes and describe possible mechanisms involved. Our data point towards the necessity to develop add-on treatments, preventing both the haemodynamic deficits and injury increase. Concerted efforts will be required in this field in the future.

Qualité de vie après hémorragie sous-arachnoïdienne anévrysmale chez les patients âgés

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Objectif: Etudier l'impact d'une hémorragie sous-arachnoïdienne anévrysmale (HSA-a) sur la qualité de vie des patients âgés.

Méthode: Etude rétrospective de 33 patients âgés de 70 ans et plus au moment de l'HSA-a.

Résultats: 19/33 patients, tous en bon grade clinique, ont bénéficié de l'exclusion de l'anévrisme. Parmi ceux-ci, 26% (5/19) sont décédés pour des causes liées à l'HSA et 53% (10/19) ont pu regagner leur domicile et vivre de façon indépendante (mRS 0–3). 40% des survivants n'ont ressenti aucun changement de leur qualité de vie, 30% une diminution modérée et 30% une diminution sévère. 7 patients en bon grade clinique initial n'ont pas été traités pour des raisons autres que

l'HSA-a (co-morbidité, etc.). 57% (4/7) sont décédés, alors que les 43% restants ont pu retrouver une vie indépendante (mRS 0–3). Aucun patient avec un grade clinique initial WFNS ≥3 n'a pu regagner son indépendance.

Conclusions: Le score clinique initial (WFNS) reste le meilleur outil pronostic. Une séparation claire apparaît entre un score de 2 et 3 (GCS 13–14 sans ou avec déficit neurologique) suggérant que chez les patients âgés, un déficit neurologique secondaire à l'HSA exclu la possibilité de retrouver une vie indépendante.

Neurologie du comportement

Thérapie des troubles morphosyntaxiques d'un patient avec une aphasie fluente: une étude de cas

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L'aphasie de Wernicke se caractérise par des difficultés syntaxiques et morphosyntaxiques, regroupées sous le terme de paragrammatisme.

L'objectif de cette étude est d'examiner si chez un patient avec une aphasie fluente les difficultés morphosyntaxiques à l'écrit peuvent être diminuées par une thérapie.

Le patient a souffert d'une ischémie de la branche inférieure de l'artère cérébrale moyenne gauche. 14 mois après, une thérapie est entamée, 16 séances en 11 semaines au total. La tâche consiste à décliner correctement les constituants d'une phrase donnée.

Dans l'épreuve contrôle, on constate une diminution des erreurs de flexions (test de tendance monotone, coefficient de corrélation de Spearman Rho = –0,609; p = 0,0432. Les erreurs lexicales de la classe ouverte et de la classe fermée (Butterworth, Howard 1987) diminuent significativement (Rho = –0,537; p = 0,0162 respectivement Rho = –0,672; p = 0,0027). Par la thérapie, les mécanismes morphosyntaxiques se sont améliorés dans la production écrite de phrases. Cette amélioration se généralise à la production de textes. Les effets thérapeutiques sont spécifiques, significatifs et stables.

Spatial representation of numbers: role of right parietal lobe

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Numerical abilities may in part rely on a representation of quantities where magnitude is organised by spatial proximity, along a "mental number line" extending from left to right. We show that unilateral spatial neglect after right hemisphere damage may impair such mental representation of numbers. For example, when asked to judge whether a single number shown at fixation is smaller or larger than "5", patients with neglect are selectively slower to respond to "4", but when asked to compare numbers to "7" they are selectively slower to respond to "6". This demonstrates a deficit to form a representation for numbers located on the left of a reference point along the "mental number line" and suggests a role of the right parietal lobe in representing numerical quantities.

When far space is lost in mind

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Focal unilateral brain damage can induce an impaired exploration of contralesional space. This phenomenon, known as hemispatial neglect, has been described for perceptual space as well as representational space. Moreover, perceptual hemispatial neglect can occur for different spatial dimensions such as within (near) or beyond (far) hand-reaching distance. Although predicted, the same double dissociation has not been found in the representational domain, yet. Here, we report a patient with a selective spatial deficit for far but not near representational space subsequent to a small haemorrhage of the right lateral temporal lobe with an oedema involving the lateral temporo-occipital areas. This patient did not show a perceptual neglect in near or far space. However, she systematically omitted elements on the left hemispace with regard to her imagined self-position, but only in far and not near representational space. These findings, consistent with neurophysiological and neuropsychological studies, confirm (1) the

existence of distinct neural mechanisms and (2) different brain areas that are relevant for the coding of near and far representational space. The modular nature of human visual space lets us predict the case of an isolated representational neglect restricted to near space.

