Clinical review

Neurocardiogenic syncope

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Syncope is a common problem that many clinicians may encounter in various outpatient settings. Neurally mediated syncopal syndrome includes carotid sinus syndrome, situational syncope, and neurocardiogenic syncope (also known as vasovagal syncope), which is the most common cause of syncope in both children and adults, accounting for 50-66% of unexplained syncope.[1] [2] The distinction between neurocardiogenic syncope and other causes of fainting is essential, as the prognosis and treatment are different.

Sources and selection criteria

We selected articles from the PubMed database by using the search words "syncope" and "neurocardiogenic syncope." Inclusion criteria were articles published in English, in peer reviewed journals, between 1980 and 2004. Exclusion criteria were articles not published in English, case reports, and articles not published in peer reviewed journals. We incorporated guidelines from the American College of Cardiology, European Society of Cardiology, and American Heart Association, along with a summary of clinical trials. We selected 31 references for this review.

Definition and incidence

Syncope is defined as a transient loss of consciousness, with loss of posture (that is, falling). Commonly described as "fainting," "passing out," or "blackout," syncope accounts for 3% of visits to emergency departments and 6% of all admissions to hospital.[1] [3] It occurs relatively often in all age groups, ranging from 15% in children aged under 18 years to 23% in elderly patients aged over 70.[4] The prevalence and incidence of syncope increase with advancing age,[5] with a 30% recurrence rate.[3]

Neurocardiogenic syncope, with a mean prevalence of 22% in the general population,[2] is defined as a syndrome in which "triggering of a neural reflex results in a usually self-limited episode of systemic hypotension characterized by both bradycardia (asystole or relative bradycardia) and peripheral vasodilation."[6]

Causes of syncope

Syncope is a symptom, not a disease, and can be classified according to the underlying cause: neurological,

P+ *Additional references and protocols for tilt testing are on bmj.com*

Summary points

Syncope, commonly described as "fainting," is a symptom, not a disease, and can be classified according to the cause, the most common of which is neurocardiogenic syncope

Neurocardiogenic syncope (also known as vasovagal syncope) is a benign condition characterised by a self limited episode of systemic hypotension

Stimulation of the cardiac C fibres results in vasodilation and increased vagal tone, with consequent reduction in cardiac filling and bradycardia, with ensuing syncope

Differential diagnoses include carotid sinus hypersensitivity (resulting from an extreme reflex response to carotid sinus stimulation) and orthostatic hypotension (failure of the autonomic reflex response)

The mainstay of management is education of the patient to avoid situations that predispose to syncope, with anxiety management, coping skills, and reassurance of the patient and others that this is a benign condition

metabolic, psychiatric, and cardiac[7]; cardiac syncope is the most common form. Cardiac syncope includes syncope due to mechanical or structural heart disease, cardiac arrhythmias, and neurocardiogenic syncope (box 1).[7]

Neurocardiogenic syncope is caused by an abnormal or exaggerated autonomic response to various stimuli, of which the most common are standing and emotion.[8] [9] The mechanism is poorly understood but involves reflex mediated changes in heart rate or vascular tone, caused by activation of cardiac C fibres.[2]

Pathophysiology of neurocardiogenic syncope

Stimulation of the cardiac C fibres is implicated in neurocardiogenic syncope.[7] An abnormal autonomic response occurs, resulting in vasodilation and increased vagal tone, with subsequent reduction in car-

diac filling and bradycardia, which ultimately leads to syncope (fig 1). Stimulation of the medullary vasodepressor region of the brain stem may occur owing to activation of various receptors, such as cardiac C fibres (mechanocardiac receptors), cardiopulmonary baroreceptors, cranial nerves, cerebral cortex, and gastrointestinal or genitourinary mechanoreceptors (fig 2).[2][4][7]

In hypovolaemia and other conditions of reduced preload, sympathetic tone is increased, resulting in hypercontractility of the volume depleted ventricle (with increase in myocardial inotropy and chronotropy), with subsequent stimulation of the cardiac C fibres.[3][7][10] This results in a combination of parasympathetic enhancement (bradycardia) and decreased sympathetic tone (hypotension), with ensuing syncope.[3][10]

