As the caption already reveals, I intend to speak exclusively about the European classification of chronic hepatitis, without considering any American or Japanese publications.

Long before the clinical picture of chronic hepatitis found the world-wide interest it receives today, KALK in 1947 described its clinical symptomatic, and he saw and interpreted the laparoscopic findings on the surface of the liver during the course of the disease. His interest centered on the large liver as the principal symptom, and he laparoscopically recognized the successive stages of the large white, the large colored and the large "Höckerleber".

At that time KALK was under the impression that the chronic hepatitis resulted from the acute virus hepatitis and that it was the preliminary stage of hepatic cirrhosis. I shall mention later how far this idea corresponded to our present knowledge.

Only at a later date was it possible to obtain the histological picture of chronic hepatitis...in 1959 by myself, in 1966 by SCHMID, Zurich, in 1967 by BIANCHI, Basel, and thereafter by many others.

Then the necessity arose to correlate the clinical with the morphological findings, all the more as communication between clinicians and pathologists was rather difficult because of differing nomenclature.

For this reason, the E. A. S. L. in 1967 asked an international group of morphologically interested clinicians and hepatologically interested pathologists...I was among them...to try to work out one competent nomenclature, to be used by all concerned. That was done after the problem had been thoroughly discussed on the basis of numerous cases of chronic hepatitis. The result of our discussion was published in 1968 in English, French and German. We tried to find a classification that would be easily understandable, short and to the point, and I may say that we apparently succeeded. Our recommendation was accepted within a short time and practically without opposition, at least in Europe.

In a preamble we stressed the fact that we were guided by morphological view points, but that for the final diagnosis in each case both morphological and clinical findings must equally be taken into consideration. Pathological histology by itself in many cases does not permit a final diagnosis. This shows once again that a close contact between clinicians and pathologists is inevitable when material drawn from the liver by biopsy is to be assessed.

Now to our proposal:

1. Chronic persisting hepatitis

Chronically inflammatory infiltrates are present in the portal areas without any significant widening of these areas and with completely intact lobular architecture. Piecemeal-necroses are absent or are negligible.


You can see that all specimen basically reveal the same findings. That means that during the course of five years no development took place, neither to the better nor to the worse; the process remained static during the whole time.

That is the typical clinical and histological course of chronic persisting hepatitis. This diagnosis is justified whenever a hepatic condition exists for at least one
year and when several histological controls always show the same findings. Thus, we observe here the process of an illness with a mostly favourable prognosis. Such cases as a rule heal after a few years. A change into the aggressive hepatitis is extremely rare.

The pointing-out of the necessity of controls already shows that the histological findings in chronic persisting hepatitis are not pathognomonic. The same or at least very similar changes in the liver are also encountered in subsiding or considerably subsided acute hepatitis and as non-specific reactive hepatitis accompanying infectious general diseases, rheumatism, lupus erythematosides, parasitosis and also carcinoma.

I now show you slides of a reactive hepatitis in rheumatism.

Knowledge of and attention to the anamnesis and the clinical findings are especially important for the assessment of these particular histological changes. Observation of the process will finally lead to the definite decision.

2. Chronic aggressive hepatitis

Here the portal areas are chronic-inflammatorily infiltrated. The infiltrates encroach upon the adjacent lobular regions, and active intra-lobular septa develop in conjunction with piecemeal-necroses. Thereby the lobular architecture is altered, without nodular regeneration yet, however. This attack of the mesenchyme upon the lobules is the reason for calling this hepatitis “aggressive”. In addition, signs of an acute hepatitis may be present.

Cases of chronic aggressive hepatitis may be moderately active, as you will see in the following pictures, showing an increase of connective tissue with indicated alteration, but without real inflammatory activity; or they may be vigorously active. In the latter case they show lively cellular infiltration, a mass of liver cell necroses, and progressive fibrous alteration.

The so-called lupoid hepatitis is a chronic aggressive hepatitis with an especially high inflammatory activity. In our opinion, this form of disease represents a clinical syndrome without characteristic histological findings. We, therefore, did not consider it in our presentation.

So far the results of our discussion group.

I would like to make a short reference to another form of chronic hepatitis recently mentioned at our clinic in Kassel, which has been called...more or less appropriately... “chronic necrotizing hepatitis”. This applies to cases in which an impressive clinical picture shows frequently recurring acute necrotic attacks. The patients are usually middle-aged women (between 30 and 50). The prognosis is less favourable than in the other forms of chronic hepatitis. After a number of necrotic attacks, a post-necrotic cirrhosis often develops which frequently leads to an inevitable fatal ending.

The histological control of these cases by means of liver biopsy does not show a uniform picture. Some cases do not reveal any morphological characteristics at all, because the alterations are not widely spread and may be missed in the biopsy.

But on the other hand one does encounter a characteristic picture. Here, at the beginning there are confluent micronecroses which are changed into microscars. These confluent microscars change into larger scar areas with insular parenchymal remains in the scarred area beside large sections of normal and unchanged parenchyma.

This chronic necrotizing hepatitis therefore is an intermittent partial liver necrosis with signs of chronic scarring inflammation. Further details will soon be published.

I shall at first show you pictures of the early stage with the confluent necroses and the beginning formation of scars. Then come confluent microscars with parenchymal remains beside the normal unchanged parenchyma.

As a rule, the prognosis of chronic persisting hepatitis is favourable. Hardly ever does it change into an aggressive hepatitis or even a cirrhosis.

However, chronic aggressive hepatitis, particularly at the stage of high inflammatory activity, frequently changes into a complete hepatic cirrhosis. Originally we assumed that about 50% of such cases would have turned into a cirrhosis after a few years, but this figure is probably far too high. Today we are convinced that no more than 10-20% of chronic aggressive hepatitis
will lead to a cirrhosis. This improved prognosis may be partially credited to the therapy, but it is also due to the fact that we are now better able to diagnose the cases correctly; and above all, we have learned that a complete cirrhosis should be less often suspected than was done earlier.

As already mentioned, the prognosis of chronic necrotizing hepatitis is particularly bad in view of the possible change into a complete cirrhosis. In these cases a postnecrotic cirrhosis will occur. We shall then see broad scarred areas with insular parenchymal remains in-between which are more or less well defined by regeneration.

All cases of chronic hepatitis can result from an acute virus hepatitis. Chronic persisting hepatitis in particular is strongly linked to acute virus hepatitis, with a rate of well above 50%. Aggressive and also necrotizing chronic hepatitis are less often preceded by an acute hepatitis. Diagnoses by means of biopsy left us with the impression that only about 10% have an acute preliminary stage. However, the finding of Australia antigens in more than 50% of the cases suffering from chronic aggressive hepatitis contradicts our assumption.

Yet, to what degree the Australia antigen is specific to virus hepatitis needs still to be clarified in my opinion.

A considerable number of cases of chronic aggressive hepatitis is certainly primary chronic analogous to primary chronic rheumatoid arthritis, primary chronic glomerular nephritis and endocarditis.

In numerous cases of chronic aggressive hepatitis that are negative with respect to Australia antigen, immunologically demonstrable cell antigens are found which through formation of antigen-antibody complexes possibly determine the progress, the selfperpetuation of the chronic hepatitis. This is indicated by the results obtained from examining semi-thin sections after embedding of methacrylate and silver impregnation... according to MOVAT. However, at this time a definite etiological interpretation is not yet possible. But we hope that the progress in immunology will lift the fog around the etiology of the chronic hepatitis. On the other hand I believe that morphological investigation will contribute little, if anything, to the further understanding of the phenomenon of chronic hepatitis.