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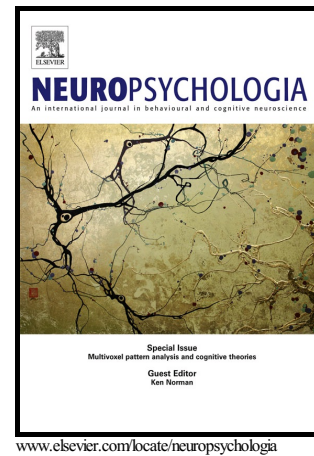
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Social cognition impairments after aneurysmal subarachnoid haemorrhage: associations with deficits in interpersonal behaviour, apathy, and impaired self-awareness

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**Social cognition impairments after aneurysmal subarachnoid haemorrhage:
associations with deficits in interpersonal behaviour, apathy, and impaired self-
awareness**

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Abstract

Behavioural disturbances are frequently found after aneurysmal subarachnoid haemorrhage (aSAH). Social cognition impairments have been suggested as a possible underlying mechanism for behavioural problems. Also, aSAH is likely to result in damage affecting frontal-subcortical circuits underlying social cognition. Therefore, we aimed to investigate social cognition after aSAH and its associations with behavioural problems (deficits in interpersonal behaviour, apathy, and impaired self-awareness) and focal as well as diffuse brain damage. 88 aSAH patients (in the subacute phase post-aSAH) and 60 age-, sex- and education-matched healthy controls participated. Tasks for emotion recognition, Theory of Mind (ToM), and empathy as well as questionnaires were used. Cortical infarctions in frontal and non-frontal areas on MRI, aneurysm circulation and aSAH-related events were taken into account. Compared to healthy controls, aSAH patients performed significantly worse on tasks for emotion recognition, ToM and empathy. Poor performance on ToM and emotion recognition was associated with proxy-ratings indicating impaired interpersonal behaviour and apathy and with indications of impaired self-awareness. No associations were found between deficits in social cognition and frontal or non-frontal cortical lesions on MRI. Also, aneurysm circulation and aSAH-related events such as hydrocephalus, vasospasm, and treatment method did not explain why and how social cognitive deficits did occur after aSAH. In conclusion, emotion recognition, ToM and empathy were clearly impaired in aSAH patients and these deficits were related to apathy and deficits in interpersonal behaviour as reported by proxies and to impaired self-awareness. This association strengthens the assumption of impaired social cognition as an underlying construct of behavioural problems after aSAH. Consequently, social cognition tests and proxy-ratings should be used in clinical practice, irrespective of lesion location on MRI or aneurysm circulation, to improve the detection and treatment of apathy and deficits in interpersonal behaviour after aSAH.

Keywords: social cognition, subarachnoid haemorrhage, apathy, self-awareness, magnetic resonance imaging

1. Introduction

Aneurysmal subarachnoid haemorrhage (aSAH) is an acute bleeding in the subarachnoid space, the area between the pia mater and the arachnoid membrane, and is caused by the rupture of a cerebrovascular aneurysm. It is a serious neurological condition associated with high mortality and morbidity (Nieuwkamp et al., 2009) with negative consequences for everyday life functioning (return to work, leisure activities) of survivors (Buunk, Groen, Veenstra, & Spikman, 2015; Passier, Visser-Meily, Rinkel, Lindeman, & Post, 2011). Common sequelae of aSAH are anxiety, depression, fatigue, and cognitive deficits in memory, executive functions, and language (Al-Khindi, Macdonald, & Schweizer, 2010). Furthermore, behavioural disturbances are frequently found after aSAH (Al-Khindi et al., 2010), involving inadequate interpersonal behaviour (Ogden, Utley, & Mee, 1997; Storey, 1970), apathy (Marin, Biedrzycki, & Firinciogullari, 1991), and impaired self-awareness (Buchanan, Elias, & Goplen, 2000; Hutter & Gilsbach, 1995; Hutter & Kreitschmann-Andermahr, 2014).

In recent years, impairments in social cognition have increasingly been put forward as a possible explanation for these behavioural problems. Social cognition refers to the mental capacities needed to recognize and understand the behaviour of others and to react appropriately in social situations (Adolphs, 2001; Amodio & Frith, 2006; Beer, John, Scabini, & Knight, 2006). A distinction can be made between ‘hot’ and ‘cold’ social cognition, which are related to two different systems (Frith & Frith, 2010): the mirror system and the mentalizing system. ‘Hot’ social cognition allows people to understand others’ feelings and

show empathy (Blair, 2003), which intrinsically guides their behavioural responses to others and motivates their actions. Important components are emotion recognition and affective empathy, that is the ability to share the emotions of others, while recognizing that they are distinct from one's own (Baron-Cohen & Wheelwright, 2004). Such capacities are related to the mirror system, which facilitates understanding of others' emotions by a mechanism of motor resonance, with mirror neurons being the possible substrate. Consequently, the perception of others' emotions is converted into one's own experiences (Gallese, Keysers, & Rizzolatti, 2004; Keysers & Gazzola, 2006). 'Cold' social cognition, which corresponds with the mentalizing system, entails thinking about something from another's perspective. It is the ability to understand others' behaviour based on intentions, thoughts, and beliefs, i.e. to have a Theory of Mind (ToM) (Castelli, Frith, Happe, & Frith, 2002; Leslie, 1987). More specifically, 'cognitive ToM' is similar to knowledge about others' intentions and beliefs, and 'affective ToM' to the understanding of others' emotional states. Affective ToM is often used interchangeably with 'cognitive empathy' (Shamay-Tsoory, 2011). The aforementioned aspects of social cognition are crucial for effective social communication and therefore for appropriate social behavioural functioning (Henry, von Hippel, Molenberghs, Lee, & Sachdev, 2016; Kennedy & Adolphs, 2012). The various aspects of social cognition, i.e. emotion recognition, ToM and empathy, are underpinned by several interconnected brain regions and circuits. Generally, it is assumed that the neural substrates of social cognition involve a frontal-subcortical circuit, comprising the orbitofrontal cortex and ventromedial prefrontal cortex as important regions (Adolphs, 2009; Channon et al., 2007; Lieberman, 2007; Tekin & Cummings, 2002). Evidence has been found for a mediating role of the frontal cortex together with temporoparietal areas and the hippocampus in regulation of social responses and generating a context for social information (Lieberman, 2007; Phillips, Drevets, Rauch, & Lane, 2003). The amygdala circuitry, with its interconnections with

