Improving quality is harder than it looks

If only people understood their condition and its treatment better, they would be more likely to adhere to treatment and thus have better outcomes—right? No. An education intervention in hospital and post-discharge for people with heart failure with reduced ejection fraction was tested in a cluster randomised trial called CONNECT-HF. It did not improve clinical outcomes (defined as rehospitalisation for heart failure and all-cause mortality) or care (defined as the percentage of recommendations followed).

On one hand, this negative trial result could be seen as disappointing. On the other hand, resources devoted to such endeavours could potentially be allocated to strategies focused on altering prognosis. Or we could quibble about whether an education intervention would be more effective if delivered differently—for example, digitally or in a more personalised way, or targeted to certain patients, such as those with less social support or those with many other comorbidities.

 Quitting smoking after lung cancer diagnosis

Yes to smoking cessation. Obviously, always. But is there a point when it is too late? After the diagnosis of lung cancer, for example, does quitting smoking alter prognosis? Sheikh and colleagues performed a prospective cohort study to address this question in 517 smokers with early stage non-small cell lung cancer in Russia. The difference in survival between those who quit and those who continued smoking was stark: it was 21.6 months longer in those who quit. In an adjusted analysis there was a clear reduction in all-cause mortality, cancer-specific mortality, and disease progression.

Of course, this is observational research, so the relationship could be confounded. For example, people who are more likely to quit may also be less likely to have other comorbidities, and the mechanism of improved prognosis might not be smoking cessation alone. Still, it’s hard not to be convinced by these data.

 Covid-19, strokes, and heart attacks

Katsoularis and colleagues’ Swedish study confirmed covid-19 as a risk factor for myocardial infarction and stroke. A cynic might say that any critical or inflammatory illness is a cardiovascular risk factor. It seems that there is more to it than just that, although this study only compared the risk to that of the background population, so we don’t know. This study (the first to use self-controlled case series methodology in this area) supports the theory that covid-19 predisposes a person to thromboembolic events, but it cannot determine how specific that risk is to covid-19 compared with, say, other serious viral infections. However, it is generally accepted to carry a higher risk than influenza.

The authors suggest that these acute cardiovascular complications of covid-19 warrant prioritisation of prevention strategies. I’m not sure this study makes prevention any more important than it was already, but for people and organisations who aren’t yet convinced perhaps this is important. I think the take-home message here is more that therapies could be targeted at preventing or reducing the impact of the cardiovascular complications from covid-19, and perhaps to be vigilant for the onset of myocardial infarctions and strokes.

 Disruption to routine childhood vaccination

The pandemic has destroyed many good things for so long that it is easy to be apathetic about things we once considered essential and routine. One of the greatest (but not that much talked about) successes of the modern era is widespread childhood vaccination, such as for measles and diphtheria-tetanus-pertussis. Globally, millions of children have missed doses for these vaccines, leaving them under-vaccinated or unvaccinated against preventable diseases at the end of 2020. Children in north Africa and the Middle East were particularly affected. Causey and colleagues’ modelling study indicates that these gaps are likely to extend throughout 2021. Targeting resources to make up the missed doses is going to be key.

 Covid-19 in vaccinated healthcare workers

If you wanted to research how protective vaccines are, you’d do a randomised trial, wouldn’t you? You’d randomise people to vaccine or placebo and see how many people got infected and how many severely so. That’s the most unbiased assessment. But there’s another piece of the puzzle—what makes people less likely to be protected by the vaccine?

Bergwerk and colleagues’ case-control analysis of more than 1000 fully vaccinated healthcare workers found that the infected workers (cases) had lower neutralising antibody titres than the controls (uninfected vaccinated workers). Reassuringly, most of the cases were mild or asymptomatic. Most cases (85%) involved the B.1.1.7 variant. I suppose this is also reassuring because at least the vaccine protected against what it was initially designed to protect against, and we just have to wait for the booster research.

Alex Nowbar is a clinician at Imperial College London
Does depression screening in primary care improve mental health outcomes?

