Deficits in decision-making in patients with aneurysms of the anterior communicating artery

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Summary

Patients with aneurysmal subarachnoid haemorrhage (SAH) secondary to ruptured anterior communicating artery aneurysms (ACoA) often suffer from neuropsychological sequelae including personality and behavioural changes. In this study, 31 patients with ruptured aneurysms of the ACoA resulting in SAH [mean age 50.9 years, NART (National Adult Reading Test) IQ 108.7] were compared with a group of 29 normal controls (mean age 51.9 years, NART IQ 109.7) on a specific task of decision-making. A similar task has been imaged previously and shown to activate regions involving the ventromedial prefrontal cortex. Patients with ACoA rupture showed no significant difference from controls on the task in their speed or quality of decision-making. They did, however, exhibit increased risk-taking behaviour, placing higher bets in a measure of the task which involved choices between actions associated with differing magnitudes of reward and punishment. ACoA patients demonstrated true risk-taking behaviour as opposed to simple impulsivity. Such a deficit in decision-making may be a result of direct damage to the orbital prefrontal cortex itself (as a result of micro-ischaemia or infarction after ACoA aneurysmal rupture) or to a disconnection in the ventromedial circuits from distant or generalized brain damage.

Keywords: subarachnoid haemorrhage; anterior communicating artery; decision-making; cognitive deficits

Abbreviations: ACoA = anterior communicating artery aneurysm; ANOVA = analysis of variance; CANTAB = Cambridge Neuropsychological Test Automated Battery; NART = National Adult Reading Test; SAH = subarachnoid haemorrhage

Introduction

Patients with subarachnoid haemorrhage (SAH) due to ruptured aneurysms of the anterior communicating artery (ACoA) have historically been observed to suffer from a poor neuropsychological outcome (Walton, 1956; Storey, 1967; Logue et al., 1968; Sonesson et al., 1987). Accounts of such patients classically outline aspects of a triad of symptoms known as the ‘ACoA syndrome’: memory loss, confabulation and altered personality (Lindqvist and Norlen, 1966; Logue et al., 1968; Sengupta et al., 1975; Okawa et al., 1980; Gade, 1982; Alexander and Freedman, 1984; Damasio et al., 1985; Vilki, 1985; Steinman and Bigler, 1986; Laiacona et al., 1989; Stenhouse et al., 1991; DeLuca, 1992). In recent times, the more dramatic changes associated with the full-blown ACoA syndrome have been seen with less frequency because of improved surgical techniques and other therapeutic interventions (Eslinger and Damasio, 1984; Teissier du Cros and Lhermitte, 1984). Nevertheless, a similar set of symptoms, often of reduced severity, may still be observed in modern-day patients with ACoA aneurysmal rupture. For example, many such patients, in addition to memory problems of varying severity, continue to exhibit the described personality alterations, such as impulsivity, disinhibited behaviour, apathy, emotional lability, depression, problems in decision-making, organizational difficulties and poor judgement in social situations (Okawa et al., 1980; Alexander and Freedman 1984; Eslinger and Damasio 1984; Irle et al., 1992; Ogden et al., 1997). These changes in aspects of memory, emotionality and judgement, which appear to be chronic and resistant to change (Steinman and Bigler, 1986), continue to have unfortunate consequences for patients and their families. Loss of job, financial difficulties and changes in interpersonal relationships, e.g. divorce, are among the sequelae not infrequently reported (Ljunggren et al., 1985; Saveland et al., 1986; Ogden et al., 1990, 1994; Vilki et al., 1990; Hutter et al., 1995). For this reason, the study of functional changes in post-SAH patients continues to be important.
The neuropathological bases for the memory and personality changes in SAH secondary to ACoA rupture remain a matter of debate. The range of symptoms and its similarity to that suffered by patients with prefrontal lobe damage has for many years led researchers to suggest ischaemic damage to the frontal lobes and the ‘frontal dysexecutive syndrome’ as a possible explanation for the sequelae of ACoA rupture, in particular for the personality changes observed (Storey, 1967; Logue et al., 1968; Eslinger and Damasio, 1984; Viliki, 1985; Parkin et al., 1988; Stenhouse et al., 1991). Damage to the frontal lobes would be a plausible effect of ischaemia or infarction of cortical areas in the territory of the perforating branches of the ACoA (Alexander and Freedman, 1984; Damasio et al., 1985). Studies of ACoA rupture patients, however, have not invariably demonstrated a ‘frontal lobe syndrome’, tests of frontal lobe function often being found to be unaffected (Ahola et al., 1996). For example, not all studies have found a group deficit in the Wisconsin Card Sorting Task, a classical test of executive function (e.g. McKenna et al., 1989; Shoqerir et al 1990; Anderson et al., 1991; Rousseaux et al., 1996). In our recent paper on 47 patients with ACoA (Mavaddat et al., 1999), using the Cambridge Neuropsychological Test Automated Battery (CANTAB) (CeNeS Ltd, Cambridge, UK), which has the ability to localize dysfunction and has been validated extensively using groups of neurosurgical patients with either frontal or temporal lobe excisions, we found normal performance compared with controls matched for age and National Adult Reading Test (NART) IQ on tests of spatial span (Milner, 1971; Owen et al., 1990), spatial recognition memory (Sahakian et al., 1988), the Tower of London test of planning (Shallice, 1982; Owen et al., 1990) and Attentional Set Shifting (Grant and Berg, 1948; Sahakian et al., 1990), all of which have been shown previously to be impaired in patients with frontal lobe lesions (Owen et al., 1991; Sahakian and Owen, 1992). In addition, while patients performed poorly compared with controls on a test of spatial working memory, a test sensitive to both frontal and temporal lobe dysfunction, they made no more within-search errors (i.e. looking back into a spatial location previously searched for a hidden token in the same trial), nor did they use an inefficient strategy, which is normally associated with damage to the prefrontal cortex (Owen et al., 1990).

