Post stroke depression

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Depression following stroke is common. Although it is highlighted as an issue in stroke guidelines, guidance on diagnosis or management is not given. This paper presents the original research from a literature review of Medline and the Cochrane Database on stroke and depression, and discusses some of the clinical implications of the findings.

Mood disturbance following stroke can take several forms. This review will mainly discuss depression; covering its incidence, clinical features, associations and therapeutic options. The review also briefly considers emotional lability, which is also relatively common and can be mistaken for depression. Adjustment reactions, abnormal illness behaviour, adoption of the 'sick role' or institutionalization and anxiety disorder can also occur after stroke but these are not specifically dealt with here.

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CLINICAL SIGNIFICANCE

Post stroke depression (PSD) can present as an obstacle to rehabilitation. It has an independent negative effect on longer term recovery of physical function, cognition (Morris et al, 1992) and social function (Clark and Smith, 1998). Patients who are depressed tend to stay in hospital longer. There is also evidence of a significant relationship between depressive symptoms and stroke mortality (Everson et al, 1998), even after adjustments for age, sex, race and risk factors. Although in 60% of patients the depression will resolve at 12 months irrespective of treatment, those in whom it has not will have a high risk of chronic depression (Astrom et al, 1993). One study has examined the incidence of suicide following stroke and reported a significantly increased risk, especially in females and in those less than 60 years old (Stenager et al, 1998). It might be argued that formal assessment of suicide risk in any patient with depressive symptoms should be included in all stroke management protocols.

TABLE 1.
Suggested risk factors for post stroke depression

No proven association	Increased risk	Odds ratio
Age of patient	Female patients	n/a
Volume of the lesion	Past history depressive illness	2.3–2.9
Side of the stroke	Increased impairment	n/a
Pathological type of stroke	Increased disability	1.8–2.9
Aetiology of stroke	Increased handicap	n/a
Location of lesion	Patients with dysphasia	n/a
	Patients living alone	n/a
	Patients with little social contact outside the home	n/a
	Cerebral atrophy on scan increases risk of depression at 3 years	n/a

n/a = data not available

INCIDENCE

Several studies have examined the incidence of depression following stroke with different results. Most studies excluded patients with dysphasia and patients with moderate or severe cognitive impairments, although one group of researchers have developed a questionnaire for carers as a proxy for patients with dysphasia (Sutcliffe and Lincoln, 1998). Incidence rates between 15 and 55% have been reported (Morris et al, 1992; Astrom et al, 1993; Burvill et al, 1995; Herrmann et al, 1998; Kotila et al, 1998; Pohjasvaara et al, 1998). Reasons for this wide variation may lie in differences in the diagnostic tools used, in the timing of the assessment following stroke and in patient selection. The incidence of depression is reported higher in hospital (22–55%) than in community studies (11–25%).

CLINICAL ASSOCIATIONS

Several studies have examined risk factors for the development of depression following stroke (*Table 1*). Groups of patients at higher risk have been identified, and perhaps increased awareness will allow earlier diagnosis and treatment.

AETIOLOGY

The aetiology of depressive illness in general is the subject of much research; it is evident that genetics, personality traits and the environment

all contribute. The monoamine neurotransmitters noradrenaline, serotonin and dopamine and their receptors have been widely studied and indirect evidence from the response of pharmacological agents acting on these pathways suggest they have a role in the aetiology of depressive illness (Gelder et al, 1996).

The specific aetiology of PSD has also been studied. It has been postulated that the psychological reaction to disability and social handicap has a role. Depression occurs more than twice as often following stroke than in age and sex matched controls (Wade et al, 1987; Burvill et al, 1995) and is more common following stroke than in other populations matched for physical disability; Folstein et al (1977) found that stroke patients were significantly more depressed than orthopaedic patients with equal levels of disability. Therefore it seems unlikely that adjustment issues are the only cause, but it does not exclude the possibility that there may be specific adjustment issues in stroke.

