

## ***Neuropsychological (mood and cognition) consequences of stroke***

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## Introduction

Stroke is the lead cause of disability in United Kingdom (UK) adults, third lead cause of death as well as a national and international research priority. Direct costs to the National Health Service (NHS) are £3 billion, with indirect care costs of £2.4 billion. Half of all stroke units in England have no access to psychology services and on recent survey only two in ten stroke survivors considered they had received the support they need to cope emotionally. There is much service development work to do.

Neuropsychological (mood and cognition) stroke consequences are prevalent, complex and overlapping. This article guides on assessment formulation and treatment of these aspects.

## Clinical assessment of neuropsychological stroke consequences

Any comprehensive stroke neuropsychological assessment process must involve patient interview, and consented interviews with a spouse/relative and rehabilitation team member(s) who know the patient well.

### *Cognitive Impairment*

Stroke is an acquired brain injury. Cognitive deficits occur in 30-44% of stroke survivors and associate with negative treatment response, poor functional outcome and reduced quality of life (Lincoln et al, 2012). Routine cognitive screening after stroke is recommended e.g. National Stroke Strategy, and many UK hospitals have adopting this. There remains a lack of consensus however as to how and when in the care pathway this should be positioned. An early screening strategy might seem appealing but could be impracticable and confer unfavourable clinical/organisational outcomes such as over-diagnosis (Lees & Broomfield, 2014).

Comment [AA1]: Grammar needs attention here.

An initial cognitive screen should form part of any stroke psychology assessment, irrespective of referral reason or cognitive data already collected. The purpose and possible results should be explained to the patient and consent sought. Repeatable Battery for the Assessment of Neuropsychological Status (RBANS) and Montreal Cognitive Assessment (MoCA) are good start points. A hypothetico-deductive reasoning approach to testing must be followed.

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Stroke patients have a variety of disabilities which affect assessment, including cognition itself. Impaired attention, executive function, mental speed and language are common so achieving a true measure can be problematic when assessment requires a high level of cognitive demand and/or verbal responding. Clinicians need to take account of this, pay careful attention to qualitative (performance) data and where necessary employ bespoke tests if standard measures are not suitable. A collateral history is important. Patient awareness of cognitive/other stroke deficits must be determined as this will influence rehabilitation goals. Do not over burden patients with lengthy testing; fatigue is common and if too many tests are progressed, results could be invalid on this account.

### *Adjustment Distress*

Adjusting psychologically to stroke can be challenging. Depending on stroke type and lesion location, permanent mobility, vision, sensory, cognitive and communication deficits are possible. Life role, self-image and sense of identity may be profoundly impacted. When training colleagues, I [NB] ask they imagine themselves, today, sustaining a disabling stroke. This illustrates powerfully the rapid, permanent changes faced by stroke patients and their families.

There is little concordance between severity of stroke lesion and mood disturbance. Most all patients (assuming intact intellectual awareness) will grieve to some degree

Comment [AA3]: grammar

although emotional passivity following stroke can occur (Lincoln et al, 2012). Anger, frustration, anxiety and sadness are common emotional responses in the first weeks, usually (but not always) a signature of 'normal' adjustment. In this context, tears may be a sign of progress rather than a mood disorder, as patients realise what has happened and what has changed. Distress about distress is common ("Am I losing my mind as well?").

### *Post-Stroke Depression (PSD)*

Not all stroke survivors adjust. On pooled analysis, one third develop major depression (Hackett et al 2005), often once rehabilitation stops and patients return home. PSD impairs functional outcome, raises suicide risk and heightens mortality, independent of stroke type. Rates are highest in younger, poorer stroke survivors.

It can be complex to disentangle PSD symptoms from 'direct' stroke effects. Fatigue, reduced initiation and diminished concentration could reflect PSD, or be unrelated to mood. Careful assessment is needed. Yale Question and Patient Health Questionnaire-2/9 are validated screening measures with recommended stroke cut-offs (see Lincoln et al, 2012). These will complement a careful diagnostic interview and provide baseline and treatment change data. Hospital Anxiety Depression Scale (HADS) has good sensitivity specificity for stroke but it is not in the public domain, so costs to use.