Clinical signs and symptoms

Although presentation of neurocardiogenic syncope is similar to that of other types of syncope, loss of consciousness in patients with neurocardiogenic syncope may be preceded by prodromata such as nausea, diaphoresis, lightheadedness, blurred vision, headaches, palpitations, paraesthesia, and pallor,[3][7][10][11] which usually occur in the upright position (with downward displacement of 300-800 ml of blood[3]) and resolve almost immediately when the patient assumes the supine position.[7] In addition, after recovery, patients with neurocardiogenic syncope may complain of a "washed out" and tired feeling.[4][7]

Assessment of the symptoms and setting may yield clues as to the possible cause of the syncope. Syncope after cough, defecation, and micturition suggests
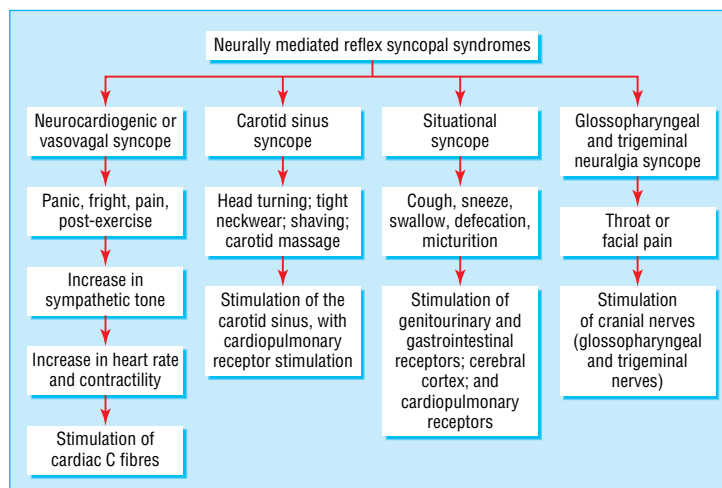
Box 1: Causes of syncope[4][7]

Cardiac causes

• Structural cardiac or cardiopulmonary disease (aortic stenosis, mitral stenosis, pulmonary stenosis, left atrial myxoma, aortic dissection, acute myocardial infarction, cardiac tamponade, pulmonary embolism, obstructive cardiomyopathy)

• Cardiac arrhythmias (tachyarrhythmias, bradyarrhythmias)

• Neurally mediated syncopal syndrome (includes neurocardiogenic or vasovagal syncope, carotid sinus syncope, and situational syncope)

• Orthostatic (or postural) hypotension

Metabolic causes

• Hypoxia

• Hypoglycaemia

• Hyperventilation

Psychiatric causes

• Somatisation disorders

• Hysteria

• Panic

• Fright

Neurological causes

• Seizure disorders

• Transient ischaemic attacks

• Subclavian steal syndrome

• Normal pressure hydrocephalus

Fig 1 Pathophysiology of neurally mediated reflex syncopal syndromes

situational syncope; syncope associated with throat or facial pain (glossopharyngeal or trigeminal neuralgia) is indicative of neurally mediated syncope with neuralgia; and syncope after pain, fear, or noxious stimuli suggests neurocardiogenic syncope.[4] Carotid sinus syncope may occur with rotation or turning of the head or pressure on the carotid sinus (for example, carotid massage, shaving, tight collars or neckwear, or tumour compression).[4]

Diagnosis

A thorough assessment of associated symptoms, setting, drugs, and family history and a physical examination often provide important clues to the cause and help to guide baseline testing. However, the history and physical examination are non-diagnostic in more than 50% of patients with neurocardiogenic syncope.[6] Structural cardiac disease and cardiac arrhythmias must be ruled out, especially in elderly people, who have a higher incidence of syncope. Differential diagnoses include carotid sinus hypersensitivity and orthostatic hypotension.

