



The Parkinson's  
Disease Academy

# Master Strokes

Issue 10 - Winter 2008

## Editorial

"Like I always say, there's no 'I' in "team". There is a 'me', though, if you jumble it up." *Gregory House MD*

In a previous editorial I commented on the enormous strides that have taken place in improving services for patients with Parkinson's disease. I did, however, enter a note of caution and recognised that we could not be complacent, particularly with regard to protecting and developing the role of specialist Allied Health Professionals. I wish to return to this theme.

The development of PD services can be seen as an evolutionary process. The first step is usually for a doctor to declare an interest and start running a dedicated clinic for PD. At this stage the priority is for them to improve and maintain their knowledge of the diagnosis and management. The next step is to establish a service proper and this might be defined as happening with the appointment of a PD Nurse specialist. Most geriatricians have access to physiotherapy, occupational therapy and speech and language therapy services and patients will benefit from appropriate referral and treatment.

The final goal of any service, however, should be the establishment of a true multidisciplinary *team*. This is more than the sum of its parts and the synergies created when different professionals work together take the service to a different level. For this to happen it is important that each team member is recognised as an expert in PD in their own right and is able to benefit from building up their experience in the condition as well as accessing high quality specialist education. Indeed Peter Fletcher, at the recent Advanced Masterclass meeting, promoted the definition of a specialist as "someone who spends a significant amount of their time in a PD practice and who furthers their education by spending time with other specialists".

A key aim of the BGS Movement Disorders Section is to promote such team-working and to support education. Through supporting the PD Academy in running the Masterclasses great progress has been made, particularly in supporting clinicians through the early stages as they develop their services and this has played a significant part in the improved geographical spread and better access for patients to specialist clinics.



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## The newsletter for graduates and mentors of The Parkinson's Disease Academy

Editors: Sue Thomas & Doug MacMahon. Other faculty members:

Prof David Burn, Professor in Movement Disorder Neurology and Honorary Consultant, Institute for Ageing and Health, Newcastle;

Prof Carl Clarke, Professor of Clinical Neurology and Honorary Consultant Neurologist, Birmingham;

Dr Peter Fletcher, Consultant Physician, Cheltenham; Dr John Hindle, Consultant Geriatrician, Llandudno;

Dr Jane Liddle, Consultant Geriatrician, Sheffield; Dr Graeme MacPhee, Consultant Physician, Glasgow;

Dr Helen Roberts, Senior Lecturer and Honorary Consultant in Geriatric Medicine, Southampton;

Dr Dorothy Robertson, Consultant Geriatrician, Bath; Dr Jagdish Sharma, Consultant Geriatrician, Grantham;

Dr David Stewart, Consultant Physician, Glasgow; Dr Richard Walker, Consultant Geriatrician, Northumbria

With a few exceptions, however, the Masterclasses have been aimed at senior doctors – either consultants or registrars coming towards the end of their training. In general Nurse Specialist colleagues are reasonably well-served for educational opportunities but it has been recognised for some time that our AHP colleagues are less well provided for. Of course the ‘Science to Practice’ meetings run by Dorothy Robertson over many years have been a beacon of light in the darkness in providing programmes orientated towards the multidisciplinary team. Budgetary and time constraints, with many AHPs having difficulty in accessing study leave funding, have, however, meant that maintaining such national meetings has proven very difficult.

Recognising the above, the BGS MD Section is hoping to establish a series of PD educational courses aimed specifically at AHPs and a small group comprising Peter Fletcher, Graeme Macphee, Brian Wood

and myself is taking this forward. These courses will be structured to provide the core background information that a therapist specialising or hoping to specialise in PD will require. Importantly, the aim is to deliver these courses locally using local experts. The rubric for this initiative is ‘Professional Partnerships in Parkinsons’ (PPiP). The first, pilot meeting is taking place on the 27<sup>th</sup> February 2009 in Glasgow and, if this proves successful, we hope to roll it out around the country. More anon.

I’ll leave you a rather more inspiring quote than the one I started with:  
‘Players win games, teams win championships.’ *Bill Taylor*

**Dr. David A. Stewart**  
**Associate Medical Director**  
**Emergency Care and Medical Services,**  
**Mansionhouse Unit**  
**Victoria Infirmary, Glasgow**  
**Founder Faculty Member PD Masterclass**

## Breaking News...



The All Party Parliamentary Group (APPG) for Parkinson’s disease is to hold a public inquiry into access to health and social care services for people with Parkinson’s and their carers.

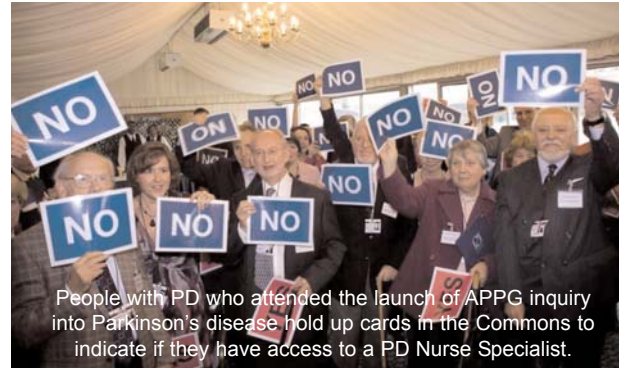
The APPG, which is chaired by Baroness Gale, is a cross-party group of Peers and MPs with an interest in Parkinson’s disease. The inquiry is being launched in the light of recent evidence pointing to inequity of access to key health and social care services for people with Parkinson’s and their carers in different parts of the country. In particular, the report **Life with Parkinson’s – room for improvement** highlighted specific problems around access to key health and social care services and support for carers. For example, it revealed that nearly four in ten people with the condition have not spoken to a Parkinson’s disease Nurse Specialist in the past 12 months, and one in three carers of people with Parkinson’s are still unaware of their right to a carer’s assessment. The inquiry takes place three years after the publication

of the first NICE Clinical Guideline for Parkinson’s disease (applicable in England, Wales and Northern Ireland), four years after the publication of the Department of Health’s National Service Framework (NSF) for Long-term Neurological Conditions and nearly two years after publication of **Fulfilled Lives Supportive Communities** by the Welsh Assembly Government. These sought to set benchmarks for key aspects of health and social care to which people with Parkinson’s/carers are entitled. A reception at the House of Commons was held on 19<sup>th</sup> November to launch this inquiry and Parkinson’s Academy Faculty member Dr Doug MacMahon spoke at the launch about the importance of submitting evidence to this inquiry. Dr MacMahon was a member of GDG that advised NICE on production of the Guidelines. He stated to the audience at the launch which included Health Minister Anne Keen MP that the NICE Guidelines have not been implemented fully – stating that the British Geriatrics Society Movement Disorders Section and Parkinson’s Disease Society have evidence of this, both at

anecdotal level, and from a series of audits performed across the country by attendees at the Parkinson's Disease Academy. He also said that good care should not cost any more than indifferent, unorchestrated care (and may cost less, through fewer admissions and better scheduled out-patients), and that good quality care makes a real difference to real people!