For patients with severe cognitive/ communication impairment, the Depression Intensity Scale Circles (DISCS) have moderate sensitivity/specificity. Pictorial emotion charts can be employed for mood scaling. A number of versions of an observer rating scale, the Stroke Aphasic Depression Questionnaire (SADQ) are available. Close working with SLT will assist. Depression screening protocols for the post-acute stroke setting have been developed (Kneebone et al, 2010). Cognitive behavioural maintaining factors must be considered in PSD assessment. It is critical to listen closely to patient and to ask about their thoughts when mood changes for the worst. The Stroke Cognitions Questionnaire, simple (three-column) thought diaries and weekly activity schedules will aid assessment. Depressed stroke survivors show more negative and fewer positive cognitions than non-depressed stroke survivors with heightened focus on what "should" rather than "can" be achieved. Black and white thinking is common ("Unless I do everything as before the stroke, I cannot be happy") with unhelpful content of inadequacy ("I am useless now"), reliance on others ("I am a burden to everyone") and unrealistic recovery ("I should be better now"). Baseline distortions occur where stroke survivors unhelpfully contrast current functioning with how they were the day before the stroke rather than the days after. Such thinking errors are important to detect as they maintain PSD via a cognitive feedback loop: depressed affect promotes negative thinking, further lowering mood and so on. Patient attention becomes focused on deficits, causing demoralisation, hopelessness and apathy. Confidence, personal agency and activity levels are eroded and stroke recovery rate may slow (Broomfield et al, 2011).

### *Post-Stroke Anxiety (PSA)*

Anxiety is common in stroke survivors, affecting 18-24% (Campbell Burton et al, 2012). Like PSD, PSA impairs functional outcome and life quality with highest prevalence amongst younger, socially deprived patients (Broomfield et al, in press). Generalised Anxiety Disorder is a typical presentation with prominent worry (stroke recurrence, family, future) poor relaxation and fear avoidance of rehabilitation and previous activities. There may be symptom overlap with PSD and an association with disturbed sleep. Fear of falling is also common (see Lincoln et al 2012).

There are few validated PSA measures to complement clinical interview. Some studies suggest the HADS Anxiety sub scale has quite low specificity, so stroke patients with anxiety disorders could be missed (see Lincoln et al, 2012). Sensitivity and specificity

data are not available for the Beck Anxiety Inventory. Validation of the Behavioural Outcomes of Anxiety (BOA) and the Tension Rating Circles, an appropriation of the DISCS, is awaited.

Careful assessment of worry content and pattern is important. Clinicians should also determine which relaxation methods (relaxation schedule, music listening) patients actively use, and how often. Frequently, it is none.

### *Post-Stroke Emotionalism (PSE)*

Post stroke emotionalism (PSE) is poorly understood yet affects 21% of stroke survivors at 6 months. Ability to control emotional expression is lessened and sudden unheralded crying episodes, not under normal social control, occur daily or more often usually in response to appropriate stimuli or thoughts but out of proportion. PSE associates with left frontal and temporal lesions and there could be psychological/behavioural maintaining factors (e.g. social avoidance based on fear of disordered crying). PSE disrupts rehabilitation, social functioning and work participation. Almost nothing is known regarding PSE persistence and which factors keep it going. Definitive research is needed.

PSE can overlap with PSD so careful assessment is needed. There are few validated diagnostic instruments. The Center for Neurologic Study-Lability Scale (CNS-LS) has clinical utility but lacks stroke validation. The Pathological Laughter and Crying Scale (PLACS) is used by some. To accurately identify PSE, most often we rely on standard diagnostic interview questions (House et al, 1989):

1. Have you been more tearful since the stroke than you were beforehand? Have you actually cried more in the past week/month(s) (not just felt like it)?
2. Does the weepiness come suddenly, at times when you weren't expecting it? (Suddenly means with only a few moments or no warning, not after several minutes trying to control yourself)
3. If you feel the tears coming on, or if they have started, can you control yourself to stop them? Have you been unable to stop yourself crying in front of other people? Is that a new experience for you?

