

The Palliative Care Handbook

Guidelines for clinical management
and symptom control, featuring extensive
support for advanced dementia



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Central nervous system

Dementia

Dementia is an insidious, global deterioration of cognition without impairment of consciousness. More than 100 causes are recognised, though most of these are exceedingly rare:

- a terminal disease (albeit slow) with a median survival of 7 to 10 years post-diagnosis
- prevalence of 10% in over 65-year-olds, 20% in over 80-year-olds, 40% in 90-year-olds, and for indigenous Australians the prevalence is 3 to 5 times that of non-indigenous Australians
- About 1% of all dementia is considered early-onset (age < 65). In indigenous Australians, early-onset disease is defined by an age of onset <50 years

Types

- Alzheimer's is the most common (70% of all dementias)
 - predominant early deficits are episodic memory and orientation to time.
- vascular (30% of all dementias)
 - accompanies a history of cardiovascular events (CVA/TIA)
 - islets of retained functioning
 - language is preserved
 - dysexecutive syndrome
 - gait disturbance
 - subcortical signs
- frontotemporal (FTD – 10% of all dementias; commonest cause of early-onset disease)
 - can occur in those with Motor Neurone Disease (10 to 15%)
 - disinhibition, apathy and loss of empathy
 - hyperorality, lability, poor insight and compulsive, perseverative behaviours
- Lewy body dementia (LBD)
 - Parkinsonism
 - visual hallucinations and cognitive fluctuations
 - cognitive fluctuations typically marked
 - REM-Sleep behaviour disorder
 - vulnerability to delirium
 - extreme sensitivity to antipsychotics – quetiapine is the agent of choice.
- treatable causes
 - depressive pseudodementia
 - subdural and hypothyroidism
 - B₁₂/folate deficiency
 - syphilis

- others
 - Parkinson's disease (essentially very similar to Lewy body dementia), Huntington's, alcoholic, post traumatic brain injury, paraneoplastic, post encephalitic

Note that mixed types of dementia become increasingly common with age, and that end-stage dementia (regardless of cause) tends to assume a common phenotype. With the exception of Lewy body dementia, determining the exact type of dementia in a palliative/end-stage setting is much less important than recognition and appropriate treatment of a behavioural syndrome.

Assessment

- Take an extensive history (in end-stage dementia this will invariably need to be from a family member or close caregiver).
- Formally assess mental state, including the use of cognitive screening tools e.g. Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA) - where the patient retains verbal skills. (see 'Useful resources' p. 162)
- In many cases a formal cognitive evaluation will not be possible in advanced dementia, but the broader mental status examination remains invaluable, particularly in relation to:
 - General appearance and behaviour. Is the patient agitated, distressed, vocalising? Are there any signs of drug side effects (Parkinsonian facies, resting tremor, dyskinetic movements - oro-lingual dyskinesias are particularly common), dystonic reactions, motor tics or perseveration?
 - Affect - does the patient's expression reflect sadness, anxiety, anger? Are they guarded and suspicious? Lability may reflect frontal involvement, and should be differentiated from depression.
 - Perception. Does the person appear to be responding to external stimuli?

Behavioural and Psychological Symptoms of Dementia (BPSD)

- delirium
 - a careful history is vital. The biggest single risk factor for delirium is the presence of pre-existing cognitive impairment, so those with dementia are at vastly increased risk. Reduced cognitive reserve lowers delirium threshold.
 - A history of acute deterioration (cognitive, functional, behavioural) in the setting of previously stable impairments should always suggest delirium, and should be treated as such.
- depression (treat early initially with a SSRI or mirtazapine)
 - this is a difficult diagnosis to make in the presence of advanced dementia, where the patient's ability to report symptoms accurately is compromised
 - clinicians are advised to fall back on the presence of 'hard-core' biological symptoms of depression in this setting (recent change in sleep or appetite patterns, complete anhedonia, self-harm behaviour)
 - if there is any doubt, erring on the side of a trial of treatment is often advisable. Depression should be on the list of differential diagnoses for most behavioural disturbances in dementia, and modern antidepressants are much less toxic than the antipsychotic drugs that might otherwise be prescribed

