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Neurological Content and Impact on Personality Assessment Inventory Scale Elevations

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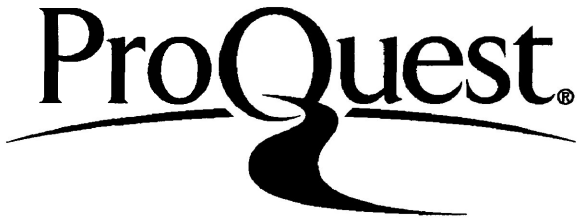

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On completion of a particularly arduous task many people say that they often did not believe that they would ever finish. However, I always believed that I would. Every few weeks or so I was called upon by my children to provide evidence and promises for "the end." I could not have believed otherwise. I thank Jennifer, Jacqueline, Michael, and Elise for making me believe, and for providing such inspiration each and every day.

Abstract

The Minnesota Multiphasic Personality Inventory (MMPI/MMPI-2), the most frequently used measure of personality and emotional functioning in clinical neuropsychological settings, does not meet current psychometric standards. The Personality Assessment Inventory (PAI; Morey, 1991) is a promising alternative to the MMPI, however its usefulness or clinical utility has not yet been formally examined. The present study was conducted to identify items of the PAI that might reflect neurological symptoms and to compare scale scores before and after adjustment for this content. All 344 PAI items were examined by three neurospecialists. Twenty items were identified by these raters as representing physical effects produced by acquired brain injury (ABI) and experienced by at least one in four patients. Fifteen of these items were keyed on either the Somatic scale or the Schizophrenia Thought Disorder subscale. PAI profiles for 62 ABI patients were corrected for this content using the method of Gass (1991) for prorating scale scores on the MMPI-2 (i.e., differential endorsement neurological vs. non-neurologcal items). Although this correction significantly reduced mean sample scores across all affected scales (F [8, 54] = 45.22, $p < .001$), the frequencies of T scores > 69 were most reduced for the Somatic scale, from 32% to 8%, and the Schizophrenia Thought Disorder subscale, from 45% to 16%. Difference scores between ABI and normative responses were calculated. Nine of 20 neurological items were among the highest discriminators between these two populations.

Neurological Content and Impact on Personality Assessment Inventory Scale Elevations

Of all behaviours that follow brain injury, those related to changes in personal motivation and the expression of emotional responses remain the most puzzling, both for researchers and the families of those with acquired brain injury (ABI). Analyses of the utility of psychometric tests of personality and emotional status to neuropsychological research and clinical practice have concluded that these measures are not theoretically useful (Prigatano, 1987). In other words, although they may be helpful in refining observations of psychiatric dysfunction, they provide little insight into the neuropsychologically mediated behaviour, characterological effects, or reactionary involvements that encompass the etiology of post ABI behaviour. However, the idea persists that these tests may be useful for providing information to the clinician regarding the current emotional state or the personality traits of people who have sustained a brain injury.

The call for investigations into the cause of post ABI behaviour (e.g., Prigatano, 1987) stems from the significant expenditure of societal resources in terms of health care for these individuals and their families. Slagle (1990) reported the estimated societal cost as reaching $4 billion annually in the United States as of 1984. Perhaps this is not surprising when one considers that conservative estimates have suggested that 100%, 67%, and 10% of patients with severe, moderate, and mild degrees of injury, respectively, will have ongoing neurologic impairment (Kraus, 1987; Sorenson & Kraus, 1991). The estimates for neuropsychiatric sequelae are most certainly as high (Dikmen, 1998), or higher in certain subpopulations (e.g., mild head injury, Mittenberg, Tremont, Zielinski, Fichera, & Rayls, 1996).

Of the more than 2 million new cases of head trauma that occur annually in North America (Fann, Katon, Uomoto, & Esselman, 1995; Iverson, 1998), those that undergo a neuropsychological evaluation will more likely than not also complete the Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1967) or the Minnesota Multiphasic Personality Inventory-2 (MMPI-2; Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989) as an objective measure of psychological functioning (Alfano, Finlayson, Stearns, & Neilson, 1990; Dunn & Lees-Haley, 1995; Wooten, 1983). However these tests present many problems for the interpretation of post ABI behaviour.

Problems with MMPI/MMPI-2 Application to the Case of Acquired Brain Injury

Some investigators (e.g.,Alfano et al., 1990; Cripe, 1996; Gass & Russell, 1991; Lezak, 1995; Meyerink, Reitan, & Selz, 1988; Prigatano, 1987) have stated that the MMPI and its current update, the MMPI-2, fail to provide accurate descriptions of ABI motivational and emotional behaviours. The failure can be attributed to two general problems. The first problem lies in the poor construct validity of the tests. From the perspective of current psychometric standards, these measures of personality and psychopathology suffer from major theoretical and structural problems. The second problem lies in the lack of knowledge of personality and psychopathology in individuals with acquired brain injury. Specifically, there is no theoretical and little empirical basis on which to compare psychopathology and personality of individuals with ABI to psychiatric or normal populations (i.e., "organic vs psychiatric," or "primary vs secondary" distinctions; Lishman, 1978; 1988; Miller, 1997; 1998; and Malloy & Duffy, 1994; respectively).

Problem 1: Weak Construct Validity of the MMPI

The MMPI scales represent the result of an overreliance on past diagnostic practices and empirical keying techniques (Helmes & Reddon, 1993). Major theoretical concerns about the test's suspect diagnostic criteria and its lack of a consistent measurement model extend to that of the heterogeneous scale content itself. Individual items on the MMPI were selected for scale inclusion based on post hoc analyses of criterion group responses. Therefore, a single item may contribute to a number of different clinical scales, and bear little logical or theoretical relationship to the construct(s) that it measures. The MMPI content scales are associated with increased construct validity. However, they also share items, and require reference to the main clinical scales for their interpretation.

Cripe (1996) described the MMPI as a "murky measure" for neuropsychological assessment. He reported several underlying beliefs and misconceptions that misguide clinicians' thinking. Many neuropsychologists believe that the MMPI measures general emotional adjustment in neurologic patients. As Prigatano has explained (1986), this is far from the truth of the matter. Most probably, this concept has been driven by the logical assumption that elevations on the MMPI are linked to maladjustment. For example, if some people elevate on a scale for reasons of maladjustment, then anyone who elevates on this scale is maladjusted. Or if the normal reference group is well adjusted, then anyone who deviates from this group is maladjusted. Further, if both psychiatric patients and neurologic patients elevate on a particular MMPI scale, then both are elevating for the same reasons and are to be considered maladjusted. This kind of reasoning is only valid if there was only one invariant reason why such an elevation could occur. Further, this reasoning also ignores evidence that 30% of normal controls have elevations on one or

more MMPI scales (Cripe, 1999).

There is also a misconception that the scales on the MMPI reflect unitary traits or states and further, that increasing elevations on these scales represent more and more of a particular trait or mental state. The truth is that the MMPI scales were never psychometrically designed to measure unitary traits or states, or to reflect continuums of these. Rather, the scales were constructed in a manner that would suggest that, as a scale elevates, the probability that the person belongs to the normal reference group is decreasing. However, the idea persists, incorrectly, that increasing elevations reflect more and more of a particular condition (i.e., a T score of 50 means none, a T score of 70 means some, a T score of 90 means a lot more, and a T score of 100 means severe amounts of a particular condition).

While the authors of the MMPI contended that face validity was unnecessary, and hence the content of any given item could bear little obvious relation to any scale that contained it, they paradoxically provided a correction factor (i.e., the "K" scale) to modify test takers' responses. The need for a correction factor of any kind implies that some internal or external factors (i.e., other than the personality traits being measured) are interacting with MMPI items to produce different ways of responding. Investigations (Paulhus, in Helmes & Reddon, 1993; O'Connor & Stefic, 1959; Winfield, 1953) have identified a number of additional influences, or external factors, other than the "personality traits" that MMPI authors believed were related to test taker's reason for responding in a particular direction. While some of these factors are beyond the scope of the present study, those specifically related to the ABI population follow.

Problem 2: Responses of the ABI Population Versus Normal or Psychiatric Populations

Observations that the self-report of individuals from the brain-injured population are misrepresented within profile configurations (Alfano et al., 1990; Gass, 1991; Gass & Russell, 1991; Lezak, 1995; Meyerink et al., 1988; Prigatano, 1987) deliver evidence of the second contributor to MMPI / MMPI-2 inefficacy. Resulting largely from the poor construct validity of this test, the neurological symptoms of the ABI population are often misconstrued as psychiatrically-related. If all individuals who were administered the MMPI comprised a homogeneous population, then all that would be required in the way of norms would be an adequate sample of this population. However, this is not the case. Investigations, within a variety of population settings, have reported significant differences in specific response sets between groups, such as normal males (Long & Graham, 1991), closed head injury (Gass & Wald, 1997), and multiple sclerosis patients (Meyerink, Reitan, & Selz, 1988), and the MMPI norms.

Extrapolating from these observations, Prigatano (1987) stated that the fact that the MMPI was standardized on psychiatric patients presented the problem of discerning the applicability of patterns of test scores and test interpretation to individuals with various forms of brain damage. For example, the endorsement of items that contribute to the Schizophrenia (Sc) scale may simply reflect confusion in thinking secondary to brain injury as opposed to a psychiatrically mediated thought disturbance.

Lezak (1995) related similar concerns. While conceding that the selection and construction of MMPI scales was based on stringent statistical discrimination techniques, she added that research findings had been inconsistent in terms of providing characteristic trends within the brain-injured population. While some very general pattern tendencies that

characterize the responses of many patients with neurological disorders have been observed, to some extent these patterns of MMPI profiles are artifacts of the test items and scale compositions themselves. Alfano and his colleagues (1990), on reviewing the applicability of the MMPI to the assessment of personality and emotional status of the neurologic population, cautioned MMPI use with regard to its literal interpretation in such cases. Based on their analyses of MMPI responses these researchers suggested that a more specific, directly applicable, and shorter measure of personality and emotion status was necessary. In general, elevated MMPI profiles are common within brain damaged populations, due in large part to significant elevations in a combination of neurotic triad scale scores (Hypochondriasis [Hs], Depression [D], Hysteria [Hy]), and/or Schizophrenia (Sc) and Psychopathic Deviate (PD) scale scores (Alfano et al., 1990; Gass & Russell, 1991; Lezak, 1995; Wooten, 1983).

Gass and his colleagues (Gass, 1991; Gass & Russell, 1991), in an attempt to improve the validity of MMPI profiles for the head-injured population, identified those neurologically-related items that may be misinterpreted as psychiatric symptoms. By devising a algorithm for reinterpreting the self-report of these individuals, Gass was able to modify the profiles of those individuals who reported fewer nonneurologically-related items as compared to neurologically-related items. For those individuals who reported symptoms clearly in excess of the characteristic neurological sequelae of head trauma, profiles would remain relatively unchanged.

Some researchers have suggested that Gass's correction factor approach for MMPI-2 interpretation is inappropriate for forensic cases (Dunn & Lees-Haley, 1995), while others (e.g., Cripe, 1997) state that this correction results in the loss of much

information concerning ABI behaviours. Cripe (1997) goes beyond the argument for or against the use of a neurological factor by stating that the use of alternative psychometric measures of psychopathology and personality is necessary to elucidate factors that contribute to post ABI behaviour as well as the degree of rehabilitation achieved by these individuals. While measures such as the Symptom Checklist-90 (SCL-90; Derogatis, Lipman, & Covi, 1973) are useful for the evaluation of patients who are not able to complete longer test forms, they do not provide as much information to the clinician concerning the patient's current level of emotional and social functioning. In his review of the concept of personality and assessment of brain impaired patients Cripe urges neuropsychologists to review some of the more recently developed tests in clinical psychology including the Personality Assessment Inventory (PAI) (Morey, 1991). Although he states that the PAI is a "promising alternative" to the MMPI, Cripe cautions that, because there has been no research with the PAI and neurologic patients, the extent to which the self-report of neurologically-related symptoms is misconstrued within psychiatric constructs is unknown. While other alternatives are availableAnother measure of emotional functioning, the Symptom Checklist-90 (SCL-90; Derogatis, Lipman, & Covi, 1973)

The use of an alternative measure, such as the PAI (1991), represents the first step toward meeting the needs of both clinicians and researchers to clarify the involvement of emotional and personality factors in the expression of ABI symptomology. It seems reasonable to assume that researchers *and* clinicians would benefit from the use of an assessment technique that was well-constructed in psychometric terms.

