Parkinson’s disease, Motor Neurone Disease, and Multiple Sclerosis

Malcolm Steiger
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PD – clinical features

- Tremor
- Rigidity
- Bradykinesia

- +/- Postural instability
Parkinsonian tremor

- 3-6 Hz
- At rest, more than posture
- Asymmetrical
- Associated with rigidity and bradykinesia
- Increased with distraction / anxiety
Parkinsonian Gait

- Slow, shuffling
- Narrow base
- Progressive difficulties with time
  - Freezing
  - Start hesitation
  - Festinating
  - Propulsion / retropulsion
Autonomic symptoms

- Urological:
  Urgency, frequency, nocturia.
- Sexual dysfunction – impotence.
- Orthostatic hypotension and progressive cardiac sympathetic denervation.
- Constipation – slow colonic transit times, decreased rectal contractions.
- Increased perspiration particularly in “off”.


Atypical features

- Poor response to Rx (MSA)
- Autonomic failure (MSA)
- Pyramidal / cerebellar signs (MSA)

- Early postural instability (PSP)
- Mainly axial symptoms (PSP)
- Ophthalmoplegia (PSP)

- Gait apraxia (CV disease, NPH)

- Hemiatrophy; persistent hemiparkinsonism (HPHA)

- Dementia (DLB)
The importance of a long term treatment plan – Life expectancy in the general healthy population

United Kingdom 2005 Life Expectancy Statistics - Government Actuary's Department

UK Actuary's Department
Overall aim of treating PD

■ To improve QoL and maintain motor function by giving patients:

- An individualised management plan
- Relief from the main symptoms
- Longest delay of motor complications
- Minimised side effects
When do you start treatment?

- Prospective QoL study in drug naïve (DN) and new monotherapy (MT) PD cases

- DN cases still DN at 11 months have significantly worse PDQ39 than those DN now on Rx

- MT cases have no worsening of PDQ39 at 18 months compared to start of study

- Should we delay the start of treatment?

Chaudhuri 2005
Myths in therapy of PD

- Delayed initiation of dopaminergic therapy for parkinsonism is advocated
- Therapy may lose effectiveness with time
- In addition, it has been suggested that early treatment with levodopa may hasten disease progression.
- A number of studies have found no evidence to support this belief e.g. Diamond and Markham found that disability from parkinsonism is related to disease duration not duration of levodopa treatment
Biological v chronological age

- ‘Because DA monotherapy causes significantly less dyskinesia, treatment of early PD in the younger, healthier patient generally begins with DAs’

- Many patients at diagnosis are suitable for initiating on a DA but taking into consideration
  - Employment issues
  - Life expectancy
  - Morbidity
  - Current lifestyle

Samil 2004
Parkinson’s disease is a significant health problem in developed countries.

1% of people over 65 years suffer from the condition.

Progression of disease associated with decline in quality of life and increasing health care costs.

Associated increase in health care usage.
PD patients are more likely to be hospitalised.
■ 3x more likely to use skilled nursing facilities.
■ 2x more likely to use home care compared to other patients with chronic illnesses.
■ Also, higher comorbidity levels in PD patients contribute to higher medical expenditure.
■ Particularly trauma, mostly due to falls, diabetes, dementia and cerebrovascular disease.
■ (Murman et al Neurology 2003)
Findley et al, Movement Disorders 2003.

- Mean annual cost of care for all patients by age was £5,993
- NHS costs accounted for 38%
- Social service costs for 34% of costs of care
- Drug expenditure accounted for:
  - 24% of overall costs in < 65 year age group
  - 10% in > 85 years
Initiation with L-dopa?

- For initiation with L-dopa
  - Most effective symptomatic Rx for PD\(^1\)
  - Cost effective in the short term

- Against initiation with L-dopa
  - Significant risk of motor fluctuations including\(^1\):
    - Wearing off phenomena
    - On-Off motor fluctuations
    - Dyskinesias

Olanow 2001
Elldopa study

- Designed to test the hypothesis that L-dopa is toxic to dopaminergic neurons
- Demonstrated clinical improvement in those randomized to L-dopa compared with placebo after 9 months, even after the 2 week wash-out
- Explanations for the better clinical improvement in patients randomized to symptomatic therapy include:
  - Inadequate washout period
  - Direct neuroprotection
  - Restoration of intrinsic basal ganglia compensatory mechanisms by L-dopa, the beneficial effects of which persisted beyond the withdrawal of L-dopa
Ell-dopa study

- RCT of L-dopa as Rx in early PD
- Placebo vs 3 regimens
  - L-dopa at a dose of 150mg daily
  - L-dopa at a dose of 300mg daily
  - L-dopa at a dose of 600mg daily
- There was a strong dose response benefit (improvement in UPDRS) which persisted through to week 40
- Adverse events were significantly higher for those patients receiving 600mg L-dopa
  - Especially dyskinesias, nausea, infection, hypertonia & headache
- There was a greater decrease in \([^{123}]\beta\)-CIT uptake among those patients receiving L-dopa, especially the L-dopa 600mg daily group

PSG 2004
Which drugs to use when starting treatment in early PD is a complex decision that depends on factors such as:

- disease severity
- functional disability
- psychosocial handicap

as well as:

