NEUROLOGY HANDBOOK

"QUICK TIPS"

ADVICE ON THE MANAGEMENT OF COMMON NEUROLOGICAL PROBLEMS

By Dr John Bowen
Consultant Neurologist

Fourth Edition 10.02.18

"very useful for GPs"
- *Dr Peter Coventry GP, Shropshire*

"pragmatic and refreshingly honest"
- *Dr Andrew Wass GP, Rutland*

-

Disclaimer

Acknowledgements

Acknowledgement & thanks are extended to Dr P.Davies (Headache Chapter), Professor M Reuber (Blackouts Chapter) & Dr O Bandmann (Parkinson's disease & related disorders Chapter), Consultant Neurologists and experts in their fields, who reviewed and improved the subsections indicated: to Dr Jon Stone, Consultant Neurologist, from whose work most of the Functional Chapter has (with permission) been pinched: to Drs Peter Coventry & Andrew Wass, General Practitioners for their helpful comments and advice: to the staff of the Neurology Department at Shrewsbury and Telford NHS Trust for their thoughts and contributions: and to my wife, Gillian, for her patience.

Princess Royal Hospital\Royal Shrewsbury Hospital

Consultants

Dr. Piers Newman PRH

Dr John Bowen RSH

Royal Shrewsbury Hospital Secretaries: Tel: 01743 261105: Fax 01743 261288

Melanie Jones: melanie.jones17@nhs.net
Sue Preece : susan.preece4@nhs.net
Tracy Nurse tracy.nurse1@nhs.net

Princess Royal Hospital Secretaries: Tel: 01952 641222 ext. 4817 / 4430; Fax 01952 282873

Bharti Patel bharti.patel10@nhs.net
Brie Rowe brie.rowe@nhs.net
Natalie Caunt: natalie.caunt@nhs.net
Jackie Dainty: Jackie.dainty@nhs.net

Nurse Specialists

Epilepsy (Cross-Sites)
Conor Smyth: Epilepsy Specialist Nurse 01743 352540. Mobile 07866362075.
Email : conor.smyth@nhs.net

Multiple Sclerosis (Cross-Sites)
MS Admin Coordinator: 01952 641222 ext 4048
Lorna Baker: Multiple Sclerosis Specialist Nurse Email: lornabaker1@nhs.net
Alison Mourghton: Multiple Sclerosis Specialist Nurse

Parkinson's disease
Marie Haywood: Parkinson's Specialist Nurse Mobile 07801 404521 Email : marie.haywood@nhs.net

Motor Neuron Disease
Lisa Sievwright: MND Co-ordinator (Community)/ Severn Hospice. Mobile:07884432593

Neurophysiotherapists - Cross Site

Carol Napper. Clinical Specialist Physiotherapist (in the management or Spasticity
Email: carole.napper@nhs.net

Sandy Ramsey. Clinical Specialist Physiotherapist. Email: sandy.ramsay@nhs.net

Rachel Leigh. Clinical Specialist Physiotherapist Email: Rachel.leigh2@nhs.net

Anita Evans. Clinical Specialist Physiotherapist Email: anita.evans2@nhs.net

CONTENTS

WELCOME TO NEUROLOGY: 6 TIPS TO LESS STRESS?

For many clinicians (and very many patients) symptoms to do with the brain and the nervous system are especially concerning.

Much of this concern is based on uncertainty which feeds anxiety which both amplifies and multiplies symptoms and things gets worse. Increased access to information and investigation does not seem to have improved matters so in an attempt to help some tips are offered below which reflect recurrent themes seen in the clinic over the last decade or two. Though much may appear obvious and self-evident, I hope some of this might prove useful in your clinic.

1. Neurological symptoms are a lot more common than neurological diseases.

To take one example, we all see a lot of patients with headache. Sometimes we worry. More often patients worry.

We looked a 6 months of consecutive 2 week wait cancer referrals – mostly headache. There were no primary brain tumours, not one! Two patients had metastases, neither had headache.

Brain tumours usually present with focal signs and/or focal seizures. If your patient is worried about a brain tumour and none of the above is present it's fine to explain this and allay their concerns.

2. Do you have any idea what it could be Doctor?

Of course you will, usually. And all but one (hopefully!) of your ideas will be wrong. So the value of sharing such potentially worrying information with an already uncertain and anxious patient even under the guise of "I think it's unlikely but I just want to rule it out" is questionable at best.

You will routinely ask the patient + spouse/partner what they think might be wrong & if stress is suggested it is quite likely to be just that. I would suggest that if the response is "I haven't got a clue / you're the Dr" etc, push on further. "What's the worst case scenario(s) you have considered?"

Anecdotally (the antidote to evidence-based) I have had many patients leave delighted with their fears relieved, seemingly wanting nothing more.

If one doesn't know what is going on, what can one say other than just that- with whatever explanation & reassurance can be justified e.g. insufficiently specific, no sign of anything unpleasant, not ringing any alarm bells I'm pleased to say, etc.

3. To scan or not to scan – that is the question.

Never before have more scans been done (with less waiting) than now. Inevitably the pick-up of trivial & incidental findings is rising. As the volume & speed with which scans "must" be done outstrips capacity to do them (in cost and manpower) the unintended consequence of losing the target pathology in a sea of normal/ incidental findings emerges. Scanning to reassure when the risk of incidental finding exceeds the risk of finding relevant pathology by at least 2 orders of magnitude is questionable at best. I explain this to patients who want a scan yet of course lack knowledge of the concept of incidental findings. For example 3-5% of us have unruptured intracranial aneurysms though the annual risk of subarachnoid haemorrhage is ~ 9/100,000.[1]

4. It's All in Your Head

Long ago studies concluded that about a third of new neurology outpatients and half of all neurology clinic patients had functional symptoms (physical symptoms unexplained by physical disease). This is numerically the largest group in an unselected neurology practice. What this figure is in primary care others will know but it's unlikely to be small.

Clues to functional disorders include a high number of symptoms including pain (almost invariable), disproportionate fatigue, concentration problems, and symptoms that leave you bereft of any physical diagnosis. Certain neurological symptoms like dropping things and multifocal evanescent tinglings are very common and feelings of detachment and out of body experiences (derealisation and depersonalisation) are clear indictors of psychological factors at play. Other co-existent functional disorders (irritable bowel syndrome / fibromyalgia) & medically unexplained complaints often co-exist.

I would strongly recommend a glance at the neurosymptoms website (www.neurosymptoms.org) and reading Dr Suzanne O'Sullivan's book - It's All in Your Head (Chatto & Windus).

5. Is Neurology the optimal referral destination?

It's always good to find a point of agreement and we can agree that the brain is pretty important. I hope we can also agree that getting the most out of your Neurology service is pretty important too, though "Right patient right time right place" is one of those oft repeated sayings that fails the test of "should arguing for the opposite be clearly absurd then the comment is without value".

Transient loss of consciousness with little to suggest a seizure should go to cardiology, swiftly if clearly not vasovagal as the potential cardiac mortality can be worryingly high, far more so than a seizure which is less likely if there is nothing to suggest it happened.

Dizziness without features to suggest brain involvement is most commonly peripheral and most appropriately assessed by ENT colleagues, assuming it's not lighthedness in which case think heart (& consider over treated hypertension!) & if referral required cardiology will serve the patient better.

Visual disorders should be first seen ophthalmology as cataracts, glaucoma, & broken down squints are not well managed in neurology. Furthermore, odd looking discs are best clarified by eye specialists.

Hydrocephalus and lumps in the head need neurosurgeons, based at regional centres.

Psychological / psychiatric / learning disability / rehabilitation /Chronic Pain related disorders are most likely to benefit primarily from appropriate specialised services rather than Neurology.

A pragmatic and focussed referral pattern would facilitate optimal use of our current regular consultant workforce of 2.

6. It's good to Talk

There will be times when things don't go well. This needn't mean anybody has messed up or doesn't care or is a bad person. It's also the case that not every moment of every day is joyous and mood swings, bad days, and illness are not restricted to one side of the desk. If things aren't great and a chat might help it is usually possible to return phone calls within one or two working days (annual / study leave/ disease permitting) and if mobile numbers are left this makes the task easier.

1. Rooij N. et al. J Neurol Neurosurg Psychiatry. 2007 Dec; 78(12): 1365–1372.

HEADACHE

The Essentials

REGULAR USE OF SIMPLE AND COMPOUND ANALGESICS
ARE THE COMMONEST PREVENTABLE CAUSE OF CHRONIC HEADACHE
AND
RENDER ANY OTHER ATTEMPTS TO IMPROVE HEADACHE USELESS

Further information freely available on

British Association for the Study of Headache website (www.bash.org.uk).

Headache (like any pain) is a symptom not a diagnosis.

Primary headaches (migraine, tension type) are common.

Secondary headaches (tumour, intracranial bleed) are rare and seldom if ever present with isolated headache in alert patients with a normal neurological examination.

Headache – Causes & incidences

Condition	Incidence/year
Migraine	1 in 11
Chronic Daily headache*	1 in 25
Medicine Overuse Headache	1 in 50
Cluster Headache	1 in 1000
Subarachnoid Haemorrhage	1 per 10,000
(Brain Tumour	1 in 10,000)

*Headache more days than not for > 6 months

Headache – Diagnosis

Since the vast majority of headaches are primary, routine investigation (scanning) of headache without specific worrying features (see below) seldom uncovers relevant positive findings. Inevitably most abnormalities found are incidental not causative. This point is worth discussing with patients seeking a scan “for reassurance”.

Brain Scanning in Headache

Types of Pt	**Nº with serious Abnormality**	**%**	**95% CI**
Migraine	2/1086	0.2	(.02-0.7)
TTH	0/83	0	(0-4.4)
Chronic HA	7/1445	0.5	(0.2-1)
Asymptomatic	4/1000	0.4	(0.1-8)
Asymptomatic	Meta-analysis	2.7	Nº needed to scan- 37

* BMJ 2009; 339:b3016

Primary headache diagnosis therefore is clinical, with migraine and tension type headache constituting numerically the majority of presentations. Much “sinus” headache is migraine.

More iWELCOME TO NEUROLOGY: 6 TIPS TO LESS STRESS?

WELCOME TO NEUROLOGY: 6 TIPS TO LESS STRESS?

WELCOME TO NEUROLOGY: 6 TIPS TO LESS STRESS?

nformation on scanning in headache can be found at Appendix V

Temporal pattern of headache is of key diagnostic relevance.

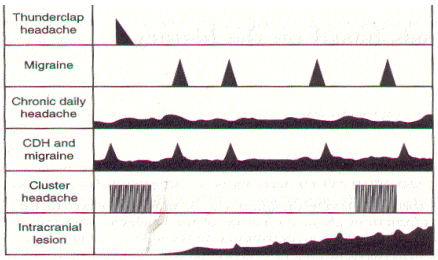


Figure 4.1 Temporal patterns of headache; chronic daily headache (CDH)

Questions to ask about Headache

Circumstantial	Specific
Antecedent health	Onset (time to maximum intensity of pain in minutes), main site, radiation, character, severity (1-10), duration, frequency, aggravating & relieving factors, periodicity, associated phenomena (especially neurological symptoms).
Activity at onset	
Course of headache since onset	
Effect on usual activities	
Drugs taken before (incl. recreational) & since (esp OTC analgesics)	
Past medical & family history of headache / head disease	Worries, concerns

Why do you think you have this headache?

Return to Contents Page

MIGRAINE

IHS diagnostic criteria for migraine without aura:-

A: at least five attacks fulfilling criteria B-D

B: headache attacks lasting 4-72 hours

C: headache has ≥ 2 of the following:

- Unilateral location
- Pulsating quality
- Moderate or severe pain intensity
- Aggravation by/causing avoidance of routine physical activity

D: during headache at least one of:

- Nausea and / or vomiting
- Photophobia and phonophobia

E: not attributed to another disorder

Remember your PIN?

A brief validated screening instrument with a 93% (if 2/3 questions answered "yes") and 98% positive predictive value (if 3/3 questions answered "yes") for the diagnosis of migraine can be recalled by the acronym **PIN.**

Does the light bother you more when you have a headache – **P**hotophobia

Do your headaches limit your ability to work, study or function – **I**mpairment

Do you feel nauseated or sick in your stomach with your headache - **N**ausea

Further Diagnostic tips

- Premonitory Phase
  - Feeling on "top of the world"
  - Hungry & craving
  - Very tired, irritable, yawning a lot
- Aura
  - May not occur
  - **Positive** visual (zig zags/ shimmerings) & sensory (tingling) symptoms usually <1hr
  - **Spread over 5-30 minutes**
- Headache
  - Episodic
  - Intensity rises over time –(> 10mins – hours)
    - **(not usually of abrupt onset)**
  - Throbbing, constant, +/- "jabs & jolts"
  - Retro-orbital ache / stabbing / prickling pain
  - Often unilateral, can be anywhere!
  - Photophobia, phonophobia, osmophobia
  - Aggravated by head movement
  - Sleep benefit
- Resolution
  - Exhaustion/apathy can last days

- May have paradoxical euphoria
- Sensitive to head pain on shaking/jarring head

Management

A firm diagnosis is a good start!

(Headache diaries (Appx IV) including self- medication records assist diagnosis)

- Trigger Avoidance / Manipulation
  - (diet/ alcohol/ avoidable stress/fatigue/ regular sleep pattern/ hormones)
- Drug Treatment / Cessation of regular analgesics in chronic headache (see below)

Acute Symptomatic Treatment

Step 1

Aspirin 600mg-900mg soluble or Ibuprofen 400-800mg +/- buccal prochlorperazine (3mgm)

Migramax or Other NSAIDs +/- antiemetics eg domperidone oral / suppository

Step 2

Triptan + NSAIDs

Step 3

Triptan + NSAIDs +Antiemetics

Consider

Timing of Drug Delivery:	Earlier better
Route of Delivery:	If vomiting intranasal / injectable better
Tolerability/Efficacy:	These vary between triptans & patients
Expectations:	Try for 3 attacks and switch triptans if needed

Return to Contents Page

Persisting Problems?

Points to consider:

1. **Regular analgesics perpetuate chronicity in all headaches**
2. **All acute treatments (including triptans) can induce rebound headaches if taken to excess (more days than not)**
3. **Route of delivery**

   (nausea / vomiting – consider sublingual/ nasal/ rectal

   (diclofenac, domperidone)/ s/c routes (prochlorperazine), antiemetics
4. **Timing of treatment**

   (early treatment more likely to work, especially antiemetics)
5. **Responses to different triptans**

   (Vary between patients try different ones if necessary)

   (Even in best responders efficacy < 70%. Worth trying on three occasions)
6. **Prophylaxis need**

   If rising or > twice weekly triptan requirement

   If taking triptans > twice per week – high risk of rebound phenomena
7. **Migraineurs commonly have other non-migraine primary headaches** e.g. tension type headaches, idiopathic stabbing headaches
8. **Contraindications to both triptans (& ergotamine)** - uncontrolled hypertension & IHD, cerebrovascular disease. Avoid beta blockers with ergotamine.
9. **Insufficient dose /treatment course** (Max tolerated dose/3 months duration)
10. **Unrealistic expectations** (Improvement not cure is goal / HA diaries help objective assessment)
11. **Coexistent Issues** (Unaddressed concerns/ Depression/Alcohol /Substance misuse)
12. **Incorrect diagnosis** (Secondary HA)
13. **Tough Headache!**

Emergency home treatment:

IM Diclofenac 75mgm / Parenteral Antiemetics

OR

IM Chlorpromazine 25 – 50mgm

(Parenteral Narcotics **not** advised)

Prophylactic Treatments

Indications:

1. Increasingly frequent migraine
2. Two or more disabling migraine per week
3. Inadequate relief from acute symptomatic treatment AND
4. Patient wishing to engage in prophylaxis

Caveats:

1. Analgesics (especially opiate based drugs / triptan / ergotamine) excess (This renders prophylaxis ineffective and needs correcting first.)
2. Depression
   (Caution with beta blockers, consider tricyclics and depression Rx)
3. Other options (trial off OCP, review other potentially "headachogenic" Rx such as dipyridamole, nitrates, antihypertensives)

Prophylactic Drugs[1d]

Start low, go slow,

End-points – efficacy / intolerable side-effects

If tolerated but ineffective raise the dose

Assess over blocks of 2 months using headache diaries

Drug Name	Start Dose	Incremental rate*	Target Dose	Max Dose	Cautions/ Contraindications
Topiramate[1a] **(reduces efficacy of COCP at doses > 50mg bd)**	25 mgm od	25 mgm/ fortnite	50 mgm bd	100mg bd	Teratogenicity[£] Weight loss, tingling, urinary calculi[$]
Amitriptyline	10 mg nocte	10 mg/wk	50 mg nocte	1.5mg/kg day	Sedation, Dry mouth, Confusion
Propranolol***	10mgm od**	10mgm/wk	20 mg tds or Half Inderal LA 1tab/od	160mg /day	Asthma, Diabetes, Vascular disease, Depression
Riboflavin	400mg od	-	400mg od	400mg od	

* Incremental rate determined by headache frequency. Allow sufficient time at each dose to assess response using headache diaries (Appx IV) if necessary

** Atenolol or Metoprolol may be substituted if side-effects troublesome

$ Ensure 3 litres fluid per day. Caution if history of urinary calculi.

*** Can use 10 mgm tds if concerned about potential side-effects (i.e. if already complaining of fatigue, low mood etc). Use 5 mgm Rizatriptan dose with propranolol

£ 5%

NB Agents also shown to have prophylactic activity include:

pizotifen, valproate*, venlafaxine, flunarizine, & candesartan

*https://www.gov.uk/drug-safety-update/valproate-and-of-risk-of-abnormal-pregnancy-outcomes-new-communication-materials

Return to Contents Page

Botulinum Toxin & Migraine

NIHCE approved the use of Botulinum toxin for the prevention of migraine in patients with chronic migraine in 2012[1c].

Starting Criteria are:

1. Headaches on at least 15 days per month
2. With migraine on at least 8 of these days
3. Without (or with*) concomitant excessive analgesic use **and**
4. Three different preventive agents have failed to help previously.

*Where present this has been "addressed"

Stopping Criteria are:

1. Less than 30% reduction in headache days after 2 courses of injections

Or

2. Fewer than 15 days per month each month for 3 successive months

REGULAR USE OF SIMPLE AND COMPOUND ANALGESICS
ARE THE COMMONEST PREVENTABLE CAUSE OF CHRONIC HEADACHE
AND
RENDER ANY OTHER ATTEMPTS TO IMPROVE HEADACHE USELESS

MIGRAINE – SPECIAL CIRCUMSTANCES

Migraine & pregnancy

- Paracetamol in moderation is safe in pregnancy & breast feeding.
- Aspirin is safe **except near to term**.
- Prochlorperazine is unlikely to cause harm throughout pregnancy & lactation.
- Metoclopramide & domperidone are probably safe in the second & third trimesters.
- Migraine improves, remains unchanged, or worsens (~1/3 each) during pregnancy & lactation, so prophylaxis can be avoided.
- Generally best avoided. Propranolol and amitriptyline are probably safest but counselling regarding risks (intrauterine growth retardation with beta blockers and possible increased rate of spontaneous abortion with tricyclics) & benefits should come first & be documented. NIHCE offers no comment on this above seeking specialist advice.

- Triptans may be considered[1d] following discussion of needs for treatment and associated risks (evidence of harm to mother or fetus is sparse).

Return to Contents Page

Catamenial (Menstrual) Migraine[1bi]

Defined as migraine without aura occurring on or between 2 days before menstruation and day 3 of bleeding (days -2 to + 3) only (pure menstrual migraine) or in combination with migraine at other times +/- aura (menstrually-related migraine), confirmed by diaries in at least 2/3 of (not less than 3) cycles. Prophylaxis should be tried for at least 3 cycles before dismissed as ineffective.

