In the late 19th century words like “Oh my, I'm feeling rather faint” were uttered regularly. It was the early Greeks who first identified female hysteria and later the diagnosis became associated with Freud. Hysteria or vapours, as it was also known, was associated with women of the higher social classes. When these women encountered something offensive to their elevated moral sensitivities, they might respond with a faint. The stimulus was thought to be too great for their delicate constitution to endure. However, as women came to be understood as far from delicate, the vapours diagnosis began to disappear. Nonetheless, fainting is still a common occurrence and often bears resemblance to this earlier idea that a highly emotional or painful stimulus can result in a faint.

Syncope is the medical term for fainting. It is a sudden, usually temporary, loss of consciousness (LOC). Forty percent of the Irish population are expected to experience at least one episode of syncope in their lifetime. In Ireland, it accounts for up to six per cent of acute medical admissions and three per cent of emergency consultations (O’Dwyer, Hade, Fan, Cunningham, & Kenny, 2010). Recognising the scope of this condition, professional medical societies such as the European Society of Cardiology (ESC) have published clinical guidelines to direct a more effective management strategy when dealing with syncope (Moya et al., 2009).

We will now briefly outline the physiological aspects of the common faint according to the ESC guidelines and provide an overview of how syncope presents in primary care. This will be followed by an exploration of the psychological aspects of syncope such as its impact on quality of life (QOL) and the psychological models that may explain syncope. Finally, we will discuss the non-pharmacological treatment of syncope and psychological approaches to treatment.

**Mechanism, Triggers and Symptoms**

Vasovagal Syncope (VVS), also referred to as Neurocardiogenic Syncope and Neurally Mediated Syncope, is regularly cited as the most common cause of syncope. In the ESC guidelines, VVS falls within a group of syncope known as Reflex Syncope. This refers to a varied set of conditions in which cardiovascular reflexes that are normally useful in controlling the circulation become intermittently inappropriate, in response to a trigger. There is a drop in heart rate (bradycardia) and blood pressure (hypotension), which reduces blood flow to the brain, and this in turn results in a temporary LOC (Van Dijk, & Sheldon, 2008).

A wide variety of conditions can trigger VVS, including physical strain, psychological distress, dehydration, bleeding or pain (Kapoor, 2000). Emotional VVSs may also occur in relation to a threat of injury, the...
sight of blood or extreme physical pain. In others, the activation of receptors in the ventricular wall or in other organs (e.g., the bladder or the oesophagus) may activate the reflex, leading to the faint. Knowing the various triggers for VVS is clinically important, as recognising them may be instrumental in diagnosing syncope (Moya et al., 2009). Importantly, in many cases of VVS patients have some warning signs. These signs include dizziness, nausea, pale skin, blurred vision and sweating (Marchiondo, 2010). However, these warning signs may themselves become triggers for certain patients as they form part of a psychological feedback loop, which we will discuss later. After an episode, symptoms may persist because of continued low blood pressure.

Presentation at Clinic/ Diagnosis

Patients who present at a clinic with syncope are often fearful of impending sudden death. Cardiac problems are assessed and ruled out. Some patients are referred for a Tilt Table Test (TTT), which assesses the physiological susceptibility in patients with VVS (Blount, Morris, Cheng, Campbell, & Brown, 2004). However, the efficacy of TTT in diagnosis has not been completely supported (e.g., Levine, 1999), so clinical history taking remains the method of choice in clinical settings. Most syncope is benign in nature and, with accurate diagnosis and appropriate treatment, it can be resolved in the majority of patients (Moya et al., 2009). Most psychologists will only receive a diagnosis and appropriate treatment, it can be resolved in the majority of patients (Moya et al., 2009). Most psychologists will only receive a referral when the primary care physician has ruled out an identified physical cause. In such cases, there may be psychological mechanisms that are precipitating and sustaining the incidence of syncope, such as catastrophisation or blood phobia, which we will discuss in the following sections.

Psychological Factors/Impact of Syncope

The experience of VVS for patients is at best inconvenient and at worst may be perceived as threatening and disabling. Many patients with recurrent syncope have been shown to have a reduced QOL, similar to that of patients with severe rheumatoid arthritis or chronic back pain (Linzer et al., 1991). QOL appears to decrease as the frequency of syncope increases and impairment is evidenced in many dimensions, for example in mobility, activities of daily living and self-care (Rose, Koshman, Spreng & Sheldon, 2000). Problems in relationships with friends, family and spouses have also been noted (Linzer et al., 1991) while VVS is also associated with school and work absences (Newton, Kenny, & Baker, 2003). The impact upon QOL is reversible and improves when the frequency of syncope is reduced. This can be seen, for example, when syncope are reduced on pacemaker implantation (Sheldon, Rose, Flanagan, Koshman, & Killam, 1996).

