Revista Española de Neuropsicología
Vol. 1, No. 4, 83-112

ISSN: 1139-9872

The Phenomenology Of Social Withdrawal Following Brain Trauma

La Fenomenología del Retiro Social tras Traumatismo Craneoencefálico

Roy Sugarman
Brenthurst Centre for Rehabilitation Medicine, Greenside, South Africa

Social withdrawal, according to much of the literature, is the prevailing problem following traumatic brain injury (Cattelani, Lombardi, Brianti & Mazzzucchi, 1998). Even though intellectual and functional sequelae are frequent too, both in adults and children, it appears from multiple regression analysis that the presence of such behavioural problems has the most severe and pervasive impact on all aspects of caregiver function (Marsh, Kersel, Havill & Sleigh, 1998). Despite this, and the increasing awareness of rehabilitation professionals of this presentation, the phenomenon appears to require more careful observation and evaluation. There is increasing evidence of concurrent neuropsychological and psychiatric illnesses that modify the course of this condition. An evaluation of some of the literature that has emerged in the last few years indicates that social withdrawal as a behavioural phenomenon occurs in the context of failed inhibition of emotions, perception and motor activity, and as such represents a collapse of the organism's ability to compete in social environments. A diagnostic model of thinking is proposed as a method of overcoming the present and lamented method of accepting symptoms at face value.

La Fenomenología del Retiro Social tras Traumatismo Craneoencefálico

De acuerdo con la literatura el retiro social es el principal problema tras un traumatismo craneoencefálico (Cattelani, Lombardi, Brianti & Mazzucchi, 1998). Aunque las secuelas intelectuales y funcionales también son frecuentes en adultos y en niños, parece que la presencia de problemas comportamentales tiene una influencia más severa y duradera en aspectos relacionados con el cuidador (Marsh, Kersel, Havill & Sleigh, 1998). A pesar de esto y el crecimiento de conciencia de los profesionales de la rehabilitación, el fenómeno require de observación y evaluación cuidadosa. De hecho, hay evidencia de enfermedades neuropsicológicas y psiquiátricas que modifican el recorrido de esta condición. Se realiza una evaluación de la literatura que ha surgido en los últimos años la cual indica que el retiro social como fenómeno comportamental ocurre en el contexto de falta de inhibición de emociones, percepción y actividad motora y representa un colpaso en la habilidad del organismo para competir en ambientes sociales.

Executive dysfunction has been singled out as being at the core of the loss of social autonomy (Mazaux et al., 1997). Contemporary psychiatric research conclusively demonstrates that although major brain pathways are specified in the genome, the detailed connections that ensue are socially mediated by experience in the world, and the brain is thus jointly fashioned by genotype and phenotype routes (Eisenberg, 1995).

Despite this, no explanatory model has been developed for social withdrawal, and the condition remains only one of a barrage of sequelae described in the literature as emanating from frontal trauma. In a similar vein, most of the emotional and behavioural consequences of brain insult have been described, but again the explanations remain vague. Together with the social withdrawal that attends the affective disorders, this phenomenon remains the major challenge facing rehabilitation specialists, given the need of the patient to return to competition in social spheres (Morton & Wehman, 1995). There is a need to penetrate the phenomenology of social withdrawal, and utilise existing theory to frame a therapeutic approach to address this issue. The hypotheses lead to the conclusion that following brain insult, failures of self-regulation emerge result in rapid cycling mood disorders which have social withdrawal as a major sequel, and should not be diagnosed as disorders of personality alone.

Overview

The theory that the complication of social withdrawal is purely on of the major sequelae of frontal injury is overturned by an appreciation of the widely discrepant conditions in which it is described, not only in traumatic brain injury (Klonoff, Snow & Costa, 1986; Spatt, Zebenholzer & Oder, 1997), but in stroke (Kishi, Kosier & Robinson,1996), dementia (Lopez et al., 1995), Down syndrome (Geldmacher et al., 1997), epilepsy (Austin, 1996), schizophrenia (Gruzelier & Kaiser, 1996), psychosis (Olin & Mednick, 1996), anorexia (Rosenvinge & Mouland, 1990), bipolar spectrum disorders (Perugi et al., 1998), temporal lobe epilepsy and borderline personality disorder (Sugarman & Hartman, 1998), panic disorder (Okasha et al., 1994), atypical panic disorder (Edlund, Swann & Clothier, 1987), substance abuse (Turner & Mayr, 1990), behavioural inhibition (Kerr et al., 1997), aggression (Evans & Short, 1991), attention deficit hyperactivity disorder (Kuhne, Schachar & Tannock, 1997; Stanford & Hynd, 1994), the mentally retarded (Lund, 1986; Reid & Ballinger, 1995), the elderly (Ungvari & Hantz, 1991), those with frontal asymmetry (Fox et al., 1995), and with frontal lobe degeneration (Miller et al., 1991).

Other examples include widely disparate conditions of human existence, namely chronic fatigue syndrome (White et al., 1995), post liver transplant (Plutchik et al., 1998), dialysis (Villa et al., 1998), carcinoma, (Schuth, 1984), muscular dystrophy (Harper, 1983), Rett syndrome (Harding, Tudway & Wilson, 1985), Diogenes' syndrome (Williams et al., 1998), diabetes (Zangerle & Rathner, 1997), low birth weight (Hoy et al., 1992), abuse of CNS stimulants (Schiorring, 1981), war trauma and post-traumatic stress disorder (Johnson et al., 1997; Mollica et al., 1997), precocious puberty in girls (Sonis et al., 1985) in pre-menstrual women (Goodale, Domar & Benson, 1990), in response to neuroendocrine activation in some studies (Dabbs, Jurkovic & Frady, 1991; Granger, Weis & Kauneckis, 1994), and even in depression, when stressors are imposed by strangers (as opposed to intimates) (Meyer & Hokanson, 1985), or in response to complete dentures (Friedman, Landesman & Wexler, 1987), neonatal death (Tudehope et al., 1986), the use of methylphenidate in the mentally retarded (Handen et al., 1991), and in being the only child (Xin et al., 1992).

The need for a unifying model is clear, despite previous attempts at the psychological level (Cochrane, 1983), that do not appear to result in effective rehabilitation strategies.

As an obstacle to rehabilitation, social withdrawal has been neglected (León-Carrión, 1997) possibly because it appears to be described, rather than explained in the literature (Akiskal, 1994). Also, no reasonable model for the underlying neurological substrate exists, despite a swing towards describing such characterological changes in neural terms in recent years (Ratey, 1995).

However, in related conditions, attempts are being made to accurately define and expiate models to address neurological antecedents of psychobiological presentations, e.g. in Attention Deficit Hyperactivity Disorder (Barkley, 1998a; 1998b), or to search for underlying genetic links, e.g. in schizophrenia (Kendler, Gruenberg & Strauss, 1982).

It is clear that behavioural inhibition and social withdrawal, although behaviourally similar, should be differentiated conceptually and empirically, as they present disparate risks for later outcomes (Kerr et al., 1997). Social withdrawal has been treated most often as a unitary trait, a diagnosis, which it clearly is not (Harrist et al., 1997).

Diagnosis, meaning literally 'through knowledge' has unfortunately been reduced to meaningless description. In the contemporary scientific analysis of neuropsychiatric sequelae, the process of diagnosis has been tainted by reductionism, and heterogeneous affective syndromes are forced

to fit regardless of their obvious differences from each other regarding pre-morbid development, course and outcome (Himmelhoch, 1984).

This is no more clearly pointed out than in Othmer, Othmer & Othmer (1998), who accuse psychologists of accepting psychiatric symptoms at face value without relating them to normal brain functions for which the symptom signals malfunction.

In order to avoid doing this with the phenomenon of social withdrawal, the author takes as point of departure the caveat of Akiskal (1994, p. 35):

(It) is necessary to properly attend to the personal needs of the patient and the evolving human drama or tragedy. Formal clinical diagnosis however, necessitates a more penetrating approach, which must consider the phenomenology of the illness, its course, familial patterns and putative aetiologic factors.

Diagnosis as description unfortunately therefore displaces diagnosis as hypothesis, and thus creates descriptive menus such as the DSM-IV (1994).

This more modern approach to research appears vital in such phenomena as social withdrawal, given that such researchers as Thomsen (1992) and Bond (1975) have found that social-emotional factors do dominate in the years following insult. The linkage to the affective disorders, and the deterioration of emotional and behavioural aspects are often neglected, despite the patient's often complete indifference to the surrounding environment that results from classic frontal syndromes (Emilien & Waltregny, 1996).