Step 1. Mefenamic acid 500 mgm tds-qds from onset to end of menses

Step 2. Transdermal oestradiol 100 microgm patch from 3 days before onset for one week thereafter OR

Oestradiol 1.5mg gel for day -3 for 7 days

Step 3. Frovatriptan 5 mg bd day 1, 2.5 mg bd days 2-6 starting 2 days before onset of menses[1bii, 1d]

Step 4. Tricycling (9 weeks on OCP with one week off) with combined oral contraceptives (if migraine unassociated with focal symptoms and if oestrogen containing OCPs haven't aggravated migraine in patient previously), desogestrel (Cerazette), subdermally implanted etonogestrel (Implanon) without oestrogen, and injectable depot progestogens.

Further information on British Association for the Study of Headache website (www.bash.org.uk).

Migraine & Hormonal Contraception

May trigger onset of or worsening of pre-existing migraine. May improve migraine!
Concern exists that migraine and the COCP are risk factors for stroke in young women.
Evidence mixed.

Relative contraindications to ethinylyoestradiol COCPs:

Migraine with aura.[1d]
Migraine without aura but with one or more additional risk factors for stroke
Migraine treated with ergot derivatives (but not triptans)
(Progestogen-only contraception is acceptable with migraine of any type).

Migraine and Patent Foramen Ovale

Insufficient evidence to support routine closure.[1e]

Return to Contents Page

MEDICATION OVERUSE HEADACHE (MoH)

Headache occurring on more days than not as a consequence of regular overuse of acute or symptomatic headache medication (simple/compound analgesics/NSAIDs/opioids/triptans) or 10 or more days per month.
Seen in patients with pre-existing primary HA who with such therapy develop new or worsening HA. Migraine or TTH features are common.

Withdrawal of analgesics is the essential step in managing CDH[3a, b]

One third significantly improve

One third modestly improve

One third no change

Virtually nobody worse off 4-6 weeks after withdrawal

Temporary worsening can occur especially coming off opiates

Mean reduction of headache days of 46% (N=175)[3c]

Suggestions for planned drug withdrawal

1. Explain why its being advised

   You have a chronic headache that keeps returning because of the painkillers. There is a 1 in 2 chance you will improve if we do this and no chance you will improve if we don't.

2. Explain what's involved

   You need to stop you pills as quickly as possible. Your headaches may worsen temporarily but then there is a good chance things will improve. It is difficult to say how long this will take exactly but in most patients it's between 5 and 10 weeks.

3. Be realistic!

   There are no guarantees except one. If your patients and their tablets don't part company there is no reason to think their headaches are ever likely to improve. If the headaches become intermittent, as they often do on successful withdrawal of perpetuating medication, then other medicines may be introduced that have a much better chance of helping. These medicines are most unlikely to be effective you are taking pain-killers at the same time.

4. Use a headache diary (noting days of headache and intensity 1-10)

   This can be an extremely helpful and empowering experience for patients undergoing this process.

5. Discuss and negotiate

Whether to initiate preventative treatment at the time of analgesic withdrawal or wait 5-10 weeks and then reconsider. The argument for the former is that it may make the transition off analgesics to a headache-free (or more realistically headache-reduced) state more likely. The argument for the latter is that some patients will not need preventative therapy at all once analgesics are withdrawn so this group (which cannot of course be predicted on an individual basis) may have very little to gain by taking preventative therapy (other than side-effects!).

CHRONIC DAILY HEADACHE (CDH)

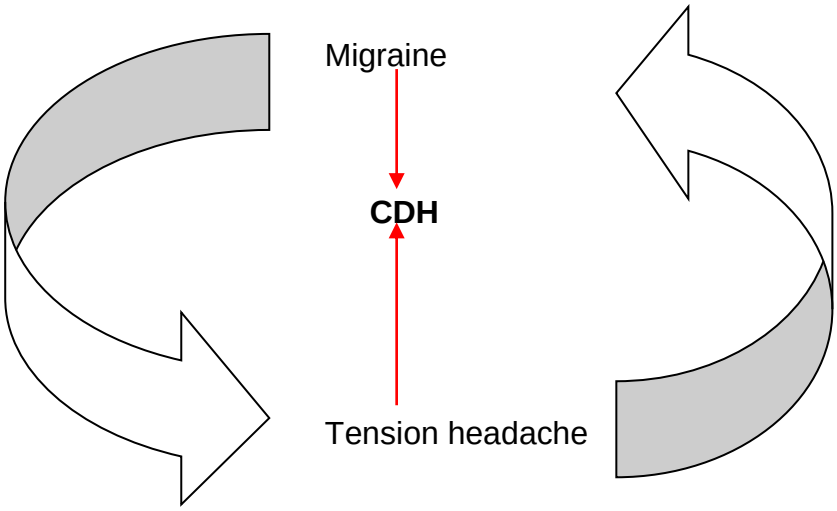
Definition

A descriptive term rather than an aetiological diagnosis

- Headache more days than not for 6 months or longer
- Usually arise in one of two settings:-
  - An episodic headache that has become more frequent until it's daily
    - Eg Chronic (formerly "transformed") migraine
  - A headache that is daily from onset
    - Eg New Persistent Daily Headache (NPDH)
- Various causes < or > 4 hrs in duration

Prevalence ~ 4 % of general population / ~ 2 million in UK (common!)

Many patients with chronic daily headache overuse acute headache medication but not all have medication-overuse headache as some will continue to have frequent headaches despite withdrawal of such medication.



Many patients with CDH are depressed[2a] and anxious.[2b]

Management

1. A good hearing and thorough examination*[2c]
2. Identification of excessive muscular activity, coexistent depression, anxiety & hyperventilation with explanation and management
3. Withdrawal of offending "headache treatment"[2d]
4. Refined diagnosis (chronic migraine/ tension type headache/ hemicrania continua / NPDH)

5. Non-analgesic based drug therapy if desired
6. Acupuncture (evidence base weak)

*A recent outpatient study revealed that only 0.9% of consecutive headache patients without neurological signs had significant pathology[2c]

Return to Contents Page

CDH - Drug-based Treatment

These drugs are unlikely to work if analgesics continued to be used.

Drug Name	Start Dose	Incremental rate	Target Dose	Max Dose	Cautions
Amitriptyline	10 mgm nocte	10 mgm per fortnight	50 mg nocte	1.5 mg/kg/day	Sedation, Dry mouth, confusion
Topiramate[2e,f] **(reduces efficacy of COCP at doses > 50mg bd)**	25 mgm od	25 mgm/ fortnite	50 mg bd	100 mgm bd	Teratogenicity (5%) Weight loss, tingling, urinary calculi[$]
Valproate [4a]* Slow release	200 mgm nocte	200 mgm per fortnight	600 mg nocte	1.6G nocte	Teratogenicity (10%) Weight gain, tremor,

$ Ensure 3 litres fluid per day. Caution if history of urinary calculi.

*****Sodium valproate is useful if when tension type headaches & migraine co-exist, or if migrainous features are present in the history or if an increasing frequency of migraine –like headaches have transformed in to CDH.
Sodium valproate is associated with an increased risk of spina bifida in infants conceived during maternal use (1-2% compared to background risk of 0.2 – 0.5%). Other additional adverse physical & cognitive sequelae in offspring born to mothers taking valproate at conception & during pregnancy. It is imperative that these are discussed in full with all women of child bearing potential prior to commencing this drug. It is strongly recommended that such discussions are documented in medical notes.
Additional side-effects of which all patients should be advised before commencing valproate include the potential for weight gain, tremor, and very occasionally hair loss (which is reversible).
Valproate does not reduce the efficacy of the oral contraceptive pill.

The MHRA has published guidelines to assist clinicians prescribing valproate[4ai]

Tension-Type Headache (TTH)

Thought to be commonest HA in primary care. Often reassurance sufficient. Many manage with simple analgesia though risk of MoH needs explainaing. Chronic TTH can be helped with amitriptyline or topiramate[4b] (schedules above) or acupuncture[1d]

Return to Contents Page

CLUSTER HEADACHE

https://ouchuk.org/cluster-headaches-overview

Also referred to as migrainous neuralgia.

Defined as a severe, strictly unilateral headache or face pain lasting from 15 minutes to 3 hours, 1-8 times per day, occurring in bouts of weeks to months, with marked periodicity. Intense pain is orbital, commonly associated with ipsilateral conjunctival injection, lacrimation, and blockage of the nostril. Restlessness during attacks helps distinguish this from migraine (most migraineurs prefer to remain still during a migraine attack).

80-90% of patients experience bouts with intervals free of such headaches.
Prevalence believed to be ~ 1:1000. Marked male predominance ~ 4:1
Usually starts 20-40 yrs old. Diagnosis is clinical. There are no tests for this.
Despite the primary nature of this headache, its chronicity and severity have led to a general recommendation to investigate with magnetic resonance imaging to exclude secondary (retrorbital / para-sellar) structural abnormalities, though pick up rates remain very low if the clinical presentation is typical.

Treatment

Acute Attacks

Sumatriptan injection 6 mgm s/c[5] ≤ 2 per day

(intranasal sumatriptan or zolmitriptan can be used but takes longer to work)

High concentration (7-15L/min) oxygen via rebreathing mask*.[6]

* Oxygen requested on Home Oxygen Order Form (HOOF-A)
Patient to complete Home Oxxygen Consent Form (HOCF)
Supplier for West Midlands Air Products 0800 373580
(Oxygen non rebreathing masks available from Flexicare Medical Limited, (www.flexicare.com)

Suppression of bouts

1. Verapamil[1d,7] 80 mgm tds (check ECG first for heart block / WPW and prior to and following dose increases over 360 mg/day)

If tolerated but ineffective

Increase by 40 mgm tds to 120 mgm tds and assesses response

If tolerated but ineffective check ECG and if satisfactory

Increase by 40 mgm tds to 160mg tds

If tolerated but ineffective check ECG and if satisfactory

Continue to increase by 40 mg tds up to but not above 320 mg tds checking ECG before each increase

2. Prednisolone[8] 60 mgm daily for 7 days tapering by 5 mgm alt days until cluster recurs

Then hold for 2 days at previous dose & continue tapering thereafter.

The usual caveats & cautions apply to steroid use in cluster headache.

Prolonged (> 6weeks), frequently repeated (> 2/year) steroid use is discouraged.

Cluster Headache - Indications for referral

1. Diagnostic Uncertainty
2. Failure to tolerate / respond to medications outlined above
3. First presentation or no spontaneous resolution

** See Appendix I for useful resources*

Other Trigeminal Autonomic Cephalagias

(Paroxysmal Hemicrania and SUNCT*)

Rare but worth knowing about since they are diagnosed on purely clinical grounds and are treatable.

UNILATERAL, SHORT-LASTING, HIGH-FREQUENCY

Feature	Cluster Headache	Paroxysmal Hemicrania	SUNCT*	Tic Dol$	Hypnic HA	ISH^
(M:F)	4:1	1:3	2:1	F>M	5:3	F>M
Duration	15-180 mins	2-45 minutes	15-120s	< 1s	15-30m	<30s
Frequency	1-8/day	1-40/day	1/day – 30/hour	Any	1-3/N	Any
Location	Orbital	Orbital	Orbital	V2/V3 > V1	Holo	Any
Triggers	Alcohol/GTN	Mechanical	Cutaneous	Cut	Sleep	None
Autonomic	+	+	+	(+V1)	-	-

Treatment	Verapamil	Indomethacin	Lamotrigine	CBZ	Caffeine?	?

* SUNCT Short-lasting unilateral neuralgiform headache with conjunctival injection and tearing

$ Tic Doloreux – Trigeminal Neuralgia

^ Idiopathic Stabbing Headache

Return to Contents Page

HEADACHE

When to worry	Why	Action
1. Abrupt onset (and worst ever)	**suggests intracranial bleed** –	Admit
2. **New** onset in:		
i. > 55 year old	**? Giant cell arteritis**	– Urgent ESR/CRP
ii. Patient with a cancer past or present	} **Increased chance of 2^0 HA**	} F/R/Ad*
iii. Patient with HIV	}	} F/R/Ad*
3. Focal signs (not known to be old)	**suggesting focal pathology**	-} F/R/Ad*
4. Exertional/ Positional/ Nocturnal	**suggests raised IC pressure**	-} F/R/Ad*
5. Headache + fever / altered behaviour / fits	**Secondary headache**	- } F/R/Ad*

***F/R/Ad** fax referral or admit depending on clinical scenario & level of concern,

WELCOME TO NEUROLOGY: 6 TIPS TO LESS STRESS?

References

1a. Brandes JL, et al. Topiramate for migraine prevention: a randomized controlled trial. JAMA 2004; 291(8):965-73

1b i. MacGregor EA. Diagnosis and management of menstrual migraine. Progress in Neurology & Psychiatry.2011:15(2):11-16

1b ii. Silberstein SD, et al. A randomised trial of frovatriptan for the intermittent prevention of menstrual migraine. Neurology 2004; 63:261-269

1c. Botulinum toxin type A for the prevention of headaches in adults with chronic migraine. NICE technology appraisal guidance [TA 260] Published date: June 2012

1d NIHCE.Clinical Guideline (150); Headache: Diagnosis and management of headaches in young people and adults.2012

1e. https://www.nice.org.uk/guidance/ipg370

2a. Lance JW & Curran DA. Treatment of chronic tension headache. Lancet 1964; I: 1236-1239

2b. Gainotti G, Cianchetti C, & Taramelli M. Anxiety level and psychodynamic mechanisms in medical headaches. Res Clin. Stud. Headache 1972; 31:302-304

2c. Sempere A, et al.Neuroimaging in the evaluation of non-acute headache. Cephalagia 2005; 25:30-5

2d. Zeebug P, et al.Probable medication overuse headache: the effect of a two-month withdrawal period.Neurology 2006; 66:1894-88

2e. Diener HC, et al.Topiramate reduces headache days in chronic migraine: a randomized, double-blind, placebo-controlled study.Cephalalgia 2007:27(7):814-23

2f. Silvestrini M, et al. Topiramate in the treatment of chronic migraine.Cephalalgia 2003; 23(8):820-4

3a. Schnides P, et al. Long term outcome of patients with headache and drug abuse after inpatient withdrawal: five-year follow-up. Cephalagia 1996; 16:481-485

3b.Meskunas CA, et al.Medications associated with probable medication overuse headache reported in a tertiary care headache centre over a 15-year period. Headache 2006; 46:766-72

3c. ZeebergP, OlesenJ, Jensen R. Discontinuation of medication overuse in headache patients: recovery of therapeutic responsiveness.Cephalalgia 2006; 26:1192-8
4a. Mathew NT & Ali S. (1991). Valproate in the treatment of persistent chronic daily headache. An open label study. Headache 31, 71-74
4ai. https://www.gov.uk/drug-safety-update/valproate-and-of-risk-of-abnormal-pregnancy-outcomes-new-communication-materials
4b. Clarke C, Howard R, Rosser M, et al.Neurology: a Queen Square textbook. 1st edn. UK: Wiley-Blackwell, 2009
5. Goadsby PJ. Cluster headache and the clinical profile of sumatriptan. Eur Neurol1994; 34 (Suppl.):35-39
6. Fogan L. (1985). Treatment of cluster headache: a double blind comparison of oxygen vs. air inhalation. Arch Neurol. 42, 362-363
7. Leone M, D'Amico D, Frediani F, et al. Verapamil in the prophylaxis of episodic cluster headache: a double blind study versus placebo. Neurology 2000; 54:1382-5
8. Jammes JL. The treatment of cluster headaches with prednisone. Dis Nerv Syst.1975; 36:375-6

Return to Contents Page

BLACKOUTS

DIAGNOSIS

Many potential causes but commonest (constituting ~95% seen in clinic) are:

Syncope	typically underdiagnosed
Epileptic Seizure	typically over diagnosed*
Non-Epileptic seizure	typically underdiagnosed

*Misdiagnosis rates reported to vary from 16%-32%.[8]

Distinguishing syncope from seizures is usually straightforward.

Difficulties can arise if / with:

1. Undue emphasis is placed on a single feature such as incontinence (people fainting with a full bladder can be incontinent: people convulsing with an empty bladder cannot) or twitches or jerks (commonly seen in syncope especially if patients held up or unable to lie flat).
2. Partially witnessed accounts, minor head injuries (which may involve trauma to face or tongue and be considered "tongue biting")
3. Alcohol (+/- head trauma) impairs patients' recollection of the prodromal syncopal symptoms thus obscuring a diagnosis of syncope.
4. Underappreciation of the significance of the situation in which the blackout occurs (toilets – micturition syncope, mid-painful swallow – oesophageal syncope, restaurants, aeroplanes – high risk situations for syncope: crowded supermarkets & shopping centres – common trigger sites for anxiety hyperventilation and "funny turns") can result in over diagnosis of epileptic seizure, too.
5. Overemphasis on a "positive family history" – common in syncope & non-epileptic attack disorder.
6. Lack of awareness of differential diagnosis – "all that twitches is not epilepsy"

Four Big Red Flags for sinister syncope (See T-LOC form below):

1. **Exertional trigger/ sitting or supine symptom onset/ lack of trigger.**
2. **Cardiac symptoms and/or Cardiac Signs**
3. **Past Medical and/ or Family History of (hereditable) heart disease/sudden death**
4. **Abnormal ECG.**

These should prompt urgent CARDIOLOGY assessment

Transient Loss of Consciousness (T-LOC)

A

Questions to diagnose true syncope.	Yes	No
Was LOC complete?		
Was LOC transient with rapid onset and short duration?		
Did the patient recover spontaneously and completely with no sequelae?		
Did the patient lose postural tone?		

If the answer is yes to all → high likelihood of true syncope. Go to B.
If the answer to any of the questions above is no → exclude other forms of LOC first.

Non syncopal causes of transient LOC.
Intoxications: Drugs and alcohol Epilepsy Trauma – concussion Metabolic disorders: ↓glucose ↓ P_aO_2 ↓ P_aCO_2

Stratification of Risk in Syncope: Clinical and ECG Criteria

B

Clinical Features	Tick
▪ Syncope during *exertion,* when *sitting or supine.*	
▪ Absence of prodromal symptoms or predisposing factors or precipitating factors.	
▪ Preceded by *palpitations* or accompanied by *chest pain* or *shortness of breath*	
▪ History of *structural heart disease* or heart failure, presence of ventricular arrhythmia or *coronary artery disease*.	
▪ Family history of *SCD* or an inherited cardiac condition	
▪ Examination suggestive of *obstructive valvular heart disease*	
▪ Syncope associated with *trauma*	
▪ SBP < 90mmHg	
▪ Acute drop in haemoglobin.	
▪ Severe electrolyte disturbances e.g. ↓K^+ ↓ Mg	

ECG Changes	Tick
ECG changes favouring bradyarrhythmias	
▪ High degree AV blocks – Mobitz Type II 2° heart block, complete heart block, tri-fascicular block.	
▪ Bi-fascicular block	
▪ IVCD QRS >120msec	
▪ Asymptomatic sinus bradycardia (<50bpm), sinoatrial block or sinus pause > 3 seconds – all in absence of negatively chronotropic drugs.	
ECG changes favouring tachyarrhythmias	
▪ Pre-excited QRS complexes – WPW syndrome	
▪ ↑QT_c / ↓ QT_c	
▪ Brugada syndrome: RBBB pattern + ↑ST V1-V3	
▪ Q waves suggesting myocardial infarction	

▪ Non sustained VT	
▪ Arrhythmogenic RVD: Negative T waves in right precordial leads (V1-V3) + epsilon waves + prolonged S upstroke in V1-V3	

C **Actions**

No high risk clinical and ECG features	Consider discharge.
High suspicion of vasovagal syncope (supporting features '3Ps' – Posture, Provoking factors and Prodromal symptoms.	Consider discharge.
Presence of one or more	Inpatient specialist cardiovascular assessment
Presence of non highlighted high risk features	Admit, investigate and treat.
>65 years with TLoC without prodromal symptoms.	Inpatient specialist cardiovascular assessment

Distinguishing epileptic from non-epileptic seizures can be difficult, however, most diagnoses are suggested on clinical grounds from evaluation of patient and witness accounts. Overdependence on, or indiscriminate application of, investigations such as brain imaging and routine EEG[11] recordings may be unrewarding & on occasion frankly misleading.

