Laparoscopic Heller Myotomy and Dor Fundoplication for Achalasia

Analysis of Successes and Failures

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Background: In the treatment of achalasia, surgery has been traditionally reserved for patients with residual dysphagia after pneumatic dilatation. The results of laparoscopic Heller myotomy have proven to be so good, however, that most experts now consider surgery the primary treatment.

Hypothesis: The outcome of laparoscopic myotomy and fundoplication for achalasia is dictated by technical factors.

Setting: University hospital tertiary care center.

Design: Retrospective study.

Patients and Methods: One hundred two patients with esophageal achalasia underwent laparoscopic Heller myotomy and Dor fundoplication. Fifty-seven patients had been previously treated by pneumatic dilatation or botulinum toxin. The design of the operation involved a 7-cm myotomy, which extended 1.5 cm onto the gastric wall, and a Dor fundoplication. Esophagrams, esophageal manometric findings, and video records of the procedure were analyzed to determine the technical factors that contributed to the clinical success or failure of the operation.

Main Outcome Measure: Swallowing status.

Results: In 91 (89%) of the 102 patients, good or excellent results were obtained after the first operation. A second operation was performed in 5 patients to either lengthen the myotomy (3 patients) or take down the fundoplication (2 patients). Dysphagia resolved in 4 of these patients. The remaining 6 patients were treated by pneumatic dilatation, but dysphagia improved in only 1. At the conclusion of treatment, excellent or good results had been obtained in 96 (94%) of the 102 patients.

Conclusions: These data show that a Heller myotomy was unsuccessful in patients with an esophageal stricture; a short myotomy and a constricting Dor fundoplication were the avoidable causes of residual dysphagia; a second operation, but not pneumatic dilatation, was able to correct most failures; and that the identified technical flaws were eliminated from the last half of the patients in the series.

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Until recently, surgery for achalasia was usually reserved for patients with residual dysphagia after pneumatic dilatation. The excellent results of minimally invasive myotomy, however, have recently convinced many gastroenterologists and surgeons that surgery should become the primary treatment for this disease.1

Laparoscopic Heller myotomy and Dor fundoplication improves swallowing in more than 90% of patients.2,3 The causes of the few failures are still not completely understood. To identify the technical elements that affected the outcomes, this study analyzes the successes and failures of this operation.

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PATIENTS AND METHODS

Between October 1993 and July 2000, 102 patients with esophageal achalasia had a laparoscopic Heller myotomy and Dor fundoplication at the University of California, San Francisco. There were 48 females and 54 males, whose ages ranged from 14 to 85 years (mean, 46 years). The median duration of symptoms was 48 months. Fifty percent of patients had been previously treated by pneumatic dilatation (mean, 2 dilations per patient; range, 1-12), and 23 patients were treated by intraspheincteric injection of botulinum toxin (mean, 1.6 injections per patient; range, 1-3), either alone or in conjunction with pneumatic dilatation.

PREOPERATIVE EVALUATION

Symptoms

The ability to swallow was graded before and after surgery as follows: excellent (no dysphagia); good (occasional dysphagia); fair (frequent dysphagia requiring dietary adjustments); and poor (severe dysphagia preventing the ingestion of solid food) (Table).

Barium Swallow

The gastroesophageal junction was abnormally narrow in 92% of patients. The maximal esophageal diameter was less than 4 cm in 52% of patients, between 4 and 6 cm in 26% of patients, and more than 6 cm in 22% of patients. The esophageal axis was sigmoid-shaped in 7 (32%) of 22 patients whose esophageal diameters exceeded 6 cm and in no patient with an esophageal diameter less than 6 cm. In 2 patients, the barium swallow showed a corkscrew distal esophagus characteristic of diffuse esophageal spasm.

Endoscopy

No patient had a mechanical stricture of the esophagus diagnosed on esophagoscopy.

Esophageal Manometry

Manometry was performed following an overnight fast using techniques previously described. The mean ± SD resting lower esophageal sphincter (LES) pressure was 21 ± 11 mm Hg. Lower esophageal sphincter relaxation in response to swallowing was partial or absent in all patients.

