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Investigating the 'latent' deficit hypothesis: Age at time of head injury, implicit and executive functions and behavioral insight.

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Caption: Latent deficits and behavioral insight

ABSTRACT

This study investigated the 'latent deficit' hypothesis in two groups of head-injured patients with predominantly frontal lesions, those injured prior to steep morphological and corresponding functional maturational periods for frontal networks (≤ age 25), and those injured >28 years. The latent deficit hypothesis proposes that early injuries produce enduring cognitive deficits manifest later in the lifespan with graver consequences for behavior than adult injuries, particularly after frontal pathology (Eslinger, Grattan, Damasio & Damasio, 1992). Implicit and executive deficits both contribute to behavioral insight after frontal head injury (Barker, Andrade, Romanowski, Morton & Wasti, 2006). On the basis of morphological and behavioral data, we hypothesised that early injury would confer greater vulnerability to impairment on tasks associated with frontal regions than later injury. Patients completed experimental tasks of implicit cognition, executive function measures and the DEX measure of behavioural insight (Behavioral Assessment of the Dysexecutive Syndrome: Wilson, Alderman, Burgess, Emslie, & Evans, 1996). The Early Injury group were more impaired on implicit cognition tasks compared to controls that Late Injury patients. There were no marked group differences on most executive function measures. Executive ability only contributed to behavioral awareness in the Early Injury Group. Findings showed that age at injury moderates the relationship between executive and implicit cognition and behavioral insight and that early injuries result in long-standing deficits to functions associated with frontal regions partially supporting the latent deficit hypothesis.

Keywords: Developmental, trajectory, cognition, awareness

INTRODUCTION

Pathology to anterior brain regions produces a constellation of deficits, although diminished self-regulation, poor behavioral insight, and other socio-emotional problems are the most incapacitating and resistant to rehabilitative efforts (Ponsford, 2004; Bach & David, 2006). Behavioral insight refers to the capacity to acknowledge and exhibit awareness of functional deficits post-injury and is considered a useful marker of more general socio-behavioural problems (Hart, Seignourel & Sherer, 2009; Flashman & McAllister, 2002). Diminished insight is associated with poor rehabilitation outcome, increased maladaptive behavior, heightened caregiver distress and reduced goal-directed self-regulatory behavior (Craik et al., 1999; D'Argembeau et al., 2005; Oddy, Coughlan, Tyerman & Jenkins, 1985; Sherer et al., 1998; Wise, Ownsworth & Fleming, 2005). Importantly, accurate self-appraisal (high behavioral insight) contributes significantly to post-injury emotional control and adaptive socio-cognitive functioning (Schmitz, Rowley, Kawahara & Johnson, 2006). Recent imaging data show that self-appraisal accuracy is associated with activity to ventromedial prefrontal cortex (Rosen et al, 2010). Although functional recovery is deleteriously affected by reduced behavioral insight, injury severity, locus, extent and age at time of injury are also important determinants of outcome (Anderson et al., 2009).

Executive and implicit processes and behavioral insight

Both executive and implicit functions are thought to contribute to insight into one's own behavior, often termed self-evaluative accuracy or behavioral awareness **(**Morris

& Hannesdottir, 2004; Toglia & Kirk, 2000; Schmitz et al., 2006). Executive functions are super-ordinate processes that operate across, inhibit or initiate other cognitive processes to produce integrated sequences of behavior (Miyake et al., 2000). Executive deficits are typically associated with pathology to frontal regions and these functions may dissociate post-injury with patients showing selective or multiple executive function deficits (Stuss & Alexander, 2007; Sullivan, Riccio & Castillo, 2009). Diverse executive functions have been associated with post-injury awareness and include, self-regulation (Bogod, Mateer & MacDonald, 2003), sustained attention (O'Keeffe et al., 2007), and planning and mental flexibility (Ownsworth, McFarland & Young, 2000). Findings have been difficult to replicate mainly due to the use of single and heterogeneous measures of executive function and diverse methodologies across studies (Ownsworth & Fleming, 2005).

Implicit processes operate by activating existing knowledge, strengthening links between concepts, or encoding new information in the absence of conscious awareness of learned information or the acquisition process. Implicit knowledge is acquired in parallel in contrast to explicit serial processing, facilitating or biasing behavioral responses outside of conscious volition. The term implicit cognition is used within the experimental literature to encompass the processes measured by a broad range of tasks testing behavioural and brain responses to subliminal, unnoticed or unattended stimuli or regularities between stimuli. These processes are thought to play an important role in 'real world' contexts, facilitating adaptive behavioral responses (Lieberman, 2000; Frith & Frith, 2008). Patients with lesions to frontal regions show diminished or abolished implicit learning on Serial Reaction Time tasks (SRT - Nissen & Bullemer, 1987), and lack of priming on a mere exposure effect task

(Zajonc, 1980) compared to control data (Beldarrain, Grafman, de Valesco, Pascual-Leone & Garcia-Monco, 2002; Morton & Barker, 2010), a pattern that maintains in patients with frontotemporal lesions (Barker, Andrade, Romanowski, Morton & Wasti, 2006).

The SRT is a choice reaction time task incorporating a regular but lengthy sequence of targets. Implicit learning is expressed as faster responses to sequence compared to random trials, without awareness of the presence of a sequence. In mere exposure effect tasks processing of subliminal or unattended stimuli primes a preferential response (increased liking) to those stimuli. In models of behavioral awareness implicit processes such as these are thought to operate by augmenting metacognitive awareness (mediated by executive functions), and by guiding behavioral responses in the absence of explicit awareness (Ownsworth, Clare & Morris, 2006; Toglia & Kirk, 2000; Morris & Hannesdottir, 2004). Absence of priming and inability to acquire an implicit sequence suggests disruption to mechanism(s) that might normally guide behavior in the absence of awareness (Barker et al., 2006). Whilst models of behavioral awareness typically include executive and implicit components, the implicit component is generally under-specified with limited explanation of how implicit processes modulate awareness, and is not measured experimentally. In addition, behavioral awareness models do not account for the possibility that implicit processes are disrupted after neuropathology (Ownsworth, Clare & Morris, 2006; Toglia & Kirk, 2000; Morris & Hannesdottir, 2004; Schacter, 1990). One aim of the present study was to investigate the contribution of implicit cognition to behavioral insight and to test whether age at injury affected this contribution.

Neuroplasticity and the 'latent deficit' hypothesis

There is ongoing debate about whether cognitive behavioral effects of head injury vary depending on age at time of head injury (Kolb, 1995; Anderson et al., 2009). This debate is particularly relevant to individuals with frontal pathology as these regions undergo a protracted period of maturation that may confer developmental vulnerability to pathophysiological processes (Paus, Keshevan & Giedd, 2008). Traditionally, theories of neuroplasticity and age at injury have presented conflicting findings of either preserved function after early compared to later injury (Kennard, 1940), or intractable functional deficits due to abolition of elementary functions underpinning cognitive systems (Hebb, 1949). A possible reason for contradictory findings is that consequences of early brain injury (to frontal regions in particular) are not fully apparent until later developmental periods when cognitive resources driving autonomy, independence, goal-directed, prosocial and sexual behavior are most in demand (Williams & Mateer, 1992). More data are needed to address this question as adolescents and young adults (particularly males) are highly represented in the head-injured population and frontal and temporal regions are particularly vulnerable to trauma after insult due to their position within the skull (Sosin, Sacks & Webb, 1996; Kolb & Whishaw, 1990), trajectory of the brain on impact, and proximal and distal connections with other brain regions. Early frontal insult may therefore result in graver deficits, because the brain is maturing and consequently vulnerable, that are latent in the sense that they emerge later in development. Findings from longitudinal case studies (Anderson, Catroppa, Morse, Haritou & Rosenfeld, 2005; Anderson, Damasio, Tranel & Damasio, 2000; Anderson, Bechara, Damasio, Tranel & Damasio, 1999; Barlow, Thomson, Johnson & Minns, 2005; Eslinger, Biddle & Grattan, 1997; Eslinger, Flaherty-Craig & Benton, 2004; Tranel & Eslinger, 2000), and group studies