Huge diagnostic value comes from both

what patients and witnesses say about attacks

and also

how patients themselves describe their attacks.

Cause	Prodrome	Posture	Provoking Factors/ Situational Associations	Appearance	Comments
Syncope	**Minutes**	**Upright**	**Heat, Hunger, Fear Restaurants/ Toilets/ Aeroplanes/ Dentists/ Supermarkets**	**Pallor, sweaty, may twitch. Any "confusion" settles within minutes or less**	**Provoking factors common but may not always be recalled. Incontinence can occur.**
Epileptic Convulsion	**None / Seconds**	**Any**	**Rare Flashing lights Hyperventilation**	**Rhythmic jerking, noisy breathing, red or purple colour, confused & amnesic afterwards for many minutes**	**Any time**
Non-Epileptic Convulsion	**Variable (may be hours or days)**	**Any**	**Stress, company Doctors Surgery**	**Eyes closed, Side to side head jerking, Weeping after attack. Not cyanosed, pelvic thrusting, carpet burns to face, injuries can occur.**	**Provoking situations History of psychological stressors/ illnesses common. High frequency events (eg ≥ daily) in otherwise normal brain**

Further features that may alert clinician to diagnosis of non epileptic seizures

Feature in history[9]	Non-Epileptic seizures	Epileptic seizures
Onset < 10 yrs. old	Unusual	Common

Change in semiology	Occasional	Rare
Worse with anticonvulsant drugs	Occasional	Rare
Fits in presence of doctors	Common	Rare
Recurrent "status"	Common	Rare
Multiple unexplained physical Symptoms (esp pain)	Common	Rare
Multiple operations/ invasive tests	Common	Rare
Psychiatric Treatment	Common	Unusual

In addition aspects of how patients talk about their attacks may be informative[10]

Features	Patients verbal output	
	Non-Epileptic seizure	**Epileptic Seizure**
Subjective symptoms	avoided / sparse	Volunteered, detailed
Focus on memorable Sx In episodes (First, last, worst)	Difficult tend to generalise	Easy, often made spontaneously
Focus on seizure description	Difficult	Easy
Seizure described by negation (I don't know/ I can't hear/ I can't remember)	Common & absolute "I feel nothing" "I don't know anything"	rarely and usually in context ("I can recall this but I can't recall that")
Danger of situation Serious Consequences	Emphasised more than Seizure symptoms	Not/ or the converse

INVESTIGATIONS – LIMITATIONS AND PITFALLS

EEGs

- About 50% of patients with epilepsy show epileptiform discharges on a single EEG. And 50% don't
- 1 in 200 healthy adults without epilepsy have frank epileptiform discharges on EEG[12]
- Only epileptiform discharges should be considered a diagnostic indicator.
- 10 – 20% of healthy population have non-specific changes
- Positive predicative value of abnormality is reduced by low prevalence of condition in population under study (i.e. if most patients referred for EEG don't have epilepsy the likelihood of an abnormal EEG being a "false positive" climbs)

Scanning

- Normal in idiopathic generalised epilepsies & 70% of focal epilepsies.
- Abnormal scans neither substantiate a diagnosis of epilepsy nor a cause of seizures

ECG

- Mandatory for all "blackouts? cause"

Blood Tests

- Basic haematology & biochemistry (including LFTs & glucose) - highly recommended for all "blackouts? cause" other than simple faints

Indications for referral

- Suspected seizure disorder
- Diagnostic uncertainty despite adequate witness account
- On-going seizures despite drug treatment
- No neurologist review ever
- No neurologist review in last 2 years **and the patient or you would like one**
- Female contemplating pregnancy not under secondary care

Return to Contents Page

If you suspect a syncopal event secondary to cardiac dysfunction then referral to cardiology is strongly advised (rather than referral to neurology or synchronous referral to neurology and cardiology)

Prior to referral:

- Advise not to drive (it is the event itself and the DVLA that dictate fitness to drive and not investigation results)
- Baseline Bloods including FBC, renal & liver FTs & glucose & ECG (enclosing ECG with referral)
- Give patient / partner information on first aid / safety (SaTH Suspected Seizure Information Handout - Appendix IID)
- Do **not** start AEDs without discussing with neurologist first

In particular DO NOT START valproate in women of child bearing potential

MAKING A REFERRAL

Seizures are considered urgent and therefore not suited for Choose and Book appointment systems. Provided you and your patient are happy with the choice of SATH you are strongly advised to:-

Refer seizures or suspected seizures by Fax to

01743 261288(RSH)
01952 282873 (PRH)

See Appendix IIB

FOR FLOW CHART/ CHECKLIST/ PROFORMA

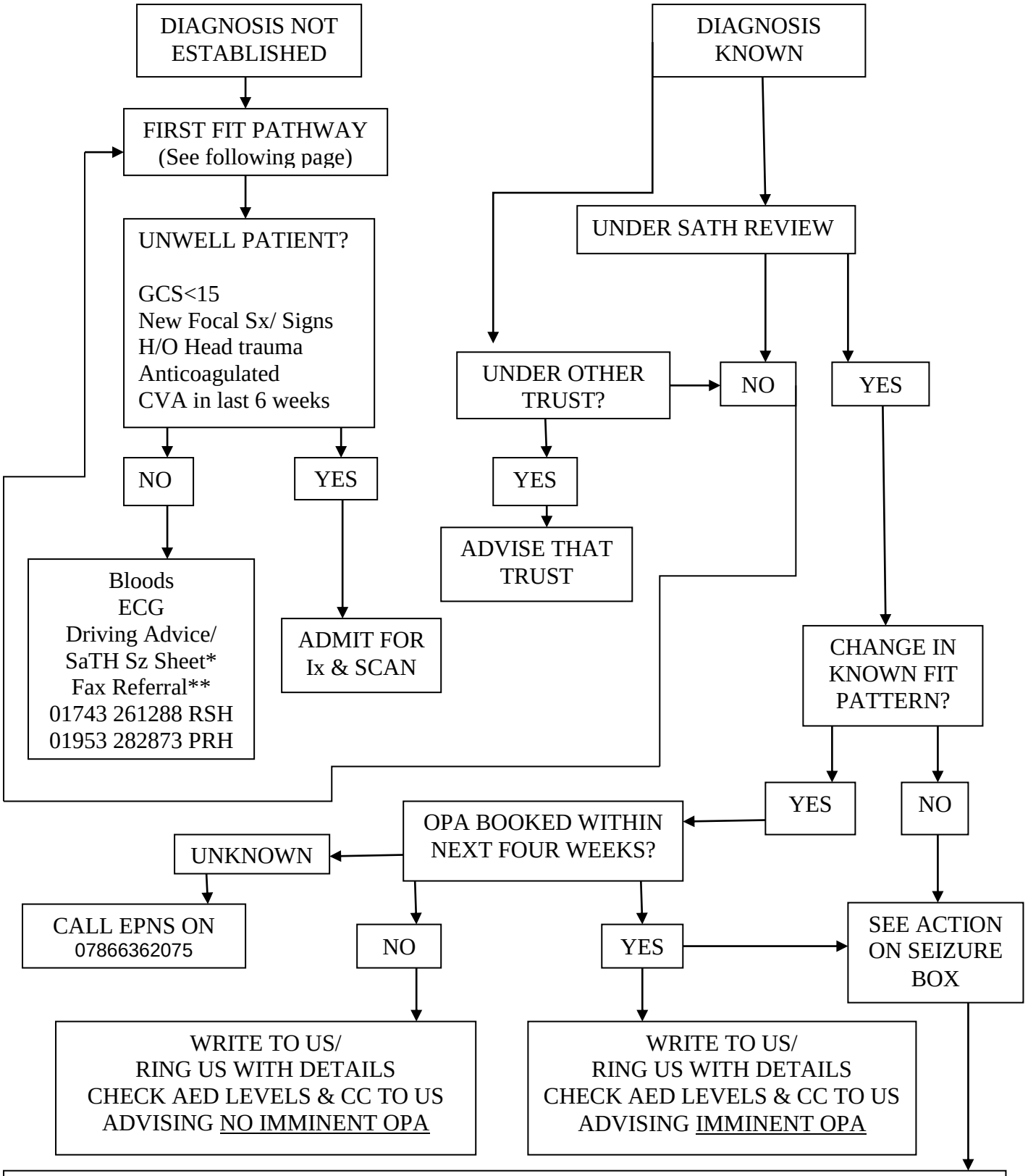
If you subsequently send pre-faxed referral by “snail” mail it would help enormously if you could indicate that the referral has already been faxed.

Tips on referral below

[NB: EPNS – Epilepsy Nurse Specialist]

Return to Contents Page

Seizure Referral Pathway



ACTION ON SEIZURE:

1. Consider causes:
    a. Patient –related. Sleep pattern (deprivation/alteration), food (irregular, stimulants), intercurrent illness fever, vomiting, diarrhoea, stress (personal, professional)
    b. Pill related – compliance (commonest cause): check prescription history/ side-effects /AED levels for concordance (& cc to SaTH clinician)
2. Action:
    a. Address cause if identified and copy outcome to SaTH clinician
    b. If no cause found request patient keeps seizure diary & brings diary to next OPA

First seizure- Recommended Pathway

Patients with a (suspected) first fit should be streamed into 2 groups:

1. First seizure in someone who has recovered fully and is otherwise well with no focal symptoms or signs.

i.e. well, no focal neurology, low suspicion of malevolent underlying lesion

Advise not to drive

Baseline Bloods (including LFTs & glucose) & ECG

Give to patient / partner SaTH Information Handout for Suspected Seizures*

Refer by fax (01743 261228 RSH or 01952 282873 PRH) **

* SaTH Suspected First Seizure Information Sheet – Appendix IID
** First Fit Referral Proforma – Appendix IIC

2. First seizure in someone who isn't well, hasn't recovered fully, has focal symptoms or signs, has no responsible adult to care for them

i.e. unwell, febrile, altered mental status, persistent or severe headache, neurological signs, recent head trauma, anticoagulated / bleeding diathesis, possible underlying malevolent brain lesion (history of cancer/ HIV), infection, past history of stroke/TIA

Admit to hospital for assessment, bloods, ECG +/- CXR and urgent head scan.

Medical assistance should be sought & provided if:-

a. The person has been injured in the seizure
b. Breathing difficulties occur after the seizure
c. Sequential seizures occur without recovery in between
d. The seizure exceeds five minutes & the usual duration is unknown
e. It is the person's first seizure (or the person is not known to have seizures)

Helpful Information for you, your patients, & their loved ones

Downloadable free handout for patients regarding emergency first aid can be found at:
http://www.epilepsysociety.org.uk/10-first-aid-steps-when-someone-has-convulsive-seizure

The National Society for Epilepsy
Chesham Lane, Chalfont St Peter
Bucks.SL9 0RJ
Tel 01494 601 300
www.epilepsysociety.org.uk/

Epilepsy Action
Tel: 0808 800 5050
https://www.epilepsy.org.uk

Return to Contents Page

Women & Epilepsy

Discuss & record issues surrounding
Contraception, pre-pregnancy planning, conception, child care, breast feeding, & menopause[12]
Review diagnosis, drugs, doses.
Recommend referral if contemplating pregnancy

Teratogenicity	2-10% risk on AEDs (Gen population 2-3%) *No consensus on optimal dose/timing
Folic Acid supplementation*	5mgm preconceptually & throughout pregnancy

Discuss contraception with women of child bearing potential:

Contraception

Enzyme Inducers OCP efficacy Reduced	**Non-Enzyme-Inducers** OCP efficacy Not Reduced
(50, 75, or 100 µgm oestrogen pill needed*)	
Carbamazepine	Valproate
Topiramate**	Lamotrigine
Phenytoin	Gabapentin
Primidone	Zonisamide
Phenobarbitone	Levetiracetam
Oxcarbazepine	Pregabalin
Perampanel >12mg/day	Ethosuximide
Rufinamide	Clobazam/Clonazepam
Eslicarbazepine	Lacosamide
	Perampanel < 12mg/day
	Zonisamide

Not an enzyme inducer though **does reduce efficacy of OCP at doses > 200mg/day

* Even on higher oestrogen pills, full contraceptive efficacy cannot be guaranteed but failure rates appear lower (~7%) than barrier methods alone (15-20%). Contraception can be enhanced by combining higher oestrogen OCP with double dose progestogen content and continual use.

Progestogen only pills (POPs) are not recommended with enzyme inducing AEDs. Non-enzyme inducers probably have little effect on POPs but the reduced efficacy of POPs mean that this form of contraception (without additional barrier methods) is not recommended. Depo-Provera now recommended 12 weekly with inducing AEDs. Norplant not recommended with enzyme inducing AEDs. IUDs and barriers contraception are unaffected by anticonvulsants.

Anticonvulsants (AEDs) – Do's & Don'ts

Do:

1. Seek advice if:
   - dosing schedule unclear
   - side-effects occur, persist, & are intolerable
   - seizures worsen
   - drug interaction suspected
   - **Patient on Vigabatrin – referral of such patients STRONGLY ADVISED**
   - Female patient of child bearing potential on valproate*
   - Patient on AEDs and planning a pregnancy
   - Patient wishing to come off AED

3. Check AED drug levels if:

- Compliance uncertain (or poor response despite seemingly adequate dosing)
- Pharmacokinetic drug interaction suspected
- To guide dose escalation

NB The end point is seizure control with tolerable/ no side effects. No indication to routinely check levels once this state is reached, though knowledge of level as future reference point can have utility.

a) Phenytoin

Check phenytoin levels before altering dose (equilibration takes 2 weeks)
Alter daily phenytoin doses by 25mgm only (zero order pharmacokinetics)

3. Recommend folic acid supplements (5mg/day) for fertile women on AED especially valproate
Register (with patients consent) all pregnancies in women on AEDs with the UK Epilepsy Pregnancy Register – Tel: 0800 3891248

4. Think about bone health in at risk patients on enzyme inducing AEDs & Valproate
(www.shef.ac.uk/frax) (https://riskcalculator.fore.org/)

Don't

1. Routinely check AED levels if seizures are controlled and side-effects negligible
2. Alter AED dose if seizures are controlled and side-effects negligible
3. Commence women of child bearing potential on Valproate without prior neurological advice ⁎
4. Commence adults on Vigabatrin without prior neurological advice
5. Withdraw / alter previously successful AED therapy without prior neurological advice
6. Forget to advise patients of potential side-effects of AEDs
    - Dizzyness, sedation, forgetfulness – virtually all, often settle over time
    - Weight gain, tremor, teratogenesis including cognitive delay – valproate
    - Rash – Lamotrigine, Carbamazepine
    - Bruising, sore throats, mouth ulcers – Carbamazepine (urgent FBC)
    - Weight loss, peripheral tingling, urinary calculi (topiramate & zonisamide)
    - Hirsutism, facial coarsening, & gum hypertrophy - Phenytoin

⁎ Sodium valproate is associated with an increased risk of spina bifida in infants conceived during maternal use **(**1-2% compared to background risk of 0.2 – 0.5%). Other additional adverse physical &cognitive sequalae in offspring born to mothers taking valproate at conception & during pregnancy have been reported and it is imperative that these are discussed in full with all women of child bearing potential prior to commencing this drug. Additional side-effects of which all patients should be advised before commencing valproate include the potential for weight gain, tremor, and occasionally hair loss which is revers

Valproate does not reduce the efficacy of the oral contraceptive pill.
It is **strongly recommended** that such discussions are documented in medical notes.
The MHRA has published guidelines to assist clinicians prescribing valproate[14]

Return to Contents Page

Switching AED Formulations[15]

The Medicines and Healthcare Products Regulatory Agency (MHRA) has issued new advice about oral AEDs and switching between different manufacturers' products of a particular drug. This includes switching between branded original and generic products, and between different generic products of a particular drug. The MHRA notes that different AEDs vary considerably in their characteristics, which influences the risk of whether switching between different products may cause adverse effects or loss of seizure control.

Following a review of the available evidence, the MHRA's Commission on Human Medicines has classified AEDs into 3 categories:

 Category 1 – phenytoin, carbamazepine, phenobarbital and primidone

For these drugs, doctors are advised to ensure that the patient is maintained on a specific manufacturer's product.

Category 2 – valproate, lamotrigine, perampanel, retigabine, rufinamide, clobazam, clonazepam, oxcarbazepine, eslicarbazepine, zonisamide and topiramate

For these drugs, the need for continued supply of a particular manufacturer's product should be based on clinical judgement and consultation with patient and/or carer, taking into account factors such as seizure frequency and treatment history.

 Category 3 – levetiracetam, lacosamide, tiagabine, gabapentin, pregabalin, ethosuximide and vigabatrin

For these drugs, it is usually unnecessary to ensure that patients are maintained on a specific manufacturer's product unless there are specific concerns, such as patient anxiety or risk of confusion or dosing errors.

Author's comments:

A case by case approach is recommended taking into account individual patient characteristics including comorbidities, cognition, seizure control, driving status, lifestyle, & patients views. Minimising disruption wherever possible should be the goal.

Checking AED level(s) for compliance acutely when seizures occur (& randomly on occasion when seizures are controlled) can be informative with respect to medication concordance.

Suggested Drug Schedules for commonly used AEDs can be found in Appendix IIA

Return to Contents Page

Potential Grants / Benefits for People with Epilepsy

Medication-Related

Free prescriptions If you have epilepsy and take epilepsy medicines, you are entitled to free prescriptions in the UK.

Travel – Related

Free bus pass If you would be refused a driving license because of your epilepsy, you may be entitled to free or reduced price bus travel.

Disabled Persons Railcard reduced rail fares in England, Scotland and Wales.

Work-Related

Access to Work for employment related expenditure.

Employment and Support Allowance (ESA) for people with an illness or disability that prevents work.

Personal Independence Payment (PIP) A benefit to help with some of the extra costs of living with a long-term health condition or disability. PIP is not currently available in Northern Ireland.

Care-Related

Attendance Allowance for personal care for those with a disability, aged 65 or over.

Carers Allowance for those caring for a person with epilepsy and substantial care needs.

Disability Living Allowance (DLA) for those looking after a child with a disability or health condition.

Disabled Facilities Grant for those needing to make changes to their home because of their epilepsy.