There is no consistent evidence of any relationship to lesion location. Abnormalities in serotonin pathways have been suggested as causal factors. Lower levels of the serotonin metabolite 5-hydroxy indoleacetic acid (5HIAA) have been found in the cerebrospinal fluid of depressed compared to non-depressed stroke patients (Bryer et al, 1992). Patients following stroke have reduced serotoninergic responsiveness (prolactin response to d fenluramine) when compared with controls (Ramasubbu et al, 1998). More work is required in order to improve our understanding of PSD; further understanding of the biochemical abnormalities may allow targeted pharmacological therapies to be developed.

CLINICAL ASSESSMENT

The common symptoms and signs of depressive disorder in general are shown in *Tables 2* and *3* (Gelder et al, 1996).

From *Tables 2* and *3* the difficulty in the diagnosis of depression following stroke will be apparent: facial weakness can look similar to an unhappy expression, movement and function may be reduced, impairments of speech and language cause difficulty in assessing thoughts, cognitive impairments may be a direct consequence of the stroke lesion, particularly the apathy that can occur following frontal lesions. The atypical ward environment can confound the assessment: eating habits are changed, sleep disturbance is common and patients may be adjusting to their new abilities or simply be bored. This difficulty in clinical diagnosis has led some to explore the use of formal assessment tools to assist in the diagnosis of depression following stroke.

ASSESSMENT TOOLS

The 'gold standard' in the diagnosis of general depression is the Present State Examination. This requires training to administer and is time consuming. Other scales have been developed to help in the diagnosis of depression. These tools are not diagnostic — most are designed to screen for high-risk patients who warrant further assessment. Some have been developed for use in physically ill patients but few have been validated in stroke.

Assessment tools in stroke must allow for the physical symptoms that may exist as a result of the stroke itself and must also allow for patients who may have language or cognitive impairments limiting the ability to complete the questionnaire.

Table 4 shows some of the tools currently used and quotes sensitivity, specificity, positive predictive value and misclassification rate for their use following stroke, where available.

There is no ideal assessment tool. The Hospital Anxiety and Depression Scale (HADS), General

TABLE 2.
Symptoms of depressive disorder

Low mood and feeling of misery	
Loss of interest and pleasure from activities	
Pessimistic thinking	Past
	Present
	Future: may lead to hopelessness and suicidal ideation
Biological features	Energy loss
	Sleep disturbance
	Diurnal variation of mood
	Anorexia and weight loss
	Loss of libido
Psychotic features	Delusions: guilt, worthlessness, nihilism
	Hallucinations: auditory, persecutory

TABLE 3.
Mental state examination

Appearance/ behaviour	Looks unhappy, may be tearful, avoid eye contact
	May appear neglected
	Psychomotor retardation, slumped posture
	Agitation, irritability, sleep disturbance or reaction to perceptual disturbance may be observed
Speech	Slow, soft, lacks expression
Mood	Low mood. Flat affect with lack of reactivity
Thoughts	Depressive themes
	May include suicidal ideation or delusions
Cognition	Impairments on formal testing, particularly concentration

Depression Scale 15 and General Health Questionnaire are probably most commonly used. The General Depression Scale 15 has been recommended by the Royal College of Physicians as a useful screening tool for depression in elderly patients. Although it has been used in medical rehabilitation settings, there are, as yet, no data available on its use in stroke. Perhaps the HADS is the preferred tool, as it is short and relatively easy to complete: this is an important consideration if it is to be used as a screening tool although the thresholds may be better set at 6–7 in the stroke population. As there is no gold standard for diagnosis, treatment may have to be considered on the basis of these 'screening tools'. Further studies are required to validate threshold HADS scores in relation to specific treatments.

TREATMENT OPTIONS

Conservative management

Evidence from several studies indicates that there is a tendency for PSD to improve spontaneously over time. At 2–6 weeks following stroke, 50% of patients in one study improved independent of treatment, although spontaneous recovery after 7 weeks was infrequent (Andersen et al, 1994). Approximately 50% of patients in another study who were depressed at 3 weeks or 6 months were no longer depressed at 1 year (Wade et al, 1987); another study reported a slightly higher recovery rate of 60% at 1 year (Astrom et al, 1993). Despite evidence that spontaneous recovery occurs, conservative management may allow depression to needlessly hamper the rehabilitation process.