- age
- employment status
- cognitive impairment
- co-morbidity
Elldopa study
- PD patients were randomized to rasagiline or placebo for 6 months, after which the placebo group received rasagiline.
- Those patients randomized to receive rasagiline early had better clinical (UPDRS) scores at 12 months than those transferred from placebo 6 months later.
- The results of this “delayed start” trial cannot be explained by symptomatic effect.
Early treatment with rasagiline: TEMPO UPDRS scores

Primary analysis: 371 subjects

- Rasagiline 1 mg
- Rasagiline 2 mg
- Delayed 2 mg rasagiline

* p=0.05
** p=0.01
Initiating DAs for new cases

- DAs can be as effective at maintaining patients ADL over 5-10 years as L-dopa

- DAs are effective at controlling resting tremor

- Over a third of patients can be managed for at least 5 years before supplemental L-dopa is required

- Over 10 years patients started on ropinirole have a significantly lower risk of developing dyskinesias than patients started on L-dopa

- It is essential to titrate patients to a therapeutic dose of a DA - Patients on low dose agonists can see worsening of symptoms due to presynaptic endogenous dopa release inhibition (Kellett & Steiger).

Reasons for NOT initiating a DA

- Significant cardiovascular history
  - e.g. heart failure, severe angina
- Significant cognitive impairment
  - e.g. dementia

See also specific contraindications, warnings and monitoring requirements of individual DAs

Ropinirole, pramipexole, cabergolline, pergolide & Bromcriptine SPC
There is a much greater risk of developing dyskinesias in patients managed on L-dopa versus DAs.

Rascol 2000
A number of clinical trials have indicated that early initiation of treatment results in a slower decline in UPDRS scores than if treatment is initiated later.

Initiation of dopaminergic treatment can be associated with unwanted side effects that may include gastrointestinal disturbances, cognitive problems, and sedation.

But, symptomatic improvement and the hypothetical long-term neuroprotective and neurotrophic benefits.

The most important advance for PD will come with the development of a treatment that will slow or prevent disease progression.
**Stages of Parkinson’s Disease**

- **Presymptomatic phase of disease**: c 4-10 years
- **Abnormal rate of dopaminergic neuron deterioration begins**
- **Onset of symptoms**
- **Diagnosis**
- **Maintenance**
- **Complex**
- **Palliative**
- **Death**

- **Disease can be managed without dopaminergic therapy for c 1 year**
- **Symptoms/motor complications can be managed with dopaminergic therapy for c 5-10 years**
- **Motor complications develop c 5 years**
- **Cognitive decline c 5 years**

**Dopaminergic neuron number and function**

**Severity of the disease**

Adapted from Olanow 2001 & Baker 2003
Managing complex motor problems

Advancing Parkinson's disease

Motor complications

Motor fluctuations

Suboptimal clinical response

End of dose or “Wearing-off” phenomenon

Delayed “on” or no “on” response

Unpredictable “off” episodes

Dyskinesia

Peak dose

Dystonia

Diphasic

Freezing

Adapted from Olanow 2001
Dyskinesias

- Choreathetoid
- Can significantly interfere with gait, balance and quality of life
- Erratic L-dopa levels in advanced disease provokes dyskinesia

Adapted Thanvi 2004
Strategies for fluctuation

■ More levodopa
■ Add agonist to levodopa
■ COMT inhibitors
  ☐ Entacapone (“Stalevo”)
■ Madopar dispersible 62.5 / 125
■ Apomorphine
■ Surgery
■ DUODOPA
Rationale for adding DAs to L-dopa

- DAs can reduce "off" time by 20%
- DAs can reduce the dose of L-dopa
- DAs can improve CGI scores compared to placebo
- DAs improve UPDRS score

Liberman 1998, Mizuno 2004
Titrating dopamine agonists – in patients on L-dopa monotherapy

- Use the same regime as de novo patients

- Titrate gradually until there is a noticeable therapeutic effect

- The concurrent dose of L-dopa may be reduced gradually – the optimal balance should be determined by the clinician

- Once a therapeutic response is observed ONLY increase dose when motor symptoms deteriorate
COMT inhibitors improve L-dopa kinetics

Olanow 2001, Ruottinen 1996
COMT inhibitors improve L-dopa kinetics

Olanow 2001
MAO-B Inhibitors

- Two alternatives
  - Selegiline
  - Rasagiline
- Once a day
- Side effects may include: Back pain, headache, dizziness, insomnia, hallucinations

Once a day rasagiline reduces mean daily off time in an effect similar to entacapone

<table>
<thead>
<tr>
<th>Drug</th>
<th>Reduction in mean daily ‘Off Time’</th>
<th>Increase in ‘ON Time’ without troublesome dyskinesia’s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rasagiline</td>
<td>-1.18 hrs</td>
<td>+0.85 hrs</td>
</tr>
<tr>
<td>Entacapone</td>
<td>-1.2hrs</td>
<td>+0.85 hrs</td>
</tr>
</tbody>
</table>

