Editorials

Myocardial Revascularization — Bypass Surgery or Angioplasty?

In this issue of the *Journal,* the results of the Bypass Angioplasty Revascularization Investigation (BARI), a large clinical trial comparing coronary-artery bypass grafting (CABG) with percutaneous transluminal coronary angioplasty (PTCA), are presented.[1] BARI enrolled 1829 patients who were followed for an average of 5.4 years. The authors concluded that for patients with multivessel coronary artery disease and suitable anatomy who prefer to avoid major surgery, "angioplasty offers a reasonable alternative with an expectation of similar overall survival rates and survival rates free of Q-wave myocardial infarction."

This carefully worded conclusion is similar to that derived from a recent meta-analysis of eight smaller trials with a total of 3371 patients with single-vessel coronary disease (732 patients) or multivessel disease (2639 patients) followed for 1 to 4.7 years.[2] Readers may interpret these collective data to mean that PTCA and CABG have equivalent outcomes. The choice of procedure for individual patients may then be based on other factors, such as shorter hospital stays and avoidance of major surgery with PTCA, or lower rates of subsequent procedures and recurrent angina after CABG.[1-6] But is such a simple conclusion acceptable?

The aim of BARI was to determine whether an initial strategy of PTCA, as compared with CABG, compromises clinical outcome.[7] The primary end point was mortality at five years, which was found to be 13.7 percent for PTCA and 10.7 percent for CABG. The observed survival advantage at five years with CABG was 2.9 percentage points, with a 95 percent confidence interval of −0.2 to 6.0.[1] Thus, the study did not establish with certainty that an initial strategy of PTCA is actually equivalent to CABG in patients with multivessel disease and clinically severe angina or objective evidence of ischemia. In fact, the confidence interval is consistent with the possibility that the survival rate with CABG may be superior by as much as 6 percentage points. Stated differently, the data are consistent with a 50 percent higher mortality rate with PTCA.

Although not statistically significant, a somewhat higher mortality rate with PTCA was also reported in two other trials, the Emory Angioplasty Versus Surgery Trial (EAST)[5] and the Coronary Angioplasty versus Bypass Revascularization Investigation.[8] Combining all available data,[1,2] my colleagues and I calculated the risk ratios for mortality to be 1.25 (95 percent confidence interval, 0.75 to 2.07) at hospital discharge (favoring PTCA), 0.86 (95 percent confidence interval, 0.63 to 1.16) at one year, and 0.89 (95 percent confidence interval, 0.74 to 1.08) overall (favoring CABG). These differences are not statistically significant, and the confidence intervals are wide, because the total number of patients in these studies was limited. Thus, although a survival advantage with CABG is suggested, there is insufficient statistical power to be certain.

The relevance of small differences in survival may be questioned. Yet differences of similar relative and absolute magnitude have been reported in larger trials claiming important treatment benefits for patients with moderately elevated cholesterol levels or myocardial infarction. If differences in mortality on the order of 1 percent (10 deaths per 1000 patients treated) are considered relevant, larger trials are required to exclude such differences and to demonstrate equivalence.[9] It is remarkable that such larger trials are commonly conducted for medical treatment regimens, whereas comparative trials of interventional procedures such as BARI do not have sufficient statistical power to demonstrate or exclude similar differences. Furthermore, the difference between treatment regimens, if any, may become apparent only after long-term follow-up.

In BARI, myocardial infarction during the initial hospitalization was more frequent after CABG than after PTCA. Nevertheless, the rates of survival free from Q-wave myocardial infarction (80.4 percent after CABG and 78.7 percent after PTCA) were similar at five years.[1] The investigators correctly point out that the rates of infarction were underestimated, because only Q-wave myocardial infarctions were reported. This explains why the rates are lower than those in other recent trials in which serum creatine kinase MB values were used to detect infarctions associated with PTCA.[7] The interpretation of a composite end point, such as survival without myocardial infarction, becomes difficult if the various components are not affected in the same way. How should we interpret the somewhat better survival in spite of somewhat more frequent myocardial infarctions with CABG in BARI? Clearly, in this situation, more weight should be given to the survival advantage, albeit small. It is better to survive with an infarct than not to survive at all.

