The association between communication impairments and acquired alexithymia in chronic stroke patients

Authors: Hannah Hobsona, b\*, Evangeline Grace Chiuc, Chloe Ravenscroftc, Kate Partridgec, Geoffrey Bird c, d, Nele Demeyerec

a Department of Psychology, University of York, York, YO10 5DD

b Department of Psychology Social Work & Counselling, University of Greenwich, Old Royal Naval College, Park Row, Greenwich, London SE10 9LS

c Department of Experimental Psychology, Anna Watts Building, Radcliffe Observatory Quarter, Woodstock Road, Oxford, OX2 6GG

d Social, Genetic and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology and Neuroscience, De Crespigny Park, Denmark Hill, London, SE5 8AF

**\*Corresponding author address, telephone and email:** Department of Psychology, University of York, York, YO10 5DD, Tel: 01904 323190, Email: [hannah.hobson@york.ac.uk](mailto:hannah.hobson@york.ac.uk)

Word count for abstract and manuscript text (excludes references and tables/figures): 5160

# Abstract

Introduction: Language dysfunction has recently been suggested to be one route to alexithymia, an impairment in recognising and communicating one’s own emotions. Neuropsychological evidence is needed to investigate the possibility that acquired language problems could underlie acquired alexithymia. Method: This project examined data from a large group of chronic stroke patients (*N* =118) to test whether self-reported or behavioural measures of language and communication problems were associated with alexithymia. We also examined the impact of hemisphere of damage on alexithymia. Results: We found no differences in alexithymia levels for patients with observed language impairments on brief tests of picture naming, comprehension and reading vs unimpaired patients. However, self-reported communication difficulties were found to be associated with higher scores of alexithymia, even after controlling for depression and anxiety. Patients with left versus right hemisphere damage did not differ in their alexithymia scores. Conclusions: We found partial support for the language hypothesis of alexithymia. We discuss potential reasons for the discrepant findings between the self-report and objective language measures, and suggest that self-report measures may be more sensitive to milder, more pragmatic language impairments, as opposed to the severe structural language impairments measured by the cognitive screening tests.

**Keywords:** Acquired brain injury; Language; Emotional awareness; Neuropsychology; Aphasia; Emotional impairments.

The association between communication impairments and acquired alexithymia in chronic stroke patients

Alexithymia is the term used to describe difficulties identifying and communicating one’s own emotions (Taylor, Bagby, & Parker, 1991). It is best considered a sub-clinical trait (it is not a clinical condition in its own right), and is elevated across a range of psychiatric populations, including autism, eating disorders and schizophrenia (Hill, Berthoz, & Frith, 2004; Schmidt, Jiwany, & Treasure, 1993; van ’t Wout, Aleman, Bermond, & Kahn, 2007). In addition to a higher prevalence of alexithymia in these and other clinical groups, alexithymia has also been linked to well-being and emotion regulation in the general population (Honkalampi, Hintikka, Tanskanen, Lehtonen, & Viinamäki, 2000; Pandey, Saxena, & Dubey, 2011; Saxena, Dubey, & Pandey, 2011). Alexithymia has thus been considered a trans-diagnostic risk factor for poor emotional functioning (Valdespino, Antezana, Ghane, & Richey, 2017).

Given its predictive power for these outcomes, researchers have sought to understand the aetiology of alexithymia. The precise cognitive factors giving rise to alexithymia are a topic of intense scrutiny (Brewer, Cook, & Bird, 2016; Hobson, Brewer, Catmur, & Bird, 2019). However, alexithymia represents dysfunction in what might be considered a set of higher-level skills; the abilities to recognise, discriminate, label and communicate one’s emotional states. These skills would presumably rely on a variety of perceptual, cognitive and affective processes, and we might expect there to be different cognitive routes to alexithymia, whereby different deficits in processes required for effective emotional processing manifest in a similar alexithymic phenotype. Thus, impairments in any one of a number of cognitive processes may underlie alexithymia. It has been proposed that one such cognitive deficit that could result in alexithymia is language impairment (Hobson et al., 2019; Hobson et al., 2018).

As described in Hobson et al (2019), one framework for investigating the nature of the association between language and alexithymia is to seek evidence from both developmental groups (such as developmental language disorder, or children born with hearing impairments) and populations with late-acquired difficulties (for example, patients who have developed aphasia following a traumatic brain injury or stroke). Bringing together findings from these two separate groups has been fruitful when attempting to understand the cognitive and neurobiological systems underpinning language (see Bishop, Nation, & Patterson, 2014). Indeed, combining evidence from groups with developmental language problems and groups with acquired language problems will shed light on a) whether language processes play a developmental role in alexithymia, and b) whether language processes are continuously required for emotion processing.

If the role of language in emotion processing is limited to a developmental role, then late-acquired language problems – such as those acquired after brain injury – should leave emotion processes untouched. In this vein, studies of patients with Traumatic Brain Injuries (TBI) have suggested a link between language processes and alexithymia that continues aftr development. Verbal abilities are correlated with alexithymia traits in TBI patients (Henry, Phillips, Crawford, Theodorou, & Summers, 2006), and patients with and without alexithymia differ significantly in their verbal ability scores (Wood & Williams, 2007). More recent investigations of TBI have found that acquired naming difficulties, and damage to regions of the inferior frontal gyrus considered to support language, are associated with alexithymia (Hobson et al., 2018).

Another population that could provide useful insight into the role of acquired language problems in alexithymia is stroke patients. Studying alexithymia in stroke could provide important insights into the neurobiological underpinnings of emotional deficits (see Ricciardi, Demartini, Fotopoulou, & Edwards, 2015), but such research could also be used to support better emotional outcomes in stroke survivors. Alexithymia has been suggested as an explanation for the socio-cognitive and socio-affective problems reported in stroke patients (Nijsse, Spikman, Visser-Meily, de Kort, & van Heugten, 2019). Alexithymia may also have a role in a range of different mental health problems that frequently onset after stroke: alexithymia is a risk factor for post-stroke depression (Spalletta et al., 2001; Spalletta, Ripa, Bria, Caltagirone, & Robinson, 2006; Su, 2016), as also appears to be the case in short-term post-stroke PTSD (Wang, Chung, Hyland, & Bahkeit, 2011). Anxiety is a common mental health problem following stroke (Barker-Collo, 2007); as yet, it is unclear whether alexithymia predicts post-stroke anxiety as it does depression, but anxiety is predicted by alexithymia in other populations (e.g. Eizaguirre, de Cabezon, de Alda, Olariaga, & Juaniz, 2004).