Rascol 2005
Subcutaneous injections and infusions

- **Apomorphine**
  - Used to treat disabling motor fluctuations that persist despite ongoing treatment with L-dopa and/or DAs
  - Administered either:
    - As a rescue Intermittent injection by patient
    - Continuous infusion
  - It is essential that a patient is established on domperidone two days prior to initiation to avoid severe nausea and vomiting
  - An apomorphine challenge needs to be carried out by a PDNS in a specialist clinic or as a day case with medical backup
  - Dose is dependent on the outcome of the apomorphine challenge.
  - Long term use of apomorphine via infusion can be associated with reduction in dykinesia.
Duodopa

- Duodopa consists of the active substances L-dopa and carbidopa.
- It is administered by continuous infusion into the small intestine.
- Costly and requires special funding on a case by case basis.
Dementia in PD

Associated with:

- Reduced quality of life of patients and caregivers
- Rapid functional and motor decline
- Loss of independence / Behavioural problems
- Increased care in the community and/or Nursing Home care due to increasing disability.
Dementia in PD

- 26% at baseline; 80% at 8 years
- Most look like Lewy body dementia
- "subcortical dementia"
  - Slowing
  - Executive dysfunction
  - Memory impairment
  - Depression
- Resembles Alzheimer's (cortical features)

- Pathology = LBD / AD / mixed
- P.D. Dementia can be difficult to treat.
- Consider quetiapine as antipsychotic of choice.
- The effects of Cholinesterases are modest yet significantly effective in many patients.
- Useful in reducing hallucinations, and increasing attention and alertness
- May preserve the independence and well-being of the patient.
- Reduce the burden on caregivers.
- Involving primary care at the start more likely to aid communication and clinical effectiveness.
Conclusion

- Management of PD in the individual patient requires an holistic approach involving patient, family and carers.
- Problems of motor and non-motor type need to be recognised and predicted.
- Strategies in dealing with the illness throughout the disease are recognised.
- A team approach involving patient, family/carers, primary care, therapists, PD Nurse Practitioners, and social services can assist in reducing the impact of the disease.
Motor Neurone Disease
Relaying Messages in the Motor System

[Diagram showing neuronal pathways and muscle connections]
Upper motor neurone signs

- Dysarthria
- Emotional lability
- Spasticity, clonus
- Paresis
- Decreased fine movements
- Brisk reflexes
- Upgoing plantars
Lower motor neurone signs

- Wasting
- Weakness
- Fasciculations
<table>
<thead>
<tr>
<th>Medulla</th>
<th>Upper Motor Neurone lesion</th>
<th>Pseudo Bulbar Palsy (other causes - including stroke)</th>
<th>Tongue spastic, no fasciculation Speech spastic explosive, dysarthria Increased reflexes Emotional lability</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Upper &amp; Lower Motor Neurone lesions</td>
<td></td>
<td>Dysarthria Dysphagia Wasting of tongue Jaw jerk reflex increased</td>
</tr>
<tr>
<td></td>
<td>Lower Motor Neurone lesion</td>
<td>Bulbar Palsy</td>
<td>Tongue - shrunken, wrinkled - fasciculating Speech slurred Dysphagia Paralysis of diaphragm</td>
</tr>
<tr>
<td>Cortico Spinal Tract</td>
<td>Upper Motor Neurone lesion</td>
<td></td>
<td>Spastic weakness Stiffness Increased reflexes Extensor plantar responses</td>
</tr>
<tr>
<td>Anterior Horn cells</td>
<td>Lower Motor Neurone lesion</td>
<td></td>
<td>Flaccid weakness Muscle wasting Muscle fasciculation</td>
</tr>
</tbody>
</table>

Subtypes

- Progressive muscular atrophy
- Amyotrophic lateral sclerosis
- Progressive bulbar palsy
Motor Neurone Disease (Amyotrophic Lateral Sclerosis)

- A motor system degeneration with combined upper and lower motor neurone involvement
- Progressive
- Disease of adulthood (some exceptions)
- 10% genetic; SOD1 gene mutation identified in 20% of familial cases
- Incidence 2.4/100,000
- Prevalence 6.8/100,000
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Percutaneous endoscopic gastrostomy

Gastrostomy tube

Jejunal feeding tube
Percutaneous endoscopic gastrostomy and enteral nutrition in amyotrophic lateral sclerosis
Overnight pulse oximetry

Signs of hypoventilation

• Disturbed sleep
• Frequent awakenings - patient not always aware, pulse ↑
• Nightmares
• Morning headaches
• Excessive daytime sleepiness
Non-invasive Positive Pressure Ventilation (NIPPV)

Non-invasive ventilation using an interface as opposed to invasive ventilation using endo-tracheal intubation.

