CLINICAL PERSPECTIVES

Fainting, emancipation and the 'weak and sensitive' sex

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In the Victorian era, it was considered feminine and appropriate for young women to faint. Women with a frail and weak appearance were even considered to be ideal beauties. This fashion obviously did not apply to young men, who were expected to be strong and healthy. Some say that the corset women wore in order to supposedly slim the body and make it conform to a fashionable silhouette by reducing the waist and thereby exaggerating the bust and the hips was the cause of their tendency to faint. 'Tightlacing' with extreme waist constriction was thought to impede venous return.

Although the position as well as the clothing of women has changed dramatically since the Victorian era, fainting is still considered a disorder mainly affecting women. This assumption is correct. Among medical students a prevalence of 40–45% of fainting episodes in females compared to 25% in males has been reported (Ganzebooom *et al.* 2003). Furthermore, the number of presentations for fainting in general practice and in emergency settings is approximately four times higher for young females than for young males (Olde Nordkamp *et al.* 2009).

The search for the mechanisms involved in the higher fainting rate in females has a long history. Differences due to sex hormones, body stature and psychological factors influencing reflex responses have all been considered. The hypothesis that the effects of sex hormones are involved goes back to observations by Soma Weiss, an astute clinician and clinical investigator, who held the prestigious Hersey academic chair at Harvard around 1940. 'In female patients there is a tendency for the attacks to occur at the time of menstruation' he wrote almost 75 years ago in his famous monograph entitled 'Syncope and Related Syndromes' (Weiss, 1935). Fluctuations in baroreflex sensitivity were supposed to be involved. A large number of studies have been published since, focusing on baroreflex mechanisms and other haemodynamic changes during the menstrual cycle like skin blood flow. In a recent issue of *The Journal of Physiology* Fu *et al.* (2009) re-address the issue. The authors conclude that other than sympathetic baroreflex control mechanisms contribute to sex differences in orthostatic tolerance in young humans. Interestingly, the question whether fainting is actually at its peak during the period of menstruation as suggested by Soma Weiss seems to have received little further attention in the literature apart from a study under medical students. This study reported that menstruation was presented as a reason for vasovagal episodes in 6–9% of women with syncope (Ganzeboom *et al.* 2003). From a clinical perspective it seems much more likely that fainting during the period of menstruation is triggered by abdominal discomfort rather than by subtle haemodynamic changes.

In teenagers and young adult females, the heart rate in the supine position tends to be higher and in the upright position values exceed those in males by about 10 beats min^{-1}. Systolic blood pressure is reported to be lower in females than males. These findings have been attributed to cardiac anatomical sex differences. Fu *et al.* reported in an earlier study that women have smaller, less distensible left ventricles such that the maximal slope of the Frank–Starling curve is greater in women than men (Fu *et al.* 2004). To date this study, seems to provide the most plausible argument to explain some of the sex differences in orthostatic tolerance.

Fainting is a unique human condition. It is usually triggered by postural or emotional stimuli when the patient is conscious and the body upright. An intriguing recently described condition is 'sleep syncope'. In this condition, patients wake up feeling faint already and pass out in bed or on the way to the toilet (Jardine *et al.* 2006). These patients are predominantly female, with more fainting episodes triggered by blood-injection-injury phobias than regular fainters. Young female subjects also have a much higher incidence of presyncope and symptoms of orthostatic intolerance (Romme *et al.* 2008). It seems therefore reasonable to assume that psychological factors may also be involved in the higher fainting rate in women.

Less traditional factors involved in the high fainting rate in women need to be considered. Women tend to have a lower creatine kinase level (CPK) and lower CPK levels are associated with a higher incidence of fainting (Brewster *et al.* 2009). Three factors crucial to maintain orthostatic normotension could be involved due to 'lack of energy' caused by the lower CPK. First, a diminution of the capacity to constrict systemic blood vessels resulting in a diminished vasoconstrictor capacity. Second, diminished swift skeletal muscle responses with more downward pooling of venous blood. Third, CPK is thought to fuel sodium retention in the kidney, with a low CPK leading to diminished salt retaining capacity. The effects of body iron stores should also be considered. Iron deficiency is extraordinarily common in young females and an association between decreased serum ferritin and a tendency to faint has been reported. An effect of iron on catecholamine metabolism, peripheral vasodilatation due to the anaemia or preferential splanchnic vasodilatation has been suggested (Stewart, 2008).

The mechanisms underlying the higher fainting rate in women will remain to fascinate clinicians and physiologists interested in integrative studies. It seems highly unlikely that 'a single fainting factor' will ever be identified, since women differ from men physiologically and psychologically in many aspects, whatever emancipation may say.

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