While the PAI has been used more recently with increasing frequency in a variety of psychological assessment settings, including clinical neuropsychological assessments (M. A. Mountain, personal communication, February 29, 2000), and has been suggested as a favourable alternative to the MMPI in guidelines for neuropsychological assessment of the ABI population, to date there has been no systematic analyses of the effect of neurological content on PAI profiles.

The Personality Assessment Inventory (Morey, 1991)

The PAI was developed to provide measures of constructs that are of greatest importance to clinicians for evaluative purposes. It has been described by clinicians and researchers as a welcome improvement to the existing standard of psychometric tests of personality and emotional functioning (Bell-Pringle, Pate, & Brown, 1997; Boone, 1998; Helmes & Reddon, 1993; Rogers, Ornduff, & Sewell, 1993; Rogers, Ustad, & Salekin, 1998; White, 1996).

A study of the preliminary use of the PAI as a screening instrument within an adult mental-health setting argues that the PAI has proved a more accurate diagnostic predictor of DSM-IV diagnoses than that provided by the traditional psychiatric approach (86.7% versus 62.8%) (White, 1996, p. 38). Others who compared the accuracy of the MMPI-2 and the PAI for making a specific diagnosis observed correct classification of 86% of these patients based on a single scale of the PAI, while a similar diagnosis using MMPI-2 three-scale configuration was correct for only 9% of the same patients (Bell-Pringle, Pate, & Brown, 1997).

Described as providing a useful instrument for differentiating specific forensic groups (e.g., "abused," "dangerous or psychopathic offenders"), the PAI has been used

increasingly for forensic purposes in correctional populations, both as a screening device and within corrections-based psychiatric hospitals (Rogers, Ustad, & Salekin, 1998; Wang et al., 1997). Those working within these settings have stated their need for test instruments that are short, easy to read, able to assess response sets and response styles, able to detect and assess the potential for violence (e.g., suicidality and aggression), able to detect malingering, as well as being able to assess severe Axis I disorders and problematic Axis II disorders. The PAI has been successful in such settings, producing a data base from which population-specific characteristic profiles have emerged, as well as providing superior performance for meeting the above-mentioned specified needs. As such, further investigation of PAI profiles for different forensic populations appears a productive area for future research.

Morey, the author of the Personality Assessment Inventory, has described its use in the diagnosis and treatment planning for patients with cognitive disorders (1991). While the PAI does not detect the presence of this type of dysfunction, it provides discriminant validity that is useful for assessing both emotional and personological aspects of a client with suspected or confirmed cognitive impairment. During ongoing test construction and validation procedures (Morey, 1996), particular patterns of scale elevations have been observed to coincide with organic and cognitive disordered clinical sample groups including elevations of somatic scales (SOM-H score usually representing the greatest elevation of the three), depression scales (vegetative symptoms or DEP-P), and the schizophrenia thought disorder subscale.

Test Construction

Unlike the MMPI, the PAI was developed based on a construct validation framework that emphasized the importance of both rational and empirical methods of scale

development (Morey, 1991). Item development and selection was assisted by adherence to a theoretically informed approach. Following the sequential construct validation strategy proposed by Loevinger (1957) and Jackson (1970), the PAI went through four iterations, as well as the consideration of an additional number of item parameters. Morey was particularly concerned that no single quantitative item parameter should be used as the sole criterion for item selection. Each PAI scale was constructed to include those items that addressed the unabridged range of severity of the construct (i.e., milder to more severe forms). By so doing, milder items could most effectively distinguish clinical subjects from normals, while those that reflected more severe pathology would similarly discriminate among diverse clinical groups.

Test Reliability and Validity

The internal consistency and temporal stability of the PAI scales reported by Morey (1991) have been reproduced in more recent experimental settings (Boone, 1998; Schinka, 1995). Internal consistency alphas for the PAI full scales for normative (N = 1,000), college student (N = 1,051), and clinical (N = 1,246) samples (1991) are consistently high (median alphas of .81, .82, and .86, respectively). The temporal stability of the PAI full scales in community and college samples, as assessed through test-retest reliability measures (Morey, 1991), is quite good, with mean absolute T score change values on the order of two to three T score points for most of the full scales. Further, the configural stability of the 11 clinical scales within this population, at a median correlation of .83, indicates a substantial degree of stability in profile configurations over time.

Although the clinical relevance of the PAI to contemporary issues of personality and psychopathology is superior to that of the MMPI, in large part, due to its excellent

construct validity, it has been limited by the sparsity of literature associated with its usage (White, 1996). This does not detract from the lengthy and comprehensive validation procedures that went into its construction however. The PAI manual alone contains correlations of individual scales with more than 50 concurrent indices of psychopathology (1991). Following test construction, a number of the best available clinical indicators (e.g., Beck Depression Inventory, Beck Anxiety Inventory, Wiggins scales, State-Trait Anxiety Inventory [1983], Hamilton Depression Scale, Diagnostic and Statistical Manual of Mental Disorders semi-structured interviews) were administered concurrently to various samples in order to determine convergent and discriminant validity.

Perhaps the best current indicators of PAI validity come from a number of recent investigations in both clinical and forensic settings, some mentioned previously within the PAI utility section, that have found the test to be superior for diagnostic, as well as treatment planning purposes. Many of these researchers have found the PAI validity scales to perform reliably in the assessment of the potential influence of certain response tendencies on PAI test performance (i.e., typical profiles have emerged). Two of these 4 validity scales, Inconsistency and Infrequency, assess deviations from conscientious responding, while the other two scales, Negative Impression and Positive Impression, assess efforts at impression management by the respondent.

Valid responses within PAI protocols are facilitated by both the face validity of PAI items, and the low reading ability level required for their understanding. Further, because the development of the PAI placed priority on both convergent and discriminant validity, clinical interpretations of PAI protocols are relatively straightforward.

Administration and Interpretation

The PAI is a self-administered, 344-item inventory that allows responses on a four-alternative scale: "totally false," "slightly true," "mainly true," and "very true." Reading level analyses of these items indicate that a reading ability at the grade four level is required to complete the test, comparatively lower than other tests (Schinka & Borum, 1993). Further, the PAI professional manual itself is well laid out (i.e., organized and visually appealing), replete with examples, and easy to read.

Comprised of 22 nonoverlapping full scales, the PAI provides 4 validity scales, 11 clinical scales, 5 treatment scales, and 2 interpersonal scales. Nine clinical scales and one treatment scale contain a number of conceptually derived subscales that were designed to facilitate the coverage of the full scope of complex clinical constructs, as well as the interpretive process. Six response distortion indicators for the PAI were recently evaluated for their effectiveness in conditions of positive impression management, malingering, and honest responding (Morey & Lanier, 1998), in order to validate and improve their specific contributions to such test interpretation.

As opposed to MMPI interpretation, the interpretation of PAI responses at the item level is meaningful, as the content of each item was assumed to be critical in determining its relevance for the assessment of the construct (Morey, 1991). Interpretive hypotheses may therefore be generated at four different levels: the individual item level, the subscale level, the full scale level, and the configuration level.

Subscales, developed to aid the isolation of core elements of the different clinical constructs that the PAI measures, serve to clarify the meaning of full-scale elevations. Full scale T scores may be compared to two referents: those expected scores based on the

community or the clinical sample norms. The configuration of the PAI profile, the highest interpretive level, provides a combination of information that is greater than any of its parts. A number of approaches are provided for studying the configural use of profile data. With respect to mean profiles, the PAI manual (1991; Chapter 9) provides those of 24 different groups, isolated on the basis of diagnosis (e.g., major depression) or a particular behaviour problem (e.g., recent suicide attempt). Further, one and two-point code types associated with 14 different diagnoses are identified (1991; Appendix J).

Cluster analyses, used to determine PAI modal profiles, have made it possible to express a greater range of information within profile interpretations. The mean profiles for 10 different clusters that are frequently observed in clinical practice are accompanied by a narrative describing the expected response style, current emotional status, behavioural problems, and other possible historical factors based on empirical research with such populations.

Treatment Planning

As treatment planning is a critical issue for neuropsychological assessment, an instrument that can glean important information relevant to this process will enhance treatment-related decision-making. The PAI can identify the particular needs of the patient and advise regarding the choice of setting for treatment, the need for medications, the patient's suitability for psychotherapy, the selection of therapeutic targets, and assessment of change (Morey, 1996).

The assessment of treatment motivation is based on the general assumption that an individual is more willing to make an effort to change current behaviour patterns if they are dissatisfied with their current level of functioning. Further indications of treatment

efficacy involve the self-report of patients who are willing to engage in the following activities; an honest report regarding self and difficulties, the recognition of symptoms as psychological in nature, both introspection and curiosity conerning their behaviour and motives, the consideration of new ideas as well as the perspective of others, the expectation to achieve realistic goals for treatment, and putting forth a reasonable amount of effort to achieve a successful outcome.

In addition to providing an account of the individual's potential to benefit from treatment the PAI predicts impediments and assets to the treatment process by identifying personal levels of friendliness, likability, conscientiousness, self-discipline, impulse control, defensive style, internalization, empathy, parental factors, and social supports.

Summary and Purpose of Present Study

The reality of clinical practice with neurological populations remains one where clinical methods are continually compared and optimized for their use with these populations. The MMPI and MMPI-2, although frequently used in clinical neuropsychological evaluations fail to meet current psychometric standards. The PAI does meet contemporary psychometric requirements, and has been successful in a variety of clinical and research settings. However, the usefulness of the PAI for clinical neuropsychological evaluations cannot be judged in absolute terms (i.e., based solely on its psychometric properties). Investigations that explore the applicability of PAI theoretical constructs to neurological populations are necessary for examining the possible utility of this test within a clinical neuropsychological context.

Preliminary clinical use suggests that those PAI scales that measure constructs associated with somatic complaints (i.e., Somatic Complaint [SOM] subscales contain

items that reflect complaints and concerns about physical functioning and health matters in general), depression (DEP subscales), confused thinking (e.g., the Schizophrenia Thought Disorder subscale [SCZ-T]), and emotional lability (e.g., the Borderline Affective Instability subscale [BOR-A]) are frequently elevated (M. A. Mountain, personal communication, February 29, 2000). Other clinical use has suggested that the symptoms of mild head-injury are highly represented in elevated SOM and DEP PAI scales (Cripe, 1999).

The purpose of the present study was three-fold: (1) to examine the extent to which neurological content within the PAI affects the characterization of post ABI behaviour along psychiatric dimensions; (2) to examine neurological content in terms of its structure and dimensionality within an ABI sample; and (3) to examine endorsements that discriminate ABI from normative, and clinical from normative PAI samples.

The following three hypotheses were made. First, that neurological content on the PAI results in the overestimation of psychopathology on the SCZ-T, DEP-P and three SOM subscales. Second, that selected discriminant items ABI versus PAI normative sample would differ from those selected discriminant items PAI clinical versus normal samples.

Method

The present study examined the extent and impact of neurological content on the PAI profiles of an ABI sample following previous methods for neurologically related item identification and neurological adjustment (Gass, 1991; Gass & Russell, 1991).

