Syncope

A Diagnostic and Treatment Strategy

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Definition of SYNCOPE

"…a sudden and transient loss of consciousness that may result from a wide spectrum of cardiovascular, neurologic and metabolic abnormalities"

- Self-limited loss of consciousness and postural tone
- Relatively rapid onset
- Variable warning symptoms
- Spontaneous complete recovery

Greek words: 'syn' = 'with' 'koptein' = 'to cut' or interrupt

Definition of SYNCOPE

Section I:

Prevalence and Impact

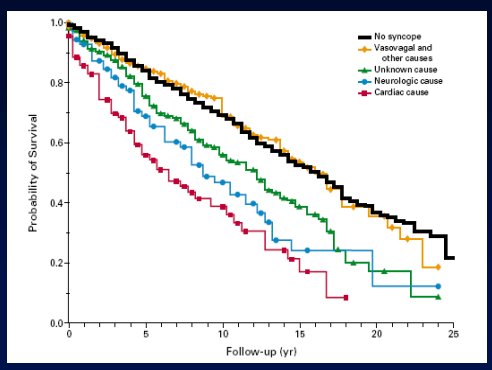
Syncope Reported Frequency

Individuals <18 yrs	15%
Military Population 17- 46 yrs	20-25%
Individuals 40-59 yrs*	16-19%
Individuals >70 yrs*	23%

*during a 10-year period

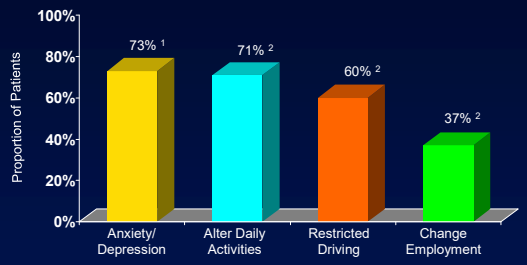
Brignole M, Alboni P, Benditt DG, et al. Eur Heart J, 2001; 22: 1256-1308.

Syncope Mortality



Soteriades ES, Evans JC, Larson MG, et al. Incidence and prognosis of syncope.*N Engl J Med.* 2002;347(12):878-885. [Framingham Study Population]

Impact of Syncope



[1]Linzer, *J Clin Epidemiol*, 1991.
[2]Linzer, *J Gen Int Med*, 1994.

FALLS IN THE ELDERLY

- Each year, more than one third of persons over the age of 65 fall.
- In half of such cases falls are recurrent
- One in 10 falls results in serious injury.
- Falls are responsible for two-thirds of the deaths resulting from unintentional injuries.

Major health and socioeconomic problem

Terra Mitica, Benidorm, Spain

Syncope:
A Symptom…Not a Diagnosis

Syncope: Aetiology

Cardiac related

Neurally-Mediated	Orthostatic	Cardiac Arrhythmia	Structural Cardio-Pulmonary	Non-Cardio-vascular
1 • Vasovagal • Carotid Sinus • Situational ➢Cough ➢Post-micturition	2 • Drug Induced • Dehydration • ANS Failure ➢Primary ➢Secondary	3 • Brady ➢Sick sinus ➢AV block • Tachy ➢VT ➢SVT • Long QT Syndrome	4 • Aortic Stenosis • HOCM • Pulmonary Hypertensio • PE • ARVC	5 • Psychogenic • Metabolic e.g. hyper-ventilation • Neurological
24%	11%	14%	4%	12%

Unknown Cause = 34%

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Section III:

Diagnosis and Evaluation Options

Initial Evaluation
(Primary Care/Clinic/Emergency Dept.)

- Detailed history
- Examination
- Investigations

Syncope
Evaluation and Differential Diagnosis

History – What to Look for

- Complete Description
  - From patient and observers, mobile phones-videos
- Associated/Prodromal Symptoms
- Onset
- Posture
- Duration of Attacks
- Sequelae

Neurally-mediated syncope

- Absence of cardiac disease
- Long history of syncope usually occurring before 40 yrs.
- After sudden unexpected unpleasant sight, sound, smell or pain
- Prolonged standing or crowded, hot places
- Nausea, vomiting associated with syncope

Tunnel vision, tinnitus, yawning

- During or in the absorptive state after a meal
- With head rotation, pressure on carotid sinus (as in tumours, shaving, tight collars)
- After exertion

During a meal

3 P's : Provocation, Prodromal, Postural

Thank you very much for referring this pleasant 41-year-old gentleman for a cardiology opinion. Approximately one week ago, he was at a work event and had approximately five pints of beer in the evening. This is more than his normal intake, which amounts to about five pints in a week. The following day, he had a big breakfast as he was staying in a hotel and ate more than he usually does. He was at a meeting and whilst sitting, presenting, started to feel unwell with blurred vision, lightheaded and sweating. He later stood up, felt unsteady on his feet and lost consciousness. He quickly recovered but started to feel ill again with sweating. On arrival of the paramedics, it was commented that his blood pressure was low and his ECG showed a ventricular rate of 75 beats per minute (I have reviewed the ECG). After laying on the floor for a while and drinking a lot of fluids, he started to feel better. He felt tired for the rest of the day and by the following day he was back to normal. There has been no previous history of syncope or tendency to faint in the past.

He suffers with mild asthma.

His current medication consists of a Ventolin inhaler.

Syncope caused by orthostatic hypotension and other syndromes of orthostatic intolerance

- After standing up
- Temporal relationship with start of medication leading to hypotension or changes of dosage
- Prolonged standing especially in crowded, hot places
- Presence of autonomic neuropathy – Diabetes Mellitus or parkinsonism
- After exertion

[redacted] made an appointment for a cardiology review as he was concerned with his overall cardiovascular health. He is short of breath on exertion which can be associated with feeling light-headed. He also gets short of breath if he stands up quickly. There is no associated chest pain, orthopnoea or paroxysmal nocturnal dyspnoea. Neil has a sedentary lifestyle with little in the way of exercise.

He was diagnosed with having Type II diabetes mellitus eight years ago and was initially commenced on Metformin and Gliclazide. Approximtely sixteen months ago he had an ulcer on his left foot which was slow to heal and his podiatrist noticed a BM of 32 mmol/L. He was taken immediately to Watford Hospital for further diabetic management and was commenced on insulin. His glucose must have been high for some time as he has now developed diabetic retinopathy with decreased vision in his left eye, erectile dysfunction and diabetic neuropathy causing paresthesia in his feet. He also has irregular bowel movements, feels bloated after eating, has difficulty swallowing and suffers with indigestion. His collection of symptoms almost certainly reflects an autonomic neuropathy.

His past medical history includes an appendectomy thirteen years ago.

His current medication consists of Novorapid and a slow acting insulin. He was taking Cialis which he stopped as it was ineffective.

His father is alive and suffers with Type II diabetes mellitus.

Mr. Leggett is married and has an 18 year old daughter and a 16 year old son. He is an ex smoker since the age of 23 years having smoked for only 5 years. He works in recruiting. He drinks alcohol occasionally.

On examination: pulse 62 beats per minute, regular. JVP not elevated. Lying blood pressure 150/94mmHg., standing at one minute 96/70 mm. Hg. He had no symptoms of dizziness during his postural drop. Heart sounds S1 plus S2. His chest and abdomen were unremarkable with no abdominal bruits. The posterior tibial and dorsalis pedis arteries were bilaterally palpable. There were no carotid bruits.

49 year old male

Cardiac syncope

- Presence of severe structural heart disease
- During exertion, or supine
- Preceded by palpitation or accompanied by chest pain
- Family history of sudden death

Abnormal ECG

Epilepsy versus syncope

	Epileptic seizure	Neurocardiogenic syncope
Symptoms pre event	Aura (déjà vu, jamais vu), chewing, lip smacking, abnormal stereotypical behaviour	Situational, nausea, vomiting, abdominal discomfort, yawning, dizziness, sweating, blurred vision. Improvement lying down
Findings during LOC	Tonic-clonic movement, 1-2min., rhythmic, hemilateral clonic movements	Myoclonic jerks~80%, <15-30 sec.,
	Blue	Pallor
Tongue biting	Common (side)	Uncommon/rare (tip)
Incontinence	Common	Common
Symptoms after the event	Prolonged confusion > 10min., aching muscles	Short duration (<30sec), nausea and vomiting

Re: [redacted] 03/08/1968

Diagnoses:

1. Large left MCA infarct July 2014
2. Hypertrophic cardiomyopathy (asymmetrical septal hypertrophy IVSD 1.7 cm)
3. MRI scan 23rd October 2015 showed marked asymmetrical septal hypertrophy (23 mm), mild LVOT obstruction and SAM of the mitral valve at rest. Extensive fibrosis in the hypertrophied septum and anterior wall with near transmural and circumferential extension to the mid and apical segments.
4. Coronary angiogram June 2015 showed unobstructed coronary arteries
5. Post-CVA seizures

I reviewed this gentleman today in clinic. Since he was last seen in April 2016 he has had two seizures. His wife found him leaning to the left with rhythmical contractions of the arm and legs. He then falls asleep for half an hour and on recovery he is a little confused. His seizures are associated with tongue biting and urinary incontinence.

There is no separate history of syncope or palpitations. He gets short of breath on walking 100 yards. Furosemide made no difference to his symptoms which he discontinued as it only increased his urinary frequency.

His current medication consists of Lipitor 40 mg daily, Warfarin 5 mg daily, Bisoprolol 2.5 mg daily, Episenta 20 mg bd and Ramipril 1.25 mg daily.

RED FLAGS Symptoms and Signs

Syncope with:

- No warning
- With Exercise/ exertion.
- Palpitations

(sequence of events v. important i.e. if palpitations & THEN dizziness = more likely cardiac)

- Chest pain/ SOB
- Being supine
- Cardiac hx
- Signs of heart failure
- Abnormal ECG
- Prolonged LOC, post recovery confusion for longer than a minute or so.
- FH of sudden death even neonatal deaths, Cot deaths, drowning
- New onset or severe headaches
- Frequent recurrence, severe injury or driving involvement e.g. PSV,HGV drivers.