Academy graduates are encouraged to respond to this inquiry through the APPG for Parkinson's Disease Secretariat; either electronically (in MS Word or Rich Text format) by email to [ecogbill@parkinsons.org.uk](mailto:ecogbill@parkinsons.org.uk) or by hard copy to

Emily Cogbill,  
 APPG for Parkinson's Disease Secretariat,  
 c/o Parkinson's Disease Society, 215  
 Vauxhall Bridge Road, London. SW1V 1EJ



## Pain in Parkinson's Disease



In his original description of 'the shaking palsy' in 1817, James Parkinson noted that patients often described pain or 'rheumatism' as a symptom of the disorder(1). Since then, the recognition of pain as a significant symptom in Parkinson's disease has grown particularly in recent years. A review of pain in PD published in 1998 suggested that the frequency of pain in this population was 40% (2); however more recent work has suggested that pain is somewhere between 60-85% (3-5). The reason for the discrepancy is that the earlier studies largely restricted their recording of pain to PD related pain.

The causes of pain in PD are multifactorial and various classification systems have been used. One such system is:

- ◆ Pain unrelated to PD (eg osteoarthritis)
- ◆ PD related pain (eg cramps and dystonias)
- ◆ Pain indirectly related to PD (eg pressure sores/ or secondary to falls)
- ◆ PD treatment related pain (eg peak dose dystonias)

The frequency of these pains in a study by Lee et al (n=123) (3) was 50.9%, 42.5%, 4.2% and 0.3% respectively. The study highlighted the fact that the most common pains were non PD related (osteoarthritis in around 75%) and that these pains were significantly more severe than the PD related pains. Furthermore the

study showed that many patients had more than one pain (40% had 3 or more pains) and that 51% had pain which had a "moderate or dominating effect on their day". These findings demonstrate both the breadth of pain experienced and the impact which pain could have on patients. In keeping with other studies within elderly populations, these pains were also often undertreated with analgesics (6).



Hence, in general, the PD population often has pain which is poorly assessed and undertreated.

### Assessment of pain in PD

The key to treating pain optimally is assessing pain fully. It is therefore vital to assess every individual pain in terms of its position, radiation, onset, periodicity, character, precipitating factors, relieving factors, and severity. It is also important to look at the both previous and current analgesics that may have been used. Of particular importance in PD is

the relationship of the pain to the PD medication in that it may help determine whether a review of dopaminergic treatment is required rather than the use of analgesics.

Within palliative medicine, intrinsic to the assessment of pain is the concept of 'total pain'. In other words pain has not just a physical effect but also has an impact on psychological, social and spiritual domains. Consequently, pain assessment in PD should determine the effects of pain on lifestyle, mood, sleep, relationships as well as what meaning the patient attaches to the pain. It has been shown by Negre-Pages et al (5) that when compared with controls PD patients had higher depression and anxiety scores and reported poorer sleep. All these factors can affect the experience of pain and highlight the importance of an holistic approach to pain.

### Management of pain

The management of pain in PD directly follows from the comprehensive holistic assessment. Based on a proposed mechanism and underlying cause, a management plan can be proposed. For example, if the pain is thought to be PD related then it would reasonable first to consider either reviewing the dopaminergic treatment (especially if the pain is related to the times of the medication) or using non drug

therapies (such as physiotherapy for cramps). If this is not successful, then use of the WHO analgesic ladder may be helpful (7).

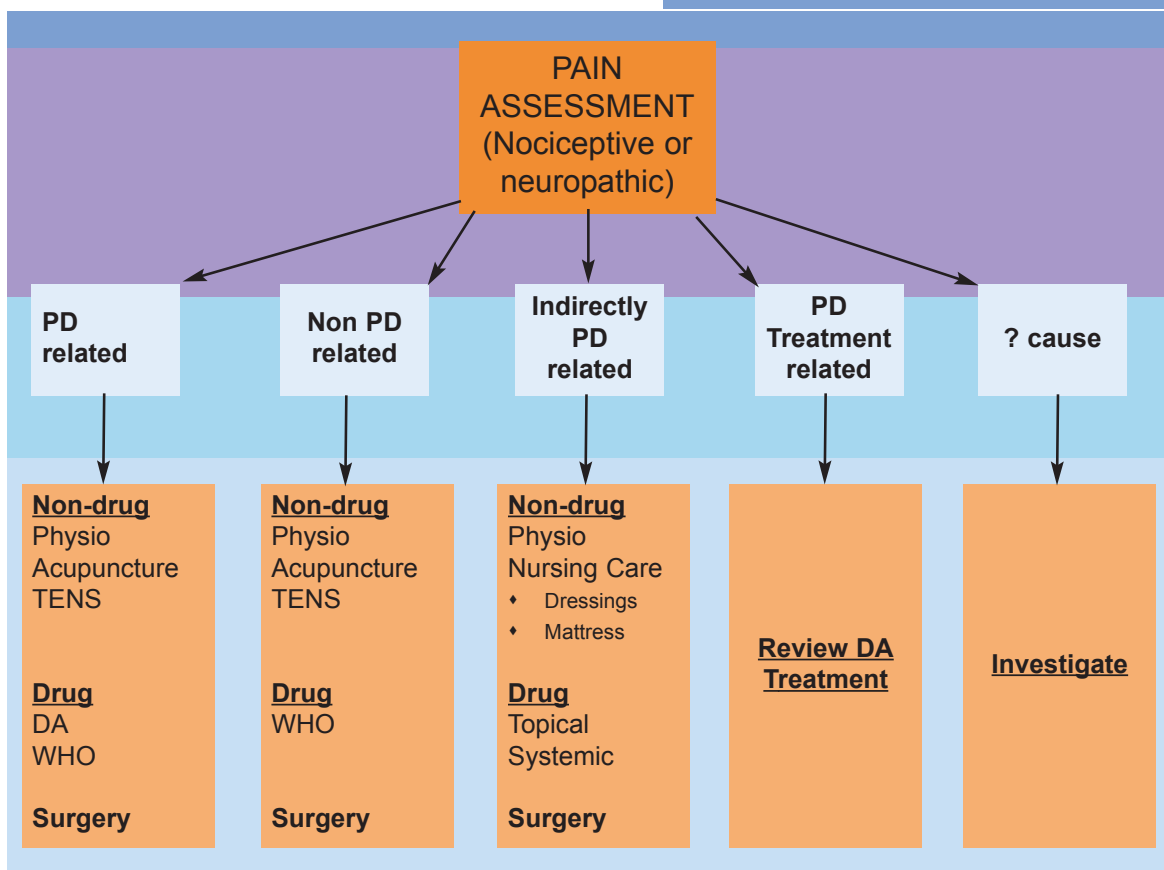
Simultaneously an assessment of the patient's mental state is vital in order to exclude depression, which again is underdiagnosed in PD. Finally, if required, advice can be sought from a chronic pain team or Specialist Palliative Care Team.

### Conclusion

Pain in PD is very common and can adversely affect the lives of patients and their families. It is also often under-diagnosed and as such undertreated. However, a simple stepwise approach of impeccable broad holistic assessment and a tailored management plan using the whole MDT may have a significant effect on patients' quality of life.

**Dr Mark Lee, MBChB, MRCP, MD**  
**Consultant in Palliative Medicine**  
**St Benedict's Hospice, Sunderland**

Figure 1 Algorithm for the treatment of pain in PD.