In BARI, as in the other studies, CABG provided more extensive revascularization, with an average of 3.1 grafts per patient, than PTCA, for which angioplasty was attempted for an average of 2.4 lesions.[10] This difference, along with the problem of restenosis, resulted in higher rates of angina and repeated revascularization procedures after PTCA.[1-6]

A subgroup analysis in BARI revealed a major advantage of CABG over PTCA in patients with treated diabetes. In fact, the higher mortality after PTCA

TABLE 1. ESTABLISHED AND POSSIBLE DIFFERENCES BETWEEN CABG AND PTCA.

CABG		PTCA	
ADVANTAGES	DISADVANTAGES	ADVANTAGES	DISADVANTAGES
Established Differences			
Results in more complete revascularization Provides excellent relief of angina Associated with fewer subsequent procedures	Involves major surgery (possible perioperative problems) Requires longer hospitalization and higher initial costs	Major surgery avoided in 70 percent of patients Associated with shorter hospitalization and lower initial costs	Results in less complete revascularization Angina may recur Associated with more subsequent procedures
Possible Differences			
Possibly better long-term survival	Associated with more early infarctions	Associated with fewer periprocedural infarctions	Possibly poorer long-term survival

(as compared with CABG) was accounted for by the higher rate in this subgroup of patients, which made up 19 percent of the study population. This important observation would have major implications for therapy in clinical practice if confirmed by data from other trials. However, in EAST, the mortality rate among diabetics in the PTCA group was not higher than in the CABG group,[5] although this population was much smaller. Additional analyses from the other trials may help to clarify this issue.

Many factors influence the choice of procedure for revascularization in a given patient (Table 1). The decision by the physician and the patient will depend on the weight given to the various factors.

In studies of interventional procedures, technology may change during the long interval between the enrollment of patients and the reporting of results. Consequently, the results of a trial may apply to procedures that are not fully comparable to those used currently. Indeed, major advances have been made in interventional cardiology. The use of blockers of platelet glycoprotein IIb/IIIa receptors may halve the rate of periprocedural myocardial infarction and improve long-term outcome.[7] Furthermore, the use of coronary stents improves outcome, particularly since the introduction of better deployment techniques, better antithrombotic regimens, and heparin-coated stents.[11] Also, surgical techniques continue to improve with more frequent use of internal-thoracic-artery grafts and, more recently, minimally invasive surgical procedures.[12]

In the past, CABG and PTCA were distinct procedures performed by separate groups of operators. In the near future we may expect more of a team approach and the development of larger revascularization centers in which different procedures are performed by a closely coordinated team of operators with different backgrounds. Such developments necessitate repeated reassessment of the relative strengths and weaknesses of surgical and percutaneous revascularization procedures. Studies of adequate size remain warranted in spite of the findings in BARI and other comparisons of coronary surgery and angioplasty.[1-6] In the meantime, the conclusions of the BARI investigators stand: in patients with myocardial ischemia and multivessel disease, who constitute 12 percent of all candidates for revascularization, surgery is the established therapy, and angioplasty is an acceptable alternative.

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Pregnancy and Renal Disease

Of all the medical disorders that add risk to pregnancy, renal disease has long ranked among those most feared by physicians. Not only are the renal and vascular manifestations of preeclampsia the most common medical complication of pregnancy, affecting 5 to 7 percent of previously healthy women, but also intrapartum worsening of renal function, along with increased fetal morbidity and mortality, has been the fate of a large proportion of pregnant women with underlying diseases of the kidney, at least in the past.[1] Careful longitudinal studies of large numbers of women with varying degrees of renal impairment are necessary to permit us to advise concerned patients intelligently about the risks of pregnancy. The results of the study by Jones and Hayslett of 82 pregnancies in 67 women with moderate or severe renal insufficiency, reported in this issue of the *Journal*,[2] will help obstetricians and internists do just that. Such data are not easy to collect, because fertility is decreased in women with renal impairment.

For doctors who need to counsel women with renal impairment who are contemplating pregnancy, certain caveats must be kept in mind in interpreting these results. First, some women who do well may be lost to follow-up after pregnancy because they do not seek medical advice. On the other hand, longitudinal surveys of the outcome of pregnancy cannot include women who contemplated pregnancy but did not in fact become pregnant, perhaps on their doctors' advice. Such women may have a poorer prognosis than those who actually become pregnant.