prefrontal and temporal brain areas, was found also to be involved in social cognition (Siegal & Varley, 2002).

Traumatic brain injury (TBI) and stroke often result in damage to these prefrontal-subcortical brain circuits underlying social cognition. In line with this, several studies on TBI (de Sousa, McDonald, & Rushby, 2012; Henry et al., 2016; McDonald, 2013; Ryan et al., 2016; Spikman et al., 2013) and stroke (Blonder, Pettigrew, & Kryscio, 2012; Happe, Brownell, & Winner, 1999; Martory et al., 2015; Wilkos, Brown, Slawinska, & Kucharska, 2015) found impairments in social cognition, as well as associations between these impairments and behavioural disturbances. As early as in 1978, Lezak related impairments in social perceptiveness to personality changes after brain injury. More recently, deficits in interpersonal behaviour have been related to impaired emotion recognition (May et al., 2017; Radice-Neumann, Zupan, Babbage, & Willer, 2007; Spikman et al., 2013) and ToM (McDonald, 2013; Milders, Fuchs, & Crawford, 2003) after TBI. Research on stroke also revealed an association between impaired emotion recognition and interpersonal behavioural impairments (Yuvaraj, Murugappan, Norlinah, Sundaraj, & Khairiyah, 2013). In addition, the relationship between apathy and social cognition has been investigated in patients with acquired brain injury (ABI) and neurodegenerative disorders. In these studies, associations between apathy and impaired social judgements (Njomboro, Humphreys, & Deb, 2014), impaired ToM (van der Hulst, Bak, & Abrahams, 2015), and emotion recognition deficits (Robert et al., 2014; Rosenberg, McDonald, Rosenberg, & Frederick Westbrook, 2016) have been found. Furthermore, deficits in self-awareness have been associated with impairments in ToM after TBI (Bivona et al., 2014), amyotrophic lateral sclerosis (van der Hulst et al., 2015), and schizophrenia (Pijnenborg, Spikman, Jeronimus, & Aleman, 2013). To conclude, studies in various, neurological, patient groups strongly suggest that impairments in social cognition underlie behavioural disturbances.

However, in aSAH, only few studies investigated deficits in social cognition. Brand et al. (2014) investigated empathy in a small sample of aSAH patients, finding impairments when compared to healthy controls. Furthermore, in a previous study we reported impaired emotion recognition after aSAH (Buunk et al., 2016). To date, there are no studies investigating to which extent there are impairments in a broad range of aspects of social cognition after aSAH, nor whether such impairments are related to behavioural disturbances, in particular deficits in interpersonal behaviour, apathy, and impaired self-awareness.

Because aSAH is likely to result in damage affecting frontal-subcortical circuits underlying social cognition, depending not only on aneurysm location, but also due to treatment modalities and additional aSAH-related events such as vasospasm (with possible delayed cerebral ischemia) and hydrocephalus, impairments in social cognition are likely to be found. The anterior communicating artery (ACoA) aneurysm accounts for almost 40% of all aneurysms, and a ruptured ACoA aneurysm can lead to ventromedial or unilateral frontal lobe lesions. Impairments in aspects of social cognition have been described in a few patients with an ACoA aneurysm (Heberlein, Padon, Gillihan, Farah, & Fellows, 2008). An early study showed that behavioural problems and personality changes were more likely to be found in aSAH patients with ACoA aneurysms (Storey, 1970). However, more recent studies found no relationship between aneurysm location and behavioural problems (Bottger, Prosiegel, Steiger, & Yassouridis, 1998; Hutter, Gilsbach, & Kreitschmann, 1995). Likely, not only focal (frontal) damage, but also diffuse neural injury post-aSAH disturbs important connections in the circuits supporting social cognition, but the relation between brain damage after aSAH and social cognition has not been investigated before.

In the present study, our aim was to investigate whether and to which extent a broad range of aspects of social cognition was impaired in the subacute stage post-aSAH. This period, 3 to 6 months after aSAH, is generally regarded as a clinically relevant moment to

evaluate SAH patients (Zweifel-Zehnder et al., 2015). To comprise different aspects of social cognition, we investigated both ‘hot’ social cognition, i.e. emotion recognition, and ‘cold’ social cognition, that is: ToM and cognitive empathy. Also, we investigated the relationship of deficits in these aspects of social cognition with behavioural problems, more specifically deficits in interpersonal behaviour, apathy, and impaired self-awareness. It is hypothesized that, in line with prior research in other groups and given the likelihood of damage to neural circuits that underlie social cognition post-aSAH, social cognition would be impaired and related to behavioural problems. Additionally, we aimed to clarify the relationship between deficits in social cognition and focal as well as diffuse brain damage, as this has not been done before. Therefore, both frontal and non-frontal cortical lesions on Magnetic Resonance Imaging, aneurysm circulation, treatment method (clipping or coiling) and additional aSAH-related events, like hydrocephalus (acute and/or chronic) and vasospasm, were taken into account. We expect that our findings will contribute to a better understanding of the nature of behavioural disturbances after aSAH.

