Fluid dynamics of CSF in adult hydrocephalus is quite different compared to the conditions of hydrocephalus in infants (2). Main reasons for the difference between adult and infantile hydrocephalus are the closed cranial sutures and the physical properties of grey and especially of white matter in adults.

Figures 1 and 2 (1, 4, 6, 12) illustrate the aetiology and the pathological consequences of hydrocephalus in adults. Since the underlying diseases causing hydrocephalus may occur in infants and children but also in adults (Fig. 1), we have limited our investigations to those conditions which occur predominantly in adults (Fig. 2).

An important factor influencing the pathophysiology of adult hydrocephalus is due to the differences in diameter and elasticity of the ventricular wall and the surrounding tissue as well as to the different pressure gradients in the single portions of the ventricular system and the cisternae. The frontal lobes are a special example of this. Here nearly all subarachnoidal space between bone and brain substance is missing. Increasing pressure of the CSF therefore affects the frontal brain substance immediately. The consequences are circulatory disturbances of the frontal cortex manifesting itself especially in the venous system with tortuous dilatations and with a predominant dilatation of the anterior portion of the lateral ventricles in a very early stage (8). Clinically this correlates to psychic disturbances and occasionally to EEG-changes concentrated especially over the frontal lobes (Fig. 3).

The situation of the third ventricle is quite different from that of the anterior horn: Roof and lateral walls are formed by the compact thalamic nuclei, but floor and especially caudal and anterior borders are formed by membrane-like very soft tissue. These are the ventricular portions, which may be fast and easily dilated up to an extreme degree. The ball-like dilatation of the third ventricle is also supported by the neighbourhood of the basal cisternae. Dilatation of the recessus supraopticus, infundibularis and suprapinealis may lead to transparent and paperlike walls (9) with corresponding encephalography (Fig. 4). This morphological change may be accompanied by endocrine and visual disturbances. In one of our patients we observed a Parinaud-syndrome, which we believe to be caused by the pressure of the enormously dilatated recessus suprapinealis against the lamina quadrigemina (3).

Aquaeduct and IVth ventricle - which are the physiological narrowings - may compensate even extreme degrees of stenosis for quite a long time. But small and unimportant disturbances are apt to cause the decompensation of this very labile steady state. Figure 4 demonstrates the X-ray picture of a high degree aquaeduct stenosis, which decompensated suddenly after an acute respiratory infection. Figure 5 shows the
Table 1. Causes of hydrocephalus in adults

1. Inflammation
2. Post meningitis
3. Post trauma
4. Developmental abnormalities
5. Space occupying processes
6. Subarachnoidal hemorrhage
7. Diffuse neoplastic processes in the subarachnoidal space
8. Demyelinating processes and chronic oedema of the white matter

Table 2. Disturbances in cases of hydrocephalus in adults

1. Frontal lobe
   1.1 Psychosyndroma
   1.2 Frontal EEG-disturbances
   1.3 Disturbance of venous circulation
   1.4 Marked widening of cornua anteriora

2. Third ventricle
   2.1 Blow up of recessus
       (supraopticus, infundibularis, suprapinealis)
   2.2 In some cases: visual disorders
       endocrinological disturbances

3. Aquaeduct and fourth ventricle
   3.1 Paresis of brain nerves (nn. III, IV, VI)
       extrapyramidal symptoms
   3.2 Forking, stenosis, dilatation of aquaeduct
   3.3 Obliteration of foramina of Luschka and Magendie
   3.4 Atrophy of cerebellar white matter following
       increased intracranial pressure

4. Disturbances of CSF-resorption
   4.1 Common signs of increased intracranial pressure
   4.2 Communicating, symmetrical hydrocephalus
   4.3 Obliteration of the subarachnoidal space

Pathological processes affecting the subarachnoidal space, which frequently leads to symmetrical internal hydrocephalus without any stenosis or blockade of the internal ventricular system. A space occupying lesion does not exist in these cases and only in the Risa-test (7, 11) a disturbance of distribution may be obvious. So the diagnosis frequently is made at autopsy, where an obliteration of the subarachnoidal space can be demonstrated histologically. In these diffuse subarachnoidal processes in which neither an increased pressure of the CSF in the ventricular system exists nor a stop of the CSF drainage can be demonstrated, the hydrocephalus should be attributed to a reduction of the subarachnoidal space responsible for CSF-resorption.