(Ewing-Cobbs et al., 2006) support this 'latent deficit' hypothesis

Morphological maturation of anterior structures

Morphological studies reveal a phase of steep maturation occurring in late adolescence and early adulthood to anterior brain regions. Temporal and spatial maps of MRI scans show patterns of maturation from around age 14 to age 26 localised to large regions of frontal cortex and lenticular nuclei with little change to other regions (Sowell, Thompson, Holmes, Jernigan & Toga, 1999). Gogtay et al., (2004) found that changes in gray matter volume follow a non-linear pattern across different brain regions, with loss of gray matter density (either through synaptic pruning or intra-cortical myelination - Paus, 2005) to dorsolateral prefrontal cortex and posterior superior temporal gyrus only evident after age 16-17 years in healthy adolescents. In a longitudinal study of 8 healthy subjects aged 11 to 17.5 the greatest mean increase in white matter volume was found in frontal lobes (ranging from 8.4 - 26.8%) across the two time points, indicating rapid maturation of these regions during late adolescence compared to other brain regions (Riddle et al., 2008). Subcortical tracts (extending into frontal regions) and corticospinal tract continue to undergo change up to age 25 (Lebel, Walker, Phillips & Beaulieu, 2008), and there is some indication that maturational change may continue to age 30 in superior temporal cortex (Sowell et al., 2003).

Morphological changes correspond with socio-cognitive maturation occurring through adolescence and early adulthood (Blakemore & Choudhury, 2006). Evidence indicates that psychosocial and logical reasoning functions continue to mature until around age 25 (Steinberg, 2007). Executive functions follow a similar trajectory, with protracted development extending through adolescence into early adulthood (Anderson et al., 2001; Huizinga, Dolan & van der Molen, 2006; Levin et al., 1991;

Lin, Chen, Yang, Hsiao & Tien, 2000; Romine & Reynolds, 2005). The same may be also true of implicit cognition. Adults outperformed 7-11 year old children on an implicit sequence-learning task, showing a significantly larger learning effect and more rapid learning (Thomas et al., 2004; but see Meulemans, van der Linden & Perruchet, 1998). Other studies found that schoolchildren performed worse than adults on implicit memory tasks (e.g. Vaidya, Huger, Howard & Howard, 2007 but see Hayes & Hennessy (1996) for developmental invariance argument), and older adults performed significantly better on a symbolic implicit learning task than younger adults (Bo & Seidler, 2009). Overall there are little data on the developmental variability of implicit processes in later childhood through adolescence to adulthood.

To summarize, patients with frontal lesions show impaired executive function across a range of measures and diminished or abolished implicit cognition (measured by SRT, HCD and mere exposure effect tasks) across studies. There is evidence that both executive function and implicit cognition make separate contributions to behavioral insight also impaired after injury (Barker et al., 2006; Schacter, 1990; Morton & Barker, 2010). Impaired behavioral insight is thought to be a marker of intractable socio-behavioral problems post-injury. Frontal regions show steep morphological changes during adolescence extending into adulthood and are consequently vulnerable during this period (Paus, 2005). The present study tested the hypothesis that brain injury sustained during this period would have graver consequences for executive function, implicit cognition and behavioral insight in adulthood than similar injuries sustained later in the lifespan. We also tested whether age at injury moderated the expected relationships between these variables.

We selected SRT and mere exposure effect implicit tasks to measure implicit cognition as they are thought to correspond to mechanisms governing tacit non-verbal decoding and encoding of information respectively (Lieberman, 2000), and are sensitive to frontal pathology across patient-based studies. We selected a battery of standardized executive function tasks to contrast with single measures of executive function used in other behavioral insight studies. Behavioral insight was measured using the DEX Questionnaire (Behavioral Assessment of the Dysexecutive Syndrome – BADS, Wilson, Alderman, Burgess, Emslie, & Evans, 1996) a standardized measure widely used in clinical assessment. Though we are not concerned with the precise neural substrates of functions measured here imaging data show frontal activation during SRT (Seidler et al., 2005; Honda et al., 1998; Wong, Bernat, Bunce & Shevrin, 1997) and behavioral insight task performance (Schmitz et al., 2006) supporting neuropsychological findings and the assumption that these functions are disrupted after frontal pathology.

Two groups of closed head injury patients with pathology mostly confined to frontal regions completed the study. Patients who sustained insult during developmentally sensitive periods (up to age 25) on the basis of maturational and behavioral data were categorized as 'Early Injury,' the 'Late Injury' group sustained injury from age 28 onwards. Age- and IQ-matched controls allowed comparison of the extent of implicit deficits across the two patient groups. In line with previous work, we predicted that patients would be impaired on implicit experimental tasks compared to matched controls.

METHOD

We investigated the effect of age at time of head injury in 32 traumatically brain injured (TBI) patients with mostly frontal lesions on implicit cognitive tasks, executive function ability and a measure of behavioral insight used in clinical assessments. For the Early Injury group, imaging data showed pathology to frontal and temporal regions in four cases, ten cases had frontal pathology (only identified via medical records and clinical presentation for Case 2 and Case 5 as they were considered too high risk to transport for scans), Case 3 was acollosal anteriorly, Case 10 had lesions to anterior corpus callosum, and Case 15 had lesions to centrum semiovale (Table 2). In the Late Injury group, 5 cases had frontotemporal pathology, only identified by CT and medical records for Case 5 who had an aneurysm clip due to a frontal bleed predating a frontal head injury, and Case 14 who had ocular metal fragments. Case 1 had bilateral temporal lobe contusions. The remaining 10 had frontal contusions, identified by CT for case 8 who could not be scanned due to a metal eye socket. Age- and IQ-matched controls completed experimental tasks to provide normative data. Using a range of measures allowed us to explore possible age of injury effects on severity of functional deficit and contribution of executive and implicit processes to post-injury behavioral insight.

Participants

Ethical approval was granted by Sheffield South, North, Barnsley and Doncaster NHS Research Committee. All participants gave informed consent and were a minimum of 18 years of age at test. Time since injury duration varied from two to a maximum of ten years post-injury at time of testing to safeguard against measuring acute rather than chronic effects of injury (Lezak, 2004). Patients were recruited if

they had sustained anterior injury at putatively sensitive developmental periods or later in adulthood, showed behavioral problems post-injury, and were impaired on at least one executive function subtest. Half of the patient group sustained insult in adolescence through early adulthood (*n* = 16 range, 12-25 years: Early Injury group) and half in later adulthood (*n* = 16, range 28-55 years: Late Injury group). All were right handed and none had any marked physical disability (see Table 1 results section). Patients were predominantly male reflecting regional demography with the exception of two females in the Late and one in the Early Injury group so our sample may be somewhat skewed. National demographics drawn from NICE (National Institute for Clinical Excellence) guidelines 2007 indicate that males constituted 70-88% of all head-injured hospital admissions in the UK for that year. In relation to our sample these figures indicate that at the top of end of the range for a cohort of 32, 2.6 participants should be female approximately in line with our sample. Regional demography may fluctuate due to differences in cause of head-injury, falls being the primary cause with assaults as the secondary cause at national levels. For our recruitment area assaults were the primary cause of head injury (followed by road traffic accidents), which may explain the low female to male ratio in our sample (direct.gov.uk). Occupational status was as follows for the Early Injury group at time of test: Resident in a rehabilitation unit (*n* = 5), independent with rehabilitative support (*n* = 10), employed in semi-skilled work (*n* = 1). Late Injury patients were classified as: Resident in a rehabilitation unit (*n* = 4), independent with rehabilitative support (*n* = 4), employed in semi-skilled work (*n* = 8). Patients with a history of alcoholism, depression, or drug addiction were not included in the study.

All patients were MRi scanned on a 1.5 Tesla Eclipse scanner, Marconi Medical

Systems (Cleveland, Ohio) unless there were medical or behavioral contraindications (e.g. metal aneurysm clips, violent behavior). Scan sequence and parameters were TR, TE, TI (fluid attenuated inversion recovery FLAIR only), slice thickness 4mm with 2mm gap, T2 fast spin echo (FSE) in 3 planes (axial, coronal & sagittal), slice thickness 5mm with 1mm gap and T1 volume RF-FAST, slice thickness 1mm. Imaging data were analyzed using the Template Method (Damasio & Damasio, 1990). Regions were identified on the basis of Brodmann's Area by the Consultant Neuroradiologist (Table 2). Glasgow Coma Scale (GCS) scores at emergency admission and Post-Traumatic Amnesia (PTA) duration are also reported (Table 1).