Examination

- Cardiovascular
  - Pulse
  - Blood Pressure – supine and upright (1, 3 min.)
  - Heart murmurs
  - Signs of Heart Failure
  - Carotid sinus massage (>60 years)

Abnormal BP fall is defined as a progressive and sustained fall in systolic BP from baseline value >_20 mmHg or diastolic BP >_10 mmHg, or a decrease in systolic BP to < 90 mmHg.

Carotid Sinus Massage

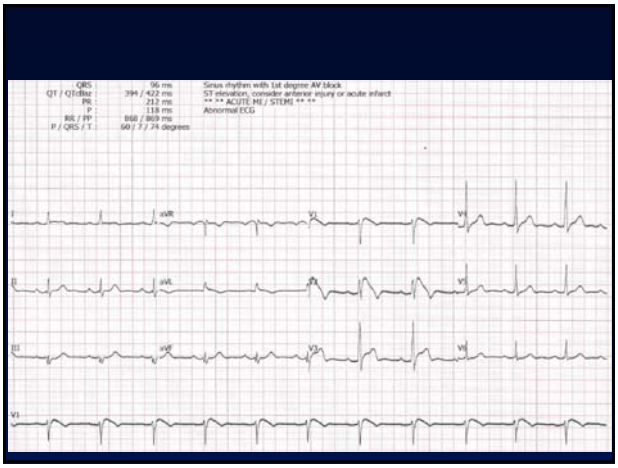
- Outcome:
  - 3 sec asystole and/or 50 mmHg fall in systolic blood pressure with reproduction of symptoms =

  Carotid Sinus Syndrome (CSS)
- Contraindications
  - Carotid bruit, known significant carotid arterial disease, previous CVA, MI last 3 months
- Risks
  - 1 in 5000 massages complicated by TIA

Investigations

12-Lead ECG

- Normal or Abnormal?
  - MI (Q waves, ST-T wave abnormalities)
  - Severe Sinus Bradycardia/pauses
  - Bundle Branch block, Axis deviation, AV Block
  - Preexcitation (WPW), Long QT, Brugada
  - Tachyarrhythmia (SVT, VT)
- Short sampling window (approx. 12 sec)

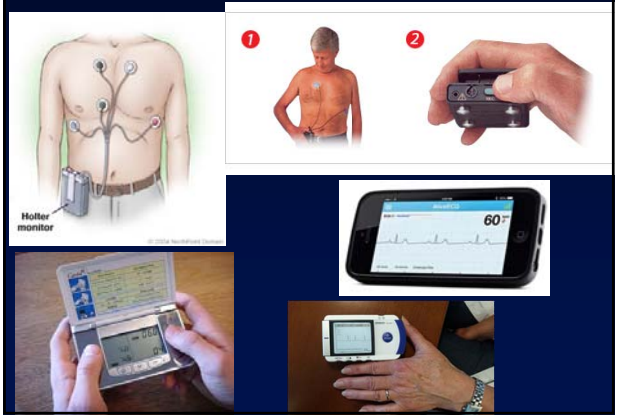


The Beginning: 1949 HOLTER Analysis

Montana physician
Dr Norman Jeff Holter

Rocky Mountain Med J
1949; 747-751

Holter monitor VS Event monitor



Conventional Diagnostic Methods/Yield

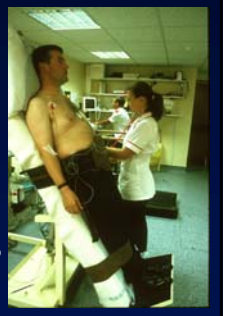
Test/Procedure	Yield (based on mean time to diagnosis of 5.1 months[7])
History and Physical (including carotid sinus massage)	49-85% [1,2]
ECG	2-11% [2]
Echocardiography	
Electrophysiology Study without SHD*	11% [3]
Electrophysiology Study with SHD	49% [3]
Tilt Table Test (without SHD)	11-87% [4,5]
Ambulatory ECG Monitors:	
• Holter	1-2% [7]
• External Loop Recorder (2-3 weeks duration)	20% [7]
• Insertable Loop Recorder (up to 14 months duration)	65-88% [6,7]
Neurological † (Head CT Scan, Carotid Doppler)	0-4% [4,5,8,9,10]

[1] Kapoor, et al *N Eng J Med*, 1983.
[2] Kapoor, *Am J Med*, 1991.
[3] Linzer, et al. *Ann Int. Med*, 1997.
[4] Kapoor, *Medicine*, 1990.
[5] Kapoor, JAMA, 1992
[6] Krahn, *Circulation*, 1995
[7] Krahn, *Cardiology Clinics*, 1997.
[8] Eagle K., et al. The Yale J Biol and Medicine. 1983; 56: 1-8.
[9] Day S, et al. Am J Med. 1982; 73: 15-23.
[10] Stetson P, et al. PACE. 1999; 22 (part II): 782.

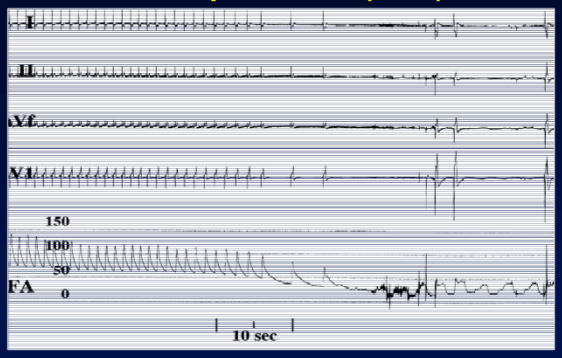
* Structural Heart Disease
† MRI not studied

TILT TABLE TEST

- PREPARATION:
  - 2 hour fast,
  - no drugs for 5 half-lives before
- TEST:
  - Rest quietly for 20-40 mins
  - Head-up tilt 60-80 degrees
  - Duration 45 mins
- Continuous monitoring HR / BP
- Resuscitation equipment / staff



Head-Up Tilt Test (HUT)



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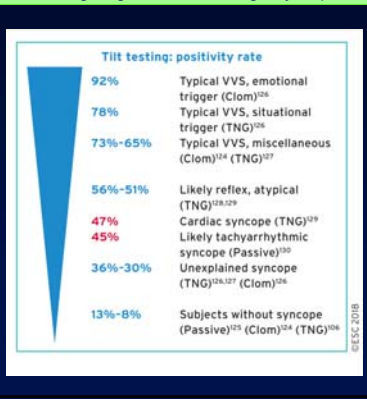
Tilt testing should now be considered a means of exposing a hypotensive tendency rather than being diagnostic of Vasovagal syncope



ESC guidelines 2018

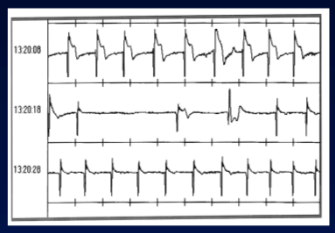
Implantable Loop Recorder

Patient Activator

Reveal® Plus ILR

9790 Programmer

83 yo woman
Bradycardia: Pacemaker implanted



Reveal ® ILR recordings; Medtronic data on file.

Conventional EP Testing in Syncope

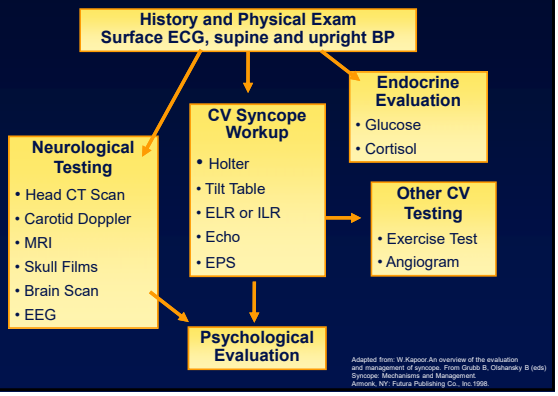
- Limited utility in syncope evaluation
- Most useful in patients with structural heart disease
  - Heart disease……..50-80%
  - No Heart disease…11-50%
- Relatively ineffective for assessing bradyarrhythmias

Brignole M, Alboni P, Benditt DG, et al. Eur Heart Journal 2001; 22: 1256-1306.

Electroencephalogram

- Not a first line of testing
- Syncope from Seizures
- Abnormal in the interval between two attacks – Epilepsy
- Normal – Syncope

Unexplained Syncope Diagnosis



Adapted from: W Kapoor An overview of the evaluation and management of syncope. From Grubb B, Olshansky B (eds) Syncope: Mechanisms and Management. Armonk, NY: Futura Publishing Co., Inc 1998.