The role of self regulation: personality, psychometry and social withdrawal

Following on in this way, the records of 1400 neurologically suspect, medico-legal and clinical patients were evaluated retrospectively, focussing on those patients or family who mentioned spontaneously or on directed questioning that social withdrawal was a problem. Although this was never a symptom in isolation, but rather given in passing as one of the usual constellation of symptoms expected post-traumatic brain injury, it has not been treated with much rigour in the common literature (e.g.Lezak, 1995).

In keeping with this, the reported sequelae intimated that characterological psychopathology was present, despite its obvious clinical absence both pre- and post-morbidly. The affective components were neglected however, and it was easy to see how unwary clinicians would label the affective components as Axis-II disorders, and focus more on the usual sequelae related to hypoarousal and dyscontrol.

In all cases two factors emerged.

The first was the regular complaint of change of personality, by now understood together with various degrees of awareness of deficit, as one of the inevitable sequelae to brain trauma (Prigatano, 1996; 1997). Other authors disagree with this (Spatt et al., 1997), although they acknowledge that patients tend to have difficulties in monitoring their specific problems of control of behaviour in social relationships.

The second was more difficult to evaluate as the interviewers often disguised the client's comment on mood swings in terms of increased temper rather than disinhibited temper control. Many families described mood swings: this was then usually clarified in a leading way by the interviewers. This was then interpreted as disinhibition of temper control, also documented elsewhere as a common sequel to such trauma (Lezak, 1995), and hence the underlying inference was lost that this might not be a unitary phenomenon, or have mood components.

However, further perusal of the interview documents showed that the patient's loss of control is most often sudden and short-lived, and followed almost immediately by an extended period of withdrawal. This was reported variously as depression, sulking, petulance, moodiness, isolation, distance, and various other terms as far removed from reality as "pre-menstrual" in some cases.

Affect versus personality

In order to determine which self-reported symptom clusters could be reliably targeted as affect rather than personality variables in social withdrawal, Sugarman and Hartman (1998) embarked on a review of 59 Millon Clinical Multiaxial Inventory III (MCMI-III) records. These were evaluated using NCS Microtest Q software version 4.0c, in order to create a perfect inter rater coefficient. Trends were sought without scientific rigour, given the known difficulties with self-report (Thompson, Batzel & Wilkus, 1992).

34 of these patients with unequivocal recent (approximately 2 years post-injury) highspeed mild-moderate traumatic brain injury (TBI) were evaluated, given the better prospects in this group for rehabilitation, but all showed significant executive dysfunction on detailed neuropsychological assessment, or were excluded. Secondly, severe brain injury is not a necessary criterion for social withdrawal (Finset et al., 1995). The remainder

was made up of a mixed group of clinical patients with personality disorder, and some temporal lobe epilepsy patients (TLE).

In the 25 remaining patients, a psychiatric group overall, 100% of the Borderline Personality Disorder (BPD) patients reported anxiety, as did the TLE patients if taking them alone as a diagnostic group.

In keeping with the idea of social withdrawal, 68% of the TBI patients scored significantly on passive-aggressive scales, with only 45% of psychiatric patients in contrast, and removing the BPD patients, this drops to 34%.

The superficial examination of the full neuropsychological protocol demonstrates at least a *prima facie* case for the link between executive dysfunction and passive-aggressive behaviour, the closest the Millon comes to predicting social withdrawal (McCann & Dyer, 1996), apart from self-defeating behaviour and avoidant coping.

Dysthymia was reported in 65% of the TBI cases, against exactly the same in the psychiatric group overall. This rises to 80% of the pure TLE group and 73% in the BPD group. In contrast, major depression was reported in only 35% of both groups, and 60% in the TLE subgroup. Self-defeating and avoidant behaviour made up 56% and 53% respectively in the TBI group, with 50% claiming a new dependency.

Another feature of the TBI group was a tendency to debasement in 56% of the respondents, with only 34% of psychiatric patients (BPD removed), 54% in the BPD patients standing alone, and 60% in the TLE patients.

Most importantly, the mood swings reported clinically were not evident in the Millon records as Bipolar disorders, suggesting again the subsyndromal nature of these swings when compared to DSM-IV criteria (Koopowitz & Hartman, 1996a/b; 1997 a/b).

A diagnosis of BPD could not be made pre-accident in any of the TBI patients, but post-accident 38% could receive such a classification, and in the TLE group, 60% could, to the unwary simply evaluating the MCMI-III at face value.

Interestingly, schizoid features appear in 41% of TBI patients, but in only 20% of the known TLE patients, all of whom were on mood-stabilising anti-epileptic medication. 34% of the other psychiatric patients record such features.

In keeping with Prigatano (1996, 1997) and Thomsen (1992), the patients' report on interview of these features underestimated the prevalence of the Millon criteria. Even the over-sensitive Millon Inventory (James Butcher, 1998, personal communication) failed to realistically estimate the extent of the psychopathology. The patients in the anxious group also rated

their injuries as significantly more severe and their cognitive functioning as significantly worse than objective neuropsychological testing revealed, in keeping with Fann et al., (1995).

In summary, it appears that in TBI versus general psychiatric populations, the psychometry and clinical interview could lead the examiner to conclude there is personality disorder present. However another set of hypotheses is also possible given the discrepancies between psychiatry groups and the TBI group:

Executive dysfunction is directly linked to personality changes in the TBI patients, leading to rapid cycling, often subsyndromal mood disorder (Sugarman & Hartman, 1998), which results in subsequent social withdrawal after a brief, mixed, anxious, irritable, aggressive affective episode, as it does so often in bipolar spectrum disorder, type II and especially III (Akiskal, 1995; 1996), and often confused with BPD (Wehr et al., 1998). This hypothesis needs to be evaluated in the context of a model that illuminates both on the role of executive dysfunction in socialisation and gregarious human behaviour, and the mood components of human interaction.

Certainly, disinhibition syndromes have been linked to affective disorders (Shulman, 1997), and social withdrawal is more frequent in affective disorders (Johnson et al., 1995). Up till now, the connection between the affective disorders and cognitive disorders has received theoretical attention elsewhere, and this literature provides the basis for directed analysis of the phenomenon of social withdrawal in the context of evolution and socialisation.

Russell Barkley and Antonio Damasio: The role of self-regulation in the socialisation of primates

Barkley (1998a, 1998b) refers in passing to Damasio (1995) in his expiation of the role of self-regulation, but these authors have much in common. Like Akiskal, Damasio is also sceptical of science's presumption of objectivity, seeing scientific results in neurobiology as nothing other than provisional approximations. Damasio (1995), makes passing reference to social dysfunction as a function of time and working memory, noting that Phineas Gage lost the ability to plan his future as a social being. Gage suffered in essence from temporal myopia (Barkley's term) in that he could not decide in the here and now what course of directed behaviour would best serve his temporally distant survival (pp. 33 & 56). Damasio goes on to

describe Barkley's theme of 'prepotent response' as typical of the prefrontally impaired patients whom:

> Emerge…..with a normal intellect…..unable to decide properly, especially when the decision involved personal or social matters (Damasio, 1995, p. 43).

Damasio (1995) reasons that the social domain is different to, say, the realm of numbers, and destruction of the neurological areas that subserve these social functions causes a deficit of application, or of performance, not of skills, as these latter remain intact (p. 49). This perspective finds itself applied later, in Barkley (1998a; 1998b) in his contention that in deficits of self-regulation, the patient has the social skills, but cannot apply them, a deficit of performance at the point of performance, not of skill. The hypothesis is made that the ability of the patient to cope with environmental demands, in other words, compete with other primates, is a function of the suppression of outward behaviour in favour of hidden behaviour, a form of 'poker face' if you will. This is an act of volition, dependent on goal setting and inhibition, as in Lezak's (1995) formulation of executive functioning.

Defining the issues

It is widely understood that active, tackling coping based on social and emotional resources and problem-analysis is superior in many ways to passive coping, with social withdrawal (Heim, 1998), and this latter problem is life threatening, at the core of suicidal syndromes (Ahrens & Linden, 1996; Spirito, Overholser & Stark, 1989). However, few authors have embarked on defining and developing the conceptual model necessary for rehabilitation of such conditions.

With Russell Barkley's emailed permission, the following definitions are adopted in their entirety, and are paraphrased from his works cited above, without direct reference, and inform then on the approach to these entities that rehabilitation should take.