Patients were matched to controls for age, years of education and Full-Scale IQ (WAIS III Wechsler, 1997). Controls provided normative data for implicit experimental tasks and where possible spouses, siblings or significant others were recruited to match for demography. Patients completed the Wechsler Memory Scale-R (WMS-R Wechsler, 1987) to screen for memory impairments that might account for performance deficits, The National Adult Reading Test (NART Nelson, 1991) to measure any IQ decline from pre-morbid levels, a range of executive function measures with good reliability and sensitivity indices, and a clinically used measure of behavioral insight (Table 3). Use of standardized neuropsychological measures with published normative data obviated the need to measure control performance on these tasks (other than the WAIS III for IQ matching). Early Injury patients (M = 5.7. SD, 1.6, range 0-9) and controls (M = 4.8, SD, 2.7; range: 2-11) t (1,30) = .37, p = .82, and Late Injury patients (M = 6.5, SD, 2.1, range: 2-10) and controls (M = 5.8, SD, 2.8, range: 3-9) t (1,30) = .41, p = .79 two-tailed fell within normal ranges for anxiety and depression on the Hospital Anxiety and Depression Scale (HADS

Zigmond & Snaith, 1983).

Procedure

Test administration was counterbalanced across participants. Testing typically took place across several sessions with rest breaks determined by the participant; some patients were only able to concentrate for 15-20 minutes in any given session, others completed longer test sessions interspersed with brief five minute breaks. The SRT task was programmed with rest breaks hard-wired (duration determined by participant key press) after each block of 50 trials. The mere exposure effect task had no rest breaks as the task only took ten minutes to complete.

Executive function measures

The Hayling and Brixton tests measure response initiation, inhibition and rule detection (Burgess & Shallice, 1997). The Behavioral Assessment of the Dysexecutive Syndrome (BADS, Wilson et al., 1996) provides a composite score from a range of executive function subtests measuring temporal sequencing, rule shifting, strategy initiation and action plan development, planning and goal-directed ability. The Wisconsin Card Sort Test (WCST, Heaton, 1981) measures the capacity to shift and maintain set. The C.O.W.A. (Controlled Oral Word Association Test – FAS version – Benton & Hamsher, 1989), a measure of verbal fluency, was also administered to patients and controls as a filler task between acquisition and test in the mere exposure effect task, although we are only interested in patient data here.

Measure of post-injury behavioral insight (DEX – Wilson et al., 1996)

The DEX is a 20-item scale with scores ranging from 0-4 (4 being the most severe).

Questions sample emotional or personality change, behavioral change and motivational and cognitive changes. It comprises two versions, one completed by the patient (Self-rating: DEX-S), and the other by a clinician/ significant other (Independent-rating: DEX-I) who has close daily contact with the patient. DEX-I ratings are used clinically as a metric of degree and extent of post-injury behavioral insight. In line with convention we took correlation between Self- and Independent-ratings as a measure of awareness of type of deficit experienced and discrepancy score as a measure of degree of awareness of deficit (Hart, Seignourel & Sherer, 2009). Self-ratings were subtracted from Independent-ratings to produce a DEX-Insight score, a measure of the patient's awareness of behavioral, cognitive and emotional deficits. This measure is thought to produce a more accurate discrepancy score than comparison of Self and Independent rating means because it takes account of rating differences by question.

Implicit cognitive tasks

We used two implicit experimental tasks previously shown to be sensitive to neuropathology, a measure of implicit judgment (mere exposure effect task Zajonc, 1980) and a measure of implicit performance (serial reaction time task-SRT Nissen & Bullemer, 1987). Performance on these tasks is thought to recruit the same mechanisms that mediate non-verbal encoding and decoding respectively (Lieberman, 2000).

Serial Reaction Time task

This task was programmed in Psyscope (Cohen, MacWhinney, Flatt & Provost, 1993) and presented on a Macintosh Powerbook 5300. Participants completed a practice

session before beginning the task. At acquisition, participants responded as quickly as possible to a single target circle appearing at one of four screen locations evenly spaced in a row. The target was a 1cm diameter closed white circle on a black background. Each target location corresponded to a key on the keyboard of the computer (v, b, n or m) respectively. Circles disappeared when the appropriate key press was made and reaction times (RT's) were recorded. Response-stimulus interval was 200 msec. Locations of the circle on the screen followed a predetermined 10 trial sequence, A B C D B C B D B C (see Seger, 1997). Participants were randomly assigned to one of two screen assignments to counterbalance the frequency of stimulus circle presentation at inner and outer locations.

Stimuli were presented in six acquisition blocks of 50 trials (five x 10 trial sequence repetitions) constituting the learning phase of the task, with rest breaks between each block. At test, participants completed three blocks of 50 trials (150 trials in total), two random blocks flanking a sequence block. Test phase followed immediately after the acquisition phase without warning to participants. Typically, participants respond more slowly to random block trials compared to sequence trials at test indicating that the sequence has been learned. For random block trials, target locations were hard-wired into the programme in a pseudo-random order to ensure that performance differences between sequence and random blocks at test did not result from mere learning of first order frequency information. A random block of trials was also completed at the beginning of the acquisition phase to confound any automatic judgment by participants that circles might follow a pattern.

After the task participants completed an explicit knowledge questionnaire (Seger, 1997). They were informed that circles followed a sequence and rated how certain

they were of the presence of a pattern, described any pattern that they had noticed, then rated (overleaf) how sure they were that the sequence consisted of a) ten positions (correct) and b) 12 or more positions. We used Seger's (1997) scoring method for the explicit task and her criterion that a score of sixteen or over showed explicit knowledge of the sequence. Each of the three test blocks (two random and one sequence block) produced 50 reaction time values, divisible as five repeats of ten trials. We calculated the median RTs for each of the five repeats of ten trials. The five medians for each block were combined to produce three means, one sequence mean and one mean for each random block. The two random block means were combined to produce a single mean. The sequence mean was subtracted from the random mean to provide a single learning score for each participant.

Mere exposure effect task

The mere exposure effect typically primes a preferential response to targets compared to foils. This task assessed participants' preference for previously heard stimuli over foils. Participants listened to one of two lists of fifteen disyllabic Finnish words, matched for likeability, recorded on compact disc and presented aurally through headphones (see Andrade, Englert, Harper & Edwards, 2001). The word list was presented twice at a rate of one word per 1.5 seconds. Use of word lists as targets or foils was counterbalanced across participants. After the acquisition phase, the C.O.W.A. (Controlled Oral Word Association Test – FAS version – Benton & Hamsher, 1989) was administered for three minutes as a distractor task. This task was chosen specifically to prevent explicit rehearsal of prime words prior to test stage so that any preference effects could be reliably ascribed to tacit processing. Participants then heard a test list containing all 30 words, targets and foils, recorded in random

order with a 4 second inter-stimulus interval. They were asked to guess whether the words meant something good or something bad on the basis of their sound, rating each word as "very nice/good", "slightly nice/good", "slightly nasty/bad" or "very nasty/bad". The aim of this instruction was to imply that there was a correct answer on each trial to discourage participants from making a global judgment about the sound of Finnish words and consequently rate each word identically.

Task scores

Response sheets were scored as follows: three points for "very nice", two points for "slightly nice", one point for "slightly nasty", and 0 points for "very nasty". Individual preference priming scores were calculated by subtracting the sum of preference ratings for foil words from the sum of preference ratings for target words for each participant. A negative or zero score indicated that priming did not take place whilst a positive value indicated that previously exposed words were preferred to new words.