The good news in this report is that with advances in the care of newborn babies, fetal survival has improved remarkably. This is important because almost 60 percent of infants born to women with a serum creatinine concentration exceeding 1.4 mg per deciliter (124 μmol per liter) are premature — six times the expected rate in the general population — and because the babies are also small for their gestational age. Both problems are more marked, the worse the mother's renal insufficiency. Nevertheless, fetal mortality in the present report was only 7 percent, as compared with rates ranging from 12 to 88 percent in earlier, smaller series of similar women.[3-6] With the advent of modern intensive care for newborns, survival is routine even when babies are born as early as 26 to 27 weeks of gestation.

The news is not so good for the mother. What is clear from all the published data is that, as the woman's degree of renal insufficiency increases, the risk that renal function will worsen during pregnancy rises sharply, a tendency that is further exaggerated by the presence of hypertension. Katz et al. found that renal function declined during pregnancy in 16 percent of women with mild renal disease (those with initial serum creatinine concentrations of less than 1.4 mg per deciliter), most of whom were normotensive.[7] Of the women followed by Jones and Hayslett whose initial serum creatinine concentrations were 1.4 mg per deciliter or more, almost half had a pregnancy-related decline in creatinine clearance of at least 25 percent, and in three quarters of these women, the decline in renal function persisted or progressed further after delivery. Among women whose initial serum creatinine concentrations were between 1.4 and 1.9 mg per deciliter (124 and 168 μmol per liter), the chance of a pregnancy-related exacerbation was roughly 40 percent, and the loss of renal function persisted after delivery in about half of those affected.

The risk of pregnancy-related renal damage was found to be particularly high in the subgroup of women whose initial serum creatinine concentrations were 2.0 mg per deciliter (177 μmol per liter) or higher. Of 20 pregnancies in such women, serum creatinine concentrations rose in the third trimester in 13 (65 percent). Worsened renal function persisted or progressed after delivery in almost all these women, rapidly reaching end-stage renal failure in seven (35 percent). Thus, unlike pregnancy in women with only mild renal disease, pregnancy in women with moderate or severe renal disease tends to exacerbate renal injury in a way that is largely irreversible. This conclusion is supported not only by the data of Jones and Hayslett, but also by earlier studies involving smaller numbers of women.[3-6] Although some of the late decline in kidney function may be attributed to the natural progression of the underlying disease, it is notable that in the present study all the women with peripartum worsening for whom preconception data were available had decreases in the glomerular filtration rate during pregnancy that exceeded those predicted.

There are, of course, exceptions to the general rule. One of the study patients had an initial serum creatinine concentration of 3.7 mg per deciliter (327 μmol per liter) and had no change in renal function either during pregnancy or six weeks after delivery. Many nephrologists and obstetricians can recount such anecdotes. An optimistic physician or a woman yearning to bear a child may prefer to view

the glass as half full. But a sober analysis of all the risks involved in pregnancy for a woman with renal impairment should include a realistic assessment of the dangers of worsening renal function. What data we have indicate that a pregnant woman with a serum creatinine concentration exceeding 2.0 mg per deciliter has a chance of approximately one in three of having end-stage renal failure necessitating dialysis during or shortly after pregnancy. The chances are better than even that renal function will be worse after the pregnancy than it would have been if pregnancy had been avoided. Manifestations of preeclampsia in susceptible women with renal disease may be reduced somewhat by treatment with low doses of aspirin,[8] but this treatment is still controversial.[9]

Why does pregnancy exacerbate renal disease? One notion is that the worsening is paradoxically related to the increase in glomerular perfusion characteristic of normal pregnancy. But this hypothesis seems contradicted by the facts at hand. Most women whose renal function deteriorates during pregnancy have no evidence of early hyperperfusion, such as an initial fall in the serum creatinine concentration.

A more attractive hypothesis is that the preexisting renal disease somehow sets the stage for the cascade of platelet aggregation, formation of fibrin thrombi, microvascular coagulation, and endothelial dysfunction in the kidney and elsewhere that is at the heart of the phenomenon of preeclampsia. An imbalance between the production of vasoconstrictor products of arachidonic acid (thromboxane) and vasodilatory ones (prostacyclin) has long been posited in preeclampsia. It may be important that renal overproduction of thromboxane is regularly elicited by experimental renal insufficiency.[10] The consequent superimposition of preeclamptic microangiopathy on already-damaged kidneys might cause poorly reversible or persistent renal damage in some women. Further collaborative studies of these phenomena by nephrologists and obstetricians should provide clearer insights into both the physiology of pregnancy and the nature of renal disease.