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This is one of a series of occasional articles that highlight areas of practice where management lacks convincing supporting evidence. The series adviser is Nai Ming Lai, clinical editor. You can read more about how to prepare and submit an Education article on our Instructions for Authors pages: www.bmj.com/about-bmj/resources-authors/article-types

Depression is usually identified when patients report symptoms or when clinicians recognise them through routine assessment of patient wellbeing. Screening can potentially increase rates of depression recognition. Depression screening involves administering a symptom questionnaire to all patients not known or not suspected of having depression. It is intended to identify symptomatic people who may not otherwise be recognised or seek treatment.1 2 A cut-off is used to classify positive and negative results, with further assessment of those with positive results, and, as appropriate, management. The Patient Health Questionnaire-9 (PHQ-9) is among the most used depression screening tools in primary care.3

In the UK, the National Institute for Health and Care Excellence (NICE) encourages general practitioners to be alert to possible depression, but not to screen routinely.4 The National Screening Committee recommends against screening.5 Depression screening in general practice was financially incentivised by the UK Quality and Outcomes Framework from 2006 to 2013 but was subsequently removed owing to disappointing results; almost 1000 patients had to be screened for each new depression diagnosis and almost 700 for each new antidepressant prescription.6 In North America, the Canadian Task Force on Preventive Health Care (CTFPHC) recommends against screening,7 whereas the US Preventive Services Task Force (USPSTF) recommends screening all primary care patients “with adequate systems in place to ensure accurate diagnosis, effective treatment, and appropriate follow-up.”8 This is described as, at a minimum, dedicated nursing staff to manage the screening process and protocols for referral to evidence based behavioural treatments. More intensively, it involves components such as dedicated staff training programmes, mental health specialists to conduct assessments, trained therapists, and co-payments for medications.9 Only 3% of US adult ambulatory care visits in 2015 included depression screening, however, even though it has been recommended by the USPSTF since 2009.10

Screening tool cut-off points are typically set to maximise combined sensitivity and specificity, but this does not cover important clinical considerations, such as minimising false positive screens, identifying patients with high symptom levels, or...

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WHAT YOU NEED TO KNOW

- International guidelines and practice differ regarding screening for depression; it is not currently recommended in the UK
- Little high quality evidence is available from primary care settings on the benefits of depression screening in improving mental health outcomes for patients
- Instead of routinely screening all patients in primary care, engage patients in discussions about their overall wellbeing, including mental health, and be alert to clinical cues that could suggest depression

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UNCERTAINTIES

In the UK, the National Institute for Health and Care Excellence (NICE) encourages general practitioners to be alert to possible depression, but not to screen routinely. The National Screening Committee recommends against screening. Depression screening in general practice was financially incentivised by the UK Quality and Outcomes Framework from 2006 to 2013 but was subsequently removed owing to disappointing results; almost 1000 patients had to be screened for each new depression diagnosis and almost 700 for each new antidepressant prescription. In North America, the Canadian Task Force on Preventive Health Care (CTFPHC) recommends against screening, whereas the US Preventive Services Task Force (USPSTF) recommends screening all primary care patients “with adequate systems in place to ensure accurate diagnosis, effective treatment, and appropriate follow-up.” This is described as, at a minimum, dedicated nursing staff to manage the screening process and protocols for referral to evidence based behavioural treatments. More intensively, it involves components such as dedicated staff training programmes, mental health specialists to conduct assessments, trained therapists, and co-payments for medications. Only 3% of US adult ambulatory care visits in 2015 included depression screening, however, even though it has been recommended by the USPSTF since 2009.

Screening tool cut-off points are typically set to maximise combined sensitivity and specificity, but this does not cover important clinical considerations, such as minimising false positive screens, identifying patients with high symptom levels, or...

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SOURCES AND SELECTION CRITERIA

We reviewed systematic reviews conducted to support depression screening guidelines for adults in general practice or in other populations (women during pregnancy or postpartum, children and youth) by the United Kingdom National Screening Committee, the CTPHC, and the USPSTF for randomised controlled trials (RCTs) that investigated the effects of depression screening interventions on health outcomes. We then searched for more recent trials via Medline, Medline Epub Ahead of Print, in-process and other non-indexed citations, PsyCInfo, Embase, CINAHL, and the Cochrane Registry of Controlled Trials through March 12 2021. Search terms, which included “depression,” “depressive disorder,” “mass screening,” and “screen*” are available online (https://osf.io/ptqdk/). We searched for RCTs that compared outcomes among participants randomly assigned to screening versus no screening. To avoid conflating effects of screening and different treatment options, we limited our analysis to RCTs in which participants in both arms had access to similar options for depression management.11 13 We excluded trials that compared communication or management strategies among patients with positive depression screens or an established diagnosis of depression, because in practice decisions about screening need to occur before screening results are known.
What is the evidence of uncertainty?

Successful depression screening would require patients without known depression to agree to be screened, identification of a significant number of new cases while limiting false positive screens, and effective treatment of newly identified cases. Trials of screening programmes must determine eligibility and randomly assign before screening, exclude patients already known to have depression or in depression treatment, and provide similar depression care options to patients in screened and unscreened trial arms to avoid confounding screening and management effects.1 11 A 2008 Cochrane review12 reported that interventions that included depression screening did not reduce depressive symptoms (five randomised trials; standardised mean difference—0.02, 95% confidence interval -0.25 to 0.20). Only one trial included in the review13 randomly assigned participants not known to have depression to be screened or not screened and appropriately separated screening and treatment effects.