What factors appear to predict alexithymia in stroke survivors? Thus far, data have suggested that damage to the right hemisphere is more strongly associated with acquired alexithymia than left hemisphere damage (Spalletta et al., 2001, 2006). Spalletta et al (2006) compared alexithymic versus non-alexithymic stroke patients, and found that the alexithymic group were significantly more likely to have right hemisphere damage than the non-alexithymic group. An earlier paper (Spalletta et al., 2001) compared patients with right versus left hemisphere damage following stroke on their average alexithymia score, and categorization as non-alexithymic, borderline alexithymic or alexithymic (using conventional cut-offs). They reported that while 48% of right hemisphere damage patients were alexithymic, only 22% of patients with left hemisphere damage were alexithymic. However, these relative percentages reflect sample sizes of just 10 and 6 patients (of their total sample of 48 patients) respectively. Notwithstanding this issue, average continuous alexithymia score was significantly higher in the right hemisphere damaged group.

Together, these findings have been interpreted as supporting the theory that the right hemisphere is dominant for emotional processes (see Borod et al., 2010 for a review of evidence from patients with unilateral brain damage). Nonetheless, the left hemisphere appears to play a role in at least some elements of emotion processing; a meta-analysis of fMRI studies examining processing of emotional faces concluded that both hemispheres are activated during these tasks (Fusar-Poli et al., 2009). Indeed, given that the left hemisphere is typically dominant for language processes, the language hypothesis of alexithymia put forward by Hobson et al (2018) would predict that left hemisphere damage, that disrupts language processes, should increase alexithymia.

In addition to region of damage, it is pertinent to consider how behavioural language problems pattern with alexithymia in stroke. Aphasia occurs in approximately one third of acute stroke cases (Engelter et al., 2006). In previous studies (e.g. Spalletta et al., 2001, 2006) individuals with aphasia were excluded, a decision that may also have decreased evidence of an association between left hemisphere damage and alexithymia, as patients with acquired language problems due to damage to left-hemisphere language areas would have been excluded.

If communication problems were a contributing factor to alexithymia and therefore poorer mental health outcomes, we would arguably expect an association between acquired communication problems and depression in stroke populations. It had previously been argued that stroke patients with left hemisphere damage were at greater risk of depression (Robinson & Price, 1982), however a systematic review of depression in stroke patients did not find evidence of higher rates of depression in patients with left versus right lesions following stroke in hospital studies (Carson et al., 2000). They did note a higher incidence in patients with right hemisphere lesions in community studies, but argued that such studies excluded aphasic patients, likely confounding the association between hemisphere of damage and depression.

The present study seeks to test the hypothesis that acquired language problems are associated with alexithymia, extending previous neuropsychological evidence from patients with TBI to consider evidence from patients who have had a stroke. This project was also the first to examine alexithymia in stroke in a representative sample of stroke patients, in which individuals with acquired language problems were not excluded, and to include both behavioural and self-report measures of acquired language and communication problems in relation to alexithymia. We also examined whether we could replicate previous reports of an association between alexithymia and right hemisphere damage, without the exclusion of patients with aphasia.

# Method

## Participants

125 participants were recruited from a cohort of chronic stroke research volunteers at the Translational Neuropsychology Group. Patients were originally recruited through the Oxford Cognitive Screening programme, in which patients from an acute stroke unit are screened at presentation, and followed up after 6 months with a home visit (Demeyere, Riddoch, Slavkova, Bickerton, & Humphreys, 2015; OCS-Tablet and OCS recovery studies, NHS REC reference 14/LO/0648 and 18/SC/0550 respectively). All patients were thus at least 6 months post-stroke (see Table 1 for average time since stroke at the point at which self-report questionnaires were administered). These participants had provided consent to be contacted about research, and provided study specific informed consent to participate in this study (Medical Sciences Interdivisional Research Ethics Committee, reference R59378/RE001).

3 participants did not complete the alexithymia questionnaire (described below), and were thus excluded from the analyses. Furthermore, 4 participants were determined to have had a Transient Ischemic Attack, and were thus excluded, leaving a final sample of 118, comprising of 55 females and 63 males. The mean age of patients was 73.14 years (SD = 11.37). Information regarding type of stroke and lesion site were available from medical records and confirmed by clinical CT scan at acute stage. The type of stroke for the sample (*N* = 118) were: 95 Ischemia and 23 Haemorrhage; lesion lateralisation: 57 unilateral right hemisphere, 51 unilateral left hemisphere, and 10 bilateral. For the analysis of lesion laterality on alexithymia scores, participants with no clear lateralization of damage [bilateral lesions, subcortical lesions (e.g. midbrain or cerebellar lesions), or with no visible lesions on the CT scans] were excluded. Lesion analyses are the subject of a separate investigation and will be reported separately. Table 1 summarises the characteristics of the reported sample.

[Table 1]

## Procedure

Observed language and self-reported communication scores were collected during the 6-month post stroke follow up home visits, as part of the standard Oxford Cognitive Screening programme. For this specific project, self-report questionnaires were also collected for alexithymia, depression and anxiety. Patients were given the choice of completing these questionnaires via the telephone (N = 62), during the OCS Recovery home visits (N = 52) or questionnaires returned via the post (N = 4). Collection of the self-report data was overseen by a trained speech and language therapist (EGC) to ensure patients could sufficiently comprehend the questions and that responses were reliable. Reliability analyses were conducted for each self-report measure, both for the full sample and for those with language impairment only.

## Measures

Behavioural data on language impairment was collected via the OCS (Oxford Cognitive Screen; Demeyere et al., 2015, 2016) or the BCoS (Birmingham Cognitive Screen; Humphreys et al 2012). Depression and anxiety are typically highly correlated with alexithymia (Eizaguirre, de Cabezon, de Alda, Olariaga, & Juaniz, 2004; Marchesi, Brusamonti, & Maggini, 2000), and therefore including them is important to ensure that associations between language impairment and alexithymia are not the result of shared association with depression or anxiety; thus, in addition to questionnaire measures of alexithymia and communication problems, we also collected measures of depression and anxiety. All measures are described below. Behavioural measures are described first, followed by questionnaire measures.

