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H. Krayenbühl, Zürich (Managing Editor)

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Preface

There are two important reasons for commencing this new series of publications entitled "Advances and Technical Standards in Neurosurgery": 1. the lack of any organized common European postgraduate training system for young neurosurgeons and 2. the language barriers, which impede the exchange of neurosurgical findings in Europe more than in other parts of the world.

The fact that the English language is well on the way to becoming the international medium at European scientific conferences is a great asset in terms of mutual understanding. Therefore the Editors have decided to publish all contributions in English, regardless of the native language of the authors.

All contributions are submitted to the entire editorial board before publication of any volume.

Our series is not intended to compete with the publications of original scientific papers in other neurosurgical journals. Our intention is, rather, to present fields of neurosurgery and related areas in which important recent advances have been made. The contributions will be written by specialists in the given fields and will constitute the first part of each volume.

In the second part of each volume, we shall publish detailed descriptions of standard operative procedures, furnished by experienced clinicians; in these articles the authors will describe the techniques they employ and explain the advantages, difficulties and risks involved in the various procedures. This part is intended primarily to assist young neurosurgeons in their postgraduate training. However, we are convinced that it will also be useful to experienced, fully trained neurosurgeons.

The descriptions of standard operative procedures are a novel feature of our series, and in this it differs from the similarly entitled series "Progress in Neurological Surgery"; also, our series will be mainly, but not exclusively, a forum for European neurosurgeons. We intend as well to make available the findings of European neurosurgeons which are published in less familiar languages to neurosurgeons beyond the boundaries of the authors' countries and of Europe, and we aim to promote contacts among European neurosurgeons.

The Editors do hope that neurosurgeons throughout the world, and not only in Europe, will profit by the new series "Advances and Technical Standards in Neurosurgery".

The Editors

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A. Advances

Non-operative Management of Intracranial Hypertension

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With 20 Figures

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I. Pathophysiology of Intracranial Hypertension

The brain, by being encased in a container with rigid walls, is unique among the organs of the human body. The restricted ability of the intracranial contents to expand implies that the intracranial pressure (ICP) is a parameter of central importance for the intracranial dynamics.

A comprehensive account of the pathophysiology of intracranial hypertension is beyond the scope of this article. However, familiarity with intracranial dynamics is of fundamental importance for the management of patients with intracranial hypertension. Therefore and with respect to the torrent of new information which has been presented during the last two decades it is essential to begin with a short survey of this field.

1. Cerebrospinal Fluid Dynamics and Intracranial Pressure

The site of the cerebrospinal fluid (CSF) production has long been a controversial issue. At present, in accordance with the early views of Dandy and Blackfan (1914) and Weed (1935) it seems to be generally agreed that the major fraction of the CSF is formed in the cerebral ventricles.

CSF is at least partly produced by means of active transport and "secretory pressure" is supposed to be one of the main forces behind the CSF flow and the increase in CSF pressure that occurs after obstruction of fluid pathways. It seems highly probable that in originally incomplete obstruction an increase of the ventricular fluid pressure (VFP) may displace the supratentorial part of the brain and cause further blocking of CSF pathways thus starting a vicious cycle. In arrested hydrocephalus subsidiary routes of absorption are supposed to be utilized. Being in continuity with the ventricles, the extracellular space of the brain tissue is a probable pathway for such diversion of CSF.

In their classic works, Key and Retzius (1875) and later Weed (1914) produced convincing evidence that the main site of the CSF absorption is in the arachnoid villi. This conception is still generally accepted. Recent investigations in animals indicate that the reabsorption takes place through open channels with a valve mechanism for unidirectional flow and is exclusively dependent on the difference in hydrostatic pressure between the subarachnoid space and the dural sinuses (Welch and Friedman 1960, Davson et al. 1970). Investigations in man indicate that there is no CSF absorption below a pressure of approximately 5 mm Hg ("opening pressure") and that a linear relation exists between the CSF pressure and the absorption rate above this limit (Cutler et al. 1968,

Lorenzo et al. 1970). It may be assumed that the absorption mechanism plays an important role for the intracranial pressure homeostasis and pressure/volume relationships as well as for the ability of the CSF to act as a "spatial buffer" under pathological conditions (see below). For review of literature see Davson (1967) and Johnson (1972).

2. Interrelations between ICP and Intracranial Hemodynamics

This relationship is reciprocal in so far as variations of the cerebral vascular resistance and the intracranial venous pressure may cause variations of the ICP (Roy and Sherrington 1890, Ryder et al. 1952) and, on the other hand, variations of the ICP may cause changes of cerebral perfusion pressure and cerebral blood flow (CBF). The influence of the ICP on the CBF is determined by the fact that the blood pressure in the draining cerebral veins is approximately equal to the ICP (Noell and Schneider 1948, Rowan et al. 1972). This means that the cerebral perfusion pressure equals the difference between the systemic arterial pressure (SAP) and the ICP. Thus, the formula for CBF can be written:

$$\mathrm{CBF} = \mathrm{k}\,\frac{\mathrm{SAP} - \mathrm{ICP}}{\mathrm{CVR}}$$

where CVR is the cerebral vascular resistance. This relationship is of fundamental significance in the production of ischemic brain damage in intracranial hypertension. The autoregulation of the CBF—i.e. the vasomotor mechanism, which maintains an adequate CBF in spite of variations of the cerebral perfusion pressure—may thus be elicited not only by a fall of the systemic blood pressure like in other organs, but also by an increase of the ICP (Wolff and Forbes 1929, Fog 1933, Noell and Schneider 1948, Evans et al. 1951, Lassen 1964, Zwetnow 1968, 1970). Furthermore, there are strong reasons to believe that a vasodilatory response to increase of ICP is involved in the mechanism producing certain acute elevations of the ICP in patients with intracranial hypertension (see below, page 13).