The paucity of findings in a number of studies in the ACoA rupture group in tests classically sensitive to frontal lobe dysfunction may suggest that frontal lobe pathology does not contribute significantly to the symptoms that have been described in these patients, or may support the possibility of resolution of any frontal deficits in the early postoperative phase (D’Esposito et al., 1996). Alternatively, the lack of uniform findings of frontal deficits may confirm the growing recognition that patients with a damaged frontal lobe may suffer from organisational difficulties in daily life and yet do well in standard frontal tests (Eslinger and Damasio, 1985; Shallice and Burgess, 1991a; Ahola et al., 1996). Damasio proposes that such patients suffer symptoms secondary to lesions of the ventromedial or orbital prefrontal cortex, while most classical frontal tests tap into functions subserved by the dorsolateral prefrontal cortex (Damasio, 1996).

Recently, it has become possible to dissociate functionally between the two areas of the prefrontal cortex. Bechara and colleagues found that a small group of patients with bilateral lesions of the ventromedial prefrontal cortex (some of whom had suffered ACoA rupture) performed normally in standard frontal tests of executive and mnemonic function but were impaired in a test which models aspects of real-life decision-making (Bechara et al., 1994). In this test, Bechara and colleagues asked subjects to make selections from four decks of playing cards. Each card selected was associated with a certain reward or penalty, in the form of facsimile money. Two of the four packs were associated with high rewards (penalties) and penalties, and two with small rewards and penalties. They found that patients who had damage to their ventromedial cortex drew more frequently from packs with high rewards and penalties compared with normal controls and those with dorsolateral prefrontal lesions, who found the most productive strategy to be selecting from the packs with small rewards and penalties (Bechara et al., 1994). Thus, patients with bilateral ventromedial lesions took greater risks in their decision-making, which closely reflected their symptoms of poor social judgement and organizational difficulties.

In a study by Rogers and colleagues (Rogers et al., 1999a), a computerized decision-making task related to the gambling task of Bechara and colleagues (Bechara et al., 1994) was found to be impaired in a group of patients with orbital prefrontal lobe lesions but not dorsolateral prefrontal lesions. A similar task has been imaged and shown to involve a neural network that includes the inferior and orbital prefrontal cortex (Rogers et al., 1999b). In this task, subjects are required to make choices or decisions between contingencies that are presented visually. Several measurements are taken, including the speed of decision-making, the quality of the decision-making and a measure of risk-taking behaviour.