**Interface:**
- Full face mask
- Nasal mask
- Mouth piece
- Nasal pillows
Advantages of NIV

- RCT has shown it to prolong life
- Avoids complications of intubation – infection
- Rests the respiratory muscles – decreases work of breathing
- Provides adequate $O_2$ and clearance of $CO_2$
- Patients can continue to communicate, eat and drink
- May improve quality of life
Disadvantages

- It is important that patients are aware that NIV will not “treat” their MND—it is used to palliate symptoms
- Prolongation of life may lead to reduction in quality of life
- Added burden to carers
- Patients can become increasingly dependent on the ventilator
- Mask problems
Multiple Sclerosis
Outline

- Relevant background
- Diagnosis
- Relapse
  - Identification
  - Management
- Disease modifying treatment
  - Current thinking
- Medical review
Multiple Sclerosis

- Commonest cause of acquired neurological disability in young adults
- Long term personal/family/financial implications of MS demand accurate and prompt diagnosis
- Misconceptions amongst patients and professionals about the nature and prognosis of MS
- Evolving treatment options in (early) relapsing disease
- Many symptoms amenable to treatment
  - Which can improve quality of life
MS: Facts and figures

- Affects 1 in 600-800 in the UK
  - 2-4/GP list

- F:M ratio 3:1
  - Incidence clearly increasing in women

- Age range 10-60, peak at 20-40

- Natural history 30 years+, limited impact on life expectancy for the majority

- At 15 years from onset:
  - 1/3 no/minimal disability
  - 1/3 moderate disability
  - 1/3 significant disability
Disease evolution in MS

Inflammation

- (relapses)

Axonal injury/demyelination

- (persistent relapse related disability)

Anti-inflammatory agents

Axonal (nerve) degeneration

- (late - progression)

Repair strategies?

Neuroprotection?
MS spectrum

Asymptomatic

Definite MS

‘Malignant’ MS

‘Of uncertain prognosis’

Single episode demyelination

(CIS – clinically isolated syndrome)
Making a diagnosis of MS

- Does the clinical syndrome fit?
- Do the investigations fit?
- Is there a better explanation?
- Is there dissemination in time *and* space?
Clinical features

**History**
- Temporal pattern
  - 85% RR onset
- Minor antecedent events
  - Episodic fatigue
- Uhtoff’s/Lhermitte’s
  - Other paroxysmal symptoms
  - Other characteristic sensory symptoms
- Family history

**Examination**
- Typical clinical features
  - Asymmetric SP
  - Subtle INO
  - Disc pallor
- Red flags
  - Joint disease
  - Rash
Investigation

- Should be targeted!

- In most:
  - MR brain +/- cord
  - VER (?)
  - Bloods: FBC/ESR, B12 + F, A. Abs

- In some
  - CSF (*but not with steroids...*)
  - Other......
MR in MS

- Always review the scans!
  - Beware the General Radiologist..

- Lesion distribution/morphology
  - Peri-ventricular
  - Callosal/‘Dawson’s finger’
  - Juxta-cortical
  - Posterior fossa
  - Cord
Diagnostic issues in 2009

- Non-specific/minor MR findings
  - Over-reporting in patients with non-specific symptoms

- Other inflammatory disorders
  - Anti-phospholipid syndrome
  - Sjogren’s

- CADASIL

- Other demyelinating conditions
  - NMO/NMO-spectrum disorders
Who should I be worried about?

- High early relapse rate
  - >2 in first 2 years
- Disabling relapses
  - Motor/sphincter
- Incomplete recovery
  - Signs
- Later onset disease (RR)
  - Shorter RR phase
- High MR lesion load
  - >10 lesions

- Miss NA, Age 30
  - 3 relapses in 2 years from onset
  - Motor involvement in attacks
  - Minor residual disability (EDSS 1.5) at 2 years

- What is her risk (untreated) of EDSS 6 or greater at 10 years?
  - ~80%
Relapsing MS

- Prolonged RR phase more likely in younger patients
- Relapses are unpredictable and (untreated) occur on average every 18 months
- Infections and stressful life events increase risk of relapse
- Less than 50% of relapses require treatment
  - Sensory vs motor/cerebellar
- High dose steroids accelerate recovery but probably don’t change outcome.
  - Methylprednisolone 1g IV x3 or 500mg oral, 5 days
DMT’s in MS - Background

- 12 years of disease modifying therapy
  - Interferon/Glatiramer Acetate
  - Benefits largely confined to RRMS

- Limited evidence of long-term benefit
  - Disability/delay SPMS

- Move to early treatment
  - CIS: ETOMS, CHAMPS, BENEFIT, (PRECISE)

- Highly variable natural history
  - Difficult to predict in early disease?

- New (more effective/higher risk) therapies
  - Tysabri, CAMPATH-1H, Mitoxantrone
  - Where do they fit?
‘Emerging’ therapies

- Natalizumab (Tysabri)
- Alemtuzumab (CAMPATH)
- Mitoxantrone

…First or second line??
Natalizumab (Tysabri)

- Relapses
  - Reduced by 68%
  - Onset of action – within 12 weeks

- Disability
  - 42% reduced risk of disability progression

- Safety
  - Very well tolerated (patients felt ‘well’)
  - But….
    - 3 cases of PML (in 3000) – longer term risk unknown
Months before and after Campath-1H treatments

Correct to 1 June 2004

91% reduction in relapse rate after Campath-1H.

Before treatment
2.21 relapses/patient/year

After treatment
10 investigator-confirmed episodes, 0.19 relapses/patient/year (p<0.0001).

