Woodlands Hospital
NHG Health

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SingHealth

Red Flags & Green Flags

Common presenting complaints, and how to handle them

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What this webinar is — and isn't

1 The goal

Finding thresholds for acceptability in uncertainty.

You will rarely have complete certainty in an initial consult. The aim is to build a working sense of *how much uncertainty is safe to carry* for a given presentation — and when that threshold has been crossed.

We will also touch on examples of how I communicate this principle to the patient

✗ What this is NOT

Not an encyclopaedic, WebMD-style list of every neurological disease.

But there will be examples of diseases to have on your radar

All of neurology is a two-step process

WHERE SPOIL?

- Which neuro system is affected?
  - High cortical?
  - Basal Ganglia?
  - Autonomic?
  - Cerebellar?
  - Pyramidal?
    - Brain
    - Brainstem
    - Spinal cord
    - Anterior horn cell
    - Nerve root
    - Peripheral nerve
    - Neuromuscular junction
    - Muscle

ALL OF NEUROLOGY IS MERELY A 2.5 STEP PROCESS

WHY SPOIL?

- VITAMIN-C

WHERE ELSE SPOIL

- Other physiological systems
- Other neurological systems

BASIC PRINCIPLE 2

How neurology organises itself

1. **Epilepsy**
2. **Dementia & Cognition**
3. **Stroke / Vascular**
4. **Headache**
5. **Neuroimmunology**
6. **Neuromuscular**
7. **Movement Disorders**

A simple map — not the focus of today, but useful scaffolding for where each complaint in this talk sits.

First objective: make sure the patient is safe

At every level of care, the job is not to name the disease first. The job is to decide whether there is a localisation that

a) ***makes sense*** for a pathology, and

b) what level of acuity that pathology might have.

Localises and fits a coherent pattern

Findings converge on a plausible anatomical or physiological site, and the story hangs together for a specific process.

→ Concerning. Warrants active work-up.

Doesn't localise / doesn't fit

Findings are diffuse, inconsistent, non-anatomical, or don't cohere into a recognisable pattern for any single pathology.

→ Reassuring. Perhaps may be construed as non-specific.

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Dear colleague,

Please kindly review this patient who is keen to see specialist to
evaluate "cold/shivering" sensation over left parietal region