The area of cortex supplied by the anterior communicating artery and its perforators includes the orbital or ventromedial aspects of the frontal lobes and it is, therefore, this area that would be expected to be predominantly affected by any ischaemic damage subsequent to aneurysmal rupture. Thus, while the ‘classic frontal tests are not selectively sensitive to the mainly medial frontal (damage) which may follow the rupture of an anterior communicating artery’ (Ahola et al., 1996), anatomically we might predict impairment in a test designed to investigate specifically ventromedial prefrontal function. Having found no significant impairment in our group of ACoA rupture patients in a number of standard tests of frontal lobe function that are thought to require dorsolateral prefrontal activity for their performance, such as the Attentional Set Shifting task and the Tower of London task (which has been used in the functional imaging setting and shown to produce activation in regions including the
dorsolateral prefrontal cortex) (Baker et al., 1996), we have now studied the performance of our patients in a test dependent on the integrity of the ventromedial input, similar to that devised by Bechara and colleagues (Bechara et al., 1994). Any deficits found in this task would enable us to make inferences about the cause of the symptoms, particularly the personality changes, seen after SAH in these patients.

Methods
This study had ethical approval from the Cambridge Local Research Ethics Committee and consent was obtained according to the Declaration of Helsinki.

Patients
Adults below the age of 70 years achieving a favourable neurological recovery (Glasgow Outcome Scale score 4 or 5) after open surgery for a ruptured ACoA 6–24 months previously were identified from the Addenbrooke’s Neurosurgical SAH audit (Seeley et al., 1998). The neurosurgical management in all patients had followed a standardized protocol (Hutchinson et al., 1996; Whitfield et al., 1999; Mavaddat et al., 1999).

Of 47 patients identified [who had previously performed the CANTAB battery of tests (Mavaddat et al., 1999)], 31 returned to participate in the study. Of the 16 who did not take part, one was deceased, six declined to return and nine were no longer contactable. There were no differences in any demographic variables of interest between those who participated and those who did not. Twenty-nine control subjects were chosen to match the patients in age and NART IQ. These were drawn from a group of normal controls with no neurological or psychiatric history. Informed consent was obtained from all patients and control volunteers. The mean age and NART IQ was 50.87 years (SD 9.76) and 108.74 (SD 10.28), respectively, for the patient group and 51.9 years (SD 11.37) and 109.76 (SD 7.72) for the control group. There were 16 males and 15 females in the patient group, and 14 males and 15 females in the control group.

Neuropsychological assessment
Premorbid verbal IQ was measured using the NART (Nelson, 1982). Patients were asked to complete a Beck Depression Inventory (Beck, 1970), and family/carers a rating of the patient’s memory. N.M. completed the Neuropsychiatric Inventory (Cummings et al., 1994) for each patient.

Decision-making (Rogers et al., 1999a)
The decision-making task (gamble) was performed using a touch-screen computer (Datalux Touch Sensitive screen and Carry I portable hard disk). Subjects were seated ~0.5 m from the computer screen and asked to make responses by touching the screen with their dominant hand.

In this task the subject is shown a screen containing 10 boxes with differing ratios of blue to red boxes (Fig. 1). The subject is told that there is a yellow token that has been randomly hidden by the computer behind one of the red or blue boxes. The subject then has to decide whether they think the token is hidden behind a red or a blue box and register this decision by touching either the response panel marked ‘red’ or the response panel marked ‘blue’. After making this initial choice, the subject is asked to place a bet on this choice being correct. The available bets appear in sequence in a box positioned near the right-hand side of the display. Each bet is displayed for 5 s, before the appearance of the next bet in the sequence. The subject is asked to select any bet by touching the box in which the sequences appear. Immediately after the selection, the box hiding the yellow token opens to reveal its location. This is accompanied by either the message ‘You win!’ and a short rising musical scale or ‘You lose!’ and a low tone. If the subject chooses the correct colour the amount of bet is added to the total points score; if the subject chooses the wrong colour, the bet is subtracted.

The task is performed in two separate conditions: an ‘ascending’ condition, in which the first bet offered is small and is replaced by larger bets, and a ‘descending’ condition, in which the first bet is large and is replaced by smaller bets. Each bet represents a fixed percentage of the current total points score, although this is never made clear to the subject. Five bets are offered on each trial, so that in the ascending condition the order of available bets is 5, 25, 50, 75 and 95% of the available points, with the reverse order in the descending condition. In both conditions, each bet is presented together with a short tone whose pitch corresponds to the size of the bet: higher tones accompany larger bets and lower tones accompany lower bets. If the subject fails to select a bet by the end of a sequence, the last bet is chosen automatically. The subject is given an initial 100 points at the start of the sequence and is asked to increase this total by as much as possible. If the subject’s score falls to just one point, the current sequence ends and the next begins.