### Oxford Cognitive Screen/Birmingham Cognitive Screen

Patients were seen from 6-months post stroke, and assessed using either the Oxford Cognitive Screen (OCS: Demeyere et al, 2015) or the Birmingham Cognitive Screen (BCoS: Humphreys, Bickerton, Samson, & Riddoch, 2012). In the present study, data from the picture naming, spoken word comprehension, and sentence reading tasks were examined. For both the OCS and BCoS, in the picture naming task, patients were presented with greyscale drawings and asked to name the object in the picture. For both batteries, the sentence reading task required patients to read a sentence aloud. In the OCS, in the comprehension task, patients are presented with simultaneous pictures and asked to point to an item (e.g. “Can you point to the fruit for me?”). In the BCOS, the comprehension measure is based on how many times instructions needed to be repeated for 4 target tasks (personal orientation, orientation in time and space, anosognosia and rule-finding). The level of understanding was then judged as follows on a 3 point scale as follows: 1=poor understanding even after repetition, 2=relatively good understanding but instructions need often to be repeated, 3=good understanding, almost no need to repeat the instructions.

There was limited variability in the scores on the observed language assessments, given the nature of the cognitive screening tools with few items, the time of testing post stroke, and high rates of recovery and improvement for all aphasia types within the period of 6 months following stroke (e.g. (Pedersen, Jorgensen, Nakayama, Raaschou, & Olsen, 1996, or Demeurisse et al., 1980); indeed no more than 15% of the patients assessed showed impairments on any of the individual behavioural tasks, which is in keeping with the good recovery many patients show from initial language problems (Laska, Hellblom, Murray, Kahan, & Von Arbin, 2001). Participants were categorised into either impaired/not impaired on the language tasks, using the standard thresholds for the OCS/BCoS. We opted to denote these patients’ problems as “language impaired” rather than “aphasia”, given that grouping was done on the basis of this cognitive screen rather than a full neuropsychological examination.

### Toronto Alexithymia Scale

To measure alexithymia, the Toronto Alexithymia Scale (TAS-20) (Parker, Bagby, Taylor, Endler, & Schmitz, 1993) was used. This is a 20-item scale, including three sub-scales examining difficulties identifying feelings, expressing feelings and externally orientated thinking. Participants responded using a 5-point Likert scale. Higher scores indicate greater alexithymia. Cronbach’s alpha for this measure for the current sample was .79, and thus this measure can be considered to have fair internal reliability. Considering the reliability of responses for patients in our language-impaired group, Cronbach’s alpha was .77, and thus was still reliable for patients with communication difficulties. As per previous investigations (Spalletta et al., 2001), the rate of patients surpassing the threshold for high alexithymic traits was elevated with respect to the general population; 22% of the whole sample were at or above the criterion for high alexithymia traits (10% of the general population are usually expected to meet this threshold; Franz et al., 2008).

### Stroke Impact Scale

The communication section of the Stroke Impact Scale (Version 3.0) (Duncan, Bode, Min Lai, & Perera, 2003) (SISCOM) was used as a self-report measure of communication difficulty following stroke. The SISCOM includes 7 questions requiring participants to rate their difficulties in the last week on a 5-point scale. Higher scores indicate greater self-reported ability. Cronbach’s alpha for the SISCOM for the current sample was .84, and thus this measure can be considered to have good internal reliability. The reliability for the patients with language difficulties on the SISCOM was .86; thus, reliability was still good for patients with communication problems.

It was considered possible that a significant association between alexithymia and the SISCOM could simply be a reflection of general poor self-esteem/low belief in one’s abilities (i.e. there would be an association between reporting one had poor emotional insight and poor communicative abilities because of a belief that one was poor at everything). The mobility subscale (SISMOB) of the Stroke Impact Scale was thus also included in analyses to test this interpretation. This subscale was deliberately selected as the construct of mobility problems seemed unlikely to overlap with communication or emotion, beyond poor self-esteem. This subscale has 9 items, answered on a 5-point scale. For the current sample, Cronbach’s alpha for the SISMOB was .94, indicating high internal reliability.

### Hospital Anxiety and Depression Scale

Hospital Anxiety and Depression Scale (HADS: Zigmond & Snaith, 1983) is a 14-item questionnaire, with 7 items assessing anxiety symptoms, and 7 assessing depressive symptoms, on a 4 point scale. Higher scores indicate greater anxiety/depression symptomatology. Previous research has shown these measures to be appropriate for use with stroke patients (Ayis, Ayerbe, Ashworth, & Wolfe, 2018). In the current sample, Cronbach’s alpha was .74 for the anxiety scale, and .77 for the depression scale. For the sample of patients with language problems, reliability was .75 and .75, for anxiety and depression respectively. Thus, across the whole sample and for those patients with language problems, the HADS subscales had fair internal reliability.

# Results

Analyses of alexithymia and self-reported communication problems via the SISCOM are reported first. Alexithymia scores were then compared between the impaired/not impaired groups, as defined by patients’ performance on the behavioural OCS tasks. Because comparisons of raw alexithymia scores would leave the possibility that differences could be confounded by the effect of increased depression/anxiety in the language-impaired groups, these group comparisons were also run on alexithymia scores that had been corrected for depression and anxiety statistically; specifically analyses were conducted on residual scores from a regression predicting alexithymia scores from depression and anxiety (see Hogeveen, Bird, Chau, Krueger, & Grafman, 2016 and Hobson et al., 2018). As reported below, the self-reported communication and behaviourally observed language problems showed discrepant relationships with alexithymia. We therefore also compared the self-reported communication problems between the impaired/not impaired groups to understand the relationship between these two modes of measurement of communication problems. Finally, we examined whether we replicated previously reported associations between alexithymia and lesions in the right hemisphere, and considered potential interactions with stroke type.

## Self-reported communication impairments and alexithymia

The degree to which self-reported communication problems (measured via the SISCOM) predict alexithymia (measured by the TAS-20) was examined using standard multiple regression. Anxiety (HADS-A) and depression (HADS-D) symptoms were also entered as predictors, along with gender, age at stroke, years in education and time since stroke (in months). While depression, anxiety and alexithymia are commonly highly correlated, all Variance Inflation Factors (VIFs) were below 2, indicating minimal problems with multicollinearity.

Overall, the model was significant: *F* (7, 102) = 8.78, *p* <.001, *R*2 = .38. Table 2 summarises the results for the individual predictors. Significant predictors included SISCOM (β = -0.24, *p* =0.01), anxiety (β = 0.22, *p* =0.03), depression (β = 0.20, *p* =0.05) and education (β = -.21, *p* = .02).