Neurocardiogenic syncope results from excessive autonomic reflex activity, which shows as abnormal vascular tone and heart rate. In contrast, orthostatic hypotension is a failure of the autonomic reflex response.[12] Orthostatic hypotension affects 5% of the

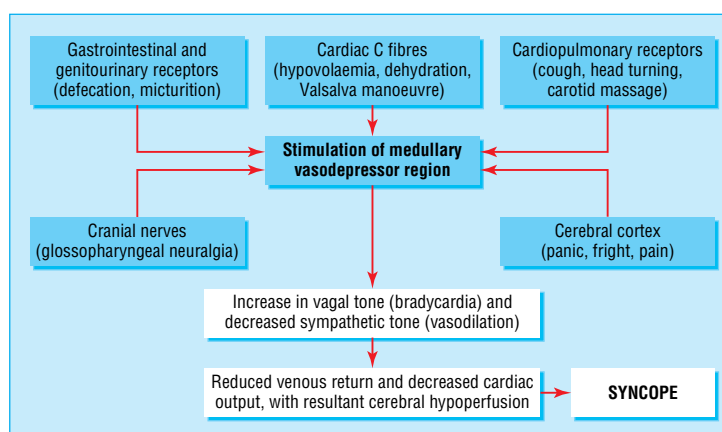**Fig 2** Activation of receptors in neurally mediated syncopal syndromes

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Box 2: Indications and contraindications for tilt table testing[6]

Indications
- Recurrent syncope or single syncopal episode accompanied by physical injury or motor vehicle crash or occurring in a high risk setting (for example, pilot, surgeon, commercial vehicle driver) and no evidence of structural cardiovascular disease; or presence of structural cardiovascular disease but other causes of syncope ruled out by diagnostic testing
- Syncope induced by or associated with exercise
- Further evaluation of patients in whom an apparent specific cause of syncope has been established (for example, asystole, high atrioventricular block) but susceptibility to neurocardiogenic syncope may affect treatment plan

Contraindications
- Syncope with severe left ventricular outflow obstruction (for example, aortic stenosis)
- Syncope in presence of severe mitral stenosis
- Syncope in setting of known critical proximal coronary artery disease
- Syncope in setting of known critical cerebrovascular disease

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population and 7-17% of patients in acute care settings.[13] It is more common in elderly people and is attributed to an age related decrease in physiological function (reduction in baroreceptor sensitivity) and polypharmacy (including various vasoactive drugs).[13] Orthostatic hypotension is a drop in blood pressure on assuming an upright posture and is due to failure of the autonomic system to compensate for venous pooling in the lower extremities, which results in reduced venous return, decreased cardiac output, and cerebral hypoperfusion.[13] Carotid sinus massage is done to rule out carotid sinus syndrome or hypersensitivity as the cause of syncope. This procedure should be avoided in patients with carotid bruits or a history of cerebrovascular events or transient ischaemic attacks, because of the risk of neurological complications.[4] Carotid sinus hypersensitivity is defined as "syncope or presyncope resulting from an extreme reflex response to carotid sinus stimulation."[14] This reflex response has two components:
- A cardioinhibitory component, due to enhanced parasympathetic tone, manifested by slowing of the sinus rate or prolongation of the PR interval and advanced atrioventricular block, alone or in combination
- A vasodepressor component, due to decreased sympathetic activity, resulting in loss of vascular tone and hypotension, independent of changes in heart rate.[14]

Carotid sinus hypersensitivity is diagnosed when a ≥ 50 mm Hg reduction in systolic blood pressure or a ventricular pause of ≥ 3 s occurs when a 5-10 s carotid sinus massage is done.[4] Although the condition is rare before the age of 40, the prevalence increases with age and with comorbidities (cardiovascular, cerebrovascular, and neurodegenerative).[4] The condition is recognised in up to 45% of elderly patients with syncope, falls, and dizziness.[15]