2. Methods

2.1 Patients and procedure

All aSAH patients that were admitted to an University Medical Centre in the Netherlands between 2010 to 2012 were eligible for inclusion. Aneurysmal SAH diagnosis was determined on computed tomography (CT) on admission, in combination with CT angiography and/or digital subtraction angiography to confirm the presence of a symptomatic intracranial aneurysm. Patients were excluded in case of current or previous neurological conditions, psychiatric disorders, or substance abuse, age under 18 years, and insufficient proficiency of the Dutch language. Neuropsychological and MRI assessment were performed at approximately three to six months post-SAH. Healthy controls (HC) were either relatives of aSAH patients or recruited through an advertisement in a local newspaper. Exclusion criteria for HC were neurological conditions, psychiatric disorders, or substance abuse affecting cognitive functioning. The information on previous psychiatric disorders and neurological disorders was obtained by interviewing healthy controls before participation. Demographic characteristics of patients and healthy controls can be found in table 1. Furthermore, for comparison between patients' and proxies' scores on questionnaires, a group primarily made up of patients' spouses filled out informant ratings. The study protocol was approved by the Medical Ethical Committee (nr. 2009.164) of the University Medical Centre and was conducted in accordance with the Declaration of Helsinki. All participants gave a written informed consent.

2.2 Social cognition

2.2.1 Emotion recognition. The subtest Ekman 60 Faces Test of the Facial Expressions of Emotion – Stimuli and Tests (FEEST) (Young, Perrett, Calder, Sprengelmeyer, & Ekman, 2002) was used to assess emotion recognition. In this test, sixty faces expressing the emotions

Fear, Anger, Happiness, Surprise, Sadness and Disgust, are shown to the participant, who has to select which label best describes the emotion shown. Separate emotion scores range from 0 to 10, total scores from 0 to 60. The FEEST has shown reliability and validity (Young et al., 2002) and has been used in numerous studies assessing facial emotion recognition in neurological disorders (Calder, Keane, Manes, Antoun, & Young, 2000; Ietswaart, Milders, Crawford, Currie, & Scott, 2008; McDonald & Saunders, 2005; Sprengelmeyer et al., 1996).

2.2.2 Theory of Mind. The Cartoon Test (Happe et al., 1999) consists of 12 humorous cartoons, classified in two conditions: ToM and non-ToM. In the six ToM cartoons, the joke is related to what a character did not know or mistakenly thought. For example, one ToM cartoon is a picture of a man kissing a woman, while at the same time playing table tennis. He tries to deceive his mother, who sits in the adjacent room, by letting her think he is only playing table tennis. In the six non-ToM cartoons, the joke is based on a physical abnormality or violation of a social norm. An example of a non-ToM cartoon is a picture of a laboratory with one of the people shrunken, with the caption “Looks like Janssen found out something interesting”. Participants have to describe the humorous intention and their answers are scored from 0 (incorrect) to 3 (full and correct explanation) per cartoon, leading to a maximum total score of 36. The Cartoon Test has been used before in studies investigating ToM after ABI (Bibby & McDonald, 2005; Happe et al., 1999; Milders, Ietswaart, Crawford, & Currie, 2006) and effect sizes were moderate to large (Martin-Rodriguez & Leon-Carrion, 2010).

A shortened version of the Faux Pas Test (FP) (Stone, Baron-Cohen, & Knight, 1998) examines the ability to judge inappropriate behaviour in social situations. Ten short stories are read aloud by the experimenter, participants are allowed to (re-)read the stories. In half of the stories, a faux pas occurs: someone unintentionally says something awkward. Three

questions about the story are asked. First, a detection question is asked to discover the faux pas (“Did anyone say something awkward?”) and in case of an affirmative answer an identification question (“Who said something awkward?”) is asked. If both the detection and identification question are correct, this determines the FP-Detection scores (range 0 to 10). Lastly, only in faux pas situations, an affective question is asked (“How do you think Jeanette felt?”), to examine the empathic understanding of how the victim of the faux pas would feel. These answers form the FP-Empathy scores (range 0 to 5), providing an index for cognitive empathy (Shamay-Tsoory & Aharon-Peretz, 2007). Higher scores indicate better performance. The Faux Pas Test has been previously used to investigate ToM in neurological populations (e.g. Bibby & McDonald, 2005; Cox et al., 2016; Geraci, Surian, Ferraro, & Cantagallo, 2010; Milders et al., 2006; Stone, Baron-Cohen, Calder, Keane, & Young, 2003). The psychometric properties of the Faux Pas Test have not been officially evaluated, but it has been demonstrated to discriminate between patients with social cognitive impairments and typical peers (Gregory et al., 2002).

2.3 Behavioural problems

2.3.1 Apathy

The Apathy Evaluation Scale (AES) (Marin et al., 1991) is an 18-item scale that quantifies apathy. Items (for example “She/he gets things done during the day” and “She/he has initiative”) are rated on a 4-point scale, resulting in total scores between 18 and 72. Higher scores represent more severe apathy and a cut-off score of ≥ 34 was used for the diagnosis of apathy (Kant, Duffy, & Pivovarnik, 1998). Total scores of the AES self (AES-S) and informant (AES-I) version, to be completed by a partner or relative, were used. This scale has been evaluated in a group of neurological patients, including stroke patients, showing substantial support for the reliability and validity to evaluate apathy (Marin et al., 1991).