Thank you
```

This is the lens under every red flag / green flag judgement in this talk.

PART TWO

Common Presenting Complaints

1. Approach to headache
2. Approach to weakness & sensory loss
3. Approach to movement disorders
4. Approach to dizziness, funny turns, LOC, and gait difficulties

Every section below shares one recurring framework:

LOCALISE → RED FLAGS → MANAGE / REFER / ED

SECTION 2 OF 5

Approach to Headache

LOCALISE → RED FLAGS → MANAGE / REFER / ED

Red flag headache diseases

Subarachnoid haemorrhage

Thunderclap onset — maximal intensity within seconds to minutes.

Giant cell arteritis

New headache, age 50+, jaw claudication, visual symptoms, scalp tenderness.

Venous sinus thrombosis

Persistent headache, worse lying flat, risk factors for hypercoagulability.

Mass lesion / raised ICP

Worse in the morning, with Valsalva, or new neurological deficit.

Idiopathic intracranial hypertension

Young, overweight, visual obscurations, pulsatile tinnitus.

Meningitis / encephalitis

Fever, neck stiffness, photophobia, altered mental state.

Not exhaustive — the shared pattern: sudden or changed, systemic, or accompanied by a new deficit.

Some green flags

- Long-standing, stereotyped pattern the patient recognises as “their usual headache”
- No systemic features — no fever, no weight loss, no scalp or jaw symptoms
- Positive family history of a similar pattern
- Completely normal neurological examination
- Typical migrainous accompaniments: photophobia, phonophobia, nausea, unilaterality
- Reproducible, identifiable triggers (sleep loss, dehydration, stress, hormonal timing)

Green flags reassure — they don't eliminate the need for clinical judgement on their own.

Example documentation

55F
.
History revisited
has history of headaches since youth
bitemporal tightness
not a/w visual symptoms
occasional N&V
no focal neurology
.
since 5-6 months ago
increase in frequency from 1-2x/mth to 3-4x/mth
not thunderclap
lasts approx. 4-5 hrs each time
similar character to before, with addition of throbbing. mostly left, sometimes right
not debilitating
no postural relation
no visual change
no amaurosis symptoms
no limb girdle pain
no jaw claudication
no clear triggers
not symptomatic at present moment
appetite NAD, able to tolerate food well
.
o/e
alert GCS 15
fundoscopy bilat partially observed optic disc margins, distinct
reflexes 2+ throughout plantars flexor
sensation intact throughout
power 5/5 all 4 limbs prox and distal
gait narrow based
no dysmetria
.
impression
migraine
green flags: intermittent, low frequency, clear periods of wellness. symptoms not suggestive of raised ICP, IIH, no focal neurology
.
Plan
ESR in view of age >50, low index of suspicion
simple analgesia
hold off preventors for now
TCU 3m rv
no clear indication for imaging at the moment

Green flags reassure — they don't eliminate the need for clinical judgement on their own.

Approach to management of migraine

Lifestyle

- Regular sleep & meal timing
- Hydration
- Headache / trigger diary
- Moderate, consistent caffeine
- Stress & routine management

Relievers

- Triptans, taken early in the attack
- NSAIDs ± antiemetic
- Avoid overuse — watch for medication-overuse headache
- Treat the attack, not the day after

Preventers

- Consider once attacks are frequent or disabling
- Beta-blockers, amitriptyline, topiramate
- CGRP-targeted agents for refractory cases
- Reassess response over 8–12 weeks

SECTION 3 OF 5

Approach to Weakness & Sensory Loss

A localisation approach

These aren't a checklist to memorise. They're examples of the where/what and red/green reasoning from Part One, applied to a single presenting complaint.

A	**Diabetic neuropathy**	*Concordant, severity-matched, length-dependent*
B	**Inflammatory neuropathies & MMN**	*“Potentially sinister” — doesn't fit the metabolic pattern*
C	**Stroke**	*Hyperacute by definition — Bamford / OCSP syndromes*