[Table 2]

In order to consider whether this significant effect could be due to general feelings of impairment/low self-esteem, a further separate standard multiple regression was run with the addition of the SISMOB. We also included modality of questionnaire collection (home versus phone, as only 4 participants returned data via post) to check whether this could have impacted the results. The model remained significant overall: *F* (9, 108) = 7.45, *p* <.001, *R2* = .40. Significant individual predictors included: SISCOM (β = -0.21, *p* =0.03), education (β = -.21, *p* = .01), modality of questionnaire collection (β = 0.24, *p* =0.04), and anxiety (β = 0.22, *p* =0.03). For a full list of standardised Betas, significance and squared semi-partial coefficients for this regression, see Table S.1, in the Supplementary Materials. VIFs for time since stroke and modality of response were 2.13 and 2.08 respectively; this reflected the fact that those assessed over the phone had longer periods between their stroke events and the data being collected. All other VIFs were below 2.

## Observed language impairments and alexithymia

OCS/BCoS data were available for 116 patients, of which 11.0% were impaired on picture naming, 2.5% were impaired on spoken word comprehension, and 15.3% were impaired on sentence reading (1 additional patient’s data was not available for the sentence reading task). A participant was considered to have language impairment if they fell below the cut off for one or more of these tasks, leading to 31 patients being classed as having a language impairment, and 84 being classed as having typical language (within the normative ranges). These groups were compared on the total TAS z-scores (after controlling for depression and anxiety) and raw TAS total via independent samples t-tests. These comparison were not significant for either TAS z-scores: *t* (111) = .04, *p* = .48 (one-tailed), *d* = .001 (See Figure 1), TAS raw scores: *t* (113) = -1.34, *p* = 0.09 (one-tailed), *d* = .28.

[Figure 1]

## Agreement between self-reported and behavioural language measures

Given the discrepant findings regarding alexithymia for the self-reported communication impairments and the behavioural language impairments, we also compared the impaired/not impaired groups on their SISCOM scores. Given the high number of non-language impaired participants reporting no problems, the data were not normally distributed, and thus the non-parametric Mann-Whitney test was used. There was a significant difference between these groups: *U* = 870.50, *Z* = 2.40, *p* = 0.02 (see Figure 2).The language-impaired participants reported worse communication skills (*Mdn* = 30.50, IQR = 10.75 *N* = 30) than the non-impaired participants (*Mdn* = 34.00 , IQR = 5.00, *N* = 82), with a small effect size (*r* = 0.23).

[Figure 2]

## Alexithymia in right versus left hemisphere damage

For patients with clear lateralisation of stroke damage, we compared patients with right versus left hemisphere damage on their alexithymia scores. Patients with right hemisphere damage (*N* =57, Mean TAS score = 48.30, SD = 13.94) were not significantly higher in alexithymia scores than those with left hemisphere damage (*N* = 51, Mean TAS score = 51.45, SD = 11.40): *t* (105.18) = -1.29, *p* = .20 (two-tailed), *d* = .25. When this was examined with only patients who were not language-impaired, there was still not a significant difference between patients with right hemisphere damage (*N*=37, Mean TAS Score= 46.57, SD = 12.38) and left hemisphere damage (*N*=28, Mean TAS Score = 51.14, SD = 11.21); *t* (63) = -1.46, *p* = .15 (two-tailed), *d* =.17.

Potential effects of stroke type (haemorrhage vs. ischemic) and interactions between stroke type and hemisphere of damage were explored: there were no significant differences between haemorrhage and ischemic stroke patients in terms of their SISCOM (*p* = .23, two-tailed, *d* = .31) or alexithymia scores (*p* = .74, two-tailed, *d* =.08), nor any significant interactions between these factors on SISCOM or alexithymia scores (TAS: *p* =.63, Partial Eta Squared = .008; SISCOM: *p* =.96, Partial Eta Squared = .001).

# Discussion

This study aimed to examine the role of language impairment in acquired alexithymia, in a large group of stroke patients. While previous studies of alexithymia have excluded stroke patients with language impairments, this project assessed a representative sample of chronic stroke patients, not selected based on lesion side or any particular cognitive impairment. Different findings arose from the self-reported versus observed language measures: while self-reported communication impairments were predictive of alexithymia, alexithymia did not differ between language-impaired versus not impaired stroke patients, as determined by a stroke-specific cognitive screen. Fewer years in education also predicted alexithymia, an association that has been reported previously in other samples (Lane, Sechrest, & Riedel, 1998). Participants assessed at home were also found to be more alexithymic than those who reported their alexithymic traits over the phone. The findings thus only partly support the language-hypothesis of alexithymia put forward by Hobson et al (2019). Here, we discuss potential explanations, including what different aspects of communication and language the behavioural versus self-report measures may represent.

The difference in associations with alexithymia between the two communication measures (self-report versus behavioural tests) could reflect a number of things. First, it could be the case that the cognitive screening subtests, relative to the self-report measures, are less sensitive. Subtle language and communication problems might disturb daily communication for the patients, yet not be reflected in performance on the cognitive screening tests. The lack of association between the behavioural language measures and alexithymia could thus be symptomatic of the smaller impaired group size, or because these measures do not reflect the communicative impairment some patients are experiencing.

Second, it could be the case that the two measures index rather different aspects of language. The language skills assessed by the behavioural assessments relate to structural language problems. However, patients’ daily communication with others may be disrupted due to pragmatic language impairments. Indeed, few structural language assessments are sensitive to pragmatic language problems, even though these difficulties have very real impacts on participants’ communication with those around them. An example in the field of developmental disorders of language and communication comes from studies of the overlap between autism and developmental language disorder: parents of children with autism self-reported communication difficulties, but these were not apparent in behavioural tests (see Bishop, 2010). The authors suggested that the behavioural tests were not sensitive to the pragmatic language difficulties experienced by these participants. In the present study, the SISCOM scores of language impaired/not impaired groups (categorised on the basis of their behavioural tests) were compared, and it was found those with behaviourally-defined language impairments did report poorer communication at the group level. This would suggest that the SISCOM and behavioural tests reflect overlapping constructs at least to some extent, but future work should consider more in-depth assessment of pragmatic language problems.