Section IV:

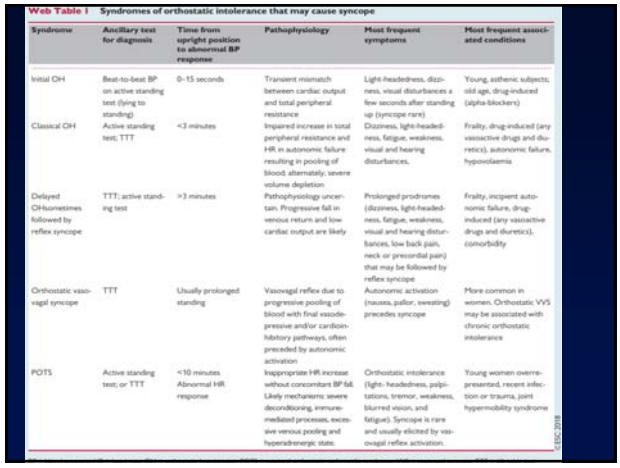
Specific Conditions and Treatment Options

Principal Causes of Orthostatic Syncope

- Drug-induced (very common)
  - Diuretics
  - Beta blockers
  - Antihypertensives
  - Tamsulosin/Indoramin

  Elderly - polypharmacy
- Secondary autonomic failure
  - Diabetes
  - Alcohol
  - Amyloid
  - Spinal cord injuries
- Alcohol
  - Orthostatic intolerance apart from neuropathy
- Primary autonomic failure
  - Multiple system atrophy
  - Parkinsonism

Fall in SBP >20 mm Hg or fall in DBP>10 mm Hg within 3 minutes of standing



Web Table 1 Syndromes of orthostatic intolerance that may cause syncope

Syndrome	Ancillary test for diagnosis	Time from upright position to abnormal BP response	Pathophysiology	Most frequent symptoms	Most frequent associated conditions
Initial OH	Beat-to-beat BP on active standing test (lying to standing)	0–15 seconds	Transient mismatch between cardiac output and total peripheral resistance	Light-headedness, dizziness, visual disturbances a few seconds after standing up (syncope rare)	Young, asthenic subjects; old age, drug-induced (alpha-blockers)
Classical OH	Active standing test; TTT	<3 minutes	Impaired increase in total peripheral resistance and HR in autonomic failure resulting in pooling of blood, alternately, severe volume depletion	Dizziness, light-headedness, fatigue, weakness, visual and hearing disturbances.	Frailty, drug-induced (any vasoactive drugs and diuretics), autonomic failure, hypovolaemia
Delayed OH sometimes followed by reflex syncope	TTT; active standing test	>3 minutes	Pathophysiology uncertain. Progressive fall in venous return and low cardiac output are likely	Prolonged prodromes (dizziness, light-headedness, fatigue, weakness, visual and hearing disturbances, low back pain, neck or precordial pain) that may be followed by reflex syncope	Frailty, incipient autonomic failure, drug-induced (any vasoactive drugs and diuretics), comorbidity
Orthostatic vasovagal syncope	TTT	Usually prolonged standing	Vasovagal reflex due to progressive pooling of blood with final vasodepressive and/or cardioinhibitory pathways, often preceded by autonomic activation	Autonomic activation (nausea, pallor, sweating) precedes syncope	More common in women. Orthostatic VVS may be associated with chronic orthostatic intolerance
POTS	Active standing test; or TTT	<10 minutes Abnormal HR response	Inappropriate HR increase without concomitant BP fall. Likely mechanisms: severe deconditioning, immune-mediated processes, excessive venous pooling and hyperadrenergic state.	Orthostatic intolerance (light- headedness, palpitations, tremor, weakness, blurred vision, and fatigue). Syncope is rare and usually elicited by vasovagal reflex activation.	Young women overrepresented, recent infection or trauma, joint hypermobility syndrome

Postural Orthostatic cardiac syndrome (POTS)

- Dysautonomia
- Orthostatic intolerance, young females (12-50 yrs).
- Increase in heart rate by 30 beats/min (>40 bpm, 12-19 years), from baseline or >120/min within 10 minutes from lying to standing. No fall in BP
- Symptoms: headache, fatigue, nausea, weakness, sweating, anxiety, palpitations, dizziness, vertigo, presyncope, tremulous, dyspnoea/hyperventilation, sleep disorder
- Labelled "neurosis" or "panic attacks"

Associated with deconditioning, recent infections, chronic fatigue syndrome, joint hypermobility syndrome, and a spectrum of non-specific symptoms such as headache and chest pain.

Pathophysiology: heterogeneous, ? deconditioning, immune-mediated processes, excessive venous pooling, and a

Neurally-Mediated Reflex Syncope (NMS)

- Vasovagal syncope (VVS)
- Carotid sinus syndrome (CSS)
- Situational syncope
  - post-micturition
  - cough
  - swallow
  - defecation
  - blood drawing
  - post prandial
  - etc.

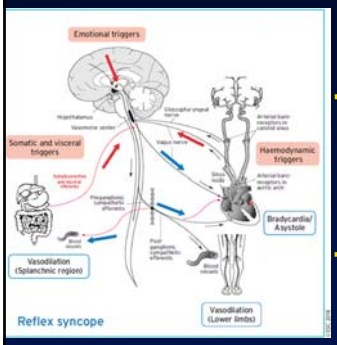
Prevalence of VVS

In general:

- VVS patients younger than CSS patients
- Ages range from adolescence to elderly (median 43 years)
- Pallor, nausea, sweating, palpitations are common
- Amnesia for warning symptoms in older patients

The older you are:
- Consider other causes besides is neurally mediated reflex syncope
- The less likely the cause is benign

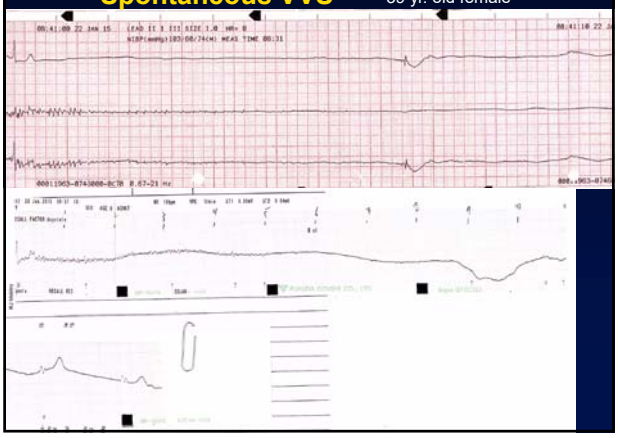
Vasovagal Syncope (VVS): Clinical Pathophysiology



- Neurally Mediated Physiologic Reflex Mechanism with two Components:
  - ↓ Cardioinhibitory (HR) — Variable contribution
  - ↓ Vasodepressor (BP) — Variable contribution
- Both components are usually present

Spontaneous VVS

39 yr. old female



DOB 29/01/1975
erts SG13 7JU

Diagnoses:
1. Neurocardiogenic syncope with a significant cardioinhibitory response with asystole up to at least 11 seconds
2. Asthma
3. Mother has idiopathic pulmonary fibrosis

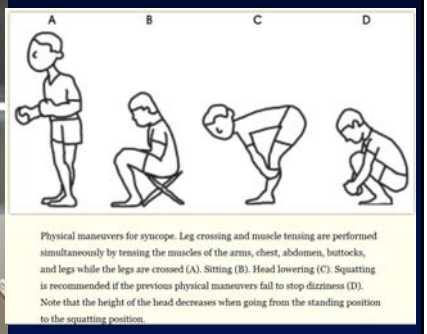
I reviewed this lady today in clinic. As you know she was admitted back in January 2015 with presyncopal and syncopal episodes. At that time she was under a lot of stress at her work. On the evening before her admission, she ate some olives which had been opened for approximately a week. In the early hours of morning she awoke feeling unwell with diarrhoea and vomiting. She then had a syncopal episode in the toilet sustaining a laceration to her scalp which was subsequently glued. On recovery from the syncopal episode she remained unwell and lightheaded and called her father who then called the ambulance. Whilst in the ambulance she continued to feel nauseated and weak and I understand there was an attempt to insert a Venflon during which she had another syncopal episode with an asystolic pause of at least 11 seconds (the ECG tracing was cut at this point and therefore I was unable to determine the full duration). Whilst being monitored in hospital she felt unwell sitting in the chair and again she had another syncopal episode with a similar asystolic pause. She was commenced on IV hydration and remained well. She has a past history of fainting in the past and some of these episodes included whilst being on a plane, following immunisations before travelling and another episode when she was sick associated with vomiting in the past. Following IV hydration she improved and was subsequently discharged. Her echocardiogram was normal. There is no family history of sudden unexpected death.

Since discharge she has remained well and I am glad to hear that she has not had any further syncopal episodes. I have arranged for her to have a cortisol level for completeness. I will review her again in three months' time and if all is well I think we can discharge her from clinic.

Management Strategies for VVS

- Optimal management strategies for VVS are a source of debate
  - Patient education, reassurance, instruction
  - Fluids, salt, diet
  - Tilt Training
  - Support stockings
  - Counter-pressure maneuvers
- Drug therapies
- Pacing
  - Class II indication for VVS patients with positive HUT and cardioinhibitory or mixed reflex

Counter Pressure Manouvres



Physical maneuvers for syncope. Leg crossing and muscle tensing are performed simultaneously by tensing the muscles of the arms, chest, abdomen, buttocks, and legs while the legs are crossed (A). Sitting (B). Head lowering (C). Squatting is recommended if the previous physical maneuvers fail to stop dizziness (D). Note that the height of the head decreases when going from the standing position to the squatting position.

VVS: Pharmacologic Rx

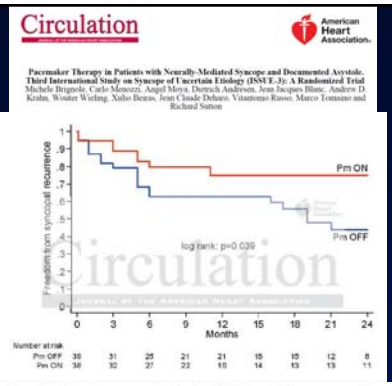
- Salt /Volume
  - Salt tablets, 'sport' drinks, fludrocortisone
- Beta-adrenergic blockers
  - 1 positive controlled trial (atenolol),
  - 1 on-going RCT (POST)
- SSRIs
  - 1 controlled trial
- Vasoconstrictors (e.g., midodrine)
  - 1 negative controlled trial (etilephrine)

VVS: Tilt-Training

- Objectives
  - Enhance Orthostatic Tolerance
  - Diminish Excessive Autonomic Reflex Activity
  - Reduce Syncope Susceptibility / Recurrences
- Technique
  - Prescribed Periods of Upright Posture
  - Progressive Increased Duration

Status of Pacing in VVS

- Reserved for patients who have failed medical therapy
- Most useful in cardio-inhibitory syncope (>3 sec. with sycope or asymptomatic and >6 sec.)
- Dual chamber pacing
- Sophisticated algorithims – rate drop response



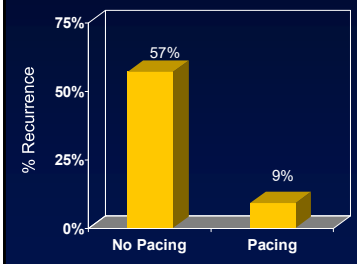
Conclusions - Dual-chamber permanent pacing is effective in reducing recurrence of syncope in patients ≥40 years with severe asystolic NMS. The observed 32% absolute and 57% relative reduction in syncope recurrence support this invasive treatment for the relatively benign NMS.