D	**Parkinsonism**	*Often described by patients as “weakness”*
E	**Musculoskeletal mimics**	*Caveated — check the distribution fits*

Diabetic neuropathy — an example of “fits the pattern”

GREEN FLAG

When the pattern is concordant

- Symmetrical, length-dependent (“glove and stocking”) sensory loss, worse distally
- Severity broadly tracks disease severity — persistently elevated HbA1c over time
- Other end-organ evidence of diabetic microvascular damage: established nephropathy, retinopathy
- The story coheres: one process, one plausible mechanism, matched severity

This is the green-flag reasoning from Principle 2 in action: it localises, and the pathology it points to explains the whole picture — no further chasing required.

Inflammatory or malignant neuropathies— “potentially sinister”

RED FLAG

When the pattern doesn't fit a metabolic story

- Guillain-Barré syndrome / CIDP — ascending, progressive, areflexic, often over days to weeks
- Multifocal motor neuropathy (MMN) — asymmetric, motor-predominant, follows individual nerve territories rather than a length-dependent glove-and-stocking pattern
- Neoplastic/Paraneoplastic neuropathies related to MM, monoclonal gammapathies, paraneoplastic disease
- Amyloidosis
- Common thread: asymmetric, motor-predominant, or progressing faster than a metabolic process would explain

Treated here as a category, not a checklist — the point is recognising “this doesn't look diabetic”, not naming the exact syndrome.

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Stroke — hyperacute by definition

Sudden-onset focal neurology is a red flag regardless of severity. The Bamford (OCSP) classification describes the hyperacute syndromes:

TACS	PACS	POCS	LACS
Total Anterior Circulation	Partial Anterior Circulation	Posterior Circulation	Lacunar
All three of: hemiparesis/sensory loss, homonymous hemianopia, higher cortical dysfunction (e.g. dysphasia, neglect)	Two of the above three, or higher cortical dysfunction alone	Brainstem, cerebellar, or isolated homonymous hemianopia — often subtler, easily missed	Pure motor, pure sensory, or sensorimotor — no cortical signs, small vessel territory

Hyperacute onset is itself the localiser — the syndrome tells you roughly where, before any imaging.

Parkinsonism, misread as “weakness”

Patients frequently describe bradykinesia and rigidity as “weakness”

Listen for the actual descriptors: *“slow”, “stiff”, “dragging my leg”, “my writing has shrunk”* — rather than a true reduction in power on formal testing.
This is a mimic worth catching early: the workup for “weakness” and for parkinsonism diverge quickly, and formal power testing is usually preserved.

Most easily elicitable green flag:

Chronic, slowly progressing (ie not hyperacute)

Musculoskeletal mimics — a caveated green flag

GREEN FLAG

When the distribution doesn't fit a neurological pattern

- “Weakness” confined to a single joint movement
- Reminder: check specifically for rotator cuff pathology — a commonly missed cause of apparent shoulder “weakness” that is actually pain-limited or tendon-related
- The caveat: this is only reassuring once you've confirmed the distribution genuinely doesn't fit a root, plexus, or peripheral nerve pattern, AND DOCUMENTED IT DOWN

Green flags still require you to have actively excluded the red-flag pattern — not just to have not thought of one.

SECTION 4 OF 5

Approach to Movement Disorders

Define the common phenomenologies

Tremor

Rhythmic, oscillating movement around a joint axis

Dystonia

Sustained or intermittent muscle contractions causing abnormal postures

Chorea

Brief, irregular, unpredictable “dance-like” movements that flow between body parts

Myoclonus

Sudden, brief, shock-like jerks

Tics

Stereotyped movements or sounds, often suppressible, preceded by an urge

Bradykinesia / rigidity

Slowness of movement and increased resistance to passive movement

Tremor, in more detail