- Mirtazapine is a useful drug in this patient group. It has beneficial effects on sleep, appetite and anxiety that occur early in the course of treatment and which are independent of its antidepressant effects
- The minimum antidepressant dose of mirtazapine is 30mg. If treating depression there is generally no advantage to commencing at a lower dose (often justified on the basis of minimising sedation...mirtazapine is an inverse agonist at the histamine receptor, however, and thus is more sedating at lower doses)
- agitation/aggression (consider low dose short term antipsychotics, benzodiazepines)
 - identify precipitants (can be difficult)
 - avoid confrontation
 - if the issue is agitation alone, antipsychotics hold no advantage over benzodiazepines, and are considerably more toxic
 - an intermediate half-life benzodiazepine with no active metabolites (e.g. oxazepam 7.5-15mg, temazepam 10mg) is the safest choice
 - there is evidence for the use of low-dose risperidone in the management of aggression, but the effect size is small (~0.2)
- anxiety
 - peaks in early/mid stages
- delusions (treat with antipsychotic)
 - particularly paranoid
 - beware ‘delusions of theft’ and ‘misidentification delusions.’ These may well be beliefs that have arisen as the artefact of cognitive impairment and/or to reflect neurological impairment (e.g. prosopagnosia) and are not likely to be antipsychotic responsive
- hallucinations
 - visual (up to 50% in LBD, although 20% of Alzheimer’s patients will hallucinate at some stage during the course of the disease)
- sleep/wake cycle reversal/sundowning
- loss of insight/judgement
- wandering (60% of patients)
 - pacing and lapping (exclude akathisia)
 - (dangerous) eloping i.e. getting lost, accidents
- rejection of care
 - of food, hydration (consider artificial hydration) and hygiene

Complications

- eating and swallowing difficulties, cachexia
- infections – pneumonia, urinary tract
 - in pneumonias, the mortality is sevenfold that of a non-dementia patient
 - treat if symptomatic, antibiotics have limited efficacy
- falls – due to impulsivity, frailty, benzodiazepines and other sedatives
- pain – common in very elderly (50%)
 - may present behaviourally (non-verbally, crying, irritability)

- roughly 70% of patients with significant BPSD are likely to have under-treated or unrecognised pain as a contributing factor
- adverse reactions to drugs
 - antipsychotics – sensitivity (Lewy body disease), parkinsonism, akathisia, acute dystonic reactions, sedation, peripheral oedema, chest infections, accelerated cognitive decline, stroke risk (3 fold that of non-dementia patients, 1.5 fold mortality), hypotension
 - benzodiazepines – sedation, falls

Treatment

As curative treatment does not exist, ensure that end-of-life discussions/advance directives/appointment of enduring power of attorney all happen early before loss of capacity. The environment of care is important – it should be simple, safe, involve attentive and patient staff, include support and education for family and carers, person-centred, proactive, include distractions, activities, routine, memory cues and benign paternalism.

- Mild – cholinesterase inhibitors may have temporary cognitive benefit
- Moderate – focus on quality of life and maintenance of function
- Severe – maximise comfort, avoid aggressive, burdensome or futile treatments, avoid enteral tube nutrition, consider a secure facility, allow a natural death (AND)

Depression.....

In end-of-life care it is important to distinguish between clinical depression and profound sadness.

- depression is a pervasive sense of misery
- sadness is a normal response to loss which waxes and wanes but enjoyment and future planning are retained
- most terminally ill patients do not become clinically depressed
- prevalence is about 15% (compared with 5 to 10% in the general population), most commonly in the early cancer stages
- reaching a diagnosis of depression in terminal patients is difficult as the usual physical symptoms of depression in the otherwise well such as anorexia, weight loss, sleep disturbance are often already present in patients with malignant disease whether they are depressed or not
- the psychological symptoms are more discriminative
- asking 'Are you depressed?' provides a bed-side assessment of mood
- suicide is rare, however, fleeting suicidal thoughts and fluctuating 'will to live' in cancer patients are common and not necessarily pathological
- requests for euthanasia and/or physician assisted suicide are more common although, as for suicide, this is not limited to depressed patients
- clinical depression is under-recognised and under-treated yet it is generally very responsive to treatment
- the cause of depression is unknown but imbalances in neurotransmitters, especially serotonin, in the brain may play a part

