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Zazula, Tania Olga

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PERCEPTION AND PRODUCTION OF INTONATION IN MODERATE AND SEVERE HEAD INJURIES

by

TANIA ZAZULA

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date

Steven Mattis, Ph.D.
Chairman of Examining Committee

August 31 1984
date

Herbert D. Saltzstein
Executive Officer

Dr. Steven Mattis

Dr. Jeffrey Rosen

Dr. Lou Gerstman

Supervisory Committee

The City University of New York

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by

Tania Zazula

Adviser: Dr. S. Mattis

Moderately and severely head-injured patients were evaluated on their ability to perceive and produce affective vocal intonation and on their ability to conceptualize affective terms. Difficulty in the production of prosody has been identified as a source of social difficulties in patients with neurologic dysfunction. Accurate perception of prosody may play an equally important role in communication and social interaction. Head-injured patients present with a myriad of cognitive sequelae. Difficulties in communicative skills, as part of these sequelae could further impede successful social and occupational reintegration post-trauma.

Patients enrolled in this study were all moderately or severely injured head trauma victims. They were evaluated on a perceptual affective intonation task, a task requiring the production of intonation, an affective concept formation task, and the Vineland Social Maturity Scale. Selected tests from a standard neuropsychological battery were also administered. A group of patients' relatives and hospital employees served as a control group.

Results of this study indicate that patients were significantly poorer than controls in their perception and production of vocal intonation, but not in the conceptual identification of affective terms. Pathologic and neuropsychologic measures indicate that brainstem

compression was associated with deficits in the perception of intonation, while right hemispheric anterior pathology was associated with difficulties in producing affective intonation. Brainstem compression and the presence of right hemispheric pathology were associated with lower scores on the Vineland Social Maturity Scale, indicating that patients with brainstem compression and associated perceptual intonational difficulties as part of their cluster of sequelae are more likely to have difficulty in social and occupational adjustment post-trauma.

The present study has identified deficits within the perception and production of affective prosody in head trauma patients. These findings illustrate the need for studying and quantifying suspected difficulties in other aspects of perceptual and motor affective functions and for the development of an affective therapy for head-injured patients that will parallel other forms of cognitive therapy.

Table of Contents

List of Tables

List of Figures

INTRODUCTION

I. Development and Purpose of the Present Study

The present study was developed as an effort to examine and quantify an apparent difficulty in perceiving and producing vocal intonation in a series of moderately and severely head injured patients. These patients have sustained injuries which leave them with a variety of persistent cognitive and sensory-motor sequelae, particularly in the areas of memory, language, planning, abstract reasoning and motor coordination (Tabaddor, Mattis, Zazula, 1984; Jennett and Teasdale, 1981; Levin and Grossman, 1978; Strub and Black, 1981). Although some survivors of trauma lose insight into the extent of their disability and remain euphoric and childlike or indifferent, many patients are acutely aware of their post-traumatic deficits (Strub and Black, 1981). Understandably, they become anxious and depressed over their inability to cope with tasks that were at one time automatic and executed with facility.

Much has been written regarding the "personality" change that often accompanies post-traumatic symptomatology (Jennett and Teasdale, 1981; Levin and Grossman, 1978; McKinley et al, 1981). These changes include irritability, hypersensitivity, lability of affect, blunted affect, unexpected rage reactions, lack of empathy, lack of drive or initiative, social inappropriateness, social withdrawal and depression. The cluster and severity of symptoms observed vary from patient to patient depending on the nature of the pathology, however, whatever pattern these character changes take, they cause more stress, more misunderstanding and more alienation of family and friends than do purely physical or isolated cognitive sequelae (Oddy and Humphrey, 1978; Bond, 1975; Dikman and Reitan, 1977).

Clinical observations of the moderately and severely head injured patients admitted to Jacobi Hospital within the past four years have confirmed reports in the literature of some peculiarity of affective response. A particular pattern became discernible in the patients that I had evaluated. The major characteristic of this pattern was social awkwardness. Patients often appeared tentative in their dealings with family members and especially in their interactions with hospital personnel. They did not react appropriately to signals of approval or impatience or annoyance. A patient might smile, for example, while a relative is angry and impatiently rushing him or remain unreactive to signals of approval whether in facial expression or tone of voice. The patients gave the appearance, at times, of completely misperceiving the intent of non-verbal signals communicated to them. Patients who would come to our trauma center to informally visit the staff could linger for an inappropriate length of time, regardless of how busy the staff member was. The conclusion of a conversation, for example, which is generally communicated through a gesture, a facial expression or a finality in tone of voice was completely disregarded by some patients. At times, one could easily misinterpret the resulting behavior as indifferent, insenstive or inappropriate. Alternatively, patients would sometimes appear confused, as if not knowing whether to stay or leave. A possible hypothesis regarding this socially awkward behavior is that patients were having difficulty picking up on the auditory or facial cues that are a part of nonverbal communication.

