jections spans 40 years using doses of up to 100 mg, but it has always been emphasised that injections must be given into the gluteal muscles, in which fat cells are intermingled with muscle cells, but never into the thigh or deltoid muscles, which contain no fat cells.

The finding of Phipps et al. that gram-stained fluid aspirated from the site showed no viable bacteria confirms my own findings that aspirations from progesterone injection or implantation sites are always sterile. Consequently, their extensive and diverse use of antibiotics would have little effect. Perhaps there is a need to emphasize that whenever the words "intramuscular progesterone" are used they should be linked with the words "injected into the gluteal muscles."

Dr. Katharina Dalton
London, England
November 28, 1988

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2. Greenblatt DJ, Allen MD: Intramuscular injection size complications. JAMA 240:542, 1978
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Reply of the Author:

I appreciate the opportunity to respond to Dr. Dalton's letter. Dr. Dalton's vast experience with progesterone administration is well known and respected, but I believe her conclusions relating to our report to be in part unfounded.

The manufacturer's package insert for the progesterone preparation we used (50 mg/ml in peanut oil; Eli Lilly, Indianapolis, IN) does not recommend the use of one intramuscular (IM) injection site over another. Our patient used the vastus lateralis muscles bilaterally because of the ease of self-administration of IM injections into these sites, and her previous experience with such injections for other medications.

The technique advocated for gluteal IM injections of progesterone by Dr. Dalton[1] uses a 1.5-inch (3.8 cm) needle. It has been clearly demonstrated that, for a substantial majority of women, because of the thickness of the layer of fat overlying the gluteal muscles, such a technique in fact will not result in a truly IM injection, but rather a deep subcutaneous one.[2,3] Thus, Dr. Dalton's success with this technique over the years is unlikely to be related to the intermingling of fat cells with gluteal muscle fibers as she states. Nonetheless, it may well be that, for progesterone injections, the gluteal region is a superior site compared with the anterior thigh, perhaps precisely because the injection is given into the subcutaneous fatty tissue and not muscle. Certainly the use of subcutaneous injections does not lead to the development of myofibrosis, which may occur as a consequence of repeated IM injections.[4]

Based on the clinical findings and our patient's response to clindamycin, all of the physicians involved with her care, including an infectious disease specialist, were unanimous in the belief that a substantial secondary bacterial infection had occurred. Thus, I believe that the extensive use of antibiotics as described was absolutely necessary. Our patient's myositis was quite different from the gluteal region sterile abscesses Dr. Dalton has described as occurring in some of her patients,[1] which in fact are probably subcutaneous abscesses.

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January 17, 1989

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Progesterone for Diagnosis of Ectopic

To the Editor:

I wish to comment on a paper recently published in *Fertility and Sterility* in which a single serum progesterone (P) measurement was used for timely diagnosis of early ectopic pregnancy. In this manuscript by Yeko et al.,[1] a dilatation and curettage was recommended if the serum P level was <15 ng/

ml. They did not fear harming a viable intrauterine pregnancy since 17 of 17 patients with an intrauterine pregnancy and P level <15 ng/ml had spontaneous abortions.

However, they make no mention of any attempts to salvage these pregnancies with progesterone therapy. We aggressively treated 27 patients with intrauterine pregnancies presenting with an initial P level during their first trimesters of <15 ng/ml with parenteral P and P by vaginal suppositories. Nineteen (70%) successfully completed their first trimesters.

Patients unwilling to risk the loss of a potentially viable fetus might better be followed by conventional serial human chorionic gonadotropin levels and pelvic sonography with a transvaginal probe.

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Reproductive and Medical Endocrine Associates, P.C.
Melrose Park, Pennsylvania
October 4, 1988

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Does Minimal Endometriosis Cause Infertility?

To the Editor:

Rodriguez-Escudero et al.[1] properly conclude that normal fecundity is possible with endometriosis and azoospermia treated with donor insemination. In a small sample, they report fecundity of 0.20 monthly. In contrast, they found 0.06 fecundity for endometriosis without anatomic distortion treated expectantly.

Although some women with endometriosis do not have reduced fecundity, it is misleading to compare women with infertility with those with no chance for fertility. Women with minimal endometriosis[2] are a heterogeneous population. The possible contribution of minimal endometriosis to subfertility is most appropriately studied in women with infertility in whom no other cause can be found.

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December 12, 1988

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Reply of the Authors:

The possible role of minimal endometriosis in female infertility is very controversial. The prevalence of minimal endometriosis in a normal population is unknown, and it is impossible to ascertain whether, when discovered in the evaluation of an infertile couple, the endometriosis is a causal phenomenon or an incidental finding.

I do not see the point of Drs. Tummon and Colwell's letter. Why is it misleading to compare a group with minimal endometriosis treated by therapeutic donor insemination (TDI) with a group not treated with TDI? Furthermore, the first group fulfills the last statement of Drs. Tummon and Colwell's letter. The fact is that after correcting the azoospermia, the fecundity rate of minimal endometriosis was normal.

Therefore, we think that it is fairly possible that minimal endometriosis ought to be considered as a coincidental finding rather than as a causal factor of infertility.

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