Tests

Once cardiac arrhythmias, structural heart disease, and non-cardiac causes of syncope have been ruled out, head up tilt testing is usually the first line of testing. Tilt testing is an orthostatic stress test, used when neurocardiogenic syncope is suspected. In people without neurocardiogenic syncope, tilting causes a reduction in venous return, with subsequent baroreceptor stimulation and increased α and β adrenergic tone, averting syncope. In patients with neurocardiogenic syncope, tilting causes decreased venous return, but sympathetic tone increases with stimulation of cardiac C fibres. This

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Box 3: Indications for permanent pacing in neurocardiogenic syncope and carotid sinus hypersensitivity[14]

Class I
- Recurrent syncope caused by carotid sinus stimulation; minimal carotid sinus pressure induces ventricular asystole of >3 s duration in the absence of any drug that depresses the sinus node or atrioventricular conduction (Level of evidence C)

Class IIa
- Recurrent syncope without clear, provocative events and with a hypersensitive cardioinhibitory response (Level of evidence C)
- Syncope of unexplained origin when major abnormalities of sinus node function or atrioventricular conduction are discovered or provoked in electrophysiological studies (Level of evidence C)

Class IIb
- Neurally mediated syncope with significant bradycardia reproduced by a head up tilt with or without isoproterenol or other provocative manoeuvres (Level of evidence B)

Class III
- A hyperactive cardioinhibitory response to carotid sinus stimulation in the absence of symptoms
- A hyperactive cardioinhibitory response to carotid sinus stimulation in the presence of vague symptoms such as dizziness, lightheadedness, or both
- Recurrent syncope, lightheadedness, or dizziness in the absence of a hyperactive cardioinhibitory response
- Situational vasovagal syncope in which avoidance behaviour is effective

Strength of recommendation
Class I—Conditions for which evidence or general agreement exists that a given procedure or treatment is beneficial, useful, and effective
Class II—Conditions for which conflicting evidence or a divergence of opinion exists about the usefulness or efficacy of a procedure or treatment
Class IIa—Weight of evidence or opinion is in favour of usefulness or efficacy
Class IIb—Usefulness or efficacy is less well established by evidence or opinion
Class III—Conditions for which evidence or general agreement exists that a procedure or treatment is not useful or effective and in some cases may be harmful

Strength of evidence
Level A—Data from multiple randomised clinical trials or meta-analyses
Level B—Data from a single randomised trial or multiple non-randomised trials
Level C—Consensus opinion of experts

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leads to stimulation of the medullary vasodepressor region of the brain stem, resulting in sudden reduction in sympathetic tone (vasodilation) and concomitant increase in vagal tone (bradycardia), with consequent syncope. Tilt testing is considered positive if the original symptoms are reproduced, along with an abrupt drop in blood pressure, heart rate, or both.[4 10] Box 2 summarises indications and contraindications for tilt testing.[6] See bmj.com for protocols for tilt testing, including pharmacological provocation.[6]

Other ways of evaluating syncope can be grouped into three main categories: electrocardiographic recordings (including event recorders); analysis of heart rate variability (to assess susceptibility to neurocardiogenic syncope); and other tests (including Valsalva manoeuvre) to assess the autonomic function. However, more research is needed to determine the diagnostic value of these methods.[6]

Treatment of neurocardiogenic syncope

Treatment consists of education, manoeuvres to avert syncope, drug treatment, and pacemakers. Education, the mainstay of treatment, includes avoidance of predisposing situations (for example, dehydration, stress, alcohol consumption, extremely warm environ-

ments, tight clothing),[4 10] anxiety management and coping skills, and reassurance of the patient and others that this is a benign condition. Drug treatments include β blockers, α agonists, selective serotonin reuptake inhibitors, fludrocortisone, disopyramide, scopolamine, and anticholinergic agents.