In addition, some patients present with a peculiarity of vocal expression. Their tone of voice is plaintive, their vocal expression seems contrived or feigned or laughter sounds artificial. Others have speech with a monotonous, dragged out quality to it. Volume is not always modulated appropriately and in some patients remains constant and hypophonic. Patients with this expressive pattern appear as though they are constantly disinterested in events or people around them and they appear depressed. Families have complained that these patient characteristics place stress on family interaction and hospital staff members were observed to be less patient and less jovial and talkative to patients whose voice and general demeanor were monotonous. These observations led to the hypothesis that a combination of expressive and receptive difficulties in these patients makes them appear stilted, uncomfortable and unfriendly, lacking in the alertness and smoothness necessary for effective social interaction. In addition, the described characteristics have been observed to alienate persons dealing with these patients on a daily basis, that is, both family and hospital staff. Clinical follow-up visits to the trauma center at Jacobi Hospital led to the observation that moderately and severely head injured patients can present with these behavioral characteristics for as long as one year post-trauma, and in certain cases, even longer.

Pilot data confirmed the clinical impression that patients were not utilizing the non-verbal affective cues presented to them. When presented with a triad of photographs of different people and asked to choose the two whose facial expressions conveyed similar affect or when asked to find the facial expression in a series of photographs that conveyed a particular emotion, patients made an inordinate number of errors as compared to non head-injured adults. When presented with a photograph of an expressive face and asked to explain what that person might be feeling, patients again made an excessive number of errors. Patient responses to all three tasks were recorded verbatum and examined to determine whether a particular pattern of errors was detectable. The qualitative errors observed across all these tasks were divided into three categories and they accounted for all errors observed. Category 1 errors consisted of confabulatory responses that had no bearing on any detail within the stimulus. Generally, patients would make up a statement about the face in the photograph whose emotional expression they were required to label. The statement was apparently constructed in order to provide a rationale for imposing a particular emotion as an answer. Category 2 errors consisted of focusing on a non-affective detail within a photograph as an aid in responding. A non-affective cue, for example, would be a non-facial feature of the person in the photograph, such as hairstyle, presence or absence of glasses, or focusing on an article of clothing. This particular detail would then become the reason for a person's affective state according to the patient. Category 3 errors consisted of a continual changing of responses.

Figure I provides an example of each category of error made by moderately and severely head-injured patients. In contrast to the examples of errors, Figure I also portrays some of the responses made by a patient who sustained a mild head injury. She presented with none of the features of social awkwardness seen in the more severely injured patients. The mild trauma patient's responses throughout all the pilot tests were characterized by constant appropriate reference to facial expression and facial features. There was no confabulation nor reference to non-affective cues in guiding her responses. One hypothesis derived from the above data is that some head trauma patients may have difficulty in extracting and utilizing the information provided by facial expression.

On a task that required the identification of the emotion in a speaker's tone of voice, patients made many more errors than normals. Similarly, patients' verbal utterances were often judged by normals to be monotonous, lacking in prosodic features. This pilot data was obtained by having patients listen to recordings of the same sentence with various intonations and also by asking patients to produce a sentence in a particular emotional tone and tape recording their responses. Non head-injured adults used as raters frequently labeled patients' speech as "flat" or "neutral". Patients had difficulty in discriminating such basic intonations as anger, happiness and anxiety. Preliminary data on the perception and production of intonation supported the hypothesis that some moderately and severely head injured patients have difficulty in evocative emotional speech as well as in the perception of emotionally intoned speech.

The pilot data concerned with the perception and production of vocal intonation forms the framework for the present study. The tests utilized in this study were desiged to quantify difficulty in perceiving or producing the prosodic elements of speech used to convey affective information. The particular area of prosody was chosen for study because disorders were clincally apparent in this head trauma population. Furthermore, there are reports in the literature of neurological and developmentally disabled populations who present with prosodic difficulty and for whom this disorder is significantly debilitating both socially and occupationally (Wing, 1981; Weintraub and Mesulam, 1983; Bryan, 1977; Tucker, Watson and Heilman, 1977; Ross and Mesulam, 1979; Ross, 1981) and these will be discussed in detail later.