Rehabilitation process

The rehabilitation process itself may also bring about improvement by maximizing independence, increasing social stimulation and relieving pain and other unpleasant symptoms (Wade, 1992). One study showed a leisure rehabilitation programme had a beneficial effect on mood measured by the Nottingham Health Profile, although not when measured by the Wakefield Depression Inventory (Drummond and Walker, 1996).

Stroke family care worker

A randomized controlled trial (RCT) (Dennis et al, 1997) has examined the impact of a stroke family care worker on various outcomes following stroke. The worker had a social work background and was able to identify unmet needs, access health, social and voluntary services and provide a counselling role. The treatment group

TABLE 4.
Tools used for assessment post stroke

Assessment tool	References	Notes	Sens	Spec	PPV	MR
Hospital Anxiety and Depression Scale (HADS)	Zigmond and Snaith (1983), Abiodun (1994), Burvill et al (1995), Spinhoven et al (1997), O'Rourke et al (1998)	Validated in primary care and medical patients. Weighted to psychological symptoms. Gives probability of disorder				
		Cut off 3–4	94%	32%	25%	56%
		Cut off 4–5	83%	44%	26%	48%
		Cut off 6–7	83%	8%		
General Health Questionnaire (28) (GHQ)	Burvill et al (1995), Bridges and Goldberg (1986)	Screening tool for primary care and medical patients. Takes <10 minutes. expressed as judgment of psychiatrist as case or non case. Cut off 5–6	78%	81%	50%	20%
General Health Questionnaire (30)	O'Rourke et al (1998)	Cut off 8–9	80%	76%		
Montgomery Asberg Depression Rating Scale	Montgomery and Asberg (1979)	10 themes, 4 point scale for each. Interviewer has definitions. Psychological symptoms rated				
Beck Depression Inventory (BDI)	Beck et al (1961)	21-item patient completion scale. 4–6 statements per item. Cut off 9–10	92%	75%		
Geriatric Depression Scale (GDS 30)	Yesavage et al (1982–83), Burvill et al (1995)	30-item scale, cut-off 10–11. Simple to administer, no trained interview skills required	84%	66%	53%	28%
15 item Geriatric Depression Scale (GDS 15)	Lesher and Berryhill (1994)	15-item scale — shorter form of GDS 30, validated reliability compared to GDS 30. Not studied in stroke				
Visual analogue scales	Davies et al (1975)	Mark along 10 cm line as marker of mood				
Stroke Aphasia Depression questionnaire	Sutcliffe and Lincoln (1998)	Carer completes as proxy for dysphasic patients. Correlates with HADS. 21 items+ validated a 10-item version. Detects mood, not syndrome or prediction of treatment response				

Sens=sensitivity, spec=specificity, PPV=positive predictive value. MR=misclassification rate

expressed significantly greater satisfaction than the control group in certain aspects of their care. However, there were no differences in physical or mood-related outcomes in patients or carers.

Cognitive behavioural therapy

Cognitive behavioural therapy is of proven benefit in depressive illness. Its effect in PSD has been studied using Beck Depression Inventory and HADS (Lincoln et al, 1997). Severe strokes were excluded. The Beck Depression Inventory suggested a significant benefit although the HADS or activities of daily living score did not. The study was not controlled. Further controlled studies are required to identify which patients are able to engage in cognitive behavioural therapy and are most likely to benefit.

Drug treatments

The treatments for depression following stroke are similar to those for other depressive illnesses.

Tricyclic antidepressants: These inhibit the reuptake of noradrenaline and serotonin. They are inexpensive, well studied and have no proven long-term toxicity. Side-effects are common and often limit therapeutic treatment (*Table 5*).

A double-blind placebo-controlled RCT (Lipsey et al, 1984) has compared nortriptyline to placebo in 34 patients with PSD measured by the Hamilton Depression Scale (HDS), Zung Depression Score (ZDS) and Present State Examination. There was a significant beneficial effect of treatment by 3 weeks, which persisted at 6 weeks. Dropout rates were comparable.