Sourced from:

https://www.epilepsy.org.uk/info/benefits

References

8. Juarez-Garcia A.et al. http://dx.doi.org/10.1016/j.seizure.2006.08.005
9. Reuber M,Elger CE.Psychogenic nonepileptic seizures: review and update.Epilepsy Behav 2003;4:205-16
10. Schwabe M, et al. Differential diagnosis of seizure disorders: a conversation analytical approach. Soc Sci Med 2007;65(4):12-24
11. Benbadis SR, Tatum WO. Overinterpretation of EEGs and misdiagnosis of epilepsy. J Clin Neuophysiol 2003;20:42-4
12. Gregory RP, Oates T, Merry RTG. EEG epileptiform abnormalities in candidates for aircrew training. Electroencephalog Clin Neurophysiol 1993;86:75-7
13. https://www.nice.org.uk/guidance/cg137/chapter/1-Guidance
14. https://www.gov.uk/drug-safety-update/valproate-and-of-risk-of-abnormal-pregnancy-outcomes-new-communication-materials
15. https://www.nice.org.uk/guidance/cg137/evidence/evidence-update-544389949

Return to Contents Page

MULTIPLE SCLEROSIS

(MS Trust www.mstrust.org.uk)
(MS Society of Great Britain and Northern Ireland Tel: 0808 800 8000 / www.mssociety.org.uk)

An inflammatory disease of the Central Nervous System (CNS)

Prevalence ~ 1:800 Usual onset 18 – 55 years

Early accurate diagnosis can be difficult and few neurological symptoms if "Googled" by patients will not suggest MS as a possible cause. Statistically speaking most patients with transient or scattered sensory symptoms will not have MS and whilst it will be appropriate to accept the possibility of "inflammation" if the presentation is strongly supportive of such, much anxiety can be avoided by NOT suggesting a diagnosis of MS (or indeed any other neurological diagnosis) unless strong evidence exist to justify such potentially distressing news. If unsure it is fair (and truthful!) to say so & explain why the uncertainty exists and agree a plan.

In general terms:

1. MS symptoms develop in otherwise well individuals
   a. (systemic features unusual)
2. Focal symptoms evolve (spread/worsen) typically over days/weeks
3. Distribution of symptoms reflect central (rather than peripheral) origin
   a. (i.e. not usually confined to root or peripheral nerve territory)
4. History of a previous episode(s) of CNS inflammation may be found
   a. Non-specific isolated/recurrent dizziness, tingling <24 hours insufficient
5. Commonest alternative diagnosis is anxiety based/ functional symptoms
   a. View symptoms in context of co-morbidity
6. Persistent neurological symptoms in the absence of objective clinical evidence rarely lead to the development of MS*

* Boster A, et al.Mult Scler.2008;4(6):804-8

If you suspect a patient has an MS related problem the following action is suggested:-

Diagnosis not established

Situation	Recommended Action
Patient has focal worsening symptoms	}
OR	Fax Referral to 01952 282873
Patient has disabling symptoms	}
Patient has static non-disabling symptoms	}
OR	Refer in usual manner
Patient has improving symptoms	}

Diagnosis has previously been established

For acute deteriorations resulting in loss of function rendering independent existence in existing circumstances actually or potentially unsafe – admit.

For less severe deteriorations -

Inform MS nurse service 01952 641222 Ext 4430 or: 07971 497902 / 07974 30541

Management

Acute relapses

- May not "need" treatment at all if not functionally incapacitating since:
    - High dose short course steroids may accelerate rate (but not degree) of recovery and though low risk are not zero risk therapy.
    - Repeated courses of oral steroids for minor/ tolerable relapses are **not** encouraged
    - Prior to considering steroids seek and manage acute infection as potential cause of deterioration
- Patient reporting of suspected relapses to MS Nurse service is encouraged

Maintenance therapy

Disease Modifying Treatments (DMTs)

- Reduce relapse frequency by ~ 1/3 – 1/2
- Indicated for relapsing remitting MS with relapses
- **May** help secondary progressive MS if dominant decline is caused by relapses
- Established benefit on primary progression remains uncertain,
- Specialist referral for advice on secondary progression recommended
- Agents currently given via injection or oral routes.
- Optimal DMT individualised according to patient /disease characteristics.

Symptomatic Therapy

- Best delivered in multidisciplinary context
- Neurological guidance recommended
- Neuropathic pain drugs (Amitriptyline, Duloxetine, Carbamazepine, Gabapentin, Pregabalin) less likely to help non-neuropathic pain and should not be continued if ineffective. Opiates best avoided if possible
- Neuropathic drug schedules suggested in Appx IIC
- Physical therapy helpful as adjunct to pharmacotherapy for spasticity

- Consider comorbid mood & sleep disturbances (very common)
- Bowel & bladder care paramount & MS nurse service available to advise & coordinate as necessary

Denise Cooper	Multiple Sclerosis Specialist Nurse	Cross Site and Community
Mobile:	07974930541	
24hr voice mail	01952 641222 Ext 4430	
Kate Watkiss	Multiple Sclerosis Specialist Nurse	Cross Site and Community
Mobile	07971497902	
24 hr voice mail	01952 641222 ext 4430	

Carol Napper	Clinical Specialist Physiotherapist (in the management or Spasticity)
RSH	Mon-Thursday
Tel:	01743 261105
Email:	carol.napper@sath.nhs.uk

Return to Contents Page

PARKINSON'S DISEASE & RELATED DISORDERS

Parkinsonism is a clinical term encompassing varying degrees of rigidity/ bradykinesia/ rest tremor

Causes:

- Idiopathic Parkinson's Disease (IPD) – most common
- Drug related Parkinsonism (many dopamine antagonists)
    - Antiemetics, vestibular sedatives, major tranquillisers
        - Maxolon, Stemetil, Stugeron, Stelazine
- Neurodegenerative disorders such as multiple system atrophy (MSA), progressive supranuclear palsy (PSP). Typically, these patients have symptoms & signs beyond those found in IPD.

Diagnostic clues for Parkinsonism

Symptoms

- Tremor at rest, micrographia, difficulty rolling over in bed, shoulder stiffness, "unhappy" face, problems doing up buttons, dragging one leg, slowing down.
- History of dopamine antagonist/ antipsychotic therapy ingestion implies drug induced/aggravated parkinsonism

Signs

- Reduced (unilateral) arm swing when walking, asymmetrically reduced rapid shoulder shrug,
- Micrographia, asymmetrical "pill-rolling" rest tremor*, rigidity, bradykinesia (a reduction in amplitude, speed or both of repetitive movements such as opposing finger and thumb. (*Postural tremor can also commonly occur in IPD)

IPD was only confirmed pathologically in 76% of patients diagnosed by Neurologists[12]. Misdiagnosis rates in primary care are likely to be higher and may approach 50%. In view of this the following action is strongly recommended.

If you suspect a patient has Parkinsonism then:-

1. Review drug records and if possible taper / stop dopamine antagonists/ refer back to psychiatry if on psychotropic Rx for review.
2. If not possible stop offending dopamine antagonist. If not switch to Domperidone, or Ondansetron.
3. Refer to Neurology if < 70 of Geriatricians if > 70
4. Please do not start medication for Parkinsonism prior to referral.

If medication considered urgent please phone (07791487444 / 01743 261105 RSH or 07791487444/ 01952 641222 PRH) for advice

Once diagnosis confirmed by neurologist a drug schedule will commonly be recommended.

Return to Contents Page

Drug treatment of idiopathic PD remains somewhat controversial reflecting the lack of universally accepted evidence regarding the “right” treatment.

Use of non-ergot based dopamine agonists is standard owing to concerns regarding fibrotic side-effects of ergot agonists especially with respect to “cardiac valvulopathy”[13a]

LevoDopa Resistant Features of Parkinson’s disease

Some patients with long-standing disease may eventually develop problems that are unresponsive to L-Dopa. This is not invariable, however, & need not be raised with patients in early stages of the illness or those with comorbidities that are less likely to reach advanced stages.

These can include:

- On-freezing (feet getting stuck to ground in the medicated “on” state)
- Loss of postural reflexes leading to falls
- Speech and swallowing difficulties

Awareness of the dopa-resistant nature of these problems is vital since

- Further escalation of drugs will only increase adverse side-effects
- Other strategies may be required (OT / Physio/ SALT input)
- Other possible causes should be considered (postural hypotension / visual disturbance / ill-fitting carpets/ footwear / oesophageal disease etc)

Non-Motor Symptoms of Parkinson’s disease

Non-motor symptoms (NMS) are BIG problems. They can be clustered by thinking of the culinary delight known as Big MACS

MACS:

Mood disturbances & anxiety

Autonomic symptoms (constipation, faints, urinary & erectile difficulties)

Cognitive symptoms (hallucinations, dementia)

Sleep disturbances and others.

These are increasingly recognised as potent sources of morbidity and NMS adversely affect quality of life.[13b-d]

Dopaminergic treatment for these non-motor symptoms (other than those directly related to “on” or “off” states) may be helpful though responses are variable & can be incomplete.

Awareness and active pursuit of NMS may impact positively on both PD patients and carers.

Mood

(Depression, Anxiety, Delirium, Dementia)

Depression:

- Affects ~50% of PD pts.
- Poor sleep, lack of interest, and anorexia suggestive of diagnosis. Apathy is an important differential.
- Usually not responsive to L-Dopa. Tricyclics / SSRI (generally not combined) can help

Anxiety

- Very common
- See Functional & Mood Disorders Chapter for more (P42-46)

Autonomic Dysfunction

Dizziness /Postural hypotension

Withdraw antihypertensives

Add salt to diet and encourage fluid intake of at least 2 litres/day

Elevate head of bed

0.1 mg morning Fludrocortisone (increasing to 0.3) but supine hypertension may be a problem and hypernatraemia/ hypokalemia may occur. Midodrine can help 2.5 – 10 mg tds. Supine hypertension is principal concern

Drooling

- SALT input / Cherry stones/ amitriptyline/ intra-oral atropine eye drops/ btox/ more dopaminergic Rx

Constipation

- Check not side-effect of medication
- Typically due to slow transit time so bulking laxative make it worse.Movicol, Senna , Bisacodyl can be tried.May need Glycerine/ Bisacodyl suppositories or phosphate enemas if impacted
- May be unrelated to PD!

Bladder dysfunction

- May be unrelated to PD!
- Only attributable to PD if PD diagnosis > 10 yrs. old
- Common in Multiple System Atrophy
- Consider non-PD causes if PD diagnosis secure but < 10 yrs.
- Referral for urological opinion preferable to “trial of treatment”
- Urinalysis (& MSU if abnormal), serum creatinine, KUB USS, with pre & post micturition bladder USS recommended pre-referral and ISC considered if post mic volume exceeds 100 ml
- Anticholinergic therapy e.g. tolterodine can help reduce frequency but can result in confusion

Erectile dysfunction

- Phosphodiesterase inhibitors (if not on nitrates or hypotensive)
  Eg Sildenafil can help though hypotension can occur
- Serum testosterone worth checking and correcting if low

Return to Contents Page

Cognition

Delirium

- Abrupt onset with disordered attentional and fluctuation
- Risk factors include previous episodes, older age, medical comorbidities, prior cognitive impairment, &PD! Common triggers can be considered under acronym “Ms Dippeo” (**M**eds, **S**epsis, **D**ehydration, **I**mpaction, **P**ain, **P**yrexia, **E**lectrolytes, **O**ther medical problem)
- Management – address and optimise precipitating factors. See Appendix V for more

Cognitive Impairment / Dementia

- Seen in ~80% a decade post diagnosis, though mild in many. Subcortical in type, slowness in thought and reasoning, inflexibility in task switching especially for new or unfamiliar tasks.
- Seen as consequence of PD, Dementia with Lewy Bodies (DLB), Alzheimer’s disease (AD), or Vascular dementia (VaD)
- DLB diagnosed within first year of parkinsonian Sx with
    - Fluctuating alertness,attention,cognition
    - Visual hallucinations
    - Parkinsonism (minimal/absent tremor)
    - REM-sleep BD
    - Marked sensitivity to dopamine antagonists

Sleep disturbance

- Multifactorial aetiology. .
- Potential causes include night time stiffness due to nocturnal parkinsonism/dystonia, & nocturia all disrupting night sleep. Optimising these is initial Mx.

Treatment

All dopaminergic agents (levodopa & all dopamine agonists)

Excessive day time sleepiness can occur and patients advised accordingly, especially patients on Dopamine agonists. Particular reference to driving and operating machinery should be stressed and these activities discontinued until sleepiness ceases.

Levodopa

Early common: Nausea & Vomiting – Rx domperidone 10mg-20mg tds*
Early less common: Postural hypotension, sleep attacks
Chronic: fluctuations, dyskinesias. Neuroleptic malignant syndrome on sudden withdrawal.

Domperidone

*MHRA publication regarding cardiac complications unaccompanied by practical suggestions regarding safer alternatives. The author suggests explaining very small risk to patients of harm (< 1 in 100) restricting use to symptomatic requirement for shortest time needed in lowest effective dose, checking ECG in patients > 60 before and after initiating therapy for changes in conduction times, and asking patient to be vigilant about and report any new cardiac symptoms. Alternative suitable antiemetics include ondanseron and cyclizine.

Dopamine Agonists (pramipexole, ropinirole, rotigotine, apomorphine)

Early common: Nausea & Vomiting – Rx domperidone 10mg-20mg tds

Early less common: Postural hypotension, hallucinations, sleep attacks – patients must be forewarned and driving cease should these occur

Behavioural changes including disinhibition, hypersexuality, hoarding, gambling, excessive spending, paranoia, hallucinations especially in elderly

Leg swelling

Ergots *(cabergoline, pergolide) – seldom used. Refer if you have patients on this who are not already under secondary care but do NOT stop drug.*

Monoamine Oxidase Inhibitors

Dry mouth, hypotension, alerting effect, hallucinations, confusion (esp. older patients), weight loss, joint pain

Avoid if active peptic ulceration present.

With SSRIs v small risk of serotonin syndrome (dyskinetic confused hypertensive patient). Rx stop offending agent).

NB No cheese and wine reactions with MAOI (B) in recommended doses.

Amantadine

Nausea, leg swelling, rash (livedo reticularis), insomnia (last dose no later than 4 pm), confusion, psychosis, seizures

Catechol-o-methyl transferase inhibitors (COMTI)

Urine reddy brown. Nausea, abdominal pain and diarrhoea. Dyskinesia may appear or worsen.

Anticholinergics

Hallucinations, confusion (esp. older patients), dry mouth, dry eyes, constipation, urinary hesitancy. Contra-indicated in closed angle glaucoma, prostatism, pre-existing cognitive impairment

Parkinson's disease and Visual Hallucinations

These are common and in fact are a diagnostic feature of Lewy body pathology, occurring in ~ 40% of patients with PD.

They are usually rich in detail involving people or animals (especially insects) and in the early stages at least a degree of insight is retained.

Risk factors for developing visual hallucinations in PD are age and cognitive decline though other factors that should be considered are:-

- Metabolic derangement including infection & dehydration
- Drugs (anticholinergics, CNS acting agents including dopa agonists)
- Other non-PD brain insults (rare)

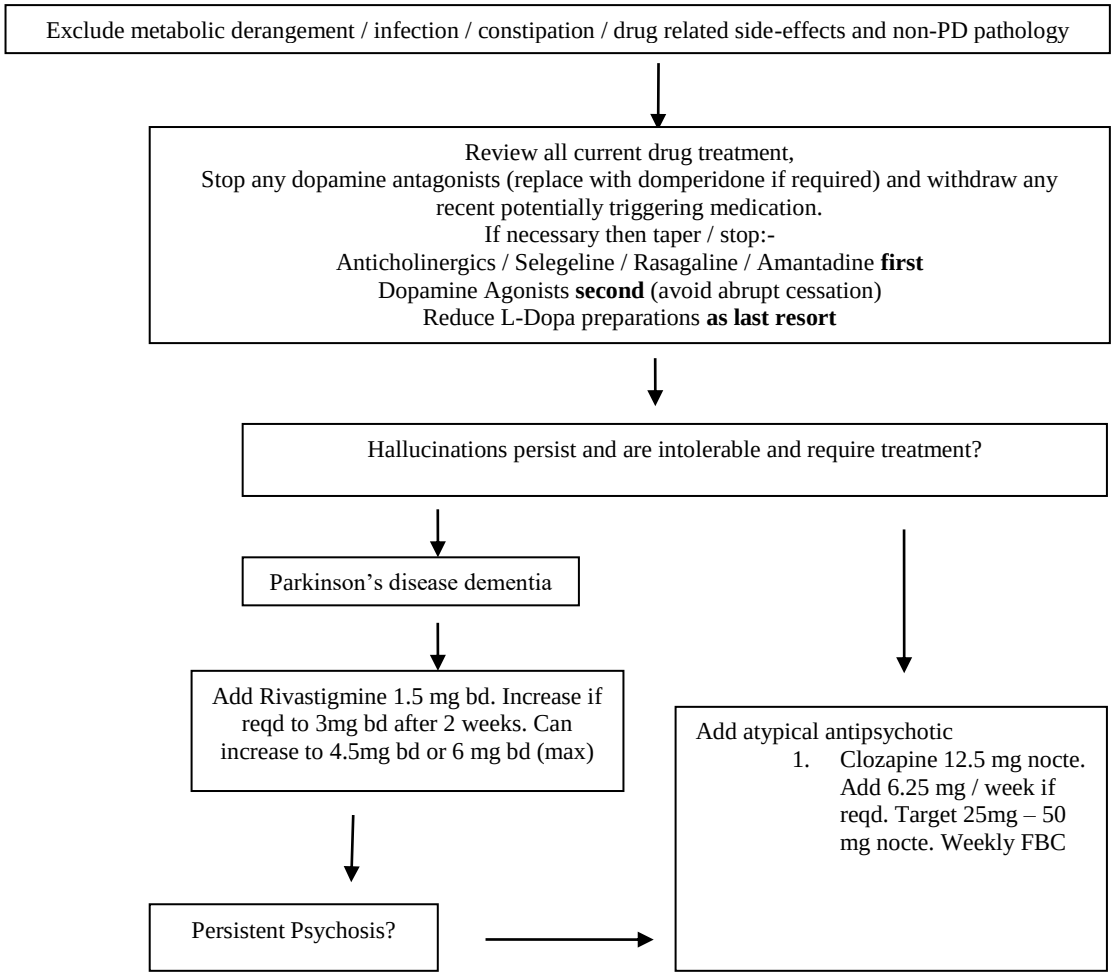
Once these factors have been actively sought and excluded, symptoms can usually be improved by manipulation of Parkinson's disease therapy (in a downward direction) though this commonly comes at the cost of worsening parkinsonian motor features.

Drugs that can be tried to actively suppress hallucinations include Rivastigmine (which can also help especially if a background dementia co-exists) and Clozapine (See Appendix IIIB)

.

Referral to Neurology and Psychiatry for guidance on such drug manipulation is strongly advised

Parkinson's disease and Visual Hallucinations[13e]



References

12 Hughes et al. (1992) Journal of Neurol Neurosurg Psychiatr; **55**:181-184

13a. Antonini A et al. (2007) Lancet Neurology; **6:**826-29

13b. Schrag A, et al (2000) Journal of Neurol Neurosurg Psychiatr; **69**:308-12

13c. Aarsland D, et al (2000) J Am Geriatr Soc;**48**: 938-42

13d. Findley L,et al (2003) Mov Disord;**18**:1139-45

13e. Poewe W. (2008) Practical Neurology; **8**(4):238-241

Return to Contents Page

ESSENTIAL TREMOR

A POSTURAL (generally symmetrical) tremor (without rest component) usually affecting the hands often progressing slowly over years/decades. Differential in includes dystonic tremor, often asymmetrical or task specific and may include family history.
A positive family history and tremor relief from alcohol ingestion are helpful diagnostic clues but their absence does not exclude the diagnosis

.

Treatment

Often unsatisfactory although doses must be increased to maximum tolerated levels before it can be concluded that they don't work for individual patients. These agents are most likely to confer benefit.

