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Psychosocial function in the first year after childhood stroke

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ABBREVIATIONS

SDQ Strengths and Difficulties Questionnaire

[abstract]

AIM Childhood stroke disrupts brain development and emerging neural networks. Motor, cognitive, and language deficits are well recognized, yet little is known about psychosocial function after childhood stroke. This study aims to describe psychosocial function within the first year after childhood stroke, and to identify factors associated with outcome.

METHOD Thirty-seven children were involved in a prospective, longitudinal study investigating recovery over the first year after childhood stroke. Children's social functioning was assessed at 6- and 12-months poststroke and psychological function at 12-months poststroke, using standardized measures.

RESULTS Mean social function was poorer at both 6- and 12-months poststroke, compared to prestroke. Psychological problems were more common than expected, with emotional difficulties and hyperactivity–inattention most significantly affected. Poorer social function was associated with older age at onset, acute neurological impairment, and prestroke social impairment. Social and psychological problems were associated with parent mental health.

INTERPRETATION While not all children are affected, psychosocial impairment affects a significant minority after childhood stroke. Older age at onset, acute neurological impairment, prestroke social problems, and poorer parent mental health were associated with deficits. Identifying early predictors of poorer outcome will facilitate early intervention. Of particular importance is parent mental health, suggesting support for families may improve child outcome.

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What this paper adds

- Children are at elevated risk of psychosocial impairment after childhood stroke.
- Psychosocial impairments may begin to emerge within the first 12 months of recovery.
- Children who have stroke onset at an earlier age appear to have better social outcomes.
- Parent mental health is associated with child psychosocial outcomes.

[main text]

Childhood stroke can occur between 1 month and 18 years of age and is increasingly recognized as a significant cause of childhood disability. Long-term neurological impairment, including motor, cognitive, and language deficits, has been reported in 50 to 60 per cent of children after childhood stroke.[1,2] Due to rapid brain development, childhood stroke has the potential to cause impairment in already developed skills and disrupt the development of emerging skills and the neural networks underpinning them. Psychosocial skills have protracted development throughout childhood and adolescence, placing them at risk of disruption.

Intact psychosocial function is critical to many aspects of long-term adjustment, including mental health, quality of life, academic progress, employment, and functioning within the community. A recent review found the literature to date to show that children are at elevated risk of psychosocial impairment poststroke,[3] yet these studies are often limited by small sample sizes and are generally cross-sectional in design. Studies have generally utilized social domain scores from broader measures or non-standardized tools. Further, no study has prospectively captured psychosocial function at specific time points after childhood stroke, and factors associated with outcomes have largely been unexplored.

Despite the limitations in the literature, there is evidence that children are at risk of social impairment after childhood stroke. A study using qualitative questions to examine peer and family integration in 16 children after childhood stroke reported significant changes in friendships with peers.[2] Using a quality of life measure, another study found that children reported reduced social acceptance, and parents reported mood instability and decreased social support from peers.[4] Other studies have also reported reduced social and peer relationship scores on parent-report quality of life measures.[5–9]

Psychological outcomes have received more attention in the literature, with a variety of internalizing (anxiety, inattention) and externalizing problems (aggression, hyperactivity, emotional lability) reported.[3] Max and colleagues explored psychiatric disorders in a sample of childhood stroke (*n*=29) and a chronic illness control group.[10,11] They reported 59 per cent of children with stroke had developed psychiatric disorders compared to only 14 per cent of controls.[10] The disorders with the highest rate of prevalence in this sample were attention-deficit–hyperactivity disorder (ADHD) (49%), anxiety disorders (31%), mood disorders (21%), and personality change (17%). In a study of 133 children with stroke, parents reported significantly poorer emotional well-being, compared to healthy controls,[8] though rates of impairment were not reported. Other studies have generally consisted of sample sizes fewer than 20, but have reported high rates of emotional problems (25%),[7] externalizing behaviours (44%),[2] and ADHD (50%).[4]

