pared to normal C57Bl mice and (Na⁺, K⁺)-ATPase is altered in its phosphorylation regulation by K⁺ ions and phenytoin (which was found to activate glial (Na⁺, K⁺)-ATPase). Ca²⁺-ATPase is deficient in synaptosomal and astrocytic membranes of cortex and brainstem of DBA/2 mice. Glutamate uptake is increased in cultured glial cells from audiogenic mice. An increase of glutamate content was also found in the cortex of epileptic mice, with no change of GABA levels. That increase could result from a metabolic shunt between polyamines and GABA in DBA/2 mice. Seizure spread from the brainstem to the cortex could then result from an imbalance between protective (carbonic anhydrase, glutamate uptake) and deficient ([(Na⁺, K⁺)-ATPase, Ca²⁺-ATPase] glial systems controlling neuronal excitability.

9 Neuron-glia relationships in epilepsy. II. Experimental and human partial seizures. T. Grisar, D. Guillouil, Liège, Belgium; M. Heeren-Bureau, Los Angeles, CA, USA

Astrogliosis is often associated with focal epilepsy in animals as well as in humans. The molecular contribution of glial cells in partial epilepsy has been investigated in our laboratory over the past ten years. We measured enzymatic specific activities and initial velocities of bulk isolated glial and synaptosomal fractions. We examined the phosphorylation level of purified neuronal and glial catalytic subunits of ([Na⁺, K⁺]-ATPase and studied its regulation by K⁺ ions and phenytoin. Our results indicate that glutamate (Na⁺, K⁺)-ATPase is specifically altered in the epileptogenic cortex of cats with a creative lesion and human with intractable partial seizures. That alteration of the glial enzyme could contribute to seizure spread and ictal transformation, through an impairment of extracellular K⁺ regulation. Monoconal antibodies against the catalytic subunits of the enzyme and sequencing studies are presently done in normal and epileptic human brain, in order to better define the defect of the glial enzyme. We will review those results, stressing the importance of neuron-glia relationships in the molecular basis of partial epilepsy and in relation to the in vivo PET metabolic studies.

10 Open and double-blind study of gamma-vinyl GABA in chronic epilepsy. H. Ring, A. Heller, E.H. Reynolds, London, UK

Gamma-vinyl GABA (Vigabatrin) is a selective, enzyme-activated, irreversible inhibitor of GABA transaminase. Recent studies have demonstrated the efficacy of this compound in patients receiving multiple concurrent antiepileptic drugs. The aim of this study was to investigate the efficacy and toxicity of Vigabatrin in patients receiving only one other antiepileptic drug. Following a 2 month baseline period of observation 31 patients aged 18 to 65 experiencing at least 4 partial and/or generalised seizures per month entered a 2 month open phase of add-on therapy with Vigabatrin. 19 patients who showed at least a 50% fall in seizure frequency in the open phase were then randomised and blindly allocated either to continue Vigabatrin or to substitute placebo for the active drug for a further 2 months. In the open phase overall there was a 58% fall in seizure frequency. In the double blind phase the active drug was associated with a continuing 56% fall in seizure frequency, while the placebo was associated with a 22% increase in seizure frequency compared with baseline. In the open phase 12 patients were withdrawn, 6 because of less than 50% fall in seizure frequency and 6 because of side effects, including headache, drowsiness, and depression. Vigabatrin is a valuable new drug.

11 Visual (VEP), brainstem auditory (BAEP) and somatosensory (SEP) evoked potentials during long-term vigabatrin therapy. A. Tartara, V. Così, R. Callicico, C.A. Galimberti, R. Manni, E. Perucca, A. Ludic, Pavia, Italy; J. Münford, Strasbourg, France

Vigabatrin (VGB) is a highly effective new antiepileptic drug which is currently undergoing extensive clinical testing. Concern about the safety of the drug has been expressed, based on the fact that in laboratory animals relatively large doses can induce neurotoxic histological changes. Since these changes, at least in dogs, are associated with a slowing of the central conduction time of SEPs, we considered appropriate to monitor prospectively evoked potentials in 18 epileptic patients (age 19–59 years) at repeated intervals before and during long-term add-on treatment with VGB (2–4/day). After 6 to 36 months (median 18 months) of continuous VGB therapy, no changes were observed in cortical latency and amplitude of SEPs and in I–II, III–IV and I–V interpeak latencies of BAEPs. VEPs P-100 latencies tended to decrease during VGB therapy (becoming closer to values found in normal controls) while P-100 amplitude was unchanged. Serum levels of associated anticonvulsants (usually carbamazepine and/or phenobarbital) were not significantly affected by VGB. When interpreted within the context of animal finding and human tolerability data, these results provide indirect evidence against the occurrence of neurotoxic effects during VGB therapy in epileptic patients.