2.3.2 Deficits in interpersonal behaviour.

A subscale of the Patient Competency Rating Scale (PCRS) (Prigatano, Fordyce, & Zeiner, 1986) was used to measure deficits in interpersonal behaviour: PCRS-interpersonal and emotional (PCRS-IPE, range 0-40) (Sveen, Bautz-Holter, Sandvik, Alvsaker, & Roe, 2010). A sample item is: “How much of a problem do you have in recognizing when something you say or do has upset someone else?” Additionally, the total score (range 0-116) of the PCRS was used, measuring problems in daily activities, including deficits in psychosocial situations. For example, patients are asked: “How much of a problem do you have in consistently meeting your daily responsibilities?” One of its questions, concerning driving ability, was not taken into account, due to the fact that by Dutch road traffic law, SAH patients are not allowed to drive until 6 months post-SAH. Both a self-evaluation version (PCRS-Patient) and a proxy version (PCRS-Proxy) were administered. The ability to perform activities was scored from easy (0) to cannot do (4). For analysis, a dichotomy was used: ‘no problems’ (≤ 29 and ≤ 10 for PCRS and PCRS-IPE respectively) and ‘problems’ (> 29 and > 10 for PCRS and PCRS-IPE respectively). Both versions of the PCRS have a high internal consistency and test-retest reliability (Fleming, Strong, & Ashton, 1996; Prigatano & Altman, 1990).

2.3.3 Self-awareness

The PCRS has been extensively used to measure self-awareness after ABI, by using discrepancies between patients and proxy reports (e.g. Noe et al., 2005; Prigatano et al., 1998; Wallace & Bogner, 2000). Secondly, the convergent validity coefficients of the different AES versions imply that one version of the AES could be used as an alternative for the other (Marin et al., 1991). Consequently, significant differences between the patient and informant version can be interpreted as impaired awareness of problems. Self-awareness was investigated by using the difference scores of the AES ($\text{AES-Dif} = \text{AES-S} - \text{AES-I}$) and

of the PCRS (PCRS-Dif = PCRS-Patient minus PCRS-Proxy). Furthermore, since the structures of these questionnaires allow global minimal differences while still showing significant discrepancies on each question, interrater agreement scores (Kappa, κ) between patient- and proxy-ratings on the PCRS and AES were used. A negative difference score or a weak interrater agreement between patients and proxies was interpreted as an indication of impaired self-awareness.

2.4 Imaging data and SAH-related events

The MRI scans were made on a Siemens 1.5 Tesla system with a regular head coil. The scan sequences comprised the following transverse and coronal T2 weighted turbo spin-echo (T2 TSE) sequences: TR/TE 6360/111 ms, slice thickness 4 mm, matrix 512x (368 – 424) pixels. The Fluid-Attenuated Inversion Recovery (FLAIR) sequences comprised the following: TR/TI/TE 9520/2500/119 ms, slice thickness 4 mm, matrix 512x (368 - 416) pixels. Cortical infarctions in frontal areas (FrCLs) were defined by Brodmann Areas (BA) A4, 6, 8-12, 24, 25, 32, 44-47 taken together and cortical infarction in non-frontal areas (nFrCLs) by the remaining BA taken together (Martin, 2003). These infarctions were registered by experienced neuroradiologists (LCM and PL), blinded to the study's hypotheses and results. Aneurysms were divided in anterior circulation (anterior communicating artery, anterior cerebral artery, middle cerebral artery, posterior communicating artery, internal carotid artery, ophthalmic artery, and anterior choroidal artery aneurysms) and posterior circulation (basilar artery, posterior cerebral artery, superior cerebellar artery, and vertebral artery aneurysms), thereby including all aneurysm sites present in our patient group.

All patients with acute symptomatic hydrocephalus were treated with temporary external ventricular drainage or external lumbar drainage. Hydrocephalus that persisted beyond the acute stage was treated with ventriculo-peritoneal (VP) shunting. Vasospasm was monitored

using TransCranial Doppler sonography (TCD), with a Lindegaard ratio (Lindegaard, Nornes, Bakke, Sorteberg, & Nakstad, 1989) of greater than 3 being indicative for vasospasm, irrespective of the presence or absence of (related) neurological signs or symptoms.

2.5 Data analysis

Analyses were performed with the Statistical Package for the Social Sciences (SPSS^a) version 23.0. The Dutch classification system of Verhage was used to describe educational level (Verhage, 1964), ranging from 1 (primary school) up to 7 (university), dichotomized in lower (1-3) and higher (4-7) education. Clinical condition on admission was recorded using the World Federation of Neurological Surgeons (Teasdale et al., 1988) score, which was dichotomized in low (1-3) and high (4-5). To test for differences on social cognition tests between patients and HC, t-tests and, in case of not-normally distributed data, Mann-Whitney U tests were used. Effect sizes were calculated for all between group comparisons (Cohen's *d*). Pearson correlations were used to investigate relationships between social cognition and behavioural problems. Kappa (κ) was used to test interrater agreement of patients and proxies on the PCRS and AES, whereby values of <0.60 were interpreted as weak, ≥ 0.60 as moderate, and ≥ 0.80 as strong, according to guidelines for clinical research studies (McHugh, 2012). Spearman correlations and between-group differences were calculated to detect associations between social cognition tests and cortical lesions on MRI, aneurysm circulation, and aSAH-related events (hydrocephalus and vasospasm). For all analyses, an alpha level of 0.05 was set and Bonferroni-Holm corrections were used in case of multiple comparisons.

3. Results

In a two-year period, 148 aSAH patients were admitted to the University Medical Centre. Of these patients, 15 died in the hospital. Of the remaining 133 patients, 45 patients were approached but declined to participate or could not participate because their clinical condition did not allow for a detailed neuropsychological evaluation. Therefore, 88 patients were included in this study.