## References

1. Parkinson J. An Essay on the Shaking Palsy. Sherwood, Neely, and Jones. Paternoster Row, London 1817
2. Ford B. Pain in Parkinson's disease. *Clinical Neuroscience* 1998; 5: 63-72
3. Lee M A, Walker R W, Hildreth A J, Prentice W M. A Survey of Pain in Idiopathic Parkinson's disease (IPD). *Journal of Pain and Symptom Management* 2006; 32(5): 462-469
4. Defazio G, Berardelli A, Fabbrini G et al. Pain as a nonmotor symptom of Parkinson disease: Evidence from a case-control study. *Arch Neurol* 2008; 65:1191-1194
5. Negre-Pages L, Regragui W, Bouhassira D, Grandjean H, Rascol O, DoPaMiP Study Group. Chronic pain in Parkinson's disease: The cross-sectional French DoPaMiP survey. *Movement Disorders* 2008; 23: 1361-1369
6. Pitkala KH, Strandberg TE, Tilvis RS. Management of nonmalignant pain in home-dwelling older people: a population based survey. *Journal of the American Geriatric Society* 2002; 50(11): 1861-1865
7. World Health Organisation. Cancer pain relief: Office of Publications, World Health Organisation, 1986

# New Nurse Support Programme for APO-go



A new programme has been launched to support the initiation and delivery of APO-go therapy in hospital and community. This service will be welcome additional support for consultants and PDNS who may be limited in their ability to start APO-go therapy for patients. There have been difficulties in the past where, for example, there was no PDNS, district nurse or community matron in post able to support patients requiring apomorphine therapy.

This new service, sponsored by Britannia Pharmaceuticals, addresses these problems through a dedicated team of nurses being made available to work in partnership with NHS professionals to commence and deliver therapy to patients either in hospital or at home. This dedicated service, independently facilitated by Alchemy, is able to cover all aspects of APO-go therapy delivery including education, counselling, clinical support and development, patient support, therapy monitoring, audit, mentorship, adherence to and sharing of principles of best practice as well as emergency out of hours support.

The programme is conducted in an open and transparent manner. Confidentiality of patient information is assured in line with the Data Protection Act and all the service nurse advisors work within the Nursing and Midwifery Council Code of Professional Conduct. In addition they adhere to the requirements of the Data Protection Act, Caldicott Principles and ABPI Code of Practice. The clinical aspect of the programme is run by a nurse manager and senior nurse adviser. Both are experienced PDNSs and have in depth experience in the management of APO-go therapy.

The team of nurse advisers is able to liaise with staff in hospital, clinic, day hospital or primary care to help with therapy initiation, clinical support and onward transfer to primary care. This will ensure a comprehensive service for patients and carers where one may previously have not been available. The nurses can also undertake evaluation and monitoring in primary care, liaise with key personnel regarding all clinical and patient interaction and support the development of clinical services around APO-go therapy. Throughout, the lead consultant retains clinical responsibility for the patient, and the PDNS, if in post, will lead on management of the patient. The service will be conducted through an honorary trust contract, thereby providing additional clinical support for patients.

Britannia Pharmaceuticals feels this is an opportunity for it to consolidate its long-standing commitment to the NHS and patients and views this as a valuable resource in its NHS Industry partnerships

**Further information for the service can be obtained from Sonia Channa at Britannia Pharmaceuticals on 01635 568400.**





# Bowel Dysfunction and Parkinson's Disease

## Introduction

Parkinson's disease (PD) is a common, progressive movement disorder affecting 3% of the population over 65 years of age and is a significant cause of morbidity and an inroad into health care resources. The prevalence and incidence of PD has consistently been observed to increase with age with the mean age of onset at 55-60 years old. PD is associated with the degeneration of dopaminergic neurons in the substantia nigra, leading to a reduction in the levels of the neurotransmitter dopamine. It is thought that 60 to 80% of PD patients suffer from constipation (Ueki and Otsuka 2005). Furthermore it appears there is an association between the frequency of bowel movements and the future risk of developing PD (Abbott et al 2001). This is demonstrated by the presence of both Lewy neurites and Lewy bodies, (a hallmark of PD pathology), in the dorsal nucleus of the vagus nerve during the earliest stage of the disease (Braak et al 2006). It is thought that these lesions then extend upwards through the brain stem to eventually cause disturbance in the substantia nigra. Although this article is predominantly concerned with bowel dysfunction, it is important to consider the pathophysiology underlying all the gastrointestinal changes that can impact on PD bowel dysfunction.

## Pathophysiology

The pathophysiology of the gastrointestinal tract in PD is complex, involving the autonomic, central and enteric nervous system dysfunction. The well-documented symptom of excessive amounts of saliva in their mouth is not due to excess production but rather to impaired and infrequent swallowing. Thus parkinsonian drooling is actually a reflection of dysphagia and posture. Studies suggest that dysphagia becomes more prevalent as the disease progresses (Pfeiffer 2003). Dysphagia in PD is thought to be the consequence of the central nervous system degeneration; however, peripheral mechanisms may also be involved, characterised by the presence of Lewy body formation in the oesophageal

myenteric plexus. Dysphagia has a direct impact on nutritional and fluid intake, which in turn can lead to the development of idiopathic constipation – see Figure One.

Similarly, the nutritional and fluid intake of PD patients can be affected because of gastroparesis (delayed gastric emptying). Gastroparesis can produce a variety of symptoms that may include early satiety, abdominal discomfort, bloating and nausea. Little is known of the exact pathophysiology of

Figure One

Simple (Idiopathic / Primary) constipation No underlying causative illness.	Extrinsic factors that originate outside the body
Associated with life style	i.e. reduced dietary fibre, reduced fluid intake, reduced mobility, environmental changes (eg lack of privacy, poor toileting posture)
Commoner amongst the elderly	often the result of reduced mental and physical function. Dementia occurs in the latter stages of PD but may parallel motor progression from the disease outset.

parkinsonian gastroparesis but it is thought that the vagal nerve mechanisms are disrupted. Pfeiffer (2003) suggests that the presence of gastroparesis poses a potential hurdle to effective pharmacological treatment of PD, as levodopa absorption occurs primarily in the proximal small intestine. Any delay in gastric emptying could therefore result in a delayed response time to medication.

The onset of constipation in PD can be the consequence of enteric nervous system

abnormalities (peripheral) or central nervous system degeneration or a combination of both. The peripheral dysfunction is due to the loss of dopaminergic neurons in the myenteric plexus of the large intestine and the presence of Lewy neuritis and Lewy body pathology. Transit time studies indicate that slow bowel transit time is the major cause of decreased stool frequency in PD patients, reflecting the enteric nervous system pathology affecting peristaltic activity (Sakakibara et al 2003).

### Figure Two

#### Valsalva manoeuvre

**This involves inhaling and forcing the diaphragm and chest muscles against a closed glottis to increase both the intra thoracic and intra abdominal pressure which is transmitted to the rectum.**

Under ideal circumstances adopting the correct posture for defaecation raises the intra abdominal pressure through contraction of the diaphragm and abdominal muscles. Intra-abdominal pressure can be further increased by initiating the Valsalva manoeuvre (see [Figure Two](#)). However, patients with PD show a less pronounced increase in abdominal pressure on coughing and during the Valsalva manoeuvre. (Sakakibara et al 2003). The neuronal degeneration in the central nervous system relevant to straining at stool is yet to be clarified in PD (Sakakibara et al 2008). Raised intra abdominal pressure causes the pressure in the rectum to rise. The pressures exerted by the internal and external sphincters decrease. This mechanism is important, as rectal pressure must be higher than anal pressure for defaecation to be effective. Relaxation of the puborectalis muscle then occurs. Relaxation of this muscle allows for widening and lowering of the anorectal angle with perineal descent allowing faeces to pass more easily into the anal canal. Co-ordination between the abdominal contraction and pelvic floor relaxation is vital to the process of defaecation. Patients with PD experience a lack of synergy between the pelvic floor skeletal muscle (puborectalis muscle) and the anal sphincter muscle. This phenomenon is known as animus. The inappropriate contraction of the puborectalis and external

sphincter muscles results in excessive straining and indicates disruption to the central nervous system.