12 Epileptic activity following transient cerebral ischemia in mongolian gerbil: an electrophysiological and morphological study. M. G. Marzani, G. Santoni, G. Sancesario, R. Massa, G. Bernardi, Rome, Italy

Changes of cortical and hippocampal electrical activity before, during and after transient 10-min telencephalic ischemia induced by bilateral occlusion of common carotid arteries was studied in 25 mongolian gerbils. The EEG from frontal cortex and hippocampus was bilaterally recorded through chronically implanted electrodes. Epileptic activity was quantified by a computerized program which allowed automatic spike detection and monitored its temporal and spatial distribution. Recording sessions were performed before, during carotid clamping and in the following seven days, every 12 h for periods of 3h continuously. The EEG activity was found to become flat within 20–30 sec from the onset of ischemia and it began to reappear between 20–30 min after recirculation reaching control values in 16–24 h. Epileptic activity, associated with clinical motor signs, appeared during carotid occlusion while immediately after only an interictal activity persisted with a maximum peak after 18–36 h from clamping and subsequently slowly decreased, disappearing between the 6th and 7th day. Morphological evidence showed alterations consisting of a selective necrosis of CA1 hippocampal neurons which started approximately 72 h after clamping and was completed on the 6th–7th day. These results show that transient cerebral ischemia induces an alteration of neuronal excitability, probably due to an abnormal release of excitatory transmitters and an ionic equilibrium disturbance, followed by neuronal death.

Oral session 17: Infection of the nervous system

1 AIDS in children: neurological abnormalities in a series of 87 infants. M. Tardieu, S. Blanche, C. Rouzioux, C. Griscelli, Paris, France

Eighty-seven HIV-infected children (82 cases of maternalfoetal transmission, 5 of post perinatal transfusion) have been prospectively followed. Thirty three patients (38%) expressed neurological abnormalities consisting in intellectual and motor dysfunctions (respectively 80 and 83% of the 33 patients) and in a bucco-lingual dyspraxia (40% of cases). The neurological alterations were correlated in intensity and frequency with: a) the intensity of the immunological dysfunc
tion; b) an early onset of the disease; c) the mode of transmission of the virus: neurological alterations were more frequent among children infected by blood transfusion or whose mothers were IV-drug users than among African or Caribbean patients. Abnormalities on CT scans were found in only 37.5% of the patients with neurological alteration.
The presence of viral specific antigens and antibodies was determined in the blood and CSF of 36 patients both with and without CNS disease. Viral antigens was isolated from the CSF of only 3 patients all having a rapidly evolving CNS disease. Viral antibodies were isolated in all the CSF samples but nine. A low or negative antibody titer in the CSF has been observed in 10 patients with a poor neurological outcome. Forty patients are currently treated with AZT. The results of a pilot study indicate that neurological signs could be improved by AZT treatment.

2
Neuropathological changes induced by cytomegalovirus in AIDS. Study of 14 cases. F. Labrousse, L. Matthiessen, C. Fontaine, C. Marche, C. Vedrenne, Paris, France

Fourteen neuropathological observations of cytomegalovirus (CMV) infection of the nervous system (NS) collected among 75 autopsy cases in AIDs, are reported. The study group consisted of: 13 males (9 homosexuals, 3 bisexuals, 1 with no known risk) and one female (no known risk); 13 Caucasians and one African; the mean age was 42 and the mean duration of AIDS 14 months. Various non specific neurological disorders were noticed: confusion, agitation, focal deficit, cerebellar ataxia and in one case peripheral neuropathy. The morphological diagnosis of an acute CMV infection was based on the finding of typical inclusion-bearing cells and on the immunohistochemical demonstration of CMV antigen. Four cases were studied by electron microscopy. The basic histopathological changes (often mixed in the same case) were: necrotizing ventriculo-encephalitis (10), focal parenchymal necrosis (8), isolated inclusion-bearing cells (6), granular ependymitis (2) and encephalitis and myelo-meningo-radulcellitis (1).

Microglial nodules and diffuse rod cells were observed in 10 cases out of 14. However, identical changes were present in other patients without histological evidence of CMV infection, but presenting toxoplamosis and/or HIV encephalitis. This lead us to think that microglial reaction alone is not CMV specific and that CMV lesions can be classified into the five categories previously described.