Three key measures for this task were analysed. (i) The speed of decision-making was measured by how long it took the subject to decide which colour of box was hiding the token. (ii) The quality of decisions was measured by how much of the time the subject chose the most likely outcome. By changing the ratio of red to blue boxes from trial to trial it was possible to examine more closely the subjects’ decision-making behaviour. For example, whereas a 4 : 6 ratio represents a more balanced contingency, a 9 : 1 ratio represents a clearer choice and the opportunity to bet more points. It would be more effective to choose the colour with the highest number of boxes represented; therefore, we measured how much of the time the subject chooses the most likely outcome. (iii) Risk adjustment was the measure of the rate at which a subject increased the percentage of the available points bet in response to more favourable ratios of red to blue boxes. The offering of bets in both ascending
and descending conditions provided an opportunity for determining whether the decision-making behaviour was based on impulsivity or risk-taking. If the subjects bet highly in both ascending and descending conditions, this could be seen as reflecting genuine risk-taking behaviour, while betting early in both the ascending and the descending condition would rather suggest impulsivity of action.

**Data analysis**

Data were analysed using the Statistical Package for the Social Sciences SPSS V8.0 (SPSS, Chicago, Ill., USA). Some data were transformed where appropriate (Tukey, 1977). The data shown in the figures always represents untransformed values. The principal measures were subjected to repeated measures analysis of variance (ANOVA) with the following between- and within-subject factors: group (ACoA versus controls), condition (ascending versus descending), decision (red versus blue) and ratio (6:4 versus 7:3 versus 8:2 versus 9:1). The proportion of trials on which subjects chose the most likely outcome (i.e. the quality of decision-making) was arcsin-transformed, as is particularly appropriate whenever the variance is proportional to the mean (Howell, 1997). In those instances where the additional assumption of homogeneity of covariance in repeated measures ANOVA was violated, as assessed using the Mauchly sphericity test, the number of degrees of freedom against which the F-ratio was tested was reduced by the value of the Greenhouse–Geisser epsilon (Howell, 1997). Pearson’s product moment correlation coefficients were calculated in the correlational analyses. Since multiple statistical comparisons were made, significance is reported at $P < 0.01$ and trends at $P < 0.05$.

**Results**

There were no significant differences between the groups in age, NART IQ and sex ratio [age, $F(1,58) = 0.141, P = 0.708$; NART IQ, $F(1,58) = 0.186, P = 0.668$; sex, $\chi^2 = 0.067, P = 0.796$].

**Speed of decision-making**

The mean deliberation times for deciding whether the yellow token was hidden behind a blue or red box for each ratio are
shown for the ACoA patient group and the control group in Fig. 2A. There was no significant difference in mean deliberation time between the ACoA and control groups \([F(1,58) = 1.564, P = 0.216]\). There was no effect of the ratio of coloured boxes on deliberation time \([F(2.2,128.7) = 0.046, P = 0.966]\) and no significant interaction between group and ratio \([F(2.2,128.7) = 1.887, P = 0.151]\).

**Quality of decision-making**
The mean percentage of choices of the most likely outcome are shown for the ACoA subjects and the controls in Fig. 2B. These were significantly different at varying ratios \([F(2.4,136.6) = 4.83, P = 0.006]\), the percentage of choices of the most likely outcome being increased at the more favourable compared with the less favourable ratios (for both groups: ratio 6 : 4, 88.7%; ratio 7 : 3, 91.1%; ratio 8 : 2, 93.1%; ratio 9 : 1, 93.3%). However, there were no significant differences between the ACoA group and the controls in their ability to make optimal choices \([F(1,58) = 0.394, P = 0.532]\). There was also no group \(\times\) ratio interaction \([F(2.4,136.6) = 1.287, P = 0.281]\). There was an effect of condition \([F(1,58) = 17.01, P = 0.0001]\), all subjects making better-quality decisions in the descending condition than in the ascending condition. This was probably due to practice effects as all subjects performed the ascending condition before the descending condition. There was, however, no group \(\times\) condition interaction \([F(1,58) = 0.946, P = 0.335]\).