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HELICOBACTER PYLORI IN THE STOMACH — A PARADOX UNMASKED

IN 1993, a National Institutes of Health Consensus Conference declared infection with *Helicobacter pylori* to be an important cause of duodenal and gastric ulcers.[1] Soon thereafter, the International Agency for Research on Cancer classified *H. pylori* as a group 1 carcinogen, a definite cause of gastric adenocarcinoma in humans.[2] In this issue of the *Journal*, Hansson and colleagues report that patients with duodenal ulcers have a decreased risk of gastric cancer, whereas those with gastric ulcers have an increased risk.[3] Thus, a paradox in the epidemiology of gastric disease is unmasked. The incidence of gastric cancer correlates positively with the incidence of gastric ulcer but negatively with the incidence of duodenal ulcer.[4] If *H. pylori* causes each of these diseases, why is it that they do not all occur in parallel?

H. pylori is a highly successful microbe, infecting most of the world's population. Once established in the stomach, the organism persists for decades, seemingly impervious to the acid and peristaltic activity in its environment. In most cases, *H. pylori* silently coexists with its host, causing no symptoms or signs. But this relation is not always benign. Gastric cancer develops in approximately 1 percent of infected persons, and 20 percent have duodenal ulcers. The findings of Hansson et al. argue that separate paths lead to these diseases — that is, *H. pylori* infection can progress to gastric cancer or duodenal ulcer but seldom to both. This being the case, other factors besides the infection must foster one disease while militating against the other. Examples of potentially important cofactors are the genetic characteristics of the host and the microorganism; exogenous elements, either infectious or noninfectious; and the time of life when the infection is acquired — early in childhood or later. All these undoubted-

ly influence the outcome of *H. pylori* infection, but the last best explains the paradox reported by Hansson et al.

Gastric cancer is a leading cause of death from cancer in the developing world, where infection with *H. pylori* in early childhood is the rule.[5] In infected children, duodenal ulcer is rare. However, by early adulthood, the infection acquired in childhood has frequently progressed to multifocal atrophic gastritis, a pathologic condition characterized by patchy loss of gastric glands that secrete protein and acid.[6] By mechanisms as yet unknown, multifocal atrophic gastritis and the associated hypochlorhydria predispose patients to both gastric ulcers and cancer. In contrast, duodenal ulcers are rare in patients with multifocal atrophic gastritis, since even in the presence of *H. pylori,* the precept "no acid, no ulcer" holds. In short, childhood infection predisposes patients to gastric ulcers and stomach cancer, but it inhibits the development of duodenal ulcers.

In industrialized countries, deaths due to gastric cancer, gastric ulcers, and duodenal ulcers are decreasing.[7,8] As might be expected, these declines parallel a diminishing incidence of *H. pylori* infection, particularly in children.[9] Thus, the prevalence of infection is declining, and the average age at the time infection is acquired is rising. Multifocal atrophic gastritis is uncommon when infection occurs after childhood. It may still occur, but at older ages, a change that has the effect of pushing back by five years the age at which gastric cancer is diagnosed in industrialized countries.[9] In young adults, acid-secreting capacity is maintained in the face of mucosal damage caused by *H. pylori,* and duodenal ulcer disease is endemic.

This age-dependent explanation of the outcomes of *H. pylori* infection fits the temporal pattern of gastric diseases described by Sonnenberg.[7] Among people born before the Industrial Revolution, both gastric cancer and duodenal ulcers would have been unusual. Their short life expectancy would have limited the incidence of gastric cancer, and high rates of *H. pylori* infection in children would have limited the incidence of duodenal ulcers by causing the early onset of multifocal atrophic gastritis. For cohorts born during the Industrial Revolution, hygiene in childhood remained poor and *H. pylori* infection was almost universal in children.[7] But at the end of the 19th century, a rapid increase in life expectancy coincided with the highest mortality from gastric ulcers and gastric cancer in the past 150 years. As hygiene and economic conditions gradually improved, the age at which *H. pylori* infection was acquired in subsequent birth cohorts shifted upward. Rates of gastric cancer correspondingly declined. The incidence of duodenal ulcers, by contrast, increased and then decreased as the prevalence of infection declined. In this way, the peak mortality due to duodenal ulcer disease followed the peak mortality from gastric ulcers by one to two decades. Today, the rates for all three diseases are declining in parallel.