3
Light chain type composition of CSF and sera oligoclonal IgG bands in asymptomatic HIV seropositive (AHS) subjects and AIDS patients with central nervous system (CNS) involvement. L. M. E. Grimaldi, A. Castagna, D. Maimone, R. Pristera, A. Lazzarin, R. P. Roos, Chicago, IL, USA; Milano and Bolzano, Italy

One of the regulatory proteins of the HIV tat gene, NF-KB, also controls kappa chain production in activated B lymphocytes (Nature 326:711, 1987). An abnormal kappa chain production may contribute to the dysregulated humoral immune response seen in HIV infection. To investigate this issue, we quantitated IgG light chains by ELISA and delineated light chain composition of oligoclonal bands by isoelectric focusing (IEF) followed by immunofixation (IF) with anti-light chain antibody in specimens from HIV infected patients. Samples included 3 sera and 5 CSF from 8 adult AIDS patients with neurological involvement and 4 sera and 3 CSF from 4 adult AHS subjects in whom we had previously shown oligoclonal IgG bands. Kappa/lambda ratio was elevated in the majority of the samples. Five of eight AIDS and 4/7 AHS samples had all IgG bands of kappa type, while only 2 AIDS and none of the AHS samples had IgG bands that were only lambda. Kappa chains predominance was seen in 2 CSF and one serum from AHS subjects while lambda chains predominated in only one AIDS serum. One free kappa chain band was seen in one AIDS CSF. Lambda chain predominance and a relatively low kappa/lambda ratio correlated with the presence of cryptococcal CNS infection. However, an additional 15 months old patient with connalal AIDS and no evidence of cryptococcal CNS infection had a CSF IEF banding pattern suggestive of an IgG lambda monoclonal gammopathy. In conclusion, our finding of an abnormally elevated kappa/lambda ratio and of oligoclonal IgG bands with kappa light chain predominance suggests that NF-KB may play a role in the pathogenesis of humoral immune response abnormalities in HIV infection.

4
Selective neurotransmitter changes during persistent viral infections of CNS cells. O. Boesphhig, S. Guerroui, M. Tardieu, Le Kremlin-Bicêtre, France

Patients suffering from chronic encephalitis have signs of severe CNS impairment despite the fact that few or no intra CNS cytopathic lesions are observed, suggesting alterations of specific CNS cells metabolic activities during the persistent viral infection. We analyzed, in vitro, metabolic activities related to neurotransmission on neurons and astrocytes during a persistent coronavirus (MHV3) infection. Cultured cortical neurons replicate the virus but are morphologically unaltered for a 7 day period postinfection, whereas astrocytes appear morphologically resistant but chronically infected (J Virol 1986) 60, 2:574–582. Virus-infected and control cultures were similar for protein and DNA contents, 3H thymidine incorporation and intracytoplasmic enzyme activities specific for neurons (choline acetyl transferase, glutamic acid decarboxylase) or for astrocytes (glutamine synthetase). In sharp contrast the maximum velocity of the neuronal 3H-GABA uptake inhibited by DABA were decreased in infected cultures as early as the fourth day p.i., whereas another neuronal specific uptake (3H-choline uptake inhibited by hemicholinium, 3H methyl glucose) or neuronal specific membrane receptor activity (3H methyl clonazepam binding) were preserved. On infected astrocytes the binding of the non neuronal cell specific benzodiazepine (3H-RO5-4846) revealed in increase of the dissociation constant of the high-affinity site (Kd 3.6 versus 1.5) without alteration of the maximum binding; no alteration of 3H methyl glucose uptake was observed. This in vitro model of virus induced selective membrane alterations afford an approach of the in vivo consequences of chronic virus infection of the CNS.

5
Toxoplasmic encephalitis of immunocompetent and nude mice: distribution of toxoplasma antigen, inflammatory cells, and expression of MHC-determined antigen. G. Schwendemann, D. Schütler, Essen; J. Löhler, Hamburg; H. Hof, Würzburg, Federal Republic of Germany