19/22 2 treatments
4 have had 3 treatments 12-30 months after 2nd treatment
Time to Sustained Disability

Number needed to treat with alemtuzumab to prevent an additional patient from acquiring fixed disability over 2 years, compared to IFNB-1a:

- 5.8 - 8.1

Risk Reduction:

- 66%
- 88%

P < 0.01

P < 0.001
MS medical ‘review’

- **Symptom review**
  - Common symptoms
    - Mobility, spasms, bladder
  - Hidden symptoms
    - Fatigue, sexual dysfunction, depression

- **Drug therapy review**
  - Interactions/exacerbating MS symptoms

- **Suitability for DMT?**
  - RR? Relapses?

- **Therapy services**
  - OT/PT
  - SALT
  - Social work

- **Signposting**
  - Appropriate local services
  - Carers

- **Screening**
  - Cardiovascular
  - Cancer
MS: resources

■ Local
  □ MS nurse specialists: 0151 529 5645
  □ MS specialist therapists (OT/PT/Psychol): 0151 529 5071
  □ MS Neurologists: Prof Young, Dr Wilson, Dr Boggild, Dr Jacob
    (via WCNN: 0151 525 3611)

■ National
  □ UK MS Society
  □ MS Trust
Conclusion

- Holistic approach begins in the diagnosis and extends throughout the disease course
- Important to recognise key features of these illnesses but also recognise that patients symptoms may not always be directly due to their neurological illness.
- Partnership with Primary Care is essential.
- Team approach works well recognising our different but synergistic roles working with the patient and their carer(s).
Clinical Hot Topics for General Practice

Dr Denis O’Brien
The Elms Surgery Liverpool
Managing Back Pain - An Update

Denis O’Brien 4th July 2009, RCGP Hot Topics Meeting
Mechanical (non-specific) LBP

“Non-specific low back pain is tension, soreness and/or stiffness in the lower back region for which it is not possible to identify a specific cause of the pain. Several structures in the back, including the joints, discs and connective tissues, may contribute to symptoms.”

NICE May 2009
“Back pain should be viewed as a chronic problem with an untidy pattern of grumbling symptoms and periods of relative freedom from pain and disability interspersed with acute episodes, exacerbations, and recurrences.”

Croft et al (1998)
What’s new?

• Not very much!
• Alexander technique (BMJ 2008;337:a884)
• NICE Guidance – back pain >6 weeks <12 months (May 2009)
The size of the Problem

- Back pain is the third most common bodily symptom after headache and tiredness.
- 5-7% of adults have almost constant back pain.
- 60% of people with LBP also have neck problems.
The Size of the Problem 2

- Pts attending with back or neck pain are more likely to consult with stress and mental disorders.

- Pts attending GPs with back pain also attend more frequently with other complaints.
Figure 1.1  The rising trend of low back disability from 1953–1954 to 1994–1995. Based on annual statistics from the UK Department of Social Security.
Adults with Back Pain (Period Prevalence)

- Today: 14 – 30%
- This month: 30 – 40%
- This year: 36 – 37%
- Lifetime: 60 – 80%
Recovery Rates from LBP Episode

- Two days: 30%
- One month: 50%
- Six weeks: 90%
- Twelve months: 97%
Rates of Return to Work

- Back to work in:
  - 2 days - 35%
  - 7 days - 67%
  - 2 weeks - 75%

- Still off work after:
  - 1 month - 16%
  - 6 months - 4%
  - 12 months - 3%
Diagnostic Triage

- Ordinary (simple, mechanical) backache
  - 95% SIMPLE

- Nerve root pain
  - < 5% SCIATICA

- Serious spinal pathology
  - < 1% SERIOUS
Physical Examination

• Important

• Limitations of SLR
Sciatica - characteristics

• Unilateral limb pain > back pain
• Radiates into foot
• Numbness / paraesthesia in dermatomal distribution
• SLR induces more leg pain (nerve tension)
• Localised neurology
Case Study

• 38yo male. Referral letter: 6m Hx. No trauma. Limited SLR. No neuro signs.

• RTA Dec 06. Truck Driver. Truck flipped over in high winds. Litigation with former employers about safety. Flashbacks / loss of confidence.

• GP records – also RTA July 07!
Investigations – Plain X-ray

• 1.5 million in UK each year (0.6 million by GPs)

• 3% of all medical / dental x-rays, 12% total radiation

• Equivalent to 120 Chest x-rays
Investigations - MRI

• 200,000 L-spines in UK per year – 25% of total

• Studies of MRI in asymptomatic subjects:
  – bulging discs - 20 - 79%
  – herniated discs - 9 – 76%
  – degenerative discs - 46 – 91%
What to Tell Patients

• Most of us will get back pain.
• It does not make much difference whether male or female, young or old, tall and thin or short and fat.
• There is little evidence to suggest that LBP is ever work related.
What to Tell patients 2

• Back pain is a symptom not a disease
• The cause is rarely detected
• Most episodes occur spontaneously
“Many patients are reluctant to accept, and many doctors or therapists to admit, the limitations of treatment for back pain”
“The choice of treatment often reflects the skills of the professional rather than the needs of the patient.

Who you see is what you get! ”

The Back Pain Revolution.

Gordon Waddell
“Know as much about the patient who has the back pain as about the back pain the patient has”

The Back Pain Revolution.
Gordon Waddell
The back pain “market” is a humming, economic machine that produces billions in revenue annually.