Drug treatment
β blockers are preferred as initial treatment,[10] as they are believed to reduce the degree of mechanoreceptor activation and block the effects of circulating catecholamines.[4] However, randomised controlled trials fail to support the efficacy of these drugs, showing no difference from placebo.[4 16 17] Furthermore, β blockers may worsen syncope through their negative chronotropic effects and atrioventricular node blocking effects.[3]

α agonists work by increasing peripheral vascular resistance and reducing vascular capacitance (to cause increased venous return).[3 10] Midodrine, an α agonist, has been shown to be effective in several randomised controlled clinical trials.[18–20]

Selective serotonin reuptake inhibitors selectively block serotonin, which has been shown to induce vagally mediated bradycardia and blood pressure lowering.[3 10] Selective serotonin reuptake inhibitors have been used to treat syncope, but their efficacy has been documented in only one randomised controlled trial of 68 patients to date.[21] Side effects of these agents include nausea, insomnia, weight gain, and sexual dysfunction.[3]

Fludrocortisone, a mineralocorticoid that promotes renal reabsorption of sodium to cause increased blood volume,[3 10] has been used in the treatment of vasodepressor syncope in both children and adults.[22 23] Vascular volume and preload are maintained through the resultant sodium and water retention by fludrocortisone, thereby preventing activation of the cardiac mechanoreceptors.[3] However, caution is needed in elderly patients because of the risk of hypertension, cardiac failure, and oedema.[24]

Disopyramide, a class Ia antiarrhythmic agent with anticholinergic and negative inotropic effects, is not considered first line treatment because of the risk of proarrhythmic and anticholinergic side effects (dry mouth, constipation, blurred vision, and urinary retention).[3] Enhanced vagal activity in vasodepressor syncope is counteracted by using anticholinergic agents,[10] which are useful when syncope is due solely to increased vagal tone and not to vasodilation.[3] Scopolamine, an anticholinergic agent, has central nervous system depressant effects and has been used successfully in some patients with syncope.[3]

The table summarises clinical trials of various drugs. Most randomised, placebo-controlled clinical studies to date show no differences between the treatment and placebo groups. Other trials that have shown benefit from treatment were not randomised. Most clinical trials, whether randomised or not, had small sample sizes (ranging from 11 to 68 participants) and involved only short term treatment and follow up (most ranging from one week to six months).

Pacemaker treatment
In most people with neurocardiogenic syncope, a fall in blood pressure precedes bradycardia, so pacing may be ineffective in most patients. However, dual chamber pacing may be effective in reducing symptoms if there is a large cardioinhibitory component.[14 25] The cardio-

inhibitory component results from enhanced parasympathetic tone, manifested by slowing of the sinus rate or prolongation of the PR interval and advanced atrioventricular block, either alone or in combination.[14] Box 3 lists indications for permanent pacemaker treatment.[6] Box 4 outlines a treatment protocol, along with strength of supporting evidence and strength of recommendation.[4]

Conclusion

Syncope is associated with considerable morbidity (including injury due to falls or motor vehicle crashes) and poses a potential danger if episodes occur during critical activity (such as participation in sport, driving, operating heavy or critical machinery). Suspension of driving and piloting privileges after syncopal episodes varies according to different state and country laws. The mortality due to syncope varies according to the cause: cardiac causes (arrhythmias or cardiovascular disease) have a 20-30% mortality compared with 5-10% mortality for non-cardiac causes.[3] Although frequent or recurrent episodes can negatively affect quality of life and employability, neurocardiogenic syncope is generally considered a benign condition as episodes are self limiting. Understanding of the pathophysiology of neurocardiogenic syncope is necessary to guide appropriate management. Finally, more randomised controlled clinical trials are needed to assess the efficacy of the various treatment strategies used.

