AMENORRHOEIC ATHLETES: AT RISK OF DEVELOPING OSTEOPOROSIS?

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The growing involvement of women in sports, while enhancing general physical fitness, is being paralleled by an increasing incidence of medical complications. Among these are menstrual disorders, including a delay in the onset of menstruation, oligo- or amenorrhoea, and inadequacy of progesterone secretion during the luteal phase of the cycle. At first, only sporadic accounts of such disturbances appeared, suggesting that pre-existing functional derangements of cyclicity were likely to be responsible. However, the sheer volume of clinical reports now casts doubt on this view, as does a recent prospective study, in which a high proportion of women with previously normal cyclicity were observed to develop menstrual dysfunction while engaging in a graded exercise programme (Bullen et al, 1984).

Physiological interpretation is complicated by the fact that clinically similar disorders of menstruation are frequent accompaniments of weight loss and emotional stress, both of which occur often in subjects undergoing physical training and athletic competition. However, in a sports setting, exertion *per se* appears to be a significant component of a multifactorial process. Thus: (1) the menarche has been observed to occur promptly during injury- or vacation-related suspensions of exercise in ballet dancers studiously avoiding a concomitant increase in body weight (Warren, 1980), and (2) menstrual dysfunction, while more frequent in women permitted to lose weight during participation in a graded exercise programme than in those who maintained their weight, was a common occurrence in both groups (Bullen et al, 1984).

The pathogenesis of the exercise-associated menstrual disorders is not yet understood. Menstrual rhythmicity is known to depend upon an exquisitely programmed stimulation of ovarian steroid secretion by pulsatile release of the pituitary gonadotrophic hormones, follicle-stimulating hormone (FSH) and luteinising hormone (LH). Pituitary secretion, in turn, reflects episodic stimulation by the hypothalamic gonadotrophin-releasing hormone (GnRH), which may be activated by a neural

oscillator. A variety of neural and hormonal inputs influences the synthesis and release of GnRH and the pituitary response. Disturbances in gonadotrophic pulsatility are reflected in aberrations of menstrual cyclicity and, if sufficiently severe and prolonged, in its suspension.

Exercise delivers a strong stimulus to the endocrine system, evoking a complex pattern of changes in the circulating levels of many hormones. Concentrations of plasma insulin fall, while those of ACTH, β -endorphin, met-enkephalin, growth hormone, prolactin, glucagon, catecholamines, melatonin and prostaglandins rise. The pertinence of these changes to the clinical disorders of reproduction is uncertain. However, it is of interest that the administration of ACTH, β -endorphin, prolactin and melatonin to experimental animals can delay sexual maturation, impair oestrous cyclicity and blight luteal function. The repetition of exercise tends to dampen the acute rise in hormonal levels, which is virtually extinguished in the case of certain hormones (e.g., met-enkephalin) but apparently is better maintained for others (e.g., β -endorphin). Tonic elevations of the above-mentioned hormones have not been detected in the body fluids of women athletes. However, a persistent depression of circulating levels of the reproductive hormones (oestrogens, progesterone, LH and, to a lesser extent, of FSH) is characteristic of the exercise-induced hypogonadism.

Clinical speculation regarding organs that might be affected unfavourably by prolonged stimulation by low levels of oestrogen in the absence of progesterone has, until recently, centred on the endometrium and breast. However, current research is focussing attention upon bone as a possible target of interaction between the sex steroid hormones and exercise stress.

Physical exertion exerts a major influence upon bone mass and upon the remodelling of bone. Immobilisation results in bone loss and activity in bone restitution. The manner in which muscular activity induces these effects

is not understood. Among the mechanisms postulated are; (1) neural influences, perhaps mediated by piezo-electric currents, (2) mechanical stress and strain consequent upon weight bearing and muscle tension, and (3) changes in vascularisation and blood flow.

In several instances, participation in sports has been found to be accompanied by increased bone mass. For example, Nilsson and Westlin (1971) noted the femur to be more dense in male athletes than in healthy age-matched non-athletes, as measured by photon absorption, with greater density in the leg of preference. Jones et al (1977) observed relative humeral hypertrophy in roentgenograms of the playing arm of professional tennis players of both sexes.

Hypo-oestrogenism, an important factor among several known to play a role in the genesis of post-menopausal osteoporosis, also occurs in amenorrhoeic athletes. Therefore, the outcome of attempts to prevent bone attrition in women following the climacteric by means of exercise regimens is of interest in a sports context. From measurements of total body calcium, Aloia et al (1978) concluded that a mild exercise programme (i.e., 1 hour/day, 3 days/week) retarded the development of osteoporosis in women who were approximately 5 years past the menopause. Smith et al (1981) found that a light to moderate physical activity programme, one designed around a chair, increased the bone mineral content of the radius by 2%, as measured by photon absorption, in 69-95 year old women in a nursing home. Krolner et al (1983) noted that an 8 months' moderate training load increased the mineral content of the lumbar spine, but not that of the forearm, as measured by dual photon absorptiometry, in healthy 50-73 year old women who had previously sustained a Colles' fracture.

That bone attrition rather than accretion may result from vigorous exercise by amenorrhoeic athletes is indicated by recent study of Drinkwater et al (1984). These investigators compared the vertebral mineral density of 14 amenorrhoeic athletes (11 runners and 3 crew members) with that of 14 eumenorrhoeic athletes. The latter were selected from a pool to match the amenorrhoeic women with respect to sport, age, weight, height and the frequency and duration of daily training sessions, in that order of priority. A satisfactory match was achieved for all physical characteristics, including body composition (as determined by hydrostatic weighing), a property not included in the original profile. The athletic history of the two groups differed significantly, the mean weekly mileage of the eumenorrhoeic group being 24.9 and that of the amenorrhoeic group 41.8 miles. The dietary intake of total calories and of fat by the amenorrhoeic athletes was lower than that of the eumenorrhoeic subjects but the difference did not quite achieve statistical significance, while calcium intakes were similar. Hormonal assays for

plasma oestradiol, progesterone, testosterone and prolactin concentrations were performed on four samples of blood obtained at intervals. These revealed significantly higher oestradiol, prolactin and progesterone levels in the eumenorrhoeic women, and equivalent testosterone concentrations in the two groups.

Measurements of regional bone mass were made by single photon absorptiometry for the appendicular skeleton (the distal radius of the non-dominant arm at two sites) and by dual photon absorption for the axial skeleton (vertebral bodies and disc interspaces of the individual vertebrae L₁ through L₄). No differences were found between the amenorrhoeic and eumenorrhoeic groups with respect to the mineral content of the radius, but that of the vertebrae was significantly lower in athletes belonging to the amenorrhoeic group, and was lower in runners than in crew members.

Comparison of these results with population norms revealed close correspondence of the radial measurements without regard to the menstrual status of the athletes. Likewise, there was good agreement between vertebral mineral density in the eumenorrhoeic athletes and that predicted from a normal population by means of an age-based regression equation. However, the mean vertebral mineral density in the amenorrhoeic group corresponded to that of women 51.2 years of age. In two athletes, the vertebral mineral density fell below the fracture threshold of 0.965 g/cm², as defined by Riggs et al (1982).

As Drinkwater et al (1984) point out, confirmation of these findings is required, as is extension to other skeletal areas, in particular to those subjected to repeated mechanical stresses in specific sports. Elucidation of the mechanisms responsible for the hypo-estrogenism and menstrual disturbances is of scientific and potential clinical importance. Ultimately, such research should lead to prophylactic measures enabling women to benefit from vigorous participation in sports, without compromise of bone integrity or other deleterious consequences of hypo-oestrogenism.

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