**Risk adjustment**
The mean percentage of bet scores for the patient and control groups are shown in Fig. 2C. There was a significant effect of ratio, the amount bet increasing as a function of the ratio of red to blue boxes (for both groups: ratio 6 : 4, 41.6%; ratio 7 : 3, 50.9%; ratio 8 : 2, 58.2%; ratio 9 : 1, 61.5%). Patients with ruptured ACoA showed markedly increased bets at each ratio compared with the control group \([F(1,58) = 26.43, P = 0.0001]\) (controls: ratio 6 : 4, 33.4%; ratio 7 : 3, 42.3%; ratio 8 : 2, 49.9%; ratio 9 : 1, 52.6%; patients: ratio 6 : 4, 49.3%; ratio 7 : 3, 59.1%; ratio 8 : 2, 65.9%; ratio 9 : 1, 69.7%). This analysis was performed only for those trials in which subjects chose the most likely outcome as it was only in these trials that it was possible to assess the subjects’ response to the available opportunities to earn reward represented by different ratios. There was no group \(\times\) ratio interaction, patients betting higher regardless of the ratio of coloured boxes \([F(1.96,113.96) = 0.063, P = 0.937]\). All subjects placed larger bets in the descending than in the ascending condition \([F(1,58) = 37.254, P = 0.0001]\). There was no group \(\times\) condition interaction, indicating that the choices were not a consequence of impulsive behaviour alone \([F(1,58) = 0.155, P = 0.696]\). Figure 3 demonstrates that all but one of the 31 patients placed higher bets than the mean percentage of bets placed by the control group.
Correlations and other analyses

There was no correlation between performance on any measure of the decision-making task and depression as scored by the Beck Depression Inventory, the Neuropsychiatric Inventory, family/carer rating of memory or any tests of the CANTAB battery performed previously. There was also no correlation with the grade of haemorrhage or the Glasgow Outcome Scale score at discharge. Patients who had earlier surgery (within 3 days after ictus) performed better on a measure of the task than those who had later surgery (i.e. >3 days after ictus). An ANOVA showed a significant difference in performance between the early (n = 19) and late groups (n = 12) in the probability of choosing the most likely outcome \( F(1,29) = 6.466, P = 0.017 \). This clinically relevant finding is of especial significance in view of our previous study, which revealed no differences in performance for any of the background CANTAB tests between the two surgical groups (Mavaddat et al., 1999).

Discussion

This study shows that patients with rupture of an ACoA leading to SAH have specific deficits in their decision-making ability. Whereas in the computerized decision-making task developed by Rogers and colleagues (Rogers et al. 1999a), patients with ACoA rupture showed no significant difference from controls in the time it took them to make their choice of coloured box (speed of decision-making) and in their ability to choose the most likely outcome (quality of decision-making), they exhibited increased risk-taking behaviour in placing higher bets on their chosen colour of box. The decision of how much to bet involves choices between actions associated with differing magnitudes of reward and punishment and measures the willingness of patients to risk some of their already accumulated reward in the hope of earning yet more reinforcement. Our patients bet higher than controls at every ratio of the coloured boxes and placed these higher bets in both the ascending and the descending condition of the task. This supports true risk-taking behaviour as opposed to simple (motor) impulsivity. The study confirms that patients with ACoA rupture, like patients with ventromedial lesions of the prefrontal cortex and those with frontal-variant frontotemporal dementia (who have similar personality and behavioural changes), are impaired in decision-making. In the absence of confirmatory neuro-anatomical data on all patients, it seems likely that the impairments shown by the ACoA group were due to either direct damage to the orbital prefrontal cortex itself (as result of micro-ischaemia or infarction) or to a disconnection in the ventromedial circuits from distant or even generalized diffuse brain damage. The differences in measures of impaired decision-making between the ACoA group and each of the other two patient groups suggests differences in the extent and nature of network damage involved in each condition. Our ACoA rupture group, like the frontal variant frontotemporal dementia patients, showed no impairment in the quality of their decision-making and placed higher bets than their control counterparts (Rahman et al., 1999), but they did not show the statistically significant increased deliberation times of the other patient groups. However, on average they did
exhibit an increase in the time taken to make their choice, thus lending support to the argument that they were again not acting simply on impulse.