These aspects should present in the absence of depressed mood (inner sadness) although some patients will report distress due to the tearfulness. It is important to consider co morbid anxiety. PSA could maintain emotionalism by preventing habituation to social and cognitive triggers of emotional outbursts.

### Clinical formulation of neuropsychological stroke consequences

The central endpoint of clinical assessment is a detailed formulation. This will consider all possible explanatory hypotheses (neurological, physical, psychological, social) for the aetiology and maintenance of stroke mood and cognitive consequences, and map out potentially reversible cognitive and behavioural maintaining factors. The interplay of mood, cognition and physical aspects on functional outcome should feature, alongside patient values, pre-morbid coping style and wider familial social and relational frameworks (Wilson & Gracey, 2009). The 'predisposing, precipitating, perpetuating, protective' framework is helpful to determine in what individual way(s) cognitive behavioural or cognitive rehabilitation methods need to be augmented, for that specific patient. Failure to do this will result in poorer treatment outcome. This point is not radical; any clinician working with a specific client group such as stroke must ensure that the therapy provided is an optimal fit for the individual at that time, and not the other way around (Broomfield et al, 2011). An excellent diagrammatic formulation template which adapts well for stroke is published in Clinical Psychology Forum (Doherr, 2013).

## Treatment of neuropsychological consequences of stroke

### *Cognitive Impairment*

There is considerable evidence of positive treatment effects for cognitive rehabilitation (Cicerone et al 2011). As much of this research is single case level or involves traumatic brain injury patients, stroke clinical guidelines generally conclude there is not yet sufficient evidence to fully support or refute cognitive rehabilitation, whilst recommending such strategies be implemented, tailored to patients' needs.

Clinical experience suggests cognitive rehabilitation can have a profound and positive impact in stroke. Direct retraining (e.g. Attention process training) and compensatory methods (e.g. logical safe place and electronic reminder) can be combined, depending upon assessment results, domain(s) of deficit, formulation and local resources. The target of cognitive rehabilitation must be to help patients achieve personally meaningful functional goals. It is important to keep in mind the key interplay between mood and cognitive factors. A goal for rehabilitation could be to improve confidence in ability to read for short periods of 5-10 minutes, even if no significant sustained attention or language deficit is revealed on assessment.

We argue stroke services should adopt the Cognitive Assessment and Rehabilitation Pathway for Stroke (CARPS). This encourages an inter-disciplinary team-based approach to cognitive rehabilitation of stroke (Taylor & Broomfield 2012).

### *Adjustment Distress*

Maintaining the mental health of NHS stroke patients is everybody's responsibility, not just psychology. When training colleagues, we emphasise staff take the time to listen to patients, to help them talk through their feelings. Often, this is all that is required.

At Glasgow, we have developed in patient information groups: Stroke Education and Discussion (SEED). Patients (7 to 10 stroke survivors) attend weekly psychology led sessions: What is Stroke?; Coming to terms with stroke; Coping with stress after stroke; Life after hospital- Changing roles & activities. Sessions have structured content but allow discussion. These are popular and appear effective to enhance well being and confidence to cope. Participants report provision of information and the social nature allowing group reflection, to be beneficial. A formal evaluation is planned.

Research is lacking, however psycho-education to 'normalise' distress can be greatly therapeutic, and confers hope. Patients begin to understand why they feel e.g. low, that this is normal, and that mood symptoms should settle with time. Presenting a 'stages of grief' model during formulation helps. Whilst not how psychologists exactly conceptualise stroke adjustment (Taylor et al, 2012), such simple (not simplistic) formulations are more easily comprehended. It is important to build and maintain a good therapeutic relationship allowing patients a 'safe place' to process what has happened and adjust. Self-coping should be fostered. Addressing life-trajectory (new post stroke roles and relationships), maintaining activity levels and disputing unhelpful beliefs will assist. Often, adjusting stroke patients only need to be seen once or twice.