We can rule out that the associations between self-reported communication problems and alexithymia are simply reflective of poor self-esteem or shared method variance. If this were the case, we would expect not just the SISCOM component of the Stroke Impact Scale but also other components of this scale to predict increased alexithymia. We examined whether self-reported mobility problems were also predictive of alexithymia: self-reported difficulties in mobility were not associated with alexithymia, and even after adding this measure to our regression, self-reported communication problems remained a significant predictor. Thus, the association between alexithymia and SISCOM is not merely reflective of low self-esteem or shared method variance, given the specific association between alexithymia and self-reported communication problems.

In addition to associations with language, we also examined the associations between alexithymia and hemisphere of damage. Previous findings suggesting alexithymia is more likely to result from right than left hemisphere stroke were not replicated (Spalletta et al., 2001, 2006). The number of patients included in this comparison were double those reported in Spalletta et al (2001), so potentially the disagreement between ours and Spalletta et al.’s findings reflect increased statistical power. In addition, a key difference between our sample and the samples of previous studies is that our exclusion criteria did not include language impairment. It is possible previous studies, by adopting criteria that excluded patients with language problems, missed a role for left hemisphere damage in alexithymia. An additional analysis was conducted to investigate whether limiting our analyses only to patients who were not considered to have language impairment (as per the selection criteria in previous studies) could have led to the different results of the present study: even when language impaired patients were not included in the analyses, patients with right hemisphere damage did not show significantly higher alexithymic traits. Our findings thus suggest that alexithymia can results from either right or left hemisphere damage, and highlight the need for clinicians to be watchful for emotional deficits following brain injury, regardless of hemisphere of damage.

The reported associations are thus only partly supportive of the hypothesis that language processes have a role in alexithymia. Unlike in Hobson et al (2018) associations between alexithymia and behavioural language measures were not found. It is possible this is due to the use of briefer screening instruments in the current study. In Hobson et al (2018) the full Boston picture naming task was used, but the OCS/BCoS measures employed here are designed to provide a rapid and holistic picture of a patient’s functioning, rather than a detailed neuropsychological language assessment.

There are pressing outstanding questions that future research should seek to address. The first is the direction of the association between alexithymia and communication problems. While the language hypothesis of alexithymia as outlined in Hobson et al (2019) argues that language dysfunction underlies problems in emotional functioning, it is logically possible that the relationship could go in the other direction, or be bi-directional in nature. Individuals with alexithymia have reduced empathy (Bird et al., 2010; Grynberg, Luminet, Corneille, Grèzes, & Berthoz, 2010) and report increased interpersonal problems (Zarei & Besharat, 2010): these problems could lead to reduced opportunities to communicate socially, and thus be reflected in self-reports of communicative dysfunction in the SISCOM. This model would perhaps also explain why associations arose for the SISCOM but not with observed language assessment outcomes. A more detailed self-report measure of communicative dysfunction could help to disentangle such questions in the future. Although the reliability of the self-report measures was acceptable, observer report measures of alexithymia and communication problems, given by family members or those close to patients, may also provide additional insight.

A practical point for future researchers to resolve is that it is unclear why modality of response (over the phone versus home assessment) would impact alexithymia scores. Even after including this variable in the regression, SISCOM continued to independently predict alexithymia, so this effect was not due to differences in the communication abilities of home versus phone assessed patients. Participants had the option of how they wished to return their data, and possibly those assessed over the phone were more frail: phone-assessed participants scored higher on the SISMOB than those assessed at home, more time had passed since their stroke, and tended to have higher anxiety symptoms (although aside from anxiety, these factors were also included in the regressions and were not predictive of alexithymia in themselves).

Another possibility for future research would be to utilise the typical recovery from aphasic symptoms during the first few months post-stroke to examine whether short-term language impairments produce transient alexithymic problems. In the current study, the questionnaire measure for alexithymia was delivered at 6 months post stroke, or later, after the majority of recovery from aphasic symptoms would have taken place. There was no measure available for alexithymia at the acute stage. Nonetheless, future research examining alexithymia and aphasia in stroke patients could take both acute and chronic measures of both constructs to investigate this question further.

Finally, if language difficulties lead to alexithymia in stroke, it would be expected that post-stroke depression and anxiety would also pattern, at least partially, with aphasia. There is some support for the idea that depression after stroke is increased in aphasic cases (Carota et al., 2011; Salminen, Saarijärvi, Äärelä, Toikka, & Kauhanen, 1999), and that post-stroke anxiety is associated with left hemisphere damage (Barker-Collo, 2007) although such an association could reasonably be due to associations between depression or anxiety and the severity of general disability following stroke, rather than a specific role of language in emotional functioning per se. In the present sample, the language impaired group did report higher anxiety and depression symptoms.

To conclude, the present study found associations between self-reported communication impairment and alexithymia. Behavioural language impairments were not associated with alexithymia, and patients with left versus right hemisphere damage did not differ in their alexithymia. These results may indicate an association with pragmatic or more subtle communication impairments and alexithymia, or could reflect a relationship between communication problems and alexithymia in which alexithymia-related interpersonal problems underlie communicative breakdown, and feelings of communicative incompetence.

# Acknowledgements

The authors have no conflicts of interest to declare. This study was supported by the Stroke Association UK (TSA LECT 2015/02) and the National Institute for Health Research (NIHR) Oxford Biomedical Research Centre (BRC). The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health.

# References

Ayis, S. A., Ayerbe, L., Ashworth, M., & Wolfe, C. DA. (2018). Evaluation of the Hospital Anxiety and Depression Scale (HADS) in screening stroke patients for symptoms: Item Response Theory (IRT) analysis. *Journal of Affective Disorders*, *228*(June 2017), 33–40. http://doi.org/10.1016/j.jad.2017.11.037

Barker-Collo, S. L. (2007). Depression and anxiety 3 months post stroke: Prevalence and correlates. *Archives of Clinical Neuropsychology*, *22*(4), 519–531. http://doi.org/10.1016/j.acn.2007.03.002

Bird, G., Silani, G., Brindley, R., White, S., Frith, U., & Singer, T. (2010). Empathic brain responses in insula are modulated by levels of alexithymia but not autism. *Brain*, *133*(5), 1515–1525. http://doi.org/10.1093/brain/awq060

Bishop, D. V. M. (2010). Overlaps between autism and language impairment: phenomimicry or shared etiology? *Behavior Genetics*, *40*(5), 618–29. http://doi.org/10.1007/s10519-010-9381-x

Bishop, D. V. M., Nation, K., & Patterson, K. (2014). When words fail us: Insights into language processing from developmental and acquired disorders. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *369*(1634). http://doi.org/10.1098/rstb.2012.0403

Borod, J. C., Bloom, R. L., Brickman, A. M., Nakhutina, L., Curko, E. A., Borod, J. C., … Curko, E. A. (2010). Emotional Processing Deficits in Individuals With Unilateral Brain Damage With Unilateral Brain Damage, *4282*. http://doi.org/10.1207/S15324826AN0901

Brewer, R., Cook, R., & Bird, G. (2016). Alexithymia: A general deficit of interoception. *Royal Society Open Science*, *3*(150664).