Psychological symptoms of major depression may include

- hopelessness
- anhedonia (loss of pleasure)
- morbid guilt and shame
- worthlessness and low self esteem
- request for physician assisted euthanasia
- persisting suicidal ideation
- lowered pain threshold
- decreased attention and concentration
- cognitive slowing
- impaired memory
- indecisiveness
- early morning wakening
- ruminative negative thoughts
- nihilistic and depressive delusions
- feeling of unreality

Depression in older people and people with dementia

It is worth noting that the 'textbook' symptoms of major depression as they appear in references such as DSM-V have not been validated in older persons. Many older people with depression will not use the word 'depression' to describe their feeling state, but will instead use terms such as 'anxiety,' or 'I'm just worried, doctor.' Taking these terms at face value may lead to the inappropriate prescription of anxiolytics. Older persons also tend to express their depression more frequently in terms of somatic symptoms than younger persons do, which can clearly present diagnostic difficulties in a setting where palliative care is being provided.

Similarly, the diagnosis of depression in the setting of dementia is fraught. In cognitively intact populations, the diagnosis is made on the basis of symptom self-report. In advanced dementia, however, most patients will be unable to reliably verbalise their symptoms. The psychological distress that depression causes may instead be expressed in terms of externalising behaviours, which may include agitation, aggression, pacing and calling out, themselves common behavioural and psychological symptoms of dementia (BPSD). Two of the more reliable 'biological' symptoms of depression in the setting of dementia are recent worsening in sleep or appetite.

SSRI antidepressants are considered first-line pharmacological management for symptoms of BPSD. One of the likely reasons for their apparent success in controlling BPSD is that many cases labelled as BPSD are, in fact, cases of depression manifesting as disturbed behaviour. In a similar vein, while drug treatment trials of depression in the setting of dementia have been disappointing/contradictory, part of the problem inherent in such trials is a lack of certainty around diagnosis. In other words, these trials may well have included persons with undifferentiated BPSD, rather than depression.

A number of screening tools for depression in dementia exist. Perhaps the most commonly used tool is the Cornell Scale for Depression in Dementia. Clinicians should be wary of placing too much faith in the Cornell, however, as it has not been validated in patients with an MMSE of 10 or less, nor in patients with significant BPSD.

The role of antidepressants in treating depression in advanced dementia is controversial, and is likely to remain so, given the methodological problems in 'true case' ascertainment. When in doubt, however, clinicians are advised to err on the side of a trial of treatment.

Risk factors

- inadequate symptom control - unrelieved pain, nausea
- poor quality of life
- lack of social support
- past and/or family history of depression
- older age
- substance abuse
- misinformed prognosis
- polypharmacy
- specific drugs
 - steroids, cytotoxics, antibiotics, anti-hypertensives, neuroleptics, sedatives, beta-blockers, opioids
- immobility
- advanced malignant disease

Differential diagnosis

- adjustment/grief reaction (sadness)
- 'vital (physiological) exhaustion'
- demoralisation (a state of existential despair, meaninglessness and hopelessness but not of anhedonia and joylessness)
- delirium/sedation
- detachment (the terminal shedding of attachments)
- 'giving up' (affect neutral, rational, decisive)

Management

- mild to moderate depression
 - support, empathy, clarification of stressors or precipitators, explanation, cognitive therapy, symptomatic relief
- severe depression
 - supportive psychotherapy plus drug therapy
 - drug therapy - antidepressants are effective in 50 to 70% of cases
 - > a therapeutic trial is usually appropriate
 - > if in doubt, refer to a specialist psychiatrist
 - > SSRI e.g. escitalopram, sertraline, fluoxetine

- > although full response to antidepressant therapy may not be evident for 4-6 weeks, the lack of any response in the first 10-14 days should prompt consideration of a dosage increase or change of agent.
- > alternative agents include mirtazapine, duloxetine and venlafaxine
- > mirtazapine – can be useful due to its beneficial early effects on appetite, sleep and anxiety, which can be expected to occur well in advance of its antidepressant effects.
- > tricyclic antidepressants should be avoided, as the doses required for adequate response are likely to produce significant anticholinergic side effects, and may thus precipitate delirium, particularly in those with dementia.
- psychostimulants e.g. methylphenidate
 - > not as effective as SSRIs - may help retarded/withdrawn, frail patients for a few weeks only
 - > a response may be achieved from small doses (5 to 30 mg each morning) within days either alone or in combination with an SSRI - watch for additive serotonergic effects. Modafinil may be a useful alternative to methylphenidate

Delirium

Toxic confusional states, like delirium, are common in people who are dying.