RESULTS

We analyzed patient group data separately (and compared to matched controls for implicit tasks) to determine possible group differences as an effect of age at injury on implicit, executive and DEX measures with all other variables held relatively equal (IQ, Glasgow Coma Scale scores, etc., see Table 1 and 2). Early Injury patients were well matched to controls for IQ, t (1, 30) = .35, p = .72 two-tailed, with no significant decline from pre-morbid IQ scores t (1, 15) = 1.0, p = .32 two-tailed (see Table 1 for

Full-Scale scores). The Late Injury group were also well matched to controls for IQ, t (1, 30) = -.73, p = .47 two-tailed, similarly with no decline in IQ from pre-morbid levels t (1, 15) = -1.2, p = .24 two-tailed, and IQ scores did not differ for the two patient groups t (1, 30) = .65 p = .52 two-tailed. Number of years of education was similar for Early Injury patients (M = 11.5, SD 1.3, range: 11-16), and controls (M = 11.7, SD 1.0, range: 11-14), t (1, 30) = -59, p = .56 two-tailed, and Late Injury patients (M = 11.7, SD 1.9, range: 10-18), and controls (M = 11.1, SD 0.8, range: 11-14), t (1, 30) = 1.2, p = .24 two-tailed. Patient groups did not perform significantly differently on the WMS-R (Wecshler, 1987) measure of memory and attention (Table 3). Early Injury patients were 5.9 (SD 2.6, range: 2-10) mean years since injury, Late Injury patients were 4.2 (SD 1.6, range: 2-9) mean years post-injury t (1, 30) = 2.3, p = .03 two-tailed with the Late Injury group less far on in the recovery process.

[insert table 1 here]

Neuropathology data

Glasgow Coma Scale scores t (1, 30) = .56, p = .76 two-tailed, were not significantly different for the two groups. There was no significant difference in duration of Post-Traumatic Amnesia t (1,30) = -1.8, p = .082 two-tailed although the Late Injury group had a greater mean duration of PTA than the Early Injury group (Table 1). For ease of comparison Brodmann's regions were categorized as follows: Dorsolateral prefrontal cortex (BA 8, 9, 44, 45 and 46), ventromedial prefrontal cortex (BA 10, 11, 12, 13, 14 and 47), and 'other' frontal and temporal regions. Pathology was heterogeneous and varied in location and severity within and between groups. In general terms, there were no marked differences in degree of pathology at the group level although Late

Injury patients had greater incidence of bilateral frontal pathology than Early Injury patients (Table 2). Data correspond by case across tables for ease of comparison.

[insert table 2 here]

Patient scores on executive function measures and measure of behavioral insight

Each patient was impaired on at least one executive function subtest (including BADS subtest scores) as criterion for inclusion to the study. The Late Injury group performed more poorly on the Hayling test of response initiation and response inhibition t (1, 30) = 3.0, p = .005 two-tailed, and the Brixton subtest of rule detection t (1, 30) = 2.1, p = .048 two-tailed than the Early Injury group (see Table 3 for descriptive data). Group means were not different for BADS, WCST, and C.O.W.A. tests of executive function (Table 3).

[insert table 3 here]

Measure of behavioral insight (DEX –Wilson et al., 1996)

We compared Self-ratings (DEX-S) of the two patient groups to Independent-ratings (DEX-I) made by spouses, siblings or significant others. Marked behavioral problems and lack of insight into post-injury deficits is typified by higher and contrasting DEX-I compared to DEX-S ratings across items (i.e. patients may rate themselves as having deficits but lack correspondence between Self- and Independent-raters for *type* of deficit(s) identified signifying poor behavioral insight). Early Injury patients rated themselves as most affected by 'lack of insight and social awareness,' 'shallowing of affective responses,' 'distractibility,' and 'lack of concern for social rules.' Independent-raters scored 'impulsivity,' 'confabulation,' 'euphoria,' 'perseveration,'

‘restlessness,’ ‘shallowing of affective responses,’ and ‘impaired temporal sequencing’ most highly. Total Self- (M = 29.1, SD 17.8, range: 0-71) and Independent-ratings (M = 36.1, SD 20.2, range: 3-77) were computed for each person. Pearson’s correlation showed no significant relationship between DEX-S and DEX-I scores r (16) = .38, p = .13 two-tailed indicating diminished awareness of type of deficit exhibited post-injury.

Late Injury patients rated ‘unconcern for social rules,’ ‘shallowing of affective responses,’ and ‘aggression’ most highly. Independent-ratings corresponded, but also included high ratings for the ‘inability to inhibit responses’ items. Again, total Self- (M = 36.9, SD 17.8, range: 3-65) and Independent- (M = 41.2, SD 18.5, range: 1-71) ratings were computed for each person. DEX-S and DEX-I scores showed a good level of behavioral insight into type of deficit identified as problematic by Late Injury patients r (16) = .57, p = .02 two-tailed. Discrepancy between DEX-S and DEX-I ratings was measured by calculating a DEX-Insight score for each patient group in line with recommendations by test authors and others (Burgess, Alderman, Emslie, Evans, & Wilson, 1998; Bennett, Ong & Ponsford, 2005; Hart, Seignourel & Sherer, 2009). DEX-Insight scores are computed by subtracting Self- from Independent-ratings. Early Injury patients had a mean DEX-Insight score of 6.9 (SD 20.9, range: -21-48), contrasted with a mean score of 4.2 (SD 16.9, range: -19-42) for Late Injury patients. DEX-Insight scores were not significantly different for patient groups t (1,30) = .41, p = .69 two-tailed, although mean DEX-Insight score was higher for the Early Injury group. Correlation analysis between time since injury and DEX-Insight showed that duration of time since injury was not associated with degree of behavioral insight r (32) = .02, p = .96.

Implicit experimental tasks: Patient and matched control data

Serial Reaction Time task

Several patients were slower on the sequence compared to random blocks at test (Early Injury group = 5, Late Injury group = 5) producing a negative learning score that accounts for large standard deviations in both patient groups compared to controls. For the Early Injury group, mean learning score was significantly lower for patients (9.6, SD 106.5) than controls (96.4, SD 40.5), t (1, 30) = 3.04, p = .005 two-tailed. Mean explicit measure scores were below the threshold of 16 and similar for patients (6.8, SD 4.7), and controls (8.4, SD 3.6), t (1, 30) = -1.05, p = .31 two-tailed; explicit knowledge of the sequence did not account for sequence learning differences between patient and control groups. In contrast, Late Injury group mean scores (33.1, SD 141.8) were not significantly different from control scores (86.7, SD 79.4), t (1, 30) = -1.32, p = .21 two-tailed although learning scores were lower for the patient group. Mean explicit measure scores were also similar for patients (7.8, SD 1.9), and controls (8.7, SD 3.1), t (1, 30) = -.87, p = .39 two-tailed. Findings indicate that Early Injury patients were more impaired than Late Injury patients compared to matched controls on this task. It is unlikely that impaired explicit cognition mediated these effects because patient groups were not different from controls in this respect.

Mere exposure effect priming scores were analyzed non-parametrically because ratings constituted ordinal data. For Early Injury patients mean prime scores (1.7, SD 5.7) did not differ from control scores (2.8, SD 2.9), Mann-Whitney U: z = -.89, p = .38 two-tailed. However, the difference between target and foil preference ratings only reached statistical significance for controls z = -2.76, p = .006 two-tailed, and z =

-.97, p = .33 two-tailed for patients, showing a mere exposure effect in controls that was absent in the patient group. Mean prime scores were different for Late Injury group patients (1.1, SD 3.8), and controls (3.2, SD 3.3), z = -1.97, p = .04 two-tailed. Wilcoxon Signed Ranks Test confirmed a priming effect for controls, z = -2.94, p = .003 two-tailed, but not for patients, z = -.88, p = .31 two-tailed, again indicating no mere exposure effect in patients compared to controls.

Classification of WAIS, WMS-R and executive function scores by case.