CLINICAL OUTCOME

Good or excellent results were obtained after the initial operation in 91 (89%) of the 102 patients; specifically, in 41 (82%) of the first 50 patients (October 1993 to September 1997), and in 50 (96%) of the last 52 patients (November 1997 to July 2000). Eleven patients had either persistent (8 patients) or recurrent (3 patients; average symptom-free interval, 11 months) dysphagia. Based on the results of the postoperative workup and the review of the video records of the operation, the following 3 causes of failure were identified.

Short Myotomy

Four patients had a short myotomy. The myotomy was too short distally in 2 patients and too short proximally in 2 patients.

The 2 patients in whom the myotomy was too short distally had persistent dysphagia. Barium swallow radiographs showed persistent distal esophageal narrowing,
and manometry revealed a residual high-pressure zone that was approximately 1 cm long (Figure 2). A review of the video records revealed that the fat pad overlying the gastroesophageal junction had not been removed during the course of these operations, and consequently, the myotomy had been made shorter than intended (intraoperative endoscopy had not been used in either patient). In 1 of these patients, pneumatic dilatation permanently corrected the dysphagia; the other patient refused further treatment.

Two patients had diffuse esophageal spasms in addition to achalasia (Figure 3A), and after the first operation (ie, the laparoscopic Heller myotomy), dysphagia persisted. In both patients, postoperative manometry showed an LES pressure of 8 mm Hg and an absence of esophageal peristalsis (Figure 3B); the results of pH monitoring were normal. A left thoracoscopic myotomy was then performed, which eliminated the swallowing difficulties. The potential need for a 2-stage surgical treatment had been anticipated in these patients from the outset of surgery.

### Constricting Dor Fundoplication

Four patients had either persistent (2 patients) or recurrent (2 patients; symptom-free interval, 11 months) dysphagia due to mistakes in construction of the Dor fundoplications. Their postoperative manometry and pH monitoring measurements were normal. Video esophagrams showed no distal narrowing, but passage of contrast media from the esophagus into the stomach was slow.

In 3 patients, the video record of surgery showed that all the stitches on the right side of the fundoplication (rather than just the top stitch) had incorporated the right pillar of the crus in addition to the fundus of the stomach and the esophagus (Figure 1B). Furthermore, apical stitches had not been used (Figure 1B). Pneumatic dilatation was unsuccessful in these 3 patients. Subsequently, the fundoplication was dismantled in 2 patients (a second myotomy was performed in 1 patient), and their dysphagia resolved. Heartburn occurred afterwards; this was treated with acid-suppressing medications.

In 1 patient, the video record of surgery showed that the short gastric vessels had not been divided, and the body of the stomach, rather than the fundus, had been used to construct the fundoplication. This resulted in tension that pulled the right pillar of the crus toward the left, distorting the esophagus. The dysphagia was not improved after pneumatic dilatation, and the patient refused a second operation.

### Effect of Esophageal Diameter on the Results of Heller Myotomy

Good or excellent results were obtained in 75 (94%) of 80 patients whose esophageal diameter exceeded 6 cm, and in 21 (95%) of 22 patients whose esophageal diameter was less than 6 cm. On comparison, $P$ was not significant.

At present, 96 (94%) of the 102 patients judge their ability to swallow as excellent or good.

### Transmural Scarring Caused by Previous Treatment

A satisfactory myotomy could not be accomplished in 3 patients because of transmural fibrosis (a stricture) at the gastroesophageal junction. This unexpected finding, which had not been diagnosed preoperatively, had resulted from pneumatic dilatations in 1 patient and from intrasphincteric botulinum toxin in 2 patients. In these patients, the operation took longer, and the esophageal mucosa was perforated as the myotomy was being performed. Pneumatic dilatation was tried postoperatively without success. Subsequently, 1 patient had a second operation that involved takedown of the Dor fundoplication and a distal extension of the myotomy. Dysphagia persisted.