When ICP is experimentally increased, autoregulation is capable of maintaining normal CBF until the cerebral perfusion pressure has been reduced to about 40 mm Hg (Zwetnow 1970). On further increase of ICP, i.e. further reduction of the cerebral perfusion pressure, there is a critical decrease in CBF leading to an intracranial state characterized by prolonged vasodilatation, a CBF which is passively dependent on the systemic blood pressure, and an impaired or abolished response of the resistance vessels to changes in the perfusion pressure and $PaCO_2$ ("cerebral vasomotor paralysis", Langfitt et al. 1965, 1966). If this process continues the cerebral circulation may be further impaired by progressive brain swelling, first from congestion and later from intractable brain edema. One can assume that the well-known picture of non-filling of intracranial arteries on angiography and total infarction of the brain is the end result of such a development (cf. section on brain edema).

In treating patients with intracranial hypertension it is important to bear in mind that a defective autoregulation means that both CBF and ICP is more dependent on the systemic arterial pressure than under normal conditions. The

combination of vasoparalysis and rise in blood pressure may cause a disastrous rise in ICP and seems to cause the final catastrophy in many cases of intracranial hypertension (see Fig. 3*c*). On the other hand, defective autoregulation means an increased risk of ischemic complication from fall in blood pressure.

The dependence of the cerebral vascular resistance on the PCO_2 of the arterial blood and the co-variation of the intracranial venous pressure with the intrathoracic pressure form the physiologic basis for the central role of the respiration in the management of patients with intracranial hypertension. During the last 15 years the respirator has become more and more established as a tool in the treatment of patients with traumatic and non-traumatic brain lesions. The importance of the effect on ICP of hyperventilation in the treatment of intracranial hypertension as well as the risk of cerebral ischemia at low $PaCO_2$-levels are issues still under discussion. They will be dealt with below (page 40).

3. Brain Edema

Edema of the brain tissue may cause forceful swelling of the brain and by influencing intracranial pressure/volume parameters it may interfere with the intracranial dynamics. (To avoid confusion the terms brain swelling and brain edema should be kept apart; brain swelling may be caused by edema *and* by blood congestion.) The pathophysiology of brain edema will not be treated in any detail. However, the importance of brain edema for the production of intracranial hypertension warrants a short account of basic ideas and clinically important data.

In accordance with Klatzo (1972) current pathophysiological concepts may be summarized as follows: Cerebral edema is defined as an abnormal accumulation of water in the brain tissue. The water may be localized mainly within the cells (cytotoxic edema) and is then related to a functional disorder of the cell membrane; or it may accumulate mainly in the extracellular space as a result of increased vascular permeability permitting an increased outflow of water, Na^+-ions and protein molecules from the blood (vasogenic edema). Disorders of permeability may be caused by various kinds of lesions to the vascular endothelium. In this connection the "tight junctions" of the endothelium are of special interest since it may be assumed that temporary reversible opening of tight junctions may occur in a number of pathological conditions in the brain.

Brain edema may cause or contribute to the production of intracranial hypertension in a variety of brain lesions due to mechanical trauma, ischemia, hypoxemia, neoplasm, and toxic agents. With regard to the frequent occurrence of edema in such common diseases as cerebral contusion and cerebral infarction it is probably the principal cause of intracranial hypertension.

From a clinical point of view, the progressive nature of brain edema is of particular importance. One example is the perifocal edema which surrounds localized cerebral contusions and transforms them into expanding lesions. An even more striking example is the intractable brain edema, which occurs in the final state of intracranial hypertension and which by its inherent force appears capable of arresting the cerebral circulation by compression of intracranial vessels. This circulatory arrest may be preceded by a rise in blood pressure and abolished autoregulation (see Fig. 3 C). The edema may thus be an ex-

ample of what Langfitt has called "hydrostatic brain edema", i.e. edema due to increase in capillary and venous pressures caused by dilatation of cerebral resistance vessels in combination with high arterial blood pressure (Langfitt et al. 1967, Marshall et al. 1969, Schutta et al. 1968).

Another clinically important observation has been reported by Klatzo et al. (1967). In animal experiments they found that the speed of progression and the ultimate extent of edema around an experimentally induced brain lesion is largely influenced by the systemic blood pressure. This relationship must always be taken into account when the mechanism behind a therapeutic effect upon brain edema is discussed. The clinical importance pertains to the control of systemic arterial pressure in patients with intracranial hypertension (see page 28).

