Porcine Parvovirus (PPV) causes reproductive failure in pigs and is widespread throughout the world. Several studies were performed in pig herds [2, 3]. In this study, the antibodies titers in the sera of sow in one pig herd have been examined for 4 years on the same animals to observe the evolution of antibodies levels and of the PPV infection among the breeders.

MATERIALS AND METHODS: In a breeding and fattening pig unit holding 100 sows, serum samples of 3 to 5 sows of different ages were regularly collected each month during 4 years (from 1977 to 1980). The sows were individually identified and the sera-sample were collected from the same sows at each time. Of course, during these 4 years, some sows have been culled and others introduced. In this herd, the gilts are bred in the farm and in usual conditions, no breeder are coming from outside. Antibodies were detected by haemagglutination inhibition test (HAI) [1, 5]. The antibodies titers were expressed as the reciprocal of the highest dilution of serum inhibiting haemagglutination of 4 units of virus. A serum is considered as positive if antibodies have been detected after the dilution 1/20th. Antibodies or haemagglutinating antigen were detected in 70% of the sera from the same sow. The sera were collected in the farrow. The titers from February sow was, in general, stronger in the sows. The results did not show any cross-reaction at the same moment, as it is shown in the table 2, 97% of the tested sows were infected in May 1980.

RESULTS: In October 1977, the table I shows that, among the 4 groups included in this study, none of them has high titers of PPV antibodies in their sera (sows n° 3, 4, 5, 7), whereas others are not infected as no antibody is detected in the sera (sows 1, 2, 6). From December to February a seroconversion is observed in several sows and the sow n° 7 is a good example of this phenomenon. But, in successive sows, antibodies were not infected at this time in the sera of the INP haemagglutination inhibiting antibody appeared in their sera. This case is illustrated by the sows 2 and 6. At this period, reproductive disorders (maimed fetuses) have been observed in some litters, and the reproduction was limited in the herd. No serological investigation was performed. Then, all the gilts were brought in the fattening units and introduced in the breeding unit. At this time, the herd has no PPV antibody in their sera. No seroconversion is observed in these sera in May 1978 and until December 1979. As it is shown on the table I (sows 1, 6, 10, 12). Besides, the table 2 shows that, in proportion as young sows are introduced in the breeding unit, the percentage of the infected sows is 40%. The results do not show the replacement of the PPV infected sows by non infected gilts and not a decrease of the level of sero antibodies. Indeed, the table 3 shows clearly that PP antibodies titers are remarkably constant in the same sow during 4 years by taking account of technical variations. So, it seems that PPV haemagglutinating antibodies are persisting, without any variation, a very long time in the sera of the sows.

DISCUSSION: The results of this study show that PPV infection seems to occur in the herd after the infectious period in December 1977. Indeed, all the sows whose serum was free of antibodies did not show any seroconversion even after a contact with older infected sows. The epidemiological factors responsible of the spread of the virus in the herd are still unknown; some work demonstrated that the contact of herd management could have an influence on the probability of the infection of gilts or sows from infected sows [6]. In the case previously described, the conditions of herd management could have had an influence on the immunity level of the herd. Two years later, in December 1979, spontaneous recrudescence of the virus could have been occurred in the herd. The outcome of the infection could be, at this moment, a low immunity level of the herd or to still unknown epidemiological factors.