M Brignole et al. Circulation. 2012 May 29;125(21):2566-71

Carotid Sinus Syndrome (CSS)

- CSS may be an important cause of unexplained syncope / falls in older individuals

Role of Pacing in CSS -- Syncope Recurrence Rate



Class I indication for pacing (AHA and BPEG)
Limit pacing to CSS that is:
- •Cardioinhibitory
- •Mixed

DDD/DDI superior to VVI

(Mean follow-up = 6 months)

Brignole et. Al. Diagnosis, natural history and treatment. Eur JCPE. 1992; 4:247-254

Syncope Due to Arrhythmia or Structural CV Disease

Principal Causes of Syncope due to Structural Cardiovascular Disease

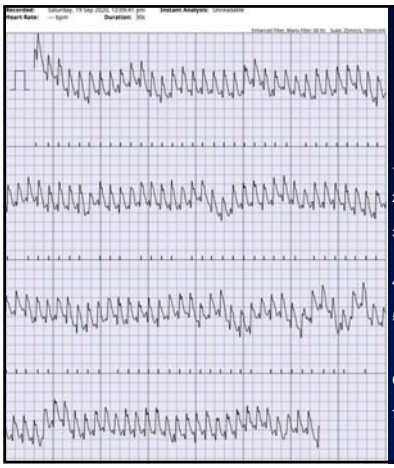
- LV systolic dysfunction
- Acute MI / Ischemia
  - Acquired coronary artery disease
  - Congenital coronary artery anomalies
- HOCM
- Acute aortic dissection
- Pericardial disease / tamponade
- Pulmonary embolus / pulmonary hypertension
- Valvular abnormalities
  - Aortic stenosis, Atrial myxoma

Syncope Due to Cardiac Arrhythmias

- Bradyarrhythmias
  - Sinus arrest, exit block
  - High grade or acute complete AV block
- Tachyarrhythmias
  - Atrial fibrillation / flutter with rapid ventricular rate (e.g. WPW syndrome)
  - Paroxysmal SVT or VT
  - Torsades de pointes

Drug-Induced QT Prolongation

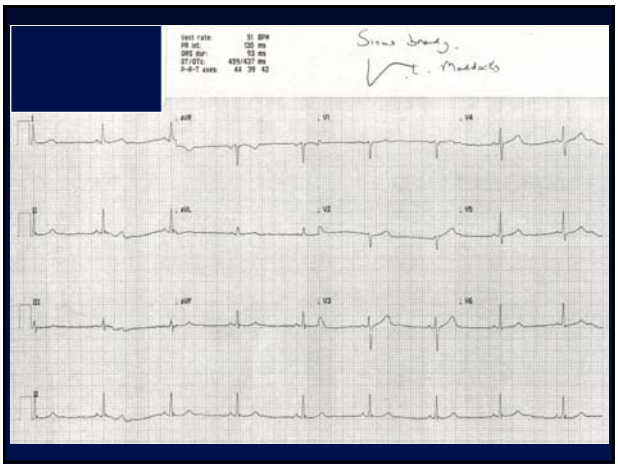
- Antiarrhythmics
  - *Class IA* ...Quinidine, Procainamide, Disopyramide
  - *Class III*...Sotalol, Ibutilide, Dofetilide, Amiodarone, (NAPA)
- Psychoactive Agents
  - Phenothiazines, Amitriptyline, Imipramine, Ziprasidone
- Antifungal Agents
  - Fluconazole, cotrimoxazole, itraconazole), ketoconazole
- Antibiotics
  - Erythromycin, Pentamidine,
- Nonsedating antihistamines
  - (Terfenadine), Astemizole, azelastine, diphenhydramine, ebastine*, hydroxyzine,
- Others
  - (Cisapride), Droperidol (http://www.sads.org.uk/drugs_to_avoid.htm)



44 yr. old male

On flecainide 50 mg and bisoprolol 2.5 mg – pill in pocket

1. Paroxysmal atrial fibrillation
2. Structurally normal heart on echo
3. Possible pericarditis/myocarditis and pneumonia aged 19
4. Hay fever
5. Normal CT coronary angiogram, with a calcium score of 0 and no coronary artery disease (2014)
6. Gout
7. Fractured left ankle, left hip and left shoulder – 2015.



Treatment of Syncope Due to Bradyarrhythmia

- Class I indication for pacing using dual-chamber system wherever adequate atrial rhythm is available
- Ventricular pacing in atrial fibrillation with slow ventricular response

Treatment of Syncope Due to Tachyarrhythmia

- Atrial Tachyarrhythmias;
  - AVRT due to accessory pathway – ablate pathway
  - AVNRT – ablate AV nodal slow pathway
  - Atrial fib– Pharmacotherapy, pacing, ablation
  - Atrial flutter – Ablation of reentrant circuit
- Ventricular Tachyarrhythmias;
  - Ventricular tachycardia – ICD or ablation where appropriate
  - Torsades de Pointes – withdraw offending Rx or ICD (long-QT/Brugada)
- Drug therapy may be an alternative in many cases

NHS

Nati
Health and C

Quick reference guide

Issue date: August 2010 Update

Syncope associated with:

- No warning & assoc. with trauma
- Occurring whilst supine
- Cardiac History (IHD/Cardiomyopathy/ Congenital Heart Disease)

Refer within 24 hours for ***specialist cardiovascular*** assessment by the most appropriate local service, anyone with TLoC who also has any of the following:

- An **ECG** abnormality
- **Heart failure** (history or physical signs).
- TLoC during **exertion.**
- Family history of **sudden cardiac death** in people aged younger than 40 years and/or an **inherited cardiac** condition.
- New or unexplained breathlessness.
- A heart **murmur**.

DVLA
Driver and Vehicle Licensing Agency

At a Glance Guide for Medical Practioners

Feb. 2020


Transient loss of consciousness – solitary episode

	Group 1 car and motorcycle	Group 2 bus and lorry
Typical vasovagal syncope		
While standing	May drive and need not notify the DVLA.	Must not drive and must notify the DVLA.
While sitting	May drive and need not notify the DVLA if there is an avoidable trigger which will not occur whilst driving. Otherwise must not drive until annual risk of recurrence is assessed as below 20%.	Must not drive for 3 months and must notify the DVLA. Will require investigation for identifiable and/or treatable cause.
Syncope with avoidable trigger or otherwise reversible cause (for cough syncope see page 26)		
While standing	May drive and need not notify the DVLA.	Must not drive and must notify the DVLA.
While sitting	Must not drive for 4 weeks. Driving may resume after 4 weeks only if the cause has been identified and treated. Must notify the DVLA if the cause has not been identified and treated.	Must not drive for 3 months. Driving may resume after 3 months only if the cause has been identified and treated. Must notify the DVLA if the cause has not been identified and treated.
Unexplained syncope, including syncope without reliable prodrome		
This diagnosis may apply only after appropriate neurological and/or cardiological opinion and investigations have detected no abnormality.		
While standing or sitting	Must not drive and must notify the DVLA. If no cause has been identified, the licence will be refused or revoked for 6 months.	Must not drive and must notify the DVLA. If no cause has been identified, the licence will be refused or revoked for 12 months.
Cardiovascular, excluding typical syncope		
While standing or sitting	Must not drive and must notify the DVLA. Driving may be allowed to resume after 4 weeks if the cause has been identified and treated. If no cause has been identified, the licence will be refused or revoked for 6 months.	Must not drive and must notify the DVLA. Driving may be allowed to resume after 3 months if the cause has been identified and treated. If no cause has been identified, the licence will be refused or revoked for 12 months.

Cases

30.04.2014

This pleasant, young 32 year old lady from Quatar came to see me for a cardiology review with a history of syncope. She has a tendency to "faint" which started around seventeen years of age, usually around when blood is taken. She also gets dizzy in the morning when she gets out of bed and stands up quickly. She generally returns to bed and has to lie down for approximately ten minutes before she feels better. She had one episode whilst sitting, when she did not eat breakfast and suddenly felt light-headed, dizzy, sweaty and then lost consciousness for a few seconds. She quickly recovered.

She was complaining of palpitations which she describes as a big beat/missed beat that lasts for seconds. This occurs approximately once a week. She has never had any sustained rapid palpitations. She provided me with a dossier of her previous medical reports which she has had done in Qatar. I was able to find the reports from a cardiologist in 2011 around the time she was complaining of palpitations and dizziness. He felt she may have sick sinus node disease based on a 24 hour tape. I was able to review this 24 hour tape and this showed marked sinus arrhythmia with an appropriate diurnal variation of her heart rhythm. She has also had an echocardiogram in 2012 which was reported as normal and another 24 hour tape in May 2012 which was also normal. She had an exercise tolerance test in June 2012 during which she had a normal chronotropic and blood pressure response. She clearly does not have sick sinus node disease. She has always had a tendency to low blood pressure.

Her past medical history incudes an appendicectomy. She is on no regular medication.