Group means can sometimes mask the heterogeneity of deficit and sparing across individuals so we classified individual test performance for Early (table 4) and Late Injury cases (table 5) on IQ, memory and executive function subtests based upon test manual scoring criteria (impaired ability typically falls at or below the 5th percentile). BADS total score is provided here rather than BADS subtest scores because total score was used in subsequent analyses; C.O.W.A. scores are not included because this variable showed no relationship with DEX-Insight for either group. Individual scores for implicit experimental tasks are not included here because these tasks lack sensitivity at the individual rather than the group level. Data show that despite heterogeneous performance patterns across cases in both groups there were few marked differences between groups. The Late Injury group had several cases showing impaired performance on the Verbal IQ (3 borderline/1 impaired) subtest of the WAIS not seen in the Early Injury group, and the Early Injury group had a greater number of cases impaired on the General Memory measure of the WMS-R (3 impaired/3 borderline compared to 1 impaired/2 borderline: Early Injury group). The greatest distinction between the groups lay in executive function scores, with the Late Injury

group showing more cases with impaired performance on the Hayling subtest of response inhibition (8 cases compared to 4 in the Early Injury group), although groups were not significantly different in frequency of impaired ability on this measure $c^2(1, N = 32) = 1.33$, $p = .38$. Early and Late Injury groups had the same number of cases impaired on the BADS ($n = 3$) although the Early Injury group had a slightly greater number of borderline cases ($n = 5$) than the Late Injury group ($n = 2$). Case data were not notably different on other subtest measures.

[Insert table 4 and 5 here]

Our previous work (Barker et al., 2006) showed a relationship between executive function measured by the BADS (selected because it provides a global score across a range of subtests of executive functions) and implicit cognition measured by SRT task learning score. Impaired performance on both measures was associated with high DEX-Insight scores indicating poor behavioral insight after injury. In the present study, we tested whether these relationships varied as an effect of age at injury. For the Early Injury group, analyses confirmed that most executive function scores correlated negatively with DEX-Insight (high DEX-Insight score indicates poor behavioral insight), BADS r (16) = -.65, p .003 one-tailed, WCST r (16) = -.59, p = .008 one-tailed, and Hayling r (16) = -.50, p = .02 one-tailed. Neither SRT r (16) = -.34, p = .10 one-tailed, or mere exposure priming score r (16) = -23, p = .19 one-tailed, correlated with DEX-Insight for the Early Injury group.

There was no relationship between executive function scores and behavioral insight for the Late Injury group, BADS r (16) = .20, p = .23 one-tailed, WCST r (16) = .03,

p = .45 one-tailed, and Hayling r (16) = .26, p = .17 one-tailed. In contrast to the Early Injury group, there were marginally significant correlations between implicit tasks and DEX-insight scores, SRT r (16) = .39, p = .06 one-tailed, and mere exposure priming score r (16) = -.41, p = .058 one-tailed for Late Injury patients. Results suggest a different pattern of relationship between executive function, implicit cognition and DEX-Insight for each patient group. To test further whether age at time of injury moderated relationships between these variables we conducted hierarchical regressions with BADS and SRT scores as predictor variables and DEX-Insight as the criterion variable.

Interaction effects: age at time of injury, executive and implicit function and behavioral insight.

To preserve predictive power, two separate hierarchical regressions were conducted for age at injury, either BADS or SRT variables (see Table 6 and 7), and the interaction term (BADS*age at injury and SRT*age at injury, respectively) as predictors of DEX-Insight. DEX-Insight served as the dependent variable in both analyses. The interaction variables were created as the products of centred versions of BADS and SRT variables and the dichotomous age at injury variable consistent with recommendations (see Jaccard & Turrisi, 2003). Independent variables were entered in three blocks, with the relevant cognitive variable (either BADS or SRT) entered first. The second block contained the categorical variable 'age at injury' (Early Injury = 0, Late Injury = 1), and the interaction term was entered in the third block. This final step computed whether the relationship between cognitive variables and DEX-Insight was moderated by age at injury, and whether the moderation had predictive

utility beyond that of the main effects of age at injury and BADS/SRT. Change statistics in the first model show that scores on BADS explained 11% of the variance in DEX-Insight across both groups, that the injury group variable did not add to the variance on its own, and that the interaction between BADS and age at injury explained an additional 16% (R^2-Δ = .16) of the variance in DEX-Insight scores.

In the final step, the unique regression coefficients (see Table 6) indicate that the interaction between age at injury and executive function as measured by BADS scores was a significant unique predictor of DEX-Insight. This suggests that age at injury was a moderator in the relationship between executive function and behavioral insight. There was a strong inverse relationship between BADS and DEX-Insight for the Early Injury group r (32) = -.65 p = .006 two-tailed, indicating that low BADS scores were associated with low levels of behavioral awareness (high DEX-Insight discrepancy scores). There was no relationship between BADS score and DEX-Insight for the Late Injury group r (32) = .20, p = .45 two-tailed (Figure 1).

[Insert Table 6 here]

[Insert Figure 1 here]

The interaction between age at injury and implicit cognition (SRT scores) was only marginally significant. The change statistics show that beyond the individual contributions of age at injury and SRT scores, the interaction of the two explained a further 12.7% of variance in DEX-Insight scores (R^2-Δ = .127, see Table 7). This is a moderate effect with marginal significance p = .052 two-tailed. Simple effect analysis showed that the relationship between SRT and DEX-Insight followed a different direction for each patient group (i.e., positive for the Late Injury group, inverse for the

Early Injury group) although neither analyses were significant (Late Injury r (32) = .39, p = .14 two-tailed) and r (32) = -.34, p = .20 two-tailed for the Early Injury group.

[Insert Table 7 here]

[Insert Figure 2 here]

Overall, findings from interaction analyses indicate that age at time of injury strongly moderates the contribution of executive function to behavioral insight, but that moderating effects of age at injury on the contribution of implicit cognition to insight is less clear.

DISCUSSION

Closed head injury patients were assigned to Early and Late Injury groups on the basis of age at time of injury. Early Injury patients sustained insult in adolescence and early adulthood, a period of rapid morphological change to anterior regions thought to correspond with maturation of social and 'higher-order' cognitive abilities (Blakemore & Choudhury, 2006; Romine & Reynolds, 2005). Late Injury patients sustained injury from late twenties through to middle age. Patients were matched to controls for age, IQ, years of education and demography and were not different on measures of IQ, attention and memory at the group level. There was no significant decline from pre-morbid to present IQ status, although most patients showed a small decrease in IQ points from pre-morbid levels. Patients were more than two years post-injury at time of test in line with recommendations that this period represents the acute phase of recovery (Lezak, 2004), and differed significantly in this respect. Early

Injury patients had a slightly longer mean duration of time since injury than the Late Injury group, although we found no relationship between time since injury and behavioral insight. Imaging data, GCS scores and medical records revealed similar loci and extent of brain injury in the two groups, although the Late Injury group included a greater number of cases with bilateral pathology, and had greater mean duration of PTA compared to Early Injury patients. Both groups were impaired on the mere exposure effect task but the Early Injury group alone showed impaired performance on the SRT task compared to controls. Both groups had higher mean DEX-I than DEX-S ratings typically interpreted as evidence of diminished behavioral insight (Flashman & McAllister, 2002; Toglia & Kirk, 2000; Burgess et al., 1998; Hart, Seignourel & Sherer, 2009). There was evidence of some residual insight into post-injury problems for Late Injury patients shown by corresponding DEX-S and DEX-I ratings not seen for Early Injury group ratings. Indeed Early Injury patients identified wholly different deficits as problematic compared to DEX-I ratings. Results are unlikely to reflect unreliable DEX-I ratings because evidence shows that significant others make reliable deficit ratings on the DEX except in acute circumstances immediately post-injury when the patient is typically hospitalized (Bennett, Ong & Ponsford, 2005; Chan & Bode, 2008). Groups were not different on the DEX-Insight discrepancy measure signifying impaired insight into severity of deficits in both groups.

Group means were similar across most neuropsychological tests though Late Injury patients showed poorer ability on Hayling and Brixton subtests of response inhibition and rule detection falling within low average ranges. Poorer performance on these tasks by Late Injury patients may be related to impaired inhibitory control reported by

Independent-raters not identified by Self-ratings on the DEX.