Table 1 shows the demographic characteristics of 88 aSAH patients and 60 healthy controls. Patients and healthy controls were matched on age ($p = 0.72$, $d = 0.05$), sex ($p = 0.6$), and educational level ($p = 0.09$, $d = 0.29$). Also, SAH characteristics are listed in table 1: aneurysm location, severity of the SAH, treatment methods, and SAH-related events (hydrocephalus and vasospasm).

Table 1. Demographic and clinical characteristics of aSAH patients and healthy controls

	Patients N = 88	Controls N = 60
Demographic characteristics		
Sex, number of women N (%)	61 (69.3%)	39 (65%)
Age in years (M \pm SD)	53.3 \pm 9.8	52.8 \pm 11.6
Educational level (M \pm SD)	5 \pm 1	5 \pm 1
SAH characteristics		
Time at evaluation since SAH in months (M \pm SD)	4.7 \pm 1.8	
Aneurysm circulation		
Anterior	69 (78.4%)	

Posterior	19 (21.6%)
WFNS grade	
Low (1-3)	70 (79.5%)
High (4-5)	18 (20.5%)
Treatment	
Clipping	22 (25%)
Coiling	64 (72.7%)
Other/none	2 (2.2%)
Hydrocephalus	
Acute (ELD/EVD)	64 (72.7%)
Chronic (VP shunt after ELD/EVD)	18 (20.5%)
Vasospasm on TCD	66 (75%)

SAH, subarachnoid haemorrhage; aSAH, aneurysmal subarachnoid haemorrhage; WFNS, World Federation of Neurological Surgeons; MRI, magnetic resonance imaging; ELD, external lumbar drainage; EVD, external ventricular drainage; VP shunt, ventriculo-peritoneal shunt; TCD, TransCranial Doppler sonography.

3.1 Social cognition

Table 2 shows the results of Mann-Whitney U and t-tests for differences between aSAH patients and HC on measures of social cognition. Significant differences were found on measures for emotion recognition (FEEST), ToM (Cartoon Test), and cognitive empathy (FP Empathy). Effect sizes of the significant differences between aSAH patients and HC were moderate to large. The differences on the ToM scales of the Cartoon Test remained significant ($p < 0.001$) after covarying for scores on the non-ToM scales.

Table 2. Scores on social cognition tests and comparisons of results between healthy controls and aSAH patients

	Controls	aSAH patients	t / U	p	Cohen's d
Social cognition tests M(SD)					
FEEST	49.0 (3.9)	45.7 (6.7)	3.70	0.001*	0.58
Cartoon Test	23.4 (6.1)	18.4 (6.1)	4.81	<0.001*	0.83
ToM	12.6 (4.0)	9.1 (3.7)	5.29	<0.001*	0.92
Non-ToM	11.1 (3.0)	9.5 (3.2)	2.97	0.003*	0.47
FP-Detection †	9.0 (1.0)	8.7 (1.4)	2250.5	0.113	0.24
FP-Empathy	2.5 (1.2)	1.9 (1.3)	3.10	0.002*	0.48

* significant after Bonferroni-Holm correction, † Mann-Whitney U tests were used to compare the results

aSAH, aneurysmal subarachnoid haemorrhage; FEEST, Facial Expressions of Emotion – Stimuli and Tests; ToM, Theory of Mind; FP, Faux Pas.

3.2 Behavioural problems

3.2.1 Apathy. Apathy (AES score ≥ 34) was reported by 42% of the patients and by 33% of their proxies. Table 3 shows the Pearson correlations between social cognition tests and the questionnaires, self and proxy versions. No significant correlations were found between any of the social cognition measures and AES-S scores. However, significant but moderate negative correlations were found between the FEEST and AES-I scores; a higher score on the FEEST was related to a lower level of apathy as rated by relevant others. The same was true for the Cartoon Test; a better score with respect to ToM was associated with a lower level of apathy as mentioned by proxies.

3.2.2 Deficits in interpersonal behaviour

Interpersonal behaviour was rated as impaired (PCRS-IPE score >10) by 52.3% of the patients and by 37.5% of their proxies. As shown in table 3, a significant correlation was found between the FP-Detection scores and PCRS-IPE-Proxy scores, indicating a relation between poor ToM and proxy reported deficits in interpersonal behaviour. Furthermore, 37.5% of the patients and 34.1% of their proxies had a PCRS score above cut-off (> 29). No significant correlations were found between measures for social cognition and PCRS self-ratings. However, the FEEST, Cartoon Test and FP-Detection scores correlated significantly but moderately with PCRS-Proxy scores; better emotion recognition and ToM were related to less daily life problems as reported by proxies.

Significant and moderate correlations were found between the AES-S and PCRS, self and proxy version ($r = 0.55$ and 0.43 resp., all $ps < 0.05$) and AES-I and PCRS self and proxy versions ($r = 0.39$ and 0.61 respectively, all $ps < 0.05$).

Table 3. Pearson correlations between tests for social cognition and questionnaires for behavioural problems

	AES-S	AES-I	AES-Dif	PCRS-Patient	PCRS-Proxy	PCRS-Dif	PCRS-IPE-Patient	PCRS-IPE-Proxy
FEEST	-0.07 (0.50)	-0.31* (0.004)	0.26* (0.01)	-0.12 (0.33)	-0.35* (0.004)	0.35* (0.007)	0.02 (0.85)	-0.28 (0.02)
Cartoon Test	-0.04 (0.7)	-0.28* (0.01)	0.26* (0.02)	-0.08 (0.48)	-0.30* (0.02)	0.31* (0.02)	-0.01 (0.91)	-0.27 (0.03)

FP-Detection	-0.17	-0.20	0.04	-0.23	-0.34*	0.13	-0.16	-0.32*
	(0.1)	(0.07)	(0.74)	(0.05)	(0.005)	(0.32)	(0.16)	(0.006)
FP-Empathy	0.03	-0.17	0.21	-0.08	-0.24	0.17	-0.02	-0.22
	(0.76)	(0.12)	(0.05)	(0.48)	(0.06)	(0.19)	(0.88)	(0.08)

* significant after Bonferroni-Holm correction. P-values are included in brackets.