Yokoyama and Hosegawa (2007) reported a 7.1% incidence of paralytic ileus amongst 112 patients as a consequence of PD. There is limited information on paralytic ileus in the PD bowel dysfunction literature.

### Figure Three

#### Inhibitory reflex.

**Relaxation of the internal sphincter allows faeces to enter the upper anal canal and to be 'sampled' by sensitive nerve cells on the dentate line. These 'sampling cells' inform the cortical centre of the brain as to whether the rectal content is stool, flatus or liquid. Those with normal sensation can easily distinguish between flatus (which can be passed without fear of incontinence), diarrhoea (needing urgent access to a toilet) and a normal stool. It is thought that the inhibitory reflex is mediated by the extrinsic nervous system but the exact process is still not understood (de Lorijn et al 2005).**

Faecal incontinence occurs commonly amongst PD patients (Sakakibara et al 2008) and is thought to be associated with a reduced rectoanal inhibitory reflex (see [Figure Three](#)) and anal sphincter dysfunction.

#### Constipation

Constipation in PD can therefore be divided into four components:

1. Decreased frequency
2. Evacuation difficulty
3. Idiopathic
4. Iatrogenic – see [Figure Four](#), below

Patients with a slow bowel transit time will give a history of infrequent bowel actions, whereas those patients with evacuation difficulties will complain of difficulty with defaecation. Evacuation difficulties and slow bowel transit time may co-exist, with the consequence that a patient with PD may complain of both infrequent bowel action and prolonged straining (Emmanuel 2004). Constipation is a major health care issue for older patients, especially for those who are less mobile and

Figure Four

Iatrogenic constipation	Induced as a consequence of pharmacological agents
5 or more medications are a particular risk (Potter et al 2002)	As a result of taking medications to alleviate parkinsonian symptoms.
Anticholinergic medication	Dopamine deficiency leads to a functional excess of acetylcholine
Opioid medication	May be used during end stage PD

have neurological problems such as PD. Bowel care can put a great strain on carers and healthcare workers. It can be the precipitating factor for admission into a care home and has cost implications in terms of medications, containment equipment and nursing time. Indeed PD is a high risk factor for elderly care home residents developing constipation (Robson et al 2000)

### Symptoms

The fear of constipation and the need for regular bowel movements is a major concern for many people with PD. In the early to middle stages of PD the symptoms of constipation may be reasonably controlled. However, as mobility diminishes and the intake of fluids and fibre declines, constipation becomes more problematic. Indeed, it is the prevalence of faecal incontinence that contributes significantly to older people with PD being moved into care homes (Harari 2002). The embarrassment and shame associated with faecal incontinence as a consequence of profound constipation can be a genuine threat to personal dignity and quality of life (Sakakibara et al 2008, Kaye et al 2006). Faecal impaction is also a common cause of faecal incontinence in frail older people with PD. Faecal impaction describes the condition when constipation has become so severe that a large mass of faeces cannot be passed. Faeces then accumulate in the rectum and may back up in the sigmoid colon and even as far as the transverse and ascending colon. Occasionally impaction of the rectum may be due to soft poorly formed faeces as a consequence of too much osmotic laxative.

In an attempt to soften hard impacted faeces the bowel produces mucus and this can result in faecal impaction with overflow, known as spurious diarrhoea. This can be a common cause of faecal incontinence amongst the elderly. Healthcare workers often confuse the overflow caused by faecal impaction with genuine diarrhoea, leading to catastrophic consequences for the patient with the resulting omission of laxatives rather than an evaluation of laxative therapy. The appearance of faecal impaction with overflow can be distinguished from diarrhoea because of its high mucous content and small scybala pieces of faeces. It has a characteristic 'sweet' offensive smell due to bacterial fermentation.

Diarrhoea from intestinal hurry has had minimal bacterial activity and decomposition and therefore it has a sharp, acidic smell and the appearance often contains remnants of undigested food.

A patient with PD may experience a variety of constipation symptoms. Hard stool and prolonged straining appear to be the commonest symptoms whereas bloating, straining and hard stool are the most bothersome symptoms recorded by patients (Johanson and Kralstein 2007). Other symptoms range from headache and fatigue to loss of appetite and nausea and vomiting. Patients with difficulty in defecation may complain physically of a 'full bottom' and an inability to have their bowels opened. Older patients can describe constipation in terms of 'urges' and having 'long tries' (Koch and Hudson 2000).

The consequence of persistent or poorly managed constipation can lead to disabling complications. Such complications may include haemorrhoids, faecal impaction, faecal impaction with spurious overflow, urinary incontinence, urinary tract infection, rectal bleeding, general weakness and psychological disorders (Prodigy Guidance 2004).

Persistent straining at stool leads to increased intrathoracic pressure which can give rise to a reduction in coronary and peripheral circulation leading to other possible complications; such as the development of hernias, worsening of gastro-oesophageal reflux and transient ischaemic attacks (Prodigy Guidance 2004)

### Risk Assessment

Kaye et al (2006) demonstrated that constipation is a significant problem for people with PD, though there was reluctance for people with PD to talk about their bowel problems, and that a more proactive approach by healthcare professionals may enable this problem to be addressed. There is therefore a strong argument for the use of a risk assessment tool for constipation. Such a tool can help health care professionals to facilitate clinical decision-making by acting as an *aide mémoire*. Documentation of a patient's risk encourages and develops a much needed proactive approach to the management of constipation.

The importance of using a risk assessment tool for constipation is well documented (RCN 2008, Kyle 2006, Richmond 2003,). This is further endorsed by the Royal College of Physicians (Potter et al 2002) who state that the identification of risk factors for constipation in elderly patients is critical to achieving effective management of constipation. The National Institute of Clinical Excellence guidelines for faecal incontinence (NICE 2007) advocate a proactive approach to the bowel care of the infirmed older patient.

The Norgine risk assessment tool for constipation is a fairly new development (Kyle 2007a, 2007b). Minimum data currently exists on its validity or reliability and the absence of any other risk assessment tool for constipation has meant that the Norgine tool cannot be judged against a gold standard. The dynamic nature of the tool suggests that it will evolve and change over time with further research. Anecdotal evidence suggests the tool is raising the profile of constipation amongst health care workers.

### Management

Constipation is characterised by an unsatisfactory bowel action that may be both infrequent and difficult. The goal of treatment and/or management of constipation is prevention and relief. Establishing an ideal bowel action should prevent reoccurrence. An ideal bowel action is initiated by an urge to defaecate that is definite but resistible. Once defaecation commences there is no delay with the stool gliding out smoothly and comfortably followed by a pleasant feeling of relief.

Effective assessment provides nurses with the relevant information. On this, advice and interventions and management can be planned, and outcomes can be measured and

evaluation of care made. Conversely, the lack of an accurate assessment leads to either unnecessary treatment or an inappropriate reliance on containment, leading to the needless use of pads which is both expensive and potentially undignifying (Wagg et al 2006).