Toxoplasmic encephalitis (TE) due to reactivation of latent infection with toxoplasma gondii occurs in patients who are immunodeficient due to various pathological conditions, particularly in AIDS. Comparably, in a mouse model of chronic toxoplasma infection, depletion of CD4+ T cells leads to toxoplasma reactivation with a lethal TE, whereas in the acute toxoplasma infection death of mice depleted of CD4+ T cells results from overwhelming systemic infection [Vollmer et al. (1987) J Immunol 138: 3737]. To elucidate the pathogenic mechanisms involved in TE further we infected NMRI mice orally with the DX strain of toxoplasma gondii, and investigated the CNS histologically and immunocytochemically at various days post infection up to 4 weeks. Toxoplasma antigen was scattered throughout the brain parenchyma with formation of cysts and glial nodules. Infiltrating lymphocytes consisted mostly of CD8+ T cells. CD4+ T cells represented a minor subgroup, infiltrating the meninges and brain tissue without a clear conjunction with toxoplasma antigen or cysts. Concurrently there was a differential pattern of the expression of MHC class I and class II antigen in the various CNS cell types. In contrast, nude mice died about 3 weeks post infection of a necrotizing encephalitis due to overwhelming free and encysted toxoplasma. The findings may further elucidate the immune mechanisms in TE. Toxoplasma has to be added to those agents apt to induce MHC-determined antigens in CNS.

6
Early diagnosis of herpes simplex virus encephalitis (HSVE) by an isoelectric focusing (IEF)-HSV-gB overlay technique. L. M. E. Grimaldi, R. P. Roos, R. Manservigi, P. G. Spear, F. D. Lakeman, R. J. Whitley, Chicago, IL, Birmingham, AL, USA

In order to establish an early diagnostic test for HSVE, we used IEF and an IEF-overlay technique with radiolabelled HSV gB to study...
sera and 12 cerebrospinal fluids (CSF) from 12 patients with presumed (6) or biopsy proven (6) HSVE. Blood-brain-barrier (BBB) damage and increased intra-BBB IgG synthesis were detected in 5/7 HSVE patients. CSF oligoclonal bands were found in 6/11 HSVE patients. By using an IEF-HSV type 1-gB overlay technique we could confirm a clinical diagnosis of HSVE within the first 3–5 days of disease. Specifically, we detected anti-gB antibody in a broad pI distribution in 44 sera and in 46 CSF obtained 3–5 days after disease onset and in all later samples. A hematogenous origin for most of the CSF anti-body was suggested because anti-gB antibody appeared in serum before matched CSF with a similar IEF pattern. Additional local CNS production of anti-gB antibody was suggested, in some cases, because of more prominent anti-gB antibody in CSF than in the matched sera, and because some CSF oligoclonal bands had anti-gB antibody activity. Of 6 CSF from multiple sclerosis (MS) patients, CSF from only one who had cutaneous HSV type 2 lesions temporally associated with MS had evidence of anti-gB antibody production. In this case, the anti-gB antibody activity did not correspond in pI location to CSF oligoclonal IgG bands. The IEF-HSV-gB overlay technique may be a useful diagnostic test in cases of HSVE, and a valuable research tool for studying qualitative aspects of the HSV humoral immune response.

7 Prognosis of viral encephalitis. T. Büttner, W. Dorndorf, Giessen, Federal Republic of Germany

Clinical data, analysis of cerebrospinal fluid (CSF) and CT of 53 patients suffering from acute or subacute viral encephalitis were evaluated retrospectively. 16 patients (30%) died due to the disease. Complete restitution was observed in 73% of survivors; the disease in 16% resulted in severe neurological deficit. Symptoms observed by neurological examination 1 to 5 years after acute encephalitis were psychosyndrome (6), hemiparesis (4), aphasia (2), ataxia (2), dysarthria (2), disorders of eye movement (2), extrapyramidal motor disorder (2) and hemianopia (1). Disturbance of consciousness and severe focal neurological deficit worsened prognosis (towards death and poor functional recovery). Patients with a fatal course of the disease had much higher CSF lactate concentrations than survivors. Autochthonous production of IgG-antibodies, observed predominantly during the second and third week after onset of symptoms, correlated with an unfavourable functional prognosis. Prognosis of non-herpes-simplex encephalitis was worse in case 3 with pathological CCT-findings.

8 SSPE in association with pregnancy. I. Wirgin, I. Steiner, D. Kidron, T. Brenner, Jerusalem; S. Udem, New York, NY, USA; B. Rager, Beer-Sheva, O. Abramsky, Jerusalem, Israel

Certain predisposing factors are associated with occurrence of subacute sclerosing panencephalitis (SSPE), and might be of pathogenetic importance. These include measles infection in early childhood, ethnic origin and an immune compromised state (i.e., agammaglobulinemia). Disease usually presents within the first decade and is of a subacute course, culminating in death within 1–2 years. We present two women who developed SSPE during pregnancy. One aged 27 was in the second trimester. The other, aged 18 developed symptoms in the immediate postpartum period. In both patients the disease had a fulminant clinical course, reaching stage IV within two weeks. Both gave birth to apparently normal infants. Although the pathogenesis of SSPE is unknown, it is postulated that a certain immunologic setting is required. Pregnancy induces a natural immune compromised state, as well as endocrine changes. It seems that pregnancy played a role in the appearance of disease in a relatively older age, and the fulminant course of SSPE in our patients.