II. The Perception and Evocation of Prosody

Monrad-Krohn (1963) divides oral communication into the three elements of vocabulary, grammar and prosody, where prosody encompasses variations in pitch, rhythm, and stress of pronunciation. The combination of these three features of prosody allow for the expression of a variety of subtle shades of meaning and can also significantly modify the meaning of words or entire sentences. Within these prosodic components, variations in pitch (intonation) are the most important for conveying meaning. In human speech, the principal correlate of pitch is the fundamental (lowest) frequency of the periodic sound and pitch corresponds to the frequency of vibration of the vocal cords. Variations in fundamental frequency during speech give rise to intonation (Fry, 1968). Monrad-Krohn (1963) further refined his definitions of prosody into several different forms. Instrinsic prosody is comprised of the standard prosodic patterns inherent in a language. This is exemplified by the simple declarative statement ending with a lowering of pitch, for example, and a simple question ending with a distinct rise of pitch. Higher intellectual (or propositional) prosody is an extension of instrinsic prosody. It is acquired later in development and allows one to express more subtle shades of meaning. The meaning of a simple declarative statement such as "yes, he is clever" can be changed in a variety of ways depending on the placement of stress and pitch. Emotional prosody conveys emotional expressions as the placement of stress and pitch become more emphatic. Anger, for example, can be displayed by increased volume and lower pitch while joy may be expressed by loudness combined with higher pitch. Inarticulate prosody, finally, represents sounds conveying variations in pitch, stress and rhythm without resorting to words. These sounds may also convey meaning either affective or non-affective.

The prosodic faculty is therefore necessary in order to adequately convey propositional and emotional content in vocal communication. Since we react to other human beings not only in repsonse to their oral expression but in response to their reactions to our expression, the perception of prosody is also an extremely important faculty.

III. Perception and Production of Intonation and Their Neurologic Correlates

Impairment in affective and non-affective prosody has been observed in patients with right hemispheric involvement due to cerebrovascular accident or glioma. Heilman, Scholes and Watson (1975) studied patients with right temporo-parietal dysfunction and noted that patients had a deficit in the comprehension of affective speech, i.e., they made many errors (as compared to patients with left temporo-parietal lesions) in identifying emotional mood when listening to affectively intoned sentences. Tucker, Watson and Heilman (1977) replicated the above-mentioned study with CVA patients alone and found that right hemispheric patients a deficit in producing affectively intoned speech as well as a perceptual deficit. (The control groups in this study consisted of patients with left CVA's and of non-aphasic hospitalized patients with CVA's). Right-hemispheric CVA patients performed more poorly on production of intonation than the control patients without hemispheric lesions. The right hemisphere CVA patients in the Tucker et al (1977) study presented with CT findings of lesions in the distribution of the right middle cerebral artery. Ross and Mesulam (1979) reported on two patients with CVA's in the right hemisphere. Both suffered losses of prosody and emotional gesturing. Infarctions were in the right supra-Sylvian region (posterior frontal and parietal lobes). Patients presented with lateralizing neurologic signs affecting the left side of the body and had both facial and limb involvement. In both patients, expressive speech was flat and monotonous, lacking inflections and coloring. One of the patients lost the ability to cry or laugh. She was unable to express emotion at her father's funeral although she reported that inwardly she felt sadness and wanted to cry. She was finally able to force herself to cry. Her husband reported that the sound was not convincing and quite different from her premorbid expression. Neither patient had aphasic symptomatology and the perception of other people's emotions via intonation, facial expression or gestures was not impaired according to the patient's self-report. (This clinical report raised the point that disturbances in the production of prosody can be dissociated from the comprehension of affective concepts and the perception of affect in others). In the first patient, lack of prosody severely impaired her communicative ability at home and at work and caused her great distress. The second patient was described by his wife as having a changed personality which prompted him to seek help.

Ross' subsequent study of patients with right hemispheric strokes (1981) and the integration of information from studies mentioned above, led him to propose that the organization of prosody in the right hemisphere mirrors the organization of propositional language in the left hemisphere. Of particular relevance to the present study is his differentiation of motor and sensory aprosodia in the manner of motor (Broca's) and sensory (Wernicke's) aphasia. Ross' research coupled with the work of Heilman et al. (1975) and Tucker et al. (1977) led him to conclude that the perception of prosodic features involves the right temporo-parietal area while the production of prosody involves the right supra-Sylvian posterior frontal and parietal areas.