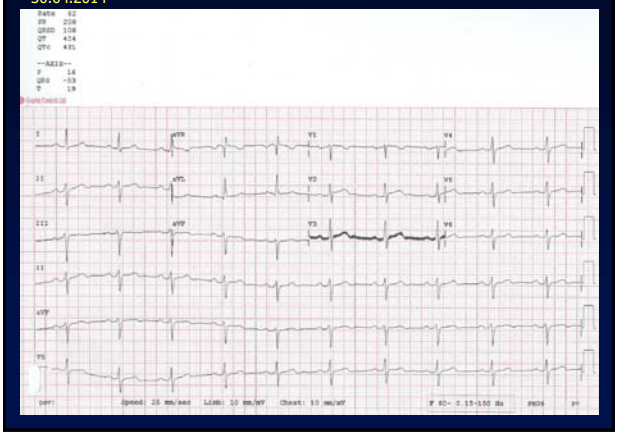
30.04.2014

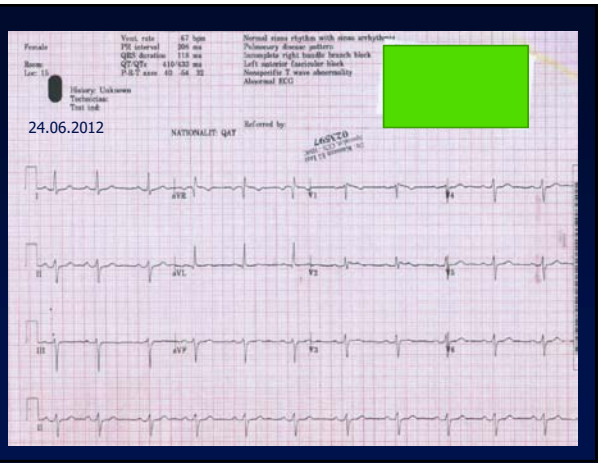
Her father died at the age of 72 with stomach cancer and her mother is alive at the age of 62 and suffers with diabetes mellitus. She has one brother aged 38 who is diagnosed as having epilepsy. He also suffers with "fainting episoses" and interestingly also has nocturnal seizures. I understand his seizures usually occur when he is sick, upset, afraid or nervous. This happens twice a year. [redacted] does not smoke and works as a secretary.

On examination pulse 62 beats per minute, regular, JVP not elevated. Lying blood pressure 107/64 mm Hg., standing at one minute 112/69 mm. Hg., pulse rate 67 beats per minute. Standing at three minutes 109/68 mm. Hg., pulse rate 70 beats per minute. Heart sounds S1 plus S2 plus a soft mild (1/6) systolic murmur at the left sternal edge and apex. Her chest and abdomen were unremarkable. Her ECG today showed sinus rhythm with left axis deviation and normal QRS morphology and conduction indices. In her medical dossier, I was able to find an ECG dated 24th June, 2012 which showed a secondary R wave in lead V1 and 2 mm J point elevation in lead V2 with a biphasic T wave. There was also an ECG from the 1st May, 2012 which was similar. I therefore repeated her ECG with leads V2 and V3 in the second intercostal space and this did not show any major difference. She had some blood tests done in June 2013 which showed normal Us and Es, full blood count, glucose, liver function tests. Her cholesterol was 6.14 mmol/L with a triglyceride of 1.2 mmol/L. I could not see a thyroid function test.

The majority of this lady's symptoms are related to her low blood pressure and most likely neurocardiogenic (vasovagal) syncope. I have asked her to increase her salt and fluid intake and I have also taught her counter pressure manoeuvres to perform when she feels light-headed or dizzy. I was quite intrigued given her ECG and her brother's history and I have asked her to send me a copy of her brother's ECG if possible. She is due to return to Qatar in due course. I would like to review her again when she next visits the United Kingdom and we can always consider performing a 24 hour tape, a repeat echocardiogram and possibly a tilt test. I have also asked her to ensure that a thyroid function test has been checked in the past for completeness. She will need to have a repeat fasting lipid profile at some point in the future and in the first instance I have recommended she makes some lifestyle changes by altering her diet.

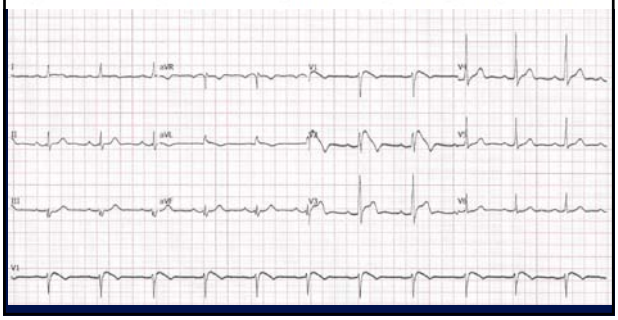
30.04.2014





Brother's ECG - Brugada syndrome

PS She was able to send me a copy of her brother's ECG. This is indeed very interesting as it showed a 5 mm. J point elevation with cove shape ST elevation and T wave inversion in lead V2. Based on this ECG pattern I think Brugada syndrome needs to be excluded and I have informed both [redacted] and her sister who accompanied her today that I would like to see her brother as soon as possible or alternatively for him to see a reputable Cardiologist/Electrophysiologist in Qatar.



I reviewed this pleasant 39-year old gentleman today for a cardiology opinion. I met his sister [redacted] in April 2014, when she was reviewed with symptoms consistent with neurocardiogenic syncope. Fatima's ECG showed a secondary R wave in lead V1 and 2 mm J point elevation in lead V2 with a biphasic T wave. She informed me about her brother [redacted] who also had recurrent syncopal episodes and I asked her to send me a copy of his ECG. This showed a type 1 Brugada pattern and I suggested that he was reviewed by a cardiologist in Qatar.

[redacted] has a history of recurrent syncope usually precipitated by blood letting, emotional stress, during exam times, diarrhea illness and pain. His first episode occurred around nine years old after jumping and hurting his pelvic bone. He knows when he is going to have an episode as he feels dizzy, weak, diminutive hearing, vision goes fuzzy with a black cloud and he then loses consciousness for approximately 1-2 minutes. He can abort a syncopal episode if he lays flat. On regaining consciousness, he feels exhausted and "not right" for up to three hours. He has two-three episodes a year. He admits that his fluid intake is poor.

At age 28 years, he had one episode when he awoke at night feeling exhausted analogous to his symptoms when he has a syncopal episode.

23/12/15

There was one episode witnessed by his mother at age 29 years when he had a syncopal episode in the evening associated with "going stiff", "shaking" and tongue biting.

In 2005 (age 29 years) whilst studying in Manchester, he was investigated by a neurologist with a cerebral MRI scan and an EEG with visual stimulation, which were both unremarkable.

He has had pyrexial illnesses without a worsening of any syncopal episodes.

He has been investigated in Qatar by a few cardiologists. His echocardiogram has been reported as normal. He underwent an exercise tolerance test on the 4th August 2015 using the BRUCE protocol. He exercised for 13:02 minutes achieving 94% of his maximum predicted heart rate and a workload of 15.2 METS. There was an appropriate BP and chronic response. There were no arrhythmias. Interestingly, the J point elevation improved at peak exercise. A 24-hour ECG analysis (4th August 2015) was unremarkable with a minimum heart rate of 42 beats/minute, maximum 121 beats/minute and a mean of 74 betas/minute. He had a tilt test (13th August 2015), which was positive at 22 minutes after GTN provocation with a period of unrecordable BP and asystole. Apparently, he was given CPR during the asystolic period. Full blood count, renal function, liver function, thyroid function, calcium, glucose and cholesterol were all nremarkable.

He is on no current medication, although he was recommended fludrocortisone, which he stopped after one dose.

His father died of stomach cancer and his mother is alive and suffers with Diabetes Mellitus. Apart from one sister (Fatima) with syncope, there is no history of sudden unexpected death.

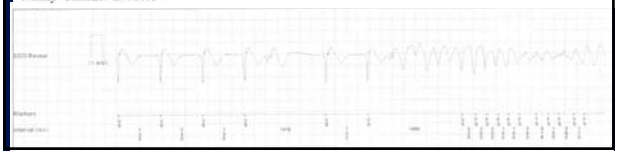
He is married, has one daughter and is expecting another child. He works as a drilling engineer.

Examination: weight 48kg, height 1.63m. Pulse 69 beats/minute, regular. Lying BP 120/80 mm Hg, standing at 1 minute 110/80 mm Hg, and standing at 3 minutes 110/80 mm Hg. Heart sounds S1+S2. His chest was clear.

4th August 2016

To: aghuran2@yahoo.co.uk

Hey Doctor. It was Thursday around 01:30 am I was sleep on bed and all of a sudden opened my eyes and I knew it's coming. I was still and I did not move a finger, all I did is praying for it to go away but it hit me so fast. During the episode I was telling my self to wake up but it was like I want to wake up then I die then I wake up then I die was like this for duno maybe 10 or more times. I opened my eyes later on vomiting and kicking and swinging my Arms around. It was really trying. I meet three Docs here and all were surprised how I survive episodes like these. I appreciate your following and support. I will advice you with any new things
Many Thanks Doctor



Dear [redacted]

78 years

Re: [redacted] Dob 23.12.1934

Diagnoses:

1. ***Mild aortic valve disease. Peak gradient 26 mm. Hg with good LV systolic function, no other significant valvular abnormalities***
2. ***Lumbar spinal stenosis treated with decompression surgery***
3. ***Hypertension***
4. ***Asthma***
5. ***Rhinitis***
6. ***Degenerative right hip disease***

Thank you very much for asking me to review this gentleman in view of his recent falls and for monitoring his aortic valve disease. I understand he is being considered for right hip replacement surgery sometime in October.

From a cardiac point of view there is no history of chest pain or increasing shortness of breath on exertion or palpitations. At the beginning of August he climbed up three stairs with some gardening tools and he felt himself falling backwards when he hit his head and sustained a laceration to his scalp. He was not sure if he was dizzy and he did not believe he lost consciousness. He apparently quickly recovered and his lacerations were treated at the Princess Alexandra Hospital where it was glued.