Participants

Professional Raters

Three neurospecialisits (a neurosurgeon and two physiatrists) examined each of the 344 items that comprise the PAI for neurological content. These experts were instructed

to identify those items that represented "actual physical (not emotional) effects commonly produced by brain injury, and observed to occur in at least 1 out of 4 patients." Each item was assessed by each rater in such a manner. No information about relationships between PAI scales and their items was given, except for the purpose of briefly summarizing the goals of the study (i.e., refining neuropsychological assessment of the ABI client and the identification of neurological symptoms within tests that assess emotional and behavioural problems).

Patients

Archival data from the hospital records of 76 individuals with acquired brain injury that had completed a PAI during a neuropsychological outpatient evaluation (over a span of three years) were examined. These records represented a number of neurological groups within the ABI population including traumatic brain injury (TBI)), stroke, anoxia, and neurodegenerative-related injuries. The main criteria for inclusion in subsequent analyses were valid response profiles as assessed by validity scales, having completed the minimum number of responses that is required for PAI interpretation (95% of items completed, or 327 of the 344 items), and a history free of psychiatric problems including alcohol or drug abuse.

Morey (1991) urges caution when interpreting the profiles of those individuals that do not meet requirements for validity scale scores (1991). PAI computer interpretation programs provide a cautionary note or no interpretation in the case of individuals who respond in a manner that suggests invalid responses due to extremes in inconsistency (ICN scale), negative impression management (NIM scale), or positive impression management (PIM scale) patterns of response. Invalid profiles were found in five cases (i.e., T scores >

70 on ICN scale, or > than 72 on PIM or NIM scales), and one case did not satisfy requirements for 95% completion. Further, eight case histories included alcohol or drug abuse and were not used in the present study.

Of the remaining 62 valid case histories, information concerning ABI diagnosis was recorded including, TBI due to motor vehicle accident, falls, or impact by blunt objects (n = 46), cerebrovascular accident (CVA) (n = 9), anoxia (n = 4), and neurodegenerative conditions (n = 3). Demographics related to age, gender, education and length of time post injury were recorded. The mean age of the 50 male and 12 female sample was 39.6 years (SD = 11.9). The mean education level achieved was 12.4 years (SD = 2.4). Twenty-five of 59 cases completed a PAI less than one year following their injury (M = 24.1 months, SD = 24.1 months). Three cases were not included in the latter groups due to undetermined dates for injury.

Procedure

Each PAI response set was examined for discrepancies between the ABI completed copy and the computer-generated report. All item scores, scale scores and subscale scores of the PAI and subject demographic data was entered into a statistical software program (SPSS Version 7.5 [1996]).

Scale raw scores for each subject were adjusted for their neurological content. Twenty PAI items had previously been identified by 2 of 3 raters as neurologically related items (NRIs). As an added measure of inter-rater reliability for the 344 items, a generalization of kappa was calculated. Using Fleiss' method for more than two raters (1971), the agreement level between the three raters for each of the items was adjusted for the agreement due to chance. The resulting kappa (.63) was not particularly good.

However, rather than improving the reliability of the ratings by excluding items, it was decided that an overrepresentation of NRIs would better suit the purposes of this exploratory analyses (i.e., versus an underrepresentation of NRIs).

Because subtracting NRI responses from scale scores may overcorrect for neurological content, the present study followed the methodology of Gass and Russell (1991) for correcting for neurological content. The total sum NRI responses for each affected scale were prorated for each subject according to the measure of total sum *non*neurologically-related item (NNRI) responses for the same scale. The equation used for correction results in reduced scale scores for those individuals who endorse a number of NRIs but relatively few NNRIs. Conversely, those scores that represent a number of NNRIs but relatively few NRIs, would remain largely unmodified from the original.

The equation used by Gass and Russell (1991) was modified in this study in order to reflect the Likert response format of the PAI. All raw scores for those scales and subscales affected by neurological content were adjusted in the following manner. The Adjusted raw score (ARS) = the sum of nonneurologically-related item responses (sum NNRIRs) + (the sum of neurologically-related item responses [NRIRs] X sum NNRIRs / total possible response sum NNIRs). In other words, the NRI response scores and the NNRI response score were summed separately. The total sum NRI score was adjusted by multiplying this total by the ratio of total NNRI responses to total possible NNRI responses. The total sum NNRI score was then added to the adjusted total sum NRI score to produce a raw score that had been adjusted for neurological content. These raw scores, further referred to as post-adjustment measures, for each neurologically affected scale were transformed into T scores using the tables for normal standardization sample statistics (Tables 1 and 2,

Appendix A, Morey, 1991).

Analyses of the Extent of Neurological Content and the Impact of Adjustment

Univariate and multivariate analyses of the effects of a correction for neurological content (i.e., within subject differences pre- and post-adjustment) were performed using a General Linear Model repeated measures design. A priori significance for these analyses were set at $p < ,01$. Analyses of within subject group effects (i.e., age, education, time post-injury, diagnosis and gender) were extended to correlational and ANOVA techniques A priori significance was set at $p < .05$ for age, education and time post-injury group factors, and at $p < .10$ for group factors related to gender and diagnosis (i.e., relatively small group or cell sizes for females, and CVA, anoxia and neurodegenerative diagnoses).

Analyses of the Structure or Dimensionality of NRIs

In order to examine both the content of NRIs and their levels of endorsement by the ABI sample, a principle components analysis (PCA) was conducted. A double-precision FORTRAN routine (in Longman, Cota, Holden, & Fekken, 1989a) was chosen to determine the correct number of factors to be extracted. Designed for parallel analysis of principal components, this routine calculates both mean and upper-percentile eigenvalues from a specified number of correlation matrices generated from random normal deviates. A comprehensive comparative study of this method of parallel analysis to that of other Monte Carlo methods suggests that it is more accurate and reliable (Longman et al., 1989b). The method for determining the correct number of factors was as follows. On observing those eigenvalues (greatest to least, and greater than one) that correspond with those of an initial unrotated PCA, factors were counted to the point where they remained greater than those of the PCA. The PCA was then repeated using the determined number

of factors and choice for rotation. Varimax rotation was chosen for the initial analyses of NRI responses.

Analyses of the Structure or Dimensionality of ABI Versus Normative, and Clinical Versus Normative Sample Groups

ABI Versus PAI Normative Samples

Item by item analyses of differential responses between the ABI and the normative PAI reference group (N = 1, 000; Table G-1, Morey, 1991) were performed using t-tests. The top discriminant responses between the ABI and the normative samples were selected based on a priori criteria for a number similar to that of identified NRIs and according to the following method. Mean difference scores that represented the highest t-test statistics were examined in terms of their deviation from the normative mean for that item. Items that represented ABI mean responses that were at least one standard deviation away from that of the normative mean, and were endorsed as at least "slightly true," in 50% of cases and as at least "mainly true" or "very true" in 25% of cases were included in subsequent analyses.

Thus, the mean ABI response to these items lay maximally outside of the range of experience for the majority of the normative sample. Subsequent PCA of ABI discriminant item responses followed the method of the previous analysis. That is, the correct number of factors was determined and varimax remained the choice for component rotation.

Clinical Versus Normative PAI Standardization Samples

Analysis of the content that underlies discriminant item responses of the PAI clinical group (N = 1, 246; Table G-1) versus normative group was conducted. The selection of items was based on criteria similar to that of the previous discriminant analysis (i.e.,

minimum of one standard deviation between means of ABI and normative group). However because frequencies of endorsement were not available for a representative subset of this population, further criteria were modified in the following manner. A minimum average clinical endorsement of "slightly true," (i.e., a mean of at least 1.0) was required. Further, in order to reduce the shared variance component of the clinical and the normative responses, a ceiling of two standard deviations was imposed on selected discriminant items (i.e., clinical standard deviation plus normative standard deviation less than or equal to 2.0). Because a representative number of clinical and normative cases were not available at the time of this study a PCA was not possible.

Results

PAI Neurologically-Related Items and Affected Scales

Twenty items were endorsed by neurospecialists as being representative of the physical consequences of acquired brain injury and observed to occur in at least one of four ABI patients (see Table 1). These items are categorized according to scale inclusion. The three Somatic Complaints (SOM) subscales including Conversion (SOM-C), Health Concerns (SOM-H), and Somatization (SOM-S) contained in total 11 of the 20 NRIs. These NRIs represent 45% of the total items of the SOM scale. This scale contains items that reflect the medical complications of ABI. Five of the eleven items refer to the general restrictions due to medical and health problems, and the difficulties inherent in treatment. These items were endorsed as "mainly true" or "very true" in 21% to 69% of cases. The remaining 6 of 11 SOM NRIs reflect specific somatic symptoms including numbness, changes in vision, and pain. These items were endorsed with less frequency, as "mainly true" or "very true" in 23% to 33% of cases.

Of the remaining affected subscales, the SCZ-T subscale is the most highly represented in the remaining NRI responses. Three of this scale's eight items were endorsed by 26% to 48% of the sample as "mainly true" or "very true." These items represent cognitive attentional difficulties. Two of the six items of the Physical Aggression (AGG-P) treatment (AGG) subscale were identified as NRIs. However, only 18% to 21% of respondents endorsed problems related to the poor control of anger as "mainly true" or "very true."

The Physical Depression (DEP-P) scale comprises that part of the Depression Scale (DEP) that assesses the somatic symptoms of the psychiatric construct. These include items related to a change in the level of physical functioning, activity, and energy and also includes symptoms related to disturbances in sleep patterns, a decrease in level of sexual interest, loss of appetite, and weight loss. Only two of these items were identified as NRIs and both of these items are related to content concerning psychomotor slowing. While 75% of the sample endorsed at least one of these items as "slightly true," 66% of these individuals reported them as "mainly true" or "very true." One of the eight items of the Physiological Anxiety (ANX-P) subscale was identified as a NRI. This item measures the experience and expression of stress in somatic form. In total 42% of the sample reported the experience of dizziness under pressure as "slightly true," while only 15% of the sample stated this was "mainly true," or "very true." The remaining and least frequently endorsed NRI is from the Negative Impression Management (NIM) validity scale. This scale contains nine items that have relatively low endorsement rates among clinical and normal subjects. The NRI represents content that would indicate exaggerated unfavourable impression or malingering in these sample groups. However, 6% of the ABI sample

endorsed as "mainly true" or "very true" the fact that they sometimes cannot remember who they are. This was reported as at least "slightly true" for 19% of the cases.

Analyses of PAI Profiles and the Effects of Neurological Adjustment

PAI Profiles

Summary statistics for PAI subscale and full-scale raw-scores are provided in Table 2 and Figure 1, respectively. The results of the neurological adjustment can seen in Figure 1 as well as Figure 2. The latter figure reports the frequency of pathological elevations in both the original sample and the adjusted sample. Of the 62 subjects, 15% exhibited pathological elevations of only one scale, 10% of two, and 30% of three or more main scales. However, 28 of the 62, or 45%, did not exhibit any pathological elevations. Further, 40% of these cases did not exhibit any significant subscale elevations. The SOM and DEP scales were the most frequently pathologically elevated of the clinical scales with 32% of sample showing T scores higher than 69. With the adjustment for neurological content, these percentages decreased to 8% and 27%, SOM and DEP, respectively. The subscales of the SOM were elevated for 31% to 34% of the ABI sample, with adjustment accounting for reduced frequencies of 19% for SOM-H, and 15% for each of SOM-C and SOM-S. A pathological elevation of the DEP-P subscale was reduced in only one case, suggesting that the relatively greater decrease in the frequency of similar pathological elevations of the main DEP scale represents the extension of NRI adjustment from the subscale level to the main scale level.