Editor, The BackLetter
Offer one of the following treatment options, taking into account patient preference:

- an exercise programme
- a course of manual therapy
- a course of acupuncture
- Consider offering another of these options if the chosen treatment does not result in satisfactory improvement.
Physical activity and exercise

• Consider offering a structured exercise programme tailored to the person: This should comprise up to a maximum of eight sessions over a period of up to 12 weeks.

• Offer a group supervised exercise programme, in a group of up to 10 people.

• A one-to-one supervised exercise programme may be offered if a group programme is not suitable for a particular person.
Manual therapy

Consider offering a course of manual therapy, including spinal manipulation, comprising up to a maximum of nine sessions over a period of up to 12 weeks.
Acupuncture

• Consider offering a course of acupuncture needling comprising up to a maximum of 10 sessions over a period of up to 12 weeks.

• Do not offer injections of therapeutic substances into the back for non-specific low back pain
Combined physical and psychological treatment programme

Consider referral for a combined physical and psychological treatment programme, comprising around 100 hours over a maximum of 8 weeks, for people who:

- have received at least one less intensive treatment

  and

- have high disability and/or significant psychological distress.
Referral for surgery

Consider referral for an opinion on spinal fusion for people who:
- have completed an optimal package of care, including a combined physical and psychological treatment programme
  and
- still have severe non-specific low back pain for which they would consider surgery.
Alexander Technique

- 579 patients with chronic or recurrent low back pain
- 144 were randomised to normal care
- 147 to massage
- 144 to six Alexander technique lessons
- 144 to 24 Alexander technique lessons
Alexander Technique

- 579 patients with chronic or recurrent low back pain
- 72 received normal care;
- 73 received six lessons in Alexander Technique;
- 73 received 24 lessons in Alexander Technique;
- 72 received exercise prescription;
- 72 received exercise prescription and massage;
- 71 received exercise prescription and 6 lessons of Alexander Technique;
- 71 received exercise prescription and 24 lessons in Alexander Technique.
Inclusion criteria

• Presentation in primary care with low back pain more than three months previously (to exclude first episodes)
• Currently scoring 4 or more on the Roland disability scale
• Current pain for three or more weeks (to exclude recurrence of short duration)
Results

• Exercise and lessons in the Alexander technique, but not massage, remained effective at one year

• Six lessons followed by exercise prescription were nearly as effective as 24 lessons
Summary

• Almost always simple
• Sinister causes usually apparent
• Explanation / reassurance key to management
• Good analgesia
• Keep active
Managing Shoulder Pain - An Update

Denis O’Brien 4th July 2009, RCGP Hot Topics Meeting
What you need to do in GP

- Make a working diagnosis
- Recognise the problems that need early referral
- Give appropriate advice
- Manage generically for first 12 weeks
- Use injections judiciously
History: Where?

- Neck/shoulder sweep ➔ Cervical
- Superior (well localised) ➔ AC joint
- Deltoid ➔ SA/GH
Shoulder Diagnoses

1. Impingement
2. Capsulitis (Frozen Shoulder)
3. Rotator Cuff Tear
4. Arthrosis (AC & GH)
5. Instability
Impingement

• aka Supraspinatus Tendinitis, Rotator Cuff syndrome, Subacromial Bursitis etc.

• Middle aged

• Pain in deltoid area

• Painful arc, impingement sign

• Pain on resisted cuff tests
Shoulder Examination

Hawkin’s Test
Frozen Shoulder

• Middle aged (40-60 years)

• Pain in deltoïd area (C5 dermatome)

• Active and passive movements restricted

• Minimal passive lateral rotation
Rotator Cuff Tear

- Extreme form of impingement
- Very painful and weak
- Suspect it if sudden or traumatic onset, >60 years
- Get a scan (USS)
- Refer to a shoulder surgeon ??
A-C joint

- Sports person (sprain) or the elderly (OA)
- Well localised pain
- “Point to the joint”
- Scarf test and tender over the joint
The Neck

- Pain in the neck
- Pain in the scapular region
- Neck movements aggravate the pain
- Often asymmetrical pain pattern
- Mechanical neck pain is managed like mechanical low back pain
Spurling’s Test
Impingement Management

- Patient Information Leaflet
- Analgesia
- Injection - 20-40mg Kenalog + 5ml 1% lidocaine. (Inject posteriorly under acromion)
- Physiotherapy
- Surgery (SAD)
Capsulitis Management

• No X-ray
• Patient Information leaflet
• Analgesia
• Injection (1 ml Kenalog, + up to 10 ml 0.25% Marcain or 1% Lidocaine)
• Physiotherapy
• Surgery
AC joint OA Management

• ? X-ray

• Analgesia

• Injection – difficult. Orange needle. Fingerbreadth in from lateral acromion. 10mg Kenalog + 0.75ml 1% lidocaine

• Surgery
Summary

• Remember the Neck
• 3 common conditions in Primary Care
  - Impingement
  - Capsulitis
  - A-C joint OA
• Pts < 35yo – refer if pain persists
Using NSAIDs -
An Update

Denis O'Brien 4th July 2009, RCGP Hot Topics Meeting
## GI event rates with NSAIDs

Table 2: NSAID-related deaths and admissions to hospital

<table>
<thead>
<tr>
<th>Event</th>
<th>UK</th>
<th>USA</th>
<th>Canada</th>
</tr>
</thead>
<tbody>
<tr>
<td>Annual NSAID prescriptions</td>
<td>25 million</td>
<td>70 million</td>
<td>10 million</td>
</tr>
<tr>
<td>NSAID-related admissions</td>
<td>12,000</td>
<td>100,000</td>
<td>3,900</td>
</tr>
<tr>
<td>NSAID-related deaths</td>
<td>2,600</td>
<td>16,500</td>
<td>365</td>
</tr>
</tbody>
</table>

Bandolier 2002
PPI prescription with NSAID /Coxib?