1 American Heart Association. Syncope. www.americanheart.org/ presenter.jhtml?identifier = 4749 (accessed 30 Jan 2004).

Drug treatment in syncope: summary of clinical trials*

Investigators	Drug	Study design	Sample	Results
Ventura et al (2002)[w1]	β blocker (metoprolol, propranolol, or no treatment)	Prospective randomised controlled clinical trial	n=56 (36 female, 20 male); mean (SD) age 44 (18) years	β blocker group had fewer recurrent episodes of syncope *v* no treatment
Madrid et al (2002)[16]	β blocker (atenolol)	Prospective randomised double blind placebo controlled clinical trial	n=50 (26 on atenolol, 24 on placebo)	Atenolol group had similar No of recurrent syncope episodes to placebo group, with no difference in time to first syncopal recurrence
Flevari et al (2002)[17]	β blocker (propranolol, nadolol, or placebo)	Randomised crossover controlled clinical trial	n=30 consecutive patients with vasovagal syncope and positive tilt test (all were serially and randomly assigned to propranolol, nadolol, or placebo for three months each, with crossover)	After nine month follow up: no difference in recurrence of syncope or presyncope among the three groups; all three treatments were equally effective in treating vasovagal syncope
Mahanonda et al (1995)[w2]	β blocker (oral atenolol) *v* placebo for one month	Randomised controlled clinical trial	n=42 (21 on atenolol, 21 on placebo); all had at least one syncopal episode or two presyncopal episodes occurring one month before presentation and had positive isoproterenol tilt test	After one month: 62% of atenolol group *v* 5% of placebo group had negative tilt test (P=0.0004), and 71% of atenolol group *v* 29% of placebo group reported symptomatic improvement (P=0.02)
Takata et al (2002)[w2]	Selective serotonin reuptake inhibitors (paroxetine/paxil) 20 mg/day *v* placebo for six weeks	Randomised double blind controlled trial	n=25 (19 completed the study: 9 on paroxetine, 10 on placebo)	Paroxetine did not attenuate sympathoinhibition or vagotonia (did not prevent syncope)
DiGirolamo et al (1999)[21]	Selective serotonin reuptake inhibitors (paroxetine/paxil) 20 mg/day *v* placebo for one month	Randomised controlled clinical trial	n=68 (42 female, 26 male); mean (SD) age 44.7 (16.5) years	61.8% of paroxetine group *v* 38.2% of placebo group had negative tilt test; 17.6% of paroxetine group *v* 52.9% of placebo group had spontaneous syncope (P<0.0001); paroxetine improved symptoms of vasovagal syncope
Kaufmann et al (2002)[18]	Selective α_1 adrenergic agonist (midodrine) *v* placebo	Randomised double blind crossover placebo controlled trial	n=12 (with recurrent neurally mediated syncope)	Midodrine significantly improved orthostatic tolerance during tilt test in patients with neurally mediated syncope (P<0.02)
Perez-Lugones et al (2001)[19]	Selective α_1 adrenergic agonist (midodrine) *v* fluid and salt tablets for six months	Prospective randomised controlled trial	n=61 (31 on midodrine, 30 on fluid and salt tablet)	81% of midodine *v* 13% of fluid and salt tablet group remained asymptomatic (P<0.001); midodrine was beneficial in treating neurally mediated syncope
Ward et al (1998)[20]	Selective α_1 adrenergic agonist (midodrine) *v* placebo for one month	Randomised double blind placebo controlled trial	n=16 (11 female, 5 male); mean (SD) age 56 (18) years	Midodrine group had more symptom-free days and fewer positive tilt tests *v* placebo group; midodrine reduced symptom frequency and symptoms during tilt test
Mitro et al (1999)[w4]	Selective α_1 adrenergic agonist (midodrine)	Prospective non-randomised clinical trial	n=41 (23 female, 18 male); mean age 34 years; with recurrent syncope and positive tilt test	95% had no inducible presyncope or syncope during repeat tilt test; the effective dose was 2.5 mg po bid in 25 patients, and 5 mg po bid in 16 patients; on mean (SD) follow up of 19 (9) months, 97% with negative repeat tilt test remained free of syncope recurrence
Yu and Sung (1997)[w5]	Anticholinergic (propantheline bromide), mean (SD) 64.3 (21) mg/day for seven days	Prospective non-randomised clinical trial	n=16 (5 female, 11 male); mean (SD) age 48.8 (15.1) years	81% of patients had no inducible presyncope or syncope on repeat tilt test; on mean (SD) follow up of 15.2 (7.4) months in 12 patients, 33% had clinical recurrence of symptoms
Da Costa et al (1993)[22]	Salt retaining mineralocorticoid (fludrocortisone) for two weeks	Prospective non-randomised clinical trial	n=11; mean (SD) age 83 (5) years; all patients had daily dizziness and had vasodepressor carotid sinus syndrome	Fludrocortisone effectively reduced vasodepressor response and relieved symptoms of vasodepressor carotid sinus syndrome