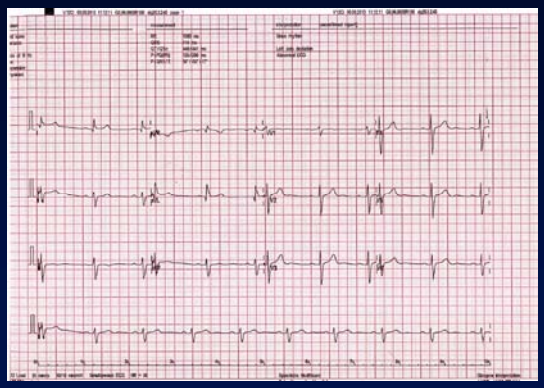
Three days ago whilst at a public meeting he was sitting down in a warm, stuffy environment. He stood up to give a small speech. Following his speech he turned around and the next thing he knew he started to fall. Again he believed he did not lose consciousness and there were no associated palpitations or feeling of dizziness prior to this episode. Recently, he has been experiencing intermittent dizziness up to three times a week, particularly if he looks up and down. He has had no symptoms whilst driving or sitting.

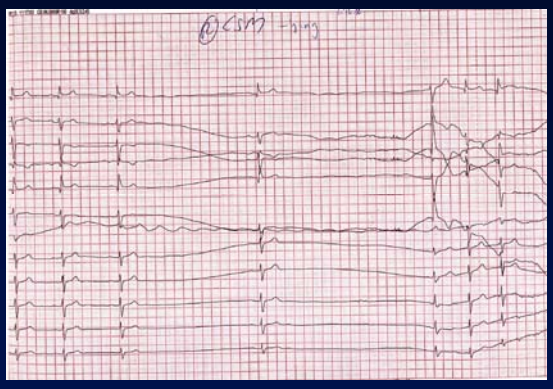
His current medication consists of Felodipine 10 mgs. daily, Bendroflumethiazide 2.5 mgs. daily, Lisinopril 10 mgs. daily, Gabapentin 600 mgs. t.d.s. and Co-Codamol.

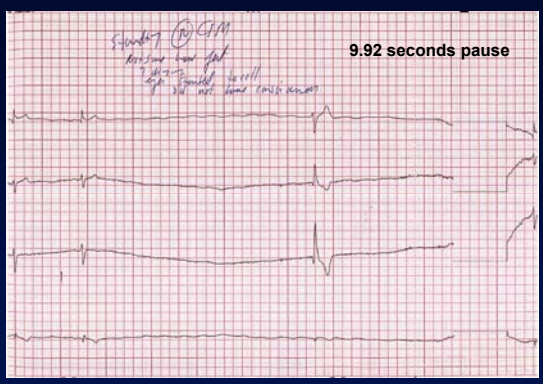
On examination pulse 66 beats per minute, regular with a normal character. JVP not elevated. Heart sounds S1 plus an audible S2 plus a 3/6 ejection systolic murmur that radiated to the base of his neck. His chest was clear. Lying blood pressure 120/80 mm. Hg., standing at one minute 110/80 mm. HG., standing at three minutes 120/80 mm. Hg. His 12 lead ECG showed sinus rhythm with first degree AV block (PR interval 204 msecs.), with left axis deviation and decreased R wave progression across the pre-cordial leads most likely due to counter clockwise rotation of the heart about the longitudinal axis. Right sided carotid massage whilst lying down showed a 3.76 second pause during which he was not symptomatic. I then stood him up and repeated right carotid massage and he had a 9.92 second pause with one ventricular escape beat that occurred at five seconds into the pause. During this time his eyes started to roll back. He was not communicative. However, he did not fully lose consciousness. I quickly asked him to lay down and interestingly he could not remember the event. This may explain why with his previous two episodes he could not recall exactly what happened.

His echocardiogram showed normal LV cavity size with isolated basal septal hypertrophy causing a sigmoid shaped septum with normal wall thickness in the other regions. He has good LV systolic function. The aortic valve was tricuspid with calcified edges and a peak gradient of 26 mm. Hg. across it. The RVSP was 23 mm. Hg and his IVC was normal in size with normal inspiratory collapse. He has normal pulmonary artery pressure. The aortic root was mildly dilated measuring 4.1 cms. at the sinuses.

This gentleman has carotid hypersensitivity syndrome and there is no doubt he will require a dual chamber pacemaker. This will need to be done prior to his orthopaedic surgery. Further to our telephone conversation, I will arrange this as soon as possible. Thanks very much for your referral and should you have any queries please do not hesitate to contact me.







Case 2

Dear [redacted]

Re: [redacted] 59 yr. Female

Diagnoses:

1. Neurocardiogenic syncope. Tilt test in 2005 at St Mary's Hospital reported by Professor Sutton and stated that although it was technically negative in that syncope was not induced, there was classical oscillation of blood pressure strongly suggesting a vasovagal diagnosis. A repeat tilt test on 11th February 2010 at the Brompton and Harefield NHS Trust was negative despite sublingual GTN
2. Dual-chamber pacemaker December 2007 for bradycardia (20 beats per minute) and pauses demonstrated on a Reveal device
3. Previous migranous headaches and vertigo
4. Hiatus hernia
5. Mild asthma
6. Normal coronary angiogram and good LV systolic function January 2006
7. Functional diarrhoea
8. Reviewed by Dr Gibbs, Consultant Neurologist for recurrent syncope in 2007 who felt that an epileptic cause was very unlikely and more likely to be hypotensive related syncope

[redacted] came for a cardiology review today. I greeted her in the waiting room and as we were walking towards the clinic room while she was trailing behind me she suddenly collapsed to the floor. As I turned around she was lying on the floor, motionless with a palpable pulse with a reasonable volume. Her eyes were flickering and her colour remained normal. 20 seconds later she completely recovered without any sequelae or ongoing symptoms. By the time we got a blood pressure machine her blood pressure was 116/47 with a pulse of 68 beats per minute. She immediately got up and walked into the clinic room. We had our consultation as if nothing had happened.

I must confess given the sequence of events today, I was not convinced that there was any cardiac component to account for her syncopal episode and I wonder whether there may be an underlying emotional element. You may wish to explore this in further details.

Although she certainly does have a history of vasovagal syncope, I do not think the sequence of events today is likely to be cardiac in origin and for that reason I have not arranged any further follow-up appointments. Should you have any further queries or like to discuss her case in more detail, please do not hesitate to contact me.

Yours sincerely,

Dictated and verified by Doctor but not signed

Dr Azad Ghuran MB ChB, MRCP, MD
Consultant Cardiologist

Case 2A

Re: [redacted] **- (dob: 19/05/66)**
[redacted]

Thank you very much for referring this pleasant 52-year-old gentleman for a cardiology opinion. Approximately two weeks ago, in the evening, he was at a bar in London and drank two rum cocktails. He had not eaten for a few hours. At about 10:00pm he decided to eat and started to feel warm, sweaty, unwell and nauseated. He walked up some stairs to go outside and leaned against a wall. The next thing he knew he was on the ground having briefly lost consciousness. He sustained bruising to his left knee, a laceration to his forehead, and abrasions to the left side of his face. He was taken to St Mary's Hospital, where he had stitches for the laceration. He had some blood tests, an ECG and was reassured that there were no significant findings. There has been no history of any headaches, focal weakness, visual disturbances, tinnitus, nasal or ear discharges.

Yesterday, whilst driving, he felt lightheaded and queasy. He said this lasted approximately eight hours. He went to bed and by the following day he was back to normal. He also drinks up to two litres of water a day, particularly during the hot weather. There has been no previous history of syncope or pre-syncope. He is usually active, goes to the gym twice a week and walks regularly.

There is a strong family history of ischaemic heart disease and last year he had a number of cardiac investigations, which included blood tests, an echocardiogram, an exercise tolerance test, an ambulatory blood pressure monitor, and a cardiac CT scan. He was reassured that his investigations were all satisfactory. He was not commenced on a statin agent. His blood pressure tends to be borderline and his home blood pressure is around 140/92 mmHg.

His past medical history includes a mole resection with early cancerous changes from his abdomen, a scrotal cystectomy and renal calculi which was endoscopically removed.

He is on no regular medication.

Case 2A

His father is alive at 78 years, having had a myocardial infarction at 53 years and later coronary artery bypass surgery. His father also suffers with hypertension and had a carotid endarterectomy. His mother is alive at 74 years, having been treated for breast cancer. There is no family history of sudden cardiac death or syncope.

He lives with his wife and has one son, 11 years. He does not smoke. He drinks between 2-4 units of alcohol a week. He is a manager in the healthcare industry.

On systemic enquiry he mentioned that he is a heavy snorer, however there is no history of any daytime hypersomnolence or lethargy.

Examination: pulse 54 beats per minute and regular. JVP was not elevated. Blood pressure, lying: 166/100 mmHg and 158/100 mmHg. Standing at one minute 150/100 mmHg and standing at three minutes 150/100 mmHg. His JVP was not elevated. Heart sounds S1 + S2. His chest and abdomen were unremarkable. There were no carotid bruits. Carotid sinus massage was unremarkable.

His ECG showed normal sinus rhythm, with a ventricular rate of 54 beats per minute and normal conduction indices.

I suspect the cause of this gentleman's symptoms is vasovagal syncope. For completeness I have arranged for him to have a 48-hour ECG, an ambulatory blood pressure monitor and blood tests. For the time being, I have not arranged a tilt test. I have asked him to provide me with copies of his previous investigations, and he will endeavour to do so when he next attends.

Case 2A

I reviewed M[redacted] today in clinic. He has episodes where he feels a little nauseated and "usteady", which tends to occur if he is driving fast. There is no history of any headaches, visual disturbances or focal neurology.

He provided me some correspondence from June 2017 from Dr[redacted] which reported satisfactory blood pressure control and a negative exercise tolerance test. His echocardiogram showed mild septal hypertrophy, with good function. There was no formal report of his cardiac CT scan and I would appreciate if you can forward to me correspondence confirming that this was indeed normal as stated by Mr Segal.

His recent ambulatory blood pressure monitor showed an overall average of 134/87 mmHg, a day average of 136/90 mmHg and a night average of 130/80 mmHg. His blood pressure is borderline elevated and the main question is whether to commence antihypertensive medication.