The organization of expressive prosody may involve the basal ganglia and premotor cortex. Monrad-Krohn (1963) noted that dysprosody may occur with lesions of the lenticular nucleus because it was so often encountered in patients with Parkinson's disease and allied neurologic disorders affecting the basal ganglia. Ross and Mesulam (1979) reported that their CVA patient who recovered prosodic speech presented with an infarction which spared most of the basal ganglia. The CVA patient in whom prosody did not recover had involvement of the right basal ganglia. The basal ganglia has rich connections to the premotor area (Penfield and Jasper, 1954) and the premotor area is believed to be involved in the smooth execution of serially organized movements (Luria, 1970): With dysfunction of the premotor cortex an impairment of any dynamic fine-skilled movements can occur, this includes ocular movement, manual movement or expressive speech. Recent regional cerebral blood flow studies have demonstrated an activation of bilateral supplementary motor areas with speech and with other complex movements (Larsen, Skinhoj, Lassen, 1978; Gelmers, in press), strengthening the hypothesis that these areas are involved in the organization and/or execution of sequences of motor activity. A clinical finding supporting this view is Gelmer's (1983) report of a patient who had signficant difficulty in speech initiation and halting speech with an infarct in the right medial frontal cortex. Marsden (1982), following an extensive review of the literature on Parkinsonian movement disorders, concluded that the basal ganglia is not involved in initiation of movement per se but in the carrying out of "motor plans". A "motor plan" is the concept of an action - for example, writing one's name in chalk on a blackboard requires different movements and different sets of muscles from signing one's name on paper with a pen, yet the signature is basically the same. Motor plans are learned activities and are executed automatically. They are comprised of hierarchically lower simple motor programs. Marsden (1982) suggests that the basal ganglia is responsible for running sequences of motor programs to complete a motor plan. Marsden reached this conclusion from evidence that patients with Parkinsonism show an increase in the time taken to execute a movement but show no abnormality in the pattern and timing of motor activity in agonist, antagonist and synergist muscles. Also, while components of a simple motor program are preserved in Parkinson's patients, the execution of a complete motor plan breaks down. A patient who needs to stand up and greet someone would get up, for example, but as he would extend a hand in welcome, could not sustain the first motor act (standing up, postural balance) and fall back into the chair. In attempting to stand up again, the hand extended in greeting might fall back, so that on the whole, the complete intended motor repertoire is not successfully completed. This clinical description of Parkinson's patients is analogous to descriptions of the production of prosody in head trauma patients made by some of the raters. That is, many raters commented that patients would, at times, begin a sentence with a distinct intonational pattern, but could not sustain it and lapsed into monotonous speech mid-sentence or towards the end. It is not inconceivable that traumatic injury to the basal ganglia in head trauma patients could account for this clinical similarity. Levin et al (1983) reported mutism in moderately and severely head-injured patients with basal ganglia injury and minimal observable cortical involvement. During recovery, speech was described as "nonaphasic" and dysarthria was observed in two patients, however, no discussion of prosodic features was presented in the paper. Damasio et al (1982), however, described right-sided lesions of the basal ganglia as producing dysarthric and dysprosodic speech, without aphasia. The right basal ganglia may participate in the "motor plans" of speech, particularly the prosodic components. According to the models proposed by Marsden (1982), if intonational features are learned and stored as motor programs, the basal ganglia may be responsible for insuring that they are linked together to execute a "motor plan" consisting of an intended intonational verbalization.

Marsden (1982) does not propose a specific mechanism for the activity of the basal ganglia. Penney and Young (1983) suggest that feedback circuits exist between the premotor area and the striatum. Dopaminergic neurons are believed to play an inhibitory role in this circuit. With the depletion of dopamine in Parkinsonism the circuit is overactivated, leading to the motor disorders observed. A model of dopaminergic insufficiency is applicable to head trauma patients. Studies of neurotransmitter abnormalities following head injury suggest that mechanical injury to neurons results in a rupture of cells with abnormal leakage of transmitters out of the damaged neurons. Elevated levels of five dopamine and dopamine metabolites (among others) have been observed in severely injured patients and indicate rupture of dopaminergic cells. Furthermore, low levels of DOPAC relative to free dopamine have also been observed. This suggests depressed functioning in damaged dopaminergic neurons (Zazula et al., in prep). Functions dependent on dopaminergic systems should therefore be disrupted following signficant trauma.