Currently, our understanding of which factors may predict psychosocial outcome after childhood stroke is limited and available findings are inconsistent. Studies exploring clinical features of childhood stroke have generally found no association with lesion characteristics,[4,7,12] yet one study reported a relationship between lesions in the ventral putamen, medial and orbital prefrontal cortex, and ADHD symptoms.[13] Studies investigating the effect of age at stroke on social function have generally found no association.[4,7] However, a recent study including self-report measures found that younger age at stroke was associated with higher levels of self-esteem and fewer problems in social interaction.[14] Research into the impact of neurological impairments on psychosocial function is largely lacking, but one study has reported that children with more severe neurological function had poorer social adjustment and social participation.[15] The impact of environmental factors such as socioeconomic status, family functioning, and parent mental health have been found to influence health outcomes following other forms of acquired brain injury, such as childhood traumatic brain injury (TBI).[16–19] Environmental factors have not been extensively explored

in the childhood stroke literature, but a recent cross-sectional studies from our team found family function and parent mental health to be associated with psychosocial outcomes.[14,20]

The aims of this study were to prospectively explore psychosocial function in the first year after childhood stroke and identify factors associated with poorer function. No previous study has used prospective, longitudinal design to examine the psychosocial domain in childhood stroke. While the first 12-months after childhood stroke is a transient phase, with recovery still taking place, examining psychosocial function during this phase may identify factors associated with longer-term difficulties. We predicted social function would be reduced at 6- and 12-months poststroke compared to prestroke, and mean scores at both poststroke time points would be lower than normative expectations. Based on the limited childhood stroke and TBI literature we predicted environmental and child factors would be associated with outcome, but neurobiological factors would not.

METHODS

Design

We employed a prospective, longitudinal design, with three time points: prestroke/acute, 6-months, and 12-months poststroke.

Participants

This study recruited a sample of children aged 3 months to 16 years, presenting to The Royal Children's Hospital Melbourne with focal arterial ischemic stroke (AIS) from December 2007 to January 2012 as described in a previous paper.[21] Children were included if brain magnetic resonance imaging (MRI) confirmed acute ischemic parenchymal infarct corresponding to one or more arterial territories. Children with previously diagnosed stroke, primarily hemorrhagic infarction, coexisting diffuse brain injury due to a traumatic or hypoxic ischemic event, and preterm infants (i.e. born before 36 weeks' gestation) were excluded.

Measures

A. Prestroke/acute

Neurobiological information. Infarct laterality, lesion location, and vascular territory affected were rated by two neuroradiologists (MD, LC) and based on visual inspection of imaging obtained at the time of diagnosis using a standardized coding system. For analysis, lesion location was collapsed into two groups: (1) discrete cortical pathology including lesions that

affected regions of the cerebral cortex, and (2) cortical/cortical and subcortical, where pathology included brain regions below the cerebral cortex, as well as the brain stem and cerebellum. Lesion size was determined by the vascular territory affected; where a major vessel was affected the lesion was coded large, where a branch was affected it was coded medium, and where a perforator was affected it was small.

Acute neurological impairment was measured using the Pediatric Stroke Outcome Measure (PSOM)[22,23] within the first week after diagnosis, rated by a trained pediatric occupational therapist. The PSOM is a detailed neurological examination, with outcome scored in terms of degree of impairment in each of language, cognition, and sensorimotor. Total impairment scores (out of a maximum of 10) were collapsed into good/poor (0 or 0.5 representing good and poor >0.5), consistent with the approach taken previously.[22]

Child functioning. Socialization from the Vineland Adaptive Behavior Scale, 2nd edition (VABS-II)[24] was used to measure prestroke social functioning. This was completed by parents within the first month after diagnosis, with instructions to rate their child's abilities before the stroke. Scores were calculated as standard scores (mean 100, standard deviation [SD] 15).