We identified four additional, more recent, trials that have evaluated depression screening in specific patient groups such as postpartum women,14 patients with osteoarthritis,15 patients after an acute coronary syndrome,16 and post-deployment military personnel.17 These trials reported mixed results or found that mental health symptoms were unimproved among participants randomly assigned to screening; three trials found no differences in mental health symptoms or wellbeing between screened and unscreened participants,131617 one trial reported both results that showed no difference and results that favoured screening,14 and one trial reported results that showed no difference and results that were worse for screened participants.15 Table 1 (bmj.com) shows trial details.

We did not identify adequately powered, well conducted trials on the benefits and harms of depression screening in general practice patient populations. The diverse populations and screening approaches used in the trials we identified, along with small sample sizes and methodological limitations in some, result in uncertainty about whether routine screening would reduce depression in general practice.

WHAT PATIENTS NEED TO KNOW

• As many as 1 in 10 patients in general practice settings may have depression, and this may be as high as 1 in 5 for patients with some chronic medical conditions
• Most mild depression symptoms go away quickly without medical attention, but this is not always the case; symptoms that are ongoing and serious enough to affect the ability to enjoy social interactions or take care of home or work responsibilities usually require treatment
• Using a questionnaire to screen for depression may not improve mental health outcomes compared with clinicians talking to patients about their experiences and concerns to determine if they may be depressed
• Effective treatments for depression are available. If you are experiencing symptoms that might be related to depression, such as sad mood, difficulty enjoying activities that you normally like, feelings of worthlessness or guilt, fatigue or lack of energy, or changes in your sleep patterns, it is important to discuss these with your healthcare provider
• Your healthcare provider can discuss with you your symptoms and can help you decide if you would like to undergo treatment, which usually involves taking medication or engaging in psychological therapy. They can also discuss advantages and disadvantages of options, and help you to determine your preferences
**RECOMMENDATIONS FOR FURTHER RESEARCH**

**Objectives:** To test whether different depression screening approaches, with standard or enhanced management options, improve mental health compared with

- not screening but providing access to the same management options
- healthcare provider education programmes which would seek to improve depression identification and management. Additionally, education programmes would ideally be tested against no-screening usual care.

**Design:** Clustered pragmatic trials with general practices randomly assigned to screening, non-screening usual care, or healthcare provider and patient education trial arms.

**Population:** All adults in general practice settings without a current diagnosis of depression and not receiving treatment for depression. In addition, screening that targets patients with risk factors (eg, social disadvantage, long term unemployment) may be considered.

**Interventions:**

Option 1 (dichotomous screening). Positive and negative results determined using an a priori defined cut-off point. Participants with positive screens are assessed for depression and, if appropriate, receive depression treatment. Treatment may be limited to treatments available in usual care or may include enhanced depression care with staff assistance to ensure accurate diagnosis, guideline consistent treatment, and follow-up.

Option 2 (risk based screening). Risk levels are determined by a model using actual screening tool scores and patient characteristics with several intervention options available (eg, watchful waiting, low intensity management option, or high intensity management option) based on risk and shared decision making.

Option 3 (education). Depression identification and management education is provided to healthcare providers to attempt to improve identification, communication with patients, and management.

**Comparison:**

Option 1 (screening or education compared with no-screening usual care). Participants in comparison trial arm are not screened for depression. Participants identified as possibly depressed via self-report or unassisted recognition by a healthcare professional are assessed for depression, and, if appropriate, receive depression treatment. Management options should be the same as in the intervention arm.

Option 2 (screening compared with education). Head-to-head comparison of screening (dichotomous or risk based) and education.

**Outcome:** The effect of depression screening on the severity of depressive symptoms, number of depression cases, suicidal thinking and attempts, and quality of life.

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**What should we do in light of the uncertainty?**

Instead of screening with symptom questionnaires, we encourage clinicians to engage patients in discussions about their overall wellbeing, including mental health. Recognise that depression may be a process that takes more than a single consultation to investigate. Be alert to clinical cues that could suggest depression, particularly among patients at risk because of factors such as family or personal history of mental health concerns, including problematic substance use, unexplained medical symptoms, or overly frequent use of medical services. These include both somatic cues, such as insomnia, anhedonia, or fatigue, and psychological cues, such as low mood or overly negative thinking. If mental health concerns are reported by a patient or are otherwise identified, provide education about depression and other common mental health conditions, including the different ways that symptoms may be experienced and, when appropriate, discuss different management options.