At the individual case level heterogeneous patterns of functional ability were evident within groups as would be expected after TBI. However, despite this heterogeneity of functional outcome shared patterns of deficit and sparing also emerged across groups. A relatively equal number of cases in both groups showed Processing Speed (WAIS-III) and Visual Memory (WMS-R) deficits on IQ and memory subtests. Both groups showed relatively similar patterns of executive function impairment but were distinguished by greater number of cases impaired on the Hayling subtest for the Late Injury group. There were more cases with impaired General Memory in the Early Injury group and more evidence of Verbal IQ deficits in the Late Injury group. It is unlikely that higher number of cases with General Memory problems mediated ability on executive and implicit tasks in Early Injury patients because executive tasks were administered with written instructions visible throughout testing, implicit task performance is not dependent on explicit memory processes (Jiménéz & Méndez, 2001), and groups showed similar levels of impairment on the mere exposure implicit task compared to controls. Both groups showed a similar number of cases with Processing Speed and Visual Memory deficits but only Early Injury patients were impaired on the SRT compared to controls making it unlikely that these deficits impeded implicit learning.

To summarize, general cognitive abilities were similar across the Early and Late Injury groups, including executive abilities with the exception of performance on the Hayling and Brixton subtests. The Late Injury group performed more poorly on these two subtests. In contrast, the Early Injury group were more impaired on the measures

of implicit cognition and showed poorer behavioral insight. Considered together these data provide little evidence to support the Kennard principle (1940) that early insult to frontal regions results in greater functional sparing than later injuries particularly in relation to executive function ability. Likewise these data offer little support to the notion that early injuries result in graver functional deficits than later injuries.

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Further analyses revealed differences in the pattern of relationships between executive measures, implicit tasks and DEX-Insight across groups. BADS, WCST, and Hayling executive scores correlated negatively with DEX-Insight scores for Early Injury patients (low EF score and high DEX-Insight score equaling poor insight and vice versa). There was no significant correlation between implicit task scores and DEX-Insight despite impaired ability on both implicit tasks in Early Injury patients.

The inverse pattern was seen for the Late Injury group, no significant relationship between executive function scores (BADS total score, Hayling, and WCST) and DEX-Insight (even though patients were more impaired on the Hayling and Brixton than Early Injury patients), and marginally significant relationships between SRT and mere exposure scores and DEX-Insight. These results suggest a definitive role of executive function to behavioral insight after early injuries. Data are less clear-cut about the contribution of implicit cognition to behavioral insight. The Late Injury group showed some residual insight into deficits based on DEX-I and DEX-S data, although DEX-Insight scores did not differ for patient groups. Late Injury patients were not impaired on the SRT task compared to matched controls unlike the early injury group, and analyses showed marginally significant correlations with implicit task scores and DEX-Insight in the Late Injury group alone. These group differences

seem best explained as an effect of age at time of injury as group means were not significantly different on neuropsychological tests, DEX-Insight, BADS or neurological variables despite within-group variability.

Our earlier work found a significant relationship between BADS and SRT scores and impairments on both contributed to behavioral insight (Barker et al., 2006). Results of hierarchical regression analyses showed that overall executive ability measured by the BADS made a significant contribution to behavioral insight in Early compared to Late Injury patients. The magnitude of the interaction term effect was large and was the strongest unique predictor of DEX-Insight scores. In contrast, there was no significant relationship between BADS and DEX-Insight scores for the Late Injury group. These data support the assumption that age at time of injury and executive ability are important predictors of behavioral insight after pathology to anterior structures (Hart, Whyte, Kim & Vaccaro, 2005; Schmitz et al., 2006). Findings might also shed some light on conflicting evidence that executive functions contribute to behavioral insight (Bogod, Mateer, & Macdonald, 2003), or conversely show no relationship with behavioral insight (O'Keeffe et al., 2007) in studies where age at time of injury is not accounted for.

The interaction effect for regression analysis with SRT and DEX-Insight was relatively weak and only marginally significant. Regression lines fell in opposite directions for the two patient groups, although neither reached significance. However, the trend towards an inverse SRT/DEX relationship in the Early Injury group is in line with the executive function/DEX relationship also seen in the Early Injury group and consistent with predictions based on earlier work (Barker et al., 2006; Hart et al.,

2005; Schmitz et al., 2006).

We can only speculate on the reasons for the difference in relationships between cognitive functions and insight as an effect of age-at-injury. Longitudinal case study findings show that early frontal injuries result in executive deficits associated with severe behavioral problems compared to later injuries (Williams & Mateer, 1992; Eslinger & Biddle, 2000; Anderson et al., 2005), though theoretically it is unclear why this should be the case: Few interpretations of these data go beyond the 'developmental sensitivity' argument. Behavioral insight is thought to depend upon the integration of several functions, including preattentive, metacognitive, implicit, executive and regulatory functions (Barker et al., 2006; Morton & Barker, 2010; Morris & Hannesdottir, 2004; Schacter, 1990), and somatic physiological markers (Damasio, 1996), though constitutive cognitive components of insight need more fine-grained specification. In normal development these contributory processes may cohere into a functional system integrated by executive function control mechanisms (Bogod, Williams, & Mateer 2003). Pathology sustained during developmentally sensitive periods may render the system vulnerable resulting in poorer functional integration. The net effect may be an executive 'hegemony' so that when executive functions are diminished during developmentally sensitive periods there are fewer and less well-integrated compensatory mechanisms in place. Injury to anterior regions in early adolescence through to early adulthood is more likely to diminish integrative and coordinating rather than 'elemental' cognitive functions in an immature and consequently inherently vulnerable functional system (Paus, 2005; Johnston, 2009; Hebb, 1949). Indeed, research shows that early insult also disrupts integrative aspects of language functional systems whilst selective ability may remain intact (Demir,

Levine & Goldin-Meadow, 2009). Executive functions are also disrupted in later injuries but evidence of dissociation between executive ability and behavior suggest the effects on behavior may be less severe compared to early injury (Barker, Andrade & Romanowski, 2004; Andrés, & Van der Linden, 2002). This hypothesis might go some way to explain our findings of a contribution of executive function to behavioral insight after early compared to late injury, an effect moderated by age at time of injury. More data are needed on the normal developmental trajectory of a range of functions and corresponding maturational change to address this possibility. The pattern of data for Late Injury patients, no significant contribution of executive function or implicit cognition to behavioral insight, indicate a possible contributory role of other processes to insight not measured here. This possibility does not correspond well with Early Injury group data, but supports the notion of a multi-componential functional system underpinning insight that may have been more robust in the Late Injury group due to normal maturation of anterior regions prior to injury. Hence evidence of residual awareness shown in the Late but not the Early Injury group.

Individual case data showed that head injury sustained as early as age 12 results in enduring deficits to executive and other functions. All patients were a minimum of 18 at test so most Early Injury patients were well on in the recovery process. This finding partially supports the latent-deficit hypothesis though whether early insult results in graver deficits than later injuries is difficult to quantify from present data. However, functional effects of early head injury were comparable with later injuries even though neurological variables showed greater incidence of bilateral pathology, greater duration of PTA and less time since injury for Late Injury patients (see also Fay et al.,

2009). This neurological profile predicts more severe head injury and greater functional deficits in Late compared to Early Injury patients that may have been masked by similar functional outcome in Early Injury patients. It is likely that both groups showed some functional recovery since the acute stage but in the absence of baseline data immediately post-injury it is not possible to determine whether this differed significantly for groups. Overall, the moderating effect of age at time of injury on executive, implicit and DEX-insight variables seems less important when injury is sustained from late twenties onwards shown by the non-significant effects for Late Injury patients in regression data.

There are several limitations of the study that future work might address. Head injured groups are intrinsically heterogeneous in functional outcome and pathology making it difficult to control for all confounds. In the present study age at time of injury varied within groups, as we were unable to recruit sufficient patients who sustained injury at the same time. This variability in age at injury may have had differential effects on maturational processes although functionally the spread of deficits was similar at the case level and early injury patients were more similar in age at time of test than at time of injury. Additionally our sample comprised only three females reflecting regional variability in head injury demographics. A greater number of female patients included in the study might have produced a different pattern of results.

To conclude, present data do not demonstrate greater impairment or greater functional plasticity after early compared to later injuries. Instead we found a significant relationship between executive function and insight and more impaired implicit cognition compared to controls after early compared to later injuries. Age at injury

moderated the relationship between cognitive processes and behavioral insight. Further research aimed at elucidating the interplay between these processes following injury should take age at injury into account. Likewise, research into the normal development of these processes should consider the development of relationships between processes as well as the individual processes themselves.

