Maternal plasma volume and disorders of pregnancy

Maternal plasma volume increases progressively during the second and third trimesters of pregnancy. The extent of the increase depends on the size of the conceptus, tending to be most in women with multiple pregnancies, and least in women with babies small for gestational age. Maternal plasma volume is also reduced in women with pre-eclampsia, a common cause of impaired fetal growth, although factors other than fetal size may contribute to the maternal hypovolaemia. Pregnant women with chronic hypertension are another group who may have reduced plasma volumes (as do non-pregnant subjects with essential hypertension), although in some studies it is not clear whether or not women with super-imposed pre-eclampsia were excluded. Thus a raised blood pressure may directly reduce plasma volume, but in pre-eclampsia, a more important factor may be a low concentration of albumin in the plasma, which is characteristic of the disorder. It is assumed that the hypoaalbuminaemia is due to albumin loss in the urine, after proteinuria develops, although this has never been proved. The possibility that albumin may leak into the extravascular tissues through abnormally permeable capillaries has not been substantiated. In women with pre-eclampsia the total intravascular albumin mass is reduced, although extravascular albumin remains unchanged and neither the exchange of albumin between the two compartments nor its catabolic rate changes. Though the cause of the low plasma albumin is thus unexplained, its reduced concentration lowers plasma oncotic pressure and predisposes towards loss of fluid from the vascular compartment. This explains the formation of oedema in severe pre-eclampsia and identifies a process which may further reduce the circulating plasma volume.

Plasma volume may be measured by safe dye dilution techniques but these are invasive and not used routinely in clinical practice. Haemoconcentration, however, may be readily assessed by measuring the packed cell volume. This reflects plasma volume depletion provided that the total red cell mass is not also reduced by blood loss or anaemia. Thus a high packed cell volume suggests hypovolaemia and increases the likelihood that there is placental insufficiency and impaired fetal growth.

It has been claimed that the hypovolaemia associated with pre-eclampsia may be the cause of poor tissue and organ perfusion leading to a maternal condition resembling circulatory shock. This is exacerbated by the increased blood viscosity which occurs in pre-eclampsia. Some workers have used plasma expanders to increase the plasma volume in such patients and claim that renal and possibly placental perfusion may be improved by such treatment. Others disagree and point out that cardiac output is well maintained or even increased in pre-eclampsia and that, although the plasma volume is reduced, the capacity of the circulation may be reduced to a similar degree so that it is not underfilled. Sudden expansion of the plasma volume increases albumin loss from the capillaries into the extravascular space. If the infused plasma expanders (colloids) leak into the extravascular space the oedema will increase and dangerous complications such as pulmonary or laryngeal oedema may ensue. A further potential problem with this form of treatment is the possibility that hypervolaemia rather than hypovolaemia may occasionally complicate pre-eclampsia.

Goodlin and his colleagues have recently reported the results of plasma volume determinations in 200 women with various complications of pregnancy. Their purpose was to determine if any simple clinical investigation could be used to identify those women with hypovolaemia. Some of the test results (renal function, packed cell volume, serum albumin concentration) are likely to correlate with plasma volume because they measure variables which are directly related. Others, such as the platelet count or changes in liver enzyme activities, should reflect the severity of abnormalities in the clotting system or in the liver in pre-eclampsia but are unlikely to be related directly to hypovolaemia. Not surprisingly, the authors conclude that no routine clinical measurement accurately identifies the pregnant women with hypovolaemia; in other words, the only way to determine plasma volume is to do so directly.

This report assumes that depletion of plasma volume is the cause, not the consequence, of both fetal growth impairment and the maternal systemic disturbances in pre-eclampsia. The authors state that “a major goal of antenatal care should be an expansion of plasma volume in pregnant women.” This assumption is unproven but could be tested by a randomised controlled trial to see if expansion of the plasma volume has any beneficial effect on the outcome of pregnancy. No such study has yet been described. The importance of maternal plasma volume in disorders of pregnancy remains unclear. On present evidence, therefore, Goodlin’s recommen-
dation that plasma volume estimations should be a part of routine clinical investigation cannot be accepted.

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