As national guidelines differ, clinicians are expected to be aware of and adhere to local guidance regarding screening. Until further evidence becomes available, it will be important to make an informed decision regarding screening in primary care after considering the benefits and harms. Depression screening would require substantial resources. Busy general practitioners must evaluate or refer all patients who have positive screens. Like other types of screening, it can also lead to overdiagnosis or misdiagnosis. Overdiagnosis occurs in depression when people with mild, transient symptoms are diagnosed and treated, but without benefit, because symptoms will subside without intervention. Misdiagnosis can occur if screening leads to some people being diagnosed and treated even though they do not meet diagnostic criteria, including people with symptoms resulting from another health condition.

Away from the context of screening, depression symptom questionnaires are often used in general practice settings for other purposes. They can be useful for assessing and discussing symptoms among patients who may be unsure if they have depression and for monitoring treatment response among patients with a diagnosis of depression.

**Competing interests:** SM, DBR, and RCZ have no relevant interests to declare. BDT is chair of the Canadian Task Force on Preventive Health Care, which develops guidelines on prevention in primary care, including on depression screening; he does not serve as a voting Task Force member on depression screening guidelines.

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**Is ongoing research likely to provide relevant evidence?**

We searched Clinicaltrials.gov, WHO International Clinical Trials Registry Platform, and ISRCTN for ongoing trials. We did not identify any ongoing depression screening trials in any setting that planned to assign people randomly not known to have depression to screening or no-screening conditions and that appropriately separated screening and management.

Research on screening tool accuracy and methods is under way. We are part of an international collaboration (https://www.depressd.ca/) that is aggregating large databases from primary studies on depression screening tool accuracy. One goal of the collaboration is to determine how clinicians might move away from a crude dichotomous screening approach and instead use individualised risk estimates based on actual screening tool scores and individual risk factors (eg, sex, age, or medical comorbidities). Such an approach could increase precision for individual patients. It could also be used to engage patients in shared decision making and to identify appropriate care options, as recommended better by NICE.

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**EDUCATION INTO PRACTICE**

- How do you ensure that patients know you are able to help them if they are depressed and want to communicate their mental health concerns to you?
- How would you discuss patients’ wellbeing with them and integrate questions about their mood and experiences that will allow you to evaluate if you should further assess for depression?
- What local referral resources do you have for patients who would benefit from additional assessment or mental health treatment, and are they accessible to patients with limited resources?
ESSENTIALS

Ward based management of behavioural and psychological symptoms of dementia

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An 81 year old woman with vascular dementia has been admitted to the hospital following a fall in the community and is currently an inpatient on the ward. You are the doctor on call and have been asked to review the patient as she appears more confused and is trying to leave the ward.

Worldwide, dementia affects 50 million people, with a predicted increase to 82 million by 2030.1 Dementia is characteristically progressive and severely affects activities of daily living. Behavioural and psychological symptoms of dementia (BPSD) refer to a cluster of symptoms of dementia relating to perception, mood, thought content, or behaviour.2 They are associated with increased care costs and are important prognosticators of admission to nursing homes. It is estimated that more than 80% of people with dementia will experience BPSD during the course of the disease.3

BPSD are challenging to manage for patients, carers, and healthcare workers, but effective management can substantially improve quality of life for both patients and carers.

As people with dementia make up a relatively high proportion of hospital inpatients, clinicians working in an acute setting are likely to encounter BPSD often.

This article aims to introduce clinicians to BPSD, their key triggers in an inpatient setting, and ward based management. We will focus on strategies employed by junior doctors, though overall care is typically within a multidisciplinary setting.

WHAT YOU NEED TO KNOW

- Behavioural and psychological symptoms of dementia (BPSD) can be exacerbated by a plethora of underlying reversible factors (such as poor pain control)
- Identifying possible environmental or clinical triggers for a patient’s symptoms is the main aim of assessment in someone with an exacerbation of BPSD
- Non-pharmacological interventions, such as managing environmental factors, are first line and should be tailored to the individual patient
- When considering pharmacological intervention, focus on medications with evidence of benefit in BPSD. Drug interventions will not be able to address all symptoms
- Pharmacological sedation should only be used in emergency situations