- if irreversible, may be an indication of impending death
- can be most distressing for patients, family and staff

Diagnosis

- abrupt onset, typically, but may be subacute in those with dementia.
- impairment of consciousness - the primary symptom which results in:
 - disorientation (to time)
 - fear and dysphoria
 - memory impairment (short term memory)
 - reduced attention span to external stimuli
 - hyperactive (frenzy) or hypoactive (retardation, torpor) but usually mixed hyperactive and hypoactive motor activity
 - reversal of sleep-wake cycle
 - perceptual disturbance (illusions, hallucinations)
 - disorganised thinking (paranoia, rambling)
 - dysgraphia (difficulties with writing)
- fluctuating symptoms ('sundowner effect')

Causes

There are often multiple organic causes but in up to 50% of cases, specific causes are not found, despite investigations. Diagnosis is dependent on the presence of an appropriate history, rather than the results of a 'delirium screen'. Causes may include:

- infection
- organ failure (liver, kidney) and underlying medical conditions

- drugs
 - sedatives
 - anticholinergics
 - opioids
 - benzodiazepine or alcohol withdrawal
 - steroids
- metabolic disturbances
 - dehydration
 - hypercalcaemia
 - hyponatraemia
 - hyper/hypoglycaemia
- hypoxia
- anaemia (severe)
- vitamin deficiency
- cerebral metastases
- cerebral haemorrhage
- epilepsy - post-ictal

Predisposing/precipitating/aggravating factors

- dementia and CNS immaturity
- any other cause of pre-existing cognitive impairment (e.g. intellectual disability, ABI)
- pain
- fatigue
- urinary retention
- constipation
- unfamiliar excessive stimuli
- change of environment
- sensory deprivation
- sleep deprivation

Management

- treat the underlying organic causes if identifiable and treatable
- treat fever, hypoxia, anaemia, dehydration, constipation, fear and anxiety and pain if possible
- ensure there is a safe and secure environment - have adequate staffing, remove potentially dangerous objects, have the mattress on the floor
- prevent sensory over-stimulation - have a single room, minimise noise and staff changes and maintain a warm and comfortable environment
- psychological interventions
 - reassurance
 - orienting aids (clock, personal belongings, presence of a supportive family member)

- cognitive strategies (clarification, reality testing, validation and repetition during lucid periods)
- emotional support (touch, empathy)
- drugs - use if symptoms are severe (in combination with above management)
 - antipsychotics (goal is to calm or pacify rather than sedate)
 - > haloperidol is traditionally the drug of choice BUT not in AIDS delirium (HIV makes the CNS more sensitive to dopamine antagonists), hepatic encephalopathy or alcohol withdrawal where benzodiazepines only should be used (see haloperidol in drug section)
 - ~ **Haloperidol regimen in acute delirium:**
 - ~ Oral (tablets, liquid) if compliant, subcut if not
 - ~ initial dosage - 0.5 to 1.5 mg orally
 - ~ repeat and titrate every 30 to 40 minutes until controlled. In general, daily doses in excess of 3mg should be avoided due to high risks of extrapyramidal side effects (EPSE) in older patients. Only in physically robust, younger patients in whom significant aggression is present should doses in the order of 5-10mg daily be used.
 - ~ maintenance - 50% of daily dose required to achieve control usually 1 to 3 mg/day (oral)
 - ~ only add anticholinergic agent e.g. benztropine 1-2 mg if acute dystonia occurs. Routine use of anticholinergic agents will worsen delirium
 - ~ extrapyramidal side effects are less pronounced with the parenteral route. Thus, if IV access is present, parenteral administration is preferable
 - > risperidone (tablets, liquid, wafers) – dosage regimen as per haloperidol
 - > olanzapine (tablets, wafers) – doses of up to 2.5mg TDS can be considered. Doses in excess of this tend to have significant anticholinergic activity, and may make things worse
 - > Quetiapine – doses of 12.5-25mg are useful for acute sedation for short periods. Tolerance rapidly develops over several days to the sedative effects of this agent, leading to a tendency towards ‘dose-creep’ over time. If rapid control of distressing psychotic symptoms is required, however, this agent is not recommended, as it must be titrated up over several days in order to avoid both oversedation and postural hypotension.
 - sedatives (should not be used alone in most cases of delirium as they may aggravate symptoms, particularly if inadequate doses are used, so use with an antipsychotic)
 - > benzodiazepines e.g. midazolam, clonazepam
 - > barbiturates e.g. phenobarbitone
 - > melatonin may be useful
 - anaesthetics e.g. propofol (rarely indicated)
 - drug-induced delirium
 - > opioid-induced - decrease dose or change opioid
 - > anticholinergic-induced - e.g. physostigmine may reverse this.