His 10-year cardiovascular risk is 11%, with a lifetime risk of 45.5% (80 years). If treatment for hypertension is commenced, his 10-year risk is reduced to 9% and lifetime risk 39%. The predicted cardiovascular event risk curves begin to separate at around 55 years. Given his recent syncopal episode, one has to be cautious in commencing antihypertensive medication, in order to not reduce his blood pressure too much. I would suggest observing him a bit longer and reviewing his home blood pressure recordings over the next six months.

In view of his intermittent nausea symptoms and recent head injury, I have arranged for him to have an MRI of his head. This was done on the 25/08/18 and reported as showing a space occupying lesion in the region of the pituitary fossa. I have therefore referred him to Mr.[redacted], Consultant Neurosurgeon at [redacted] Hospital for an urgent opinion.

Yours sincerely,

Case 4

12th February 2013

This gentleman was referred to my clinic having been taken to [redacted] Hospital in late October with a blackout. He is now aged 76 and well known to the cardiologists.

It would appear that around the time of swapping from Atenolol to Bisoprolol he had two events. His wife feels that the first event occurred about a week before his cardiology appointment on 24th October. This occurred whilst in the car and the patient was driving. She noticed his hands were clenched on the wheel and there was no response when she spoke to him. The car also wandered a little. He drove past where they should have stopped. He came to and she made him pull up but he had no idea that this had happened. He attended cardiology on 24th October where his Bisoprolol was increased. On 29th October his wife thought he was a long time upstairs. When she went up she found him standing by the basin looking vacant and unresponsive. He was pale. He then gave a shriek and collapsed backwards with some twitching of all four limbs. She called the paramedics. He had stopped twitching by the time they arrived but was confused. He was taken to A&E. He recalls going upstairs to the bathroom but the next thing he remembers is being in A&E.

Bisoprolol has been stopped and Atenolol reinstituted and he has remained well with no further events since. He remembers feeling unwell on Bisoprolol generally.

As far as his general health is concerned he has mitral valve disease and had a mitral valve repair and has been in atrial fibrillation and flutter.

On examination today his pulse was initially quite rapid but then slowed to 84/minute. He has cataract. There was no other neurological abnormality.

Case 4

Given his cardiac status and the change in the medication I suspect that this has more to do with his blackout than anything else. I note a CT scan was performed when at [redacted] and this was thought to show some periventricular lucencies suggestive of small vessel disease.

I understand he has a cardiology follow-up appointment next month and I will be writing to the cardiologists to make them aware of what has happened. I have warned him that should he have any further events he should stop driving immediately and seek medical advice.

I have discharged him.

Yours sincerely

Dictated but not signed

[redacted]

Consultant Neurologist

20th March 2013 (5 weeks later)

Diagnosis:

1. Mitral valve repair 2008
2. Atrial flutter/fibrillation
3. Syncopal episode December 2012

Referred to Electrophysiologist

I wonder if you could see this very pleasant 76 year old man. He had a mitral valve repair in 2008. LV function is preserved. He has been variously labelled as having atrial flutter/atrial fibrillation. He has remained generally well until last year when his Atenolol (25mg od) was changed to Bisoprolol with an increase in dose at his October clinic visit to 5mg daily. In December he then had 2 episodes where he was noted to be 'slightly vacant'. He then had an episode of transient syncope and attended the A & E department in [redacted]. Bisoprolol was discontinued and he was restarted on Atenolol 25mg od. Subsequently he has remained well.

His 12 lead ECG shows organised atrial activity, atypical flutter with a regular ventricular rate of 68bpm. His 24 hour holter shows organised atrial activity and some periods where the atrial activity is clearly less organised.

I wonder if he has an atypical flutter or a focal left sided AF.

I have left him on Atenolol 25mg od for the meantime. One option would be to implant a loop recorder but I wonder if you think there might be a role for ablation in the first instance.

Best wishes,

Yours sincerely

Dictated but not seen or signed by

[redacted]

Consultant Cardiologist

Case 4

Admission Date: 03/05/13 15:34

Discharge Date: 11/05/13 02:47

77 Male

Primary Diagnosis: Grand Mal Seizures (with Or Without Petit Mal)

Co-morbidity:

Infection control: MRSA status is unknown

Notes:

Presented with LOC while driving in an RTA, next thing he remembers is being in A+E. Wife stated that he was staring ahead and turned in the wrong direction. He was unresponsive to wifes shouts of distress. No tongue bitting or incontinence. 2 previous episodes first time 30 second episode of tightening hand while driving, seemed vacant. second episode was unresponsive to wife then LOC while in the bathroom No memory of incident.

PMH HTN, AF, Mitral Valve repair 5 years ago, Primary dilated cardiomyopathy.

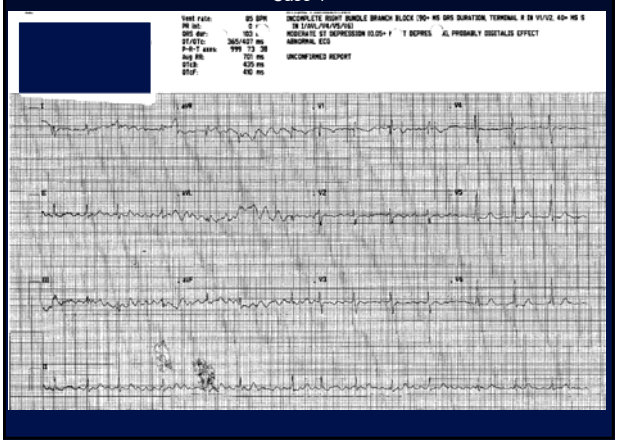
ECG showed AF

Echo showed preserved LV systolic function, Mildly dilated RV with preserved RV function, Mild MR/AR

PMH HTN, AF, Mitral Valve repair 5 years ago, Primary dilated cardiomyopathy.

Reviewed by [redacted] He felt likely diagnosis - Started on sodium valproate and advised not to drive and inform DVLA

Case 4



Case 4

Cardiac vs Epileptic Seizures

Implantable loop recorder

Contacted local cardiologist for further follow up

Also followed up by neurologist

DON'T DRIVE

Diagnoses:
1. Vasovagal syncope
2. Irritable bowel syndrome
3. Haemorrhoids
4. Hypotension

Thank you very much for referring this young 26-year-old lady for a Cardiology opinion. She suffers with syncope and presyncope. Her symptoms usually occur in the morning when she gets out of bed and before breakfast. She can develop abdominal pain, feel hot, sweaty, pale, has palpitations and then can lose consciousness. She then has to open her bowels. Her symptoms occur approximately once a month. She has seen Dr [redacted] Consultant Neurologist and has also been reviewed at the Autonomic Centre, The National Hospital for Neurology and Neurosurgery in Queens Square. She had a Tilt test during which she had no symptoms and also an ambulatory blood pressure monitor. I would appreciate if you could forward to me some correspondence from these consultations.

She has also been reviewed by [redacted] Gastroenterologist and also by Professor [redacted] Consultant Neuro-Gastroenterologist at [redacted]

Her current medication consists of vitamins, probiotic supplements, the contraceptive pill and folic acid.

There is a family history of thalassaemia trait.

She is an ex-smoker of 2½ years with occasional vapes. She drinks up to 2 units of alcohol a week. She works as a hairdresser.

Examination : Weight 57 kg, height 152 cm, BMI 25. Pulse 60 beats per minute regular. Blood pressure 93/59 mmHg. Heart sounds S1 plus S2. Her chest was clear.

ECG showed normal sinus rhythm with normal conduction indices and waveform morphology.

Echocardiogram showed a structurally normal heart.

This lady has neurocardiogenic syncope (vasovagal). She already drinks between 2-3 litres of fluid a day and also has increased her salt intake. I have showed her a website to buy thigh length compression stockings that she can use and also taught her counterpressure manoeuvres. It may be helpful if she sleeps with the head of the bed a little elevated which may help reduce further postural drop of blood pressure when she gets out of bed. If these manoeuvres do not help then I would suggest adding Fludrocortisone starting with 50 µgm and slowly increasing the dose up to 200 µgm monitoring her blood pressure.

I would like to review her again in three months' time.

Yours sincerely,

Dictated and verified by Doctor but not signed

Dr Azad Ghuran MB ChB, MRCP, MD, FESC

Case 4A

Autonomic MDT Summary

The autonomic function tests were informative. There was a low to normal resting blood pressure with no evidence for cardiovascular autonomic failure or no orthostatic hypotension. There was no postural tachycardia syndrome or autonomically mediated syncope on prolonged head-up tilt test.

The catecholamine levels are pending.

The 24-hour ambulatory blood pressure monitoring showed low to normal blood pressure profile with occasional heart rate rise, otherwise unremarkable.

Miss Avanzi has a follow up appointment in clinic to discuss the results.

With best wishes

Yours sincerely

Checked electronically

Dr [redacted]
Autonomic Clinic Fellow

AUTONOMIC UNIT
NATIONAL HOSPITAL FOR NEUROLOGY AND NEUROSURGERY
Queen Square, London WC1N 3BG Tel: 020 344 [redacted]

Provisional Diagnosis: ?Situational syncope

CLINICAL ABSTRACT

- Syncope associated with bowel movements and abdominal pain. Ongoing for 3-4 years, and has got progressively worse. Feels lightheaded, sweaty, nauseous, unable to breathe, vision closes in & loses consciousness when passing stools. Recovery with lying down.
- Thought to have sluggish gastrointestinal tract, which has led to small intestinal bacterial overgrowth.
- Dizziness & presyncope when standing at work (hairdresser).
- Random fast heart beats. Headache, fatigue.
- Bladder Dxg, Cystitis & right tubes found on cystoscopy. Recurrent UTIs.
- No hypermobile traits.
- Thalassaemia.

Surname
First name
Hospital No.
Date of birth
Ward/GP
Consultant
Cc
Requesting doctor

Sex: F

Drugs: Vitamin B12, OCP
Drugs withdrawn for test: nil

RECEIVED 11 [illegible]

Case 4A

I was pleased to review [redacted] in the autonomic clinic today. She was accompanied by her boyfriend. We were able to go through the results of the autonomic testing, which showed no evidence of cardiovascular autonomic failure, no postural tachycardia and we were not able to provoke a syncopal event. Her catecholamine levels (adrenalin and noradrenalin) were normal with an appropriate rise on tilt (adrenalin 28 supine, 35 tilted and noradrenalin 227 supine, 297 tilted).