*As noted in text, most randomised placebo controlled clinical studies to date show no differences between treatment and placebo groups. Other trials that have shown benefit from treatment were not randomised. Most clinical trials, whether randomised or not, consisted of small sample sizes (ranging from 11 to 68 participants) and involved only short term treatment and follow up (most ranged from one week to six months).

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Additional educational resources

Resources for healthcare professionals

Heart Rhythm Society (www.hrspatients.org/patients/signs_symptoms/fainting/non-cardiovascular_syncope.asp)—describes neurocardiogenic syncope and its causes, diagnosis, and treatment

Brignole M, Alboni P, Benditt D, Bergfeldt L, Blanc JJ, Bloch Thomsen PE, et al for the European Society of Cardiology. Guidelines on management (diagnosis and treatment) of syncope. *Eur Heart J* 2001;22:1256-306

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Four key ongoing research studies

Relationship of autonomic function to hypnotic susceptibility—a study to investigate the relation between susceptibility to hypnosis and regulation of the autonomic nervous system

Propranolol for syncope with sympathoadrenal imbalance—evaluating treatment with oral propranolol for a particular form of neurocardiogenic syncope characterised by a neuroendocrine pattern called sympathoadrenal imbalance

Clinical laboratory evaluation of chronic orthostatic intolerance—to identify and characterise distinct types of chronic orthostatic intolerance

Randomized study of midodrine, an adrenergic agonist, in patients with neurally mediated syncope—to determine the efficacy of midodrine, a selective α₁ adrenergic agonist, in preventing neurally mediated syncope (For more detailed information, visit www.clinicaltrials.gov and enter "syncope" as search term)

The other side of medicine

Going to work in the week before Christmas, I was reminded of an event in my school days. As 11 year olds, we were asked to write a piece of prose about Christmas. Understandably, we all set to work writing about food, presents, and festivities. Well, not quite all: one of my classmates surprised us all, and left an impression on some of us, by writing an account titled "The other side of Christmas," in which he described the Christmas experience for a homeless person. Well written, but not pleasant reading.

I am currently working as a senior house officer in paediatrics, and I have been taken aback by the generosity of the many individuals and organisations who have given their time and gifts to the children who will spend their Christmas on an acute paediatric ward. We have had visits from the local newspaper and local B-list celebrities, and there have been, quite literally, van loads of gifts arriving daily.

The ward office is now a storeroom, and the gifts piled high are remarkable, including televisions, video players, and hi fi systems. There are numerous video games and CDs, and a six foot long game of table football. There are boxes of sweets, cuddly toys, and craftwork materials. Four lucky children look set to receive a personal CD player, though it has not yet been decided how all these wonderful gifts will be distributed. How do you decide who should get the adult size mountain bike that was wheeled in?

All of this is a far cry from the build up to Christmas that I witnessed last year. Then, as a senior house officer in psychiatry, I saw the staff club together to try to buy some gifts for the men and women who would be spending Christmas as patients on the ward. There were no local benefactors and no rush of people making donations for these patients. There was certainly no visit from the local football team, who we are expecting on the paediatric ward this week. In the end, we pooled enough money to buy the men a tub of talcum powder and the women some basic toiletries.

We are really grateful for the generosity that has been shown towards the children who are unwell, but I cannot help but think of patients with mental health problems, who really are the other side of medicine.

Edward C A Barrett *senior house officer, Queen's Hospital, Birmingham*