<table>
<thead>
<tr>
<th>Box 1</th>
<th>The four main clusters of BPSD, including their associated features and their prevalence (%) in patients with dementia1–7</th>
</tr>
</thead>
</table>
| Affective | • Agitation (41.5%)  
  – Easily upset  
  – Repetition of questions  
  – Arguing or complaining  
  – Hoarding  
  – Pacing  
  – Inappropriate screaming, crying, or disruptive sounds  
  – Wandering  
  • Depression or dysphoria (58.5%)  
  – Insomnia |
| Psychotic | • Delusions (10–73%)  
  • Hallucinations (15–49%)  
  • Psychiatric symptoms (27.6%) |
| Hyperactive | • Aggression (46.2%)  
  – Physical, verbal  
  • Disinhibition (61.4%)  
  – Socially/sexually inappropriate behaviour  
  – Repetitive vocalisation  
  • Irritability/lability (44.6%)  
  – Resisting care  
  – Restlessness  
  • Motor disturbances (57.5%)  
  – Repetitive actions  
  – Wandering  
  – Rummaging  
  • Night time disturbances (66.7%)  
  – Waking during the night |
| Apathetic | • Indifference (80%)  
  • Appetite and eating problems (16.7%) |

HOW PATIENTS WERE INVOLVED IN THE CREATION OF THIS ARTICLE

No patients were involved in the creation of this article.
What are the clinical features of BPSD?

Within the literature, the clinical features of BPSD are grouped into four clusters: affective, psychotic, hyperactive, apathetic (box 1). The prevalence of each cluster and its composite symptoms is variable, but particularly common features include indifference, disinhibition, motor and night time disturbances, and depression. Agitation, irritability, and aggression are also common and can be particularly challenging for carers and healthcare staff to manage. 5 7

What’s the difference between delirium and BPSD?

Delirium and BPSD have overlapping manifestations and can also occur concurrently. BPSD are chronic symptoms in a number of domains that are secondary to underlying dementia. They are persistent, may be progressive, and can be worsened by certain triggers. Delirium is defined as a transient, usually fluctuant change in a patient’s attention, cognition, or consciousness that is associated with an identifiable but reversible precipitant and is not better explained by an underlying dementia. Anybody can develop delirium, but only people with dementia have BPSD. BPSD fluctuate over time, but can gradually worsen as the underlying dementia progresses. Likewise, patients can also suffer exacerbations of BPSD, where their baseline BPSD worsen suddenly and markedly, frequently owing to reversible triggers including delirium. Patients with BPSD are at particular risk for developing delirium superimposed on BPSD and may take longer to recover from delirium. In an acute setting, the management strategy is similar: identify and correct any triggers for the patient’s deterioration. Once the cause of the delirium is resolved, it may take up to a few months for patients to return to their baseline function.

A patient’s capacity to consent to a particular treatment can change over time

How are BPSD assessed?

People with dementia may struggle to communicate, so adapt your communication style (box 2) and take a collateral history from a family member or carer. When taking a history, ask about the history of presentation, the patient’s cognitive and functional baseline, their care needs, the home environment, and current support in the community. Ask about the patient’s baseline ability to perform activities of daily living. Are they able to feed themselves? Are they mobile? Are they incontinent? How many carers do they get in a day and what do they usually need assistance with? Do they have any particular likes and dislikes? A lot of these questions can be found in the “This is me” leaflet which can be found on the Alzheimer’s website. These details can be included in the patient’s notes so that members of the multidisciplinary team are aware of how to tailor their approach to the patient and provide support. The information can also help with planning a safe discharge from hospital and identifying the need for any additional support (eg, package of care arrangements, mobility aids, safety aids, or carer support). 10

When managing dementia patients with BPSD, undertaking a capacity assessment is vital. An assessment of capacity is specific to each decision and is independent of any prior diagnoses made. Capacity is also time-specific, meaning a patient’s capacity to consent to a particular treatment can change over time.

Finally, all patients are assumed to have capacity. For a patient to lack capacity, they must first have an impairment of brain function that could affect their ability to make decisions for themselves. If so, a capacity assessment must be undertaken. This involves identifying whether the patient can a) understand the information provided to them, b) retain that information, c) weigh up the information, and d) communicate their decision. If a patient is unable to perform any one of these processes then they do not have capacity for that specific decision and at that specific time. If the patient’s capacity is likely to change, or the decision can be delayed, then reassess capacity at a later time

BPSD may be triggered or exacerbated by several factors. Triggers can be categorised as environmental and social factors, physiological factors, and pharmacological factors. The impact of any one trigger on a patient’s clinical status is variable and differs between patients. Quite often, several triggers may contribute to a patient’s presentation. The only way to assess the impact of each trigger is to systematically identify any possible triggers, treat them appropriately, and evaluate the patient’s response to treatment. In some cases, no clear trigger for the patient’s symptoms can be identified: in such cases, manage the symptoms and reassess regularly for any triggers or precipitants that may later reveal themselves.