Advice on the trio of exercise, fluid and fibre - known as life style advice - is often recommended for promoting a healthy bowel and is still considered first line treatment for constipation (NCCC 2006). Yet evidence fails to demonstrate convincingly that these measures reduce the impact of constipation. Arguably still the best intervention for constipation is to prevent it from occurring in the first place.

### Increase fluids?

Inadequate fluid intake is thought to be a risk factor for constipation (Richards-Hall 1995, Maestri-Banks & Burns 1996, Müller-Lissner et al 2005) with the additional risk that older people often drink less in an attempt to avoid urinary incontinence. However, there is no evidence that increasing fluids can successfully treat constipation, unless there is evidence of dehydration (Müller-Lissner et al 2005). The dysphagia associated with patients with more advanced PD prevents an adequate intake of fluids. A small study by Ueki and Otsuka (2004) demonstrated that water intake amongst PD patients was significantly lower than in the control group and that PD patients tended not to feel thirsty. Fluid intake is controlled by thirst, which signals the need for more fluid. Dehydration leads to the water content of blood plasma being reduced. This creates an osmotic gradient between the capillaries in the bowel mucosa and the interstitial fluid between mucosal cells, causing water to move from the interstitial fluid to capillaries. This process leads, in turn, to the development of an osmotic gradient between the interstitial cell fluid, and the fluid in the lumen of the bowel. Water then moves out of the bowel lumen into the interstitial fluid and mucosal cells resulting in harder and drier faeces.

Whether active water drinking would ameliorate constipation is uncertain. However, a simple calculation of 30mls per kg body weight, (see fluid matrix in Figure Five, below) clearly shows the difference of a litre in the minimum fluid required for someone weighing 57kg and someone weighing 95kg suggesting that the common blanket statement of one and a half litres of fluid over twenty-four hours cannot be applied to all patients. If swallowing

difficulties exist, referral to a speech and language therapist or dietician can help.

Finally, there is some anecdotal evidence to suggest that drinking coffee instead of tea may help constipation. Coffee (not caffeine) appears to stimulate colonic activity (Brown et al 1990). Interestingly, a high coffee and caffeine intake is associated with a significantly lower incidence of PD (Ross et al 2000). However, in this study the effective ingredient appears to be the caffeine not the coffee. These results however, were not replicated amongst female patients with PD (Ascherio et al 2001). Whether caffeine provides a protective effect for preventing the development of PD requires further evaluation.

#### Gastro-colic action

Patients can be encouraged to take advantage of the morning gastro-colic reflex by having a

hot drink soon after waking and sitting on the toilet around twenty minutes later. The large bowel has a peristaltic action only 5-6 times a day. These movements are called gastro-colic reflex actions. They are triggered by distension of the stomach and it is thought that gastrin, a hormone secreted by the mucosa of the stomach, has an action in stimulating the gastro-colic reflex. The most powerful of these reflexes usually occurs in the morning, after breakfast. This strategy may not be so effective in those PD patients with gastroparesis.

#### Increase mobility?

There is some evidence to suggest that regular exercise amongst inactive individuals correlates with bowel function. Kinnunen (1991) found a statistically significant increased risk of constipation for people walking less than 0.5 km daily. The study also identified risks associated with patients walking with help, being chair bound or bed bound. PD patients not only experience decreased

mobility but postural instability and gait disturbance adds the risk of falling. A 12 week study of middle aged patients with constipation undergoing regular physical activity demonstrated a positive outcome with both the total colonic and rectosigmoid transit time decreasing significantly (De Schryver et al 2005). This evidence suggests that a regular exercise regime, through a broad rehabilitation programme, may be beneficial for those patients with PD. Bed rest or a period of immobility results in a weakening of the abdominal wall muscles. For patients with PD this leads to further difficulty in raising their intra-abdominal pressure sufficiently for effective defecation.

#### Increase fibre

In healthy individuals there is a relationship between dietary fibre intake, whole gut transit time and stool bulk and weight. This is supported by a lower incidence of constipation amongst

**Figure Five**

Patient's Weight		Minimum daily fluid intake			
Stones	kg	mls	Fluid oz	Pints	Glasses
6	38.1	1.1	40	2	4
7	44.5	1.3	47	2.3	4
8	50.8	1.5	54	2.7	4 to 5
9	57.2	1.7	60	3	6
10	63.5	1.9	67	3.4	6 to 7
11	69.9	2.0	74	3.7	6 to 7
12	76.2	2.2	81	4	8
13	82.6	2.4	87	4.4	8 to 9
14	88.9	2.6	94	4.7	9
15	95.3	2.8	101	5	10
16	101.6	3.0	107	5.4	10 to 11
17	108	3.3	114	5.7	11
18	114.3	3.4	121	6	12
19	120.7	3.6	127	6.4	12 to 13
20	127	3.8	134	6.7	13

Calculation based on 30mls fluid per 1kg of body weight from Ritz P (2001) Factors affecting energy and macronutrient requirements in elderly people. Public Health Nutrition 2(2B):561-568.

vegetarians (Nair & Mayberry 1994).

Dietary fibre is both soluble and insoluble i.e. fibres which are either soluble or insoluble in water. During the formation of faeces, fluids are drawn from the colon into the faecal matter by insoluble fibre particles. These fibres can swell up to twenty times their original size resulting in bulky faeces that can move easily through the colon. Soluble fibre is broken down by 'friendly bowel bacteria' into gas (flatus) and a gel like substance both of which aid peristalsis. Dietary fibre comes from plant food and is intimately related to starch as it is composed of long complex chains of polysaccharides. These chains cannot be broken down. Instead they maintain their bulk and in doing so stretch the bowel wall and stimulate peristalsis.

Towers et al (1994) found that constipated older patients consume fewer meals and tended to consume fewer calories, leading them to identify the amount or frequency of meals as risk factors for constipation. Older people may skip at least one meal per day (Cope 1996) and their consumption of fruit, vegetables and bread all decline significantly with age (Bennett et al 1995). The evidence therefore, suggests that a low calorific diet is a risk factor in constipation rather than a diet low in just fibre (Sheehy & Richard Hall 1998, Wald 2000) although this topic requires further research.

Increasing dietary fibre is still recommended in the management of constipation, under the guise of life style changes, yet this supposed remedy for constipation is unsupported by research (Joanna Briggs Institute for Evidence Based Nursing and Midwifery 1999). Patients with PD and dysphagia primarily require a diet of an appropriate consistency and to learn eating / feeding techniques that improve their swallowing and minimises aspiration.

It is important to note that there is now evidence which questions the efficacy of life style advice of increasing fibre, fluids and exercise for the treatment of constipation, whatever its cause (Annells and Koch 2003), suggesting further research is required.

### Pharmacological intervention

Laxatives are the most commonly prescribed pharmacological intervention for the management of constipation. The main groups of laxatives currently used regularly in practice

are identified below (Clinical Knowledge Summaries 2008).

- ◆ Bulk forming
- ◆ Stimulant
- ◆ Osmotic

There is, however, no reliable evidence that laxatives prevent constipation especially amongst patients with PD.

The recommendation to 'start with a clean bowel' (Weeks et al 2000) suggests that laxatives may be a necessary treatment option for severe constipation, but audit demonstrates that bowel problems can be linked to ineffective and or inappropriate prescribing of laxatives (Addison et al 2003). The audit outcomes indicated that patients received a combination of laxative regimes.

