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# IMAGING OF INNER CEREBRAL TRAUMA

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**Summary**

The concept of "Inner cerebral trauma" (ICT) has been preliminary defined as a characteristic topographic pattern of deep brain lesions produced by physical forces occuring within the cranial cavity in closed head injury of the acceleration/deceleration type. The lesions, based on neuropathological examinations, are characteristically localized in the "centro-axial" regions of the brain. The extent of ICT is often underestimated by CT. Due to asses the value of MR imaging, 83 patients with ICT were examined on a 1.5 T unit in different stages after trauma. The pattern of lesions, as shown with MR imaging, correlated well with neuropathological studies, suggesting a multifocal pathogenesis of severe traumatic brain injury.

RIASSUNTO

L'"inner cerebral trauma" (ICT) è stato definito come pattern topografico caratteristico delle lesioni cerebrali profonde che, nel trauma cranio-cerebrale chiuso, originano per l'azione di forze fisiche del tipo accelerazione/decelerazione. All'esame neuropatologico risulta che tali alterazioni sono localizzate a livello della regione "centro-assiale". Spesso il quadro dell'ICT viene sottovalutato alla CT, quadro ben evidenziabile alla risonanza magnetica. 83 pazienti con ICT sono stati sottoposti a risonanza magnetica, che ha confermato i risultati degli studi neuropatologici circa la patogenesi multifocale del trauma cranio-cerebrale grave.

Keywords: Inner cerebral trauma - closed head injury - magnetic resonance imaging

## Introduction

The term "inner cerebral trauma" (ICT) has been defined by Grcevic as a characteristic pattern of deep brain lesions in closed head injury of the acceleration/deceleration type. As the traumatizing forces acting the direction of the

longest diameter of the skull, the lesions are characteristically localized in the " centro-axial" regions of the brain. The ICT pattern is characterized by multiple lesions within the parasagittal region, the corpus callosum, the periventricular and paraventricular white matter, hippocampal and temporo-sphenoidal area, basal ganglia, upper brainstem, and the ponto cerebellar complex 7. In addition Gerstenbrand, subdivided the different pattern of inner cerebral trauma depending on the direction of traumatizing forces, in a "linear upper inner cerebral trauma" a "linear lower inner cerebral trauma" and a rotation trauma 6. Depending of the local strength of acting forces and the capacity of resistance of the brain tissue the lesions are subdivide in primary irreversible lesions ("epicenters") surrounded by a perifocal zone of potentially reversible tissue damage ("traumatic penumbra"). Due to the relative insensivity in the detection of small and non-haemorrhagic lesions, the extent of ICT is often underestimated by CT. Magnetic resonance imaging (MR) has proven superior to CT in the evaluation of most non-traumatic central nervous disorders. The purpose of our study was to assess the value of MR in patients with special consideration of the acute stage.

**Materials and methods**

The study includes 83 patients (71 male, 12 female), with a mean age of 25.1 years (5-69 years). Inclusion criteria were suspicious ICT on CT, or discrepancy of CT findings and clinical symptoms. Clinically, acute midbrain syndrome was present in 11 patients, prolonged midbrain syndrome in 10, apallic syndrome in 29, and post- apallic stage in 33 patients. All patients underwent a CT examination at admission to our hospital. MR-Imaging was performed within one week after the accident in 11 patients (acute stage), in 13 patients between one week and one month (subacute stage), in 39 patients between one month and one year (late subacute stage) and 20 patients later than one year (chronic stage). All MR studies were performed on a 1,5 T unit. The imaging protocol consisted of sagittal and axial T1 (500/14/TR/TE), and of axial and coronar PD/T2 (2400/15/90) weighted images.

Additionally a 3-D FLASH sequence was performed in 60 cases. All patients were monitored by continuous pulse oximetry, capnography, ECG, and blood pressure manometry. 50 patients required general anesthesia or artificial ventilation. In two patients, who died six respectively nine months after the injury (three weeks, respectively six months after MR imaging), autopsy was performed. The unsectioned brains were fixed in buffered formalin, and 28 paraffin blocks were taken from correspondig sites of both hemispheres, the brainstem and the cerebellum. Sections were stained with hematoxilin and eosin, prussian blu e, silver impregnation, and with the Masson goldner trichrome method.

**Results**

MR images were of diagnostic quality in all cases. Hemorrhagic and/or non hemorrhagic lesions were present in the periventricular/parasagittal complex in 53 of 83 patients (56.9 %), in the hippocampal/parahippocampal complex (52/83; 68.8 %), the basal ganglia (64/ 83; 68.8 %), the midbrain (45/83; 48.3 %), corpus callosum (69/83; 74.1 %), pons and medulla oblongata (30/83; 32.2 %) and the cerebellum (17/83; 18.2 %). The detailed distribution of lesions within the various anatomical regions in the acute, subacute, and chronic stage of ICT are summarized in table I. The histopathological examination of two cases confirmed the presence of focal post traumatic lesions comparable with MR, and ruled out parenchymal damage in areas without signal abnormalies.