9 Neuroradiological and immunological observations in subacute sclerosing panencephalitis: A longitudinal study of four cases. A. Alfaro, M. A. Antolin, J. Benedicto, A. Cervelló, D. G. Granda, M. Peñarrocha, Valencia, Spain

Subacute sclerosing panencephalitis (SSPE) is a rare disease of children and young adults, due to persistent infection of the CNS by measles virus. In the last 7 years, we have studied 4 patients (1 male and 3 females, aged 8 to 14 years at onset of symptoms) with characteristic EEG findings and variable degrees of intellectual impairment, visual loss, rigidity and myoclonus. In spite of continuous treatment with Inosiplex and long periods of clinical stabilization, serial CT scans showed a steady progressive cerebral atrophy in all 4 cases. Serum and CSF specific IgG and IgM measles antibody titers were measured by an enzyme-linked immunosorbent assay (ELISA) in 27 frozen samples (11 CSF and 16 sera) taken from the 4 patients at different stages of the disease. Rheumatoid factor was absorbed prior to the search for IgM. We found high levels of IgG in serum and CSF, with abnormal (CSF/serum IgG): (CSF/serum albumin) indexes, indicating an intrathecal synthesis of IgG. Oligoclonal IgG bands were present in all occasions. No IgM was detected in serum samples. On the contrary, high cerebrospinal fluid levels of IgM were observed in all samples, with a marked increase during the later phases of disease in 3 patients.

These results indicate that a persistent viral infection does exist in SSPE, with an intrathecal immune response involving the production of IgM and IgG. An intense and prolonged IgM response within the CNS seems to be a characteristic feature of the disease.

10 Cysticercosis in Spain. E. García-Albea Ristol, Madrid, Spain

Cysticercosis is not rare in Spain. We analyzed a series of 52 cases seen in our hospital from 1974 to 1987, in emigrants who were mainly from the western part of the Iberian peninsula. In accordance with the clinical and pathogenic findings that determined the clinical situation, 59.6% of the patients were classified as having parenchymal forms with several variants: "miliary" with active cysts (1.9%), "calified miliary" (42.3%), apoplexy-like (1.9%), tumoral (7.7%) and "encephalitic" (5.8%). Predominantly meningeal colonization (25%) originated an arachnoidal reaction that was either active (15.3%), with CSF pleocytosis (13.5%), or inactive (1.9%), with space-occupying lesions (tumoral form 9.6%). Larvae in the ventricular system (13.5%) produced ventriculitis with hydrocephalus (5.8%) or "tumoral" obstruction by the cyst (7.7%). Ocular (1.9%) and spinal (1.9%) cysticercosis were exceptional. The incidence of cysticercosis is declining in Spain, but its diagnostic identification is improving. Brain scan and CSF study enable classification of this highly polymorphic disease.

11 Neurobrucellosis: clinical presentation and outcome. S. M. Al Deeb, B. Yakub, A. J. Jabbar, A. Ghani, H. S. Sharif, Riyadh, Kingdom of Saudi Arabia

Brucellosis is endemic in Saudi Arabia. 12 patients with neurobrucellosis were seen at the Riyadh Military Hospital in the last 2 years. The clinical presentations were: acute meningoencephalitis in 3 patients, papilloedema with increased intracranial pressure and without focal neurological signs in 3 patients, and subacute disseminated encephalo-myelitis in 3 patients. The remaining 4 patients had progressive ataxic quadriaparesis, cerebral infarction, polyradiculitis and mononeuritis respectively. Bilateral cochlear VIII nerve palsy with profound or complete hearing loss was a common feature seen in 50% of patients. The CSF was abnormal in all cases with lymphocyte pleocytosis, high protein and low or normal glucose. The CSF brucella agglutination titre was significantly raised in all cases but brucella organisms were isolated from the CSF in only 10% of patients. The neuro-radiological findings, as demonstrated by MRI and/or CT were: brain oedema, more marked in the white matter, deep low density lesions probably due to occlusion of the penetrating arteries, and multiple white matter
periventricular high intensity lesions indicating a possible central demyelination. The neurophysiological findings in the polyradiculitides were consistent with peripheral demyelination. Other details including treatment and outcome will be presented.