Even if the aetiology is irreversible, the symptoms of delirium may be palliated. Only 10 to 20% of patients with terminal delirium should require ongoing sedation to achieve control.

Disorders of sleep and wakefulness

Sleep disturbance in people who are dying is a frequent occurrence and it requires careful assessment and management.

- sleep patterns change with age and with illness e.g. cancer
 - reduction of depth and continuity of sleep and an increasing propensity for day-time naps occurs
 - many cancer patients have difficulty falling and staying asleep
 - cytokines are implicated in these changes

Insomnia

This is common and distressing. It undermines coping strategies through tiredness.

Causes

- poor symptom control of
 - anxiety, depression, pain, urinary frequency, faecal incontinence, nausea, vomiting, delirium, cough, delirium
- environmental changes
 - admission to hospital or hospice
 - disturbance by staff or family
- fear of going to sleep and never waking up
- drugs
 - stimulants e.g. methylphenidate
 - steroids (particularly if given after noon)
 - bronchodilators
 - alcohol, caffeine
- withdrawal of benzodiazepines, alcohol or tobacco

Management

- symptom control of above
- establish good sleep hygiene
 - regular bedtimes
 - minimise daytime napping
 - reduce evening stimulants e.g. caffeine, alcohol
 - comfortable bedding
 - comfortable temperature
- relaxation techniques
- drugs
 - hypnotics
 - > short acting benzodiazepines e.g. temazepam
 - > longer acting benzodiazepines e.g. oxazepam

- > melatonin 2 to 4 mg at night
- sedative antidepressants e.g. mirtazapine 7.5-15mg nocte
- sedating antipsychotics e.g. quetiapine 25 to 50 mg at night may be considered if insomnia is resistant to above. Note that tolerance to sedation from quetiapine can occur rapidly (within several days).

Drowsiness/hypersomnia

These are common symptoms, particularly as the end-of-life approaches.

Causes

- organ failure e.g. renal, hepatic, cardiac, respiratory
- delirium (hypoactive)
- metabolic disturbances e.g. hyperglycaemia, hypercalcaemia
- fatigue or 'vital exhaustion'
- infection
- raised intracranial pressure
- drugs
 - adverse effects e.g. opioids, anticholinergics, benzodiazepines, cyclizine, levomepromazine (methotrimeprazine)

Management

- accurate assessment
- treat/remove causes where possible
- it may be unresolvable and be a natural part of the dying process

Sleep phase (circadian) disorder

(Delayed Sleep Phase Syndrome or Sleep-Wake Reversal)

- a dysregulation of the sleep-wake cycle
 - profound initial insomnia and
 - the inability to arise at desirable hours
- particularly associated with cerebral tumours
- presents a major burden for carers

Management

- shifting the circadian rhythm with behavioural strategies and bright light therapy is impractical in the terminally ill
- relief care for the family and a night nurse may be necessary as this tends to be an intractable symptom
- drugs are of limited benefit
 - sedatives e.g. benzodiazepines
 - psychostimulants e.g. methylphenidate can promote daytime alertness
 - sedating antipsychotics e.g. quetiapine 25 to 200 mg at night
 - pericyazine 20 to 30 mg at night
 - melatonin 2 to 6 mg at night

Terminal agitation

Perhaps best conceptualised as a prolonged delirium, this may indicate physical, psychological and/or spiritual discomfort. It is usually a 'pre-death' event.