Acute cognitive function was assessed using one of the following at 1-month postdiagnosis: (1) Cognition subscale, Bayley Scales of Infant Development III (BSID-III)[25] (age ≤3 .5y); or (2) IQ composite from the Kaufman Brief Intelligence Test 2 (KBIT2)[26] (≥4y). Both have mean of 100 (SD 15).

Family environment. The Social Risk Index (SRI)[27] rates a number of family factors (family structure, education of the primary caregiver, occupation and employment status of primary income earner, language spoken at home, and maternal age at child's birth) and was collected within the first month after diagnosis. Scores range 1 to 10, higher scores indicate higher social risk.

B. 6- and 12-month assessments

Family environment. The Mental Component Summary (MCS) (mean 50, SD 10) from the SF-36v2 (Quality Metric, Lincoln, RI) was used to measure parent mental health 12-months post-childhood stroke. The SF-36v2 is a 36-item, self-rated questionnaire that evaluates health status across eight health concepts, with the MCS primarily comprising items related to social, emotional, and psychological well-being. Australian norms were employed.[28]

Child psychosocial functioning. Social function was evaluated using socialization from the VABS-II at 6- and 12-months. Socialization is measured across the domains of interpersonal relationships (e.g., shows a desire to please others), play and leisure time (e.g., seeks out others for play or companionship), and coping skills (e.g. acts appropriately when introduced to strangers). Scores greater than one SD below the mean (i.e. 85) are borderline and greater than two SD below the mean (i.e. 70) are clinically significant.

Psychological function was measured at 12-months poststroke using the Strengths and Difficulties Questionnaire (SDQ).[29] Five subscales are calculated: emotional symptoms, conduct symptoms, hyperactivity–inattention, peer problems, and prosocial behaviours. Total difficulties score is obtained by combining the scores for all but prosocial behaviour. SDQ data are compared to US normative data (n=10 367) provided on the SDQ website (www.sdqinfo.com). Scores were banded into 'normal', 'borderline', or 'abnormal', where 80 per cent are expected to be 'normal', 10 per cent 'borderline', and 10 per cent 'abnormal'.

Procedure

The study was approved by the Human Research Ethics Committee of the Royal Children's Hospital, Melbourne (HREC# 27114). Participants were identified by acute care clinicians, who discussed the study with parents, and referred them to the study. Participants were all recruited within the first month after diagnosis. Written consent was obtained by parents for their participation and that of their children. All children were able to complete the cognitive assessment conducted at 1-month poststroke.

Statistical analysis

A repeated measures analysis of variance (ANOVA) was conducted to explore the trajectory of socialization prestroke and 6- and 12-months poststroke. Time was included as a discrete predictor, and Bonferroni-adjusted posthoc comparisons between times was explored. Differences in the ordinal proportions of children with social impairment (normal/borderline/clinically significant) prestroke, 6-months, and 12-months were tested using Wilcoxon signed-rank tests.

One-sample *t*-tests were conducted to compare mean socialization ratings to normative data, and because of the skew of SDQ total and subscale scores, median ratings were compared to SDQ norms using one-sample Wilcoxon signed rank tests. Effect sizes were determined by Cohen's *d*. Pearson correlation coefficient was used to investigate

whether social and psychological problems occur together in children who had normal premorbid social function. Multiple regressions were then conducted to explore the contribution of neurobiological, child, and environmental factors for the socialization domain of the VABS-II and the total difficulties domain of the SDQ at 12-months poststroke. Model improvement through predictor removal was tested using both likelihood ratio tests, and comparisons of Akaike information criterion (AIC).

All analyses diagnostics were examined to ensure meeting model assumptions. Data missingness was explored, and the above models were rerun using multiply imputed data as a sensitivity analysis. Results did not differ from analyses with listwise deletion, and the latter was applied. All analyses were carried out using Stata v14.1 (StataCorp, College Station, Texas).

