Coital rate and pregnancy-induced hypertension

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Previous workers have emphasized the possibility that pregnancy-induced hypertension (PIH) is associated with a change of sexual partner. It is noted here that any such association is far weaker than that between PIH and fecundability (the probability of conceiving in a month at risk). This latter association is so strong as to suggest its closeness to a true cause. Of the determinants of fecundability, coital rate seems the most promising to pursue as a possible cause of PIH.

Key words: coital rates/fecundability/paternal effect/pregnancy-induced hypertension

Gratacós et al. (1996) report that previously normotensive women who later develop pregnancy-induced hypertension (PIH) have a highly significantly shorter duration of unprotected sexual cohabitation (conception wait) than controls, on the average. This was so, regardless of the type of contraception previously used (condom or oral). These authors accordingly suggested that the risk of PIH in primigravidae is reduced by exposure to paternal spermatozoa. They invoke the hypothesis that PIH has an immunological basis on the ground that PIH is more common in primigravidae and “rarely affects multigravidae unless there is a change in paternity”. However, this notion that changes in paternity affect the risk of PIH has been challenged by Adams et al. (1996) on a far larger sample, namely 139 085 women, whose first and second births were live singletons for whom the fathers were identified, and occurring in Georgia 1980–1992. It is worth briefly reproducing the data of these latter authors. They reported that: (i) the rate of pre-eclampsia/eclampsia (PE/E) in first pregnancies was 4.7% and in second pregnancies 1.4%; (ii) among women who did not have PE/E in their first pregnancy, the rate in the second was 1.0% when the father was the same and 1.4% when the father was different; (iii) among the 6521 women who had PE/E in the first pregnancy, the rate in the second pregnancy was 10% when the father was the same, and 8.9% when the father was different. As these authors write: “These findings do not support previous reports that changes in paternity influence risk of PE/E”.

Nevertheless one may acknowledge that the data of Robillard et al. (1994, 1996) suggest that PIH is associated both with changes in paternity and with short conception waits. So it is interesting to consider whether these are independent risk factors in these data. This question may be addressed by assessing whether, in women who have a fresh partner, the reduction of conception wait associated with PIH is as great as in women who are having their first pregnancy. In fact, the reductions are of the same order: in both categories of women, the conception waits of cases were about one-third those of controls. I would infer that the primary variable here is the duration of unprotected sexual cohabitation rather than any change of father.

So the question arises: why should duration of sexual cohabitation be so strongly associated with PIH if, as seems possible, any association with change of, or protection of, paternal spermatozoa may provisionally be discounted?

Couples vary substantially in their fecundability (the probability $P$ of conceiving a live-birth conception in a month at risk) (Bongaarts, 1975). So those who achieve a conception in a short time (as contrasted with those who take longer) do not do so at random, but because, on the average, they have a higher fecundability. Since PIH seems so strongly associated with fecundability, it is worth specifying the individual components of fecundability. The probability $P$ of conceiving a live-birth conception in a month at risk is the product of four probabilities: $P_1$, the probability that the woman will ovulate during that month; $P_2$, the probability that insemination occurs within the fertile interval (that time period spanning ovulation during which the probability of conception is non-zero); $P_3$, the probability that (given that insemination has occurred during the fertile interval) the ovum is fertilized; and $P_4$, the probability that spontaneous abortion does not ensue.

In principle, it is possible that the association between PIH and fecundability may be due to any combination of these four parameters. But $P_2$ (which is determined by coital rate) seems a likely candidate for three reasons: (i) coital rates vary greatly between couples (James, 1981a); (ii) Marx et al. (1981) offered the suspicion that sexual activity is directly related to pre-eclampsia because (according to their observation) young pre-eclamptic patients were usually accompanied by male companions in labour rooms while controls were accompanied by female family members. These authors also documented this suspicion with data suggesting that coitus after conception was highly significantly associated with pre-eclampsia; (iii) Robillard and Hulsey (1996) report two PIH patients in protracted relationships mainly spent apart except for brief
holidays together. Coital rates rapidly decline across time in normal conjugal relationships (James, 1981b, 1983). But it is reasonable to suppose that in these cases of intermittent union, coital rates would be high during the limited times when the couples were re-united.

The data cited above seem consistent with the notion that PIH is associated with high coital rates. This may be for either (or both) of two reasons: (i) it is possible that high pre-pregnancy coital rates are associated with high maternal steroid hormone concentrations which are later associated with PIH as previously suggested (James, 1995); (ii) it is also possible that high pre-pregnancy coital rates are associated with high coital rates during pregnancy and that these in turn initiate PIH.

If high coital rates were causally associated with PIH, this would explain the association of PIH with primiparity. Coital rates prior to first pregnancies would be expected to be substantially higher than those preceding subsequent pregnancies. Moreover, any association of PIH with a change of partner would then plausibly be explained as secondary to the boost given to coital rates by a new partner.

PIH doubtless has a number of causes, but the very strong association with fecundability and, by inference, with coital rate should be pursued.

References

Received December 12, 1996; accepted April 10, 1997