A significant proportion of new-onset BPSD-type behaviours in fact represent terminal agitation. Early recognition of the syndrome enables appropriate palliative measures to be instituted early.

In the residential care setting, predictors of terminal agitation can include chest infections, unexplained fevers, poor oral intake, significant recent weight loss, the presence of bed sores, and increases in verbal and motor behaviours.

Terminal agitation is poorly recognised, and is often interpreted by care staff as a worsening of behavioural and psychological symptoms of dementia (BPSD). Early data from the Australian national Severe Behaviour Response Teams (SBRT) found that up to 10% of referrals to this service were ultimately revealed to have been on a terminal trajectory.

Causes

- physical discomfort
 - unrelieved pain
 - distended bladder or rectum
 - physical restraint
 - insomnia
 - uncomfortable bed or environment
- delirium (see delirium section)
- psychological discomfort
 - anger
 - fear
 - guilt
 - unfinished business
- spiritual discomfort/distress
 - helplessness
 - hopelessness
- drugs
 - akathisia induced by dopamine antagonists e.g. metoclopramide, haloperidol (and occasionally via sedating antihistamines such as promethazine)

Management

- assess and treat/remove possible causes
- explain what's happening to the family, patient (if appropriate) or main carers
- have the family present to reassure and support
- discuss psychological discomfort e.g. anger, fear, guilt
- drugs
 - see delirium section and anxiety and fear section

- e.g. midazolam in inadequate doses can aggravate (by disinhibition) rather than relieve restlessness in some patients
- if levomepromazine (methotrimeprazine) with a benzodiazepine are ineffective consider phenobarbitone or dexmedetomidine

Palliative sedation

This is considered when all other symptom-relieving measures have failed and the patient is clearly distressed.

Reasons for palliative sedation

- terminal restlessness (see terminal agitation)
- uncontrolled delirium (see delirium)
- severe breathlessness (see dyspnoea)
- massive haemorrhage (see haemorrhage)
- neurogenic or cardiogenic pulmonary oedema
- intractable distress

How palliative sedation is achieved

- the level of sedation should be titrated to removal of distress
- drugs
 - benzodiazepines e.g. midazolam, clonazepam
 - sedating antipsychotics e.g. levomepromazine (methotrimeprazine) (subcut 12.5 to 200 mg/24hours)
 - barbiturates e.g. phenobarbitone (subcut 600 to 1,200 mg/24 hours)
 - dexmedetomidine - experience in palliative care is limited
 - opioids
 - > BUT increasing doses may not result in increased sedation (opioids tend only to be sedating in the opioid naive) and may instead induce respiratory depression or seizures

Sedation of this type may be subject to the principle of 'double effect' which has the dual effects of intentional relief of suffering and increased risk of hastening death. Palliative sedation itself has not been shown to hasten death.

Fear and anxiety

Fear

A brief, reflexive, rational and unpleasant emotional response (being afraid) caused by anticipation or awareness of danger. A present-focused, reality-based reaction initiating avoidant behaviours. Associated with physiological and psychological arousal. May be adaptive and enhance safety, or non-adaptive.

- innate fear (pain, bleeding, being alone, odours, confined spaces, novel places)
- learned fear (dying, death, being buried alive, needles, chemotherapy)

Anxiety

Sustained and excessive uneasiness. Future-focused, irrational, grossly exaggerated response to perceived threat to the 'self', to one's existence. An intrapsychic conflict. Encourages (unsuccessful) attempts to resolve threat.

- may be a normal alerting response
- may be a symptom of a medical condition (e.g. delirium, depression, hormone-secreting tumour), or a symptom of an impending medical catastrophe
- may be the result of an adverse reaction to a drug e.g. bronchodilators, steroids, methylphenidate
- may be a symptom of Generalised Anxiety, Panic or Depressive disorders

Common anxieties and fears centre on

- being ill
- separation from loved ones, homes or jobs
- becoming dependent on others (being a 'nuisance' or 'burden')
- losing control of physical faculties
- failing to complete life goals or obligations
- uncontrolled pain or other symptoms
- abandonment
- not knowing how death will occur
- 'death anxiety' (the fear of non-being)
- spirituality