Another “safety behaviour” noted by the author among some clients with VVS is walking everywhere with a bicycle which has shopping baskets on the front basket. Clients always report surprise that nobody ever guesses they were using a bicycle as a rather large prop, nobody ever asks them “why do you never cycle the bicycle?” Other clients who have blood injury (BiT) or needle phobias will present with dental care issues as they will avoid the dentist at all costs to their teeth, and those with a blood phobia will report walking a few streets away from a hospital, as the streets and environment near that hospital tend to create a sense of unease and can precipitate feelings of a VVS commencing. Some clients will report an increase in VVS the closer a consultant’s appointment approaches, so much so that the anticipatory anxiety can in itself bring on symptoms, and the clients then reasoning that they will miss their appointment due to sickness or a number of VVS just prior to the appointment time. Of course, this information must be kept in mind when planning sessions with a client. Their avoidance behaviour might be quite strong and it would make sense that the avoidance would generalise to psychologists’ appointments too. A brief discussion about avoidance behaviour and understanding of the normalcy of this is often useful.

In anxiety disorders, avoidance and other self-protection behaviours have been shown to reinforce the belief that particular situations are dangerous and prevent the development of more adaptive ways of coping with the condition (Clark, 1986). This suggests that how people understand their condition, their experiences and their predictions about the consequences of fainting is associated with different styles of coping and adjustment, and subsequent QOL (Gracie, Baker, Freeston, & Newton, 2004). Similarly, considering that autonomic symptoms of syncope and anxiety are alike, fear arousal may amplify the physiological signs associated with syncope and lead to an increase in the fear associated with syncope. This creates a situation where the symptoms become entrenched in a psychological disorder and this influences the natural history of what is a chronic relapsing condition (Gracie, Newton, Norton, Baker, & Freeston, 2006). Considering the link between anxiety/fear and recurring syncope, psychological theories of anxiety disorders (e.g., panic disorder and phobias) can contribute to an understanding of recurring syncope, specifically emotionally triggered syncope. We will now explore some of these theories.

Relevant Theories

Catastrophic Cognitions and Vicious Cycle Theories

These models propose that patients misinterpret external or internal cues in a catastrophic manner and, as a result of these catastrophic cognitions, the symptoms are maintained (Moore & Zebb, 1999). The core thesis here is that syncopeal episodes are caused by catastrophic beliefs about certain internal bodily sensations or external stimuli, which make the patient react to these stimuli with syncope. A typical example of a catastrophic cognition is on noticing a symptom of syncope, such as sweating, the patient will have the terrifying thought, such as “I’m going to faint and hurt myself.” Triggers and warning signs form part of a causal chain of somatic sensations, which cause catastrophic thoughts, which in turn cause syncope. This formulation includes a positive feedback loop or vicious circle between bodily sensations, accompanying anxiety and anxiety generated from perceiving them. As with internal stimuli, external stimuli, for example, hearing a loud noise may form part of a catastrophic cognition, such as “there’s someone breaking into my house, I’m in danger.” For some people, this belief may be strong enough to induce emotionally triggered syncope.

Diathesis-Stress Model

The diathesis-stress model explains behaviour as both a result of biological and genetic factors, and life experiences. Blount et al. (2004) examined whether a person’s physical predisposition or diathesis combines with stressful life experiences, such as poor parental and/or personal adjustment, to yield a particular set of symptoms. They found that the frequency of children’s syncopeal episodes and the number of Emergency Room (ER) visits due to syncope were associated with their own psychological functioning and the psychological functioning of their parents. This was especially salient for those children who tested negative for VVS on the TTT. Those in the negative VVS group did not have a physical diathesis sufficient for diagnosis, yet they were symptomatic, displaying a frequency of syncope and ER visits similar to that of the children in the positive VVS group. For this group,
Table 1. Some Non-pharmacological Treatments of VVS (Alboni, 2010)

<table>
<thead>
<tr>
<th>Treatment of the impending reflex</th>
<th>Prevention of the impending reflex</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assumption of a supine position</td>
<td>Avoidance of prolonged standing</td>
</tr>
<tr>
<td>Pacemaker implantation</td>
<td>Avoidance of hot crowded environments</td>
</tr>
<tr>
<td>Physical counter-pressure manoeuvres and applied tension</td>
<td>Hydration, salt intake</td>
</tr>
<tr>
<td>Self-talk, address catastrophisation and apply muscle tension</td>
<td>Tilt training</td>
</tr>
<tr>
<td>Psychological therapy</td>
<td></td>
</tr>
</tbody>
</table>

syncope frequency and ER visits were highly associated with the stress component of the equation, in particular with their fathers' and mothers' psychological symptoms, and their own internalising symptoms.

**Behavioural Model**

Many people who experience BI phobia faint at the sight of blood. Like most specific fears, development of BI phobia may occur through multiple pathways including direct conditioning, learning through observing others (vicarious learning) or through receiving fear-relevant information (Kleinkechte, 1994). Conditioning theories of anxiety describe the process by which one learns to be afraid. Kleinkechte (1967) investigated the relationship between VVS and fear of blood and injuries and found that a greater proportion of fainters, relative to non-fainters, reported having family members who also fainted to BI stimuli. There is potential here for a conditioning theory of VVS, where both the phobia and the response to it are learned.