For the purpose of this investigation, self-regulation is taken to encompass any behaviour that changes another behaviour so as to alter a temporally distant, likely consequence. This constitutes a special form of attention to internal mental set, rather than to the environment. It is a self-directed activity, directed at changing one's goals in response to internal utility, of necessity future orientated, or in other words relative to time, and thus needing a special form of sequential memory. The future must be valued over the present, immediate, prepotent context. Such a utility must bridge temporal gaps, thus binding events over time in a continuous flow of

related information. The further the temporal stretch, the further forward in the prefrontal cortex the activation must be.

It is clear from the above that a neural mechanism for response inhibition is a *sine qua nom* for self-regulation. This refers to the capacity to inhibit what Barkley calls the environment- driven and reinforced prepotent response. In essence, in thinking an action through, you have to delay the decision-making process. Firstly this requires a capacity to interrupt any response when the information flow indicates that the response is going to be ineffective, and secondly, you have to resist distraction by inhibiting any competing response patterns in working memory.

In this way, to inform a model for rehabilitation, executive functions should be thought of as those forms of:

(a) self-directed behaviour that (b) bridge the gap in time to (c) alter future consequences by (d) delaying the prepotent response and then (e) modify that behaviour that will (f) alter the future consequence or outcome in (g) a favourable way, and fulfil Lezak's (1995) criteria for assessment.

It is easy to see how Damasio's dismissal of proximal formulations tie in with Barkley's definitions, and together illuminate on prefrontal efficacy in social contexts, whilst both take affective processes into consideration, a novel approach.

The evolution of social interaction: Social Darwinism

Reading through Barkley's work, and in direct email discussion with Professor Barkley, the social necessity of executive functioning becomes clear. This is also entrenched in the multiple works of various anthropologists.

Briefly, as *Homo erectus* emerges, the radius crosses the ulna allowing for pronation of the forearm, and *Homo habilus* can manipulate effectively using the opposable thumb. Homo *sapiens* will follow, and each succession must have the neural software to make use of this hardware, and the speech to pass the secrets of the skills onward, selectively.

But the price of this erect, tool making and manipulative stance is that mankind can no longer enjoy the speed of the predator on all fours, must not have claws that climb or slice, since the hands are now required for finesse, not power. Likewise, jaw development must allow for effective speech, not defence as in the sabre tooth tiger.

In terms of the brain, social pressures at the same time have favoured an extension in the proportion of time primates spend as juveniles, owing to the

extended complex social skills now required (Joffe, 1997). It is therefore essential that this extended period of executive development continue uninterrupted, with better social outcomes for those who sustain injuries to this area after childhood and adolescent stages are completed (Asikainen, Kaste & Sarna, 1996). In keeping with Heim (1998) above, TBI children will thus generate fewer positive assertive and more indirect passive responses to peer group entry situations (socially competitive situations) in general (Warschausky et al., 1997).

In short, man is dependent on, but becomes vulnerable to, proximal competition from his peers, and must move from a solitary existence to a gregarious one, as the wolf pack must, in his own defense. At the same time, the neocortex had to evolve to meet the demands on the software that evolution of this hardware thus created.

The difficulty that arises, and here Barkley's formulations are again pertinent, is that competition is made difficult when all and sundry in the cave era are literally and figuratively looking over the leader's shoulder, and copying what the leader, shaman, firemaker does in order to compete with him. Minuchin & Fishman (1981) note that animals and humans group in hierarchies, and this demands some subterfuge, or a 'hiding' of skills in social circumstances, in the service of effective competition. Effectively, this would mean an internalisation of speech, emotion and motor activity. In other words, public behaviour must become private (Barkley's terminology). Even in animals, this is the case (Annett et al., 1989), where social withdrawal and behavioural competition are linked. There are at least animal studies that link the brain catecholamines with the maintenance of social dominance, and with the noradrenaline/dopamine ratio of importance as well (Serova & Naumenko, 1996).

This is precisely as Barkley formulates it. He regards executive functioning as the most inherited trait. This leads to the invariate ontogenesis of self-regulation: non-verbal memory is the first of the 'special' mechanisms to develop, followed by the internalisation of affect, language and motor activity, and through a process of social Darwinism, reconstitution and the emergence of a verbal working memory system. Reconstitution is a measure of verbal as well as behvioural fluency.

Damasio finds in his cases that this is precisely the problem following prefrontal injury. He notes that reason is the ability to think and make inferences in an orderly, logical manner, and rationality as the quality of thought and behaviour that comes from adapting reason to a personal and social context (see p. 269). Not all reasoning processes are followed by a decision, and thus the patients Damasio saw could no longer act in their own

best interests, since emotionally-informed choice to decide was now absent, as emotions are prompted by a particular mental content, with feeling being the perception of these changes. Their social choices are thus nearly always wrong, and thus no creative thinking emerges.

In terms of his application of social Darwinism, Barkley uses the term 'reconstitution' to describe a process of creating novel behaviour without experience of it. This involves the taking apart and reconstitution of sequences of social behaviour so that each is novel, in essence creating hierarchies of behaviour never seen before, a form of "ideational Darwinism" (1998b, p. 243) that encompasses repeatability, variation, and natural selection in the process of creativity.

Combining the information of Barkley and Damasio illuminates on the way executive functioning impacts on social functioning. The essence of creativity is thus the combination of visual and verbal working memory. In order to compete socially, the human brain has to make all vital public behaviour private. An internal mechanism of simulation is a simulator that has as operating mechanism the inhibition of spontaneous and public thought, emotion and motor behaviour. It can thus run through all the options, attach emotional valence to them, and decide on the public, socially competitive acts that must follow for optimum results to the organism.

Finally then, the creation of internalised cognitive, motor and emotional functions allows for meta-cognition: we can reflect and clarify, self-question, problem-solve, apply rule-governed behaviour, and develop ontologies and epistemologies, namely rules about rules. This meta-cognitive ability refers to a combination of visual and verbal working memory, that allows us to comprehend and extract semantic information from social encounters, and is therefore the template for all encultured behaviour and social competition. We can think about our thoughts, decide on rules about rule-governed social behaviour, and consequently the driver of our social interactive behaviours is the prefrontal cortex, an essential neural component of competitive behaviour. The model then needs to predict neural pathways that subserve such functions.

Barkley (1998a) localises inhibition to the right orbitobasal cortex, extending back to the striatum, and essentially the caudate. Internalisation of emotion and the control of motivation is localised to the mesial prefrontal areas, and the process of reconstitution, so essential in socialization and enculturation, to the dorsolateral prefrontal areas. Again the links to Damasio's (1995) way of thinking are striking. In discussing the

internalisation of emotion, Barkley refers to Damasio's somatic markers in the prefrontal cortex in particular.

For the rehabilitation expert, this constitutes a model for diagnosis and thinking of social withdrawal.

Applications of this model in social withdrawal after TBI

IQ, or rather pure intelligence skills as opposed to performance in the social environment, is thus 'cleaved off' from adaptive functioning, and the disorders of self-regulation that arise are of a performance nature, not of skill deficits. As Wechsler himself put it in 1944, intelligence is the aggregate capacity of the individual to act purposefully, to think rationally, and to deal effectively with the environment, and the correlation of this with IQ falls away in the brain injured (Walsh, 1992). Damasio's words above are thus echoes of David Wechsler's earlier considerations.

TBI Patients have all the social skills. They simply cannot wrest control from the environment by applying these. This they would previously do by creating simulated versions of immediate social reality in their minds, applying various strategies, and then choosing the optimum outcome for re-enactment in the real world. Consequently they are stimulus bound in social interactions as well as impulsive, a trial and error animal rather than an abstract one. The process of simulation describes a particular form of working memory.

Treatment of such a disorder is not internal, but demands point of performance intervention, such as behaviour therapy and medication. These are disorders, in varying degrees, of self-regulation, volition, and capacity to review freewill. As Walsh (1992) notes, while there is no doubt that correlation exists between IQ measures and intelligent social behaviour in people with intact brains, the same cannot be said of those with brain pathology. These are socially maladaptive disorders.

These are also disorders of time, of temporal gradient and temporal myopia, with a limited temporal horizon and hence little comprehension of the future. Consequently the disorder is likely to evolve over time in a developmental way, and hence the long-term findings of Thomsen (1992) and others. The further forward in time, the further along towards the poles of the frontal areas we activate.

Adults may in this way have a far more complex array of deficits than children do, hence the misapplication of the Kennard principle (Levin, Benton & Grossman, 1982) after TBI.

This model of thinking predicts that a patient with prefrontal insult will demonstrate a set of social interactions that are based in context, contingency shaped, and context dependent. This will stand out in marked contrast to their previously attained goal-directed persistence, adaptive and internally grounded and motivated behaviours, with no goal motivation dependent on visible reward. Their IQ may be irrelevant.