This model is inferential, but the effect of the age of acquisition of *H. pylori* infection has been circumstantially confirmed. Larger families are a risk factor for the transmission of diseases from person to person. Children with many older siblings are more likely to acquire infection at an earlier age than only children or oldest children. In a study of Japanese-American adults, late birth order and a large number of siblings both increased the risk of gastric cancer, but only among people with *H. pylori* infection.[10] In contrast, family size in childhood had no effect on the incidence of duodenal ulcer disease, among either infected or uninfected adults. Indirectly, then, it appears that the rate of childhood infection with *H. pylori* affects the incidence of gastric cancer but not of duodenal ulcer.

One conclusion that should not be drawn from the study of Hansson et al. is that duodenal ulcer itself protects against gastric cancer. Gastric cancers do occur in patients with a history of duodenal ulcer, even when they have not undergone gastric resection.[11] This is so because patients with duodenal ulcers are not immune from late-onset multifocal atrophic gastritis. Moreover, the risk of multifocal atrophic gastritis in patients with duodenal ulcers may be increasing. A recent study showed that proton-pump inhibitors, by altering the gastric microenvironment, increased the rate of progression of *H. pylori* gastritis to multifocal atrophic gastritis.[12] In principle, current therapies might be advancing the cancer clock by converting relatively benign gastric inflammation into a more destructive, premalignant process. At present, there is no convincing evidence that pharmacologic inhibition of acid secretion increases the risk of gastric cancer, but the long-term use of acid-inhibiting therapy in patients with *H. pylori* infection should be viewed with some caution.

The relation between *H. pylori* and humans is evolving. In the past 200 years, gastric cancer, gastric ulcer, and duodenal ulcer have waxed and waned. In other parts of the world, diseases related to *H. pylori* continue to pose important health burdens, but in the United States, we are witnessing the decline of *H. pylori*. Improvements in hygiene and economic conditions have interfered with the transmission cycle of the organism to the point where *H. pylori* is now hard to find in children. Yet the worldwide prevalence of infection remains high. Moreover, as with shigella infections, crowded conditions in day-care centers may again provide the opportunity for early childhood transmission. The varying outcomes of *H. pylori* infection serve to remind us that our interactions with the microbial world are constantly in

flux. A better understanding of these patterns will enable us to anticipate and interrupt the long-term consequences of this deleterious parasite–host interaction.

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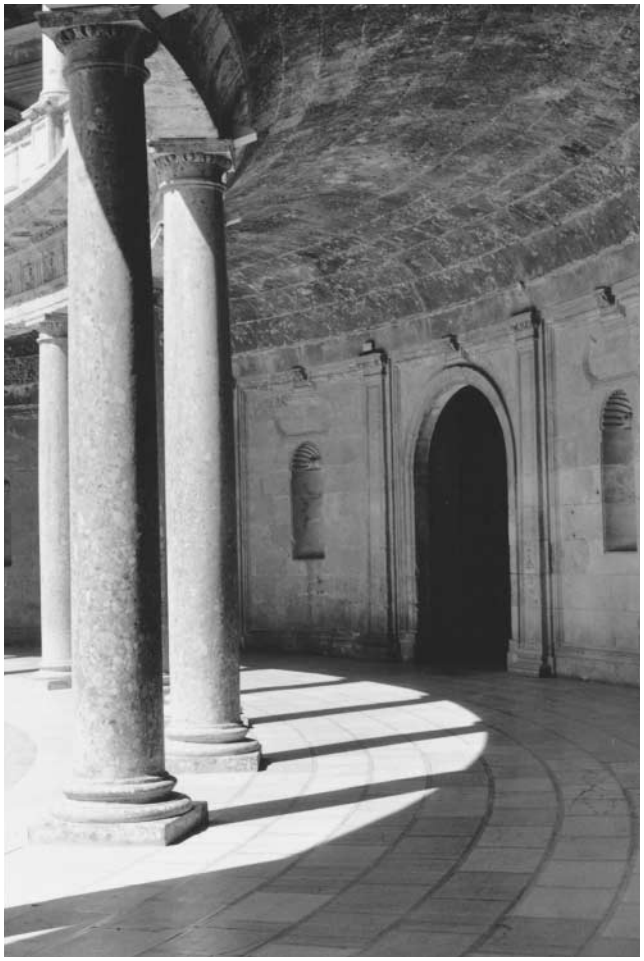

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Sounding Board

Physical and Emotional Problems of Elite Female Gymnasts

WITHIN the past five years, two U.S. female gymnasts at the Olympic level, Christy Henrich and Julissa Gomez, died from medical problems related to their sport. Christy died from complications of anorexia nervosa and Julissa from complications of spinal trauma due to a vaulting injury.