The following section highlights potential triggers to consider as part of a focused history and physical examination.

Environmental and social factors

Consider environmental factors in people with an exacerbation of BPSD. Common examples include disorientation to one’s surroundings, poor lighting, excessive noise, soiled bedding, and excessive temperature variations. Lack of environmental stimulation, as well as lack of positive social interaction
with dementia may struggle to communicate pain directly, increasing the importance of identifying non-verbal cues, such as facial expression, by using a pain assessment tool. Figure 1 offers examples of pain assessment systems that can be used depending on the patient’s cognitive state.

Many patients find invasive devices such as catheters uncomfortable, therefore consider early removal of catheters where possible.

Malnutrition may be a concern, so consider input from a dietitian. Deficiencies of vitamin B12 and folate are potential reversible causes for cognitive impairment and are commonly seen in the older population. Vitamin B12 deficiency is especially associated with reduced immune function, insomnia, cognition, and neurological function.

Appropriate management of any of these precipitants may secondarily improve BPSD.

**Pharmacological factors**

Exacerbations of BPSD can also be caused by polypharmacy. Withdrawing unnecessary medication or finding alternatives using the STOPP START medication tool can be helpful. Additionally, review medication dosages, especially for those with renal or hepatic impairment.

Key medications to be aware of are outlined in table 2. In some cases, it may be worth referring to online toolkits such as the STOPP START medication tool and discussing changes to a patient’s medication with other relevant specialists or pharmacists.

**Social and psychological factors**

Social and psychological factors can contribute to the development or exacerbation of BPSD. For patients who are in hospital, separation from a long term partner or recent bereavement may increase agitation or anxiety.

**Physiological factors**

Organic physiological derangements can precipitate or exacerbate delirium and BPSD. The patient’s history may have provided indicators of an underlying physiological trigger, such as symptoms associated with infection. Even if the history does not overtly suggest physiological factors are at play, an assessment of the clinical status of a patient through examination of relevant systems and investigation might point to a possible physiological trigger. Table 1 outlines useful investigations for patients presenting with an acute deterioration in BPSD. Further tests can be considered depending on the history. For example, a computed tomography scan of the head may be useful where the patient has a history of head injury, loss of consciousness, reduced consciousness, seizures, focal neurology, or if a patient is on anticoagulation. These investigations can also offer a helpful comparator for any future deterioration during the admission.

A useful mnemonic (box 3) outlines some key physiological factors that are often forgotten but should be considered in any case of delirium or BPSD.

Poor pain control often exacerbates BPSD symptoms, and a pain assessment is an important part of management. If a patient requires pain management, conduct regular reviews to ensure that pain is sufficiently controlled. Patients can also adversely affect the patient’s mood and lead to loneliness and frustration.

**Box 3**

Mnemonic for causes of delirium or BPSD in patients with or without dementia

| Consider | DELIRIUM factors | Drugs or withdrawal | Electrolyte disturbance | Level of pain | Infection | Respiratory failure | Impaction of faeces | Urinary retention | Metabolic disorders |
---|---|---|---|---|---|---|---|---|---|
**D**rugs or withdrawal | Electrolyte disturbance | Level of pain | Infection | Respiratory failure | Impaction of faeces | Urinary retention | Metabolic disorders |
**E**lectrolyte disturbance | Level of pain | Infection | Respiratory failure | Impaction of faeces | Urinary retention | Metabolic disorders |
**L**evel of pain | Infection | Respiratory failure | Impaction of faeces | Urinary retention | Metabolic disorders |
**I**nfection | Respiratory failure | Impaction of faeces | Urinary retention | Metabolic disorders |
**R**espiratory failure | Impaction of faeces | Urinary retention | Metabolic disorders |
**I**mpaction of faeces | Urinary retention | Metabolic disorders |
**U**rinary retention | Metabolic disorders |
**M**etabolic disorders |

**Table 1** Routine investigations in patients with delirium or new BPSD

| Bedside | Bloods | Imaging |
---|---|---|
Oximetry | Full blood count, C reactive protein | Chest radiograph |
Glucose | Urea and electrolytes, liver function tests, thyroid function tests |
Electrocardiogram | Haematinics, including iron, B12 |
Urine dip-midstream specimen | Folate |
Bladder scan | Bone profile+Mg2+ |
Blood cultures | |
Covid-19 testing | |

**Fig 1** Image showing different pain assessment scales and tools that can be utilised in clinical practice.
Management

In managing patients with BPSD, the primary objective is reducing their distress enough to allow them to engage with those around them (ie, carers, healthcare professionals, and relatives) and return to their baseline ability to perform daily activities and cognition. This is principally aimed to ensure the safety of the patient and those around them and improve the patient’s quality of life.