Consequently the patient will demonstrate the behaviour Damasio (1995) describes, where emotions do not inform on decision outcome, and the patient cannot choose optimally, but is impulsive. The patient will most often choose the most environmentally prepotent response, and not the best long-term one in terms of favourable outcome.

In the Darwinian sense, the patient cannot compete in the two social spheres, namely of interpersonal relationships and the workplace (Dikmen, Temkin & Armsden 1989; Machuca et al., 1997). Barkley and Damasio thus provide a means to a much more comprehensive model of meta-analysis of the problem of social withdrawal than is presented elsewhere in the mainstream rehabilitation literature (e.g., Miralles, 1997).

In summary, the patient loses the abstract attitude described so early on by Goldstein and others, and remains contextually bound, unable to compete, hence the need for social withdrawal or aggression. However, the situation is not a simple as that, since the model includes reference to the inhibition of emotion, bringing us full circle in our discussion. As León-Carrión (1997) has noted:

> The affective sequelae are often linked to the different cognitive deteriorations. However, one should not expect that affective deficits improve without treatment only because the affected cognitive components are improving. If the affective aspects are not attended to simultaneously, it is possible that they worsen as the cognitive aspects continue improving (p. 420).

Again there is the idea of worsening over time, as Thomsen (1992) and others suggest, and the need for attention to be paid to mood is underlined. Particularly, the patients will describe mood swings rather than elements of mood, and there is a reason for this.

The rapid cycling mood disorders, and mood

The phenomenology of the 1400 patients in this study is clear, especially in one respect. Patients with varying degrees of social withdrawal almost always exhibit subtle, bipolar spectrum rapid cycling mood disorder, but seldom if ever clinically or psychometrically are they truly manic or majorly depressed. This is not a novel finding in any population (Angst &

Hochstrasser, 1994), nor in traumatic brain injury populations (Morton & Wehman, 1995). However, such patients are described as lacking emotional depth in their 'highs' (fatuous) and often vacillating between social euphoria and social disaster, much like Angelman's "Happy Puppets" (O'Donohoe, 1995).

One would expect if the application of the Barkley/Damasio model of thinking above is correct, then rapid cycling depression/euphoria or other more subtle mood shifts are predicted. In essence, the patient will be briefly euphoric, run into some trial of daily living, fail to cope, develop a mixed mood (angry, irritable, and aggressive) and then socially withdraw. This is shown to be the case in most patient records, as it is in the literature (Clark et al., 1988) in attention deficit hyperactivity disorder.

This immediately also brings to mind the bipolar II, BPD, TLE, and other diagnostic entities that all demonstrate such shifts with social withdrawal (Sugarman & Hartman, 1998).

The essential argument is that social withdrawal represents the end point of the collapse of self-regulation, and emerges directly as an affective disorder in itself, rather than just a sequel of prefrontal injury, and perhaps an entity in itself.

Social withdrawal as affective disorder: bipolar II

Bipolar II is best defined as Akiskal did in 1995 as cyclothymic intermorbid or premorbid temperament.

The historic roots of the cyclothymic disorders, part of the subaffective spectrum, are ill defined (Brieger & Marneros, 1997) but are regarded as the likely precursor of the construct of bipolar II disorder. It is often viewed as a mild form of classic manic-depressive illness, but recently is found to be clearly different with regard to genetic, biological, clinical and pharmacological aspects (Vieta et al.,1997), and is more severe with respect to episode frequency. Like the TLE patients, bipolar II's with rapid cycling episodes experience more psychosensory symptoms (Ali et al, 1997), and this may be confusing in the bipolar II, TLE comorbid populations. The depression is however unclear, as in the MCMI-III study above.

72% of patients who look as atypically depressed as TLE and BPD patients are found to have the diagnostic features of bipolar II (Perugi et al., 1998). According to these authors, interpersonal sensitivity occurred in 94%, social phobia in 30%. Anxious and borderline personality features were highly prevalent. The authors go on further to state that the atypicality of the depression is favoured by affective temperamental dysregulation and anxiety

comorbidity, clinically manifesting in a mood disorder that is predominantly in the realm of bipolar II, with avoidant and socially phobic features, as in the above MCMI-III study above.

In the main, rapid cycling and mixed states are distinct, at least according to one of the Perugi et al. (1998) authors, namely (Akiskal, 1996). He comments that there exist ultra-rapid cycling forms where morose, labile moods with irritable mixed features constitute the patients' habitual self, and for that reason are often mistaken with BPD.

Review of the literature further supports the concept of nigral and cerebellar direct and indirect connections with thalamofrontotemporal and basal ganglia circuits compromised in bipolar spectrum disorders (Lauterbach, 1996). After stroke, affecting frontal-basal ganglia-thalamocortical circuits, mood bipolarity was found to be common (Berthier et al., 1996). Social withdrawal, like cycling mood, appears to have a definitive neural underpinning.

Impulse control disorders were compared to bipolar disorders and found to share a number of features, whilst differing in some important aspects (McElroy et al, 1996), with definite social withdrawal (Kerr et al., 1997). Hyperactivity, disinhibition of motor and mood components in bipolar disorder, and the linkage to social withdrawal have in fact been acknowledged for many years (Heninger & Kirstein, 1977).

From the perspective of social withdrawal, recent literature suggests that introversion has a negative effect on the outcome of unipolar depression, whilst extraversion has a positive influence, and indeed, in bipolar affective disorder, social dysfunction is a negative prognostic indicator (Heerlein, Gonzalez & Santander, 1998).

If the model proposed is again correct, then there should be signs of attention deficit disorder in mania, and there are, at least in adolescents (West et al., 1995). These authors emphasise the important implications for this finding. The slow shifts that may take place generally in bipolar spectrum illnesses are not exclusive, and rapid shifts in under 24 hrs do take place (Kramlinger & Post, 1996).

Nor are the bipolar spectrum disorders immune recursively from the effects of psychosocial factors, which may contribute as much as 25-30% to the outcome variance of treatment (Scott, 1995). Nearly half of bipolar patients have uniformly poor psychosocial functioning with less than 25% having steady work performance (Goldberg, Harrow & Grossman, 1995). The interaction between cycling mood and social withdrawal is thus recursive.

As with the above-cited MCMI-III study, 40-45% of subjects in bipolar groups had comorbid anxiety disorder (Borchardt & Bernstein, 1995). Like

ADHD and TBI, bipolar disorder tends to change over time, and chronify, with major social deficits that concern all social functions, and have devastating social consequences that persist for a long time, and these social aspects require psychological intervention (Pe-Na Garcia et al., 1998).

Finally, the bipolar II disorder is found to excessively load the patient for psychosocial dysfunction with statistically more educational, marital and occupational disruption and minor antisocial acts just prior to a discrete hypomanic episode. The course is more protracted and tempestuous with shorter well episodes (Akiskal et al., 1995), and the linking of these cycles to social activity, as is proposed in this study, is again supported.

To paraphrase, the patient with social withdrawal following cerebral insult is best understood by such lability of mood, masquerading as personality dysfunction, intruding into depressive episodes, creating what Akiskal et al., (1995) call an intimate weaving of state and trait. This temperamental profile appears more fundamental in defining the affective dysregulation of the bipolar II subtype than the hypomanic subtypes of the DSM-IV.

There thus appears to be sufficient evidence that the typical phenomenology of social withdrawal provided by the family and patient on clinical interview has a direct relationship with the bipolar II cyclical variation of mood, and that this directly emerges from executive dysfunction. Social withdrawal may thus best be thought of as an affective, neurological based condition, and its severity dependent on the rapidity of mood shifts and the extent of executive dysfunction. This is more useful as a starting point for rehabilitation than an explanation based on personality traits.

The question that the model must still deal with is the effect of failure of executive functioning on self-concept.

Self-efficacy and the concept of self in rehabilitation

Following the model, persons with brain injury find it difficult to simulate, or develop clear self-knowledge about what they have become, and what is left for them to do effectively. Loss of sense of self is conspicuous when they compare their present self with the past (Nochi, 1998).

Diller (1994) notes that after TBI:

Acceptance implies engagement and commitment of the self. Relations between impairments in cognition and self-concept…have not been articulated. Aside from the fact that the cognitive apparatus is diminished, a person may have to recognise that in many ways he or she is different with respect to premorbid capabilities and roles so that an integration of premorbid and postmorbid self must take place. In addition, the role of the disabled has always been denigrated so that the prospect of assuming such a role in the context of bearing the loss of the premorbid self is difficult. Finally, the shaken sense of self that is characteristic

of….TBI…..and the low self-esteem associated with TBI, must contribute to the high incidence of depression. The self-concept has been approached in rehabilitation as a static variable, or as one with only incidental or vague implications for rehabilitation. Although the 'self' is a rather difficult concept to define, one aspect is particularly pertinent for rehabilitation – self-efficacy. Self-efficacy suggests that effort makes a difference, and that the performer is responsible for the difference (p. 11).

