The Chairman announced that he had received apologies for absence from Dr Jenkins and Dr Waiter, and that Dr Buxton had come from the DHSS in place of Dr Waiter.

He then began the discussion by asking what evidence there was for the non-parenteral spread of non-A non-B hepatitis in Britain. Dr Vandervelde presented results of a continuing serological study on cases of non-B hepatitis at the Virus Reference Laboratory, Colindale (NARI 79/2). The study showed that sporadic cases of hepatitis arose which, though clinically diagnosed as viral hepatitis, lacked laboratory markers of hepatitis A virus (HAV) and hepatitis B virus (HBV) infection. These cases occurred mostly in an older age group than hepatitis A, and did not seem to be associated with intrafamilial spread. No other evidence of possible non-parenteral non-A non-B hepatitis in Britain was presented.

Discussion then turned to parenterally-transmitted non-A non-B hepatitis. Professor Zuckerman instanced an outbreak in a dialysis unit in Fulham, and the continuing occurrence of non-B hepatitis in patients receiving blood products, particularly factor VIII material. The study of post-transfusion hepatitis (PTH) conducted from the Central Middlesex Hospital had also suggested that some cases were not due to HBV. Dr Cleghorn said that his impression was that PTH must now be rare and that it would be difficult to find many cases.
1.4 million units of blood were transfused last year and very little had been heard of non-A non-B PTH. Professor Zuckerman pointed out, however, that much non-A non-B associated PTH might be anicteric, and that the risk of progression to chronic liver disease remained. Professor Sherlock, agreeing with Dr. Cleghorn that PTH was rare in the United Kingdom, was nevertheless concerned about the continued use of blood products of commercial origin. Many of these products were prepared in the United States, using blood from professional donors, and they carried a high risk of transmitting non-A non-B hepatitis.

Dr. Craske described the findings of his group, who were following up patients receiving factor VIII and factor IX preparations. Among some 1,800 haemophiliacs treated in 1978, 15 had developed hepatitis B and 20 non-A non-B hepatitis. Nine of the latter 20 cases were associated with blood products of NHS origin. There were also two or three cases per annum of non-A non-B hepatitis after administration of factor IX.

Sir William Maycock asked whether plans for the formal follow-up of cases confirmed that there was continuing follow-up of haemophiliacs under treatment.

The Chairman then asked what exactly constituted a case of non-A non-B hepatitis. It was agreed that HBV infection must be excluded by serological tests for HBsAg and anti-HBc, and that recent infection with HAV, EB virus, and cytomegalovirus must also be excluded. Blood enzyme tests, particularly SGPT, could be a useful pointer to non-A non-B infection, but there was an urgent need for specific markers of non-A non-B viruses. The Chairman suggested that Professor Sherlock and Zuckerman agreed, that until there were such markers, a survey of PTH, as suggested by Sir William Maycock was not warranted.

The Chairman drew attention to the two aspects of non-A non-B hepatitis on which the DHSS saw the need for research, and asked what studies were envisaged. Dr. Craske described the inoculation of chimpanzees in his Department. This product had caused non-A non-B hepatitis with a 10-week incubation period in patients at King's College Hospital. A paper giving his results was distributed, (NANB non-B). He intended to challenge the chimpanzees with a short-incubation agent soon. He felt that there was scope to apply the laboratory techniques for instance, the precipitin reactions between acute and convalescent sera in cases of non-A non-B hepatitis, reported by Japanese workers, (Lancet, 7 October 1978), could not be repeated elsewhere. He felt that chimpanzees were the only possible source of possible antigens and these animals were, however, expensive, their supply was limited, and maintenance costs were high.

Professor Sherlock suggested that sera should be gathered and stored until such time as specific tests for non-A non-B viruses were available. He would like to examine for markers of HAV and HBV infection stored sera from haemophiliacs studies that non-A non-B infection might severely liver already compromised by previous viral hepatitis.
quoted the view of American and German workers that up to 40% of non-A non-B infections progressed to chronic liver disease. He also had evidence of chronic liver damage in a chimpanzee inoculated with non-A non-B material.

Asked about studies on non-parenterally acquired non-A non-B infection, said that serological and epidemiological studies on sporadic non-B hepatitis would continue at the Virus Reference Laboratory. It was also intended to inoculate non-A non-B hepatitis material into marmosets in the colony there.

Sir William Maycock pointed out that it remained uncertain whether non-A non-B hepatitis virus was present in the British population and asked whether blood products of British origin were causing non-A, non-B hepatitis. Dr Craske thought that such cases certainly did occur but there was, however, no evidence of spread from the recipients of British products to other members of their family group.

Summing up, the Chairman suggested that support might be given to work with chimpanzees. Professor Sherlock should also be asked to review her cases of chronic hepatitis in relation to a history of blood transfusion, and might test them for markers of HAV and HBV infection. Dr Mortimer would brief Dr Pereira on the meeting, and requests for funds by the Public Health Laboratory Service would be considered sympathetically.

The meeting closed at 3.40 p.m.