	Essential Tremor	Parkinsonian Tremor
Character	Postural / kinetic	Rest tremor
Pattern	Bilateral, symmetric	Asymmetric, unilateral onset
Classic sign	Improves transiently with alcohol	“Pill-rolling” quality
Associations	Often positive family history	Bradykinesia, rigidity, other parkinsonism

Essential Tremor

Example documentation

65F
.
c/o bilat tremor
R>L
mostly action
no rest tremor
worsened with anxiety, coffee
does not take alcohol
mildly interferes with use of cutlery
no speech difficulty, no head involvement, no falls, no gait difficulty
no postural giddiness
.
o/e
alert GCS 15
EOM full no nystagmus
saccades/pursuit NAD
reflexes 2+ throughout plantars flexor
power 5/5. all 4 limbs prox and distal, sensation grossly intact to pinprick, no glove and stocking sensory loss
no resting tremor despite stress manoeuvres and distraction
no dysmetria bilat
bilateral R>L high frequency low amplitude postural tremor
no cogwheeling/rigidity
no bradykinesia on finger opening/closing
narrow based gait, prompt initiation, no decrease in arm swing bilat
turns in 2 steps
.
impression
likely essential tremor
no signs of parkinsonism
.
Plan
offer symptomatic medications (primidone or propranolol)
6m review symptoms for stability

Approach to parkinsonism — two steps

i Identify parkinsonism

Bradykinesia, plus rest tremor and/or rigidity. This is a clinical, examination-based diagnosis before it is anything else.

ii Screen for secondary causes and "PD+"

Not exhaustive — examples of the flags that shift thinking away from idiopathic Parkinson's disease:

- Early falls or postural instability
- Symmetric onset
- Poor or absent response to levodopa
- Early autonomic failure or dysphagia
- Vertical gaze palsy
- Causative drugs (e.g. antiemetics, antipsychotics)

iPD

Example documentation

65F
.
c/o bilat tremor
R>L
occurs at rest
feels more stiff and slow
no falls
worsening over past 6m to a year
no REM sleep behaviour disorder symptoms per partner
no postural dizziness
still able to ride bicycle
not on antipsychotic/antiemetic
no cognitive complaints
.
o/e
alert GCS 15
EOM full no nystagmus
saccades/pursuit NAD
reflexes 2+ throughout plantars flexor
power 5/5. all 4 limbs prox and distal, sensation grossly intact to pinprick, no glove and stocking sensory loss
mild resting tremor (pill rolling) R>L
midl cogwheeling R>L
no bradykinesia on finger opening/closing
narrow based gait, prompt initiation, slight decrease in arm swing R>L
no hypomimia
no stooped posture
turns in 2 steps
.
no dysmetria bilat
EOM full, vertical and horizontal motion full
mildly broken saccades
no eyelid apraxia
no limb-kinetic apraxia bilat
two point discrimination NAD
postural BP lying to standing no drop
no retropulsion on pull test
.
impression
early idiopathic PD
no signs of PD+ at present
green flags: asymmetry, no early falls
.
Plan
commence MADOPAR 62.5mg BD -> TDS
counselled on SE
3m review then 6monthly after if stable/well
PT and exercise advice

SECTION 5 OF 5

Approach to Dizziness, Funny Turns & LOC, and gait difficulty

When the history leans toward syncope

Autonomic Neuropathy

Autonomic signs and symptoms
Usually accompanied by another neurological localization (alpha synucleinopathies, peripheral neuropathy)

Endocrine

Thyroid dysfunction, adrenal insufficiency, hypoglycaemia

Cardiovascular

Arrhythmia, valvular disease, orthostatic hypotension

Not every "funny turn" belongs to neurology — the extraneurological work-up is often where the answer actually is.

The differential of loss of consciousness

Scope note: this section focuses on transient LOC — seizure vs. syncope — not peripheral vertigo.

RED FLAG

Clues pointing toward a neurological (seizure) cause

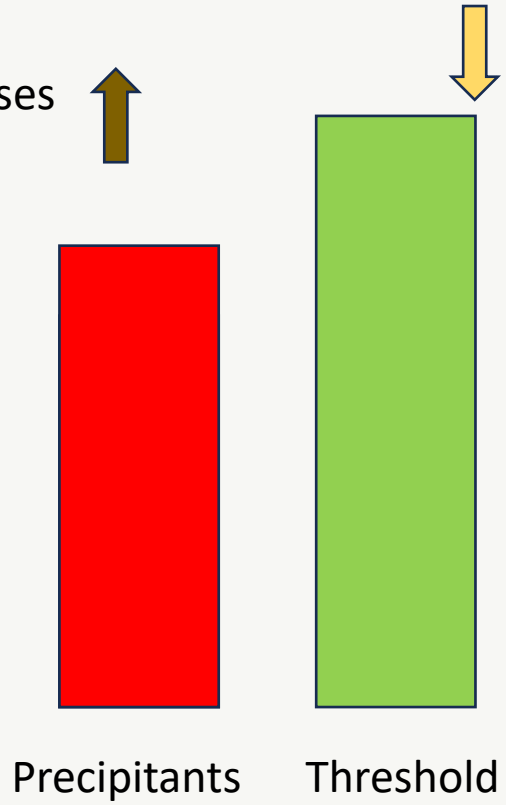
- Known focal cortical lesion on prior imaging or history
- Stereotyped symptoms and semiology — the same aura, the same sequence, every time
