Cerebral Contusions and Lacerations
A Clinical Study

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Introduction

Before presenting this study, a few preliminary remarks should be made.
When reference is made to the literature, it appears difficult to give
a precise clinical definition of traumatic cerebral lesions, and particularly
cerebral contusions–lacerations. The classical distinctions between cerebral
commotion, contusion and compression has been modified by numerous
authors. Some authors take into account the gravity of the clinical state
(Bues and Stewart 1947), or the evolution with time (Fasano 1973; Gruner

R. A. Frowein (ed.), Cerebral Contusions, Lacerations and Hematomas
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et al. 1965), or the mechanism of production of the lesions (Gurdjian et al. 1958).

In everyday practice, this distinction no longer has a precise signification: when the notion of commotion is based on a functional lesion of the brain tissue, autopsies and now the CT scan reveal the possibility of macroscopic lesions. When the notion of contusion is based on an anatomicical lesion of the nervous system, the resulting clinical manifestations depend on the location of the lesion. These manifestations can therefore be “silent” if the lesion affects a “dumb area”. Moreover, systematic use of the CT scan can demonstrate the presence of relatively large lesions in patients in a satisfactory clinical state, but with cerebral contusions–lacerations and associated tissue destruction, oedema and hemorrhage to different degrees. They are thus described under various headings in the literature (Botterel and Stewart 1947; Casella et al. 1967; Columella 1973; Feld et al. 1955; Geuna et al. 1967; Guillermain 1970; Obrador et al. 1968; Rusu 1972) and one particular aspect or another is considered by the authors. Comparison of different series is consequently difficult especially as acute subdural hematomas, which are frequently associated with these lesions, are also included with them (Fasano 1973; Lazorthes 1973), or the lesions themselves are integrated into the group of traumatic intracerebral hematomas (Stender and Schulze 1966; Teasdale and Galbraith 1981; Tönnis et al. 1965). Therefore, having recently re-examined this question using 833 personal observations selected in relation to precise criteria (angiographic, tomodensitometric and/or surgical), we have included large parts from this study (Vigouroux and Guillermain 1981). Our data generally agree with those found in the literature which cannot be exhaustive, bearing in mind the numerous publications pertaining to lesions of this type (Casella et al. 1967; Cohadon et al. 1973; Columella 1973; Da Pian et al. 1967; Geuna et al. 1967; Liguori and Troisi 1966; Stender and Schulze 1966; Tönnis et al. 1965).

Symptomatology

Traumatic cerebral lesions are usually shown by alterations of consciousness, with or without signs of localization. Numerous classifications have been made mainly concerning the seriously cranially traumatised patient and particularly those in coma (Vigouroux and Guillermain 1986). However, whether these classifications refer to a progressive deterioration in the state of vigilance, or to clinical sign scales as in the Glasgow scale, or to signs of axial involvement from rostrocaudal destruction, they take no account of the lesions. Thus, even if a contusion syndrome has been described (Basauri et al. 1973; McLaurin and Helmer 1965), the clinical