RESULTS

Sample description

Seventy-three children were identified as meeting the study inclusion criteria. Twenty-eight were not approached (five resident interstate; eight died acutely; 15 missed/not referred in time). Four declined to participate (one because of employment; three no reason given). Forty-one children were recruited. One child died of an oncological illness before 6-months follow-up. Five participants were excluded because of missing data at one or more time points and, of the 28 participants old enough to receive SDQ ratings, four did not complete the questionnaire. The social function (VABS-II) of 35 participants (one missing at 12 months) and psychological function (SDQ) of 24 participants are reported (Table I).

Social function over the first year poststroke

A repeated measures ANOVA identified a statistically significant time poststroke term for socialization ($F[2, 68]=9.17$, $p<0.001$). Model means and 95 per cent confidence intervals are presented in Figure 1. Plotted mean scores show a significantly poorer scores at 6-months, posthoc test $p<0.001$. The levelling out at 12-months was not statistically significant. They were also significantly poorer than normative expectations at 6-months ($p=0.010$) and 12-months ($p=0.033$).

Five participants had impaired premorbid socialization ratings; a sensitivity analysis was conducted to determine whether this impairment influenced the results. After removal of these five cases, the significant time term ($F[2,58]=10.43$, $p<0.001$) and posthoc premorbid to six month comparison ($p<0.001$) remained. Mean scores did not significantly differ compared

to normative expectations at either 6-months (p=0.162) or 12-months poststroke (p=0.249). Yet when compared to premorbid ratings, there was evidence that mean socialization was significantly poorer at both 6-months (p<0.001) and 12-months (p=0.001).

Social function impairment

There was no significant difference in the proportion of children in the impaired range from prestroke to 6-months (p=0.059), 6-months to 12-months (p=1.000), or prestroke to 12-months (p=0.096). However, there was a trend for increased rates of impairment over time. Prestroke ratings showed 14 per cent had socialization scores in the borderline range; this increased to 16 per cent borderline and 6 per cent in the clinically significant range at 6-months and 12-months (Fig. 2).

Psychological function 12-months poststroke

On the SDQ, compared to normative data, our sample demonstrated significantly higher mean hyperactivity–inattention (Z=2.55, p=0.011, d=0.55) only. No significant mean differences were found for total difficulties (Z=1.89, p=0.059, d=0.56); emotional symptoms (Z=1.89, p=0.059, d=0.59); conduct problems (Z=1.01, p=0.314, d=0.28); peer problems (Z=0.09, p=0.931, d=0.19); or prosocial behaviour (Z=–1.84, p=0.066, d=0.37). Further, there was a lower proportion of children in the normal range than expected across the majority of domains (Fig. 2).

Relationship between social and psychological function

The five participants with prestroke social impairment were removed, to explore the relationship between social and psychological function in children without previous social impairment. There was a strong, negative correlation between the two variables (r=–0.62, n=24, p=0.001), with lower social function associated with higher psychological problems.

Factors associated with outcome

Comparison of multiple regression models using likelihood ratio test and AIC improvement arrived parsimonious models for social and psychological function was carried out. It was found that lesion location (discrete subcortical vs cortical and subcortical), acute cognition (1-month poststroke), and social risk did show significant contribution to either model, and were excluded. For social function (F[4,30]=8.42, p<0.001, R^2=0.53), increasing age at stroke significantly (p=0.010) and impaired acute neurological function (p=0.020) predicted poorer

socialization, and higher premorbid social function significantly predicted higher 12-month social function (p<0.001). Parent mental health significantly predicted both outcomes, showing a positive relationship with socialization (p=0.012), and was the only predictor to be included in the psychological functioning model (F[1,22]=24.08, p<0.001, R^2=0.52), with a strong negative relationship with SDQ outcome (p<0.001) (Table II).