Selective serotonin-reuptake inhibitors: Selective serotonin-reuptake inhibitors (SSRIs) are attractive because a therapeutic level is soon reached. As they are a relatively recent development their long-term effects are unknown. They have a less serious side-effect profile but gastrointestinal side-effects can be troublesome and they have more potential drug interactions (*Table 6*).

The efficacy and safety of citalopram has been tested in PSD using a double-blind, placebo-controlled RCT (Andersen et al, 1994). Thirty-three patients were in each arm, with a diagnosis of depression using the HDS. Benefits were seen in the treatment group at 3 and 6 weeks, although their dropout rate was greater as a result of side-effects. (Of those entering in the study 2–6 weeks following their stroke, half recovered within 1 month, independent of treatment. In those entering more than 7 weeks after the stroke, recovery was infrequent in the placebo group.)

Atypical antidepressants: Reding et al (1986) studied the effect of trazodone *vs* placebo in a 27-patient RCT, involving patients who were seen an average of 44 days after they had had a stroke. There was no difference in dropout rate between the groups. In patients with a clinical diagnosis of PSD or abnormal ZDSs there was a tendency for Barthel scores to improve in the treatment group, although this did not reach statistical significance. There was no difference in side-effect rate.

Prophylactic mianserin had no beneficial effect on mood or function in a randomized, placebo-controlled study of 100 consecutive patients following stroke (Palomaki et al, 1999). Venlafaxine has been shown to be safe and tolerable in a small uncontrolled study by Dahmen et al (1999); 10 out of the 12 patients had a >50% reduction in HADS score following treatment. Evidence for a therapeutic effect must await a controlled study.

Stimulant drugs: Stimulants have also been studied in PSD. In a 21-patient RCT, Grade et al (1998) showed that methylphenidate was beneficial in improving HDS, ZDS and the Functional Independence Measure. Methylphenidate has also been shown to be as effective as nortriptyline in improving depressive symptoms, with a signifi-

TABLE 5.
Side-effects and drug interactions of tricyclic antidepressants

Side-effects	Anticholinergic	Dry mouth, tachycardia, blurred vision, urinary retention, constipation, erectile dysfunction, cognitive impairment
	Alpha 1R block	Drowsiness, postural hypertension, cognitive impairment
	Antihistamine	Drowsiness, weight gain
	Cardiotoxic	Conduction defects, arrhythmias
	Lowers seizure threshold	
	Risk in overdose	
Drug interactions	Increased risk arrhythmias with antiarrhythmic drugs	
	Anticholinergic/antihistamine effects increased by concomitant use	
	Hypotensive effect antihypertensives increased	

TABLE 6.
Side-effects and drug interactions of selective serotonin-reuptake inhibitors

Side-effects	Common	Gastrointestinal	Nausea, anorexia, constipation, diarrhoea
		Central nervous system	Headache, dizziness, insomnia, restlessness
	Other	Sweating, anorgasmia, ejaculatory delay	
	5HT syndrome	Especially if use lithium/$5HT_1$ agonists	
Drug interactions		May inhibit hepatic metabolism tricyclics, antipsychotics and anticonvulsants	
		Effect of warfarin may be increased	

5HT = 5-hydroxytryptamine

cantly earlier effect; 2–4 days compared to 27 days (Lazarus et al, 1994). An RCT found dextroamphetamine to be as effective as methylphenidate in improving mood (Masand et al, 1991).

Other considerations

Few data exist on optimal duration of therapy; this warrants further investigation. The three RCTs using antidepressant medication have outcomes at 6 weeks, although the optimal duration of therapy is likely to be longer if experience from depressive illness in general is relevant.

When considering pharmacological therapy it is important to be aware of the possible effects on recovery and plasticity, although there are few data from human trials. A study comparing the effects of fluoxetine and maprotiline following stroke found a difference in recovery between the two groups, suggesting fluoxetine may facilitate or maprotiline may hinder recovery in post stroke patients (Dam et al, 1996). Further studies suggest serotoninergic agents may have different effects on outcomes than noradrenergic agents, another area with potentially exciting new developments.