Pathological elevations of the ANX scale were observed to occur in 15% of cases. This frequency remained unchanged post adjustment. However, one case of ANX-P elevation was eliminated post adjustment. The Anxiety-Related Disorders (ARD) scale was significantly elevated in 16% of the cases. Twenty-four percent of the ABI sample

reported pathological elevations of Traumatic Stresses (ARD-T) (i.e., disturbing events of the past that continue to cause distress and produce anxiety). Of the Borderline Features (BOR) scale, only the Affective Instability (BOR-A) subscale was elevated at a frequency similar to that of the main scale (16% of cases). The item content of this subscale includes the report of labile and/or extreme mood. The other subscales represent uncertainty about major life issues, lack of purpose, unstable relationships, and impulsive behaviours that have a high potential for negative consequences. About 10% of the populations exhibited significant problems in one or more of these areas. Pathological elevations of the AGG main scale occurred in 16% of the cases. Neurological adjustment resulted in a T score of 69 or less in two cases for the AGG-P subscale and in three cases for the main scale.

As can be seen in Figure 2 the total raw mean scale scores for Mania (MAN) and Paranoia (PAR) did not deviate greatly from the PAI normative sample as compared to other ABI mean scale scores. However, the subscales show elevation frequencies in 3% to 5% of the cases, and in 3% to 10% of the cases, MAN and PAR, respectively. The Antisocial Features (ANT) scale was elevated in 13% of cases, with subscale elevation frequencies ranging from 8% to 15%. A NIM validity T score of 69 or greater was reported in 21% of the cases, with the neurological adjustment reducing this frequency to 19%.

Five individuals exhibited pathological reports of Suicidal Ideation (SUI), while five individuals reported a significant lack of social support (Nonsupport Treatment Scale [NON]). Three individuals reported significant Drug Related Problems (DRG) and one individual reported significant Alcohol Related Problems (ALC).

Analyses of the Effects of Neurological Adjustment

Multivariate analyses of the original and adjusted T scores for the eight affected scales or subscales of the PAI resulted in the detection of an overall significant effect for the neurological adjustment, F (8, 54) = 45.22, $p < .001$. The univariate test results are listed in Table 3. All differences between original and adjusted scale T scores were significant $p < .001$, except for the NIM scale which was significantly reduced with $p = .001$. Effect sizes were calculated and reported based on the recommendations of Rosnow & Rosenthal, 1988). The coefficient of determination for the overall effect of adjustment was $r = .46$. The coefficients of determination for individual scale effects are reported in Table 3. Further, a binomial effect-size display for the overall measure, as well as the smallest and greatest effect sizes for individual scales (NIM, and SOM-C and SOM-H, respectively) are reported in Table 4.

Investigations of the possibility for differential influence of group factors such as age (under 30, 30 to 40, 40 to 50, and 50 to 63 years old), diagnosis (TBI, CVA, anoxia, and neurodegenerative), gender, education level (12 years or less, and more than 12 years), and time post injury (one year or less and more than one year) on neurologically-related self-report and score adjustments due to neurological correction resulted in the detection of effects due to time post injury ($n = 59$) and gender.

The "one year or greater" post ABI group ($n = 33$) had significantly higher scores on both the original and adjusted T scores for the Negative Impression Management (NIM) validity scale than the "less than one year" post ABI group ($n = 26$) (F [2, 57] = 3.50, $p = .04$, and F [2, 57] = 3.33, $p = .04$, respectively). A correlational analysis revealed no significant relationship between NIM T scores and time post injury when the

variable for individual times post injury (M = 24.1 months, SD = 24.1 months) was used in the analysis.

Analyses (ANOVA) of pre and post-adjustment NIM scores following a division of patients into 4 groups ("less than 12 months," "12.5 to 24 months," 24.5 to 36 months," and "greater than 36 months") resulted in F (3, 56) = 2.27, p = .09 and F (3,56) = 2.11, p = .11, respectively. Assessment "less than 12 months" post injury (n = 22) resulted in the lowest mean NIM T scores, M = 52.23, SD = 9.5, with increasingly higher mean scores resulting from the fourth group, second group, and third group, respectively (means ranging from 57.92 to 64.43, and standard deviations ranging from 10.01 to 17.8). A correlational analysis of the malingering index scores and time post injury resulted in r = .05, p = .74, and an ANOVA using the two ABI group method failed to detect a significant difference between the mean scores of these groups, F (1, 58) = .12, p = .73. The possibility of differential effects due to time post injury on the NIM NRI (i.e., item 9) score pre- and post-adjustment was investigated. A correlational analysis using individual times post injury (n = 59) did not detect a significant relationship (r = .08, p = .57). Further, an ANOVA that included the mean NIM NRI score (i.e., item 9) of the "less than 12 months" and the "greater than 12 months" ABI groups did not detect a significant difference, F (1, 58) = 2.17, p = .15.

Gender effects were detected for both SOM-H original and SOM-H adjusted T scores, F (1, 60) = 4.18, p = .05, and F (1,60) = 3.02, p = .09, respectively. Correlational analyses resulted in r = -.26, p = .05, and r = -.22, p = .09 between gender, and SOM-H original and adjusted T scores, respectively. Although these analyses suggest that males had higher endorsement rates for the SOM-H scales, the number of males in this sample

was 4 times greater than that of females (i.e., 50 versus 12). The probability for making a Type II error is thus much greater for this situation than had these correlations been positive in nature.

Analyses of the Structure or Dimensionality of NRIs

A two-factor PCA using varimax rotation produced the matrix of neurologically-related item component loadings that are listed in Table 5. The item correlation matrix follows in Table 6. The first and second rotated component accounted for 23% and 19% of the major variance accounted for, respectively (unrotated 32% and 11%, respectively). An oblique rotation was also performed, however the item loadings did not significantly change in terms of direction of loading (i.e., first or second component or both) or in the interpretation of these factors.

Thirteen of twenty items loaded on the first component. Of these 13 items, 8 did not share loadings on the second component. These items (see Table 1) contain content that reflects cognitive difficulties or confusion (Morey, 1991; 1996), and somatic complaints due to energy loss and specific pain (i.e., "headache" and "bad back"). Shared loadings include those items related to specific somatic symptoms including "numbness," "blurred vision," "dizziness," and "pain." The remaining 8 item loadings on the second component are related to reports of the daily struggle and complications due to medical problems.

Analyses of the Structure or Dimensionality of ABI Versus Normative, and Clinical Versus Normative Sample Groups

ABI Versus PAI Normative Samples

The top twenty discriminant items ABI versus PAI normative population are listed in Table 7 in order of decreasing discriminative ability. Nine of these items are NRIs.

These items reflect problems with concentration, confused thinking, slowed movement, pain, stress related to traumatic experiences, loss of interest and pleasure, unexplained sadness, and complications and restrictions due to their health problems. Two components comprised the majority of variance accounted for (36% and 12%, first and second components, respectively). The rotated (varimax) component matrix is reported in Table 8. Rotation reduced the variance accounted for to 45% (27% and 18%, first and second rotated components, respectively). The correlational matrix for these items is reported in Table 9.

Thirteen of the twenty discriminant items loaded on the first component, with 4 of these 13 items sharing loadings on the second component. As can be seen from Table 7, the eight items that do not share loadings on component two reflect cognitive confusion, a lack of concentration, disinterest in things that used to give pleasure, and unexplainable sadness. Some of the five items that share loadings do so differentially, and those that load most heavily on the first component are related to concentration difficulties and the great effort required for daily activities. Others are related to psychomotor slowing and somatic symptoms of numbness and pain. Items that load heavily only on the second component are redundant, expressing the restrictions and complications due to health problems, as well as the report of recent major changes in life events.

Clinical Versus Normative PAI Standardization Samples

The top twenty discriminant items PAI clinical and normative sample are listed in Table 10 in order of decreasing discriminative ability. As mentioned previously PCA was not possible due to the lack of availability of a reliable sample of PAI normative and clinical responses. Two of these items are shared by the list of ABI versus normative

discriminant items. The content of these items reflect "traumatic experience" and loss of interest, and the report of suffering "from a lot of pain," respectively. Additionally, the latter item is shared by the list of NRIs. The content of the remaining items are related to "bad" or "traumatic experiences," "guilt," a loss of focused or directive attentive abilities, "nervousness" and irritability, and a loss of "pleasure," or unhappiness that is reflected in negative relationships and a loss of empathy for others (Morey, 1991).

Discussion

The results of the present study suggest that a number of the self-reported behaviours and/or symptoms that are due to the direct physical consequences of ABI are misinterpreted by PAI test protocol as psychiatric behaviours and/or symptoms. This is not surprising in itself, as the PAI was not designed for the purpose of performing differential diagnosis of neurologic and psychiatric disorders. Correction for neurological content may be necessary prior to the interpretation of ABI profiles. It is highly recommended that clinicians pay particular attention to elevations on the SOM, DEP-P and SCZ-T subscales. NRIs within these subscales expressed the majority of variance, or range for neurological symptom reports for the current ABI sample.

Neurological Content: Extent and Impact

The Identification of NRIs

The variability observed in rater agreement across all PAI items may reflect the differential experience of these raters with the ABI population. Symptom expression varies over the course of time post injury. Symptoms immediately following the injury (i.e., observed by a neurosurgeon) may differ from those observed at various points of time during rehabilitation (i.e., initial observations of physiatrists versus those made later in the

course of treatment).

Because symptom report varies both among the ABI population and along the time course post ABI for each individual the identification of NRIs to be included an adjustment for neurological content is best made by a variety of professional contacts along the course of rehabilitation. Thus, for future investigations it is recommended that both the number and the range of professional contexts be increased. For example, Alfano et al. (1990) set reliability for 2 of 3 rater agreement using 18 medical specialists within various professional contexts (i.e., similar numbers of neurologists, physiatrists, neurosurgeons and psychiatrists). This method is advocated by Jackson's (1970) construct-oriented approach to test construction.

Although this manner of identification of NRIs on objective measures of personality and emotional functioning can never provide an absolute framework from which to judge the neurological content of such tests (i.e., this will vary according to theoretical changes regarding the neurological versus psychiatric distinction), for clinical purposes these measures should not confound unique ABI neurological content within constructs for . psychiatric disorders. The results of this study suggest that this confounding largely impacts PAI psychiatric profiles. However, the range of effects are narrowly contained. That is, they are confined to a relatively small number of clinical subscales.

Adjustment for Neurological Content

Individual scale profiles were most greatly influenced by significant elevations of the DEP and SOM scales (in greater than 30% of the cases). Subscales related to somatic complaints (SOM-C, SOM-H and SOM-S), cognitive difficulties (SCZ-T), affect (DEP-A, ANX-A), symptoms of physiological depression (DEP-P) and traumatic stress (ARD-T)

were most frequently represented (greater than 20% of the cases) in the number of pathological elevations.

The adjustment for neurological content resulted in partial confirmation of the first experimental hypothesis. SCZ-T, DEP-P and all three SOM subscales were significantly reduced by the correction for neurological content. None of items on the BOR-A subscale were identified as neurologically related. However, it must be cautioned that these formerly mentioned effects (see Table 3 and Table 4) are a product of the number of NRIs within each affected scale and the frequency and magnitude for overall NRI endorsement. For example, although the number of DEP-P NRIs are equal to those of the AGG-P subscale, because DEP-P were more frequently endorsed and the magnitude of endorsement was generally higher, the neurological impact on original DEP-P scores was greater than on the original AGG-P scores. If on repeated examination of the PAI for neurological content a greater or lesser number of NRIs are identified, both the changes in endorsement levels and the spread of items across scales would result in effect sizes for scale adjustments that vary from those indicated by the present study.