FEEST, Facial Expressions of Emotion – Stimuli and Tests; FP, Faux Pas; AES-S, Apathy Evaluation Scale patient version; AES-I, Apathy Evaluation Scale proxy version; AES-Dif, Apathy Evaluation Scale Difference score (patient – proxy); PCRS, Patient Competency Rating Scale; PCRS-Dif, Patient Competency Rating Scale Difference score (patient – proxy); PCRS-IPE, Patient Competency Rating Scale-Interpersonal and Emotional.

3.2.3 Self-awareness

Mean scores on the AES did not differ significantly between patients ($M = 31.4$, $SD = 8.6$) and their proxies ($M = 31.2$, $SD = 9.2$, $t(81) = -0.26$, $p > 0.05$), and neither regarding the PCRS total score ($M_{\text{PCRS-Patient}} = 27.7$, $SD_{\text{PCRS-Patient}} = 19.5$, $M_{\text{PCRS-Proxy}} = 25.8$, $SD_{\text{PCRS-Proxy}} = 20.9$, $t(58) = 0.89$, $p > 0.05$) and the PCRS-IPE score ($M_{\text{PCRS-IPE-Patient}} = 12.7$, $SD_{\text{PCRS-IPE-Patient}} = 7.7$, $M_{\text{PCRS-IPE-Proxy}} = 11.1$, $SD_{\text{PCRS-IPE-Proxy}} = 7.7$, $t(68) = 1.74$, $p > 0.05$). However, there was a weak agreement between patients' and proxies' judgments of apathy ($\kappa = 0.37$, $p < 0.05$), as well as for the PCRS total scores ($\kappa = 0.44$) and PCRS-IPE scores ($\kappa = 0.31$), $ps < 0.05$. As shown in table 3, significant but moderate positive correlations were found between the FEEST and Cartoon Test scores and the PCRS-Dif and AES-Dif scores. Thus, lower emotion recognition and ToM scores were associated with poorer self-awareness.

3.3 Imaging data and SAH-related events

MRI data (3-6 months post-aSAH) were available from 81 patients, FrCLs were found in 33.3% of these patients and nFrCLs were found in 44.4%. Table 4 and 5 show no differences between patients with and without as well FrCLs as nFrCLs on tests of social cognition. Spearman correlations between both frontal and non-frontal cortical lesions on MRI and social cognition tests were not significant. FP-Empathy scores were significantly lower in patients with an aneurysm in the posterior circulation ($M = 1.1$, $SD = 1.1$) than in patients with an aneurysm in the anterior circulation ($M = 2.1$, $SD = 1.3$), $U = 405.5$, $p = 0.009$. No significant differences were found considering FEEST, FP-Detection, and Cartoon Test scores between the different aneurysm circulations.

No significant differences were found between social cognition scores of clipped and coiled patients (all $ps > 0.05$), between patients with and without vasospasm on TCD (all $ps > 0.05$) or between patients with and without external drainage for hydrocephalus (all $ps > 0.05$). Patients with a VP shunt for hydrocephalus performed significantly worse on the FEEST ($M = 41.3$, $SD = 6.4$) than patients without a VP shunt ($M = 46.8$, $SD = 6.4$), $t(86) = 3.19$, $p = 0.002$. No significant differences were found between patients with and without a VP shunt on other measures of social cognition.

Table 4. Comparison of results on social cognition tests between patients with and without frontal cortical lesions on MRI

Patients without	Patients with	t/U	P	Cohen's	Spearman
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	FrCL	FrCL			d	correlatio
	N = 54	N = 27				n with FrCL
FEEST	45.5 (6.9)	45.8 (6.7)	-0.24	0.8	0.04	0.01 (0.90)
Cartoon Test	18.5 (6.3)	18.7 (5.5)	-0.15	0.8	0.03	-0.06 (0.59)
ToM	9.4 (3.7)	9.1 (3.3)	0.38	0.7	0.09	-0.09 (0.42)
Non-ToM	9.2 (3.2)	10.0 (3.1)	-1.07	0.2	0.25	0.05 (0.68)
FP-	8.8 (1.4)	8.5 (1.5)	568.	0.1	0.21	-0.21 (0.07)
Detection*			5	0		
FP-Empathy	1.9 (1.4)	1.9 (1.2)	0.06	0.9	0.00	0.05 (0.69)

* Mann-Whitney U tests were used to compare the results. P values for Spearman correlations are included in brackets.

MRI, magnetic resonance imaging; FEEST, Facial Expressions of Emotion – Stimuli and Tests; ToM, Theory of Mind; FP, Faux Pas; FrCL, frontal cortical lesions.