Carota, A., Bogousslavsky, J., Annable, L., Aybek, S., Guex, P., Iaria, G., … Staub, F. (2011). A prospective study of predictors of poststroke depression. *Neurology*, *64*(3), 428–433. http://doi.org/10.1212/01.wnl.0000150935.05940.2d

Carson, A. J., MacHale, S., Allen, K., Lawrie, S. M., Dennis, M., House, A., … Summary. (2000). Depression after stroke and lesion location: a systematic review. *The Lancet*, *356*, 122–126. http://doi.org/10.1016/j.jad.2011.06.014

Demeurisse, G., Demol, O., Derouck, M., De Beuckelaer, R., Coekaerts, M. J., & Capon, A. (1980). Quantitative study of the rate of recovery from aphasia due to ischemic stroke. *Stroke*, *11*(5), 455–458. http://doi.org/10.1161/01.STR.11.5.455

Demeyere, N., Riddoch, M. J., Slavkova, E. D., Bickerton, W. L., & Humphreys, G. W. (2015). The Oxford Cognitive Screen (OCS): Validation of a stroke-specific short cognitive screening tool. *Psychological Assessment*, *27*(3), 883–894. http://doi.org/10.1037/pas0000082

Demeyere, N., Riddoch, M. J., Slavkova, E. D., Jones, K., Reckless, I., Mathieson, P., & Humphreys, G. W. (2016). Domain-specific versus generalized cognitive screening in acute stroke. *Journal of Neurology*, *263*(2), 306–315. http://doi.org/10.1007/s00415-015-7964-4

Duncan, P. W., Bode, R. K., Min Lai, S., & Perera, S. (2003). Rasch analysis of a new stroke-specific outcome scale: the stroke impact scale. *Archives of Physical Medicine and Rehabilitation*, *84*(7), 950–963. http://doi.org/10.1016/s0003-9993(03)00035-2

Eizaguirre, A. E., de Cabezon, A. O. S., de Alda, I. O., Olariaga, L. J., & Juaniz, M. (2004). Alexithymia and its relationship with anxiety and depression in eating disorders. *Personality and Individual Differences*, *36*, 321–331.

Engelter, S. T., Gostynski, M., Papa, S., Frei, M., Born, C., Ajdacic-Gross, V., … Lyrer, P. A. (2006). Epidemiology of aphasia attributable to first ischemic stroke: Incidence, severity, fluency, etiology, and thrombolysis. *Stroke*, *37*(6), 1379–1384. http://doi.org/10.1161/01.STR.0000221815.64093.8c

Franz, M., Popp, K., Schaefer, R., Sitte, W., Schneider, C., Hardt, J., … Braehler, E. (2008). Alexithymia in the German general population. *Social Psychiatry and Psychiatric Epidemiology*, *43*(1), 54–62. http://doi.org/10.1007/s00127-007-0265-1

Fusar-Poli, P., Placentino, A., Carletti, F., Allen, P., Landi, P., Abbamonte, M., … Politi, P. L. (2009). Laterality effect on emotional faces processing: ALE meta-analysis of evidence. *Neuroscience Letters*, *452*(3), 262–267. http://doi.org/10.1016/j.neulet.2009.01.065

Grynberg, D., Luminet, O., Corneille, O., Grèzes, J., & Berthoz, S. (2010). Alexithymia in the interpersonal domain: A general deficit of empathy? *Personality and Individual Differences*, *49*(8), 845–850. http://doi.org/10.1016/j.paid.2010.07.013

Henry, J. D., Phillips, L. H., Crawford, J. R., Theodorou, G., & Summers, F. (2006). Cognitive and psychosocial correlates of alexithymia following traumatic brain injury. *Neuropsychologia*, *44*(1), 62–72. http://doi.org/10.1016/j.neuropsychologia.2005.04.011

Hill, E., Berthoz, S., & Frith, U. (2004). Brief report: Cognitive processing of own emotions in individuals with autistic spectrum disorder and in their relatives. *Journal of Autism and Developmental Disorders*, *34*(2), 229–235. http://doi.org/10.1023/B:JADD.0000022613.41399.14

Hobson, H. Brewer, R., Catmur, C., & Bird, G. (2019). The role of language in alexithymia: moving towards a multi-route model of alexithymia. *Emotion Review*,  *11* (3), 247-261. http://doi.org/10.1177/1754073919838528

Hobson, H., Hogeveen, J., Brewer, R., Catmur, C., Gordon, B., Krueger, F., … Grafman, J. (2018). Language and alexithymia: Evidence for the role of the inferior frontal gyrus in acquired alexithymia. *Neuropsychologia*, *111*(December 2017), 229–240. http://doi.org/10.1016/j.neuropsychologia.2017.12.037

Hogeveen, J., Bird, G., Chau, A., Krueger, F., & Grafman, J. (2016). Acquired alexithymia following damage to the anterior insula. *Neuropsychologia*, *82*, 142–148. http://doi.org/10.1016/j.neuropsychologia.2016.01.021

Honkalampi, K., Hintikka, J., Tanskanen, A., Lehtonen, J., & Viinamäki, H. (2000). Depression is strongly associated with alexithymia in the general population. *Journal of Psychosomatic Research*, *48*(1), 99–104. http://doi.org/10.1016/S0022-3999(99)00083-5

Humphreys, G. W., Bickerton, W. L., Samson, D., & Riddoch, M. J. (2012). *BCoS cognitive screen*. Hove: Psychology Press.