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gastroesophageal reflux.\textsuperscript{5,6} In one report of the cases of 46 patients with dysphagia following a Heller myotomy, a follow-up operation was performed to correct an inadequate myotomy in 37% of patients and to correct gastroesophageal reflux in 30% of patients.\textsuperscript{5} Stricture formation from gastroesophageal reflux has been said to be the most common cause of reappearance of dysphagia 10 years or more after a Heller myotomy.\textsuperscript{5}

Figure 2. A, Preoperative manometry. High-pressure (Press) zone (lower esophageal sphincter [LES], 3 cm long), localized in 4 channels located 1 cm apart from each other. B, Postoperative manometry. Residual high-pressure zone (LES, <1 cm long), localized in the lower channel.
The present study shows that inadequate myotomy, sphincter trauma from nonsurgical therapy, and constriction from an improperly made fundoplication were the principal causes of the few unsuccessful operations. Complications of gastroesophageal reflux were not seen, but the follow-up was short. Nevertheless, we are confident, based on our pH monitoring tests, that the Dor fundoplications will help surgeons avoid this problem.2,3

TRAUMA FROM PREVIOUS TREATMENT

Intrasphincteric injection of botulinum toxin was introduced at the beginning of the 1990s as a treatment for achalasia,7 but after some initial optimism, it was found to be ineffective. Consequently, botulinum toxin has now fallen into disuse.8 This period, however, coincided with that of the present study, during which severe inflammation of the LES was seen in approximately 30% of patients treated with botulinum toxin.9,10 In 3 patients, the reaction had progressed to a transmural stricture that involved the mucosa as well as the muscle. The operations in these cases were more difficult (eg, they lasted longer, and mucosal perforations occurred), and dysphagia persisted postoperatively. By contrast, a stricture was found in only 2% of patients previously treated by pneumatic dilatation.10,11 For unknown reasons, none of these strictures had been identified by endoscopy before surgery. With the abandonment of botulinum toxin therapy, this problem should become less common.

FAILURES OF HELLER MYOTOMY SECONDARY TO TECHNICAL ERRORS

A short myotomy (distally) was responsible for 2 failures. The videotapes of these operations showed that a fat pad at the gastroesophageal junction had not been removed, and the myotomy had been taken only a few millimeters onto the stomach. This fat pad, which is a common finding, must usually be removed in order to expose the gastroesophageal junction adequately. Intraoperative endoscopy might be used to see whether the entire sphincter has been completely transected,12,13 but external visual assessment is simple and accurate as long as the fat has been cleared. Incidentally, the use of endoscopy increased the duration (30-45 minutes) and cost (by approximately $3000) of the operation.

Faults in construction of the Dor fundoplication were responsible for 4 failures. These problems can be avoided by incorporating the crus in only the cephalad stitch of each row of sutures (Figure 1A), and by placing apical stitches between the anterior rim of fundus and the adjacent anterior hiatal margin (Figure 1A), which will offset any lateral torque exerted by the fundus. While we routinely divide the short gastric vessels to minimize lateral tension on the fundus, others have reported good results without this step.3,14 In 1 patient, however, retrospective review of the video record showed that division of the short gastric vessels would clearly have eliminated excessive tension.

Some surgeons prefer a posterior partial fundoplication instead of an anterior hemifundoplication, in conjunction with a Heller myotomy, in the belief that it more effectively prevents reflux and keeps the edges of the myotomy separate.15,16 Nevertheless, the simpler Dor fundoplication has been validated as an antireflux procedure, so these arguments are unpersuasive.3,14

Two patients whose disease was a mixture of achalasia and diffuse esophageal spasm required a second operation to extend the myotomy proximally.6 We might have chosen to exclude these patients from the list of failures since a Heller myotomy was the obvious first step. Alternatively, a thoracoscopic Heller myotomy might have
been selected for such patients since the myotomy could be made to span the entire distance from the sphincter to the inferior pulmonary vein at the first operation. The drawback here then, would be the inability to thoracoscopically perform a satisfactory antireflux procedure.

EFFECT OF ESOPHAGEAL DIAMETER ON THE RESULTS OF HELLER MYOTOMY

Good or excellent results were obtained in 75 (94%) of 80 patients whose esophagus had a diameter less than 6 cm, and in 21 (95%) of 22 patients whose esophagus had a diameter greater than 6 cm, including the 7 patients whose esophagus was sigmoid (P was not significant). When sigmoid changes are present, a more extensive dissection is necessary to straighten the esophagus before the myotomy is performed. This is a maneuver that is better done laparoscopically than thoracoscopically. These results show that an esophagectomy should practically never be the first approach for patients with a dilated sigmoid esophagus. It is a last resort, reserved for severe persistent uncorrectable dysphagia following a myotomy.