DISCUSSION

Consistent with previous research,[4–9,14,20] social function post-childhood stroke was significantly poorer than expected. While group means remained within the average range, social function at both 6- and 12-months poststroke were significantly poorer compared to prestroke ratings. While, as a group, these children's social function was not outside the normal range, this suggests that their social development was negatively impacted in the year after stroke. Additionally, almost one-quarter of children were impaired at 6- and 12-months poststroke. While 14 per cent were impaired prestroke, premorbid social problems are an important risk factors for later social deficits. Further, our results show that a proportion of previously typically developing children develop social difficulties post-childhood stroke. A recent study exploring long-term outcomes after childhood stroke reported 33 per cent had impaired social life and 15 per cent were dependent in social activities.[30] Research to date suggests that social impairment does not affect all children after stroke, but it may affect a significant minority.

Overall, psychological function was not impaired compared to normative expectations, with the exception of hyperactivity–inattention. Evaluation of impairment rates also revealed a high proportion of children displaying hyperactivity–inattention, with 12 per cent borderline and 21 per cent in the abnormal range, and much higher than normative expectations of 10 per cent.[29] This is consistent with previous research which has reported the prevalence of ADHD symptoms as high as 50 per cent.[4,31] High rates of emotional difficulties were also observed, with 17 per cent borderline and 21 per cent in the abnormal range. These findings are similar to reports in cross-sectional studies, reporting high levels of emotional problems.[7,8] Higher than expected proportions of impairment were also observed for conduct problems (21% borderline; 12% abnormal) and peer problems (8% borderline; 21% abnormal).

Consistent with previous studies, we found no association between psychosocial function and lesion location.[4,7,12] Because of the complex nature of psychosocial function, there may be a lack of relationship with 'focal' measures such as lesion location. The

majority of studies have found no association with age at stroke.[4,7] In contrast, we found a significant association between age at onset and social function, with younger children showing better outcomes. This may be because of the young age of the children in this sample, with skills still developing and deficits difficult to recognize. Few studies have explored the association between neurological impairment and psychosocial outcomes. We found acute neurological impairment was associated with social function, consistent with one previous study,[15] but not psychological function.

Not surprisingly, prestroke social function was associated with poststroke social function. While 50 per cent of children who suffer a stroke are previously healthy, chronic diseases of childhood, such as cerebral artery disease and cardiac disease, frequently underlie childhood stroke.[32] This high prevalence of previous disease may impact psychosocial development, with stroke further compounding these pre-existing problems. Thus, children identified to have underlying conditions may be more vulnerable to developing impairments and these children may benefit from early intervention. Our sample size was too small to investigate the impact of stroke risk factors, but this is an important area to explore in future studies. It should also be noted that prestroke social function may be attributable to non-medical internal factors, such as the child's temperament or personality as well as environmental factors. These factors may also play a role in social function poststroke.

The family environment has been reported to be the most significant predictor of psychosocial outcome to date, with some factors playing a more important role than others.[14,20] Indirect, stable environmental factors, such as socioeconomic status and parental education, have been reported to have less impact, with more direct factors, such as family functioning and parent mental health, significant predictors of outcome. Consistent with these earlier studies, we found social risk did not predict psychosocial outcome; however, there was a strong relationship between parent mental health and both social and psychological outcome. While it remains unclear whether poor parent mental health results in poorer child functioning or vice versa, there is emerging evidence parent-based behavioural inventions aimed at improving child behaviour and social competence after brain insult also improve parent mental health.[33]

There are limitations to this study. Firstly, our sample size is quite small, which limited the analyses we were able to perform and ability to detect subtle effects. Second, the use of the socialization domain from the VABS-II as a measure of social function fails to comprehensively examine social skills. Utilizing a dedicated measure of social function may provide more detailed information regarding the nature of social problems and aid in the

development of targeted interventions. A third limitation is the sole reliance on parent ratings of psychosocial function. This may be strengthened in future research by the addition of teacher and self-report ratings, although self-report is difficult in such young children. The time since stroke is another limitation. Six- and 12-months poststroke represents a transient phase, when children are still recovering and thus difficulties they experience during this time may dissipate. However, by 1-year poststroke children are usually discharged from services and so it is important to plot these difficulties early to identify risk factors and to intervene early. We are planning a longer follow-up of these children to examine longer-term outcomes. A final limitation is the young age of the children in this study. It is possible that in such young children, particularly with many of them preschool age, psychosocial impairments may be difficult to recognize.