Additionally, there are two substantive reasons why the current findings for effects should not interpreted as generalizable to the greater ABI population. Because the PAI has not been reported in the literature concerning clinical neuropsychological evaluation, it is most likely the case that clinicians are hesitant to administer the PAI in those situations where psychiatric complications are suspected. Thus, these preliminary results may reflect the responses of a ABI population that were preferentially selected for PAI test administration. Future analyses should ensure that sampling methods preclude this possibility. Secondly, although this study did not find differences among diagnostic

groups, the detection of such effects was prohibited by the small sample size. While more than two-thirds of the sample were categorized as TBI, less than one-third of the total cases were represented within the three remaining diagnostic groups (i.e., CVA, anoxia, and neurodegenerative). Previous research using the MMPI has suggested that particular diagnostic groups report distinct neurological symptoms that are associated with their injury (e.g., cerebrovascular disease, Gass, 1992). It is unknown whether this is true for neurological content on the PAI. Further experimental analyses using larger homogeneous diagnostic groups may provide a basis for such a distinction.

Aside from the difficulties for generalizing the effects of this study to other ABI populations, the method for neurological adjustment itself proved adequate for a significant reduction in the number of pathological elevations. Reductions in the frequency of pathological scale or subscale elevations post adjustment were most noticeable for SOM and SCZ-T subscales. In terms of the main scale post adjustment frequency of pathological elevations, the greatest change was observed for the SOM scale, followed by the SCZ, DEP and AGG scales, respectively. These results suggest that a confounding of neurological content within PAI main scales occurs most frequently within SOM item endorsements.

The PAI test author (Morey, 1996) has cautioned that the SOM scale was not designed to distinguish between functional and organic somatic symptoms. That is, this particular scale should not to be used as a neuropsychological assessment instrument as it cannot provide sufficient evidence for establishing a diagnosis for physical conditions (p. 23). Rather, he describes this scale within the context of neurological populations as being useful for assessing the variability that is seen across individual psychological

reactions to declines in physical functioning and health matters generally, and as useful for assessing the extent to which physical conditions are a central concern for the individual.

Within the clinical neuropsychological context these recommendations present a major difficulty for the interpretation of SOM scale scores. Because the theoretical constructs underlying these scales are based on related psychiatric disorders, genuine health-related concerns (i.e., not abnormal reactions to normal declines in health functioning) are represented as psychopathological. Although variability in individual psychological reactions to these declines in functioning do exist, there is no valid standard for assessing what would be a normal or typical response given the extent of injury. Further, there is no other situation quite like that of the head injured. For the most part they come into their current situation from normal backgrounds. The loss of cognitive abilities or the loss of attentional and/or emotional control represents a loss of their former self and ability levels. Thus, it would seem that a failure to experience bonafide declines in cognitive and physical functional levels as a main or central personal concern would be indicative of an abnormal, or atypical ABI response, and reflective of denial (i.e., perhaps representative of the initial versus latter stages of psychic conflict that underlie the SOM-C and SOM-S theoretical constructs, or perhaps the representation of a genuine unawareness of deficits).

The method for neurological correction developed by Gass and his colleagues mitigates this paradoxical situation by reducing the neurological confounding of SOM scores. Consequently, the expression of nonneurologically related content is more clearly visible in adjusted scale scores. In this respect, Gass' method for neurological correction is quite useful. It allows the "noise" produced by neurologically related endorsements to be

reduced to a level where the "sound" of emotional or characterological involvements may be heard more clearly.

While researchers (Cripe, Maxwell, & Hill, 1995) have conducted multivariate discriminant function analyses within a number of populations (e.g., neurologic, psychiatric, random and controls) using variables related to the experience of pain, and neurologic and psychiatric factors, their results remain to be validated by experimental replication. Rather than correcting for neurological content, their study used a selected number of MMPI NRI responses (e.g., related to attention, health, sensory experiences, pain, vegetative symptoms and emotional control) in the interpretation of these analyses.

As mentioned previously, the extent of neurological content within the PAI (i.e., number of items and the range of scales) remains for future investigation, however the context for the use of these items in examinations of ABI profiles may vary. While clinical contexts require a separation of neurological content for interpreting the self-reports of personality and emotional functioning, research contexts may require the identification of NRIs and their scales for relating the self-report of neurological problems to those of emotional difficulties. For example, researchers may wish to study the relationship between particular neurological symptoms and particular subscale elevations. Perhaps characteristic patterns of physiological, cognitive, and affective responses may be observed for particular neurological symptom constellations.

NRIs: Factor Structure and Major Component Definitions

On examination of the logical content of the NRIs, the larger component of the major variance accounted for was realized in items that stated problems with concentration and thinking, specific sensory deficits or problems, and pain. The smaller

component of the major variance accounted for was realized for those items related to general health problems, and the medical or treatment complications and restrictions consequent to their injury. There was great agreement between individuals regarding the latter (i.e., greater than 80% of the sample endorsed health-related restrictions and complications as "mainly true" or "very true").

Analyses of discriminant ABI responses (ie., ABI vs normative sample) suggest that these NRIs are highly correlated with the report of unusual health problems (i.e., as compared to those of the non-brain injured population) and poor health generally, but not the number of doctors seen (even though 56% of the sample endorsed seeing a "lot of doctors" in the past as "mainly" or "very true"). This suggests that although most individuals have seen a large number of doctors, there is great variability among individuals with respect to the pattern and extent of endorsements for those neurological symptoms or items that contribute to the greater component of the major variance accounted for.

The findings for greater mean NIM scale scores for the "more than 12 months" ABI groups is perplexing in that the NIM scale represents the "fake bad" validity scale for the PAI. Malingering scores as assessed by PAI interpretations were not congruent with these results. Because the variability among scores within the greater than 12 months post injury groups was quite large, it is difficult to make generalizations about these groups. While most neuropsychological evaluations are generally conducted within three to nine months of a significant brain injury, some of these individuals continue to experience problems. The present study's "greater than 12 months" ABI patient groups were largely representative of these individuals. It would be interesting to see whether these findings

will be replicated in future studies.

Although statistical analyses of group effects for the most part did not reveal effects due to diagnosis, age, gender, education or time post injury, the power of this experiment to detect these differences was reduced due to relatively small within subject groups, particularly for the number of cases within diagnostic and gender groups. The exploratory analyses of Alfano et al. (1990) also involved a heterogeneous neurological population. Although their sample size was double that of the current sample, they do not report the investigation of possible group effects.

Discriminant Items ABI, Clinical and Normative Samples

Results for analyses of the unique character of ABI endorsements reflect both the general pattern of PAI elevations for this sample (i.e., SOM, DEP, and SCZ-T subscales) and the high endorsement of NRIs. For the most part, the magnitude and direction of NRI loadings within the discriminant items ABI versus normative sample remained unchanged. That is, the report of confused thinking and a lack of concentration remained within the larger component of major variance accounted for, while content related to poor general health or an increased number of unspecified health problems remained in the smaller component of the major variance accounted for. Further, discriminant items other than NRIs and items of the SOM subscales were most related to items that state cognitive difficulties. These items included content related to depressed or anxious affect.

The second experimental hypothesis that discriminant items ABI versus PAI normative samples would be different than those discriminant items PAI clinical versus normative samples was confirmed by the results of the statistical analyses. Discriminant items resulting from the analyses of PAI clinical versus normative data were highly

represented by content related to guilt, irritability, nervousness, and pervasive unhappiness in daily life and social relationships. Further, although two items were shared by ABI and clinical discriminant item lists, their content or meaning is different depending on the population context. For example, suffering "from a lot of pain" may have one meaning within the psychiatric context (i.e., emotional) and another within a neurological population (i.e., pain due to physical injury), while the report of "traumatic experience" by the clinical population may stem from childhood experiences and conversely as due to motor vehicle accidents for the ABI population. Thus it would appear that the endorsements of the ABI population differ significantly from those of the PAI clinical referent population.

Utility of the PAI Within Clinical and Research Contexts

The efficacy of the PAI for use in a research or a clinical context is enhanced by both an increased range and an increased level of freedom for interpretation. Within the research context, this facilitation is due to the fact that each PAI item was chosen for its representation as both a unique and a relatively equal contributor to the total scale variance. Further, because PAI scales do not share items and all clinical scales (except ALC and DRG) contain an equal number of items, an increase in power is observed for those statistical analyses that are reliant on methods that involve correlational analyses or the requirement for equal cell sizes.

Within the clinical context, each item can be interpreted either on its own or within the context of its scale representations (i.e., subscale or main scale). Rising scale scores may be interpreted as representing an increasing extent, or severity for the disorder. Further, PAI interpretation also produces considerations for differential diagnosis based on

the pattern of scale scores. Coefficients of fit for a number of diagnostic groups and cluster group analyses identities are provided (Morey, 1991; 1996).

Diagnostic issues related to cognitive and organic disorders are of particular interest to clinical neuropsychologists. Morey (1996) describes a pattern of scale elevations that is typically observed in neurological populations. SOM subscales are elevated (SOM-H score usually representing the greatest elevation of the three) along with significant elevations of the SCZ-T and DEP-P (i.e., vegetative symptoms) subscales. Fifteen individuals, or greater than 24% of the current ABI sample exhibited this specific pattern within their PAI profile. The reliability of this pattern structure remains for further study with neurological populations.

Future Directions

It is naive to believe that a test of personality and psychopathology can be attached to a neuropsychological evaluation and reliably assess the complexities of personality, emotions, and motivation that are seen in neurological populations. Further, it is a grave misconception to believe that such a test may adequately perform valid and reliable differential diagnosis of neurologic and psychiatric disorders.

More recent research (Mittenberg, Tremont, & Rayls, 1996) that has investigated the validity of MMPI-2 use with neurologically impaired patients has supported the caveat of common literature that this test "does not provide information sufficient for making differential diagnoses between psychiatric and neurologic disorders" (p. 162). Rather clinical history and neuropsychological examination procedures are necessary for making this distinction.

With this is mind, it should be remembered that although the PAI appears to be superior to the MMPI with respect to both the construction of a correction factor and the direct interpretation of ABI endorsements within clinical or psychiatric constructs, this test can never in itself provide sufficient information for making differential diagnoses between psychiatric and neurologic disorders.

Future research involving the PAI and neurological populations should focus on the development of a correction factor to be used in clinical settings. The methods proposed for the selection of NRIs have been explained previously. Further, additional experimental research using the PAI may address factors related to the etiology of characterological and reactionary involvements as defined by Prigatano (1987). Perhaps relationships may be found between characteristic PAI response patterns, the site or severity of injury, or particular diagnostic groups.

References

Alfano, D. P., Finlayson, M. A., Stearns, G. M., & Neilson, P. M. (1990). The MMPI and neurologic dysfunction: Profile configuration and analysis. The Clinical Neuropsychologist, 4 (1), 69-79.

Bell-Pringle, V. J., Pate, J. L., & Brown, R. C. (1997). Assessment of borderline personality disorder using the MMPI-2 and the Personality Assessment Inventory. Assessment, 4 (2), 131-139.

Boone, D. (1998). Internal consistency reliability of the Personality Assessment Inventory with psychiatric inpatients. Journal of Clinical Psychology, 54 (6), 839-843.

Butcher, J. N., Dahlstrom, W. G., Graham, J. R., Tellegen, A., & Kaemmer, B. (1989). Manual for the restandardized Minnesota Multiphasic Personality Inventory-2: MMPI-2. Minneapolis, MN: University of Minnesota Press.

Clark, L. A., & Watson, D. (1995). Constructing validity: Basic issues in objective scale development. Psychological Assessment, 7 (3), 309-319.

Cripe, L. I. (1996). The MMPI in neuropsychological assessment: A murky measure. Applied Neuropsychology, 3 (4), 97-103.

Cripe, L. I. (1999). Use of the MMPI with mild closed head injury. In N. R. Varney & R. J. Roberts (Eds.), The evaluation and treatment of mild traumatic brain injury (pp. 291-314). Mahwah, NJ: Lawrence Erlbaum Associates.