TREATMENT OF FAILURES OF HELLER MYOTOMY

Pneumatic dilatation was tried in 8 of 11 patients with persistent or recurrent dysphagia, but it was successful in only 1 (12%). Thus, our results contrast with those of Zaninotto et al, who reported that pneumatic dilatation was effective in 6 of 7 patients with unsuccessful laparoscopic Heller myotomies. Dilatation helped none of our patients with dysphagia resulting from a malconfigured fundoplication. The only patient who benefited from dilatation had a short distal myotomy.

When dysphagia persists or recurs after a Heller myotomy and Dor fundoplication, based on our results, one of the following may occur: (1) If the myotomy was too short distally, pneumatic dilatation should be performed. A second operation is indicated if dilatation is unsuccessful. (2) Patients with achalasia and diffuse esophageal spasm may require 2 operations before dysphagia is overcome: first, a laparoscopic Heller myotomy and Dor fundoplication; and second, a thoracoscopic myotomy carried out to the inferior pulmonary vein. (3) When the workup does not show the cause, dysphagia most often stems from tension on the esophagus by the plicated stomach. The diagnosis can be made and the problem corrected at surgery. (4) Pneumatic dilatation was of limited value for any of these problems.

Thus, laparoscopic Heller myotomy and Dor fundoplication was effective if the following technical objectives were achieved: (1) a 7-cm myotomy that extended 1.5 to 2.0 cm onto the gastric wall; and (2) a tension-free and properly oriented anterior fundoplication. Although the outcome in 10% of patients was unsatisfactory after the first operation, the reasons for the failures could be discerned, and in the second half of the series we were able to eliminate these technical flaws and their consequences. In patients with persistent dysphagia, the cause of failure can be identified, and treatment tailored to the cause will usually be effective.

REFERENCES


DISCUSSION

Bruce M. Wolfe, MD, Sacramento, Calif: I would like to compliment Dr Patti and his associates for their continuing meticulous study and reporting the cases of this very important population of patients that they have treated at UCSF. Achalasia is an uncommon disease that limits the number of patients available for clinical study and the acquisition of operative experience by individual surgeons. There is general agreement regarding the following issue: thorough preoperative evaluation being mandatory for identification of operative candidates. Although some physicians still advocate balloon di-
lation, myotomy is rapidly replacing dilatation as initial therapy. Endoscopic injection of botulin toxin results in variable, temporary symptom relief that may complicate subsequent myotomy as we have seen here. It should not be used. Regarding surgical management, there is general agreement that minimally invasive techniques are preferred. The laparoscopic approach is favored over the thorascopic approach. The myotomy must extend onto the cardia of the stomach, and a large or dilated esophagus is not a contraindication to myotomy.

Unsettled issues include the extent of the myotomy required, the role of intraoperative luminal endoscopy for assessment of the myotomy, and the role of a routine antireflux procedure at the time of the myotomy. Dr Patti and his colleagues at UCSF, as well as other authors, have previously reported good to excellent results following laparoscopic myotomy and the Dor or anterior fundoplication in approximately 90% of patients.

In the present report, Dr Patti reports the cases of 11 of 102 patients (11%) who experienced postoperative dysphagia. Four of these were because of inadequate myotomy, 4 were due to malconfiguration of the fundoplication, and 3 were due to fibrous strictures.

I have the following questions: Could you comment further on the role of intraoperative endoscopy? How many of these 11 patients with postoperative dysphagia were among the patients in whom intraoperative endoscopy was used? Second, will most general surgeons, including those with extensive laparoscopic experience, be able to adequately perform this procedure? Your group has previously reported on the impact of institutional experience as a determinant of outcome following esophagectomy, and now report an apparent learning curve for this procedure; that is, the technical problems with the anterior fundoplication in 4 patients that did not occur in the latter part of the series.

Finally, pneumatic dilatation was tried in 8 of these 11 patients but was successful in only 1. Nevertheless, you conclude at least in the written manuscript as opposed to the slide just now, that dilatation is recommended for the management of postoperative dysphagia. Is this conclusion justified?