- Propranolol (provided no contra-indications exist) see Appendix IIIA for schedule
- Primidone see Appendix IIIA for schedule
- Both
- Atenolol has little effect, Sotalol is active
- Other drugs have been tried with variable (usually disappointing) results but are worth a trial in resistant cases include: Gabapentin, Topiramate, Clonazepam, Alpraziolam,
- Botulinum toxin for head & neck tremors & OT for weights and devices to dampen down tremors.
- Referral for consideration of Deep Brain (Thalamic) Stimulation for severe disabling drug resistant cases is recommended for patients willing to consider "brain surgery."

FOCAL TREMORS AND DYSTONIAS

Troublesome focal tremors of the head, focal dystonias including cervical dystonias (torticollis etc), hemifacial spasm, are generally resistant to drug & non drug therapies, though clonazepam and Trihexyphenidyl may help if tolerated (Appx IIIB)
In selected cases these conditions may be helped by Botulinum toxin and referrals to the Botulinum Toxin service are welcomed.

Botulinum toxin pamphlets can be provided on demand for you / your patients (01952 641222 PRH/ 01743 261105 RSH).

Return to Contents Page

NEUROPATHIC PAIN

For resistant /chronic / patients in whom investigation has previously excluded remediable causes for neuropathic pain it is strongly advised that referrals are directed to Pain Services and NOT Neurology

Definition: pain initiated or caused by a primary lesion or dysfunction of the nervous system. The pain may be burning or cold, associated with numbness, tingling, or itching, or stabbing & electric shock-like. Unlike nociceptive pain, neuropathic pain serves no useful function. It is often debilitating and relatively refractory to therapy.

Clinical features: burning or cold, associated tingling, numbness, itching
May have paroxysmal electric shock type stabs
Immediate or delayed following initiating event
Distorted (unpleasant) sensory perception common
Associated features of causative neurological disorder(s)

Competing causes of pain in individual patient extremely common e.g, neuropathic pain of central and peripheral nervous system aetiology with additional musculoskeletal pain.

Amitriptyline 10 mgm nocte increasing by 10 mg / week until efficacy or side-effects intervene aiming for 1.5 mg/kg if required

or

Carbamazepine (SR)

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
200 mgm od	200mg/ week	400 mg bd	800mg bd	200mg / 2 weeks

Potential Side-effects: dizziness, sedation, reduced effectiveness of low dose oral contraceptive pill (see above)
If developing rash, unexplained bruising, mouth ulcers – see GP for review and full blood count
Or

Gabapentin (t.d.s)

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
100 mgm mane	100mg / week BNF	300 mg tds	900 mg/tds	100mg / week

Potential Side-effects: sedation

or

Pregabalin

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
25 mg od	25 mg / week	75 mg bd	150 mg bd	25 mg / week

Potential Side-effects: dry mouth, constipation, dizziness

in that order, unless contra-indications exist, for neuropathic pain

SSRIs are less effective. Paroxetine more useful for analgesia than citalopram or fluoxetine.

Return to Contents Page

FUNCTIONAL AND MOOD DISORDERS

Introduction

- Around one third of all neurology referrals are for patients with symptoms that are wholly or largely unexplained by "physical" disease.
- Around one quarter of all neurology referrals are for patients in whom a diagnosis of anxiety or depressive disorder can reasonably be made.
- Numerically, therefore, this group constitutes the largest "diagnostic" category of patients referred to neurology services.

Terminology – a source of confusion

- ICD-10 Classification *(For DSM-IV definitions see end of chapter)*
    - Somatoform disorders:
        - Somatisation disorder
        - Hypochondriasis
        - Conversion disorder (dissociative disorder)
        - Others

Alternative and perhaps more practically useful terms (that may also be perceived in less negative terms by patients) include:

- Functional Disorder
- Medically-unexplained Disorder

These terms have the advantage of describing a mechanism, not an aetiology, side-step an illogical debate about whether symptoms are in the mind or the brain, allow for the possibility of improvement, and can be easily used with patients[16].

Given the

- frequency of functional symptoms,
- the overlap with symptoms of mood disturbances of anxiety and depression (see below),
- the stigma of psychological illness,
- the explosion of access to medical information, and
- the "medicalisation" of society in general

It is to be expected that an increasing volume of primary and secondary care workload will involve the recognition and management of functional illness.

- Anxiety disorders
    - Generalised anxiety disorder
    - Panic disorder
    - Phobic anxiety

Occurrence	Behaviour	Cognitions	Somatic Sx
persistent	agitation	worry	persistence
episodic	escape	fear of sx	episodic
situational	avoidance	fear of sit	on exposure

    - Affective disorders
        - Depression disorder
        - Mania and bipolar disorder

Box 1

Diagnosis of Depression **(≥5/9 for ≥ 2 weeks)**

Core Symptoms

Must include 1 and / or 2

- Low mood most of time
- Loss of interest, motivation and pleasure

Other Symptoms

- Reduced concentration
- Fatigue
- Sleep disturbance
- Appetite / weight change
- Guilt/worthlessness
- Agitation / slowing down
- Suicidal ideation

Box 2

Diagnosis of Generalised Anxiety Disorder

Psychological Symptoms

1. Peristent worry hard to control ≥ 6 months
   Plus ≥3/6 of the following
2. Restlessness/on edge
3. Insomnia
4. Fatigue
5. Irritability
6. Poor concentration
7. Tense muscles

Much of what follows below has been distilled from an article[16] by Dr Jon Stone, Consultant Neurologist, and is reproduced with permission.

Recognition

- Given the high frequency (see above) and diversity (see below) of functional symptoms a high index of suspicion is an appropriate start.
- A past history of previous / on-going functional syndromes should be actively sought (Eg irritable bowel syndrome, chronic cough, fibromyalgia, dysmenorrhea, atypical chest pain, post-viral fatigue, globus, other functional neurological symptoms)

Evaluation

- List current symptoms and ask about fatigue, pain, sleep, and concentration. The greater the number the greater the likelihood that the primary symptom will be medically-unexplained.
- Plot time course. If onset sudden look for triggers
- Ask what patients **can** do, how they spend their time, are there good times?
- Look for dissociation (a feeling of disconnection / detachment with reality), commonly seen in panic attacks and persisting fatigue.
- What is patient's diagnosis / concern and what are they seeking from consultation
- Seek features of anxiety & depression *en passant* / at end of interview and don't expect to always find such features
- For Depression: ask – do your symptoms get you down then explore features in Box 1 (above)
- For Anxiety: ask – do you symptoms come all at once, what happens, and how do you feel (Box 2)
- Formal DSM criteria are outlined below (P.45)

Return to Contents Page

Caveats

- A past psychiatric psychotic problem does **not** mean the problem is more likely to be functional
- All physical "diagnoses" are not always correct Eg asthma (and epilepsy!)
- "Normal" people experience functional symptoms including those without anxiety & depression
- Early overt questions about depression and anxiety are unlikely to be fruitful. Pick up cues during consultation and leave more direct enquiries to the end.
- Although early life trauma (physical/emotional/sexual) are generally higher amongst patients with functional disorders, such factors are neither necessary, nor likely to be readily disclosed (initially at least) and little is lost by allowing patients to disclose such information in their own time rather than pursuing it actively.

Specific Symptoms

- Non-epileptic Attacks (NEAD: see Chapter 2)
    - 70% thrashing, 25% fall down & lie still (slumping attacks)
    - Semiological differences to epileptic attacks (See page 21)
    - Difference in manner attacks are described by patient (See page 21-22)
    - Caveats
        - Epilepsy can co-exist in 5-20% of patients with NEAD
        - Frontal lobe seizures can look very odd but a very brief (typically seconds) and high frequency often with a nocturnal preference
        - Panic / fear can occur as part of temporal lobe epileptic fits

- Weakness
    - Half of sudden onset; half of gradual onset with pain or fatigue
    - If sudden in onset may arise after physical injury , dissociative seizure, sleep paralysis, or general anaesthetic
    - Inconsistent physical findings on examination (lift legs getting onto couch but not when hip flexion examined)

- Sensory Symptoms
    - Split signs with ipsilateral pain, temperature and vibration/ joint position sense often with ipsilateral weakness, deafness & visual problems
    - Sensory symptoms that stop at groin or shoulder

- Movement Disorders & Gait Disturbances
    - Functional Disorders certainly exist but referral recommended in all

Return to Contents Page

Investigations

- Often inevitable to exclude co-existent “organic” disease and because a functional diagnosis may not be acceptable without back-up of investigations
- Incidental findings on brain MR seen in up to 10-15%
- Age related change on spinal imaging (of any kind) seen in most people > 30
- Normal tests don’t exclude neurological disease!

Are Functional Patient’s malingering?

- Generally no.
- Inconsistent history, avoidance of investigation, direct confession, evidence of inconsistency between reported and actual disability (the crippled man who spent weekends refereeing football matches for example) are pretty strong evidence for malingering but are seen in < 5% of functional patients.

Management

- Cannot begin without a confident diagnosis and if this requires secondary care input such referrals will be welcomed.
- Setting the scene prior to referral – “I suspect your symptoms may be functional but would like a second opinion on that” are preferable to “you may well have MS and you’ll have a scan. If this is negative it’ll probably be put down to stress.”
- Drugs – have a role but seldom a major one with polypharmacy for polysymptomatology a particular risk with drug-related side-effects compounding paient’s own symptoms. Antidepressants help mood, pain, and other symptoms – irrespective of overt mood disturbance but need time (2 months at least, explanation) and if tolerated but ineffective, dose escalation.
- Physical therapy – to improve pain, energy, stamina, self-esteem, confidence. Probably more important that it is continued and adopted into regular life style event than what the exact nature of the activity is. Patient must find it acceptable, enjoyable, and preferably sociable. Yoga / Pilates classes may suit.
- Psychological therapy – self help therapy may be preferred by patients. A plethora of excellent web-based resources (all free) exist (addresses at foot of chapter at Appendix I)
- Formal Referral to Psychological / Psychiatric services for Cognitive Behavioural therapy / other psychiatric intervention can be ordered if appropriate and acceptable to patient.

Return to Contents Page

Prognosis

- For severely affected patients the majority (3/4) will not do well. For this group regular support is important to limit unnecessary hospital referrals and the risk of iatrogenic harm from well-meaning but injudicious investigation and treatment.
- For less severely affected patients recognition, explanation, and support will often result in resolution of functional disorders.
- Adverse prognostic factors: chronicity, concurrent organic disease, multiple somatic complaints, reluctance to accept potential for change in health status, litigation, receipt of financial benefits, old age, sexual abuse
- Positive prognostic factors: recent onset, paucity of additional physical symptoms, concurrent mood disturbance, young, change in marital status **after** diagnosis
- Functional disorders on the basis of frequency, distress, disability, and durability are no less deserving than disorders with more clearly defined pathological substrates and no less rewarding to manage.

DSM-IV Definitions

Depression: Five of Nine (including one or more of first two) out of:

Low mood most of the time	Sleep disturbance
Loss of interest/pleasure in most things	Guilt/Worthlessness
Change in weight / appetite	Reduced concentration
Agitation/ Slowing down	Suicidal ideation
Fatigue	

Persisting and marked for 2 weeks

Panic Attack: Four of the following:

Derealisation/ Depersonalisation	Dizziness
Trembling/Shaking	Choking sensation
Chest pain/pressure	Feeling of going crazy/losing control
Palpitations	Fear of dying
Sweating	Tingling
Breathlessness	Flushes/Chills
Nausea / feeling of imminent diarrhoea	

General Anxiety: Persistent worry difficult to control > 6 months + 3 of

Restlessness/ on edge	Irritability
Insomnia	Poor concentration
Fatigue	Tense muscles

Return to Contents Page

Acknowledgement / Reference

Stone J. Functional Symptoms in neurology .Practical Neurology 2009; 9:179-189

Recommended information sources:

www.neurosymptoms.org comprehensive resource for all on functional symptoms

www.nonepilepticattacks.info information source for non-epileptic attack disorder

http://www.moodjuice.scot.nhs.uk/anxiety.asp self-help anxiety website

www.ntw.nhs.uk/pic/leaflet.php?s=selfhelp patient information on depression and anxiety

Recommended Reading (Clinicians and Patients!)

It's All in Your Head. Author::Suzanne O'Sullivan

Return to Contents Page

DIZZINESS

Dizziness (or giddiness) is a term of such inexactitude as to render it of no greater value that to indicate that all is not well.

Distinguishing

- **a sensation of feeling lightheaded** (akin to standing up too quickly)
    - suggests cerebral hypoperfusion
    - consider cardiovascular status (ischaemia, arrhythmia, valvulopathy, failure)
    - review medication (trial of reduction of antihypertensive therapy)
- **a general wooziness**
    - hyperventilation / anxiety (see chapter on **Functional and Mood Disorders**)
- **unsteadiness on the feet with a clear head**
    - implies alternative neurological substrate – referral recommended
- **an illusion of movement of one's self or one's environment** (Vertigo)

remains the key diagnostic formulation on which any therapeutic progress will depend.

Three broad presentations are common:

- Acute Vertigo
- Recurrent Vertigo
- Chronic Dizziness +/- some vertiginous features

Acute Vertigo

- Is relatively common, usually benign and usually self-limiting
- Is usually called Labyrinthitis / vestibular neuritis/neuronitis
- Presents with subacute (hours) onset of spinning vertigo almost always with nausea and vomiting. Nystagmus is typically horizontal and torsional and settles in a few days.

For examination videos see weblink below:-

http://www1.imperial.ac.uk/medicine/research/researchthemes/neuroscience/movement_balance/mabdandv/

When to refer/admit:

- Other brainstem features such as diplopia, facial weakness or sensory disturbance, dysarthria, limb ataxia, limb weakness or sensory features are incompatible with peripheral vestibulopathy and referral / admission is recommended depending on the scenario
    - hyperacute onset, rapid worsening, marked deficits – admit:
    - none of the above -refer
- Associated deafness – urgent referral to **ENT** is recommended

- Patient is unable to stand – admission for imaging to exclude post fossa stroke

Recurrent Vertigo

- Common causes of recurrent vertigo include
    - Benign Paroxysmal Positional Vertigo
        - Vertigo lasts seconds, triggered by rolling over in bed
        - Diagnosis confirmed by Hall Pike Manoeuvres (Appx V)
        - Treatment Epley Manoeuvre /Brandt-Daroff Manoeuvres (Appx V)
    - Migraine
        - Vertigo last 30 – 90 mins, other migraine features present
    - Meniere's disease
        - Fullness in ear, progressive tinnitus, ipsilateral fluctuant deafness, severe vertigo for 20 mins to several hours. **Seek ENT opinion**

Chronic Dizziness

- Establish the initiating illness / circumstances
- Dissect out current features
    - Physical & psychological (see chapter on **Functional and Mood Disorders**)
    - Review and minimize drug therapy (especially vestibular sedatives which may induce parkinsonism and are never indicated chronically.
    - Consider physiotherapy for gait retraining
    - Consider psychotherapy / CBT / relaxation therapy
    - Try Cawthorne-Cooksey Exercises (See Appx V)

Return to Contents Page

APPENDIX I

(Useful Addresses Alphabetical Order of Disease)

ANXIETY DISORDERS

http://www.moodjuice.scot.nhs.uk/anxiety.asp
www.ntw.nhs.uk/pic/leaflet.php?s=selfhelp

ATAXIA

Ataxia
Tel: 020 7820 3900 / www.ataxia.org.uk

Ataxia-Telangiectasia Society
www.atsociety.org.uk

CEREBRAL PALSY

SCOPE
Tel : 0808 800 3333 / www.scope.org.uk

CHRONIC FATIGUE SYNDROME

National ME Centre
Tel : 01708 378050 / www.nmec.org.uk

ME Association
Tel: 01375 642466

DEMENTIA

Alzheimer's Society
Tel: 0845 300 0336 / www.alzheimers.org.uk

DYSPHASIA

Speakability (Formerly action for dysphasic adults)
Tel: 020 7261 9572

DYSTONIA

The Dystonia Society
Tel: 020 7490 5671 / www.dystonia.org.uk

ENCEPHALITIS

Encephalitis Support Group
Tel: 01 653 699 599
http://glaxocentre.merseyside.org/enceph.html

EPILEPSY

The National Society for Epilepsy
Chesham Lane,
Chalfont St Peter
Bucks.SL9 0RJ
Tel: 01494 6013400 www.epilepsysociety.org.uk/

Epilepsy Action
Tel: 0808 800 5050
https://www.epilepsy.org.uk

FUNCTIONAL DISORDERS

www.neurosymptoms.org

Excellent patient info on all functional disorders

http://www.epilepsyconnections.org.uk/publications/

Patient information on Epilepsy AND PNES/NEAD

www.glasgowsteps.com Info on anxiety & depression

www.shef.ac.uk/content/1/c6/08/45/NEST%20Patient%20Booklet.pdf comprehensive patient-orientated booklet on non-epileptic attacks

www.ntw.nhs.uk/pic/leaflet.php?s=selfhelp self-help literature for panic, anxiety, depression

HEADACHE

British Association for the Study of Headache
http://bash.org.uk

The Migraine Trust
52-53 Russell Square
London WC1B 4HP
Tel: 020 7631 6970
Email: info@migrainetrust.org
http://www.migrainetrust.org/

Organisation for the Understanding of Cluster Headache

www.clusterheadaches.org.uk

Oxygen requested on Home Oxygen Order Form (HOOF)
Patient to complete Home Oxygen Consent Form (HOCF)
Supplier for West Midlands Air Products 0800 373580
(Oxygen non rebreathing masks (Part number 032 10 072) available from Flexicare Medical Limited
Cynon Valley Business Park, Mountain Ash,
Mid Glam.CF45 4ER. Tel 01443 474647)

HEAD INJURY

Headway – The Brain Injury Association
Tel: 0115 947 1917 / www.headway.org.uk

Brain and Spinal Injury Charity (BASIC)
Tel: 0870 7500000
Brain and Spine Foundation
Tel: 0808 808 1000 / www.brainandspine.org.uk

Child Head Injury Trust
http://glaxocentre.merseyside.org/chit.html

HUNTINGTON'S DISEASE

Huntington's disease Association
Tel: 0207223 7000 / www.hda.org.uk

MENINGITIS

National Meningitis Trust
Tel: 01453 768000

MOTOR NEURONE DISEASE

Motor Neurone Disease (MND) Association
PO Box 246, Northampton.NN1 2PR
Tel : 08457 626262 / www.mndassociation.org
Fax: 01604 638289/624726

MULTIPLE SCLEROSIS

MS Trust www.mstrust.org.uk

MS Society of Great Britain and Northern Ireland
Tel: 0808 800 8000 / www.mssociety.org.uk

Multiple Sclerosis (Research) Charitable Trust
www.msresearch.org.uk

MULTIPLE SYSTEM ATROPHY

www.msatrust.org.uk

NARCOLEPSY

Narcolepsy Association UK (UKAN)
www.narcolepsy.org.uk

NEUROFIBROMATOSIS

The Neurofibromatosis Association
Tel : 020 8547 1636 / www.nfa-uk.org.uk

NEUROMUSCULAR DISEASE

Guillain-Barre Syndrome Support Group
Tel : 0800 374803 / www.gbs.org.uk

Muscular Dystrophy Campaign
Tel: 020 7720 8055 / www.muscular-dystrophy.org

Myasthenia Gravis Association
http://www.myaware.org/

Charcot-Marie-Tooth (CMT) International UK
www.cmt.org.uk

PARKINSON'S DISEASE

Parkinson's UK
215 Vauxhall Bridge Road,
London.SW1V 1EJ
http://www.parkinsons.org.uk/
Helpline: 0808 800 0303

Val Mckay
Parkinson's Local Adviser
Shropshire , Telford & Wrekin
Parkinson's UK
Tel: 0344 225 3643
M : 07833237089
vmckay@parkinsons.org.uk