12
Transverse myelitis due to infection with mycoplasma. S. Tshaousho, R. Lang, Y. Smorzie, E. Kott, Kfar-Saba, Israel

Mycoplasma pneumonia has been reported to cause transverse myelitis with poor recovery prognosis. A 12½-year-old girl was admitted to the pulmonary ward due to cough, headache, severe pain in her right leg then left leg, which developed 4 days prior to admission. On the day of admission she complained about numbness in both legs which soon became paraplegic with retention of urine and feces. An atelectatic segment of upper right lobe was seen on X-rays, but disappeared after 48h. Mycoplasma was suspected. The patient was transferred to the neurological ward already paraplegic in both legs which which might render them more susceptible to damage. 3) Further studies have shown that the blood vessel density in the nerve fibre layer between the fovea and the optic disc is lower than in the neighbouring arcuate bundles. We suggest that this may provide a more likely explanation for the early appearance of a centrocecal scotoma in tobacco-alcohol ambyopia.

3
Smooth eye movements that anticipate motion. D. Boman, J.R. Hotson, Stanford and SanJose, CA, USA

Anticipatory slow eye movements (ASEM) occur prior to expected ramp or step target motion. These predictive events are restricted by visual fixation and are enhanced by extinction of a visual target prior to expected ramp motion. A percept of motion, whether produced by long or short range apparent motion stimuli, is a main stimulus for ASEM. The detection and velocity of ASEM is dependent on the clarity and velocity of real and apparent motion. Degrading the strength of a random dot stimulus motion percept reduces the velocity of preceding ASEM. ASEM also have a time course and velocity similar to the smooth predictive responses that occur during pursuit of a direction changing ramp target. Both responses move the fovea off target and in the direction of anticipated, future target motion. ASEM and smooth pursuit can be studied and compared in patients with focal neurological disorders. (Supported by NEI Grant EY03387).

4
Disturbances of saccade generation in Parkinson's disease. C. Crawford, L. Henderson, London, UK

Neurophysiological recordings in the substantia nigra, pars reticulata, of the alert monkey have revealed modulation of neuronal activity in relation to saccades (Hikosaka and Wurtz 1983). In Parkinson's disease (PD) the dopamine deficiency in the basal ganglia results in abnormal somatic motor function. However, randomly elicited saccadic eye movements in PD patients show relatively minor abnormalities (Gibson et al. 1987).

Saccadic eye movements are made in response to a wide variety of behavioural conditions. In this study we have examined the possibility that the basal ganglia are involved in saccades generated in only certain of these conditions.

Saccadic eye movements of 7 PD patients were examined in a variety of paradigms, including random, remembered, predictable and non-visually directed, and compared with 7 age matched controls. Saccades were analysed for latency peak velocity and accuracy. PDs made normal saccades to random targets and were able to make saccades to remembered targets, but these were more hypometric than those of controls. When making saccades to a predictable visual target with accompanying tone PDS had difficulty generating anticipatory saccades, but if the visual target was switched off and the tone continued PDS now improved their anticipation, but became more hypometric than those of controls.

These results will be discussed in relation to current hypotheses concerning the role of the basal ganglia in saccade generation.

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Oral session 18: Neuro-ophthalmology

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Wollaston's hemianopia. G. T. Plant, London, UK; M. C. Usselman, London, Canada

In 1824 William Hyde Wollaston described two episodes of transient hemianopia which he had experienced. From this evidence he deduced that there must be a partial decussation of the optic nerves. Although Newton had previously proposed the sem-decussation as an explanation for binocular single vision, anatomical confirmation awaited the work of Johannes Muller and Hannover. Wollaston died in 1828 of a cerebral tumour situated in the right thalamus and it has been assumed by many that the hemianopia which prompted his brilliant deduction was itself a consequence of the tumour. We present evidence that this argument may be a circular one because the PMB was first described not in studies of normal anatomy but in autopsy mate-

2
Some features of primate retinal anatomy relevant to caeco-central scotoma. G. T. Plant, London; V. Hugh Perry, Oxford, UK

Peculiar vulnerability of the "Papillomacular bundle" of retinal nerve fibres has been suggested as an explanation for the characteristic caeco-central scotoma in many toxic ambylopias. 1) We present evidence that this argument may be a circular one because the PMB was first described not in studies of normal anatomy but in autopsy mate-

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