Table 5. Comparison of results on social cognition tests between patients with and without non-frontal cortical lesions on MRI

	Patients without nFrCL N = 45	Patients with nFrCL N = 36	t/U	P	Cohen's d	Spearman correlation with nFrCL
FEEST	46.3 (6.7)	44.7 (6.9)	1.07	0.29	0.24	-0.15 (0.17)
Cartoon Test	18.3 (6.5)	18.9 (5.4)	-0.4	0.69	0.10	0.01 (0.95)
ToM	9.1 (3.9)	9.5 (3.2)	-0.54	0.60	0.11	-0.001 (0.99)
Non-ToM	9.5 (3.3)	9.5 (2.9)	0.01	0.00	0.06	-0.02 (0.86)
FP- Detection*	8.8 (1.6)	8.7 (1.2)	652	0.12	0.07	-0.20 (0.07)
FP-Empathy	2.1 (1.4)	1.6 (1.1)	1.9	0.06	0.41	-0.16 (0.16)

* Mann-Whitney U tests were used to compare the results. P values for Spearman correlations are included in brackets.

MRI, magnetic resonance imaging; FEEST, Facial Expressions of Emotion – Stimuli and Tests; ToM, Theory of Mind; FP, Faux Pas; nFrCL, non-frontal cortical lesions.

4. Discussion

To date, this is the first study that investigated a broad range of social cognition aspects after aSAH, demonstrating deficits in emotion recognition, ToM, and cognitive empathy. Moreover, this study is the first to find a relation between social cognition impairments and deficits in interpersonal behaviour, apathy and impaired self-awareness, as based on reports by patients' relevant others. These findings strengthen the assumption that social cognitive deficits underlie those behavioural problems that are frequently found post-aSAH.

In the present study, patients with aSAH performed significantly worse than healthy controls on most measures of emotion recognition, ToM and cognitive empathy. Effect sizes were moderate for empathy and large for ToM and emotion recognition, indicating that the differences were substantial. So far, only one previous study investigated empathy in a relatively small sample of patients, but still found impairments (Brand et al., 2014). We have previously reported emotion recognition deficits in the present patient group (Buunk et al., 2016). For the current study, we extended previous research by investigating multiple aspects of social cognition and found impairments in all aspects.

Social cognition has been intensively investigated in patients after TBI and in different subtypes of stroke. These studies consistently reported deficits in emotion recognition, ToM and empathy (Happe et al., 1999; Spikman, Timmerman, Milders, Veenstra, & van der Naalt, 2012). Also, impairments in ToM and emotion recognition have been described in a few cases of patients with ventromedial prefrontal brain lesions due to a ruptured ACoA aneurysm (Heberlein et al., 2008). Furthermore, a relation was found between social cognitive deficits and behavioural problems in TBI and stroke studies (Blonder et al., 2012; Spikman et al., 2012), and in line with this, similar results were expected in patients who have suffered an aSAH. This is the first study that investigated the relationship between social cognition and behavioural problems, specifically deficits in interpersonal behaviour, apathy, and impaired self-awareness, following aSAH. Apathy and impaired interpersonal behaviour were reported by more than one third of all patients and proxies, consistent with previous studies after aSAH (Caeiro, Santos, Ferro, & Figueira, 2011). Moreover, we found associations of impaired emotion recognition and ToM with apathy as reported by proxies. It is likely that social cognitive deficits, affecting the abilities to recognize behaviour and understand intentions of others, hamper patients to respond adequately to social interactions, resulting in loss of interest in social life and eventually withdrawal. Both loss of interest and

withdrawal are important characteristics of apathy. Furthermore, an association between ToM and deficits in interpersonal behaviour was found. A possible explanation for this result is that the ability to form a ToM is needed to maintain appropriate behaviour. This explanation seems to be consistent with previous research in paediatric TBI patients, that shows a prospective association between subacute ToM deficits and chronic behavioural disturbances (Ryan et al., 2016).

Interestingly, the relationship with social cognitive deficits was only found for behavioural problems as rated by proxies. Hence, likely this implies problems in self-awareness of these aspects in aSAH patients. While mean rating scores of patients were statistically indistinguishable from those of their relevant others, interrater agreement between patient and proxy-ratings was weak. These findings strongly point to aSAH patients having a limited insight into their own condition. Consequently, proxy-ratings display the actual level of behavioural problems of aSAH patients more precisely than patient ratings. Moreover, this is the first study to find an association between impaired self-awareness, as expressed in the difference score of the questionnaires, and deficits in social cognition following aSAH. These results are consistent with previous studies which showed an association between social cognitive deficits and impaired self-awareness in TBI patients (Spikman et al., 2013) and schizophrenia (Pijnenborg et al., 2013). We used discrepancy scores between patients' self-ratings and proxy-ratings (eg, family member, partner), to measure self-awareness. A possible shortcoming of this method is the inaccuracy of the relevant other to assess the behavioural problems of the patient (Maclean, Pound, Wolfe, & Rudd, 2000; Sherer, Hart, & Nick, 2003). However, comparison of the patient's self-rating with that of relevant others is the most commonly used approach to measure self-awareness after stroke (Al Banna, Redha, Abdulla, Nair, & Donnellan, 2016; Leung & Liu, 2011) and TBI (Kelley et al., 2014; Prigatano, 2005). Moreover, in our study only ratings from relevant

others, not self-ratings, were correlated with performance on social cognition tasks, validating the accuracy of proxy-ratings. From a clinical point of view, the possible causal relationship between self-awareness and social cognition impairments is interesting. Possibly, patients with low self-awareness are less capable to perceive their own cognitive, emotional, and behavioural changes and consequently, are less able to take another person's perspective. Another possibility is that social cognitive deficits hinder patients to use social feedback that could help in analysing their own behavioural changes. Moreover, our results suggest that using self-report measures alone is insufficient to detect behavioural problems in aSAH patients. Therefore, proxy-ratings and social cognitive tests should also be part of routine neuropsychological testing in this category of patients.