Richard J. Finley, MD, Vancouver, British Columbia: How many perforations did you have during the operation, and did these perforations have an effect on the outcome? Second, have you had any pseudodiverticula above the fundoplication on follow-up x-rays? If so, did they result in any long-term problems with dysphagia or regurgitation? And, finally, could you describe what you do with the anterior vagus nerve during the myotomy? Where do you start your myotomy, and where does it go in relationship to the anterior vagus nerve onto the stomach?

Cedric G. Brenner, MD, Los Angeles, Calif: Dr Patti, now we know that with the Nissen fundoplication, it is necessary to take down the short gastrics, and most centers agree with that, but what is your evidence of the necessity for taking down the short gastrics when doing a so-called tension-free anterior Dor fundoplication?

Carlos A. Pellegrini, MD, Seattle, Wash: I wanted to ask you if you could comment a little bit more on what evidence there is that these patients have a fibrotic stricture as a consequence of previous treatment, and I say that because I have been impressed sometimes by what appears to be a fibrotic structure; what appears to be a difficulty, and in 3 patients who have undergone esophagectomy eventually for impossibility to correct the problems with myotomies, the pathologist has not been able to find fibrosis, so I don’t know if you have had the opposite experience, and if so, can you state it?

I also wanted to comment on the issue of endoscopy because I personally believe endoscopy is a tremendously useful technique, not only in this operation, but in every esophageal operation. And I recognize that for many years we surgeons stayed away from doing the endoscopy because we had to wait for the endoscopists to come, pay them a separate fee, and do all the other things that increased the cost and the time involved in this operation. About 3 and a half years ago, we started doing our own endoscopies in the operating room. The unit is prepared for every case in which an esophagus is going to be operated on, and the advantage to the surgeon is tremendous. Done by ourselves, it takes no more than 5 minutes to endoscope the esophagus, to look at the cardia, and to assess whether the fundoplication or a stitch is putting enough tension on one side or not, and the whole configuration problem of the fundoplication that you showed may have been avoided if that was the case, in addition to identification of the fibrosis. So I would encourage people to do the endoscopies themselves. It is a very simple maneuver. It cannot take you more than 5 minutes, and we can repeat it several times in the same operation after you place the stitches in another place.

Haile T. Debas, MD, San Francisco, Calif: I just want to add a note of caution regarding the need for fundoplication. Clearly you will need fundoplication if you increase the myotomy onto the stomach for 1.5 or 2 cm. There is an old study that was done very well at the Mayo Clinic that shows the distal myotomy could just go 1 or 2 mm onto the stomach until you see the transverse veins, and if you do that, there is no need for a fundoplication. But clearly if you go 1.5 cm onto the stomach, you do need a fundoplication. The question I have is what is the evidence, the real evidence that a fundoplication is required if you didn’t extend that myotomy to the extent you have distally?

Dr Patti: As Dr Wolfe mentioned, this is an uncommon operation, so there is little opportunity for individual surgeons to do enough cases to master the technical nuances. Considering the known effects of volume of experience on outcome of operations that involve this much detail, it could be argued that this operation would best be confined to centers with a special expertise in laparoscopic esophageal surgery.

Early in our experience with minimally invasive Heller myotomy, we performed intraoperative endoscopy as a routine, for it allows one to see the sphincteric narrowing and to decide when the myotomy has been made long enough. With the laparoscopic as opposed to the thorascopic operation, however, the precise location of the gastroesophageal junction is visibly evident, and the myotomy can be reliably brought 1.5 to 2 cm beyond that point. Therefore, operative endoscopy was principally confined to the first few years, which paradoxically was also the period of the technical failures. Since dropping endoscopy, we have not had a case with inadequate myotomy. In other words endoscopy would not contribute to the success of the operation as it is now being performed. Incidentally, the extra charges for operative endoscopy (ie, for professional fees and OR time) amounted to about $3000 per case. The Dor fundoplication has been proven by pH monitoring to prevent reflux after a Heller myotomy. The purpose of the present communication was to show the consequences of departure from standard operative technique. Thus, in a couple of instances, the fundoplication was made in a way that interfered with swallowing. As noted in the article, pneumatic dilatation was occasionally effective for persistent dysphagia resulting from a short myotomy. Although it was ineffective when the fundoplication constricted the gastroesophageal junction, the diagnosis of this problem cannot be made with certainty until surgery. Therefore, we feel there is nothing to lose by trying pneumatic dilatation for persistent dysphagia after a myotomy.