Applying Barkley (1998a; 1998b) to Diller's discussion, it is clear that what most impacts on the prefrontal patient is the inability to wrest control of the outcome away from the environment, and to see to it that the simulated scenario becomes reality when acted upon, with as much one to one correspondence as is possible. In failure of self, the patient cannot adequately reinforce others either (Bond & Godfrey, 1997) and becomes socially uncaring (Damasio, 1994).

To defend the 'self' the patient would have to be capable of efficient working memory in order to simulate, across future time, any and all possibilities of outcome, so as to maximise the favourable outcome potential. Otherwise, as they ineffectively simulate, the are at risk for developing self-limiting belief systems about their effectiveness in altering significant events that the injury has caused in their daily lives (Moore & Stambrook, 1995).

Failure to do so affects more than just a feeling of self-efficacy, but directly disrupts the fit of the patient with their culture. Enculturation as an ongoing process of socialisation is derailed, and the patient then becomes increasing peripheral in society, in other words, alienated.

Given that the rules have changed, a necessary part of recovery is meta-analysis of the rules, in order that rule-governed behaviour can emerge again. This fails, as Barkley suggests it will, and the patient is described as socially inappropriate (Damasio, 1994).

Mood internalisation in the Barkley model is easily described as disinhibited. As one patient remarked:

> There are no barriers between the head-injured patient and the general population. We don't have the subtlety…..we can't have masks…..they seem scared of my realness…..so I am weird and they are not…..How do I live like this? (RF, patient record).

The patient poignantly describes her inability to 'hide' her inner aspirations from her peers, and her tendency to blurt out any statement of her thoughts or emotion. One might see this as personality, but it really is a function of mood. The phrases used to diagnose changes in personality:

> *Depend very largely on criteria that are actually descriptive of an affective disturbance…..In fact affective instability is one criterion, and suicide attempts another…..These phrases are mis-describing the recurrent brief depressions, and trying to ascribe them to a personality disorder* (Montgomery, 1993, p. 2).

In this case, and in the case of all brain insult, the tendency to read executive dysfunction or mood cycling as personality has plagued neuropsychiatry for years. Blumer (1984) would have us believe that as we stand at the threshold of understanding the interface of behaviour and its neural substrates, we are ignoring what is perhaps the most dramatic expression of that relationship. No psychiatric disorder such as social withdrawal was ever discovered by statistical methods, and the reason is obvious.

What clinicians recognise as a disorder distinct from all others known to them is invariably a complex entity that presents in many variations, with a linkage to and an overlap with other disorders (Blumer, 1984, p. vii).

The contribution of neuropsychology to theories of the 'self' in this way may emerge from an appreciation that the biological substratum responsible for each subject's behavioural disorder, such as social withdrawal, may be unique in that it is the final result of the interaction of a particular pathology, with a genetically unique brain, in a unique stage of development (Paradiso et al., 1995, p. 539).

Social reintegration of the self depends on recovery of multiple cognitive functions, with physical parameters of less importance (Bond, 1975). But clearly, as noted above, emotional factors weigh perhaps even more heavily with the passage of time as the sense of self-efficacy suffers and more 'simulation' failures occur at the level of self-regulation. In any helpless condition, such as paraplegia, the higher the lesion, the greater the extent of social withdrawal (Voll et al., 1995). The need to apply the strenuous control of social withdrawal may reflect on the need to overcompensate for lack of internal control mechanisms (Hart et al., 1997).

The attending therapist must thus take great care not to ignore the affective components and merely focus on the apparent personality features, with attendant dismissive treatment, and thus the need for a focussed penetration of the phenomenology of social withdrawal in the presence of rapid cycling mood and executive dysfunction.

Conclusion

Patients invariably report mood swings after TBI (Lezak, 1995), but this has not been dealt with adequately to date in the rehabilitation literature (Sugarman & Hartman, 1998). Social withdrawal is a noted sequel as well (León-Carrión, 1997) but has likewise received little attention in the rehabilitation literature, or noted to be concomitant apart from few suggestions in the literature, as noted above, despite dismal outcomes for patients psychosocially in the long term (Thomsen, 1992).

It has been argued here that disorders of self regulation attend mood swings and social withdrawal, and that the patient is thus unable to compete socially and must react with withdrawal and/or aggression followed by social withdrawal.

An investigation of 1400 protocols suggest that passive-aggressive, dependant and avoidant personality disorders predominate, as well as anxiety playing its role, and that this hides the underlying mood disorder, in this case rapid cycling mood, and leads to mismanagement of the subsyndromal patient. The patient will thus be unable to compete, and so relate to any form of debasement in self-report instruments.

The underlying disorder of self-regulation has been discussed, and credence given to the definition proposed by Russell Barkley (1998a; 1998b), which taken together with Damasio's (1994) formulations, result in a model which has the potential to better inform the rehabilitationist's own meta-cognitions of social withdrawal.

After TBI or any insult to the brain that involves the prefrontal areas, the patient can no longer compete in social situations such as the workplace, and social withdrawal is the consequence.

Therapy must therefore involve the graded introduction of the patient to competitive situations again, as failures of metacognition create an increasing sense of isolation, and are seen by others as inappropriate behaviour. This must occur in concrete social situations (Mazaux et al., 1997). Medication of course plays its role in the affective disorders in brain injury, especially the mood stabilisers (Wroblewski et al., 1997), but not without drawbacks.

In Damasio's (1995) words:

"The prefrontal sectors are indeed in a privileged position among other brain systems. Their cortices receive signals about existing and incoming factual knowledge related to the external world; about innate biological regulatory preferences; and about previous and current body state as continuously modified by that knowledge and those preferences "(p. 181)

"A…..target for biomedical efforts should be the alleviation of suffering in mental diseases. But how to deal with the suffering that arises from personal and social conflicts outside the medical realm is a different and entirely unresolved matter "(p. 266).

This is the realm of neuropsychological rehabilitation. As Ken Shulman (1999; personal communication) notes: What is needed now is a well-designed prospective study that incorporates detailed neuropsychological assessment, neuroimaging and clinical outcome evaluation. The connections with theories of self-concept are also worthy of investigation.

References

Ahrens, B. & Linden, M. (1996). Is there a suicidality syndrome independent of specific major psychiatric disorder? Results of a split half multiple regression analysis. *Acta Psychiatrica Scandanavica, Vol. 94*(2*)*, pp. 79-86.

Akiskal, H.S. (1994). The temperamental borders of affective disorders. *Acta Psychiatrica Scandanavica, Vol 89* (suppl 379), pp.32-37.

Akiskal, H.S. (1995). The bipolar spectrum: research and clinical perspectives. *Encephale, Vol 21, Special No 6*, pp. 3-11.

Akiskal, H.S. (1996). The prevalent clinical spectrum of bipolar disorders: Beyond the DSM-IV. *Journal of Clinical Psychopharmacology, Vol 16* (2 suppl 1), pp. 4s-14s.

Akiskal, H.S., Maser, J.D., Zeller, P.J., Endicott, J., Coryell, W., Keller, M., Warshaw, M., Clayton, P. & Goodwing, F. (1995). Switching form 'unipolar' to bipolar II: An 11 year prospectiv study of clinical and temperamental predictors in 559 patients. *Archives of General Psychiatry, Vol 52* (2), pp. 114-123.

Ali, S.O., Denikoff, K.D., Ketter, T.A., Smith-Jackson, E.E. & Post, R.M. (1997). Psychosensory symptoms in bipolar disorder. *Neuropsychiatry, Neuropsychology and Behavioural Neurology, Vol 10* (4), pp. 223-231.

Angst, J. & Hochstrasser, B. (1994). Recurrent brief depression: The Zurich study. *Journal of Clinical Psychiatry, Vol 55* (suppl), pp.3-9.

Annett, L.E., Ridley, R.M., Gamble, S.J. & Baker, H.F. (1989). Social withdrawal following amphetamine administration to marmosets. *Psychopharmacology, Vol. 99* (2), pp. 222-229.

Asikainen, I., Kaste, M. & Sarna, S. (1996). Patients with brain injury referred to a rehabilitation and re-employment programme: social and professional outcome for 508 Finnish patients 5 or more years after injury. *Brain Injury, Vol. 10* (12), pp. 883-899.

