as may sometimes occur after midcycle postcoital insertion. He claims for ordinary IUD usage that "There is little compelling evidence to suggest that copper IUDs reliably eliminate the likelihood of fertilization" but fails to cite any of the four principal reviews on the copper IUD's strong effects in reducing fertilization. One missed review demonstrates the direct, deleterious effects of copper on sperm motility and on sperm penetration into cervical mucus and shows that copper IUDs induce head tail separations in the few sperm reaching the oviduct.[1]

Another missed review summarizes statistical evidence of significant reductions in the percentage of IUD users with fallopian tube sperm shortly after intercourse, with greater reductions associated with copper IUDs.[2] This review also reanalyzed primate data, in IUD-fitted macacques, finding no increase compared with control cycles for the same animals in the proportion of failed implantations but a statistically significant increase in proportions of midcycle inseminations without traces of fertilization.

Demonstration of the reduction in fertilization rates in women with IUDs by study of the developmental status of ova in the oviducts of IUD users and of controls is weak, according to Spinnato, because some eggs "were actually not classifiable and could have been fertilized." There is no ambiguity, however, between the 15% of controls and 64% of IUD subjects who had, unambiguously, no signs of development (no fertilization) ($p < 0.01$).[2]

Spinnato believes that the high IUD ratio of ectopic to total pregnancies results from prevention of implantation. However, the still higher ectopic ratio associated with tubal ligation, with no known mode of postfertilization action, shows that this argument is fallacious. The critical observation about ectopic pregnancies and IUDs is their annual rate. For the sole copper IUD available in the United States today the rate is 0.2 to 0.4 per 1000 women per year, 5% to 10% of the rate in women not using contraception. A 90% to 95% reduction in ectopic pregnancy rates by this copper IUD is a powerful indicator of sharply reduced risks of fertilization.

A truly informed consent should be unbiased. An unbiased review of copper IUD actions, with the device in situ before coitus, would find its primary action is to prevent fertilization, chiefly by cytotoxic effects on sperm.

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Reply

To the Editors: Editorial requirements of the *American Journal of Obstetrics and Gynecology* required me to reduce my references from 61 to 25. I did not "miss" articles but was required to restrict my citations.

Sivin misquotes me. Throughout my article I acknowledge a spermicidal effect of IUDs. The issue I addressed was whether, as some recent authors have suggested, the available evidence supports a claim of exclusivity of this action. There is clear and compelling evidence for a postfertilization mechanism of action of IUDs that refutes such claims.

Sivin objects to my criticism of Alverez et al.[1] My concerns as indicated in my article remain firm. Sivin displays unreasonable confidence in a single cycle study of nine IUD users with uncontrolled timing and certitude of insemination (excluding five patients with ambiguous results).

Sivin argues that evidence of an increased risk of ectopic gestation after tubal ligation is somehow pertinent to this issue. Analogies are inherently weak arguments, and this one is particularly so. The mechanism of ectopic pregnancy after tubal ligation (mechanical injury to the tubes) is not particularly pertinent.

Sivin claims that there is a 90% to 95% reduction in ectopic pregnancy among copper IUD users. He provides no reference. In a study on ectopic pregnancy, Ory[2] more modestly estimates a 60% reduction in ectopic risk among IUD users versus uncontracepted control patients. An extrapolation of this data would leave potentially 40% of an IUD's mechanism as postfertilization, a major portion. This is, at best, a rough estimate.

If Sivin is citing his own study[3] of 35,496 insertions of copper-bearing IUDs, the limitations of this study should be noted. Fewer than one half of patients (15,244) were deemed to have had adequate follow up. What happened to the other 18,000+ patients is unknown. Most of the patients received their continuing health care at a different site. The bottom line is that one cannot conclude what the ectopic rate might be with such poor follow-up.

For prevention of fertilization to be the "primary" mechanism of action of IUDs, as Sivin states, requires that mechanism to explain just >50% of the action. This would still allow prevention of implantation to remain a major mechanism of action for IUDs, and that's the point. Information to practitioners and informed consent to patients should reflect this.

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