SY8 (Ludlow) postcode should please contact
Ann Henderson
Parkinson's Local Adviser,Herefordshire
Parkinson's UK
Tel: 0344 225 3645
Mob: 07900 820405
ahenderson@parkinsons.org.uk

www.theparkinsonhub.com

PROGRESSIVE SUPRANUCLEAR PALSY

PSP Association
www.pspassociation.org.uk
Tel 01327 322419

RESTLESS LEGS SYNDROME

www.is-it-rls.org

SPINA BIFIDA HYDROCEPHALUS

Association for Spina Bifida and Hydrocephalus (ASBAH)
www.asbah.org

STROKE

The Stroke Association
Tel: 0845 3033100 / www.stroke.org.uk

Different Strokes
www.differentstrokes.co.uk

TREMOR

National Tremor Foundation
Tel: 01708 386399

TOURETTE'S SYNDROME

Tourettes Action Help Line 0300 777 8427
www.tourettes-action.org.uk

TUBEROSE SCLEROSIS

Tuberose Sclerosis Association
Tel: 01527 871898 /
www.tuberose-sclerosis.org.uk

RETT'S SYNDROME

Rett Syndrome Association UK (RSAUK)
Tel: 020 8361 5161 / www.rettsyndrome.org.uk

Return to Contents Page

APPENDIX IIA

SUGGESTED ANTICONVULSANT DRUG TITRATION SCHEDULES

(Schedules may differ for certain patients and will be specified on case by case basis)
(Withdrawal rates are for elective withdrawal. Emergency withdrawal for severe adverse effects will differ and guidance should be sought on individual patient basis)

Twice daily dosing unless stated otherwise

Sodium Valproate$£ (SR) can be given once daily if preferred

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
200 mgm bd	200mg/wk	1.2 G/day	3 G/day	200mg / 2 weeks

Potential Side-effects: weight gain, tremor, teratogenesis (see below)

$ Do not combine with lamotrigine without neurology guidance)

£ The MHRA has published guidelines to assist clinicians prescribing valproate to women of child bearing potential.. These can be found at:

https://www.gov.uk/drug-safety-update/valproate-and-of-risk-of-abnormal-pregnancy-outcomes-new-communication-materials

Carbamazepine (SR)

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
200 mgm od	200mg/ week	400 mg bd	1000mg bd	200mg / 2 weeks

Potential Side-effects: dizziness, sedation, reduced effectiveness of low dose oral contraceptive pill (see above)
If developing rash, unexplained bruising, mouth ulcers – see GP for review and full blood count

Lamotrigine$ (Single daily dose)

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
25 mgm od	25mg/ 2 weeks	150 mg/day	600 mg/day	50mg / 2 weeks

$ (Do not combine with valproate without neurology guidance)

Potential Side-effects: rash – if develops stop drug & see GP

Phenytoin (Single nocte dose)

Start Dose	Rate of Increase*	Target Dose*	Max Dose*	Withdrawal Rate
250 mgm od	25mg/ 2 weeks	300 mg/day	450 mg/day	25mg / 2 weeks

* Guided by efficacy, side-effects, & drug levels

Potential Side-effects: unsteadiness, visual disturbance. Drug should not generally be increased by more than 25 or 50 mg. Two weeks needed for drug levels to equilibrate post (change of) dose

Topiramate

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
25 mgm od	25mg/ 2 weeks	75 mg bd	300 mg bd	25mg / week

Potential Side-effects: weight loss, tingling, urinary stones, teratogenesis (~5%) – best avoided by drinking 3 litres of fluid per day

Gabapentin (t.d.s)

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
300 mgm od	300 mg / 2 weeks	600 mg tds	900 mg/tds	300mg / 2 weeks

Potential Side-effects: sedation

Levetiracetam

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
250 mgm bd	250mg / week	500 mg bd	1.5 G bd	250 mg/ 2 weeks

Potential Side-effects: sedation

Zonisamide

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
25 mg bd	50 mg/week	300mg od	500mg od	50 mg / 2 weeks

Potential Side-effects: weight loss, urinary calculi - best avoided by drinking 3 litres of fluid per day

Pregabalin

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
50 mg bd	50 mg / week	150 mg bd	300 mg bd	100 mg/ 2 weeks

Potential Side-effects: dry mouth, constipation, dizziness

Oxcarbazepine

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
300 mg bd	150 mg / 2 weeks	600 mg bd	1.2G bd	150 mg / 2 weeks

Potential Side-effects: dizziness, sedation, reduced effectiveness of low dose oral contraceptive pill (see above)

Lacosamide

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
50 mg bd	50 mg / week	100 mg bd	200mg bd	50 mg / week

Potential Side-effects: dizziness, headache, diplopia, nausea, cardiac conduction problems

Perampanel

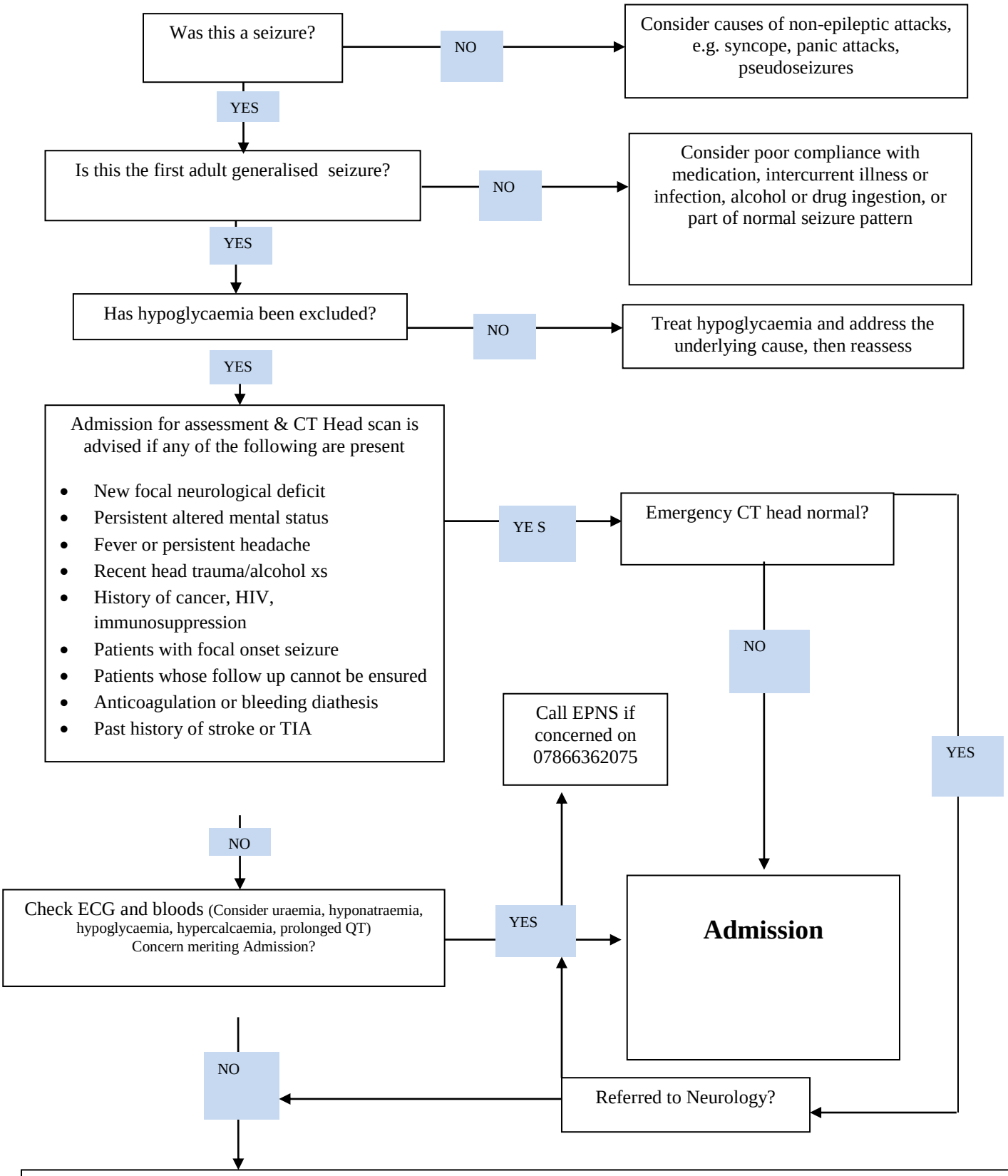
Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
2 mgm / day	2mg/ 2 weeks	4 mg /day	12 mg/day	2mg / week

Potential Side-effects: dizziness, sedation

Return to Contents Page

APPENDIX IIB

Primary Care Suspected First Fit Flow Chart



1.Give First seizure information leaflet to patient ☐
2.Fax referral proforma to Neurology Department 01743 261288 (RSH) or 01952 282873 (PRH) ☐
3. File original in patient notes. ☐
4. Post / fax witness report(s)& other relevant info & ECG to SaTH (indicate referral proforma has been pre-faxed). ☐

APPENDIX IIC

FIRST (or SUSPECTED FIRST) GENERALISED SEIZURE / CONVULSION PRIMARY CARE REFERRAL PROFORMA

PATIENT INFO ABOVE PLEASE INCL MOBILE TEL

Inclusion criteria	Patients > 16 yrs.; Clear / probable history of first convulsion (including those related to drug or alcohol ingestion or withdrawal)		√ **?**
Exclusion criteria & suggested action	Patients with known seizure already under specialist care.	If fully recovered and meets criteria (below) can be released. Check AED levels for concordance & Fax this proforma to Neurology marked **INFO ONLY**	*These seizure markers act as indicators and are not absolutes: - - Unconsciousness for more than 5 minutes. - amnesia longer than 5 minutes - injury, e.g. tongue biting - incontinence - remain conscious but with confused behaviour - headache post attack
	Syncope	Manage as syncope	
	Blackout without seizure markers*	Standard Management	

History table	**PLEASE ENSURE THE FOLLOWING ARE INCLUDED IN REFERRAL DOCUMENTS**	√ ?
Witness history	(if available) See Appendix IIE – Seizure Details Checklist	
Previous history of seizures, febrile fits, birth trauma, meningitis, head injuries		
Family history of seizures		
Possible precipitating events (alcohol, drugs, sleep deprivation)		

Investigation table

Tick if done		Result
U & Es		
Ca		
LFTs		
TSH		
FBC		
Glucose		
ECG		
CT Brain		
Name of Consultant with whom case discussed -		

Indications for ADMISSION & CT head scan

- New focal neurological deficits
- Persistent altered mental status
- Fever or persistent headache
- Recent head trauma/alcohol xs
- History of cancer/HIV/Immunosuppression
- Patients with focal onset seizure
- Patients whose follow up cannot be ensured
- Anticoagulants or bleeding diathesis
- Past history of stroke or TIA

Discharge table	**CONSIDERATIONS PRIOR TO RELEASE**	√**?**
Patient has fully recovered with no persistent neurological symptoms or signs (incl. headache)		
Normal observations (incl. temperature)		
Driving has been discussed. www.dft.gov.uk/dvla * See DVLA guidance for driving advice		
Patient has been give First seizure information leaflet		
Patient has a responsible adult to stay with following discharge and will attend follow up		

ONCE COMPLETE PLEASE FAX TO 01743 261288 (RSH) or 01952 282873 (PRH)

We think you may have had a seizure
Information for patients, family and carers

The doctor you have seen thinks you have had, or might have had, a seizure. This short leaflet contains information to help you understand what has happened and further advice on what to do if the similar problems happen again. Advice for people who have had one or more seizures is available from the sources listed at the end of this leaflet. If you want to discuss any of the issues raised in this leaflet please speak with your GP first and if you have been referred to a hospital department then further advice can be obtained by contacting that department. Shrewsbury & Telford Neurology Department contact details are given below.

What is a seizure?

A seizure (also called a fit) is a sudden disruption of brain activity caused by the nerve cells in the brain producing abnormal electrical discharges. Seizures can look and feel different.

Anyone can have a seizure and about 1 in 20 people will have a single seizure at some point in their life. This is not the same as having epilepsy. Epilepsy is a condition in which seizures recur. However, having had one seizure, there is a chance that you will have another which is why we issue this safety advice.

Sometimes it is very difficult for doctors to distinguish between seizures and other causes of disruption of brain function. Causes of attacks that can resemble seizures include some types of faint and also attacks with a psychological cause like panic attacks.

What happens next?

You will be sent an appointment to be seen in the Neurology clinic. This may be at Royal Shrewsbury Hospital or Princess Royal Hospital (SaTH) or at another hospital depending on your preferences, appointment availabilities, waiting times, etc. Following a SaTH attendance your GP will be sent a letter this hospital attendance and our usual practice is to send you a copy of that letter. If you have not received an appointment letter within three weeks of your GP telling you that you will be referred to SaTH please contact us – 01743 261105 (Royal Shrewsbury Hospital) or 01952 641222 ext. 4718 (Princess Royal Hospital)

When you come to clinic you will be asked about what happened. If someone saw what happened, it would be very helpful if he or she could come with you to provide a witness account of what they remember of the episode. If they cannot come to the clinic please ask them to give you a written account to bring to your appointment (see Page 4) or to be available at the time of your appointment on a phone number that you know so that we can speak to them on the phone to hear their account of the seizure. If your appointment time is inconvenient for the person who witnessed the event, we can phone them at a different time after the clinic if you bring their phone number with you.

For your safety, until seen in clinic, we would advise:

Shower rather than bath – or shallow quick bath when someone else is in the house and the bathroom door unlocked

Avoid climbing ladders, working at heights, horse riding, and operating machinery that would be dangerous were you to have another seizure.

If you choose to cycle we would advise you take usual safety precautions such as wearing a helmet and high visibility clothing. Consider avoiding busy roads.

If you choose to go swimming ideally have a companion in the water with you. Tell the staff at the pool about the seizure. Don't swim if you are feeling unwell. Avoid crowded times when it would be more difficult for others to notice if you have a seizure. Avoid horse riding.

Driving

Unfortunately, you have to stop driving and you will need to inform the DVLA. If you do drive you are breaking the law and your insurance company will not cover you. Driving will be discussed further at your appointment but clarification on driving will ordinarily require input from the DVLA who remain the legal authority to grant / withdraw driving privileges.

In the case of a first epileptic seizure or blackout of unknown cause, it is likely that a restriction of 6-12 months free of further episodes will be required before the DVLA will allow you to resume driving. Free bus travel, and reduced rail fares are available, see link below or telephone number for further information.

http://www.epilepsysociety.org.uk/AboutEpilepsy/Livingwithepilepsy/Drivingandtravel/Drivingandtransport#travelcosts

01494 601400

Basic first aid in the event of another seizure

Protect the person from injury (remove harmful objects from nearby)

Cushion the head

Don't try to move the person during the seizure unless they are in danger, for example in the road.

Stay with the person until they have fully recovered

Don't restrict any movements

Once the seizure has stopped, put the person on their side, into the recovery position, to help their breathing

Don’t try to put anything in the person’s mouth or give them food or drink until fully recovered or try and bring them round

Dial 999 if:

The seizure lasts 5 minutes or more (time passes very slowly in these situations)

They have injured themselves badly

Breathing difficulties continue after the seizure has ended

Another seizure occurs afterwards

Anyone witnessing a seizure should try to observe:

Was there any warning, what was the first sign?

The exact time – it started?

Did they respond when you talked to them?

Were the limbs stiff /jerking- which ones and for how long? Were they jerking together or thrashing around?

Was the head moving?

Were the eyes open?

Was the person making any noise?

Was there any change in the breathing or facial colour?

How long until it stopped? Was the end sudden or gradual? What time it stopped?

How long the seizure lasted?

If at all possible, videoing the seizure on a mobile phone would be very helpful for the doctor to determine what has happened.

After the seizure

How long before the person is able to answer simple questions accurately?

Is there abnormal behaviour e.g. wandering around?

Did they vomit, wet themselves or bite their tongue?

Did they go to sleep after or come round and get on with things?

THE PRIORITY IS TO PROVIDE FIRST AID IF REQUIRED

Factors that may increase the risk of having another seizure:

Missing or not taking your medication, if any is prescribed- many patients forget an occasional dose. If this happens take the dose as soon as you remember and then take the next dose at the next correct time.

Alcohol- up to 3 units (3 small glasses of wine or 1 ½ pints of beer) per day is generally safe. It is recommended to have no alcohol on at least 3 days each week. Some people find that even a small amount of alcohol might trigger a seizure, in which case alcohol is best avoided altogether.

Sleep deprivation- if you need to get up early, retiring to bed to enable at least 7 hours sleep is wise and avoiding having to get up in a rush by setting the alarm in good time.

Being stressed.

Flashing lights – this affects only 2% of people with epilepsy.

Medication

Is usually started after a second seizure

You will not need to pay for the prescription - you will need to ask your GP for a FP92 (A) form

Contact details for Neurology Department - SATH:

Epilepsy Nurse Specialist Tel: 07866 362075
Consultant Neurologist- PRH 01952 641222 ext. 4718
Consultant Neurologist- RSH 01743 261105

Sources of further information:
The National Society for Epilepsy
Tel: 01494 601400
www.epilepsysociety.org.uk/

Epilepsy Action
Free phone app available
Tel 0808 800 5050
http://www.epilepsy.org.uk/

Further information is available from;

- **Patient Advise and Liaison Service (PALS)** will act on your behalf when handling patient and family concerns.
- PALS, is a confidential service.
  Royal Shrewsbury Hospital, Tel: 0800 783 0057 or 01743 261691
  Princess Royal Hospital, Tel: 01952 282888

Other Sources of Information

- **NHS 111**
  A fast and easy way to get the right help, whatever the time. NHS 111 is available 24 hours a day, 365 days of the year.
  Telephone: 111 (free from a landline or mobile)
  Website: www.nhs.uk

Patient UK
Provides leaflets on health and disease translated into 11 other languages as well as links to national support/self-help groups and a directory of UK health websites.

Website: www.patient.co.uk

Return to Contents Page

APPENDIX IIE

Seizure Detail Checklist

During the seizure	✓	Please describe / comment
Did they lose consciousness? - if so, for how long?		
Did they fall down? - if so, was it forwards or backwards? - if so, did they go stiff or floppy?		
Did their colour change? - if so, did they go pale or flushed? - did they go blue around their mouth or face?		
Did they feel cold, hot or sweaty?		
Was their pulse racing or did they have palpitations?		
Did any parts of their body move? - if so, which parts (e.g. arms, legs or head)? - what sort of movements did they make? (e.g. convulsive, jerky or shuddering movements?)		
Did their head turn, and if so, which way?		
Did their face change (e.g. make chewing movements or lip smacking?)		
Did they foam at the mouth?		
Did they bite their tongue?		
Were their eyes open or closed? - did their eyes turn to one side? - did their eyelids flutter? - did they go blank and stare?		
Did their breathing change? - if so, was it noisy or laboured? - was it fast? - did they stop breathing?		
Did they do anything unusual such as fiddle with or pick at objects or their clothes?		
Were they incontinent of urine or faeces?		
After the seizure		
How did they feel after the seizure? - did they recover quickly and carry on as normal? - were they confused and if so, for how long? - did they feel agitated, aggressive or restless? - did they need to sleep and if so, for how long? - did they have a headache or feel tired? - how long did it take for them to recover fully?		
Did they injure or hurt themselves? - if so, what and how were they treated?		