Austin, J.K. (1996). A model of family adaptation to new-onset childhood epilepsy. *Journal of Neuroscience and Nursing, Vol. 28* (2), pp. 82-92.

Berthier, M.L., Kulisevsky, J., Gironell, A. & Fernandez Bents, J.A. (1996). *Journal of Neuropsychiatry and Clinical Neuroscience, Vol 8* (2), pp. 160-167.

Blumer, D. (1984). The psychiatric dimension of epilepsy. Historical perspective and current significance. In D. Blumer (Ed.), *Psychiatric aspects of epilepsy*, pp. 1-57.

Bond, M.R. (1975). Assessment of psychosocial outcome after severe head injury. In R. Porter & D.W. Fitzsimmons (Eds.), *Outcome of severe damage to the central nervous system*, pp. 147-157. Amsterdam: Elsevier.

Bond, F & Godfrey, H.P. (1997). Conversation with traumatically brain injured individuals: A controlled study of behavioural changes and their impact. *Brain Injury, Vol. 11*(5), pp. 319-329.

Borchardt, C.M. & Bernstein, G.A. (1995). Comorbid disorders in hospitalised bipolar adolescents compared with unipolar depressed adolescents. *Child Psychiatry and Human Development, Vol 26* (1), pp. 11-18.

Brieger, P. & Marneros, A. (1997). Dysthymia and cyclothymia: historical origins and contemporary development. *Journal of Affective Disorders, Vol 45* (3), pp. 117-126.

Cattelani, R., Lombardi, F., Brianti, R. & Mazzucchi, A. (1998). Traumatic brain injury in childhood: Intellectual, behavioural and social outcome into adulthood. *Brain Injury, Vol. 12* (4), pp. 283-296.

Clark, M.L, Cheyne, J.A., Cunningham, C.E. & Siegel, L.S. (1988). Dyadic peer interaction and task orientation in attention-deficit-disordered children. *Journal of Abnormal Child Psychology, Vol. 16* (1), pp. 1-15.

Cochrane, N.J. (1983). An exploratory study of social withdrawal experiences of adults. *Nursing Papers, Vol. 15* (2), pp. 22-37.

Dabbs, J.M. Jnr., Jurkovic, G.J. & Frady, R.L. (1991). Salivary testosterone and cortisol among late adolescent male offenders. *Journal of Abnormal Child Psychology, Vol. 19* (4), pp. 469-478.

Damasio, A. R. (1995). *Descartes error*. London: Papermac.

Dikmen, S., Temkin, N, & Armsden, G. (1989). Neuropsychological recovery: Relationship to psychosocial functioning and postconcussional complaints. In H.S. Levin, H.M. Eisenburg, and A.L. Benton (Eds.), *Mild head injury*, pp. 229-244. New York: Oxford University Press.

Diller, L. (1994). Finding the right treatment combinations: Changes in rehabilitation over the past five years. In A-L. Christianson & B.P. Uzzell (Eds.), *Brain injury and neuropsychological rehabilitation*, pp. 1-16. Hillsdale, N.J: Erlbaum

Edlund, M.I., Swann, A.C. & Clothier, J. (1987). Patients with panic attacks and abnormal EEG results. *American Journal of Psychiatry, Vol. 144* (4), pp. 508-509.

Eisenberg, L. (1995). The social construction of the human brain. *American Journal of Psychiatry, Vol. 152* (11), pp. 1563-1575.

Emilien, G. & Waltregny, A. (1996). Traumatic brain injury: Cognitive and emotional dysfunction. Impact of clinical neuropsychology research. *Acta Neurológica Belgica, Vol. 96* (2), pp. 89-101.

Evans, S.W. & Short, E.J. (1991). A quantitative and serial analysis of social problem solving in aggressive boys. *Journal of Abnormal Child Psychology, Vol. 19* (3), pp. 331-340.

Fann, J,R, Katon, W.J., Uomoto, J.M. & Esselman, P.C. (1995). Psychiatric disorders and functional disability in outpatients with traumatic brain injuries. *American Journal of Psychiatry, Vol. 152* (10), pp. 1493-1499.

Finset, A., Dyrnes, S., Krogstad, J.M. & Berstad, J. (1995). Self-reported social networks and interpersonal support 2 years after severe traumatic brain injury. *Brain Injury, Vol. 9* (2), pp. 141-150.

Fox, N.A., Rubin, K.H., Calkins, S.D., Marshall, T.R., Coplan, R.J., Porges, S.W., Long, J.M. & Stewart, S. (1995). Frontal activation asymmetry and social competence at four years of age. *Child Development, Vol. 66* (6), pp. 1770-1784.

Friedman, N., Landesman, H.M. & Wexler, M. (1987). The influences of fear, and depression on the patient's adaptive responses to complete dentures. *Journal of Prosthetics and Dentistry, Vol. 58* (6), pp. 687-689.

Geldmacher, D.S., Lerner, A.J., Voci, J.M., Noelker, E.A., Somple, L.C. & Whitehouse, P.J. (1997). Treatment of functional decline in adults with Down syndrome using selective serotonin reuptake inhibitor drugs. *Journal of Geriatric Psychiatry and Neurology, Vol. 10* (3), pp. 99-104.

Goldberg, J.F., Harrow, M. & Grossman, L.S. (1995). Recurrent affective syndromes in bipolar and unipolar mood disorders at follow-up. *British Journal of Psychiatry, Vol 166* (3), pp. 382-385.

Goodale, I.L., Domar, A.D. & Benson, H. (1990). Alleviation of pre-menstrual syndrome symptoms with the relaxation response. *Obstetrics and Gynaecology, Vol. 75* (4), pp. 649-655.

Granger, D.A., Weisz, J.R. & Kauneckis, D. (1994). Neuroendocrine reactivity, internalising behaviour problems, and control-related cognitions in clinic-referred children and adolescents. *Journal of Abnormal Psychology, Vol. 103* (2), pp. 267-276.

Gruzelier, J.H. & Kaiser, J. (1996). Syndromes of schizotypy and timing of puberty. *Schizophrenia Research, Vol. 21* (3), pp. 183-194.

Guerriere, D, & Mckeever, P. (1997). Mothering children who survive brain injuries: Playing the hand you're dealt. *Journal of Social Pediatric Nursing, Vol. 2* (3), pp. 105-115.

Handen, B.L., Feldman, H., Gosling, A., Breaux, A.M. & Mcauliffe, S. (1991). Adverse side effects of methylphenidate among mentally

retarded children with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry, Vol. 30* (2), pp. 241-245.

Harding, B.N., Tudway, A.J. & Wilson, J. (1985). Neuropathological studies in a child showing some features of the Rett syndrome. *Brain Development, Vol. 7* (3), pp. 342-344.

Harper, D.C. (1983). Personality correlates and degree of impairment in male adolescents with progressive and nonprogressive physical disorders. *Journal of Clinical Psychology, Vol. 39* (6), pp. 859-867.

Harrist, A.W., Zaia, A.F., Bates, J.E., Dodge, K.A. & Pettit, G.S. (1997). Subtypes of social withdrawal in early childhood: Sociometric status and social-cognitive differences across four years. *Child Development, Vol. 68* (2), pp. 278-294.

Hart, D. Hofmann, V., Edelstein, W. & Keller, M. (1997). The relation of childhood personality types to adolescent behaviour and development: A longitudinal study of Icelandic children. *Developmental Psychology, Vol. 33* (2), pp. 195-205.

Heerlein, A., Richter, P., Gonzalez, M. & Santander, J. (1998). Personality patterns and outcome in depressive and bipolar disorders. *Psychopathology, Vol 31* (1), pp. 15-22.

Heim, E. (1998). Coping: Status of knowledge in the 90's. *Psychotherapy and Psychosomatic Medical Psychology, Vol. 48* (9-10), pp. 321-337.

Heninger, G.R. & Kirstein, L. (1977). Effects of lithium carbonate on motor activity in mania and depression. *Journal of Nervous and Mental Disorders, Vol. 164* (3), pp. 168-175.

Himmelhoch, J. (1984). Major mood disorders related to epileptic changes. In D. Blumer (Ed.), *Psychiatric aspects of epilepsy*, pp. 271-294. APA.

Hoy, E.A., Sykes, D.H., Bill, J.M., Halliday, H.L., Mcclure, B.G. & Reid, M.M. (1992). The social competence of very low birthweight children: Teacher, peer, and self-perceptions. *Journal of Abnormal Child Psychology Vol. 20* (2), pp. 123-150.

Joffe, T.H. (1997). Social pressures have selected for and extended juvenile period in primates. *Journal of Human Evolution, Vol. 32* (6), pp. 593-605.

