PART XVIII

WORKSHOP ON PREGNANCY AND THE KIDNEY
Drs Lamperi and Carozzi from Genoa [1] reported on immunological studies in 14 patients with normal pregnancy, 10 patients with pre-eclampsia, and 28 non-pregnant controls. They showed that normal pregnancy is associated with an overall decrease in cellular immunity, a decrease of the ratio helper/suppressor cells, and a fall in plasma Interleukin 2 and urinary Interleukin 1 activities. On the contrary, pre-eclampsia was associated with a decreased percentage of suppressor cells and an increased percentage of helper cells, leading to a higher helper/suppressor ratio. Spontaneous lymphocyte transformation index was increased in normal pregnancy and decreased in pre-eclampsia. The authors conclude that pre-eclampsia involves an abnormal immunological response of the mother to her fetus, and a deficient mother’s immune tolerance as compared to normal pregnancy. Dr Lindheimer (Chicago) pointed out that it would be necessary to have the same tests before pregnancy in order to know whether this is a basic pattern of patients or a peculiar response to pregnancy. In answer to another question, Dr Carozzi said that none of those patients has yet completed another pregnancy.

Dr Herzog from Tel Hashomer [2] reported on renal function during pregnancy in five-sixths nephrectomized rats. The authors observed that pregnancy in those animals is associated with an increase in creatinine clearance (from 327μl/min to 525μl/min), in fluid intake, and in fractional excretion of sodium and potassium. Urinary protein excretion increases sharply during pregnancy, and returns to the control values within 24 hours after delivery. At this time, CCr remains higher than in non-pregnant animals, but FENa and FECH3O are significantly lower. The author’s main conclusion is that a very reduced renal functional mass with an already high work load, is able to significantly increase in function during pregnancy. Dr Herzog had no explanation for the diminished FENa after pregnancy. Drs Lindheimer and Davison said that it could be related to the increased CCr, resulting in a rather identical elimination of the sodium load.

Dr Gokal from Manchester [3] presented the clinical features of six women
who had a nephrotic syndrome with high blood pressure and moderate renal
gain in the late second or early third trimester of pregnancy. In all cases,
blood pressure and renal functions had been normal until the onset of this
syndrome. Fetal outcome was particularly poor, since two deaths occurred
in utero and premature cesarean section was required in three other patients.
In all cases, proteinuria disappeared and blood pressure and renal function
returned to normal within six weeks post-partum. In answer to numerous
questions Dr Gokal said that all patients were primiparas, and that none had distinct clinical features like migraine or asthma. None of them had yet com-
pleted a second pregnancy. Dr Gokal had no renal biopsies and only one placental
histology for those patients. Several participants stated they had seen some
such cases, and all agreed that the fetal prognosis is very poor. A semantic
discussion on whether or not this could be named nephrotic syndrome did not
reach a satisfying conclusion.

Dr Cosci and co-workers from Lodi [4] analysed the clinical features in 43
patients with pregnancy hypertension classified according to the ACOG criteria.
They found that maternal and fetal prognosis were worse in group I (i.e. true
pre-eclampsia) than in any other group; hypertension was more severe and less
responsive to drug therapy, fetal growth retardation was common and perinatal
mortality was high. On the contrary in group II (i.e. chronic hypertension),
blood pressure responded well to drug therapy and complications were less
frequent. Previous hypertension, as well as high blood pressure one year after
delivery (overall incidence 30%), were considerably more frequent in group II
than in group I patients. Finally, persistence of proteinuria in six per cent of
patients suggested an underlying renal disease. Most participants agreed with
Dr Cosci’s data which are in keeping with most series in the literature. There
remain, however, some difficulties in accurately classifying some patients.
The limits of ‘true’ pre-eclampsia are not easy to draw, and the significance
of groups III and IV remains obscure. Dr Lindheimer summarized his group’s
experience of renal biopsies in patients classified in group I, and he emphasized
the pitfalls of this classification.

Dr Fievet and co-workers from Amiens [5] studied the effect of evening
primrose oil (a compound which is particularly rich in linoleic and gamma-
linoleic acids) in 15 pregnant patients at high risk of pre-eclampsia. The study
was blind, and olive oil served as placebo. The bleeding time increased in treated
patients. No significant changes occurred in the various coagulation tests per-
formed, nor in tests of platelet aggregation, except for spontaneous platelet
disaggregation (after ADP induced aggregation) which was significantly increased
in the treated group. The course of pregnancies was not strikingly different
between groups, as regards hypertension, symptoms of pre-eclampsia and dura-
tion of pregnancy. The birthweights were, however, significantly higher in the
treated group. The authors concluded that a supplemented intake of poly-
unsaturated fatty acids may have a beneficial effect on fetal growth in high risk
pregnancy. The discussion focused on the selection of patients. Dr Lindheimer
argued that a selection of patients ‘at risk’ of pre-eclampsia is very empirical
and leads to heterogeneous groups whose real risk is difficult to assess. This
problem is, however, difficult to solve, for in view of the frequency of spontaneous occurrence of pre-eclampsia and fetal growth retardation, such a study in non-selected primiparous women would require huge numbers of patients.

Papers presented

1 Lamperi S, Carozzi S. *Normal pregnancy and pre-eclampsia: immunological patterns*
3 Allen R, Gokal R, Donnai P, Mallick NP. *Early pre-eclampsia presenting as nephrotic syndrome*
4 Cosci P, Surian M, Malberti F, Corradi B, Colussi G, Minetti L. *Maternal and fetal prognosis and long-term follow-up in pregnancies with hypertension*
5 Fievet P, Fournier A, Tribout B, Castier B, Coevoet B. *Renin angiotensin aldosterone system and adrenergic system in pregnancy induced hypertension. Critical role of the stimulative conditions and of the lability of blood pressure in the assessment of their pathophysiological role*