Lane, R. D., Sechrest, L., & Riedel, R. (1998). Sociodemographic correlates of Alexithymia. *Comprehensive Psychiatry*, *39*(6), 377–385. http://doi.org/10.1016/S0010-440X(98)90051-7

Laska, A. C., Hellblom, A., Murray, V., Kahan, T., & Von Arbin, M. (2001). Aphasia in acute stroke and relation to outcome. *Journal of Internal Medicine*, *249*(5), 413–422. http://doi.org/10.1046/j.1365-2796.2001.00812.x

Marchesi, C., Brusamonti, E., & Maggini, C. (2000). Are alexithymia, depression, and anxiety distinct constructs in affective disorders? *Journal of Psychosomatic Research*, *49*(1), 43–49. http://doi.org/10.1016/S0022-3999(00)00084-2

Nijsse, B., Spikman, J. M., Visser-Meily, J. M., de Kort, P. L., & van Heugten, C. M. (2019). Social Cognition Impairments in the Long Term Post Stroke. *Archives of Physical Medicine and Rehabilitation*, 1–15. http://doi.org/10.1016/j.apmr.2019.01.023

Pandey, R., Saxena, P., & Dubey, A. (2011). Emotion regulation difficulties in alexithymia and mental health. *Europe’s Journal of Psychology*, *7*(4), 604–623. http://doi.org/10.1037/e617512012-003

Parker, J. D., Bagby, R. M., Taylor, G. J., Endler, N. S., & Schmitz, P. (1993). Factorial validity of the 20-item Toronto Alexithymia Scale. *European Journal of Personality*, *7*(4), 221–232. http://doi.org/10.1002/per.2410070403

Pedersen, P. M., Jorgensen, H. S., Nakayama, H., Raaschou, H. O., & Olsen, T. S. (1996). Aphasia in acute stroke: Incidence, determinants, and recovery. *Annals of Neurology*, *38*(4), 659–666. http://doi.org/10.1002/ana.410400125

Ricciardi, L., Demartini, B., Fotopoulou, A., & Edwards, M. J. (2015). Alexithymia in Neurological Disease : A Review. *The Journal of Neuropsychiatry & Clinical Neurosciences*, (November), 1–9. http://doi.org/10.1176/appi.neuropsych.14070169

Robinson, R. G., & Price, T. R. (1982). Post-stroke depressive disorders: A follow-up study of 103 patients. *Stroke*, *13*(5), 635–641. http://doi.org/10.1161/01.STR.13.5.635

Salminen, J. K., Saarijärvi, S., Äärelä, E., Toikka, T., & Kauhanen, J. (1999). Prevalence of alexithymia and its assocation with sociodemographic variables in the general population of Finland. *Journal of Psychsomatic Research*, *46*(1), 75–82. http://doi.org/10.1016/S0022-3999(98)00053-1

Saxena, P., Dubey, A., & Pandey, R. (2011). Role of Emotion Regulation Difficulties in Predicting Mental Health and Well-being. *Journal of Projective Psychology & Mental Health*, *18*, 147–155.

Schmidt, U., Jiwany, A., & Treasure, J. (1993). A controlled study of alexithymia in eating disorders. *Comprehensive Psychiatry*, *34*(1), 54–58. http://doi.org/10.1016/0010-440X(93)90036-4

Spalletta, G., Pasini, A., Costa, A., De Angelis, D., Ramundo, N., Paolucci, S., & Caltagirone, C. (2001). Alexithymic features in stroke: effects of laterality and gender. *Psychosom Med*, *63*(6), 944–950. Retrieved from http://www.psychosomaticmedicine.org/content/63/6/944.full.pdf

Spalletta, G., Ripa, A., Bria, P., Caltagirone, C., & Robinson, R. G. (2006). Response of emotional unawareness after stroke to antidepressant treatment. *American Journal of Geriatric Psychiatry*, *14*(3), 220–227. http://doi.org/10.1097/01.JGP.0000194647.72654.a1

Su, J. A. (2016). Alexithymia and the incidence of post-stroke depression. *European Psychiatry*, *33*, S397. http://doi.org/10.1016/j.eurpsy.2016.01.1426

Taylor, G. J., Bagby, R. M., & Parker, J. D. (1991). The alexithymia construct. A potential paradigm for psychosomatic medicine. *Psychosomatics*, *32*(2), 153–164. http://doi.org/10.1016/S0033-3182(91)72086-0

Valdespino, A., Antezana, L., Ghane, M., & Richey, J. A. (2017). Alexithymia as a transdiagnostic precursor to empathy abnormalities: The functional role of the insula. *Frontiers in Psychology*, *8*(DEC), 1–7. http://doi.org/10.3389/fpsyg.2017.02234

van ’t Wout, M., Aleman, A., Bermond, B., & Kahn, R. S. (2007). No words for feelings: alexithymia in schizophrenia patients and first-degree relatives. *Comprehensive Psychiatry*, *48*(1), 27–33. http://doi.org/10.1016/j.comppsych.2006.07.003

Wang, X., Chung, M. C., Hyland, M. E., & Bahkeit, M. (2011). Posttraumatic stress disorder and psychiatric co-morbidity following stroke: The role of alexithymia. *Psychiatry Research*, *188*(1), 51–57. http://doi.org/10.1016/j.psychres.2010.10.002

Wood, R. L., & Williams, C. (2007). Neuropsychological correlates of organic alexithymia. *Journal of the International Neuropsychological Society : JINS*, *13*(3), 471–479. http://doi.org/10.1017/S1355617707070518

Zarei, J., & Besharat, M. A. (2010). Alexithymia and interpersonal problems. *Procedia - Social and Behavioral Sciences*, *5*(2), 619–622. http://doi.org/10.1016/j.sbspro.2010.07.153