The progressive nature of brain edema may be explained by the reciprocal relationship between edema and CBF. On one hand, brain edema can cause reduction of CBF owing to a general increase of ICP and probably also to a localized increase of the tissue pressure. On the other hand, a reduction of CBF can induce or aggravate edema by causing tissue hypoxia.

4. Transmission of Pressures in the Cranio-Spinal Cavity

In spite of their non-uniformity the contents of the cranio-spinal cavity have long been regarded as forming a medium for almost unrestricted transmission of pressure variations. Cerebrospinal fluid pressure has consequently been used as representative of the pressure in the whole cavity including the blood pressure in thin-walled vessels of the vascular bed (Noell and Schneider 1948). The latter assumption has recently been confirmed by experimental studies showing a highly significant linear correlation between the VFP and the blood pressure in cortical veins (Rowan et al. 1972). The implications are twofold: firstly, the ICP is dependent on how much of the systemic blood pressure is admitted into thin-walled capacitance vessels, i.e. on the vasomotor tone of the resistance vessels; secondly, it seems justified to define the cerebral perfusion pressure as the difference between mean arterial pressure and mean VFP (see above, page 5).

When a space-occupying lesion is expanding in the cranial cavity it causes pressure gradients in the brain. Owing to the plasticity of the brain tissue these gradients tend to be equalized by displacement in accordance with the law of the least resistance. Filling out of the subarachnoid spaces, herniation in the isthmuses and distortion of the brain stem are well-known consequences of such mass displacement and their clinical correlates need not be treated in this article.

As a rule, there is a rise in VFP when a supratentorial lesion causes mechanical stress on the brain stem by mass displacement. However, being part of a compensatory mechanism, displacement and distortion may be present without any significant rise of ICP provided that the lesion is expanding slowly. These two phenomena should be kept apart.

A free transmission of fluid between different parts of the CSF spaces is a prerequisite for "spatial compensation" of a space-occupying lesion by flow of CSF out of the cranial cavity (see below). By obstructing fluid pathways a tamponade of the tentorial incisura or the foramen magnum is always a serious complication. Both in patients (Hodgson 1928, Smyth and Henderson 1938,

Antoni 1946) and in experimental animals (Langfitt et al. 1964) it has been shown that such tamponade of the isthmuses may produce considerable differences in pressure between the lateral ventricles and the infratentorial and spinal spaces. Such differences are ominous since they signify that acute rises of the supratentorial pressure (e.g. plateau waves, see below) will not be fully transmitted to the infratentorial space but will cause further compression and distortion of the brain stem instead. Simultaneous continuous recording of ventricular and spinal fluid pressures has been used for the clinical diagnosis of ventriculo-spinal pressure differences and assessment of the risk of tentorial and tonsillar herniation. Since puncture of the spinal subarachnoid space means a definite risk of leakage through the hole in the dura (Lundberg and West 1965) and consequently a risk of misleading results as well as of augmented pressure difference and further herniation, we doubt whether this procedure has a place in neurosurgical practice.

5. Pressure/Volume Relationships

The core of the Monro-Kellie doctrine—that a change in volume of one of the components of the intracranial content necessitates a corresponding change of the volume of one or more of the other components—is still valid. This concept may be expressed by the formula below.

$$V_{brain} + V_{blood} + V_{CSF} + V_{expansive\ lesion} = V_{intracran.}$$

The ability of blood and CSF to pass out of the cranial cavity makes it possible for a lesion to expand. The readiness with which this shift can take place is decisive for the rise in ICP. This relation is beautifully illustrated by Langfitt's classic pressure/volume curve (Fig. 1). In his experiments, Langfitt used a slowly expanding (1 ml/hour) supratentorial balloon. One may assume that the initially slow rise of the curve represents the joint action of several compensatory mechanisms, i.e. shift of brain tissue, shift of CSF into the spinal subarachnoid space, increased absorption of CSF, and squeezing out of blood from the cerebral vascular bed (Ryder et al. 1953, Martins et al. 1972). The steep part of the curve obviously represents a stage of exhaustion of the mechanical compensation, but may also include vasodilatation elicited by the rise in ICP. The capacity for spatial compensation is thus dependent on a variety of anatomical, mechanical, and physiological factors including the form and size of the tentorial incisura, the consistency of the brain, the ability of the spinal dural sack to expand, the absorption capacity of the subarachnoid villi, the reactive ability of the cerebral vessels etc. The importance of the time factor is illustrated by the wellknown fact that a rapidly increasing hematoma causes signs of brain stem compression at a much smaller size than does a slowly expanding tumour.

Pressure/volume relationships during rapid elevations of the CSF pressure (0.08–1.45 ml/sec) have recently been assessed quantitatively in terms of "elastance", i.e. the properties of the CSF space which determine the magnitude of the immediate pressure change produced by a given rapid change in volume (Löfgren 1973). The results indicate that in such rapid increases of ICP the compensation is due to expansion of the spinal dural sack (70%) and compression of the cerebral venous bed (30%). Another clinically interesting observation by Löfgren was that in the presence of transtentorial obstruction the elastance