### *Post-Stroke Depression*

Depressed stroke survivors vary along a continuum of cognitive/communication ability. This can help determine how to adapt Cognitive Behavioural Therapy (CBT) for PSD. For patients low on cognition communication ability, behavioural therapy for PSD will be more suitable. The level of abstraction of disputation in cognitive therapy, where this is

possible, will be informed by cognitive level. Carer involvement becomes more likely as disability increases as does the need to incorporate externally supported cognitive/communication rehabilitation. The converse holds for PSD patients high on cognitive/communication ability: traditional CBT with more abstract cognitive restructuring/disputation, self-directed cognitive rehabilitation and less carer involvement is possible (Kneebone, 2012).

Recent research supports effectiveness of Behavioural Therapy for people with low mood and aphasia following stroke (Thomas et al, 2013). For PSD patients with intact communication, there is not yet evidence from randomised controlled trials to support standard CBT. We find if standard CBT is individually tailored using additional therapy components, there is benefit on mood. Baseline distortions are a helpful psycho educational tool. Translating problems into goals can be supported using Selection Optimization Compensation. The grief model is a key cognitive aspect of treatment. Resilience timelines can be employed mid-therapy to help people understand how to manage challenging situations and use prior-learning. By augmenting CBT in this way there is a better clinical fit, as the CBT takes explicit account of the trauma, acute onset and loss elements of stroke, including the physical and psychological consequences (Broomfield et al, 2011). Augmented CBT (aCBT) for stroke has not been formally evaluated. We are currently writing an aCBT stroke therapy manual.

There is evidence anti-depressants are effective for treatment of PSD, although careful monitoring is required given side effects risk (Hackett et al, 2008).

#### *Post-Stroke Anxiety*

Evidence to support psychological intervention of PSA is scarce (Campbell Burton et al, 2011). A recent trial of group based autogenic relaxation was positive with respect to reducing self-reported tension amongst stroke inpatients (Kneebone et al, 2013) and individual minimal therapist assistance CD supported relaxation shows promise (Golding et al, 2013) At Glasgow we are evaluating effect of home based autogenic relaxation for community stroke survivors.

Relaxation is low risk, and low cost, and can easily be delivered to PSA patients by trained non-psychologists. Autogenic (imaginal) methods suit best, as patients with hemiplegia, sensory disturbance and pain can still participate. Giving out a relaxation script helps maintain regular practice, which is key. Patients may record pre/post tension scores on a log as a reminder to practice, and to test for treatment effects.

Many stroke survivors fear stroke recurrence. Careful discussion of realistic risk is needed, emphasising the importance of secondary prevention including lifestyle factors. Increased self-reassurance and reversal of fear avoidance are important, and anxious patients should be helped to accept an inevitable degree of uncertainty regarding their health.

There are no proven pharmacological treatments for PSA (Campbell Burton et al, 2011).

#### *Post-Stroke Emotionalism*

There are no evidence based psychological treatments for PSE. Textbooks recommend relaxation, deep breathing, distraction and mental imagery although no trials have been attempted (House *et al* 2008).

Psycho education on PSE for the patient and family appears clinically useful. It includes reassurance the difficulty usually settles down over time, though the sparse longitudinal data suggests for 10% of PSE sufferers this is not the case at one year (House et al 2008). It is easy to assume all patients with PSE are distressed although some are not. Conversely PSE can overlap with PSD so additional attention to this may be required for

some. Antidepressants in low dose for PSE reduce emotional outburst frequency and severity (House et al, 2008). An effective psychological PSE treatment, once developed, would represent an important alternative or adjunct therapy.

### *Conclusions*

Permanent physical communication and cognition deficits arise suddenly following stroke, bringing profound role changes for the patient and family. Psychological adjustment is challenging. Distress is normal. Adjusting takes time. It is important patients understand this. Raising activity levels, encouraging self-coping and addressing post-stroke life trajectory and (unhelpful) beliefs using cognitive behavioural methods will assist. Because of the complex physical, communication cognitive and mood sequelae of stroke, careful assessment and formulation is needed. Impact of symptoms on functional outcome is important to determine and patients should be supported to achieve personally meaningful functional goals. Whilst research in stroke neuropsychology remains scarce, there are treatment methods which once augmented for stroke, can show good effect. Their correct application should ensure patient rehabilitation is not impacted and that patient mood, cognition, independence, recovery and quality of life is maximised.

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