### Bulk forming laxatives

Bulk forming laxatives are the least harmful and can be used in conjunction with the lifestyle advice of increasing fibre in the diet. Bulk forming laxatives produced a significant improvement in stool consistency and stool frequency in PD (Astarloa et al 1992) but should not be considered in PD patients with severe slow transit constipation, because they add more load to an inefficient bowel and cause more symptoms. Evidence suggests that bulk forming laxatives may be better tolerated than other laxatives, but there is little comparative evidence that there are differences between bulk and other laxatives in terms of frequency or symptoms (NHS Centre for Reviews and Dissemination 2001).

### Stimulant laxatives

These laxatives stimulate an increase in colonic motility (peristalsis) and mucus secretion when the laxative or its breakdown products come into contact with the intestinal mucosa, intramural nervous plexus or intestinal musculature (Shafik 1993). Senna is a naturally occurring plant (anthranoid laxative) and is activated in the large bowel. It is an effective short-term remedy for acute constipation however; long-term use of senna leads to tolerance and reduced effectiveness (Potter and Wagg 2005).

Bisacodyl may be given in suppository form and may be effective within 15-60 minutes. Glycerine suppositories may also act as a rectal stimulant by virtue of the mildly irritant action of glycerol (BNF 2007). Glycerine

suppositories must be moistened prior to insertion and then placed alongside the rectal wall. All bowel care suppositories need body heat in order to dissolve for activation. If suppositories are placed in the middle of faecal matter they will remain intact and useless.

Rectal preparations such as phosphate enemas and micro enemas are useful for bowel clearance. However there is a lack of evidence to support the use of phosphate enemas in the management of constipation (Davies 2004). Indeed the author suggests that healthcare professionals should be aware of the risks involved with the use of phosphate enemas and should be conversant with alternative treatments that are evidence-based. The use of phosphate enemas should be avoided in those over 65 especially the elderly frail PD patients with bowel mobility problems (Mendoza et al 2007).

#### Osmotic

This group of laxatives include mixed electrolyte solutions containing polyethylene glycol and non-absorbable sugars such as lactulose and sorbitol. Their action is to retain fluid in the bowel by osmosis. The National Prescribing Centre (2004) states that lactulose may take up to 2-3 days to have an effect and is therefore not suitable for the rapid relief of constipation. Clinical Knowledge Summaries (2008) further emphasise that lactulose should not be regarded as first-choice therapy in the management of constipation. Neither is lactulose appropriate for the prevention and treatment of constipation where gut motility is impaired (Abrams et al 1995).

Macrogols (e.g Movicol) are still relatively new laxatives. When macrogol powder is mixed with water and drunk, the water is retained in the colon via an osmotic action, which triggers receptor stimulation leading to increased colonic peristalsis. There is evidence that the macrogols are safe and effective in the treatment of constipation (Ramkumar and Rao et al 2005). Movicol sachets are dissolved in 125mls of water. This volume does not require immediate swallowing and may be sipped over the course of an hour, making it acceptable for PD patients who may find drinking large volumes of fluid difficult.

Macrogols (Movicol) are particularly useful in those PD patients where faecal impaction is suspected (Culbert 1998) as Movicol is currently the only laxative recommended for faecal impaction in the UK. However some PD

patients may find the volume of fluid required to alleviate faecal impaction impossible to drink.

#### Non pharmacological management Biofeedback

Although biofeedback has not been explored in patients with PD, biofeedback has been beneficial in individuals with animus or defaecation difficulties. The advantages of biofeedback are numerous but importantly there are no side effects. Immediate information via a rectal sensor to a computer screen enables the nurse / therapist to set an exercise regime individual to each patient. This therapy stimulates motivation and compliance, because the patient receives visual feedback. There is currently an absence of randomised controlled trials into the efficacy of biofeedback for constipation (Brandt et al 2005). However, biofeedback does appear to improve physiological outcomes e.g. propulsion of faeces during defaecation and clinical outcomes e.g. stool consistency compared with baseline data (Brandt et al 2005). Certainly evaluation of biofeedback techniques in PD patients with defaecation dysfunction would seem to be indicated.

#### Anal irrigation

Bowel irrigation has become another option in the management of faecal incontinence, constipation and rectocele (Crawshaw et al 2004) and is indicated in the National Institute of Clinical Excellence guidelines for faecal incontinence (NICE 2007). Krogh et al (2008) stress the importance of considering new treatment modalities such as the new Peristeen anal irrigation system in the management of patients with PD. Peristeen is portable, requires no batteries and gives individuals a measure of independence with their bowel management. Further research is now required to assess and establish the efficacy of this new management option.

#### Conclusion

Both constipation and faecal incontinence are highly prevalent amongst patients with Parkinson's disease. PD disease pathology has a direct effect on bowel dysfunction with both idiopathic and iatrogenic factors further impacting on these common and distressing symptoms.

The importance of preventing constipation cannot be overstressed in terms of patient well-being and in reducing costs to the NHS. There is minimal evidence to support much of the current practice associated with the

treatment of constipation, whereas evidence exists to support the risk factors for developing constipation. This suggests that health care professionals should develop a more proactive and evidence-based approach to the prevention of constipation rather than continuing with the existing reactive response to this distressing symptom. This is dependant primarily on improving the education and the

skills base of health care professionals and those with whom they work.

**Author:**

Gaye Kyle RGN BA (Hons). Dip Ed. MA.  
Honorary Lecturer, Thames Valley University,  
recognised teacher at University of Ulster.