Electroconvulsive therapy

Electroconvulsive therapy can be indicated in severe depressive illness with biological or psychotic features. It is conceivable that stimulating seizure activity in subacutely injured brain may have different consequence from its use in the general depressive illness population. Two retrospective studies have reported beneficial treatment effects in 86% and 95% of stroke patients. The safety and tolerability differ between the studies; 1/14 adverse events occurred in the study by Murray et al (1986) and 12/20 suffered significant side effects in Currier et al's study in 1992 — seven relapsed on medical maintenance, five had medical complications (including hypertension, acute pulmonary oedema, multiple ventricular arrhythmias and severe interictal delirium — all in patients with pre-existing cardiovascular disease) and three developed confusion or amnesia. There are no prospective studies looking at the effect and safety of electroconvulsive therapy in PSD. The safety in PSD is therefore not established and an RCT looking at the therapeutic effect as well as safety and tolerability seems overdue.

EMOTIONAL LABILITY

Emotional lability following stroke can lead to difficulties in assessing mental state. The incidence is probably 15% following stroke (House et al, 1990; Andersen, 1995). It is characterized by the abrupt onset of weeping (or more rarely laughing), which is uncontrollable. There is often no obvious precipitant although it can be triggered by emotion or emotional content, for example on discussing home or seeing family members.

The symptoms themselves are usually distressing for the patient, family and also for ward staff. Emotional lability can also interfere with the rehabilitation process, can delay progress and affect social functioning and it is therefore important to diagnose and manage appropriately (Andersen, 1995). Scales have been suggested and validated for the measurement of lability (Robinson et al, 1993; Brown et al, 1998). The aetiology of emotional lability is not known although it does occur more often in patients with psychological symptoms and cognitive impairments. There is no proven correlation with lesion location although it is postulated that damage to serotonergic transmission is important.

Emotional lability tends to improve over time, and explanation to the patient and family may be all that is required. Several studies have demonstrated the effectiveness of tricyclic antidepressants and SSRIs (Schiffer et al, 1985; Sloan et al, 1992; Robinson et al, 1993; Andersen, 1995; Brown et al, 1998), often with a rapid response to treatment (within 1 week). No evidence exists on optimum duration of therapy: this is another issue requiring further well designed trials.

CONCLUSION

Depression following stroke is relatively common, perhaps affecting one in four hospitalized patients. Given that depression is known to correlate with poor outcome following stroke, all patients should have a screening assessment following stroke to identify the high-risk groups. However, definite diagnosis of depression following stroke is not straightforward. Ideally there should be a standard tool for use following stroke, but none are ideal. The HADS is well studied and easy to administer; it may be the most appropriate tool if it can be further validated with standard thresholds for use following stroke. It may be that a diagnosis of PSD can never be certain but treatment should be given on the likelihood of symptom response using a specified, validated assessment tool.

There is some evidence for effective treatments using nortryptiline and trazodone but no large studies using the current commonly prescribed drugs, e.g. fluoxetine, paroxetine and sertraline. Evidence suggests that treatment is of little benefit early (<6 weeks) after stroke. An initial monitoring and supportive role is appropriate, reserving medication for symptoms persisting after 6 weeks.

It is not yet known whether treatment reverses the poorer prognosis associated with PSD. This is another area worthy of further research although

is unlikely to change our recommendations for increased awareness and treatment of this common, distressing and disabling disorder. HM

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KEY POINTS

- Post stroke depression is an obstacle to rehabilitation. It has a negative effect on longer-term recovery of physical function, cognition and social function.
- Post stroke depression is common, with an incidence in hospitalized patients of 22–55%.
- Clinical assessment is complicated by physical, cognitive and environmental factors.
- There is no ideal assessment tool. The Hospital Anxiety and Depression Scale is short, relatively simple to use and has some data for useful thresholds.
- Evidence for effective treatments exist.
- Further research is required, particularly on assessment, diagnosis and treatment with commonly prescribed specific serotonin-reuptake inhibitors.