Our findings show that early injuries result in long-standing deficits to functions associated with frontal regions partially supporting the latent deficit hypothesis. Although our data speak only indirectly to maturational imaging work, future studies might track morphological brain changes and functional ability longitudinally into middle adulthood to broaden our knowledge of the relationship between function, brain morphology and behavioral consequences of early brain injury.

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Table 1. IQ, neurological and demographic data of patients and controls.

Full-Scale IQ score Patients	Full-Scale IQ score controls	Pre-morbid IQ Patients	Age at Injury (Yrs: months)	Age at test	GCS score	PTA (days)
1. 78	84	76	12.10	20	5	18
2. 102	103	108	14.11	21	11	4
3. 88	94	90	14.9	24	4	8
4. 94	102	101	15.9	21	5	11
5. 82	96	87	17.8	20	4	30
6. 104	94	106	17.8	25	8	7
7. 102	95	90	17.6	26	13	2
8. 104	109	75	18.10	26	3	8
9. 105	97	107	18.7	24	6	28
10. 109	100	102	20.3	23	5	30
11. 104	104	100	20.2	26	3	120
12. 87	81	81	20.8	25	5	15
13. 91	95	96	21.2	31	3	14
14. 86	102	89	21.1	30	5	7
15. 110	110	110	22.11	25	6	14
16. 97	95	87	25.4	28	5	56
M = 96.4 (SD 10.0)	M = 97.6 (SD 7.8)	M = 94.1 (SD 11.3)	M = 18.6 (SD 3.3)	M = 24.7 (SD 3.3)	M = 5.4 (SD 3.1)	M = 22.1 (SD 29.7)
17. 87	90	89	28.11	33	4	3
18. 109	105	111	30.2	34	3	28
19. 75	80	78	30.5	33	3	300
20. 80	90	82	31.2	34	4	32
21. 99	104	94	33.4	37	13	3
22. 127	129	127	36.2	40	4	15
23. 85	80	86	36.1	38	3	69
24. 105	108	106	38.8	42	3	90
25. 80	84	82	39.7	43	5	40
26. 97	95	95	44.4	47	10	8
27. 87	90	87	46.5	49	6	15
28. 85	92	87	47.5	56	4	11
29. 80	85	82	48.2	52	3	18
30. 104	109	103	49.5	55	3	150
31. 100	102	102	54.3	59	3	200
32. 99	112	98	55.1	59	5	18
M = 93.7 (SD 13.8)	M = 97.2 (SD 13.5)	M = 94. 3 (SD 13.1)	M = 40.6 (SD 8.9)	M = 44.4 (SD 9.5)	M = 4.7 (SD 2.8)	M = 62.5 (SD 84.7)

Table 2. Locus of neuropathology for Early and Late Injury groups categorized by Brodmann's Areas (BA)

Patient	Right VMPFC	Right DLPFC	Left VMPFC	Left DLPFC	Additional frontal and other brain regions
Early Injury Group					
Case 1	BA (10), (11), (13), (14), (47)	BA (44), (45),(46), (8), (9)	BA (10), (11), (13)		Minimal damage to temporal pole BA (38), (21)
Case 3	Acollosal		Acollosal		
Case 4	BA (47)	BA (8)	BA (47)		Bilateral temporal gyrus
Case 6		BA (44), (45)		(44)	Moderate pathology to right temporal pole minimal to left
Case 7		BA (44), (45)			
Case 8	BA not specified (CT only)		BA not specified (CT only)		
Case 9	BA (10), (11), (13), (14), (47)	BA (44), (45),(46), (8), (9)	BA (10), (11), (13), (14), (47)	BA (44), (45), (46), (8), (9)	
Case 10			(10), (11)		Small haemorrhagic cavity in Corpus Callosum across both sides anteriorly
Case 11				BA (8), (9), (46)	
Case 12			BA (10), (11), (13)		
Case 13			BA (11), (13)		Left temporal lobe
Case 14					Global atrophy and foci of high signal to frontal regions
Case 15					Focal lesion to Centrum Semiovale
Case 16	BA (10), (11)				
Late Injury Group					
Case 1					Middle temporal gyrus Inferior temporal gyrus bilaterally
Case 2					Left frontal lobe lacuna infarcts.
Case 3	BA (10), (11), (13), (14), (47)	BA (44), (45),(46), (8), (9)	BA (10), (11), (13)		Minimal damage to temporal pole BA (38), (21)
Case 4	BA (10), (11)		BA (10), (11), (13)		
Case 6	BA (10), (11), (12), (13), (14). (47)	BA (46), (45)	BA (10), (11), (12), (13), (14). (47)	BA (45)	Left and right temporal pole
Case 7	BA (10),(11), (13), (14), (47)	BA (9)	BA (10),(11), (13), (14), (47)	BA (44), (45). (46), (9),(8)	
Case 8	Extensive pathology (BA not specified)	Extensive pathology (BA not specified)			
Case 9	BA (10), (11), (13), (14), (47)	BA (44), (45),(46), (8), (9)	BA (10), (11), (13), (14), (47)	BA (44), (45), (46), (8), (9)	
Case 10			BA (10), (11), (13)		
Case 11	BA (13), (14), (47)		BA (12), (13)		
Case 12					Global atrophy excessive for age and foci of high signal to white matter in frontal regions
Case 13	BA (10), (11), (12)		BA (14)		
Case 15			BA (47), (11), (12), (13)		Left temporal pole
Case 16	BA (10), (11), (12)		BA (10), (11)		Patchy small vessel ischaemic change in right frontal lobe

Table 3. Mean (SD) and range scores on neuropsychological measures for Early and Late Injury groups.

Neuropsychological Test	Early injury group Mean (SD), range	Late Injury Group Mean (SD), range
WAIS: Subtest scores Verbal IQ	97.6 (11.8) Range: 84 -127	91.8 (15.2) Range: 66-119
Performance IQ	93.2 (13.6) Range: 68 - 119	95.0 (15.5) Range: 72-130
Verbal Comprehension Index	97.2 (12.7) Range: 76-124	93.2 (16.1) Range: 67-116
Perceptual Organisation Index	101.1 (16.9) Range: 72-125	103.8 (18.3) Range: 78-148
Working Memory Index	97.6 (16.3) Range: 71-130	93.6 (14.7) Range: 69-126
Processing speed	81.1 (12.5) Range: 60-103	85.7 (14.1) Range: 69-120
WMS-R Subtest scores Verbal Memory	89.7 (12.8) Range: 63-111	83.3 (13.6) Range: 65-107
Visual Memory	85.2 (23.4) Range: 50-138	84.8 (16.7) Range: 50-110
General Memory	85.8 (17.1) Range 55-123	80.3 (15.8) Range: 55-108
Attention/concentration	95.1 (15.2) Range: 67-120	88.4 (17.7) Range: 62-125
Executive Function scores Hayling	5.4 (1.5) Range: 2-8	3.5 (1.9) Range: 1-7
Brixton	5.8 (2.1) Range: 1-10	4.1 (2.3) Range: 1-8
BADS total score	85.4 (20.8) Range: 43-118	84.1 (15.3) Range: 54-102
WCST	84.7 (36.4) Range: 0-125	79.1 (28.7) Range: 0-124
C.O.W.A (FAS) Total score	31.7 (8.8) Range: 19-45	33.0 (16.8) Range: 14-69

Table 4. Early injury group individual performance categorizations on WAIS III, WMS-R, and executive function tests.