Lastly, we found no evidence for a relationship between social cognitive deficits and as well frontal as non-frontal cortical lesions on MRI. Furthermore, aneurysms in the posterior circulation were related to only one test of social cognition, measuring cognitive empathy. For the measures of emotion recognition and ToM, no relation was found with aneurysm circulation within our patient group. Lastly, treatment method and aSAH-related events (hydrocephalus and vasospasm) did not explain why and how social cognition impairments did occur after aSAH; only patients with a permanent shunt (for chronic hydrocephalus) performed worse on an emotion recognition task. Evidently, for the broad range of social cognition aspects, it does not matter whether aSAH-related events are present, whether there is an aneurysm in the anterior circulation or whether there is frontal damage visible on MRI. Frontal brain circuits are assumed to have an important role in social cognition (Adolphs, 2001) and damage in these areas has been related to impaired social cognition after TBI and stroke (Shamay-Tsoory, Tomer, Berger, Goldsher, & Aharon-Peretz, 2005; Spikman et al., 2012). However, in the event of aSAH, various pathophysiological mechanisms determine the ultimate neurological deficit, such as primary damage due to the spreading of

subarachnoid blood, the disruption of neural structures due to the rapid expansion of the ventricular system in acute hydrocephalus, and the secondary brain injury due to vasospasm related ischemia and perfusion disturbance following (acute) raised intracranial pressure. This is why outcome prediction based on lesion location (i.e. the site of the symptomatic aneurysm) alone in the event of an aSAH is inaccurate. Furthermore, the frontal cortex is part of a larger network sub-serving social behaviour, in which also other cortical and subcortical areas (such as the parietotemporal areas) participate (Adolphs, 2001). Consequently, lesions in all different areas that are part of these networks can affect social cognition. Ergo; a low correlation between focal (frontal) brain damage and social cognitive deficits does not necessarily mean that frontal networks are not involved in social cognition.

The present study is subject to some limitations. Firstly, the aSAH patients in our sample were those eligible for neuropsychological assessment. However, it is likely that patients who were not able to perform neuropsychological tests have even more serious social cognitive deficits. Secondly, since we have conducted correlation analyses, we are not allowed to infer a causal relation between social cognition impairments and behavioural problems. However, we deem it plausible that relatives observe problems in social behaviour as a consequence of the fact that the patients are not able to recognize their proxies' emotions or show empathy. The plausibility of this supposed causal direction is supported by findings of a positive effect of emotion recognition training on behavioural problems and proxy-ratings of socioemotional behaviour after ABI (Radice-Neumann, Zupan, Tomita, & Willer, 2009). Another factor limiting our analysis is the trouble in imaging and localizing post-haemorrhagic brain injury after aSAH. Although conventional MRI detects structural brain damage such as cortical infarctions, Diffusion Tensor Imaging (DTI) may provide more detailed insight in the integrity and connectivity of white matter tracts involved in networks connecting the frontal cortex with other cortical and subcortical areas. Also, susceptibility weighted imaging (SWI)

is a useful technique in detecting micro-bleeds (Liu et al., 2017; Naik, Viswamitra, Kumar, & Srinath, 2014; Ryan et al., 2015) and diffuse neuropathology detected with SWI has been related to ToM deficits in paediatric TBI patients (Ryan et al., 2015). Unfortunately, DTI and SWI sequences were not part of the imaging protocol during the time of our study. DTI and other advanced MRI techniques may prove to be useful in predicting cognitive dysfunctions after aSAH, and should be taken into account in future studies. Furthermore, more recent studies underline the importance of distinguishing between vasospasm (on TCD and/or angiography) and delayed cerebral ischemia (DCI), proven by cerebral infarction on imaging (Vergouwen et al., 2010; Vergouwen & Participants in the International Multi-Disciplinary Consensus Conference on the Critical Care Management of Subarachnoid Hemorrhage, 2011). DCI is associated with worse neurological outcome (Dorhout Mees, Kerr, Rinkel, Algra, & Molyneux, 2012) and general cognitive functioning (Stienen et al., 2014), thus the relationship between DCI and social cognition after aSAH could be of interest in future studies.

In conclusion, our study shows that social cognition is impaired following aSAH and is related to deficits in interpersonal behaviour, apathy, and impaired self-awareness as indicated by relevant others. Social cognition impairments after ABI are often very disabling, causing severe problems in relationships with patients' relatives and caregivers (Koskinen, 1998), return to daily activities and rehabilitation processes (Radice-Neumann et al., 2007; Ubukata et al., 2014). Considering the importance of social cognition for adequate daily life and psychosocial functioning, neuropsychological assessments after aSAH should include social cognition tests for all patients, irrespective of the location of cortical lesions as seen on MRI or aneurysm circulation. Future research may focus on the effect of damage to connecting networks in the brain using advanced MRI techniques such as DTI and SWI. In addition, the association between social cognitive deficits and behavioural problems was

found for proxy-ratings alone, indicating limited self-awareness in aSAH patients. Impaired self-awareness has been found to affect daily functioning and quality of life after ABI (Prigatano, Borgaro, Baker, & Wethe, 2005; Spikman & van der Naalt, 2010). Therefore, we recommend the incorporation of proxy-ratings in clinical practice to enhance understanding of the behavioural disturbances that aSAH patients may encounter. Possibly, patients and their relatives may benefit from interventions aimed at social cognitive deficits, improving social outcome after aSAH.

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Highlights

- Aspects of social cognition (emotion recognition, ToM, and empathy) were impaired post-aSAH.
- Social cognition deficits were associated to behavioural disturbances after aSAH.
- An association between impaired self-awareness and deficits in social cognition was found.
- Social cognitive deficits were not related to lesion location on MRI or aneurysm circulation.