Johnson, C.R., Handan, B.L., Lubetsky, M.J. & Sacco, K.A. (1995). Affective disorders in hospitalised children and adolescents with mental retardation: A retrospective study. *Research in Developmental Disability, Vol. 16* (3), pp. 221-231.

Kendler, K.S., Gruenberg, A.M. & Strauss, J.S. (1982). An independent analysis of the Copenhagen sample of the Danish Adoption Study of

Schizophrenia V. The relationship between childhood social withdrawal and adult schizophrenia. *Archives of General Psychiatry, Vol. 39* (11), pp. 1257-1261.

Kerr, M., Tremblay, R.E., Pagani, L. & Vitaro, F. (1997). Boys' behavioural inhibition and the risk of later delinquency. *Archives of General Psychiatry, Vol. 54* (9), pp. 806-816.

Kishi, Y., Kosier, J.T. & Robinson, R.G. (1996). Suicidal plans in patients with acute stroke. *Journal of Nervous and Mental Disorders, Vol. 184* (5), pp. 274-280.

Klonoff, P.S., Snow, W.G. & Costa, L.D. (1986). Quality of life in patients 2 to 4 years after closed head injury. *Neurosurgery, Vol. 19* (5), pp. 735-743.

Koopowitz, L.F. & Hartman, L. (1996a). *Ictal phenomena in the psychiatric population: A clinical survey.* Platform presentation at the 9th National Psychiatry Congress. The Lost City, Pilansberg, South Africa.

Koopowitz, L.F. & Hartman, L. (1996b). *Subictal mood disorder: A clinical survey.* Platform presentation at the 9th National Psychiatry Congress. The Lost City, Pilansberg, South Africa.

Koopowitz, L.F. & Hartman, L. (1997a). *Temperolimbic phenomena in a psychiatric population: A clinical survey.* Platform presentation at the 14th World Congress on Psychosomatic Medicine. Cairns, Australia.

Koopowitz, L.F. & Hartman, L. (1997b). *Subictal mood disorder revisited: A clinical survey.* Platform presentation at the 14th World Congress on Psychosomatic Medicine. Cairns, Australia.

Kramlinger, K.G. & Post, R.M. (1996). Ultra-rapid and ultradian cycling in bipolar affective illness. *British Journal of Psychiatry, Vol. 168* (3), pp. 314-323.

Kuhne, M., Schachar, R. & Tannock, R. (1997). Impact of comorbid oppositional or conduct problems on attention-deficit hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, Vol. 26* (12), pp. 1715-1725.

Lauterbach, E.C. (1996). Bipolar disorders, dystonia, and compulsion after dysfunction of the cerebellum, dentorubrothalamic tract, and substantia nigra. *Biological Psychiatry, Vol. 40* (8), pp. 720-736.

León-Carrión, J.L (1997) An approach to the treatment of affective disorders and suicide tendencies after TBI. In J. Leon-Carrion, (Ed.), pp. 415-429, *Neuropsychological rehabilitation: Fundamentals, Innovations and Directions.* Florida: GR/St. Lucie Press.

Levin, H.S., Benton, A.L. & Grossman, R.G. (1982). Neuropsychological consequences of closed head injury in children. In H.S. Levin, A.L.

Benton & R.G. Grossman (Eds.), *Neurobehavioural consequences of closed head injury*, pp. 189-207. New York: Oxford University Press.

Lezak, M.D. (1995). *Neuropsychological assessment* (3rd Ed). New York: Oxford Press.

Lopez, O.L., Gonzalez, M.P., Becker, J.T., Reynolds, C.F. 3rd., Sudilovsky, A. & De Kosky, S.T. (1995). Symptoms of depression in Alzheimer's disease, frontal-lobe type dementia, and subcortical dementia. *Annals of the New York Academy of Science*, Vol. 769, pp. 389-392.

Lund, J. (1986). Behavioural symptoms and autistic psychosis in the mentally retarded adult. *Acta Psychiatrica Scandanavica, Vol, 73* (4), pp. 420-428.

Machuca, F., Martin Carrasco, J.M., Martin Gonzalez, A., Rodriguez-Duarte, R. & León-Carrión, J. (1997). Training for social skills after brain injury. In J. León-Carrión (Ed.), *Neuropsychological rehabilitation: Fundamentals, innovations, and directions*, pp. 453-468. Florida: Gr/St. Lucie Press.

Malia, K., Powell, G. & Torode, S. (1995). Coping and psychosocial functioning after brain injury. *Brain Injury, Vol. 9* (6), pp. 607-618.

Marsh, N.V., Kersel, D.A., Havill, J.H. & Sleigh, J.W. (1998). Caregiver burden at 6 months following severe traumatic brain injury. *Brain Injury, Vol. 12* (3), pp. 225-238.

Mazaux, J.M., Masson, F., Levin, H.S., Alaoui, P., Maurette, P. & Barat, M. (1997). Long-term neuropsychological outcome and loss of social autonomy after traumatic brain injury. *Archives of Physical and Medical Rehabilitation, Vol. 78* (12), pp. 1316-1320.

Mccann, J.T. & Dyer, F.J. (1996). *Forensic assessment with the Millon Inventories*. New York: The Guilford Press.

Mcelroy, S.L., Pope, H.G. Jnr, Keck, P.E. Jnr, Hudson, J.I., Phillips, K.A. & Strakowski, S.M. (1996). Are impulse control disorders related to bipolar disorder? *Comprehensive Psychiatry, Vol 37* (4), pp. 229-240.

Mcgann, W. & Werven, G. (1995). Social competence and head injury: A new emphasis. *Brain Injury, Vol. 9* (1), pp. 93-102.

Meyer, B.E. & Hokanson, J.E. (1985). Situational influences on social behaviours of depression-prone individuals. *Journal of Clinical Psychology, Vol 41* (1), pp. 29-35.

Miller, B.L., Cummings, J.L., Villanueva-Meyer, J., Boone, K., Mehringer, C.M., Lesser, I.M. & Mena, I. (1991). Frontal lobe degeneration: Clinical, neurophysiological and SPECT characteristics. *Neurology, Vol. 4* (9), pp. 1374-1382.

Minuchin, S & Fishman, H.C. (1981). *Family therapy techniques.* Harvard London: University Press.

Miralles, J. (1997). Aphasia rehabilitation. In J. León-Carrión (Ed.), pp. 313-333. *Neuropsychological rehabilitation: Fundamentals, innovations, and directions.* Florida: GR/St. Lucie Press.

Mollica, R.F., Poole, C., Son, L., Murray, C.C. & Tot, S. (1997). Effects of war trauma on Cambodian refugee adolescents' functional health and mental health status. *Journal of the American Academy of Child Adolescent Psychiatry, Vol. 36* (8), pp. 1098-1106.

Montgomery, S.A. (1993). Recurrent brief depression. *Psycholink, Vol 2* (11*)*, pp. 1-11.

Moore, A.D & Stambrook, M. (1995). Cognitive moderators of outcome following traumatic brain injury: A conceptual model and implications for rehabilitation. *Brain Injury*, Vol. 9 (2), pp. 109-130.

Morton, M.V. & Wehman, P. (1995). Psychosocial and emotional sequelae of individuals with traumatic brain injury: A literature review and recommendations. *Brain Injury, Vol. 9* (1), pp. 81-92.

Nochi, M. (1998). "Loss of self" in the narratives of people with traumatic brain injuries: A qualitative analysis. *Social Science and Medicine, Vol. 46* (7), pp. 869-878.

O'donohoe, N.V. (1995). The EEG and neuroimaging in the management of the epilepsies. *Archives of Disease in Childhood, Vol 73*, pp. 552-562.

Okasha, A., Bishry, Z., Khalil, A.H., Darwish, T.A., El Dawla, A.S. & Shohdy, A. (1994). Panic disorder: An overlapping or independent identity*?*. *British Journal of Psychiatry, Vol. 164* (6), pp. 818-825.

Olin, S.C. & Mednick, S.A. (1996). Risk factors of psychosis: Identifying vulnerable populations premorbidly. *Schizophrenia Bulletin, Vol, 22* (2), pp. 223-240.

Othmer, E., Othmer, J.P. & Othmer, S.C. (1998). Brain functions and psychiatric disorders: A clinical View. *The Psychiatric Clinics of America, Vol 21* (3), pp. 517-566.

Paradiso, S., Hermann, B.P. & Robinson, R.G. (1995). The heterogeneity of temporal lobe epilepsy: Neurology, neuropsychology and psychiatry. *The Journal of Nervous and Mental Disease, Vol 183*, pp. 538-547.