Return to Contents Page

APPENDIX IIIA

ESSENTIAL TREMOR

PROPRANOLOL SCHEDLE:

			Daily Total Dose
Propanolol	10 mgm tds	Step 1	30
	20 mgm tds	Step 2	60
	Half-Inderal LA	Step 3	80
	Inderal LA	Step 4	160
	Inderal LA+Half-Inderal LA	Step 5	240
	2 x Inderal LA	Step 6	320

Standard contra-indications to Beta Blockers apply.
End points: adequate symptomatic relief / intolerable side-effects

PRIMIDONE SCHEDULE*:*

Primidone 50 mgm nocte week 1 }
Primidone 100 mgm nocte week 2 }
Primidone 150 mgm nocte week 3 }
Primidone 200 mgm nocte week 4 }

Primidone 250 mgm nocte week 5 } Switch to 250mgm tablets

Review and if necessary increase by 125 mgm per fortnight until efficacy or side-effects intervene. Maximum dose 750 mgm nocte. If withdrawing the rate of withdrawal should mirror the rate of introduction.
End points: adequate symptomatic relief / intolerable side-effects

Whilst Propranolol and Primidone (alone or in combination) remain the mainstay of medical treatment of essential tremor, many patients find these drugs of limited value or cannot tolerate effective doses because of side effects.

The drugs outlined below are now in the “worth a trial” category for patients unhelped by Propranolol and Primidone (alone or in combination in maximal tolerated doses) based on reports of benefit in small series or “expert opinion”. As always, ineffective drugs should not be continued.

For patients significantly disabled & unhelped by optimal medical referral for consideration of Deep Brain (Thalamic) Stimulation is recommended.

Drug Name*	Start Dose	Incremental rate	Max Dose	Withdrawal rate	Cautions/Contraindications
Topiramate	25 mgm od	25 mgm/ fortnite	150 mgm bd	Teratogenicity£ Weight loss, tingling, urinary calculi$	Topiramate **(may reduce efficacy of COCP)**
Gabapentin	100 mgm mane	100mg/week	600 mg/ tds	100 mg/week	Diarrhoea Nausea Dizzyness Wt gain
Levetiracetam	250 mgm bd	250 / week	1.5 G bd	500 mgm /week	Fatigue Diarrhoea Nausea Dizzyness

£ Has potential deleterious effects in pregnancy (~5%). Referral for all women of child bearing potential is recommended prior to commencing such therapy

$ Ensure 3 litres fluid per day. Caution if history of urinary calculi.

Return to Contents Page

APPENDIX IIIB

PARKINSON'S DISEASE

(NON-ERGOT DOPAMINE AGONISTS- Ropinirole /Pramipexole)

Ropinirole Titration Schedule:

Week 1	0.25	mgm tds
Week 2	0.5	mgm tds
Week 3	0.75	mgm tds
Week 4	1	mgm tds
Week 5	2+ 1+ 1	mgm daily
Week 6	2 + 2 + 1	mgm daily
Week 7	2	mgm tds
Week 8	3	mgm tds

Hold dose if efficacy reached.
Return to maximum tolerated dose if efficacy occurs but side-effects intervene.

HOLD AND MONITOR RESPONSE
FOLLOWING NEUROLOGICAL ADVICE AND ONLY IF DIRECTED:-

Week 20:	4 mgm tds
Week 21:	5 mgm tds
Week 22	6 mgm tds
Week 23	7mgm tds
Week 21	8 mgm tds

Hold dose if efficacy reached.
Return to maximum tolerated dose if efficacy occurs but side-effects intervene

Pramipexole Titration Schedule:

BASE	**SALT**
Week 1: 0.088 mg/d	0.125 mg/d
Week 2:0.088mg bd	0.125 mg bd
Week 3:0.088 mg tds	0.125 mg tds
Week 4:0.18mg tds	0.25 mg tds
Week 5:0.35 mg tds	0.5 mg tds
Week 6:0.53 mg tds	0.75 mg tds

Hold dose if efficacy reached.
Return to maximum tolerated dose if efficacy occurs but side-effects intervene.

HOLD AND MONITOR RESPONSE
FOLLOWING NEUROLOGICAL ADVICE AND ONLY IF DIRECTED:-

Week 21: 0.7 mg tds	1 mgs tds
Week 22: 0.88 mg tds	1.25mg tds
Week 23: 1.05 mg tds	1.5 mgm tds

Hold dose if efficacy reached.
Return to maximum tolerated dose if efficacy occurs but side-effects intervene

Pramipexole Prolonged Release Titration Schedule:

	BASE	**SALT**	Tablet Scoring
Week 1:	0.260 mgs daily	0.375 mgs daily	P1
Week 2:	0.520 mgs daily	0.750 mgs daily	P2
Week 3:	1.05 mgs daily	1.5 mgs daily	P3
Week 4:	1.31 mgs daily	1.875 mgs daily	
Week 5:	1.57 mgs daily	2.25 mgs daily	P12
Week 6:	2.1 mgs daily	3 mgs daily	P4
Week 7:	2.62 mgs daily	3.75 mgs daily	P13
Week 8:	3.15 mgs daily	4.5 mgs daily	P5

Hold dose if efficacy reached. Return to maximum tolerated dose if efficacy occurs but side-effects intervene.

Ropinirole Prolonged Release Titration Schedule:

Week 1: 2mg daily
Week 2 :4 mg /day
Hold dose if efficacy reached.
Return to maximum tolerated dose if efficacy occurs but side-effects intervene.

Week 3: 6 mg/day
Week 4: 8mg /day
Hold dose if efficacy reached.
Return to maximum tolerated dose if efficacy occurs but side-effects intervene.
Can increase by 2 mg per 2 weeks to 24 mg/day

Return to Contents Page

PARKINSON'S DISEASE
DOPAMINE AGONIST SWITCHING SUGGESTIONS

Strongly recommended to be done under neurological supervision, usually as overnight swap.

Bromocriptine TDS	Pergolide TDS	Cabergoline (once daily)	Pramipexole (SALT DOSES)	Prami CR OD	Ropinirole	Ropi XL OD	Rotigotine (once daily)
1 mgm	0. 125mgm	0.5 mgm	0.125 mgm tds	0.375mg	Starter pack	NA	2 mg
2. 5 mgm	0. 25mgm	1 mgm	0. 25 mgm tds	0.75mg	1 mgm tds	4mg	4 mg
5 mgm	0. 5 mgm	2 mgm	0. 5 mgm tds	1.5mg	2 mgm tds	6 mg	6 mg
7. 5 mgm	0. 75 mgm	3 mgm	0. 75 mgm tds	2.25mg	3 mgm tds	8 mg	8 mg
10 mgm	1 mgm	4 mgm	1 mgm tds	3 mg	4 mgm tds	12 mg	10-12
12. 5 mgm	1. 25 mgm	5 mgm	1. 25 mgm tds	3.75mg	6 mgm tds	16 mg	14 mg
15 mgm	1. 5 mgm	6 mgm	1. 5 mgm tds	4.5 mg	8 mgm tds	24 mg	16 mg

NB Maximum dose of rotigotine is 16mg/24hrs

Rotigotine patches available in 2mg/4mg/6mg/8mg strength

Rotigotine patches may be combined but **must not be cut.**

ERGOT DOPAMINE AGONISTS – See monitoring requirements – Appx IV

Pergolide Titration Schedule:

Day 1: 50 µgs nocte
Day 2-4:50 µgs bd
Day 5-7:50 µgs tds
Week 2:100 µgs tds
Week 3:250+100+100 µgs tds
Week 4:250+250+ 100 µgs tds
Week 5:250 µgs tds
Week 6:500+ 250+ 250 µgs
Week 7:500+ 500+ 250 µgs
Week 8:500 µgs tds

Hold dose if efficacy reached.
Return to maximum tolerated dose if efficacy occurs but side-effects intervene.

HOLD AND MONITOR RESPONSE

FOLLOWING NEUROLOGICAL ADVICE AND ONLY IF DIRECTED :-

Week 21: 750+ 500+500 µgs
Week 22: 750 + 750 +500 µgs
Week 23: 750 µgs tds
Week 24: 1000 + 750 + 750 µgs
Week 25: 1000 + 1000 + 750 µgs
Week 26: 1000 µgs tds

Hold dose if efficacy reached.

Return to maximum tolerated dose if efficacy occurs but side-effects intervene

Cabergoline Titration Schedule:

Week 1	0.5	mgm daily
Week 2	1.0	mgm daily
Week 3	1.5	mgm daily
Week 4	2.0	mgm daily
Week 5	2 5	mgm daily
Week 6	3.0	mgm daily

HOLD AND MONITOR RESPONSE

FOLLOWING NEUROLOGICAL ADVICE AND ONLY IF DIRECTED:-

Week 21 3.5mgm daily
Week 22 4.0 mgm daily
Week 23 4.5 mgm daily
Week 24 5.0 mgm daily

Hold dose if efficacy reached.
Return to maximum tolerated dose if efficacy occurs but side-effects intervene.

Trihexyphenidyl (BENZHEXOL) SCHEDULE:
(caution in elderly or if cognition impaired)

Week 1	1mgm mane
Week 2	1 mgm bd
Week 3	1 mgm tds
Week 4	1,1,2 mgm
Week 5	1,2,2mgm
Week 6	2 mgm tds
Week 7	2,2,3 mgm tds
Week 8	2,3,3 mgm tds
Week 9	3 mgm tds
Week 10	3,3,4 mgm tds
Week 11	3,4,4 mgm tds
Week 12	4 mgm tds

CLONAZEPAM SCHEDLE:

Clonazepam	0.25 mgm daily	Week 1
	0.25 mg bd	Week 2
	0.25 mg a.m. 0.5 mg pm	Week 3
	0.5 mg bd	Week 4
	0.5mg a.m 0.75 mg pm	Week 5
	0.75 mg bd	Week 6
	0.75 a.m. 1mg p.m.	Week 7
	1mg bd	Week 8

Hold dose if efficacy reached.
Return to maximum tolerated dose if efficacy occurs but side-effects intervene.

LEVODOPA: (Sinemet, Madopar preparations)

As specified in individual patient correspondence

ENTACAPONE

As specified in individual patient correspondence

Quetiapine (bd)

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
25mg nocte	25 mg / week	50 mg/bd	100mg / bd	25 – 50 mg / week

Potential Side-effects: drowsiness, dyspepsia, constipation, dry mouth, tachycardia, leucopenia

Clozapine (single bedtime dose)

Start Dose	Rate of Increase	Target Dose	Max Dose	Withdrawal Rate
12.5mg nocte	6.25 mg / week	25 mg/nocte	50mg / nocte	See below

Withdrawal

- planned (seek neurological guidance)
- emergency (leucopenia /suspected myocarditis/cardiomyopathy) stop immediately and phone 01522 573960 for advice

Potential Side-effects: leucopenia, other bone marrow disorders, myocarditis, cardiomyopathy, anorexia, nausea, vomiting, dyspepsia, constipation, dry mouth, tachycardia, headache, tremor, seizures

Return to Contents Page

APPENDIX IV

MONITORING SCHEDULES

PARKINSON'S DISEASE

Dopamine agonists (ergot containing) – Bromocriptine, Pergolide, Cabergoline

These agents have fibrotic potential. The following monitoring is recommended**:-**

Baseline: **Clinical cardiorespiratory examination***
Urinalysis
ESR, U&E, creatinine#
Chest Radiograph#
(* ECG/ ECHO/ Lung function tests/ if clinical concern)

 # The CSM has advised these tests following prior to starting these drugs:

6 Monthly: Regular clinical reassessment for cardiorespiratory decline or novel abdominal symptoms
Perform / Repeat above investigations as clinically indicated.

Return to Contents Page

Multiple Sclerosis

Beta-Interferons (Avonex, Rebif, Betaferon)

Baseline:	FBC, U&Es, LFTs, protein electrophoresis
Month 1:	FBC, LFTs
Month 3	FBC, LFTs
Month 6	FBC, LFTs
Month 9	FBC, LFTs
Month 12	FBC, LFTs, TFTs
6 Monthly	FBC, LFTs

(Copaxone - no monitoring required)

WHO Guidelines for grading abnormal LIVER FUNCTION TESTS

WHO Grading	ALT, AST, GGT Alkaline Phosphates	Bilirubin	Recommended Management
Grade I	>UNL-2.5 times UNL	> UNL – 1.5 times UNL	Continue treatment but monitor regularly
Grade II	2.6 – 5 times UNL	1.6 –3 times UNL	Continue treatment but monitor regularly. If Grade ll persists, the dose should reduced (halved) until the LFT's have returned to grade 0 or 1; the full dose may be then recommenced at clinical discretion
Grade III	5.1 – 20 times UNL	3.1 – 10 times UNL	Interrupt treatment (or reduce/halve dose) until LFT's have returned to grade 0 or 1; recommence treatment at half dose and increase to full dose at clinical discretion
Grade IV	> 20 times UNL	> 10 times UNL	Interrupt treatment (or reduce/halve dose) until LFT's have returned to grade 0 or 1; recommence treatment at half dose and increase to full dose at clinical discretion
Grade V			Interrupt treatment

ALT= alanine transaminase; AST= aspartate transaminase; GGT= γ -glutamy1 transferase;
UNL= upper normal limit

Return to Contents Page

WHO Guidelines for grading Haemoglobin levels

WHO Grading	Haemoglobin	Recommended management
Grade I	<LNL- 10.0g/dL	Continue treatment but monitor regularly
Grade II	8.0< 10.0g/dL	Continue treatment but monitor regularly. If Grade ll persists, the dose should be reduced(halved) until haemoglobin levels returned to Grade 0or 1; the full dose may be recommenced at clinical discretion
Grade III	6.5< 8.0g/dL	Interrupt treatment (or reduce/ halve dose) until the haemoglobin levels returned to Grade 0or 1;recommence treatment at half dose and increase to full dose at clinical discretion
Grade IV	< 6.5g/dL	Interrupt treatment until the haemoglobin levels returned to Grade 0 or 1;recommence treatment giving only half dose (i.e., do not increase to full dose).
Grade V		Interrupt treatment

WHO Guidelines for grading White Blood Cell Counts

WHO Grading	White Blood Cell Count	Recommended management
Grade I	3.0 – 3. 9	Continue treatment but monitor regularly
Grade II	2.0 –2.9	Continue treatment but monitor regularly. If grade ll persists, the dose should be reduced (halved) until WBCs have returned to Grade 0 or I; the full dose may then be recommenced at clinical discretion.
Grade III	1.0 –1.9	Interrupt treatment (or reduce to halve dose)until the WBCs have returned to Grade 0 or I; recommence treatment at half dose and increase to full dose at clinical discretion
Grade IV	<1.0	Interrupt treatment until WBCs have returned to Grade 0 or I; and recommence treatment giving only half dose (i.e., do not increase to full dose).
Grade V		Interrupt treatment

Reference

1. Association of British Neurologists Guidelines for the use of Beta Interferons and Glatiramer Acetate in Multiple Sclerosis January 1999

AZATHIOPRINE

Monitoring:

Baseline: Full blood count (including platelets)

Liver function tests

TPMT
Review vaccination status and local guidelines

Weekly for 8 Weeks:
And following any dose increase:

Full blood count (including platelets)

Liver function tests

Three monthly thereafter:

Full blood count (including platelets)

Liver function tests

If any of the following are present

a) progressive anaemia
b) total WBC < 3.0 x 10^9/l
c) neutrophils < 1.5 x 10^9/l
d) platelets <100 x 10^9/l
e) progressive liver function abnormality

then:

1) withdraw Azathioprine

2) repeat blood tests at weekly intervals and when normal re-start Azathioprine at previous dose reduced by 50 mgs

3) telephone the department here on 01743 261000 Ext 1150/ 01952 641222 Ext 4430 to arrange an early appointment.

NB Drug interactions

Allopurinol - potentiates azathioprine (cut AZA dose by 25-50%)
Sulphasalazine potentiates azathioprine toxicity
ACE inhibitors
Anaesthetics
Flu vaccination: **recommended but live vaccines should NOT be administered**

Return to Contents Page

CICLOSPORIN

Monitoring

Baseline
BP check
U&Es / GFR
FBC, LFT, Lipids
Urinalysis

Review vaccination status and local guidelines

Fortnightly for the first 3 months:

Blood pressure

Serum creatinine

Full blood count (including platelets)

Liver function tests

Urinalysis

2 monthly thereafter.

Blood pressure

Serum creatinine

Full blood count (including platelets)

Liver function tests

Urinalysis

Lipids

.
If hypertension develops, please monitor and treat. Only if BP cannot be controlled would it be necessary to withdraw medication.

If any of the following are present:

a) creatinine increases by >30% from baseline
b) progressive anaemia
c) total WBC <3.0 x 10^9/l
d) neutrophils < 1.5 x 10^9/l
e) platelets <100 x 10^9/l
f) progressive liver function abnormality

then:

1) withdraw Ciclosporin

2) repeat tests at two weekly intervals and when normal re-start at previous dose reduced by 50 mgs

3) Telephone the department on 01522 573960 / 01522 573084 to arrange an early appointment.

NB Drug Interactions

NSAIDS/Aspirin levels may be elevated by Ciclosporin

Potential nephrotoxic drugs - + trimethoprim, Cipro, aminoglycosides

Ciclosporin levels may be

Elevated by	Lowered by
OCP	Phenytoin
Calcium antagonists* (eg diltiazem,nicardipine,verapamil)	Carbamazepine
Antifungals	Barbiturates
Erythromycin	Other enzyme inducers
Methylprednisolone	
Allopurinol	
Metoclopramide	

* Amlodipine & nifedipine allowable

Flu vaccination **recommended but live vaccines should NOT be administered**

Return to Contents Page

METHOTREXATE

Patient to have SaTH Methotrexate Treatment Booklet for information & monitoring records

TERATOGENIC DRUG.

Pregnancy avoid during RX & 1 month following cessation
Men not to conceive during Rx & 6 months following cessation

Dosing: Start at 2.5 mgm / week (on same day of week)
Guidance of dose increase tailored to patient but target dose 10-20 mgm as single weekly dose week (Max dose 0.5 mg/kg)

Monitoring:

Baseline: FBC.U&Es,LFTs, Procollagen Peptide 3*
CXR, USS Liver/ Preg test/ Hepatitis A,B,C serology
Review vaccination status and local guidelines
Folic acid 5 mgs daily (not on day of MTX) for prevention of gastrointestinal side effects.

1 Week FBC.U&Es,LFTs
(after test dose of 2.5 mgm and any dose increase)

Monthly:

Full blood count including platelets

Liver function tests

U & Es

6 Monthly Procollagen Peptide 3* (if remains normal liver biopsy not needed)

If any of the following are present:

a) progressive anaemia (isolated macrocytosis requires no action)
b) total WBC < 3.0 x 10^9/l
c) neutrophils < 1.5 x 10^9/l
d) platelets < 100 x 10^9/l
e) progressive liver function abnormality (ALT > 2 x baseline)
f) creatinine > 20 % over baseline
then:

1) withdraw Methotrexate

2) repeat blood tests at weekly intervals and when normal re-start at previous dose reduced by 2.5 mgs

3) seek advice from appropriate consultant

NB:

Aspirin and other NSAIDs (see national formulary appendix 1) increase Methotrexate levels and are therefore contraindicated.

Sulphonamides (especially Co-trimoxazole) have additional folate antagonist action and may lead to megaloblastic crisis. Other antibiotics can augment MTX activity increasing the risk of toxicity (Check BNF).

Alcohol should be avoided if possible.