There were 5 intraoperative mucosal perforations. Three patients had received botox injections, and 1 had pneumatic dilatations. In each, scarring was present at the gastroesophageal junction that involved the mucosa as well as the muscle
(ie, they were strictures). The perforations were all small (ie, 1-2 mm) and visually evident. We did not encounter a problem with diverticula.

Regarding the vagus nerve, if the myotomy is made in the 1-o’clock position, the vagus is easy to avoid. If the myotomy is placed in the 10-o’clock position, one may have to lift and go under the vagus in order to preserve it.

Dr Bremner brought up an important technical point. Dor fundoplications can be made more symmetrical and undistorted if the short gastric vessels have been divided. Since the passage of food in these patients is entirely the result of passive forces (ie, gravity), we prefer to optimize the geometry of the wrap. It only requires a few minutes to do and is virtually free of complications. We acknowledge, however, that good results have been reported by others who do not divide the short gastric vessels.

As Dr Debas pointed out, there are 2 kinds of myotomies. In 1, the myotomy is made only a few millimeters onto the gastric wall, a technique that has been said to obviate the need for a fundoplication. In the other, the myotomy is taken 1 to 2 cm onto the stomach, accepting that a fundoplication will be required. Using pH monitoring, we studied a series of our own patients whose Heller myotomies had been made according to the former technique and found a surprisingly high incidence of gastroesophageal reflux. This led us to question the validity of the assumption that the shorter myotomy is truly capable of simultaneously relieving dysphagia and avoiding reflux. Furthermore, gastroesophageal reflux is already present in some patients with achalasia as a result of pneumatic dilatations. Consequently, we consider a myotomy that involves a 1.5-cm gastric extension coupled with the partial fundoplication to be the best operation for this disease.

IN OTHER AMA JOURNALS

ARCHIVES OF INTERNAL MEDICINE
How Many Women Lose Bone Mineral Density While Taking Hormone Replacement Therapy?
Results From the Postmenopausal Estrogen/Progestin Interventions Trial

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Background: The frequency of bone loss among women using postmenopausal hormone therapy is unknown. Methods: We used data from the Postmenopausal Estrogen/Progestin Interventions Trial to address the frequency of bone loss among women using postmenopausal hormone replacement therapy. Of 701 women randomized to active treatment (conjugated equine estrogens alone or in combination with 1 of 3 progestins), 538 (76.7%) were adherent and had replicate bone mineral density (BMD) measures at baseline, 12 months, and 36 months. Of 174 placebo-assigned women, 132 (75.9%) were similarly eligible. Replicate BMD measures were used to calculate within-person measurement errors, which were then used to delineate cut points that defined bone losers with 97.5%, 95.0%, 90.0%, or 75.0% confidence.

Results: At the lumbar spine, during the first 12 months, 1.5% of hormone users lost BMD with 97.5% confidence, corresponding to a decline of −3% per year; during months 12 to 36, only 0.6% of treated women lost spinal BMD to this degree. An annual loss of −1% or more was the criterion for spinal bone loss at the 75.0% confidence level; 5.1% and 8.0% of hormone users met this criterion in the first year and in months 12 to 36, respectively. For the total hip, during the first 12 months, 2.3% of hormone-adherent women lost −3.0% per year or more, the 97.5% confidence definition of loss; 0.4% were so classified during months 12 to 36. To be 75.0% confident of hip BMD loss, a −1.0% per year decline in BMD was required; using this criterion, 14.3% and 11.8% of hormone users lost total hip BMD between 0 to 12 and 12 to 36 months, respectively. Among hormone-adherent women, at the spine and hip, there was virtually no overlap between women classified as bone losers in the first 12 months and those classified as such in the last 24 months. With 95.0% certainty, corresponding to an approximate loss of −2.5% at the spine and hip, 31.3% and 11.7% of placebo-adherent women lost spinal BMD in the first 12 and last 24 months, respectively. Parallel figures for the hip were 32.3% and 7.9%, respectively.

Conclusion: Bone loss while taking postmenopausal hormones is rare, and bone loss among untreated women is far from universal. (2000;160:3065-3071)

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