ing the release of gut peptides. Recently, 3,4, dihydroxyphenylserine (DL-DOPS) has been shown to be effective in treating PPH. DL-DOPS is an artificial amino acid which is converted to noradrenaline by a single decarboxylation.

Sympathetic nervous system at sleep

M. Passatore, S. Roatta

Torino, Italy

In order to have a clear picture of the mosaic showing the behaviour of the sympathetic system under conditions of wakefulness and in the different sleep stages, we still need to add a lot of tesserae in this mosaic and probably to remove a number of tesserae we believe we already possess. This is due to methodological limitations and complexity of the system, as well as to hazardous extrapolations which might have led to erroneous conclusion.

The activity of the sympathetic nervous system has been described mainly on the basis of data collected by using the following techniques: 1. determination of epinephrine and norepinephrine concentration in the blood, 2. spectral analysis of heart rate variability, 3. microneurographic recording from single sympathetic fibres, 4. monitoring several organ-specific parameters such as blood flow, sudomotor activity, cutaneous potentials, etc.. The first technique is expected to give a general picture of the sympathetic activity, but too general if we consider the large differences in the behaviour of the sympathetic outflow to different regions which has been demonstrated by electroneurography studies in man and animals. Analysis of R-R variability has been widely adopted to detect changes in the sympatho-vagal balance in the cardiovascular system, however results cannot be extended to assess sympathetic outflow either to all vascular territories or to other effector organs.

Finally, further aspects to be considered concern the different effectiveness on different organs of the sympathetic innervation, as well as the time patterns of discharge which can influence the catecholamine release and the adrenergic-peptidergic (NPY) cooperation.

These may be some of the reasons of the conflicting results found in the literature concerning sympathetic activity during various physiological and pathological conditions, including different stages of sleep.

Traumatic brainstem lesions

Acute traumatic brainstem lesions – introduction

F. Gerstenbrand

Vienna, Austria

Acute traumatic brainstem lesions may develop as primary or secondary iesions. The primary lesion is a consequence of an impact on the head type V or Va of the Innsbruck impact scheme modified according to Spatz. The head is hit directly from above or in the upper fronto-parietal region. The main lesions of the linear inner lower brain trauma (Lindenberg) are located in the midbrain and in the surrounding area (medial part of the temporal lobe, vermis region of the cerebellum). The secondary midbrain lesion is caused by tentorial herniation triggered by a supratentorial event (brain oedema, haematoma). The symptomatology of secondary brainstem lesions develops in five phases to reach the full stage (stage V) of the acute midbrain syndrome. The symptoms of the primary traumatic brainstem lesions mostly start with the full stage (phase V, the mesencephalic-pontine syndrome).

The clinical symptomatology of the acute midbrain syndrome is characterised by a disconnection of the brain and the brainstem, which causes a disinhibition of the autonomic brainstem centres and of the motor centres for antigravity reflexes. The symptoms of the full stage are tachypnoe, tachycardia, hypertension, etc. and decorticate rigidity and decerebrate rigidity, respectively, [flexed-stretched cramps, stretched synergism (Gerstenbrand and Lücking)]. Anglo-American literature (McNealy and Plum) describes the development in different stages to the mesencephalic-pontine syndrome. A lateralisation of symptoms of the acute traumatic midbrain syndrome may be seen in some cases (Gerstenbrand and Lücking), which was confirmed by Plum and Posner.

Primary and secondary acute midbrain syndrome may be followed by the acute bulbar brain syndrome (two phases), the medullary syndrome according to McNealy and Plum, which is characterised by a breakdown of the motor and autonomic regulatory centres with respiratory arrest. A remission of the acute midbrain syndrome in the reverse order of syndromes is possible. A remission of the bulbar brain syndrome has been observed in a few cases. The full stage of an acute traumatic bulbar brain syndrome leads in most cases to an irreversible breakdown of the brain functions, i.e. brain death. In most cases of primary and secondary traumatic midbrain syndrome, a traumatic apallic syndrome (vegetative state of traumatic aetiology) develops.

Early diagnosis of acute traumatic brainstem lesions and clarification of the aetiology permit successful treatment in cases of secondary, but also of primary lesions.

Acute symptomatology of primary and secondary traumatic brainstem lesions

P. E. Vos

Department of Neurology, St. Radboud University Hospital, Nijmegen, The Netherlands

Severe traumatic brain injury may cause different types of brainstem damage with bad prognosis. Primary brainstem damage due to mechanical forces initiated at the moment of injury can be focal or diffuse. Secondary damage occurs as a consequence of extrinsic forces provoked by herniation of (lesioned) remote brain structures and raised intracranial pressure. The classical brainstem syndromes are infrequently encountered in traumatic brain injury because of the high frequency of immediate fatality especially when cervical hyperextension results in disruption at the pon-

European Federation of Neurological Societies

European Journal of Neurology

Official Journal of the European Federation of Neurological Societies

Volume 6 Supplement 3

September 1999

Published by
Lippincott Williams & Wilkins
Third Floor, 241 Borough High Street,
London SE1 1GB
Tel: (+44) 0171 940 7500, *Fax*: (+44) 0171 940 7575

Abstracts of the

4th Congress of the European Federation of Neurological Societies

Lisbon, Portugal
September 7–11, 1999

CONTENTS