In this Olympic year, it is timely to discuss the psychological and physical problems associated with competitive women's gymnastics. Women's gymnastics provides a useful framework for viewing worrisome trends in other competitive youth sports. In the United States, organized athletic programs involve at least 20 million children and adolescents each year, with more than 2 million participating in all levels of competitive gymnastics.[1,2]

The development of gymnastics champions involves hard training, stringent coaching, and often parental pressure, ostensibly in the best interest of the child. Overtraining, injuries, and psychological damage are common consequences.[3,4] Parents and coaches, in collusion with the young athlete, may seek to experience vicariously the success of the child, a behavior that could be called "achievement by proxy." The recent death of seven-year-old Jessica Dubroff during her abortive cross-country airplane flight has been cited as a glaring illustration of this behavior.[5] Its hallmark is strong parental encouragement of a potentially dangerous endeavor for the purpose of gaining fame and financial reward. We suggest that in its extreme form "achievement by proxy" may be a sort of child abuse.

Elite gymnasts throughout the world begin training between the ages of five and seven years. They are often involved in serious, regimented training by the age of 10. During this critical stage of development, children experience rapid physiologic, neurologic, and psychological growth.[3] Participation in Olympic-level gymnastics may place inappropriate physical and psychological demands on these children, which may have long-term, indeed lifelong, adverse consequences.[6,7]

PHYSICAL INJURIES

Gymnastics is an intense, repetitive, high-impact sport, and most elite gymnasts do not pass through childhood and adolescence without injury.[4,8] The risk of injury increases with longer practice time, the degree of difficulty of the routines, and age-related vulnerability of the skeletal system.[1,3,6,9,10] Repetitive stress on the developing musculoskeletal system, which has a much higher cartilage content than in adults, may cause an accumulation of minor physical insults that can result in permanent injury or deformity.[11,12] These injuries include stress fractures; growth-plate fractures; wrist and elbow injuries; spinal injuries such as scoliosis, spondylolysis, and spondylolisthesis; and reflex sympathetic dystrophy.[13,14] Moreover, training more than 18 hours per week before and during puberty may alter the growth rate and prevent the attainment of full adult height.[15,16]

The pressure to practice and compete while injured compounds the risk of impaired skeletal development and permanent deformity.[17,18] Overtraining with incompletely healed injuries may contribute to reflex sympathetic dystrophy. This is a disorder characterized by an exaggerated response of the sympathetic nervous system to minor trauma, resulting in severe, recurrent, chronic pain affecting an entire leg or arm, accompanied by autonomic nervous system changes. Inactivity and inadequate treatment result in muscle atrophy, bone demineralization, and contractures.[19,20] Psychological stress has been noted to have a significant role in precipitating reflex sympathetic dystrophy.[19,20]

THE FEMALE-ATHLETE TRIAD

Gymnasts are also at risk for nutritional, endocrine, and psychiatric disorders. The "female-athlete triad," which is associated with substantial morbidity and mortality,[21,22] is characterized by disordered eating, menstrual dysfunction, and osteoporosis.[21-23] In the general population, the prevalence of eating disorders is about 1 percent for anorexia and 1 to 3 percent for bulimia.[24] Among female athletes, however, the prevalence of eating disorders is reported to be between 15 percent and 62 percent.[14,22,25,26] There is unequivocal pressure for female gymnasts to maintain a prototypic mesomorphic body appearance (i.e., to be thin and muscular). This pressure encourages atypical eating behavior, which may lead to frank eating disorders.[14,21,26,27] Oblique or deliberate comments by coaches or judges that the loss of a few pounds will improve athletes' scores may exacerbate these problems.[28,29] Judges have a profound influence on the careers of gymnasts, and they may indirectly contribute to the development of eating disorders. Disordered eating and intensive exercise may contribute to primary and secondary amenorrhea.[21,22,30,31] In addition to the physical stress on the athlete, the emotional stress of elite competition may play a part in the development of menstrual dysfunction.[32] Menstrual dysfunction, in turn, increases the risk of premature osteoporosis