NICE: Health economic modelling found that it was always more cost-effective to prescribe a PPI with an NSAID than not to do so, because of their effect in reducing serious GI problems.

- There is some evidence that co-prescription of PPI with coxibs adds some additional GI protection.

Relative risk of hospitalization for GI events

For current users the adjusted relative risk was significantly increased with use of aspirin, non-naproxen NSAIDs, rofecoxib, and other coxibs, but not naproxen or celecoxib.
Non-selective NSAIDS may be associated with a small increased risk of thrombotic CVD events. The lowest effective dose of non-selective NSAID should be prescribed for the shortest possible time.

- **Diclofenac**: has a thrombotic risk profile similar to that of at least one coxib (etoricoxib) and possibly others.
- **Naproxen**: may have a lower risk of heart attacks or strokes than selective COX-2 inhibitors.
- **Ibuprofen**: high-dose ibuprofen (which is not available over the counter) may be associated with a small increased thrombotic risk. May reduce antiplatelet effect of Aspirin.
- **Less evidence is available for other NSAIDs**, but they may be associated with a small risk of thrombotic events, especially with long duration of treatment and high doses.
Clinical Hot Topics for General Practice

Roy Farquharson
Liverpool Women’s Hospital
roy.farquharson@lwh.nhs.uk
Early Pregnancy and Emergency Gynaecology

- Diagnosis and management of -
- PUL
- Ectopic pregnancy
- Miscarriage
Abnormal Uterine Bleeding

- Indications for hysteroscopy
- Management options of POP Cerazette, Implanon, Mirena, Endometrial Ablation techniques eg TCRE Resection
- Last resort hysterectomy
- Raised BMI epidemic
Fertility

- Assisted reproductive technology (ART)
- IVF Preimplantation Genetic Screening (PGS)
- PGDiagnosis
Oncology

- Screening programmes
- Ovarian cancer Scan and CA125
- HPV vaccine
- Revised vault smears
- Specialised Centre consolidation and care provision
End of Life Care (EoLC)
Aspects of Symptom Control

G Corcoran
July 2009
The End of Life Care Programme

Adrienne Betteley
End of Life Care Programme Lead
Merseyside and Cheshire Cancer Network
Key issues – End of Life

- Establishing advance care planning systematically
- Enabling patients who wish to die at home to do so
- Establishing a supportive palliative care register across settings
- Development of joint commissioning/funding
- Establishing integrated information systems
- Equity of access for bereaved relatives for support
We Aim to:

Achieve a 10% reduction in hospital deaths through enhanced community services by 2012
Advance Care Planning

Advance care planning

Statement of wishes and preferences

Advance decisions

Lasting power of attorney
Advance care planning definition

• a process of discussion between an individual and their care providers irrespective of discipline
• involvement of others
• with agreement discussions should be:
  – documented
  – regularly reviewed
  – communicated to key persons involved in their care
Preferred Priorities for Care
(formerly known as Preferred Place of Care) (PPC)

What is it?

• An advance care plan for people with a life limiting illness who wish to have their choices and preferences recorded in relation to their care and ultimate place of death

• A patient held record which should go with the patient if they are transferred to a different care setting
Identifying and Recording Preferences

• The explicit recording of patients wishes can form the basis of care planning in multi-disciplinary teams and other services, minimising inappropriate admissions and interventions.

• In relation to your health what has been happening to you?

• What are your preferences and priorities for your future care?

• Where would you like to be cared for in the future?
Support

- Not everyone finds it easy to have conversations about death and dying
- Staff may need additional support through communication skills training or through mentor or peer support – may be a Specialist Palliative Care Nurse
The Northwest End of Life Care Pathway

- Advancing disease: 1 year
- Increasing Morbidity: 6 months
- Last Days of Life: Death
- First Days of Death
- Bereavement: 1 year
Illness trajectories

- Organ failure
- Cancer
- Sudden death
- Dementia and decline

Function over time:

A. Cancer
   - Time: High → Low → Death

B. Organ System Failure
   - Time: High fluctuations → Low → Death

C. Dementia/Frailty
   - Time: Low fluctuations → Low → Death
### Therapeutic ratio - Phases of illness

**BMJ 1991;302:1322-24**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Curative</th>
<th>Palliative</th>
<th>Terminal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Radical</td>
<td>+++</td>
<td>++</td>
<td>-</td>
</tr>
<tr>
<td>Risk</td>
<td>+++</td>
<td>++</td>
<td>+/-</td>
</tr>
<tr>
<td>Nutritional support</td>
<td>+++</td>
<td>+</td>
<td>+/-</td>
</tr>
</tbody>
</table>
Quality End-of-Life Care - Patients’ Perspectives
Singer et al JAMA 1999 281;2:163-168

