

## What about repair of recurrent rectocele?

### TO THE EDITOR:

**D**r. Marvin H. Terry Grody's description of rectocele repair, "Defect-directed reconstruction: The common-sense technique for rectocele repair" (January 2004), offered a refreshing new perspective. By identifying connective-tissue tears and repairing them individually, this approach makes more anatomic sense than "mass closure." Using Dr. Grody's method, these defects may be readily identified and repaired at the initial operation.

But what about the recurrent rectocele? Does defect-directed repair still apply? Is it even possible? Also, at the time of the primary or repeat repair, isn't it possible for attenuated Denovilliers' fascia to be present, as I believe I, and others, have encountered? By definition, isn't a rectocele a type of hernia, the result of weakened in situ fascia, abnormal collagen, or previous surgery?

The use of biological or synthetic graft sources for pelvic floor reconstruction also has been described.<sup>1</sup> Does Dr. Grody care to comment on the use of these materials for site-specific repairs or for large, recurrent, posterior vaginal wall defects using the evaluation and techniques described in his article?

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### REFERENCE

1. Kohli N, Miklos JR. Dermal graft-augmented rectocele repair. *Int Urogynecol J Pelvic Floor Dysfunct.* 2003;14:146-149.

**DR. GRODY RESPONDS:** The chances are overwhelming, in cases of recurrent rectocele seen today—when we are still in the infancy of defect-directed reconstruction—that any previous colporrhaphies were performed using the traditional, now archaic, method. This aged, misconceived technique brought together adventitial scraps and whatever was available, often under tension, from the lateral walls into the area of the midline. So the original torn sec-

tions of rectovaginal septum, retracted out into the fringes surrounding the bulging defect, still secluded and undisturbed, are waiting for discovery, to be followed, it is hoped, by reapproximation in the defect-directed repair. The size of the rectocele is totally inconsequential; the torn edges are always found at its borders.

Attenuation uncommonly is a secondary problem; usually it is a false perception because of imprecise and inadequate dissection. However, it can exist, along with tissue deterioration in cases of severe hypoestrogenism, such that defect-directed repair is not an option. In these situations, the only recourse is a prosthesis in the form of a graft—either xenograft or allograft or, my preference, synthetic polyester graft. Proximal anchorage for the graft may be a problem best solved by employing the intact cardinal-uterusacral stumps or the sacrospinous ligaments. In either instance, to prevent later enterocele, the physician should compensate for central vulnerability. Of course, local estrogen should be initiated immediately in the postoperative regimen.

A rectocele is unquestionably a form of hernia. Hernias occur secondary to disruption of connective tissue layers or fascial sheets. Regardless of the etiology of hernias elsewhere in the body, it is now an accepted fact that the herniation of the rectum into the posterior vagina, ie, rectocele, begins almost always from tears in the rectovaginal septum. Most experts today also agree that congenital collagen deficiency syndrome increases the chances of these tears.

Returning to the use of grafts, I feel they are mandatory in 2 situations:

- when the septal remnants shred as the Allis clamps attempt to reunite the edges and
- in all cases, regardless of observed quality of septal tissues, where 2 or more repair failures have occurred. ■