Pe-Na Garcia, I., Martin Gomez, C., Santamaria, C., Sanchez Cubas, S., San Pedro, L. & Lana Moliner, F. (1998). Psychosocial treatment of bipolar disorder. *Actas Luso Espania Neurologica, Psiquiatrica, Cienca Afines, Vol. 26* (2), pp. 117-128.

Perugi, G., Akiskal, H.S., Lattanzi, L., Cecconi, D., Mastrocinque, C., Patronelli, A., Vignoli, S. & Bemi, E. (1998). *Comprehensive Psychiatry, Vol 39* (2), pp. 63-71.

Plutchik, L., Snyder, S., Drooker, M., Chodoff, L. & Sheiner, P. (1998). Methylphenidate in post liver transplants. *Psychosomatics, Vol. 39* (2), pp. 118-123.

Prigatano, G.P. (1996). *Impaired self-awareness and denial of disability: Negative and positive symptoms following brain injury.* Workshop, INS annual meeting, Chicago, Illinois.

Prigatano, G.P. (1997). The problem of impaired self-awareness in neuropsychological rehabilitation. In J. León-Carrión (Ed.), pp. 301-312. *Neuropsychological rehabilitation: Fundamentals, innovations and directions.* Florida: Gr/St. Lucie Press.

Ratey, J.J. (1995). *Neuropsychiatry of personality disorders*. London: Blackwell Science.

Reid, A.H. & Ballinger, B.R. (1995). Behaviour symptoms among severely and profoundly mentally retarded patients. A 16-18 year follow-up study. *British Journal of Psychiatry, Vol. 167* (4), pp. 452-455.

Rosenvinge, J.H & Mouland, S.O. (1990). Outcome and prognosis of anorexia nervosa: A retrospective study of 41 subjects. *British Journal of Psychiatry, Vol. 156*, pp. 92-97.

Schiorring, E. (1981). Psychopathology induced by "speed" drugs. *Pharmacology and Biochemical Behaviour, Vol. 14 Suppl. 1*(0), pp. 109-122.

Scott, J. (1995). Psychotherapy for bipolar disorder. *British Journal of Psychiatry, Vol 167* (5), pp. 581-588.

Schuth, W. (1984). Psychosocial status of the patient with extensive and advanced ovarian cancer. *Onkologie, Vol. 7 suppl. 2*, pp. 6-9.

Serova, L.I. & Naumenko, E.V. (1996). Involvement of the brain catecholaminergic system in the regulation of dominant behaviour. *Pharmacology and Biochemical Behaviour, Vol. 53* (2), pp. 285-290.

Shulman, K.I. (1997). Disinhibition syndromes, secondary mania and bipolar disorders in old age. *Journal of Affective Disorders, Vol 46* (3), pp. 175-182.

Spatt, J., Zebenholzer, K. & Oder, W. (1997). Psychosocial long-term outcome of severe head injuryas perceived by patients, relatives, and professionals. *Acta Neurologica Scandanavica, Vol. 95* (3), pp. 173-179.

Stanford, L.D. & Hynd, G.W. (1994). Congruence of behavioral symptomology in children with ADD/H, ADD/WO, and learning disabilities. *Journal of Learning Disability, Vol. 27* (4), pp. 243-253.

Sonis, W.A., Comite, F., Blue, J., Pescovitz, O.H., Rahn, C.W., Hench, K.D., Cutler, G.B. Jnr., Loriaux, D.L. & Klein, R.P. (1985). Behaviour problems and social competence in girls with true precocious puberty. *Journal of Pediatrics, Vol. 106* (1), pp. 156-160.

Spirito, A., Overholser, J. & Stark, L.J. (1989). Common problems and coping strategies, II: Findings with adolescent suicide attempts. *Journal of Abnormal Child Psychology, Vol. 17* (2), pp. 213-221.

Sugarman, R. & Hartman, L. (1998). The happy puppet: Social withdrawal as and obstacle to rehabilitation following brain injury. Proceedings of the 7th National Congress in Neuropsychology. Cottesloe, Johannesburg, South Africa.

Thompson, P.M., Batzel, L.W. & Wilkus, R.J. (1992). MCMI-III assessments of patients manifesting either psychogenic or epileptic seizures. *Journal of Epilepsy, Vol. 5* (4), pp. 226-230.

Thomsen, I. V. (1992). Late psychosocial outcome in severe traumatic brain injury: Preliminary results of a third follow up study after 20 years. *Scandinavian Journal of RehabilitationMedicine Supplement, Vol 26*, pp. 142-152.

Tudehope, D.I., Iredell, J., Rodger, D. & Gunn, A. (1986). Neonatal death: Grieving families. *Medical Journal of Australia, Vol. 144* (6), pp. 290-292.

Turner, J.A. & Mayr, S. (1990). Interpersonal types among alcohol abusers: A comparison with drug abusers. *Journal of Clinical Psychology, Vol. 46* (4), pp. 500-506.

Ungvari, G.S. & Hantz, P.M. (1991). Social breakdown in the elderly, II: Sociodemographic data and psychopathology. *Comprehensive Psychiatry, Vol. 32* (5), pp. 445-449.

Vieta, E., Gasto'o, C., Otero, A., Nieto, E. & Vallejo, J. (1997). Differential features between bipolar I and bipolar II disorder. *Comprehensive Psychiatry, Vol 38* (2), pp. 98-101.

Villa, G., Capodaglio, E.M., Jurisic, D., Giorgi, I. & Salvadeo, A. (1998). Functional state and indications for a rehabilitation programme of patients in a dialysis centre: A multidisciplinarystudy with ergonomic, physiatrical and psychological evaluation. *Giornale di Italia Medicina Lavare e Ergonomica, Vol. 20* (1), pp. 31-38.

Voll, R., Mayer, U., Krumm, B. & Fichtner, H.J. (1995). Empirical results of adjustment to disability by adolescents with spina bifida and traumatic Paraplegia. *Rehabilitation (Stuttgardt), Vol. 34* (1), pp. 8-15.

Walsh, K.W. (1992). Some gnomes worth knowing. *The Clinical Neuropsychologist, Vol 6,* pp. 119-133.

Warschausky, S., Cohen, E.H., Parker, J.G., Levendosky, A.A. & Okun, A. (1997). Social problem solving skills of children with traumatic brain injury. *Pediatric Rehabilitation, 1* (2), pp. 77-81.

Wehr, T.A., Turner, E.H., Shimad, J.M., Lowe, C.H., Barker, C. & Leibenluft, E. (1998). Treatment of rapid cycling bipolar patients by using extended bed rest and darkness to stabilise the timing and duration of sleep. *Biological Psychiatry, Vol 43* (11), pp. 822-828.

West, S.A., Mcelroy, S.L., Strakowski, S.M., Keck, P.E. Jnr & Mcconville, B.J. (1995). Attention deficit hyperactivity disorder in adolescent mania. *American Journal of Psychiatry, Vol 152* (2), pp. 271-273.

White, P.D, Thomas, J.M, Amess, J., Grover, S.A., Kangro, H.O. & Clare, A.W. (1995). The existence of a fatigue syndrome after glandular fever. *Psychology and Medicine, Vol. 25* (5), pp. 907-916.

Williams, H., Clarke, R., Fashola, Y. & Holt, G. (1998). Diogenes' Syndrome in patients with intellectual disability: 'A rose by any other name'*?* *Journal of Intellectual Disability Research, Vol. 42* (Pt. 4), pp. 316-320.

Wroblewski, B.A., Joseph, A.B., Kupfer, J. & Kalliel, L. (1997). Effectiveness of valproic acid on destructive and aggressive behaviours in patients with acquired brain injury. *Brain Injury, Vol. 11* (1), pp. 37-47.

Xin, R., Chen, S.K., Tang, H.Q., Lin, X.F. & Mcconville, B.J. (1992). Behavioural problems among preschool age children in Shanghai: Analysis of 3, 000 cases. *Canadian Journal Of Psychiatry, Vol. 37* (4), pp. 250-258.

Zangerle, M. & Rathner, G. (1997). Age dependency of coping strategies in children and adolescents with diabetes mellitus. *Psychotherapy and Psychosomatic Medicine, Vol. 47* (7), pp. 240-248.

Zinner, E.S., Ball, J.D., Stutts, M.L. & Philput, C. (1997). Grief reactions of mothers of adolescents and young adults with traumatic brain injury. *Archives of Clinical Neuropsychology, Vol 12* (5), pp. 435-448.

Recibido Julio 27, 1999
Revisión recibida Febrero 10, 2000
Aceptado Febrero 15, 2000