• N=126 patients
• Dialysis (48), HIV(40), Long term care (38)
• 5 Domains
  – Adequate pain and symptom control
  – Avoiding inappropriate prolongation of dying
  – Achieving a sense of control
  – Relieving burden
  – Strengthening relationships
Quality End-of-Life Care

• “Double Effect”

• Withdrawing and Withholding Treatment

• Resuscitation Orders

• Artificial Feeding and Hydration
Double Effect - Definition
Dr. K. Foley. www.soros.org

- The administering of opioids or sedative drugs to relieve pain and suffering in a dying patient with the incidental consequence of causing respiratory depression, extreme sedation or both, resulting in the patient’s death
Double Effect - Help or Hindrance?

• Necessary protection?

• May allow inappropriate therapy - ill informed professionals

• May have perverse effect of under treatment-anxious professionals
DOUBLE EFFECT

• Still requires the use of the appropriate drug in the appropriate doses
<table>
<thead>
<tr>
<th></th>
<th>Terminal or palliative sedation</th>
<th>Euthanasia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Intention</strong></td>
<td>Relieve suffering</td>
<td>Kill the patient</td>
</tr>
<tr>
<td><strong>Procedure</strong></td>
<td>Sedating drug for symptom control</td>
<td>Administer lethal drug</td>
</tr>
<tr>
<td><strong>Outcome</strong></td>
<td>Alleviation of distress</td>
<td>Immediate death</td>
</tr>
</tbody>
</table>
Do Not Go Gentle
Dylan Thomas

- Do not go gentle into that good night,
  Old age should burn and rave at close of day,
  Rage, rage against the dying of the light
  ..........
Terminal sedation is a procedure used to relieve patients of symptoms refractory to usual treatment by decreasing the level of consciousness in a patient close to death.
Sedation and Terminal Care.
Sales, J. Euro. J. Pall. Care 2001;8:97-100

- Proportional sedation - 38%
- Sudden sedation - 15%
- Median frequency - 25%
- Average Survival 2.4 days (1.3-3.9)
### Reason for terminal sedation - Sales, J.

<table>
<thead>
<tr>
<th>Reason</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnoea</td>
<td>38%</td>
</tr>
<tr>
<td>Pain</td>
<td>22%</td>
</tr>
<tr>
<td>Delirium/Agitation</td>
<td>39%</td>
</tr>
<tr>
<td>Anxiety/Distress</td>
<td>21%</td>
</tr>
<tr>
<td>General deterioration</td>
<td>20%</td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td>6%</td>
</tr>
<tr>
<td>Haemorrhage</td>
<td>9%</td>
</tr>
</tbody>
</table>
Sedation and Terminal Care.
Sales, J. Euro. J. Pall. Care 2001; 8: 97-100

• Common medications
  – Midazolam
  – Haloperidol
  – Levomepromazine
  – Hyoscine hydrobromide
  – Diamorphine
End of Life Care – Dose Audit – Last 24hrs

• **N=80** Median Range
  - Midazolam - 12.5mg/24hrs (2.5-50mg)
  - Hyoscine – 1.2mg/24hrs (0.8 - 2.4mg)
  - Levomepromazine
    » – 12.5mg/24hrs (6.25–50mg)
  - Diamorphine – 10mg/24hrs (2.5-80mg)
A comparison of the use of sedatives in a hospital support team and in a hospice.

• “The need for terminal sedation is an indicator of impending death and not a cause of premature death”
Management of Medical Emergencies in Palliative Care

Airway obstruction

Major Haemorrhage

Perforation / Infarction / Fracture
Management of Medical Emergencies in Palliative Care

- Anticipation
- Assessment
- Diagnosis
- Treatment Plan
- Review
- Debrief
CONSENSUS - AGREE THE GOALS OF CARE

• Assess for
  – Lack of comprehension
  – Disagreement with patient preferences
  – Emotional barriers; denial, guilt, grief
  – Team conflict or mixed messages
  – Narrow understanding of ‘hope’ and ‘caring’
  – ESTABLISH TRUST
Quality End-of-Life Care

- Frequent multi-professional assessment
- Information for Patients and Carers
- Communication between professionals
- Appropriate and timely medication
- Education
- “Double Effect” - unnecessary and rare
- EXCELLENT SYMPTOM CONTROL
Artificial hydration and nutrition at the end of life. S.Ede
Euro. J. Pall.Care 2000;7:210-212

• When considering the perceptions of families and significant others, it is appropriate to consider the significance of symbolism of food and fluids

• “To slake the thirst of a dying person is deemed across times and cultures to be not only right but good”. 
Rehydration in Palliative and Terminal Care: If not – Why not?

Dunphy et al. Palliative Med. 1995;9:221-228

“It would seem reasonable to suggest that rehydrating patients who are failing to take fluids in the terminal phase of their illness, in the absence of any identifiable and potentially remediable cause of dehydration, is unlikely to confer any benefit.”
• “…However….above all, we must be responsive to the wishes of the patient, and of their carers”.