**Contact:** [gayekyle@tiscali.co.uk](mailto:gayekyle@tiscali.co.uk)

**References**

1. Abbott RD, Petrovitch H, White LR et al (2001) Frequency of bowel movements and the future risk of Parkinson's disease. *Neurology* 57:456-462.
2. Abrams WB., Beers MH. & Berkow MD (1995) Organ systems: gastrointestinal disorders. *The Merck Manual of Geriatrics*. New Jersey: Merck Research Laboratories.
3. Addison R., Davies C., Haslam D. et al (2003). A national audit of chronic constipation in the community. *Nursing Times*.99(11):34-35.
4. Annells M and Koch T (2003) Constipation and the preached trio: diet, fluid intake, exercise. *International Journal of Nursing Studies*.40(8):843-852.
5. Ascherio A Zhang SM Hernán MA et al (2001) Prospective study of caffeine consumption and risk of Parkinson's disease in men and women. *Ann Neurol* 50:56-63.
6. Astarloa R Mena MA Sanchez V et al (1992) Clinical and pharmacological effects of a diet rich in insoluble fiber on Parkinson's disease. *Clin Neuropharmacol* 15: 375-380.
7. Bennett N., Dodd T., Flatley J et al (1995) *Health Survey for England*. London: HMSO.
8. Brandt LJ, Prather CM, Eamonn MM, Quigley E, Schiller LR, Schoenfeld P, Talley NJ (2005) Systematic review on the 9. Management of Chronic Constipation in North America. *American Journal of Gastroenterology*. 100(S1):S5-S21.
10. Braak H, Rub U, Del Tredici K (2006) Cognitive decline correlates with neuropathological stage in Parkinson's disease. *Journal of Neurol Sci*. 248: 255-258.
11. British National Formulary (2007) *British National Formulary*. London: British Medical Association.
12. Brown SR., Cann PA. and Read NW (1999) Effect of coffee on distal colonic function. *Gut*. 31. 450-453.
12. Clinical Knowledge Summaries (2008) Constipation. <http://cks.library.nhs.uk/constipation#-312225> accessed 12.11.2008.
13. Cope K. (1996) Malnutrition in the elderly: A national crisis. (Publication No.017062-00147-2) Cited in: Sheehy
14. C & Richards Hall G (1998) rethinking the obvious: a model for preventing constipation. *Journal of Gerontological Nursing*. 24. (3).38-44.
15. Crawshaw AP, Pigott L, Potter MA, Bartolo DC (2004) A retrospective evaluation of rectal irrigation in the treatment of disorders of faecal incontinence. *Colorectal Diseases*. 6:185-190.
16. Culbert P et al (1998) Highly effective new oral therapy for faecal impaction. *British Journal of General Practice*, 48. p.1599-1600.
17. Davies C (2004) The use of phosphate enemas in the treatment of constipation. *Nursing Times*.100(18):32-34.
18. De Schryver AM, Keulemans YC, Peters HP, Akkermans LM, Smout AJ, De Vries WR, Van Berge-Henegouwen GP (2005) Effects of regular physical activity on defaecation pattern in middle-aged patients complaining of chronic constipation. *Scandinavian Journal of Gastroenterology* 40:422-429
19. De Lorijn F de Jonge WJ, Wedel T et al (2005) Interstitial cells of Cajal are involved in the afferent limb of the rectanal inhibitory reflex. *Gut* 54: 1107-1113.
20. Emmanuel A (2004) Constipation. Cited in Norton C & Chelvanayagam S *Bowel Continence Nursing*. Beaconsfield: Beaconsfield Publishers Ltd.
21. Harari D (2002) Epidemiology and risk factors for bowel problems in older people Cited in Potter JM, Norton C. & Cottenden A (eds) (2002) *Bowel care in older people: research and practice*. London: Royal College of Physicians.
22. Joanna Briggs Institute for Evidence Based Nursing and Midwifery. (1999) *Management of Constipation in Older Adults, Best Practice*. 3: 1-6.
23. Johanson JF and Kralstein J (2007) Chronic constipation: a survey of the patient perspective. *Alimentary Pharmacology and Therapeutics*. 25:599-608.
24. Kaye J, Gage H, Kimber A et al (2006) Excess burden of constipation in Parkinson's disease: A pilot study. *Movement Disorders* 21(8):1270-1273
25. Kinnunen O. (1991) Study of constipation in a geriatric hospital, day hospital, old people's home and at home. *Ageing*. 3(2):161-170.
26. Koch T and Hudson S (2000) Older people and laxative use: literature review and pilot study report. *Journal of Clinical Nursing* (9):516-525.
27. Krogh K, Ostergaard K, Sabroe S et al (2008) Clinical aspects of bowel symptoms in Parkinson's disease. *Acta Neurol Scand* 117:60-64.
28. Kyle G (2006) Assessment and treatment of older patients with constipation. *Nursing Standard*. 21.(8) 41-46.
29. Kyle G (2007a) Developing a constipation risk assessment tool. *Continence UK*. 1(1):38-45
30. Kyle, G. (2007b) Norgine risk assessment tool for constipation. *Nursing Times*. 20 Nov. 103(47):48-9.

31. Maestri-Banks A & Burns D(1996) Assessing constipation. *Nursing Times*. 92. (21),28-31.
32. Mendoza J., Legido J., Rubio S and Gisbert JP (2007) Systematic review: the adverse effects of sodium phosphate enema. *Alimentary Pharmacology and Therapeutics*. 26:9-20.
33. Müller-Lisner SA, Kamm MA, Scarpignato C, Wald A (2005) Myths and Misconceptions about 8
34. Nair P & Mayberry JF (1994) Vegetarianism, dietary fibre and gastro-intestinal disease. *Digestion Disorders (Di812)*:177-85.
35. National Health Service Centre for Reviews and Dissemination (2001) Effectiveness of laxatives in adults *Effective Health Care* 7. (1). ISSN: 0965-0288.
36. National Prescribing Centre (2004) The management of constipation. *MeReC Bulletin* 14. (6).
37. NCCC (2006) Parkinson's disease: national clinical guideline for diagnosis and management in primary and secondary care. London: Royal College of Physicians
38. National Institute of Clinical Excellence (NICE) (2007) Faecal incontinence. London: NICE.
39. Pfeiffer RF.(2003) Gastrointestinal dysfunction in Parkinson's disease. *Lancet Neurology*. 2(2):107-116
40. Potter JM, Norton C. & Cottenden A (eds) (2002) *Bowel care in older people: research and practice*. London: Royal College of Physicians.
41. Potter J and Wagg A (2005) Management of bowel problems in older people: an update. *Clinical Medicine* 5(3):289-295
42. Prodigy Guidance (2004) *Practical Support for Clinical Governance- Constipation*. <http://www.prodigy.nhs.uk/guidance.asp?gt=Constipation> accessed 1.11.2008.
43. Ramkumar D., and Rao S (2005) Efficiency and Safety of Traditional Medical Therapies for Chronic Constipation : Systematic Review. *American Journal of Gastroenterology*.100 (4) p.936-971
44. RCN (2008) *Bowel care, including digital rectal examination and manual removal of faeces* London: Royal College of Nursing
45. Richards-Hall G., Rakel B., Karstens M., Swanson E. & Davidson A (1995) Managing constipation using a research protocol. *MEDSURG Nursing*. 4. (1).11-21.
46. Richmond J (2003) Prevention of constipation through risk management. *Nursing Standard*. 17. (16),39-46.
47. Robson KM, Kiely DK, Lembo T. (2000) Development of constipation in nursing home residents. *Dis Colon Rectum*. 43:940-943.
48. Ross GW Abbott RD, Petrovitch H et al (2000) Association of coffee and caffeine intake with the risk of Parkinson's disease. *JAMA* 283(20):2674-2679.
49. Sakakibara R, Uchiyama T, Yamanishi T et al (2008) Bladder and Bowel Dysfunction in Parkinson's disease. *Journal of Neural Transmission*. 115:443-460.
50. Sakakibara R, Odaka T, Uchiyama T et al (2003) Colonic transit time and rectoanal videomanometry in Parkinson's disease. *Journal of Neurol Neurosurg Psychiatry* 74:268-272.
51. Shafik (1993) Constipation, Pathogenesis and Management. *Drugs*. 45. (4).p.528-540.
52. Sheehy C & Richards Hall G (1998) rethinking the obvious: a model for preventing constipation. *Journal of Gerontological Nursing*. 24. (3).38-44.
53. Towers AL., Burgio KL., Locher JL., Merkel IS., Safaeian M. & Wald A.(1994) Constipation in the elderly: Influence of dietary, psychological and physiological factors. *Journal of American Geriatrics*. 42. 701-706.
54. Ueki A and Otsuka M (2004) Life style risks of Parkinson's disease: association between decreased water intake and constipation. *Journal of Neurology*.251(Suppl 7):VII/18-VII/23

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# New Toolkit



## New toolkit to help improve management of patients with the commonly distressing condition, opioid-induced constipation

A new resource, **Opioid-Induced Constipation in Palliative Care Healthcare Professionals Toolkit**, comprising practical tools to aid consultations with patients with opioid-induced constipation (OIC), is now available. The toolkit, accredited by the Royal College of Nursing Accreditation Unit until 2011, was developed by Wyeth in collaboration with a multi-disciplinary expert working group. It aims to meet the educational need for knowledge of the causes, symptoms and treatment of OIC in palliative care patients and to encourage a proactive approach to management. The toolkit is a downloadable element of a wider online resource that can be found at [www.choices-in-oic.co.uk](http://www.choices-in-oic.co.uk).

