Mechanisms of Extradural Hematomas*

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For many years it has been recognized that extradural hematoma of traumatic origin usually results from a lacerated middle meningeal artery or one of its major branches. The classical sequence of neurologic events seen clinically occurs over a period of several hours and terminates fatally if surgical evacuation is not achieved. It is well known, however, that varied clinical forms do exist. As documentation of subacute and chronic forms and of clots forming after only minor head injury or without an overlying fracture appears in the literature, the accepted concepts of etiology become more difficult to comprehend. The purpose of this study has been to attempt to clarify some of the mechanisms operating in the development of extradural hematoma. The experiments have been carried out entirely in dogs and the hazards of translating the results to humans are recognized; nevertheless, certain general principles appear to transcend the differences of species.

The work is divided into two broad categories: 1) experimental evaluation of factors necessary for development of an extradural hematoma, and 2) observations of relationships of intracranial deformities to clinical occurrences following experimental production of extradural hematoma. The methods and results of these lines of investigation will be set forth separately followed by an attempt to integrate the conclusions into an acceptable description of the development of this lesion.

Extradural Hematoma Formation

Method. In the first group of experiments, using anesthetized dogs, burr holes of varying size were placed and a #18 short needle was fixed so that the tip was just in the extradural space. The burr hole then was sealed with acrylic. The needle was attached via a system of tubing to a source of steady pressure of fluid. The pressure within this system was recorded by a Physiograph.† Pressures were recorded simultaneously and continuously from a cisternal needle and in certain experiments respirations also were monitored (Fig. 1).

Separation of dura mater from the inner surface of the calvarium with formation of an extradural hematoma was indicated by sudden elevation of cisternal pressure and/or transient decrease of pressure in the extradural system (Fig. 2). In certain experiments the extradural pressure was allowed to continue until the animal died while in others it was discontinued immediately following dura mater-bone detachment.

By varying the size of the cranial opening, the extent of dura mater-bone detachment, and the input pressure, the total force applied to the dura mater could be varied. This force was calculated in each experiment according to the following formulae:

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1) \text{Force in grams} = \left[ \frac{\text{Pressure in gm./cm.}^2}{\text{Input pressure in gm./cm.}} \right] \times \left[ \frac{\text{Wgt. den. mercury}}{\text{Radius}^2} \right] \times \left[ \frac{\text{Area in cm.}^2}{\text{Radius}^2} \right] \times \left[ \frac{\text{in cm. Hg}}{\text{in gm./cc.}} \right]
\]

In the second group of experiments the dog's femoral artery was substituted as the source of pressure. A small catheter was introduced into the femoral artery; the tubing was filled with a heparinized solution and then attached to the cannula leading to the extradural space. The purpose of this series was to determine the effect of pulsating rather than steady pressure.

Results. Using the steady-pressure appara-

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Fig. 1. Method of simultaneous recording of cisternal and extradural pressures during formation of extradural hematoma.

Fig. 2. Physiograph recording demonstrates that, within several seconds after introduction of extradural pressure, cisternal pressure becomes elevated and steady pressure falls temporarily because of expansion of the system. Changes in respiratory rate also are noted.