Cripe, L. I., Maxwell, J. K., & Hill, E. (1995). Multivariate discriminant function analysis of neurologic, pain, and psychiatric patients. Journal of Clinical Psychology, 51 (2), 258-267.

Deisinger, J. A., Cassisi, J. E., & Whitaker, S. L. (1996). Relationships between coping style and PAI profiles in a community sample. Journal of Clinical Psychology, 52 (3), 303-310.

Derogatis, L. R., Lipman, R. S., Covi, L. (1973). SCL-90: An outpatient psychiatric rating scale: Preliminary report. Psychopharmacology Bulletin, 9, 13-28.

Dikmen, S. S. (1998). Recovery patterns: Natural history. Mild Head Trauma, 9 (3), 10-11.

Dunn, J. T., Lees-Haley, P. R. (1995). The MMPI-2 correction factor for closed-head injury: A caveat for forensic cases. Assessment, 2 (1), 47-51.

Fleiss, J. L. (1971). Measuring nominal scale agreement among many raters. Psychological Bulletin, 76, 378-382.

Gass, C. S. (1991). MMPI-2 interpretation and closed head injury: A correction factor. Psychological Assessment, 3 (1), 27-31.

Gass, C. S. (1992). MMPI-2 interpretation of patients with cerebrovascular disease: A correction factor. Archives of Clinical Neuropsychology, 7, 17-27.

Gass, C. S., & Russell, E. W. (1991). MMPI profiles of closed head trauma patients: Impact of neurologic complaints. Journal of Clinical Psychology, 47 (2), 253-260.

Gass, C. S., & Wald, H. S. (1997). MMPI-2 interpretation and closed-head trauma: Cross-validation of a correction factor. Archives of Clinical Neuropsychology, 12 (3), 199-205.

Hathaway, S. R., & McKinley, J. C. (1967). Minnesota Multiphasic Personality Inventory manual (Rev. ed.). New York: Psychological Corporation.

Helmes, E., & Reddon, J. R. (1993). A perspective on developments in assessing psychopathology: A critical review of the MMPI and MMPI-2. Psychological Bulletin, 113 (3), 453-471.

Iverson, G. (1998). Epidemic in nature. Mild Head Trauma, 9 (1), 4-7.

Jackson, D. N. (1970). A sequential system for personality scale development. In C. D. Spielberger (Ed.), Current topics in clinical and community psychology: Vol.2. (pp. 62-97). New York: Academic Press.

Kraus, J. F. (1987). Epidemiology of head injury. In P. R. Cooper (Ed.), Head injury (2nd ed., pp. 1-19). Baltimore, MD: Williams & Wilkins.

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

Lishman, W. A. (1978). Psychiatric sequelae of head injuries: Problems in diagnosis. Journal of the Irish Medical Association, 71, 306-314.

Lishman, W. A. (1988). Physiogenesis and psychogenesis in the 'post-concussional syndrome.' British Journal of Psychiatry, 153, 460-469.

Loevinger, J. (1957). Objective tests as instruments of psychological theory. Psychological Reports, 3, 635-694.

Long, K. A., & Graham, J. R. (1991). The masculinity-femininity scale of the MMPI-2: Is it useful with normal men? Journal of Personality Assessment, 57, 46-51.

Longman, R. S., Cota, A. A., Holden, R. R., & Fekken, G. C. (1989a). PAM: A double-precision FORTRAN routine for the parallel analysis method in principal components analysis. Behavior Research Methods, Instruments, & Computers, 21 (4), 477-480.

Longman, R. S., Cota, A. A., Holden, R. R., & Fekken, G. C. (1989b). A regression equation for the parallel analysis criterion in principal components analysis: Mean and 95th percentile eigenvalues. Multivariate Behavioral Research, 24 (1), 59-69.

Malloy, P., & Duffy, J. (1994). The frontal lobes in neuropsychiatric disorders. In F. Boller, & J. Grafman (Eds.), Handbook of neuropsychology (Vol. 9, pp. 203-232). New York: Elsevier.

Meyerink, L., Reitan, R., & Selz, M. (1988). The validity of the MMPI with multiple sclerosis patients. Journal of Clinical Psychology, 44 (5), 764-769.

Miller, L. (1997). Neurosensitization: A pathophysiological model for traumatic disability syndromes. Journal of Cognitive Rehabilitation, 6, 12-23.

Miller, L. (1998). Motor vehicle accidents: Clinical, neuropsychological, and forensic considerations. Journal of Cognitive Rehabilitation, 4, 10-23.

Mittenberg, W., Tremont, G., & Rayls, K. R. (1996). Impact of cognitive function on MMPI-2 validity in neurologically impaired patients. Assessment, 3 (2), 157-163.

Morey, L. C. (1991). Personality Assessment Inventory: Professional manual. Odessa, FL: Psychological Assessment Resources.

Morey, L. C. (1996). An interpretive guide to the Personality Assessment Inventory (PAI). Odessa, FL: Psychological Assessment Resources.

Morey, L. C., & Henry, W. (1994). Personality Assessment Inventory. In M. E. Maruish (Ed.), The use of psychological testing for treatment planning and outcome assessment (pp. 185-216). Hillsdale, NJ: Lawrence Erlbaum.

Morey, L. C., & Lanier, V. W. (1998). Operating characteristics of six response distortion indicators for the Personality Assessment Inventory. Assessment, 5 (3), 203-214.

O'Connor, J. P., & Stefic, E. C. (1959). Some patterns of hypochondriasis. Educational and Psychological Measurement, 19, 363-371.

Paulhus, D. L. (1991). Measurement and control of response bias. In J. P. Robinson, P. R. Shaver, & L. S. Wrightsman, (Eds.), Measures of personality and social attitudes (pp. 17-51). New York: Academic Press.

Prigatano, G. P. (1987). Psychiatric aspects of head injury: Problem areas and suggested guidelines for research. In H. S. Levin, J. Grafman, & H. M. Eisenberg (Eds.), Neurobehavioral recovery from head injury (pp. 215-231). New York: Oxford University Press.

Rogers, R., Ornduff, S. R., & Sewell, K. W. (1993). Feigning specific disorders: A study of the Personality Assessment Inventory (PAI). Journal of Personality Assessment, 60 (3), 554-560.

Rogers, R., Ustad, K. L., & Salekin, R. T. (1998). Convergent validity of the Personality Assessment Inventory: A study of emergency referrals in a correctional setting. Assessment, 5 (1), 3-12.

Rosnow, R. L., & Rosenthal, R. (1985). Focused tests of significance and effect size estimation in counseling psychology. Journal of Counseling Psychology, 35 (2), 203-208.

Schinka, J. A. (1995). Personality Assessment Inventory scale characteristics and factor structure in the assessment of alcohol dependency. Journal of Personality Assessment, 64 (1), 101-111. Schinka, J. A., & Borum, R. (1993). Readability of adult psychopathology inventories. Psychological Assessment, 5 (3), 384-386.

Slagle, D. A. (1990). Psychiatric disorders following closed head injury: An overview of biopsychosocial factors in their etiology and management. International Journal of Psychiatry in Medicine, 20 (1), 1-35.

Sorenson, S. B., & Kraus, J. F. (1991). Occurrence, severity and outcomes of brain injury. Journal of Head Trauma Rehabilitation, 6, 1-10.

SPSS for Windows, Rel. 7.5. 1996. Chicago: SPSS Science.

Wang, E., Rogers, R., Giles, C., Diamond, P, Herrington-Wang, L., & Taylor, E. (1997). A pilot study of the Personality Assessment Inventory (PAI) in corrections: Assessment of malingering, suicide risk, and aggression in male inmates. Behavioral Sciences and the Law, 15, 469-482.

White, L. J. (1996). Review of the Personality Assessment Inventory (PAI): A new psychological test for clinical and forensic assessment. Australian Psychologist, 31 (1), 38-39.

Winfield, D. L. (1953). The relationship between IQ scores and MMPI scores. Journal of Social Psychology, 8, 299-300.

Wooten, A. J. (1983). MMPI profiles among neuropsychology patients. Journal of Clinical Psychology, 39 (3), 392-406.

Table 1

PAI Subscales, Neurologically-Related Items (N =20) and ABI Percentage (%) Endorsement

Subscale	Item	Self-Report	% [c (d)]
SOM-C	3 [a]	My health condition has restricted my activities.	83 (69)
	83 [a]	I've had numbness in parts of my body that I can't explain.	56 (33)
	123 [a]	I've had episodes of double vision or blurred vision.	50 (24)
	163 [b]	There have been times when my eyesight got worse and then better again.	49 (23)
	203 [b]	I've had episodes when I've lost the feeling in my hands.	40 (24)
SOM-H	12 [a]	I've seen a lot of doctors over the years.	72 (56)
	52 [a]	My health problems are very complicated.	59 (38)
	92 [a]	It's a struggle for me to get things done with the medical problems I have.	80 (46)
	132 [a]	My medical problems always seem to be hard to treat.	42 (21)
SOM-S	72 [a]	I suffer from a lot of pain.	56 (30)
	192 [b]	I have a bad back.	48 (24)
SCZ-T	38 [b]	My thinking has become confused.	72 (40)
	78 [b]	My thoughts get scrambled sometimes.	75 (48)
	118 [b]	Sometimes I have trouble keeping different thoughts separate.	53 (26)
AGG-P	21 [b]	People are afraid of my temper.	45 (18)
	61 [b]	Sometimes my temper explodes and I completely lose control.	50 (21)
DEP-P	35 [b]	I hardly have any energy.	66 (32)
	155 [a]	I've been moving more slowly than usual.	75 (50)
ANX-P	113 [b]	Sometimes I feel dizzy when I've been under a lot of pressure.	42 (15)
NIM	9 [b]	Sometimes I cannot remember who I am.	19 (6)

Note. SOM-C = Somatic Conversion; SOM-H = Health Concerns; SOM-S = Somatization; SCZ-T = Thought Disorder; AGG-P = Physical Aggression; DEP-P = Physiological Depression; ANX-P = Physiological Anxiety; NIM = Negative Impression Managment.