There was some evidence of blood pooling and although she is not overtly hypermobile at the joints, we do see people with increased flexibility within the body that do not involve the joints.

Triggers for collapse continue to be acute abdominal pain and occasionally waking up in bed or often whilst sitting on the toilet.

We feel this syncopal response is in response to pain and while there are things that may help circulation, the best treatment is likely to be managing the pain itself.

- I have also suggested that she use a toilet posture that elevates her feet, to be in a more natural position to prevent straining.
- She could think about strengthening the core muscles in the pelvis and abdomen to see if this would help with gastric motility.
- She should continue to manage and have a good fluid intake, which is also likely to be helpful.

I was pleased to see that she is due to be reviewed by the GI team as they might have additional advice on preventing the pain. If no overt cause can be found, which happens from time to time, then she may be better in a pain management clinic for further help.

I have not arranged further follow-up at this time, however, would be happy to review her at any time in the future and we wish her all the best for the future.

With best wishes
Yours sincerely

Case 4A

I reviewed [redacted] today in clinic. I am glad to hear that she has not fainted for a while since the commencement of Fludrocortisone.

Her weight today was 47.1kg and blood pressure 112/61mmHg with a pulse of 78 beats per minute. At some point, she would like to have a family and I would suggest discontinuing the Fludrocortisone prior to becoming pregnant. I explained to her that patients are variable and some patients have increase fainting episodes whereas others have a reduction during pregnancy.

I will keep her under review and plan to see her again in 12 months' time or earlier should the need arise.

Yours sincerely,

Dictated and verified by Doctor but not signed

Dr Azad Ghuran MB ChB, MRCP, MD, FESC
Consultant Cardiologist

Case 4A

Thank you very much for referring this pleasant 85-year-old gentleman for a Cardiology opinion. Back in July 2017 he was at a restaurant for lunch and ate a lot. Whilst sitting his vision started to become dim and started to drift in and out of consciousness. The paramedics were contacted and on arrival, it was recorded that his heart rate was 46 beats per minute, blood pressure 72/52. This improved to 54 beats per minute and blood pressure of 117/66 mmHg after lying flat and given some IV fluids. He was taken to the Lister Hospital where it was felt that he had a vasovagal event.

He had an episode three months earlier after eating where his vision went dim however he did not lose consciousness. There was no history of dizziness and there was no major sequelae. He had another episode in the past which occurred 45 minutes after eating when he felt lightheaded whilst talking on the phone. He has a history of getting lightheaded if he gets up quickly from a lying position.

His past medical history includes hypertension, type 2 diabetes mellitus, TURP, cataract operations and a right knee arthroscopy. I understand he has a Nephrology appointment early next year as he has proteinuria. He is booked for a chest x-ray of his fibula fibula later today because of leg pain.

His current medication consists of Adcal D3, Aspirin 75 mg daily, Co-codamol, iron Sulphate 200 mg daily, Furosemide 20 mg daily, Tolterodine 2 mg bd, Lansoprazole 30 mg daily, Metformin 500 mg bd, Losartan 25 mg daily, Pravastatin 10 mg daily, Pregabalin 25 mg daily, Quinine Sulphate and Tamsulosin 400 µgm daily.

He lives with his wife and his two daughters live nearby. He does not smoke or drink.

Examination: Weight 79 kg, height 164 cm, BMI 30.8, pulse 76 beats per minute regular. JVP did not appear elevated. Blood pressure (lying) 148/74 mmHg with no postural drop at 1 minute or 3 minute. Heart sounds S1 plus S2 plus a grade 2/6 systolic murmur in the aortic area. His chest was clear. There were possible bilateral carotid bruits although radiation of his murmur could be an alternative explanation.

His ECG shows sinus rhythm with marked first-degree AV block (PR interval 327 milliseconds with left axis deviation). The QRS duration was 97 milliseconds.

His echocardiogram showed normal biventricular cavity size with good biventricular function. There was no significant valvular abnormalities.

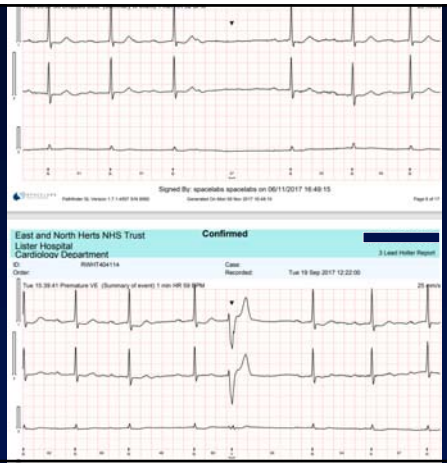
His recent 24-hour ECG showed a minimum heart rate of 50 beats per minute, maximum 89 beats per minute with a mean of 66 beats per minute. During the recording there were some episodes of nocturnal drop beats with a compensatory pause likely due to a non-conducted atrial ectopic beat. There is no significant advance degree of heart block on his 24-hour ECG monitor.

This gentleman's history is consistent with postprandial syncope and I have explained to him that he should not overeat. He should eat small meals but more frequently. He himself realised that he overate and has reduced the volume of his meal consumption. Since then he has had no further episode.

I would like to review him again once more in six months' time and if all is well I plan to discharge him from clinic.

Yours sincerely,

Case 5A



Case 5A

Case 5A

Thank you very much for informing me that this gentleman has had further syncopal episodes. I agreed with you that he needs further prolonged monitoring and I have listed him for an implantable loop recorder.

Yours sincerely,

Dictated and verified by Doctor but not signed

Dr Azad Ghuran MB ChB, MRCP, MD, FESC
Consultant Cardiologist

4 months later

This gentleman attended today for a routine interrogation of his ILR device. This demonstrated ventricular standstill/complete heart block up to 4 seconds. There were some other episodes lasting up to 3 seconds. He was asymptomatic during these episodes but given his history of recurrent syncope I have arranged to implant a dual-chamber pacemaker.

Yours sincerely,

Dictated and verified by Doctor but not signed

Dr Azad Ghuran MB ChB, MRCP, MD, FESC
Consultant Cardiologist

6 months later

Case 5A

Mr. [redacted] made an appointment for a cardiology review. He had a syncopal episode on the 26th September 2020. He was on holidays and had just eaten dinner and had a few more alcoholic drinks than usual. He returned to the cottage where he was staying. There was an open fireplace and the room was very hot. He had just had intercourse and shortly after climaxing he felt hot, thirsty, a dry throat and he subsequently lost consciousness. When he came round, he heard his wife calling him. His wife commented that his eyes were closed and he was not responded to verbal stimuli for approximately a minute. He quickly recovered but felt tired. He then went to bed and was back to normal the following day.

On the 9th October, after a poor night's sleep, with nasal and throat congestion, he woke up feeling nauseated and felt the urge to vomit. Whilst walking to the bathroom, he felt weak and then later knelt over the toilet bowl to vomit. He felt weaker, leaned against the wall and subsequently lost consciousness. He had urinary incontinence and bit his tongue. His wife, who again witnessed the event, commented that he was pale and, on recovery, he was moving his right leg as if trying to press the brakes of a car. Mr Abello commented that, when he started to come round, he had tinnitus, and he was dreaming of driving a car and trying to press the brakes. He heard his wife calling him when he came round. He felt tired. His wife contacted the paramedics, who took him to Luton Hospital. By the time he arrived to Luton Hospital, he felt a lot better. He had blood tests, a CT of his head and an ECG, which were all normal.

He subsequently had a telephone consultation with a neurologist from Luton Hospital, who felt that this was a non-neurological episode, and requested a cardiology review.

Mr [redacted] has a tendency to faint when he drinks too much, and also fainted as a teenager after playing football, particularly when he had not eaten and was not hydrated enough.

Case 5A

His past medical history includes haemorrhoids, tonsillectomy and urethral dilatation. He mentioned that when he was born he had "a hole in the heart", and was followed up for a few years at Princess Alexandra Hospital, and was subsequently discharged as everything resolved.

He currently takes Vitamin D.

His mother is alive at 66 years and has a platelet problem.

He lives with his wife and has two sons, 9 years and 14 years. He does not smoke. He drinks between 5-10 units of alcohol a week. He works as a cabinet maker.

Examination: pulse 62 bpm and regular. JVP was not elevated. Blood pressure lying down 146/84 mmHg, 142/90 mmHg and 138/84 mmHg. Standing at 1 minute 150/84 mmHg and at 3 minutes 156/86 mmHg. Heart sounds S1 + S2. His chest and abdomen were unremarkable. Carotid sinus massage was unremarkable.

His ECG showed normal sinus rhythm, with a ventricular rate of 63 bpm.

I understand you recently did some blood tests including a lipid profile. He was told that his cholesterol is mildly elevated and was recommended lifestyle changes initially.

This gentleman has a history of vasovagal syncope, and his two recent episodes sound very much vasovagal in origin. I believe the DVLA was contacted and he was advised to avoid driving for the time being by the previous medical team who saw him at Luton and Dunstable Hospital. I have arranged for Mr Abello to have an echocardiogram and a 24-hour ECG. If these are normal, then I see no reason why he should not restart driving, given that his recent syncopal episodes were vasovagal in origin.

Conclusion

- **In patients with known cardiac disease syncope should be fully investigated**
- **Diagnosis can be established in most cases with history and limited investigations**
- **Tilt table test useful in diagnosis of vaso-vagal syndrome and carotid sinus sensitivity**
- **Most patients with vaso-vagal syndrome respond to medical therapy**

Syncope

A Diagnostic and Treatment Strategy

Azad Ghuran MB ChB (Edin), MRCP, MD (Edin), FESC
Consultant Cardiologist

www.hertslondoncardiology.co.uk