Letters to the Editor

Uterine contractility decreases at the time of blastocyst transfer

Dear Sir,

Fanchin and co-workers (Fanchin et al., 2001) should be congratulated for publishing a very well written paper on the role of uterine contractions in assisted reproduction. They provide convincing evidence that uterine contractility at the time of theoretical blastocyst transfer (7 days after HCG injections or 5 days after oocyte retrieval) is significantly lower than at the time of typical embryo transfer on day 2. We fully support their conclusion that such a quiescent status of the uterus facilitates implantation and therefore offers an additional explanation for the high success rate reported after blastocyst transfer. It is in agreement with our earlier research (Lesny et al., 1998a), when we observed a decreasing number of contractions and changes in contraction patterns in oocyte donors 2, 3 and 4 days after oocyte retrieval. We were the first to evaluate junctional zone contractions during IVF cycles with the use of digitization and computer technology and are pleased that our findings are now confirmed by a larger study in which sophisticated equipment was used to assess uterine contractility.

Fanchin and co-workers were concerned about an imperfection in their study design due to embryos already present in utero on the day of theoretical blastocyst transfer (Fanchin et al., 2001). This is probably irrelevant, as our study was conducted in the absence of embryos (oocyte donors subjected to luteal support) and our observations were reassuringly similar.

We read with interest the discussion about the implications of the negative correlation between progesterone concentrations and uterine contractility. The evidence presented by the authors is very sound, but other factors influencing the luteal phase and present at the time of embryo transfer should also be taken into consideration. We observed very rapid uterine contractions immediately after oocyte retrieval, which were not noted at the time of HCG injection or even immediately prior to oocyte retrieval (Lesny et al., 1998a). This suggests the release of prostaglandins and/or other inflammatory reaction mediators from the ovaries, as described during ovulation in animal models (Espey, 1992; Kannisto et al., 1992; Wallach and Dhamurajan, 1992). It is likely that this effect persists into the luteal phase and, together with supra-physiological levels of estradiol and only very brief exposure to progesterone, could be responsible for more exaggerated contractions in an IVF cycle (Lesny et al., 1998a) when compared with natural cycles (Ijland et al., 1996). Moreover, there is no clear explanation as to whether local factors or neural stimulation are responsible for uterine contractility following a difficult embryo transfer (Lesny et al., 1998b) or the application of a tenaculum to the cervix (Lesny et al., 1999). We observed turbulent endometrial movements, reflecting junctional zone contractions, persisting for an hour after a stimulating event, but this activity could have lasted longer. As the precise mechanism for these contractions is yet to be determined, pharmacological control to reduce contractility and improve results from treatment remains elusive. We do hope that our letter will enrich discussion and emphasise the importance of this very interesting paper.

References


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