	WAIS-III subtest						WMS-R subtest				Executive function tasks				Age at Injury
Case	Verbal IQ	Performance IQ	Verbal Comp. Index	Perceptual Org. Index	Working Memory Index	Processing Speed	Verbal Memory	Visual Memory	General Memory	Attention	Hayling	Brixton	BADS Overall	WCST	Yrs:mths
1.	Low Ave.[a]	Low Ave.	Low Ave.	Ave.	Low Ave.	Border.[b]	Ave.	Ave.	Ave.	Impaired	Impaired	Impaired	Impaired	Border.	12.10
2.	Ave.	Ave.	Ave.	Ave.	Sup.[c]	Ave.	Ave.	Ave.	Ave.	Sup.	Border.	Sup.	Border.	Sup.	14.11
3.	Ave.	Low Ave.	Ave.	Ave.	Ave.	Impaired	Ave.	Impaired	Low Ave.	Low Ave.	Ave.	Ave.	Border.	V. Impaired	14.9
4.	Ave.	High Ave.	Ave.	Sup.	Ave.	Ave.	Low Ave.	Low Ave.	Border.	Ave.	Ave.	Low Ave.	Ave.	Ave.	15.9
5.	Sup.	Border.	Sup.	Ave.	Sup.	Border.	Ave.	Border.	Ave.	High Ave.	Impaired	High Ave.	V. Impaired	Impaired	17.8
6.	Low Ave.	Border.	Ave.	Low Ave.	Low Ave.	Impaired	Ave.	Impaired	Low Ave.	Low Ave.	High Ave.	Low Ave.	Ave.	Low Ave.	17.8
7.	Ave.	High Ave.	Ave.	High Ave.	Ave.	Ave.	Border.	Ave.	Border.	Border.	High Ave.	High Ave.	High Ave.	Ave.	17.6
8.	Low Ave.	Ave.	Ave.	Border.	Low Ave.	Impaired	Ave.	V. Impaired	Impaired	Low Ave.	Ave.	Ave.	V. Impaired	Ave.	18.10
9.	Ave.	Border.	Low Ave.	Ave.	Low Ave.	Impaired	Low Ave.	Impaired	Border.	Ave.	Ave.	Ave.	Border.	Impaired	18.7
10.	Ave.	Border.	Ave.	Low Ave.	Ave.	Border.	Impaired	Impaired	V. Impaired	Low Ave.	High Ave.	Low Ave.	Ave.	Ave.	20.3
11.	Ave.	Low Ave.	Ave.	Ave.	Low Ave.	Border.	Low Ave.	Low Ave.	Low Ave.	Low Ave.	Impaired	Ave.	Low Ave.	Ave.	20.2
12.	Ave.	Ave.	Ave.	Ave.	High Ave.	Ave.	High Ave.	V. Sup.	V. Sup.	Sup.	Ave.	Ave.	Border.	High Ave.	20.8
13.	Low Ave.	Low Ave.	Border.	Ave.	Ave.	Border.	V. Impaired	Low Ave.	Impaired	Ave.	Low Ave.	V. Impaired	Border.	Low Ave.	21.2
14.	Ave.	High Ave.	Ave.	Sup.	Impaired	Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	Border.	Ave.	Ave.	21.1
15.	High Ave.	Ave.	Sup.	High Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	High Ave.	High Ave.	High Ave.	22.11
16.	Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	Border.	Low Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	25.4

[a]Average, [b]Borderline, [C] Superior

Table 5. Early injury group individual performance categorizations on WAIS III, WMS-R, and executive function tests.

	WAIS-III subtests						WMS-R subtests				Executive function tasks				Age at Injury
Case	Verbal IQ	Performance IQ	Verbal Comp. Index	Perceptual Org. Index	Working Memory Index	Processing Speed	Verbal Memory	Visual Memory	General Memory	Attention	Hayling	Brixton	BADS Overall	WCST	Yrs:Mths
1.	Ave.[a]	Ave.	Ave.	High Ave.	Low Ave.	Border.[b]	Low Ave.	Impaired	Border.	Border.	Impaired	High Ave.	Ave.	Sup.[c]	28.11
2.	High Ave.	Ave.	High Ave.	High Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	Low Ave.	Low Ave.	Impaired	30.2
3.	Ave.	Low Ave.	Low Ave.	Ave.	Ave.	Border.	Low Ave.	Border.	Border.	Low Ave.	Low Ave.	Low Ave.	Impaired	Impaired	30.5
4.	Border.	Border.	Border.	Low Ave.	Low Ave.	Low Ave.	Low Ave.	Border.	Low Ave.	Impaired	Impaired	Ave.	Low Ave.	Border.	31.2
5.	Ave.	High Ave.	Ave.	Sup.	Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	High Ave.	Ave.	Ave.	Low Ave.	33.4
6.	High Ave.	Sup.	High Ave.	V. Sup.	Sup.	Sup.	Ave.	Ave.	Ave.	Sup.	Ave.	High Ave.	Ave.	Low Ave.	36.2
7.	Impaired	Ave.	Impaired	High Ave.	Low Ave.	Low Ave.	Border.	Low Ave.	V. Impaired	Low Ave.	Border.	Ave.	Border.	Ave.	36.1
8.	Low Ave.	Ave.	Low Ave.	Ave.	Impaired	Border.	Low Ave.	Low Ave.	Low Ave.	Border.	Impaired	High Ave.	Ave.	V. Impaired	38.8
9.	Low Ave.	Border.	Ave.	Low Ave.	Border.	Low Ave.	Ave.	Border.	Low Ave.	Low Ave.	Low Ave.	Impaired	Border.	Ave.	39.7
10.	Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	Low Ave.	Ave.	Low Ave.	Ave.	Ave.	Ave.	Ave.	Ave.	44.4
11.	Ave.	Ave.	Ave.	Ave.	High Ave.	Low Ave.	Low Ave.	Ave.	Low Ave.	High Ave.	Border.	Ave.	Ave.	Low Ave.	46.5
12.	Ave.	Ave.	Ave.	Ave.	Ave.	Border.	Low Ave.	Low Ave.	Low Ave.	Low Ave.	Low Ave.	Low Ave.	Ave.	Average	47.5
13.	Ave.	Ave.	Ave.	High Ave.	Ave.	Ave.	Low Ave.	High Ave.	Ave.	Ave.	High Ave.	Ave.	Low Ave.	Low Ave.	48.2
14.	Ave.	Border.	Ave.	Low Ave.	Ave.	Impaired	Low Ave.	V. Impaired	Low Ave.	Ave.	Impaired	Impaired	Impaired	Border.	49.5
15.	Border.	Low Ave.	Border.	Ave.	Low Ave.	Border.	Impaired	Low Ave.	Low Ave.	Low Ave.	Impaired	Impaired	V. Impaired	Low Ave.	54.3
16.	Border.	Low Ave.	Border.	Low Ave.	Low Ave.	Impaired	Ave.	Border.	Ave.	Low Ave.	Border.	Impaired	Low Ave.	Low Ave.	55.1

[a]Average, [b]Borderline, [C] Superior

Table 6: Hierarchical regression analysis with BADS score, age at injury and BADS/age at injury interaction term as predictor variables and DEX-Insight as the dependent variable with all head-injured participants (N = 32).

Independent variables entered	R^2-Δ	F-Δ	Df	β
Step 1: Continuous predictor variable	.11	3.68†	1,30	—
BADS score	—	—	—	-.63**
Step 2: Categorical moderator variable	.01	.16	1,29	—
Age at injury	—	—	—	-.07
Step 3: Interaction term	.16	6.59*	1,28	—
Interaction effect between BADS and age at injury	—	—	—	.43*

Note. Standardized regression coefficients (β) are shown for the model at step 3.
† $p = .07$, * $p = .02$, ** $p < .01$

Table 7: Hierarchical regression analysis with SRT score, age at injury and SRT/age at injury interaction term as predictor variables, and DEX-Insight as the dependent variable with all head-injured participants (N = 32).

Independent variables entered	R^2-Δ	*F*-Δ	*Df*	*β*
Step 1: Continuous predictor variable	.00	.03	1,30	—
SRT score	—	—	—	-.44
Step 2: Categorical moderator variable	.01	.16	1,29	—
Age at injury	—	—	—	-.07
Step 3: Interaction term	.13	4.11†	1,28	—
Interaction effect between SRT and age at injury	—	—	—	.60†

Note. Standardized regression coefficients (β) are shown for the model at step 3.
† p =.052

Legend for Figure 1. Interaction between BADS and age at injury on behavioral insight

Legend for Figure 2. Interaction between SRT score and age at injury on behavioral insight