**Discussion**

The major limitation of high-field MR imaging lies in the examination of patients in the acute stage of cerebral trauma. This limitation can be overcome by the use of specially adapted, non ferromagnetic ventilation and monitoring equipment. Due to its better contrast resolution and multiplanar capability, MR detected significantly more lesions than CT in all stages of ICT3. The pattern of ICT-related lesions in our patients, as detected with MR imaging, correlated well with our and earlier neuropathological studies. The results of our study suggest that the brain damage in patients, who survive closed head injury for sufficiently long time and who, according to the current classification, sustained a " diffuse axonal injury ", is rather multifocal and partially reversible 1. Clinically, these patients are always comatose immediatly on impact with signs a midbrain or bulbar brain syndrome, caused by dysfunction of the ascending reticular formation in the brainstem. Involvement of the brainstem is usually due to direct mechanical impact or hemorrhage. Examination during the acute stage of trauma detected traumatic lesion within the brainstem in 91 % of patients. Dependent on the degree of brainstem impairment and the severity of associated parenchymal lesion in other parts of the brain, gradual remission of midbrain syndrome or transition to apallic syndrome may be observed, leading to a defect stage with predominantly focal neurological deficits. This clinical course can hardly be attributed to widespread diffuse axonal injury, but is rather consistent with a pattern of multiple focal and partially reversible lesions. Our observations are also in accordance with the results of histopathological investigations carried out by Grcevic who suggested a pattern of primary focal, periaxial subcortical lesions in closed head injury. He described acute primary irreversible lesions surrounded by perifocal zones of potentially reversible tissue damage ("traumatic penumbra"). Preliminary results of serial neurobehavioral assessment and MR imaging in " diffuse brain injury " ca rried out by Levin et al. also suggested a multifocal pattern of lesions 8. In conclusion, improved in vivo detection of

posttraumatic lesions by MR allows a better correlation of neurological deficits and the underlying brain pathology in all stages of severe closed brain injury 4,2. The summary term of " diffuse brain injury" seems no longer justified, and should be replaced by an exact description of the type, location, distribution and size of traumatic lesions as shown by MR imaging.

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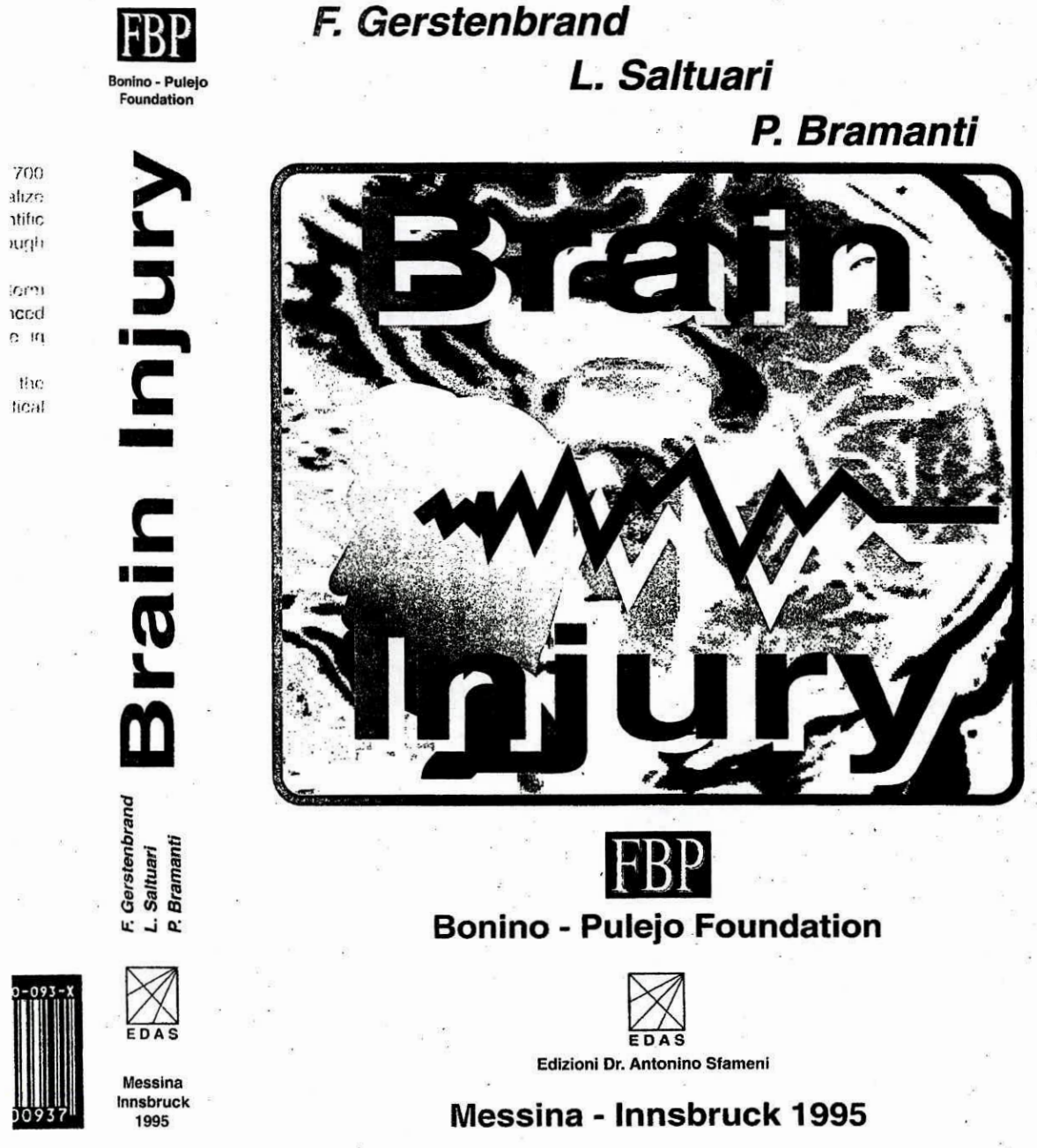

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