Neurophysiologie

Extinction of an epileptic mirror focus during functional hemispherectomy

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Functional hemispherectomy and hemispherotomy consist of physiologically isolating one hemisphere without interference with the vascularisation. This procedure is indicated in refractive epilepsy when the origin of the epileptic seizures is identified from the whole hemisphere and hereby the hemisphere can be considered non functional. The neurological deficit (such as hemiparesis and hemianopsia) is only slightly or not modified by the operation. In cases with cognitive regression due to the refractive epilepsy the development can be improved. During a hemisphectomy in the peri-insulair region with neuromonitoring ipsilateral by corticography and controlateral EEG, we observed the extinction of a mirror focus after resection of the amygdala, confirmed in the following EEG postoperatively. This observation strengthens the importance of the interhemispheric connections being the origin of mirror focus and the efficacy of surgical treatment by hemispherotomy in refractive epilepsy.

Seizure-like phenomena and propofol – a systematic review

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Data on seizure-like phenomena (SLP) in patients receiving propofol were systematically reviewed. Reports had to provide detailed information on SLP in individual patients who received propofol. Phenomena were classified according to the time point of their occurrence during anaesthesia or sedation (induction, maintenance, emergence, delayed [>30 minutes after emergence]) and their clinical presentation (generalised tonic-clonic seizures, focal motor seizures, events presented as increased tonus with twitching and rhythmic movements not perceived as generalised tonic-clonic seizures, opisthotonus, involuntary movements). In 70 patients without epilepsy SLP happened during induction in 24 (34%), during maintenance in 2 (3%), during emergence in 28 (40%), and delayed in 16 (23%). Most frequent clinical presentations of SLP were generalised tonic-clonic seizures in 30 patients (43%), events presented as increased tonus with twitching and rhythmic movements not perceived as generalised tonic-clonic seizures in 20 (36%), and involuntary movements in 11 (16%). In 11 patients with epilepsy, 7 (64%) had a generalised tonic-clonic seizure during emergence. Of all 81 patients, 26 (32%) only had an EEG, and 12 (15%) only a neurological consultation. SLP may happen in patients with and without epilepsy receiving propofol. The time point of the occurrence of SLP suggests that a change in cerebral concentration of propofol may be causal. To confirm this hypothesis, to estimate the prevalence of propofol-related SLP, and to identify patients at risk, data of higher quality are needed.

Epileptic mirror focus: neuromonitoring (EEG and corticography and dipole analysis) in 4 cases with functional hemispherectomy

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We studied and documented multifocal epilepsy, with EEG, dipole analysis and continuous neuromonitoring (EEG and corticography), in 4 children while they underwent a functional hemispherotomy for medically intractable epilepsy. These analyses enabled us to confirm the 3-D localisation of the epileptic foci as well as to visualise eventual mirror foci. Ipsilateral corticography, during the different phases of the intervention, as well as a continuous EEG, demonstrated the neurophysiological efficacy of hemispherotomy in the case of resistant epilepsy. This study also helps to better understand the electrogenesis and pathophysiology of mirror epileptic foci.

Hypothesis for propofol-induced burst-suppression: explained by vascular action or disconnection?

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The origin of burst-suppression (BS) induced by proprofolium remains debated. During 3 interventions of functional hemispherectomy the authors registered the electrical activity on line in both hemispheres before and after cortical isolation. After isolation of one hemisphere, we studied the participation of the vascular proprofolium-induced electrical activity. The lesioned tissue showed a more rapidly appearing pattern of BS. We concluded that the mechanism of action of proprofolium with induced cortical BS pattern in chronical lesions is a result of several actions; firstly the state of the cortical tissue, secondly the disconnection of ascending thalamo-cortical pathways as well as a direct cortico-cortical level of modulation.