The management of BPSD generally follows a stepwise approach: any triggers are first identified and corrected, and non-pharmacological interventions (below) are trialled. Only if this fails should you consider using medications to target BPSD. The only exception to this is in emergency situations where the risk to the patient or others requires rapid pharmacological intervention.

The recommendations made below are based on guidelines from the National Institute for Health and Care Excellence (NICE) where outlined, as well as expert opinion. However, as with most clinical decisions, it is advisable that you consult a senior clinician before making decisions. This is particularly important as many patients have multiple comorbidities and prescribers will need to consider and be cautious of drug interactions. Additionally, patients might have advanced directives or family members who can provide insight into the patient’s wishes regarding management. Assessing the patient’s capacity to ensure they can make decisions about treatment is also important.

Non-pharmacological methods

Non-pharmacological interventions include optimising the environment for the patient and providing extra stimulation via therapeutic methods. For example, a systematic review looking at 20 studies between 2005 and 2015 found evidence that music therapy, aromatherapy, physical exercise, touch therapy, and stimulating via therapeutic methods. For example, it can be done in an A-D assessment: “A” being the activating events before the behaviour; “B” describing the behaviour displayed; “C” describing how the carers or staff responded as a consequence to the behaviour; and “D” describing the de-escalation and debriefing period.

Deprivation of liberty safeguards are made under the Mental Capacity Act. They allow care homes and hospitals to keep patients in their facility with 24 hour a day supervision to provide the care that they believe to be in the patient’s best interest. Any restraint or restrictions imposed should be kept at a minimum where possible and this is only done when the patient lacks the capacity to consent to vital care offered to them. This requires approval from the local authority and requires regular review.

Where a patient is lonely, seeking psychological support or encouraging visits may improve symptoms

Where a patient is lonely, seeking psychological support or encouraging visits may improve symptoms. This may be challenging during covid-19 pandemic restrictions but video calls and activities can be coordinated with nursing and healthcare staff or medical student volunteers.

During a patient’s time in hospital, assess the carers’ wellbeing and capacity to support the patient in the community. Depression is commonly seen among carers.

![Table 2 | Examples of medications to review in patients experiencing BPSD](https://example.com/table2)

<table>
<thead>
<tr>
<th>Category of medication</th>
<th>Drug</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS acting medication</td>
<td>Narcotics (in particular meperidine)</td>
<td>Delirium</td>
</tr>
<tr>
<td></td>
<td>Benzodiazepines</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Non-steroidal anti-inflammatories</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anticonvulsants (eg, barbiturates)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Antiparkinsonian agents (eg, benzotropine, trihexyphenidyl)</td>
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<tr>
<td></td>
<td>Opioids</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anticholinergic drugs (eg, oxybutynin)</td>
<td></td>
</tr>
<tr>
<td>Cardiovascular drugs</td>
<td>Disopyramide</td>
<td>Delirium</td>
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<td></td>
<td>Antiarrhythmics</td>
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<td></td>
<td>Digitalis</td>
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<td></td>
<td>Antihypertensives (β blockers, methyldopa)</td>
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<td>Respiratory drugs</td>
<td>Theophylline</td>
<td>Delirium</td>
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<td></td>
<td>Ipratropium (nebulised)</td>
<td>Increased risk of glaucoma</td>
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<tr>
<td>Psychotropic drugs</td>
<td>Antidepressants (paroxetine in particular)</td>
<td>Increases agitation, confusion, hypotension</td>
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<td>Dopaminergic drugs</td>
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<td></td>
<td>Lithium</td>
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<td></td>
<td>Antipsychotics</td>
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<td>Tricyclic antidepressants</td>
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<tr>
<td>Anaesthetic agents</td>
<td>Ketamine</td>
<td>Vivid hallucinations or unpleasant dreams</td>
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<tr>
<td>Gastrointestinal medication</td>
<td>H2 blockers (especially cimetidine)</td>
<td>Delirium</td>
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<td>Antipsomimetics</td>
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<td>Antihistamines (first generation)</td>
<td>Diphenhydramine</td>
<td>Sedative effect, falls</td>
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<td>Chlorpheniramine</td>
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<tr>
<td>Antimetetics</td>
<td>Scopolamine</td>
<td>Delirium</td>
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<td></td>
<td>Dimenhydrinate</td>
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<tr>
<td>Antibiotics</td>
<td>Fluoroquinolones</td>
<td>Delirium</td>
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<td>Diabetic</td>
<td>Insulins</td>
<td>Hypoglycaemia</td>
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<td>Sulfonylureas</td>
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<td>Vasodilator antihypertensives</td>
<td>Hydralazine</td>
<td>Falls</td>
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<td>Minoxidil</td>
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<td>Sildenafil</td>
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<tr>
<td>Herbal</td>
<td>Mandrake</td>
<td>Delirium</td>
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<td>Henbane</td>
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<td>Jimson weed</td>
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<td>Atropa belladonna extract</td>
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<tr>
<td>Other</td>
<td>Steroids</td>
<td>Delirium</td>
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This may reduce their ability to interact positively with the patient, and could result in a cycle whereby the health of both carer and patient deteriorates. Long term management may require support for the carers at home. For example, social services can arrange a formal carer assessment which may be useful in providing financial aid, additional homecare support, and adaptations around the home. Support groups and professional counselling may also be beneficial for carers who are struggling with the pressures of their role.

