North America. Nevertheless, phase IV studies have demonstrated that rt-PA is still underused in part because of delayed referral of stroke patients to hospital, and in part due to physician reluctance to use a drug with a low therapeutic index. We stress now the need of educational programmes directed to the public, general practitioners and emergency department physicians to teach the recognition of stroke symptoms, the importance of treating stroke as an emergency and the correct use of rt-PA.

FW28-3
Guidelines for acute stroke treatment

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No abstract received

Traumatic apallic syndrome/traumatic vegetative state (TAS/TVS)

FW29–1
The traumatic apallic syndrome/traumatic vegetative state: Introduction

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The traumatic apallic syndrome/traumatic vegetative state (taS/tvS) belongs to the severest neurological diseases. Actually, the number of patients has increased in recent years. Due to the improvements in neurological rehabilitation, most cases could be treated successfully.
The definition of the traumatic apallic syndrome is based on the first publication of Kretschmer (1940). Jennet and Plum (1972) described the permanent vegetative state, but omitted the term 'permanent' after increased experiences. The term apallic was chosen to indicate that this syndrome is a functional disturbance of the brain and not caused by a cortical and/or subcortical damage of brain tissue. Permanent vegetative state signalizes the total and persistent breakdown of the brain due to an anatomical damage.
Principally there are two ways for developing apallic syndrome/vegetative state, a severe acute damage of the brain (traumatic, hypoxic, etc.) or the final stage of a diffuse and progressive process of the brain (Creutzfeldt-Jakob disease, Huntington's Chorea, etc.). The first group of patients with aS/vS, especially after an acute traumatic brain injury, passes an initial phase with the symptoms of an acute midbrain syndrome (midbrain-upper-pons-stage), developing after a transitory stage the full stage of aS/vS followed by a remission stage (80%). 30% of the patients can be resocialized.
In the treatment of taS/tvS every patient needs an active treatment in the acute phase followed by a special neurorehabilitation often to be continued over months. In some cases an improvement can be observed even after one year.
In the workshop the detailed symptomatolgy, modern diagnostic methods and the special neurorehabilitation program will be discussed.

FW29–2
Clinic and course of the TAS/TVS

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There are two causes for the development of an apallic syndrome/vegetative state: acute damage of the brain (traumatic, encephalitis, hypoxic), and end stage after progressive diffuse brain disease (Creuzfeldt-Jacob, metabolic disorders, etc).
In acute damage of the brain we see as initial stage as an acute midbrain syndrome/upper pons stage, followed by an acute bulbar brain syndrome/medullary stage, transient stage leading to the full stage of an apallic syndrome/vegetative state.
In 80% of patients with traumatic apallic syndrome/traumatic vegetative state a remission can be observed which occurs in 8 phases (Innsbruck-Remission-Scale) The remission course might stop in the first two stages. The apallic syndrome is a final stage. Basically the vegetative state of the Anglo-American literature has the same symptoms.
The apallic syndrome shows the symptoms of a disinhibition of the brainstem with coma vigile, opisthotonus, vegetative symptoms and antigravity symptoms.
A patient with apallic syndrome needs special rehabilitation and has to be treated to reach total recovery. Special centres for apallic syndrome/vegetative state have to be established, with educated medical and-non-medical professionals.

FW29–3
Morphology of traumatic apallic syndrome/vegetative state

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There is increasing evidence from clinicopathologic and experimental studies that the causes of post-traumatic apallic syndrome (AS) or (per)sistent vegetative state are: 1. diffuse axonal injury (DAI) due to axonal rupture with microglial scars and Wallerian degeneration of long fibre tracts. They occur with or without skull fracture, cortical contusions or sequelae of increased intracranial pressure (ICP) and are caused by acut shearing and tension forces in acceleration/deceleration and rotation injuries. DAI has been reproduced experimentally, detected histologically already a few days after trauma, and shows typical patterns: corpus callosum (midline, often bilaterally), fornix, ventricular walls, hemispheral white matter, dorsolateral brainstem tegmentum, cerebral and cerebellar peduncles. 2. Sequelae of ICP and transtentorial herniation: uni- or bilateral hippocampal lesions, haemorrhagic infarcts in mediobasal occipital lobes, multiple lesions in basal ganglia (thalamus, striatum, hypothalamus) and brainstem with pressure necroses in midbrain and pontine tegmentum, pes pedunculi (Kernohan's notch) and infarcts in venous and/or arterial supply areas. Secondary brainstem lesions in up to 90% of AS following blunt head injury, show close relationship to other sequelae of ICP. In patients with long survival and partial clinical remission, they are smaller than in acute fatal cases and often limited to peripheral parts of dorsolateral brainstem tegmentum and periaqueductal grey, i.e. supply areas of perforant and circumflexing vessels. 3. Ischemic brain damage in hippocampus, basal ganglia (thalamus!) and neocortex with predilection of arterial border zones, less frequent cerebellum. 4. Diffuse white matter lesions (leukoencephalopathies). All these lesions may lead to development of internal hydrocephalus. Comprehensive recent reviews of the neuropathology of AS after acute brain damage (of any cause) has identified 3 main patterns of lesions: widespread bilateral neocortical damage (laminar necrosis, diffuse gliosis), diffuse damage to cerebral white matter, and bilateral damage to thalamus and/or brainstem. The prognosis of AS after blunt head injury and other brain damage is closely related to the severity and extent of the morphologic brain lesions.

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