**Treatment**

The main goal of treatment/therapy is to prevent the recurrences of syncope, thereby addressing the infringements on QOL and preventing the associated injuries caused by falling. The majority of patients with a single VVS require only reassurance and education regarding the nature of the disorder. If a more aggressive treatment is necessary, conventional therapy usually involves advising the use of volume expanders such as increased dietary salt intake (Calkins, 1999). Various medications have also been prescribed for the treatment of VVS, but the research as to their efficacy is equivocal (Kauffman & Freeman, 2004). In VVS, such as emotionally triggered syncope, it can be more effective to explore the psychological aspects of the syncope, for example the degree of catastrophisation and the patient's psychosocial impairment. Treatment of VVS can be considered in two domains: treatment of the impending reflex (afferent pathway) and prevention of the reflex (afferent pathway) (Alboni, 2010). A list of non-pharmacological interventions for syncope can be seen in Table 1.

We will now explore the use of physical manoeuvres and psychological therapies as potential treatments for VVS.

**Counter-Pressure Manoeuvres and Applied Tension**

Most patients with VVS experience prodromal symptoms and when these begin, physical manoeuvres that can increase venous return may act to abort an attack. For example, Brigcode et al. (2002) found that isometric counter-pressure manoeuvres of the legs (i.e., leg crossing and tensing of legs, while standing or sitting) increases blood pressure during an impending VVS. Similarly, isometric arm exercises such as performing handgrip and arm tensing have been shown to be an effective way of aborting an impending VVS (Krediet, Van Dijk, Linzer, Van Lieshout & Wieling, 2002). Kim et al. (2005) examined the effectiveness of squatting in aborting an attack, and their results indicated that squatting was comparable in effectiveness to leg-crossing with applied muscle tension. However, secondary injury during a VVS is often a concern for patients and squatting has been judged as safer than leg-crossing. Given the ease of performing these manoeuvres and the evidence of their effectiveness, counter-pressure manoeuvres should be considered as first-line treatment for a patient's prodromal symptoms (Chen, Benditt, & Shen, 2008).

Furthermore, Newton et al. (2003) found that there was a significant reduction in number of syncope, improvements in QOL and ability to return to work or school among nine patients with severe VVS when they were treated using CBT and applied muscle tension. The CBT components included identifying and restructuring unhelpful beliefs, addressing maladaptive somatic attention; reducing avoidance of certain activities and situations; the use of applied tension, and addressing general difficulties such as sleep hygiene and coping with the reactions of others. Cognitive behaviour therapists argue that therapies can make a significant contribution to treatment. It may also be useful to identify the situations where spouses are maintaining avoidant behaviours for the individual with VVS and introduce spouse-assisted therapy in these cases.

**Psychosocial Factors in Treating Syncope**

A recent study has confirmed that those with VVS who have not responded to conventional treatment (i.e., increased fluid intake and counter manoeuvres, including a self-tilt training programme) have higher levels of psychosocial impairment and distress (Gracie et al., 2006). Results from this study revealed there was no difference between non-responders and responders across variables such as gender, age, past history of fainting, but non-responders were shown to be significantly more anxious and depressed than responders. They also reported more fear, worry and had significantly higher VVS-related impairment. Furthermore, high levels of self-reported psychosocial impairment at the time of diagnosis were related to non-response to treatment. This is similar to the research findings on sufferers of chronic back pain (Pincus, Burton, Vogel, & Field, 2002). Considering this, screening at the point of diagnosis could allow clinicians to identify those patients who are less likely to respond to conventional treatment (Flinn, Baker, Freeston, & Newton, 2009). Assessing patients with the Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1987) will identify those that have a propensity to panic when aroused and have low tolerance for anxious situations. Early intervention to prevent the entrenchment of symptoms and avoidance behaviour is the rule of thumb for clients who are experiencing VVS (Gracie et al., 2006).

**Psychological Therapies**

Given the high prevalence of psychological dysfunction in patients with VVS and the role of illness-related beliefs and predictions in this condition, psychological interventions can be effective in the management of VVS. Some case reports have described the successful use of applied tension and cognitive behaviour therapy (CBT) in patients with VVS. For example, Van Dijk, Velzeboer, Destree-Vonk, Linzer and Wieling (2001) described a case of syncope due to an emotionally evoked vasovagal response to seeing blood. The patient's BI phobia was treated using systematic desensitisation, muscle tensing and cognitive techniques. CBT was used to teach the patient to apply realistic and reassuring thoughts to counteract catastrophic interpretations of their physical symptoms.
Summary
Management of VVS, especially in response to fear or emotional upset, appears best served by a medical and psychological team approach. As psychologists become more involved in primary care across Ireland, knowledge of VVS and its treatment will become more salient. In addition to the medical implications, there are a number of psychological issues that can be important in the maintenance of VVS. Psychological approaches offer an alternative and/or a complementary approach to the traditional medical management of VVS.

References