Pharmacological methods
Medication may be required if non-pharmacological interventions have failed, or in emergency situations. However, medications are not effective for all BPSD and may require support for carers at home. Support groups and professional counselling may also be beneficial for carers who are struggling with the pressures of their role.

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Vancomycin induced infusion syndrome

This is vancomycin induced infusion syndrome on the back of a man in his 20s.

Vancomycin infusion (500 mg/h) was given to treat *Streptococcus viridans* infective endocarditis, but 30 minutes after initiation the patient developed a diffuse erythematous and itchy maculopapular rash on his mid to lower back associated with hypotension (blood pressure 71/42 mm Hg).

Vancomycin induced infusion syndrome is caused by mast cell mediated histamine release and cutaneous vasodilation usually secondary to infusion of vancomycin.

Drug reaction eosinophilia with systemic symptoms (DRESS syndrome) constitutes a close clinical differential diagnosis, which presents with a similar rash but also has features of lymphadenopathy, eosinophilia, and increased liver enzyme levels, none of which were present in this patient.

Vancomycin induced infusion syndrome should be suspected in patients presenting with this characteristic cutaneous reaction after vancomycin infusion. Although risk is greater when infusion rate is >1 g/h, patients might tolerate subsequent treatment without reaction if infusion is slower.

If you would like to write a Minerva picture case, please see our author guidelines at http://bit.ly/29HCBAL and submit online at http://bit.ly/29yyGSx

Progression of aortic stenosis

There’s an inverse relation between calcification in arteries and calcification in bone. People with osteoporosis are at increased risk of arterial disease.

However, treatments for osteoporosis seem to have little effect on blood vessel mineralisation. A randomised controlled trial of the bone resorption inhibitors denosumab and alendronate in people with aortic stenosis found no improvements in aortic valve calcium score or peak aortic jet velocities after two years of treatment (Circulation doi:10.1161/CIRCULATIONAHA.121.053708).

Adverse events in clinical trials

Serious adverse events in clinical trials of renin-angiotensin system blockers were about four times less common than adverse events in hypertensive patients getting similar treatment but not taking part in a trial. The reason isn’t clear. People taking part in trials may be fitter with fewer comorbidities. Or perhaps adverse events are under-ascertained and under-reported in trials. Either way, it makes translating trial results to the real world unreliable (Lancet Healthy Longevity doi:10.1016/S2666-7568(21)00092-1).

Coffee and dementia

A few years ago, an umbrella review in *The BMJ* (doi:10.1136/bmj.j5024) summarised a vast amount of data to conclude that coffee drinking was safe and unlikely to harm health. The finding that high levels of coffee consumption carry an increased risk of dementia challenges this view. Among 400,000 participants in the UK Biobank, people who reported drinking more than six cups of coffee a day were most likely to develop dementia (Nutr Neurosci doi:10.1080/1028415X.2021.1945858).

Pay it forward

Most people, if they receive a loan, expect to pay it back. An altruistic alternative, if the original donor is prepared to write off the loan, is to pay it forward by making a gift to a third person. Pay-it-forward programmes have expanded during the covid-19 pandemic, and a review in Nature Medicine (doi:10.1038/s41591-021-01401-x) discusses how they provide opportunities to reduce healthcare costs and increase uptake of interventions such as testing and vaccines.

Omega-3 fatty acids

A longitudinal study from the US reports that, over 11 years of follow-up, levels of fatty acids measured in erythrocytes were as strongly associated with mortality and cardiovascular events as traditional risk factors such as blood pressure, serum lipids, and diabetes. People in the top fifth of the distribution of erythrocyte omega-3 fatty acid levels had a life expectancy roughly five years longer than those in the bottom fifth (Am J Clin Nutr doi:10.1093/ajcn/nqab195).