Patient to have SaTH Methotrexate Treatment Booklet for information & monitoring records

MYCOPHENOLATE

Monitoring:

Baseline: Full blood count (including platelets)

Liver function tests

Review vaccination status and local guidelines

Weekly for 4 Weeks:
And following any dose increase - weekly for 4 weeks:

Full blood count (including platelets)

Liver function tests

Fortnightly thereafter for two months

Full blood count (including platelets)

Liver function tests

Monthly thereafter:

If any of the following are present

a) progressive anaemia }
b) total WBC < 4.0 x 10^9/l }
c) neutrophils < 2.0 x 10^9/l } **SEE 1 BELOW**
d) platelets <150 x 10^9/l }
e) ALT or ALK phos > x2 upper limit of normal }
f) rash, abnormal bruising, sore throat, oral ulceration **SEE 2 BELOW**
g) Diarrhoea – reduce dose and increase at slower rate

then:

1) Telephone 01743 261105 for advice

2) Withhold until full blood count result available

.

Flu vaccination: **recommended but live vaccines should NOT be administered. Those not immune to varicella zoster should receive passive prophylaxis after exposure to chickenpox or shingles**

APPENDIX V

HEADACHE DIARY

Month of – Your Name:-

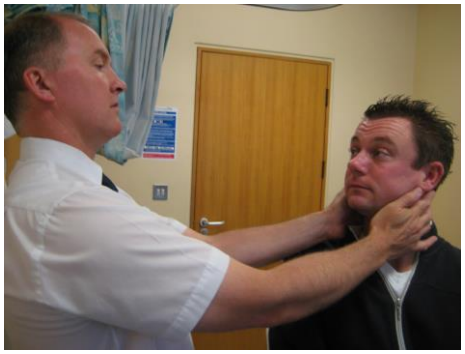
Year of - Your Date of Birth:-

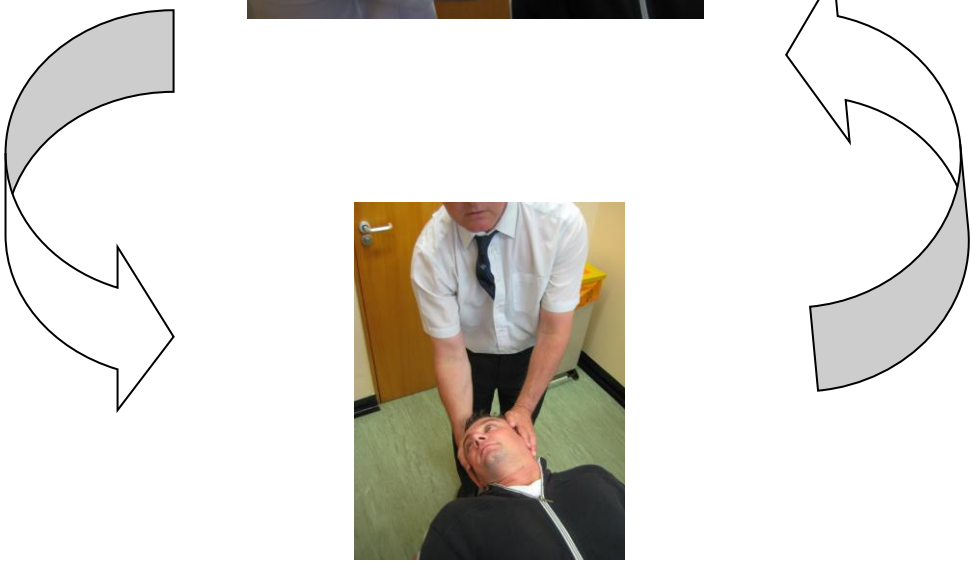
Date	Week Day	All Headache Tablets Taken (Names and numbers please)	Headache Score (0 – 10)	Running Total headache score for this month
1				
2				
3				
4				
5				
6				
7				
8				
9				
10				
11				
12				
13				
14				
15				
16				
17				
18				
19				
20				
21				
22				
23				
24				
25				
26				
27				
28				
29				
30				
31				

Return to Contents Page

THE HALLPIKE MANOEUVRE

(for right BPPV)



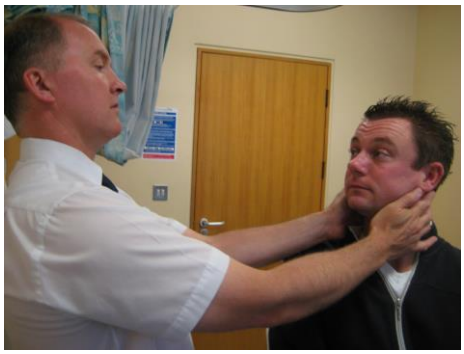


Nystagmus characteristics during Hallpike Manoeuvre

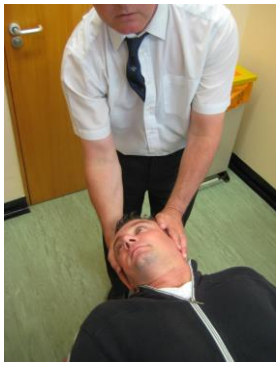
	Peripheral	Central
Latency	Few seconds	Nil
Fatigability (wears off on repeated testing)	Yes	No
Adaptation (wears off within 30 – 60 seconds)	Yes	No
Direction	Torsional	Vertical
Symptomatic	++++	+

THE EPLEY MANOEUVRE
(for Right BPPV)

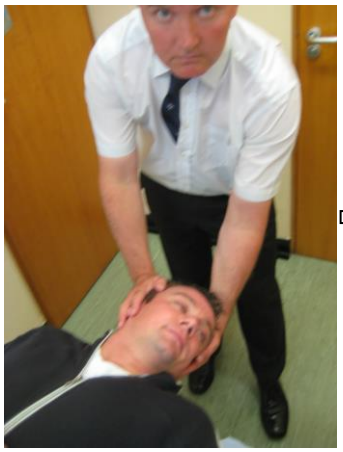
STEP ONE

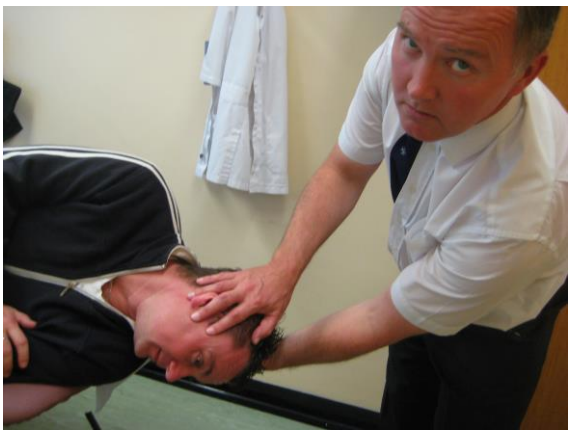


STEP Two

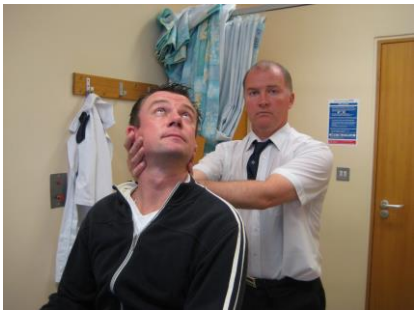


STEP THREE

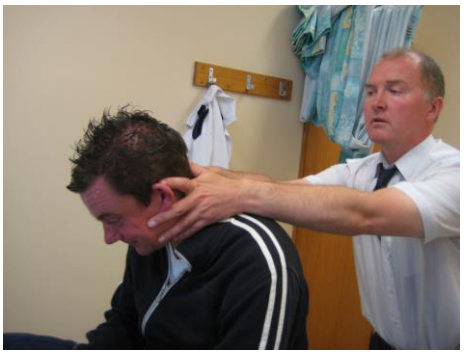




STEP FOUR



STEP FIVE

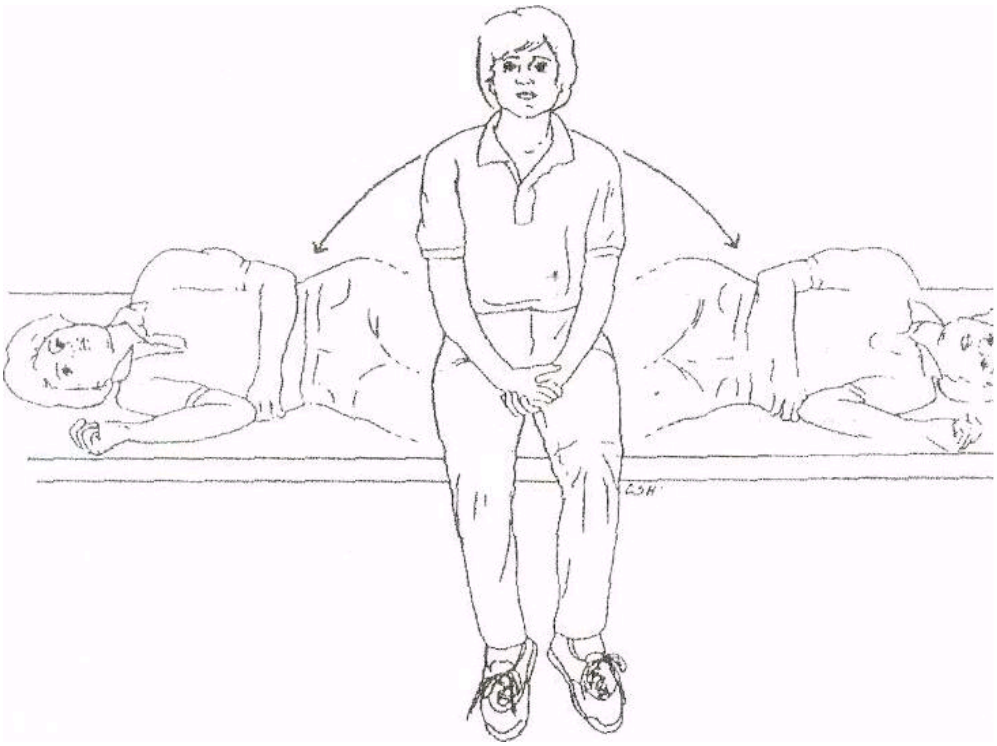


STEP SIX

Move sharply to each position and hold for 30 seconds. Initially (Step One) patient is sat up with head turned 45 degrees towards affected side. Next lay patient down (Step Two) as if performing Hall-Pike manoeuvre. Then rotate head through 90 degrees (Step Three) to opposite side with face upwards. Next (Step Four) turn patient onto side then rotate head 90 degrees so patient is facing obliquely downwards towards floor. Then raise patient to sitting (Step Five) maintaining angle between head and shoulders. Finally centre and flex head & neck so patient is looking at his/her lap (Step Six). Warn the patient before hand that each manoeuvre may induce transient vertigo. Occasionally patients may vomit and a handy receptacle is useful

THE BRANDT- DAROFF EXERCISES

A Treatment for Benign Paroxysma/Positional Vertigo



Sequence of repetitive positioning:

1. Sit on the edge of your bed.

2. Lie down sideways, to adopt the position which brings on your vertigo. *(You may close your eyes if that makes you more comfortable.)*

3. Remain in this position until the vertigo wears off (maximum of one minute).

4. Sit up for 30 seconds.

5. Lie down on the opposite side for 30 seconds (or until the symptoms subside).

6. Sit up again.

7. Try to change positions as rapidly as possible

Repeat this sequence of position flings until they no longer make you dizzy. *(You may stop if you start to feel sick, or if you still get dizzy after adopting the provoking position for the fifth time.)*

Repeat the whole exercise process THREE TIMES A DAY until you have been free of symptoms for TWO days.

COOKSEY CAWTHORNE EXERCISES

TO BE CARRIED OUT FOR AT LEAST FIVE MINUTES TWICE DAILY

THESE EXERCISES MAY MAKE YOU MORE DIZZY AT FIRST, BUT IN THE LONG TERM SHOULD HELP TO PREVENT FURTHER ATTACKS.

LEVEL 1 – EYE EXERCISES

A **Looking up then down at first slowly and then quickly - 20 times**

B **Looking from one side to the other at first slowly then quickly - 20 times**

C **Focus on your finger at arms length then move it in and out one foot - 20 times**

LEVEL 2 – HEAD EXERCISES

A **Bend your head forward and then backward with your eyes open slowly and then quickly - 20 times**

B **Turn your head from one side to the other slowly and then quickly - 20 times. As the dizziness improves these head exercises should be done with eyes closed**

LEVEL 3 – SITTING

A **Shrug your shoulders - 20 times**

B **Turn your shoulders to right then left - 20 times**

C **Bend forward and pick up objects from the ground and sit up again - 20 times**

LEVEL 4 – STANDING

A **Move from sitting to standing and back again - 20 times with eyes open then repeat with eyes closed**

B **Throw a small rubber ball from hand to hand above eye level**

LEVEL 5 – MOVING ABOUT

A **Walk across the room with your eyes open - 20 times then repeat it with your eyes closed**

Return to Contents Page

BASH Guidance for Primary Care on who to Image when Brain Tumour is suspected

(see Kernick D, Ahmed F, Bahra A et al. Imaging Patients with suspected brain tumour. Guidance for primary Care.
***British Journal of General Practice 2008*; 58: 880-885)**

The decision to investigate headache presentation is based on a number of complex factors that include the value of early diagnosis, reassurance, availability of investigation and both the patient and GP's approach to risk and uncertainty. This guidance is based upon expert opinion and the available limited evidence.

Table 1 shows the population risk of tumour with age. The overall risk of brain tumour for headache presentation in primary care is 0.09%. If the GP can make a diagnosis of a primary headache at presentation (migraine, tension type or cluster) the risk of tumour is 0.045%. If a primary headache diagnosis can not be made, the risk is 0.15%.

Table 2 shows recommended guidance for investigating various clinical presentations.

Table 3 offers guidance for GPs when managing isolated headache (no other signs or symptoms) where a diagnosis of a primary headache can not be made.

Table 1 - Relative risk of tumour. (Age, 18-29 as base rate.)

Age band years	Relative risk of tumour against base rate at 18-29 years (2.6 tumours/year/100,000 population)
18-29	1.0
30-39	1.5
40-49	2.2
50-59	4.3
60-69	6.9
70-79	7.9
80+	5.9

Return to Contents Page

Table 2 – Recommended guidance for investigating for tumour in primary care patient.

Red Flags **– presentations where the probability of an underlying tumour is likely to be greater than 1%. These warrant urgent investigation.**

- Papilloedema.
- Significant alterations in consciousness, memory, confusion or co-ordination.
- New epileptic seizure
- New onset cluster headache (imaging required but non-urgent).
- Headache with abnormal findings on neurological examination or other neurological symptoms.
- Headache with a history of cancer elsewhere particularly breast and lung.

Orange Flags **- presentations where the probability of an underlying tumour is likely to be between 0.1 and 1%. These need careful monitoring and a low threshold for investigation.**

- New headache where a diagnostic pattern has not emerged after eight weeks from presentation.
- Headache aggravated by exertion or Valsalva manoeuvre.
- Headaches associated with vomiting.
- Headaches that have been present for some time but have changed significantly, particularly a rapid increase in frequency.
- New headache in a patient over 50.
- Headaches that wake from sleep.
- Confusion.

Yellow Flags - presentations where the probability of an underlying tumour is likely to be less than 0.1% but above the population rate of 0.01%. These need appropriate management and the need for follow up is not excluded.

- Diagnosis of migraine or tension type headache.
- Weakness or motor loss.
- Memory loss.
- Personality change.

Table 3 - Clinical guidance for new presentations of isolated non-urgent headache that need a diagnostic pattern to emerge. Reduce the follow up time if orange flag headache features are present.

i) <u>At presentation</u> (Approximately 0.15% risk if a diagnosis of a primary headache is not made)

- Exclude red flags as below
- Check blood pressure, fundoscopy and consider ESR if >50years to exclude temporal arteritis.
- If no diagnosis cannot be made tell patient - *"There is no evidence of anything serious underlying your headache but I would like to review you in one month".*
- Ask patient to keep a headache diary.

ii) <u>At one month</u>

- Exclude urgent features as above. If diagnosis still uncertain:
- Assess memory and cognitive function during interview.
- Assess for symptoms that would indicate primary lesion elsewhere
- Assess for signs that would indicate primary lesion elsewhere (minimum set takes approximately three minutes.)
- Fundoscopy, pupil responses, visual fields, eye movements, facial movements (raise eyebrows to ceiling, show teeth and grimace), corneal reflexes, protrude tongue, palm drift (outstretched hands, palms uppermost), finger-nose touching with middle or index finger, finger dexterity (play piano), limb and plantar reflexes, standing feet together eyes closed, tandem gait (walk heel to toe in straight line).
- Consider blood screen to exclude systemic illness or evidence of primary tumour elsewhere - FBC, ESR, CRP, LFT, creatinine, electrolytes, glucose, thyroid function.
- If diagnosis is still uncertain tell patient - *"There is still no evidence of anything serious but I would like to review you again in another month".*

iii) <u>At two months</u>

- Exclude urgent features.
- Examination as above.
- If diagnosis is still uncertain tell patient - *"There is still no evidence of anything serious underlying your headache but we need to discuss whether it would be appropriate to have a brain scan. The best estimate is that the chances of you having a tumour are between 0.1 and 1%. However, three in every 100 people like you will show an incidental finding that may give rise to unnecessary anxiety and may have implications for future life insurance cover.*

- MRI is a more sensitive investigation and CT can miss up to 10% of lesions. Practical and economic circumstances will inevitably dictate the mode of investigation. A normal investigation does not over-rule the need for further review.
- Order blood investigations as above if not previously taken in addition to tests dependent on symptoms and history. E.g. VDRL, Lyme titres, antiphospholipids
- If patient and doctor decide against imaging review again in one month. At this time, 74% of tumours will have become apparent clinically.

Return to Contents Page

On-Off Parkinson's Disease Chart

Date Recorded: -

TIME	DRUGS TAKEN	STATE	DYSKINESIAS	CONFUSION
0600 – 0630				
0630 – 0700				
0700 – 0730				
0730 – 0800				
0800 – 0830				
0830 – 0900				
0900 – 0930				
0930 – 1000				
1000 – 1030				
1030 – 1100				
1100 – 1130				
1130 – 1200				
1200 – 1230				
1230 – 1300				
1300 – 1330				
1330 – 1400				
1400 – 1430				
1430 – 1500				
1500 – 1530				
1530 – 1600				
1630 – 1700				
1700 – 1730				
1730 – 1800				
1800 – 1830				
1830 – 1900				
1900 – 1930				
1930 – 2000				
2000 – 2030				
2030 – 2100				
2100 – 2130				
2130 – 2200				
2200 – 2230				
2230 – 2300				
2300 – 2330				
2330 – 2359				
0000 - 0100				
0100 – 0200				
0200 – 0300				
0300 - 0400				
0400 – 0500				
0500 – 0600				

Notes

1. Please enter date of study at top of page.
2. Please enter drug names & doses.
3. Please use "ON" (or shade block **GREEN**) to describe mobile (medicated) state i.e. when drugs are working and "OFF" (or shade block **RED**) to describe immobile (unmedicated) state i.e. when drugs are not working. "Intermediate" (or shade block **BLUE**) for half way between two states. If asleep please record as "SLEEP"
4. Dyskinesias are involuntary writhing movements. + = bad, ++ = very bad, **0** = absent
5. Please include any hallucinations that occur in CONFUSION column

ON OFF INT

"ON" **"OFF"** **"INTERMEDIATE"**

END OF DOCUMENT

Return to Contents Page

Trihexyphenidyl (BENZHEXOL) SCHEDULE:
(caution in elderly or if cognition impaired)

Week 1	1mgm mane
Week 2	1 mgm bd
Week 3	1 mgm tds
Week 4	1,1,2 mgm
Week 5	1,2,2mgm
Week 6	2 mgm tds
Week 7	2,2,3 mgm tds
Week 8	2,3,3 mgm tds
Week 9	3 mgm tds
Week 10	3,3,4 mgm tds
Week 11	3,4,4 mgm tds
Week 12	4 mgm tds