Management of fear

- avoid threat if possible
- forewarning and preparations
- emotional first aid
- behaviour desensitisation for phobias (a syndrome of pathological fear)
- psychotropic medications of limited effectiveness

Management of anxiety

- careful listening and attention to detail
- support to maintain independence and autonomy
- honest and open discussion about the future with the patient and family at a pace that they can accommodate
- support realistic hope for the future
- provide distractions to avoid boredom and excessive self-reflection
- attend to social and financial problems
- provide focussed spiritual care if appropriate
- psychotropic drugs - may be a useful adjunct
 - benzodiazepines e.g. lorazepam can be very effective in the short term (days to weeks) but this may fade and there is a risk of tolerance and dependency
 - beta-blockers e.g. propranolol may block the peripheral symptoms and thus ease the unease

- antidepressants e.g. escitalopram, fluoxetine may be more effective longer term than benzodiazepines

Raised intracranial pressure

Raised intracranial pressure is a life-threatening event that needs to be carefully assessed and managed to optimise quality of life and minimise symptoms.

Symptoms

- severe headache which is worse when lying down or straining
- vomiting
- convulsions
- mental - drowsiness, delirium
- diplopia
- restlessness

Causes/risk factors

- cerebral metastases (more common with some primaries, e.g. lung, breast, melanoma than with others, e.g. prostate)
- primary brain tumour
- abscess
- cerebro-vascular event
- sagittal sinus thrombosis
- secondary hydrocephalus following surgery

Management

If raised intracranial pressure is suspected look for papilloedema and signs of cerebral irritation. Computerised tomography or MRI may be appropriate.

- raise the head of the bed
- consider cranial radiotherapy or neurosurgery for malignancy if prognosis/status warrants it
- drugs
 - dexamethasone up to 16 mg per day. Avoid doses after noon as may add to insomnia. Gradually reduce dose to minimum effective. Withdraw after 7 days if ineffective (note - some anticonvulsants can reduce effectiveness – see dexamethasone page)
 - codeine (sometimes eases head pain)
 - consider anticonvulsants particularly if seizures are present
 - consider acetazolamide 250 to 500 mg once daily to bd

Convulsions

Convulsions can be distressing not only for the patient but also for the family and other carers. They should be managed effectively to reduce distress and anxiety wherever possible. It is important to have a clear history of the convulsion in order to diagnose the type (grand mal, focal, absence or status epilepticus). At times a convulsion can be mistakenly diagnosed when the true cause of loss of consciousness or absence is a syncopal attack, cardiac arrhythmia, or a transient ischaemic attack.

Causes

- previously diagnosed epilepsy, brain trauma/surgery, brain tumour/mets
- drugs
 - some lower seizure threshold e.g. phenothiazines, tricyclics
 - interactions - antiepileptics have many variable and unpredictable interactions (see individual drug pages)
 - withdrawal e.g. of steroids, alcohol
- metabolic disturbance, e.g. hypoxia, hyponatraemia, hypoglycaemia

Management

Prophylaxis

- drugs
 - consider dexamethasone if related to raised intracranial pressure (primary brain tumour/metastases)
 - sodium valproate initially 500 mg bd to tds increasing every 3 days to 1 to 2 g per day
 - levetiracetam 500 mg bd initially
 - carbamazepine initially 100 to 200 mg once daily to bd increasing by 100 to 200 mg every 2 weeks to 800 to 1,200 mg per day – consider therapeutic drug monitoring of plasma concentrations
 - phenytoin 200 to 300 mg nocte - consider therapeutic drug monitoring of plasma concentrations
 - if oral route is not available consider
 - > clonazepam 1 to 4 mg/24 hours by subcut infusion
 - > midazolam 10 to 60 mg/24 hours by subcut infusion
 - > consider the use of phenobarbitone if convulsions are not effectively managed by other agents

Grand mal convulsions or status epilepticus management

- make the patient safe, explain what is happening and reassure
- drugs
 - rectal diazepam 10 to 20 mg
 - buccal midazolam 5 to 10 mg - between the cheek and gum
 - subcut boluses of clonazepam or midazolam
 - if these measures are not effective consider the use of phenobarbitone