Rogers and colleagues point out that decision-making deficits that are attributed to damage to the orbital prefrontal cortex do not necessarily take the form of impulsive or disinhibited responding (Bechara et al., 1996) but are expressed as rather slow and ineffective deliberation about the conflicting options for action (Rogers et al., 1999b). These observations provide evidence that the orbital prefrontal cortex does not act in a unitary way as a simple inhibitory mechanism. Recent work has implicated the orbital prefrontal cortex as a site where emotional information is related to action selection, consistent with its rich interconnections with the limbic cortex. Damasio has suggested in his ‘somatic marker’ hypothesis that emotional influences via somatic markers signal a cognitive evaluation or reasoning response in networks involving the somatosensory cortex, amygdala and ventromedial frontal cortex (Damasio, 1996). His so-called somatic markers are signals that mark the ultimate consequence of the response option with a negative or positive somatic state or emotion, regardless of its predictable immediate reward or punishment (Damasio et al., 1991). After lesions of the ventromedial cortex, this emotional/somatic link is presumed to be defective, leaving the patient without the added benefit of his emotional reaction in guiding the decision-making process. This may explain why, for example in the decision-making task of Bechara and colleagues, normal subjects were found to choose advantageously before they realized which strategy worked best, whereas patients with ventromedial prefrontal damage continued to choose disadvantageously even after knowing the correct strategy (Bechara et al., 1997). In a similar vein, in Eysenck’s framework of normal versus psychopathological risk-taking behaviour, whereas the normal subject with high-risk behaviour would ‘feel the fear’ associated with the disadvantageous decision but perhaps carry on regardless, the patient with orbitofrontal damage would ‘feel no fear’ and choose disadvantageously to carrying out the ‘risky’ decision (Eysenck, 1993). An alternative explanation is that the uncoupling between cognitive and emotional factors results in the patient using only emotional information in the decision-making process (Plaisted and Sahakian, 1997). In whatever way the ventromedial cortex mediates the response reaction, clearly its dysfunction leads to poor decision-making ability beyond simple disinhibition.

This demonstration of poor decision-making and increased risk-taking behaviour in ACoA rupture patients, suggesting a dysfunction in orbitofrontal circuitry, goes some way to identifying a potential cause for the personality changes, emotional and judgemental deficiencies seen in ACoA rupture patients. However, it cannot explain the whole range of symptoms included in the ‘ACoA syndrome’. A recent point of interest has been the proposition that the memory loss and personality change in ACoA rupture patients may not be caused by a single area of damage, but is rather a result of damage to separate areas of the brain. DeLuca, for example, has proposed that, to have the full ‘anterior communicating artery syndrome’, damage both to the frontal areas and to the basal forebrain area is necessary—the so-called dual lesion hypothesis. Many researchers in the field currently agree that ischaemic damage (due to disruption of blood flow to one of the perforating arteries of the ACoA after aneurysmal rupture) to the basal forebrain cholinergic neurones and catecholamine pathways projecting to medial temporal structures (i.e. the hippocampus, amygdala and parahippocampal gyrus) may mediate the memory loss (Gade, 1982; Mesulam et al., 1983; Alexander and Freedman, 1984; Eslinger and Damasio, 1984; Volpe et al., 1984; Damasio et al., 1985; Vilikki, 1985; Phillips et al., 1987; DeLuca, 1993; DeLuca and Diamond, 1995; Selden et al., 1998). The role of the ventromedial prefrontal cortical lesion in the symptoms of ACoA rupture has rather been hypothesized as being that of producing personality changes (DeLuca, 1993; Irle et al., 1992; Eslinger and Damasio, 1984, 1985). Although we were unable to exclude a role for the frontal lobes in the memory deficits found in our neuropsychological study of the 47 ACoA rupture patients using CANTAB, we did find a pattern of deficit (impaired category fluency, pattern recognition and spatial working memory with normal strategy) resembling that seen in patients with involvement of more posterior cortical circuits, such as those with temporal lobe lesions, giving some support to the basal forebrain cholinergic/medial temporal lobe involvement theory for amnesia in ACoA rupture (Mavaddat et al., 1999). This pattern of deficit, together with the finding in ACoA rupture patients of a decision-making deficit in association with personality changes reminiscent of those in patients with damage to the ventromedial prefrontal cortical circuitry, lends support to the theory of a possible dissociation in the underlying causes of the neuropsychological sequelae seen in ACoA rupture patients. Thus, while acknowledging the range of symptoms present in ACoA patients, our findings support the emergence of two distinct neuropsychological patterns found as a group in ACoA rupture patients, in line with the dual lesion hypothesis.

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