- Post-ictal confusion, lateral tongue-biting, or an injury pattern consistent with a convulsive event
- Preceding aura (rising epigastric sensation, déjà vu, unusual smell) rather than a graying-out sensation

An overview of seizures and epilepsy

Concept of Precipitants (or Triggers), and Thresholds

Anything that causes stress to the brain

- exceptionally high or low blood sugar
- severe lack of sleep
- severe psychological stress
- infections
- infections in the brain
- etc



Precipitants Threshold

Anything that disturbs the “normal” structure and function of the brain

Examples
- Scars from previous injury
- Scars from previous strokes or bleeds
- Damage from previous brain infections
- Congenital brain lesions
- No clearly identifiable cause

Having Epilepsy means having a lowered threshold

An approach to dizziness

First question (as always): Is there objectively a localization

Vertiginous vs non vertiginous is technically a differentiator but is often unreliable

Burnish with clinical examination to exclude

1) Cerebellar disease
2) Vestibular disease
3) Proprioceptive disease (not technically dizziness)

An approach to gait difficulty

The orders system

First Order

Weakness and sensory loss

Second order

Myelopathy

Third order

Ataxia

Apraxia (higher cortical)

Example documentation

68M
.
HLD
HTN
.
c/o intermittently veering to right
.
Today
history revisited
for past 4-5 months
intermittently veering to right when walking
not accompanied by presyncopal symptoms. not a/w palpitations etc
no lateralising weakness, no speech difficulty, no diplopia
resolves promptly
no falls
accompanied by transient sense of imbalance but no vertigo per se
no loss of sensation in limbs, no paresthesias/burning sensation
.
o/e
alert GCS 15
EOM full
pursuit/saccades NAAD
no facial/tongue/palatal asymmetry
reflexes 2+ throughout plantars flexor
power 5/5 all 4 limbs prox and distal
sensation intact to proprioception and vibration in bilat LL
no visual/tactile neglect
no aphasia
visual fields full to confrontation
.
no dysmetria bilat. heel shin NAD
gait narrow based. tandem gait NAD
Dix hallpike bilat NAD
head thrust bilat NAD
Romberg's NAD
.
imp
nonspecific transient gait imbalance
?vestibular hypofunction, no explicit signs
green flags: no objective cerebellar deficits, no lateralising motor/sensory symptoms, no sign of peripheral neuropathy
.
Plan
open date
red flag symptoms advised (FAST)

CURVE BALL

WHERE SPOIL?

- Which neuro system is affected?
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ALL OF NEUROLOGY IS MERELY A 2.5 STEP PROCESS

WHY SPOIL?

- VITAMIN-C

WHERE ELSE SPOIL

- Other physiological systems
- Other neurological systems

.
O.e
AMT 10/10
prompt with conversation
able to recall recent events
able to change topic promptly
reflexes 2+ thropughout
plantars flexor
no dysmetria
tandem gait, gait NAD
no palmomental sign, no grasp reflex
EOM full no nystagmus
no myoclonus
no cogwheeling/bradykinesia/rigidity
no apraxia bilat
sensation grossly intact bilat prox and distal
.
Imp
no current explicit signs of delayed neurological syndrome
no movement disorder (myoclonus, ataxia, parkinsonism)
no cerebellar ataxia
no cortical dysfunction (no apraxia, no explicit signs of cognitive dysfunction in executive function, attention, memory as evaluated in clinic)
no sign of peripheral neuropathy on examination
.
Plan
TCU 3m KIV refer for neuropsych evaluation
follow up over 1 year at least
MRI brain to ix for basal ganglia changes or leukoencephalopathy

BEFORE YOU GO

Four things to carry back to clinic

1. Localise before diagnosing.
2. Patterns matter more than isolated symptoms.
3. A diagnosis is ideal — a defensible assessment is essential.
4. Not every symptom identifies a disease. Think in terms of whether it fits into a pathological pattern.

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Thank You

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