Pelvic floor function and childbirth

To the Editors: Klein et al. in their article on pelvic floor function after childbirth (Klein MC, Janssen PA, MacWilliam L, Kaczorowski J, Johnson B. Determinants of vaginal-perineal integrity and pelvic floor functioning in childbirth. Am J Obstet Gynecol 1997;176:403-10) claim that they were "unable to find another study that used the same perineometer that we had" and that "we were unable to find any published pregnancy or prepregnancy perineometry norms." Thus their literature review failed to discover our studies using an identical instrument in which we validated the reproducibility and established normative data in nulliparous women[1] and examined the effects of delivery on surface electromyography performance.[2] Likewise, they describe our prospective, controlled study of midline episiotomy[3] as a "retrospective study." Pelvic floor function after childbirth is an important field and I appreciate the contributions of Klein et al. I would encourage more thoroughness in their review of the literature.

John M. Thorp, Jr., MD

Department of Obstetrics and Gynecology, University of North Carolina, CB #7570, 214 MacNider Building, Chapel Hill, NC 27599-7570

REFERENCES

1. Thorp JM Jr, Bowes WA Jr, Droegemueller W, Wicker H. Assessment of perineal floor function: electromyography with acrylic plug surface electrodes in nulliparous women. Obstet Gynecol 1991;78:89-91.
2. Thorp JM Jr, Jones LG, Bowes WA Jr, Droegemueller W. Electromyography with acrylic plug surface electrodes after delivery. Am J Perinatol 1995;12:125-8.
3. Thorp JM Jr, Bowes WA Jr, Brame RG, Cefalo RC. Selected use of midline episiotomy: effect on perineal trauma. Obstet Gynecol 1987;70:260-2.

6/8/84412

Reply

To the Editors: We are pleased to respond to Thorp, recognizing that his work, which was indeed prospective, suggested the link between median episiotomy and third- or fourth-degree tears. With larger numbers, within our randomized controlled trial of episiotomy we were able or confirm that in nulliparous women median episiotomy appears causally related to almost all such trauma.[1]

Sadly, we were unable to benefit from the work of Thorp et al. cited in Thorp's first reference. We see now that these authors used a similar instrument and clearly studied and wrote about pelvic floor functioning. But they titled it "perineal floor" functioning. Such a structure does not exist. Thus our MEDLINE search under "pelvic floor" failed to locate the article.

These authors were unable to demonstrate a beneficial effect for Kegel exercises in pelvic floor functioning and speculated that this might have been due to their small numbers. They studied only 41 patients, showing great variability and range. But in our study of 459 nulliparous women we came to a similar conclusion,[2] and unreported for a similar number of multiparous subjects as well.

Unfortunately, our search failed to pick up their reference 2 because of the closeness in time between manuscript preparation and publication. In 52 patients at a mean of 46.3 days post partum, they reported results similar to ours with 359 randomized nulliparous and 341 multiparous women[1] and 459 randomized and nonrandomized nulliparous women.[2] They report, however, that women delivered vaginally had lower flick voltage than those delivered abdominally. In general, we agree, but we found at 3 months post partum in both nulliparous and multiparous women, with use of "flicks" and the more reliable 10-second "holds," that women with an intact perineum had electromyographic voltages similar to those of women with cesarean birth. Progressively lower voltages were found in subjects sustaining second-degree tears, episiotomy, and episiotomy extensions. Thus, with sufficient power, we were able to sort out the apparent protective effect of attempting to assist birth with an intact perineum. Thorp failed to cite this 1994 study[1] in the 1995 publication and apparently missed our 1992 randomized controlled trial,[3] providing normative electromyographic pelvic floor data at 36 weeks' gestation and 3 months post partum.

To really understand pelvic floor functioning, we need a prospective study among women intending to become pregnant and followed up through pregnancy, post partum, and beyond. Because we share research interests with Thorp, we will have to communicate more directly and consider collaboration.

Michael Klein, MD

Department of Family Practice, BC Women's, F412, 4500 Oak St., Vancouver, British Columbia, Canada V6H 3N1

REFERENCES

1. Klein MC, Gauthier R, Robbins J, Kaczorowski J, Jorgensen S, Franco E, et al. Relation of episiotomy to perineal trauma and morbidity, sexual dysfunction, and pelvic floor relaxation. Am J Obstet Gynecol 1994;171:591-8.
2. Klein MC, Janssen P, MacWilliam L, Kaczorowski J, Johnson B. Determinants of vaginal-perineal integrity and pelvic floor functioning in childbirth. Am J Obstet Gynecol 1997;176:403-10.
3. Klein MC, Gauthier RC, Jorgensen SH, Robbins J, Kaczorowski J, Johnson B et al. Does episiotomy prevent perineal trauma and pelvic floor relaxation? Online J Curr Clin Trials [serial online] 1992 Jul 1;2 (Doc No. 10); [6019 words; 65 paragraphs]. 1 figure 6 tables.

6/8/84413

Mechanism of action of intrauterine contraceptive devices

To the Editors: Spinnato (Spinnato JA II. Mechanism of action of intrauterine contraceptive devices and its relation to informed consent. AmJ Obstet Gynecol 1997;176:503-6) seeks revision of intrauterine contraceptive device (IUD) informed consent to emphasize the device's putative postfertilization inhibition of uterine implantation,

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.

Irving Sivin
Center of Biomedical Research, 1230 York Ave., New York, NY 10021

REFERENCES

1. Ortiz ME, Croxatto H. Mode of action of IUDs. Contraception 1987;36:37-53.
2. Sivin I. IUDs are contraceptives not abortifacients: a comment on research and belief. Stud Fam Plann 1989;20:355-9.

6/8/84469

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.

J.A. Spinnato, MD
Department of Obstetrics and Gynecology, University of Louisville School of Medicine, 550 S. Jackson St., Louisville, KY 40292

REFERENCES

1. Alvarez F, Brache V, Fernandez E, Guerrero B, Suiloff E, Hess R, et al. New insights on the mode of action of intrauterine contraceptive devices in women. Fertil Steril 1988;49:768-73.
2. Ory HW. The Women's Health Study, ectopic pregnancy and intrauterine contraceptive devices: new perspectives. Obstet Gynecol 1981;57:137-44.
3. Sivin I. Copper T IUD use and ectopic pregnancy rates in the United States. Contraception 1979;19:151-73.

6/8/84468