[a] Endorsed by three of three raters as neurologically-related

[b] Endorsed by two of three raters as neurologically-related

[c] "Slightly True"

[d] "Mainly True" or "Very True"

Table 2
ABI (N=62) Mean T Scores, Standard Deviations, and Frequency (%) of Pathologic Elevations (T>69) PAI Scales and Subscales

Scale	Subscale	M	SD	%
Schizophrenia	Thought Disorder	63.9	16.4	45
	Social Detachment	52.9	12.7	11
	Psychotic Experiences	50.2	8.9	3
Somatic	Conversion	65.7	16.1	34
	Health Concerns	64.2	11.6	34
	Somatization	59.3	14.0	31
Depression	Affective	62.3	14.6	31
	Physiological	60.7	13.1	23
	Cognitive	57.8	13.8	16
Anxiety-Related Disorders	Traumatic Stress	58.3	16.1	24
	Obsessive-Compulsive	51.8	12.1	8
	Phobias	53.0	10.1	6
Anxiety	Affective	57.3	12.7	21
	Physiological	57.4	13.1	18
	Cognitive	56.1	10.6	8
Negative Impression Management		57.4	13.5	21
Stress		56.6	11.7	18
Aggression	Physical	56.4	15.1	16
	Attitude	54.8	13.1	16
	Verbal	53.2	11.6	11

Borderline Features	Affective Instability	57.2	12.9	16
	Identity Problems	56.6	11.3	13
	Negative Relationships	53.9	12.7	10
	Self-Harm	50.3	11.3	8
Antisocial Features	Stimulus Seeking	53.1	13.7	15
	Egocentricity	52.6	14.3	11
	Antisocial Behaviours	54.7	11.5	11
Paranoia	Hypervigilance	51.3	12.3	10
	Persecution	51.3	11.1	6
	Resentment	51.2	11.6	3
Suicidal Ideation		52.0	12.1	8
Nonsupport		50.6	13.7	8
Drug Related Problems		52.7	12.0	6
Mania	Activity Level	50.8	11.0	5
	Irritability	51.7	11.8	3
	Grandiosity	50.1	9.9	3
Dominance		50.7	9.9	(>69 T) 3
				(<30 T) 2
Warmth		49.3	11.2	(>69 T) 0
				(<30 T) 3
Infrequency		52.9	8.9	3
Inconsistency		56.7	9.6	3
Positive Impression Management		49.7	10.8	2
Alcohol Related Problems		50.8	7.5	2
Treatment Rejection		47.2	10.0	2

Table 3

Mean T Score and Standard Deviation for Original and Adjusted PAI Subscales, and Effect Sizes and F Ratios for Adjustment Procedure

PAI Subscale	Original T Score M	Original T Score SD	Adjusted T Score M	Adjusted T Score SD	r [a]	F [b]
NIM	57.4	13.5	56.5	13.1	.39	11.08*
SOM-C	65.7	16.1	54.2	14.1	.84	146.23**
SOM-H	64.2	11.6	57.5	11.0	.84	140.55**
SOM-S	59.3	14.0	56.3	12.6	.73	70.01**
ANX-P	57.4	13.1	56.0	12.2	.59	33.03**
DEP-P	62.4	13.9	61.0	13.8	.79	101.75**
SCZ-T	63.9	16.4	57.8	14.2	.81	119.95**
AGG-P	56.4	15.1	53.2	14.3	.69	54.50**

Note. NIM = Negative Impression Management, SOM-C = Somatic Conversion, SOM-H = Somatic Health Concerns, SOM-S = Somatization, ANX-P = Physiological Anxiety, DEP-P = Physiological somatic symptoms of Depression, SCZ-T = Thought Disorder, AGG-P = Physical Aggression.

[a] univariate repeated measures within-subjects original and adjustment factor

[b] $df = 1, 61$. *p = .001. **p<.001.

Table 4

Binomial Effect-Size Display for Overall Effect and Lowest and Highest Subscale Effects Due to Adjustment for Neurological Content

	Adjustment Rate		
Condition	Reduced	Not Reduced	Total
Overall subscales [a]	45 cases	17 cases	62 cases
NIM	43 cases	19 cases	62 cases
SOM-C or SOM-H	57 cases	5 cases	62 cases

[a] NIM, SOM-C, SOM-H, SOM-S, ANX-P, DEP-P, SCZ-T

Table 5
Rotated Component Matrix for
ABI Neurologically-Related Items (N = 20)

Item	Component 1	2
3 [a]	-.035	.686
83 [a]	.327	.466
123	.418	.317
163	.205	.580
203	-.002	.763
12 [a]	.130	.079
52 [a]	.167	.611
92 [a]	.193	.682
132	.161	.575
72 [a]	.598	.349
192	.472	.245
38 [a]	.836	.254
78 [a]	.843	.120
118	.730	-.011
21	.525	.040
61	.612	-.046
35	.558	.228
155 [a]	.381	.477
113	.610	.491
9	.489	.204

Note. ABI (N = 62). Varimax rotation (converged in 3 iterations)

[a] Discriminant Item ABI versus Non-Clinical Normative Sample

Table 6

ABI (N=62) Neurologically-Related Items (N=20)

Item	3	83	123	163	203	12	52	92	132	72	192	38	78	118	21	61	35	155	113	9
3	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---
83	.23	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---
123	.14	.23	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---
163	.10	.32*	.44**	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---
203	.44**	.37**	.25	.50**	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---
12	-.06	-.05	.19	.06	-.04	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---	---
52	.34*	.28	.17	.24	.16	.13	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---
92	.47**	.23	.22	.40**	.36*	.10	.44**	1.00	---	---	---	---	---	---	---	---	---	---	---	---
132	.28	.23	.33*	.33*	.41**	.10	.51**	.32*	1.00	---	---	---	---	---	---	---	---	---	---	---
72	.34**	.28	.21	.18	.27	.16	.29	.42**	.21	1.00	---	---	---	---	---	---	---	---	---	---
192	.22	.29	.21	-.01	.29	.14	.12	.27	.22	.47**	1.00	---	---	---	---	---	---	---	---	---
38	.16	.37*	.38**	.37**	.19	.12	.35*	.22	.26	.56**	.28	1.00	---	---	---	---	---	---	---	---
78	.02	.31*	.47**	.19	.13	.03	.21	.29	.30*	.51**	.45**	.67**	1.00	---	---	---	---	---	---	---
118	-.09	.26	.24	.17	.03	-.03	.13	.16	.18	.26	.24	.64**	.62**	1.00	---	---	---	---	---	---
21	.05	.11	.28	.19	.09	-.02	.03	.19	.11	.39**	.31*	.34*	.36*	.09	1.00	---	---	---	---	---
61	.14	.11	.13	.11	-.13	.03	.15	.11	.10	.40**	.12	.49**	.36*	.29	.49**	1.00	---	---	---	---
35	.15	.43**	.32*	.33*	.21	-.02	.12	.32*	.10	.30*	.28	.45**	.48**	.55**	.27	.16	1.00	---	---	---
155	.36*	.21	.16	.33*	.31*	.09	.39**	.39**	.10	.27	.19	.50**	.36*	.30*	.15	.28	.27	1.00	---	---
113	.27	.38**	.30*	.37**	.39**	.14	.40**	.39**	.41**	.49**	.33*	.70**	.49**	.46**	.27	.34*	.31*	.47**	1.00	---
9	.19	.22	.16	.14	.17	.12	.22	.16	.13	.32*	.54**	.42**	.38**	.34*	.22	.27	.22	.29	.49**	1.00

One-tailed Significance: *$p < .01$, **$p < .001$

Table 7

ABI Discriminant Items (N=20) as a Function of the Mean Standard Deviation From the Normative Population Mean and the Frequency (%) of ABI Sample Endorsement

Item	Scale	Self-Report	Z	% [b (c)]
38	SCZ-T [a]	My thinking has become confused.	1.82	72 (40)
3	SOM-C [a]	My health condition has restricted my activities.	1.64	83 (69)
147	DEP-C	I can't seem to concentrate very well.	1.49	77 (46)
52	SOM-H [a]	My health problems are very complicated.	1.37	59 (38)
92	SOM-H [a]	It's a struggle for me to get things done with the medical problems I have.	1.28	80 (46)
155	DEP-P [a]	I've been moving more slowly than usual.	1.24	75 (50)
12	SOM-H [a]	I've seen a lot of doctors over the years.	1.23	72 (56)
318	SCZ-T	I can concentrate now as well as I ever could (F).	1.20	88 (75)
172	SOM-H	I've had only the usual health problems that most people have. (F)	1.15	87 (72)
323	STR	There have been many changes in my life recently.	1.13	88 (75)
72	SOM-S [a]	I suffer from a lot of pain.	1.11	56 (30)
274	ARD-T	Since I had a very bad experience, I am no longer interested in some things that I used to enjoy.	1.09	53 (30)
6	DEP-A	Much of the time I'm sad for no reason.	1.08	56 (27)
83	SOM-C [a]	I've had numbness in parts of my body that I can't explain.	1.06	56 (33)
46	DEP-A	I've forgotten what it's like to feel happy.	1.03	51 (26)
166	DEP-A	I've lost interest in things I used to enjoy.	1.03	69 (51)
4	ANX-A	I am so tense in certain situations that I have difficulty getting by.	1.01	69 (35)
112	SOM-S	I am in good health. (F)	1.01	83 (58)
78	SCZ-T [a]	My thoughts get scrambled sometimes.	1.00	75 (48)
86	DEP-A	Everything seems like a big effort.	1.00	64 (32)

Note. SCZ-T = Thought Disorder; SOM-C = Somatic Conversion; DEP-C = Cognitive Depression; SOM-H = Health Concerns; DEP-P = Physiological Depression; STR = Stress; SOM-S = Somatization; ARD-T = Traumatic Experiences; DEP-A = Affective Depression; ANX-A = Affective Anxiety.

[a] Neurologically-Related Item

[b] "Slightly true"

[c] "Mainly true" or "Very true"

Table 8
Rotated Component Matrix for Top 20 Discriminant Items
ABI Versus Non-Clinical Normative Sample

	Component	
Item	1	2
38 [a]	.794	.255
3 [a]	.026	.660
147	.652	.326
52 [a]	.146	.649
92 [a]	.156	.703
155 [a]	.450	.482
12 [a]	.059	.214
318	.511	.292
172	.068	.621
323	.162	.515
72 [a]	.578	.442
274	.508	.227
6	.740	-.149
83 [a]	.307	.385
46	.718	-.062
166	.762	.133
4	.650	.055
112	.106	.682
78 [a]	.751	.253
86	.712	.333

Note. ABI (N = 62). Varimax rotation (converged in 3 iterations)

[a] Neurologically-Related Items

Table 9

Top 20 Discriminant ABI (N=62) Item Correlation Matrix

Item	38	3	147	52	92	155	12	318	172	323	72	274	6	83	46	166	4	112	78	86
38	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---
3	.16	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---
147	.50**	.17	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---
52	.35*	.34*	.17	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---
92	.22	.47**	.30	.44**	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---	---	---
155	.50**	.36*	.36*	.39**	.39**	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---	---
12	.12	-.06	.21	.13	.10	.09	1.00	---	---	---	---	---	---	---	---	---	---	---	---	---
318	.58**	.11	.42**	.14	.21	.17	-.06	1.00	---	---	---	---	---	---	---	---	---	---	---	---
172	.25	.23	.19	.40**	.26	.24	.22	.42**	1.00	---	---	---	---	---	---	---	---	---	---	---
323	.19	.17	.34*	.36*	.21	.40**	.14	.30*	.36*	1.00	---	---	---	---	---	---	---	---	---	---
72	.56**	.34*	.54**	.29	.42**	.27	.16	.36*	.27	.21	1.00	---	---	---	---	---	---	---	---	---
274	.38**	.05	.32*	.28	.31*	.34*	.03	.27	.12	.09	.44**	1.00	---	---	---	---	---	---	---	---
6	.49**	-.02	.37*	.12	.04	.32*	.01	.25	-.04	.09	.23	.21	1.00	---	---	---	---	---	---	---
83	.37**	.23	.25	.28	.23	.21	-.05	.38**	.24	.02	.28	.20	.11	1.00	---	---	---	---	---	---
46	.41**	.05	.39**	.09	.16	.31*	.03	.18	.03	.09	.39**	.35*	.52**	.07	1.00	---	---	---	---	---
166	.64**	.10	.48**	.26	.27	.47**	.10	.32*	.11	.09	.54**	.62**	.45**	.27	.53**	1.00	---	---	---	---
4	.55**	.14	.41**	.21	.06	.34*	.07	.33*	.04	.19	.32*	.18	.56**	.31*	.40**	.35*	1.00	---	---	---
112	.18	.42**	.38**	.28	.45**	.33*	.15	.15	.26	.30*	.34*	.22	.05	.31*	.06	.07	.14	1.00	---	---
78	.67**	.02	.59**	.21	.29	.36*	.03	.59**	.27	.32*	.51**	.34*	.49**	.31*	.50**	.41**	.41**	.33*	1.00	---
86	.59**	.14	.62**	.19	.37**	.49**	.15	.35*	.23	.29	.59**	.32*	.43**	.38**	.47**	.55**	.35*	.33*	.62**	1.00