Conclusions

Despite the limitations, our results suggest that children are at elevated risk for developing psychosocial difficulties after childhood stroke, emerging within the first year of recovery. These findings are novel, because previous studies have not examined psychosocial function over the first year after childhood stroke, including reference to prestroke evaluations. We found a strong relationship between parent mental health and psychological function. This association with parental wellbeing suggests that greater support for families, particularly within the first year after childhood stroke, may have a significant effect on children's outcomes.

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Table I: Sample demographic and clinical characteristics

n	35		
Mean age at stroke onset, y (SD), range	6.8	(4.9)	0.3–16.4
Male, %	46		
Epilepsy at 12 months poststroke, %	0		
Aetiology/identified risk factors, %			
Cardioemolic	26		
Moyamoya	9		
Cervical arterial dissection	9		
Steno-occlusive cerebral arteriopathy	6		
Multiple probable/possible etiologies	9		
Undetermined etiology	43		
Lesion characteristics			
Lesion size[a], %			
Large	11		
Medium	80		
Small	9		
Vascular territory, %			
Partial MCA	51		
PCA	6		
Vertebro-Basilar arteries	20		
Multiple	23		
Laterality, %			
Left	23		
Right	40		
Bilateral	17		
Inratentorial only	20		
Location, %			
Discrete subcortical	63		
Cortical/cortical and subcortical	37		
Neurological examination			
Acute PSOM, median, IQR	2.0	1.0–3.5	
Acute PSOM impaired[b], %	20		

MCA, middle cerebral artery; PCA, posterior cerebral artery; PSOM, Pediatric Stroke Outcome Measure; IQR, interquartile range. [a]Lesion size – vascular territory affected: large, major vessel; medium, branch; small, perforator. [b]Impaired PSOM scores were total scores >0.5.

Table II: Multiple regression for the socialization domain of the VABS-II and the total difficulties domain of the SDQ

	VABS-II: socialization *n*=35			SDQ: total difficulties *n*=24		
Predictor[a]	B	SE	*p*	B	SE	*p*
Age at stroke	–0.35	0.13	0.010			
Acute neurological function (Impaired)	–0.32	0.13	0.020			
Premorbid socialization	0.53	0.13	<0.001			
Parent mental health	0.34	0.13	0.012	–0.72	0.15	<0.001
R^2	0.53		<0.001	0.52		<0.001

VABS-II, Vineland Adaptive Behavior Scale, 2nd edition; SDQ, Strengths and Difficulties Questionnaire; SE, standard error; B, standardized beta coefficient. [a]Lesion location (discrete subcortical vs cortical and subcortical), acute cognition (1-month poststroke), and social risk did not contribute to either model.

Figure 1: Observed means and 95% confidence intervals of socialization scores for premorbid, 6-months and 12-months poststroke, including (A) the total sample (*n*=35), and (B) those with normal premorbid scores (≥86, *n*=30). VABS-II, Vineland Adaptive Behavior Scale, 2nd edition.

Figure 2: (A) Percentage of participants impaired on socialization from the Vineland Adaptive Behaviour Scale: premorbid, 6-months and 12-months poststroke. (B) Percentage of participants impaired on the Strengths and Difficulties Questionnaire subscales based on normative data. ES, emotional symptoms; CP, conduct problems; HYP, hyperactivity–inattention; PP, peer problems; PB, prosocial behavior; TD, total difficulties.