Opioids are an essential and well-established treatment for pain encountered in palliative care; approximately 70 per cent of patients with advanced cancer and about 65 per cent of patients dying from non-malignant disease experience pain.<sup>1</sup> However, one of their most common side effects is constipation. OIC can be a source of distress for patients with

advanced illness and can be difficult to manage. The majority of patients who take opioids will develop OIC, with little or no tolerance to the condition.<sup>2</sup>

“Opioid-induced constipation can be a distressing and persistent condition impacting significantly on patients’ and carers’ quality of life”, said Sue Thomas, Nursing Policy Adviser, Royal College of Nursing and Chair of the expert group. “Patients with OIC often avoid discussing issues related to their bowels or may not think to ask their nurse about preventing or managing constipation before it’s too late. This toolkit offers a solution to the problem of OIC by providing practical tools to open lines of communication between nurses and patients.”

The toolkit contains a number of items for both patients and healthcare professionals. Healthcare professionals will benefit from a Guide to OIC in Palliative Care as well as a Quick Reference Guide, which includes an assessment checklist. The patient booklet contains a bowel symptom diary which encourages the identification and documentation of symptoms that require attention.



### References:

1. Colvin L, Forbes K, Fallon M. Difficult pain. *BMJ*. 2006 May 6;332 (7549):1081-3
2. SIGN Publication Number 44; Control of Pain in Patients with Cancer: A National Clinical Guideline. June 2002

ZMTX227 Date of Preparation September 2008

## ABBREVIATED PRESCRIBING INFORMATION

Consult Summary of Product Characteristics before prescribing.

**Uses:** The treatment of disabling motor fluctuations ("on-off phenomena) in patients with Parkinson's disease which persist despite individually titrated treatment with levodopa (with a peripheral decarboxylase inhibitor) and/or other dopamine agonists.

**Dosage and Administration:** Apomorphine hydrochloride is administered subcutaneously either as an intermittent bolus injection or by continuous subcutaneous infusion. Its rapid onset (5-10 mins) and duration of action (about 1 hour) may prevent an "off" episode which is refractory to other treatments. Hospital admission under appropriate specialist supervision is necessary during patient selection and when establishing a patient's therapeutic regime. Please refer to the Summary of Product Characteristics for full details before initiating therapy. Treatment with domperidone (typical dosage 20mg three times a day) before and during apomorphine HCl therapy is essential. The optimal dosage of apomorphine HCl has to be determined on an individual patient bases; individual bolus injections should not exceed 10mg and the total daily dose should not exceed 100mg. **Contraindications:** Children and adolescents (up to 18 years of age). Known sensitivity to apomorphine or any other ingredients of the product. Respiratory depression, dementia, psychotic disease or hepatic insufficiency. Intermittent apomorphine HCl treatment is not suitable for patients who have an "on" response to levodopa which is marred by severe dyskinesia or dystonia. **Pregnancy and lactation:** Caution should be exercised if prescribing apomorphine to pregnant women and women of childbearing age. Breast-feeding should be avoided during apomorphine HCl therapy. **Interactions:** Patients should be monitored for potential interactions during initial stages of apomorphine therapy. Particular caution should be given when apomorphine is used with other medications that have a narrow therapeutic window. It should be noted that there is potential for interaction with neuroleptic and antihypertensive agents.

**Precautions:** Use with caution in patients with renal, pulmonary or cardiovascular disease, or who are prone to nausea or vomiting. Extra caution is recommended during initiation of therapy in elderly and/or debilitated patients. Since apomorphine may produce hypotension, care should be exercised in patients with cardiac disease or who are taking vasoactive drugs, particularly when pre-existing postural hypotension is present. Neuropsychiatric disturbances are common in Parkinsonian patients. Apomorphine should be used with special caution in these patients. Apomorphine has been associated with somnolence and other dopamine agonists can be associated with sudden sleep onset episodes, particularly in patients with Parkinson's disease. Patients must be informed of this and advised to exercise caution whilst driving or operating machines during treatment with apomorphine. Haematology tests should be undertaken at regular intervals as with levodopa with given concomitantly with apomorphine. Pathological gambling, increased libido and hypersexuality have been reported in patients treated with dopamine agonists, including apomorphine. **Side Effects:** Local induration and nodules (usually asymptomatic) often develop at subcutaneous site of injection leading to areas of erythema, tenderness, induration and (rarely) ulceration. Pruritus may occur at the site of injection. Drug-induced dyskinesias during "on" periods can be severe, and in a few patients may result in cessation of therapy. Postural hypotension is seen infrequently and is usually intransient. Transient sedation following each dose of apomorphine may occur at the start of therapy, but this usually resolves after a few weeks of treatment. Nausea and vomiting may occur, particularly when APO-go treatment is initiated, usually as a result of the omission of domperidone. Neuropsychiatric disturbances (including transient mild confusion and visual hallucinations) have occurred during apomorphine therapy and neuropsychiatric disturbances may be exacerbated by apomorphine. Positive Coombs' tests and haemolytic anaemia have been reported in patients receiving apomorphine and levodopa. Local and generalised rashes have been reported. Eosinophilia has occurred in only a few patients during treatment with apomorphine HCl. Patients treated with dopamine agonists, including apomorphine, have been reported as exhibiting signs of pathological gambling, increased libido and hypersexuality (especially at high doses). Apomorphine is associated with somnolence. Breathing difficulties have been reported. *Prescribers should consult the Summary of Product Characteristics in relation to other side effects.* **Presentation and Basic NHS Cost:** Apo-go ampoules contain apomorphine hydrochloride 10mg/ml, as follows: 20mg in 2ml – basic NHS cost £37.96 per carton of 5 ampoules. 50mg in 5ml – basic NHS cost £73.11 per carton of 5 ampoules. APO-go pens (disposable multiple dosage injector system) contain apomorphine hydrochloride 10mg/ml, as follows: 30mg in 3ml – basic NHS cost £123.91 per carton of 5 pens. APO-go Pre-filled syringes contain apomorphine hydrochloride 5mg/ml, as follows: 50mg in 10ml – basic NHS cost £73.11 per carton of 5 syringes. **Marketing Authorisation Numbers:** APO-go Ampoules: PL04483/0064. APO-go Pens: PL04483/0065. APO-go Pre filled syringes: PL05928/0025. **Legal Category:** POM. **Date of last revision:** October 2008. For further information please contact: Britannia Pharmaceuticals, Park View House, 65 London Road, Newbury, Berkshire, RG14 1JN, UK.

**References:** 1. Pietz K, Hagell P, Odin P. 1998. Subcutaneous apomorphine in late stage Parkinson's disease: a long term follow up. *J Neurol Neurosurg Psychiatry*. 65:709-716. 2. Lees A, Turner K. 2002. Apomorphine for Parkinson's Disease. *Practical Neurology*. 2:280-287. 3. Deleu D, Hanssens Y, Northway M G. 2004. Subcutaneous Apomorphine: An Evidence-Based Review of its Use in Parkinson's Disease. *Drugs Aging*, 21(11):687-709. 4. Ellis C, Lemmens G et al 1997. Use of Apomorphine in Parkinsonian Patients with Neuropsychiatric Complications to Oral Treatment. *Parkinsonism & Related Disorders*, 3(2):103-107.

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