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and fractures[22,30,33-35] and may also increase the risk of scoliosis.[36]

ISSUES OF IDENTITY

There is enormous pressure — particularly from adults — for the child gymnast to be thin and perform well in order to conform to and promote the image of the sport.[28] Adults are often seduced by Olympic dreams and the lucrative opportunities that success may bring.[28,37] The young athlete may perceive her entire identity and self-worth as depending on her participation and success in gymnastics — a perception that may result in a unidimensional self-concept.[38] As these youngsters mature, some realize that what is happening to them is unhealthy. However, the highly organized and restrictive gymnastics programs, as well as family pressures and expectations, impede the athlete's ability to think or act independently.[39] Pain, somatoform disorders, and self-induced injury may become the only acceptable ways to escape with one's dignity (and the parent's or coach's dignity and self-esteem) intact.[40]

Many aspiring young gymnasts devote nearly all of their time to the sport and may thereby suffer from social isolation and a lack of opportunities for social development.[39] At the elite level, gymnasts work out on average 30 to 45 hours per week.[28,41] They may leave home before the age of 12 to train and in some cases are adopted by their coaches. At every competitive level, critical coaching with the goal of winning is paramount.[42] Young female gymnasts begin traveling extensively and staying in hotels and are often expected to behave as independent adults at a time when they may not even have entered puberty. Parents and coaches often attribute a child's overtraining to the child's enthusiasm and love for the sport, but this may be little more than self-deception and an abrogation of adult responsibility.

ELITE GYMNASTICS AND THE POTENTIAL FOR ABUSE

Elite gymnasts tend to be extremely obedient and disciplined and to strive for adult approval. In general, they are in awe of coaches and other adult authorities, who hold the key to potential success; consequently, they are at risk for abuse.[28] Insensitivity on the part of adults to a child's developmental requirements leads to an attempt to delay the onset of puberty, on the one hand, and the attribution of adult qualities to the child, on the other. Presenting a prepubescent girl as sexually attractive in order to gain higher scores is a matter of particular concern. "After all," as one judge said, "it is a very visual sport."[28,29] The quest for success at the Olympic level ensures that these girls may be driven beyond their physical and emotional limits. There is also an element of self-abuse involved in competing in an injured state. Sometimes physicians may also become involved in the process of getting an injured athlete back into competition too soon. Further increasing the risk of abuse is the enormous sacrifice and emotional investment made by entire families in helping the athlete achieve the Olympic dream.[41] The personal and financial sacrifices of these families may increase the stress on the child.[39]

Coaches, who may have the greatest influence over athletes and their parents, are in the best position to monitor the athlete's behavior.[43] Unfortunately, most coaches have little knowledge of child development, although gymnasts typically spend more time with their coaches during these critical developmental years than with any other person. Coaches are often role models for these vulnerable young girls, who strive to attain their mentors' approval. Given coaches' frequent lack of awareness of developmental issues, unrealistic and unhealthy physical and psychological demands are often placed on these children.

THE NEED FOR COACHING STANDARDS

Encouraging but modest efforts have been made by the elite gymnastics community in monitoring itself.[33,44] An example was the recently published preliminary report of the Female Athlete Triad Task Force.[44] Officials of national gymnastics programs are clearly concerned about the athletes' well-being. However, as members of governing bodies, they have political and financial goals that are likely to be in unavoidable conflict with the best interests of the child athletes. An external agency may be needed to establish standards and monitor health and safety requirements for the athletes. External regulation in conjunction with improved self-monitoring could be a big step forward in protecting young competitors.[43,45]

THE GOAL: PREVENTING HARM TO ELITE ATHLETES

In this Olympic year, millions of spectators will be treated to a dazzling display of elite women's gymnastics in which the main medal contenders may all be under the age of 16. It is important, however, that we understand the human story that lies behind this and other elite sports. At its best, elite gymnastics can provide a profoundly meaningful experience for the athletes, promoting their self-esteem and self-discipline and contributing to their development into productive and successful adults. At its worst, the sport can result in serious, life-endangering physical and psychological disabilities. As physicians, we need to remember that the development, health, and well-being of these gifted children must be assigned the highest priority. "Achievement by proxy" on the part of parents, coaches, and other adults (sometimes even physicians) can pose real dan-

gers to the health of these vulnerable young girls. Talented youngsters at every competitive level should be supported rather than crippled by their sport as they enter adulthood.

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