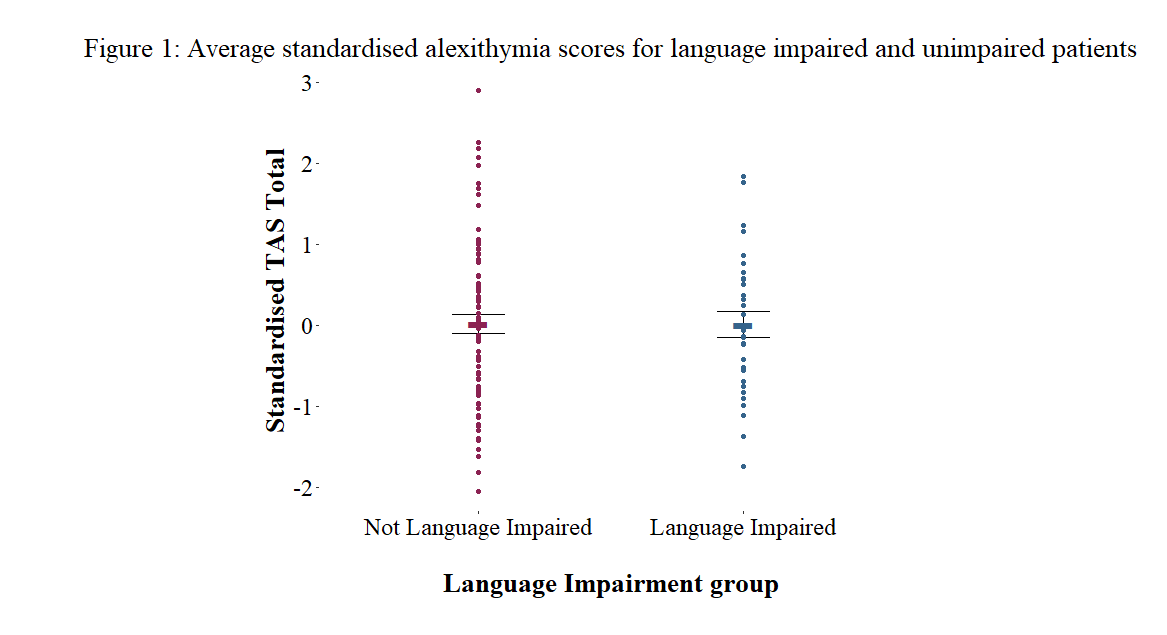
Zigmond, A. S., & Snaith, R. P. (1983). The Hospital Anxiety and Depression Scale. *Acta Psychiatrica Scandinavica*, *67*(6), 361–370. http://doi.org/10.1111/j.1600-0447.1983.tb09716.x

|  |  |  |
| --- | --- | --- |
| **Table 1** Participant sample characteristics | | |
| Sample Characteristics | Category | Proportion of Patients (N = 118)  / Mean (SD) |
| Gender | Male | .53 |
| Handedness | Left | .04 |
|  | Right | .95 |
|  | Ambidextrous | .01 |
| Aetiology | Haemorrhage | .20 |
|  | Ischaemia | .80 |
| Lesion Lateralisation | Unilateral left hemisphere | .43 |
|  | Unilateral right hemisphere | .48 |
|  | Bilateral | .09 |
| Time since stroke at questionnaire collection (months) | | 28.04 (24.43) |
| Age at stroke (years) |  | 73.14 (11.37) |
| Education (years) |  | 12.38 (2.75) |
|  |  |  |

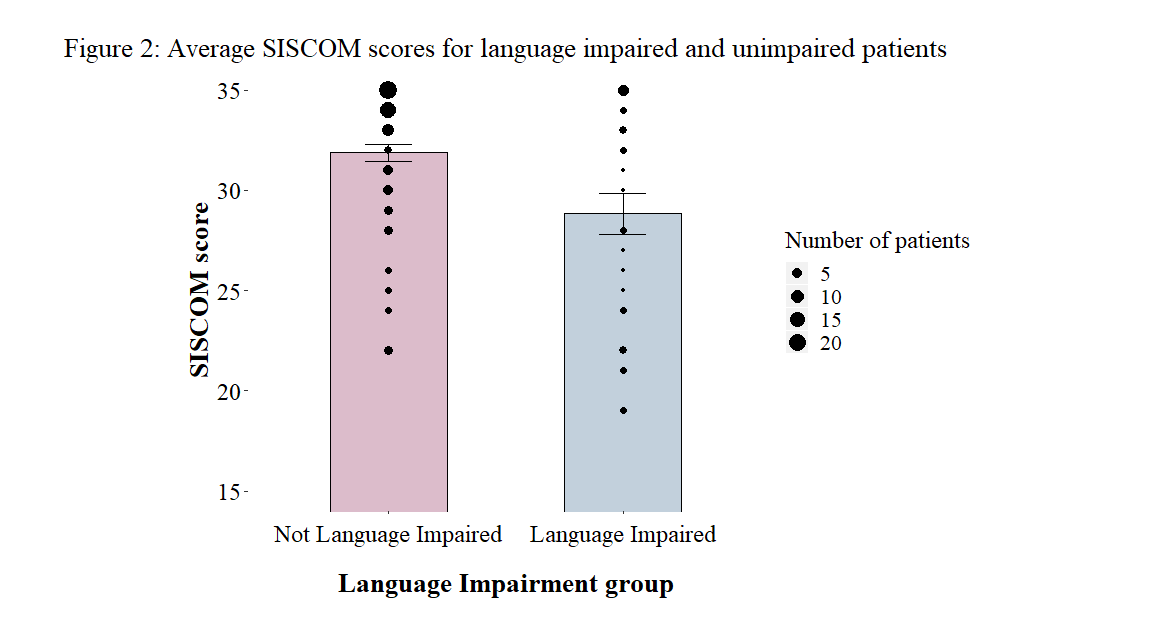
|  |  |  |  |
| --- | --- | --- | --- |
| **Table 2** Standardised Betas, significance and squared semi-partial coefficients for predictors of TAS-20 | | | |
|  | β | *p* | *Squared semi-partial coefficients* |
| SISCOM | -0.24 | 0.01 | 0.04 |
| HADS Anxiety | 0.22 | 0.03 | 0.03 |
| HADS Depression | 0.20 | 0.05 | 0.03 |
| Time since stroke | -0.09 | 0.30 | 0.01 |
| Gender | -0.03 | 0.75 | 0.001 |
| Age at stroke | -0.06 | 0.48 | 0.004 |
| Education | -0.21 | 0.02 | 0.04 |

TAS-20 = Toronto Alexithymia Scale; SISCOM = Stroke Impact Scale Communication scale; HADS= Hospital Anxiety and Depression. Note that squared semi-partial coefficients represent the unique proportion of variance attributed to the predictor, i.e. 0.01 represents 1% of variance of alexithymia explained.

**Figure 1** Standardised alexithymia scores for language impaired and unimpaired patients. The coloured bars represent the mean for either group. Error bars represent standard error.



**Figure 2** SISCOM scores for language impaired and unimpaired patients. Bars represent the mean scores for the group. Error bars represent standard error. Size of point is used to indicate the number of patients with the same score.

****