One-tailed Significance: *p < .01, **p < .001

Table 10

PAI Clinical Sample Discriminant Items (N=20) as a Function of Mean Standard Deviation From the PAI Non-Clinical Population

Item	Scale	Self-Report	Z
27	DEP-C	I feel that I've let everyone down.	1.29
253	WRM	I try to include people who seem left out.	-1.25
33	ANX-P	I often feel jittery.	1.17
105	ANX-C	I'm often to worried and nervous that I can barely stand it.	1.17
38	SCZ-T [a b]	My thinking has become confused.	1.17
46	DEP-A [b]	I've forgotten what it's like to feel happy.	1.17
194	ARD-T	I have had some horrible experiences that make me feel guilty.	1.14
114	ARD-T	I've been troubled by memories of a bad experience for a long time.	1.13
246	DEP-A	Lately I've been happy much of the time. (F)	1.11
19	BOR-N	My relationships have been stormy.	1.00
57	BOR-I	Sometimes I feel terribly empty inside.	1.00
137	BOR-I	I wonder what I should do with my life.	1.00
25	ANX-C	I often have trouble concentrating because I'm nervous.	1.00
105	ANX-C	I'm often so worried and nervous that I can barely stand it.	1.00
84	ANX-A	Sometimes I am afraid for no reason.	1.00
44	ANX-A	I can't do some things well because of nervousness.	1.00
34	ARD-T	I keep reliving something horrible that happened to me.	1.00
274	ARD-T [b]	Since I had a bad experience, I am no longer interested in some things that I used to enjoy.	1.00
72	SOM-S [a b]	I suffer from a lot of pain.	1.00
286	DEP-A	I'm almost always a happy and positive person. (F)	1.00

Note. Item sample means for normative sample and clinical sample greater than .3 and greater than or equal to .9, respectively. Item sum standard deviations normative (N = 1,000) and clinical sample (N = 1,246) less than or equal to 2.0.

DEP-C = Cognitive Depression; WRM = Interpersonal Warmth; ANX-P = Physiological Anxiety; ANX-C = Cognitive Anxiety; SCZ-T = Thought Disorder; DEP-A = Affective Depression; ARD-T = Traumatic Experiences; BOR-N = Negative Relationships; BOR-I = Irritability; ANX-A = Affective Anxiety; SOM-S = Somatization.

[a] Neurologically-Related Item

[b] ABI Discriminant Item

Figure Caption

Figure 1. PAI Normative Non-clinical (N = 1,000) and ABI (N = 62) Mean Raw Scale Scores, Original and Adjusted for Neurological Content

Note. SOM = Somatic Complaints; ANX = Anxiety; ARD = Anxiety-Related Disorders; DEP = Depression; MAN = Mania; PAR = Paranoia; SCZ = Schizophrenia; BOR = Borderline Features; ANT = Antisocial Features; AGG = Aggression.
All scales contain 24 items except for the AGG scale which contains only 18.

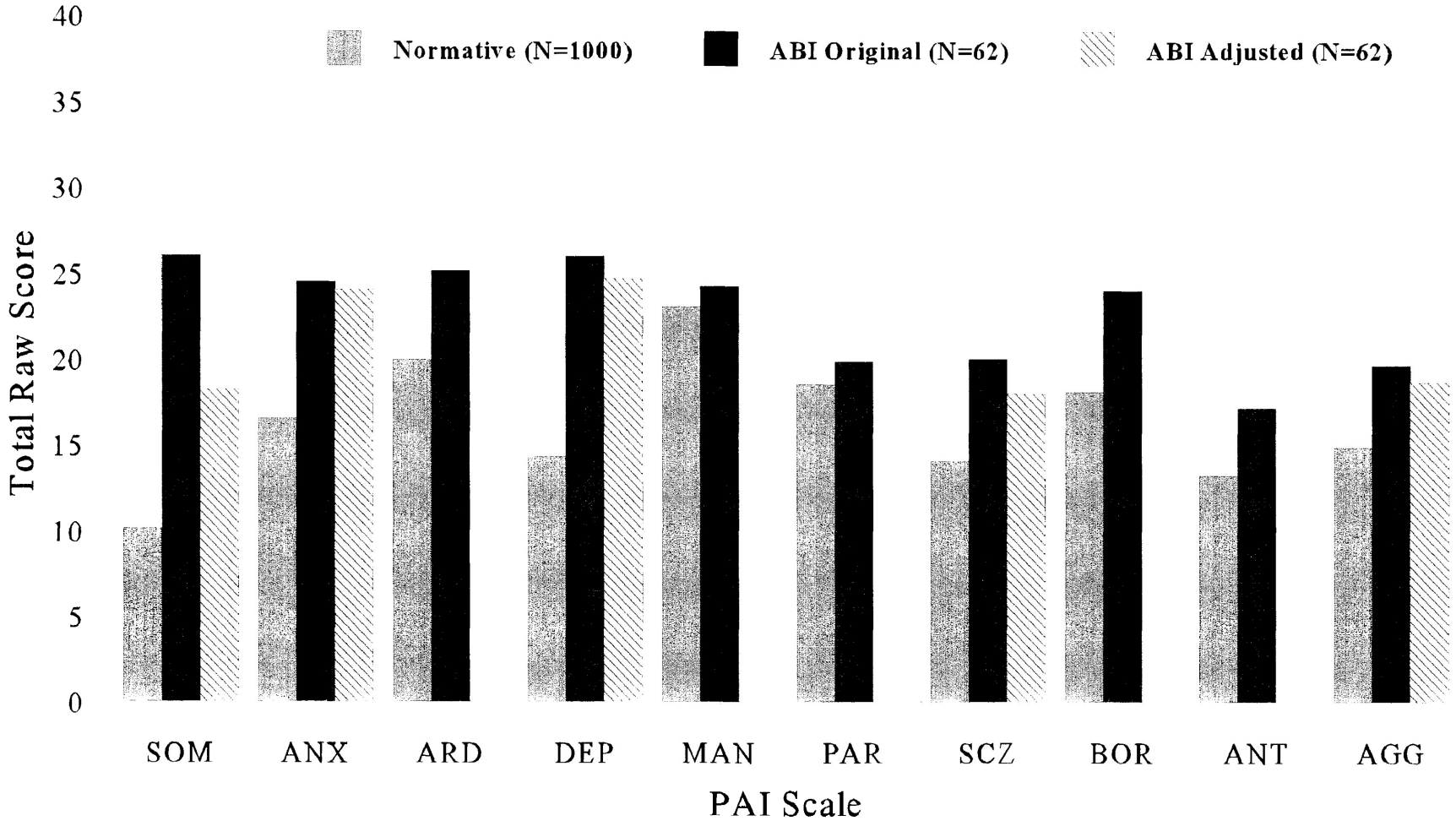
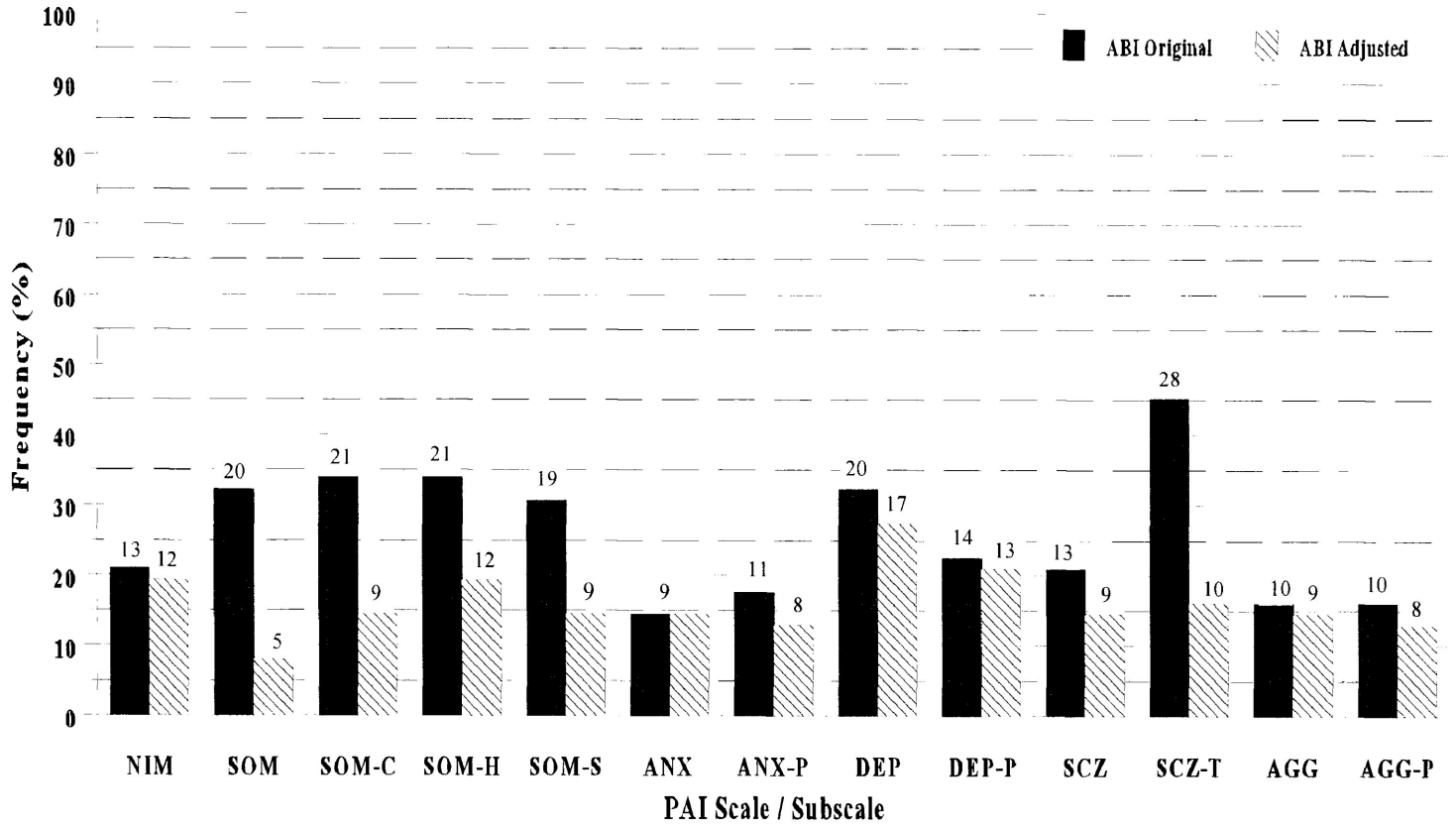

Normative (N=1000)
ABI Original (N=62)
ABI Adjusted (N=62)
Total Raw Score
40
35
30
25
20
15
10
5
0
SOM
ANX
ARD
DEP
MAN
PAR
SCZ
BOR
ANT
AGG
PAI Scale

Figure Caption

Figure 2. Frequency of ABI (N = 62) T Scores > 69, Original and Adjusted for Neurological Content.

Note. NIM = Negative Impression Management; SOM = Somatic Complaints; SOM-C = Somatic Conversion; SOM-H = Health-Related Concerns; SOM-S = Somatization; ANX = Anxiety; ANX-P = Physiological Anxiety; DEP = Depression; DEP-P = Physiological Depression; SCZ = Schizophrenia; SCZ-T = Thought Disorder; AGG = Aggression; AGG-P = Physical Aggression.


ABI Original
ABI Adjusted
Frequency (%)
100
90
80
70
60
50
40
30
20
10
0
13
12
20
5
21
9
21
12
19
9
9
11
8
20
17
14
13
13
9
28
10
10
9
10
8
NIM
SOM
SOM-C
SOM-H
SOM-S
ANX
ANX-P
DEP
DEP-P
SCZ
SCZ-T
AGG
AGG-P
PAI Scale / Subscale