The inability to sustain an intonational pattern in head trauma patients was described earlier as an analogue to the fading out of movement patterns in Parkinson's patients. Other clinical similarities, specifically in speech, are evident between the two types of patients. Scott and Caird (1983) have described Parkinsonian speech as monotonous, increased in pitch, varying in pitch, and reduced in voice intensity with an abnormal rate of production. This description is remarkably similar to the speech observed in many moderate-severe patients following head trauma. An emergent hypothesis based on all of the above descriptions and findings is that the disorders of prosody observed in head trauma patients may be related to a disruption of the circuitry involving the (right) basal ganglia and frontal premotor cortex.

To the extent that perception of intonation involves the analysis of an auditory input, the neural representation of such an auditory message involves the brainstem. Fibers from the cochlea terminate in the cochlear nucleus in the medulla. The majority of these fibers decussate here ad proceed to the medial geniculate body and then via auditory raditions reach the auditory cortex (Critchley and Henson, 1977). Lesions of the brainstem are known to disrupt auditory processing: for example, when incomplete information is presented to each ear, a fusion of the information, first at the brainstem level and then at higher levels, renders the message in more complete form cortically. Brainstem lesions interfere with this fusion effect (Bergman, Hirsch, Najenson, 1977). Pallay, et al (1984) studied patients with compression from extra-axial tumors of the posterior fossa. They report that braintem compression affects the rate of acoustic information processing. Deficits were observed in tests of time compressed speech. These findings are in agreement with reports that lesions of the central auditory nervous sytem affect perception for speech which is either distored in some way or presented in competing arrangements to the two ears. The concept is that these lesions disrupt the integrative function of the brain and are less disruptive in the peripheral organ of hearing or to language functions such as symbolization or memorization. Other auditory studies of patients with gliomas compressing brainstem regions report a reduction in speech discrimination for very intense speech (i.e., when the speech intensity level is raised) and for speech which is degraded by background competition. No hearing loss per se, that is, no abnormal response to pure tone stimulation was oberved in these patients (Dirks, 1978).

Literature on acoustic information processing indicates that the medial geniculate bodies may be particularly important in judging the timing of the arrival of the auditory stimulus (Barr, 1972). The reticular activating system is important in acoustic processing in that it is the neurologic alerting system which sensitizes cortical areas to respond to signficant stimuli (Barr, 1972). Raised ICP and brainstem compression produce dysfunction in the reticular activating system (Jennett and Teasdale, 1981) and distort thalamic fibers (Tomlinson, 1970) impeding processing at both these levels.

Although the classification of auditory characteristics that differentiate affective from non-affective speech is a major study in and of itself, certain components of affective speech can be immediately recognized. For example, angry speech may very often be louder whereas nervous speech may be higher in pitch and quicker in tempo. Difficulties in the perception of temporal patterns and higher amplitude will interfere with the interpretation of such auditory patterns. This type of perception requires extracting information at a level different from the semantic content of a statement. To the extent that fluctuations in tempo and amplitude underlie the expression of different emotional states, patients with brainstem compression may be handicapped in their perception of such information.

In summary, there is evidence that the right hemisphere is involved in the perception and production of prosodic features. Furthermore, specific areas within the hemisphere may be involved: posterior areas are implicated in perception and anterior (frontal) and subcortical (basal ganglia) areas have been associated with the production of prosodic features. Finally, disruption of brainstem pathways may also interfere with the processing of the acoustic intonational speech.

Specific studies of prosody in brain damaged populations have focused primarily on stroke and tumor patients. The study of these problems in a head trauma population is of interest because head trauma produces cerebral damage that is diffusely distributed throughout the white matter, frequently involves both hemispheres and is rarely, if ever, as specific as that which may be observed with stroke or tumor patients. At the same time, many trauma patients present clinically with the same deficits and behavioral characteristics as the right hemisphere stroke or tumor.

IV. General Behavioral and Affective Effects of Head Trauma

Head injury results in a variety of cognitive and affective sequelae. A discussion of these will serve to illustrate the complexity of post-traumatic symptoms. The interaction of these varied sequelae with the observed communicative deficits in affective prosody can make social re-adaptation for the trauma patient extremely difficult. This is particularly true where sequelae include affective disorders. Heilman and Valenstein (1979) report that while people dealing with the brain-injured readily attribute motor or sensory deficits to neurological dysfunction, they tend to ascribe emotional disorders to interpersonal, intrapersonal and/or environmental conflicts. Agitation and depression in hemiparetic patients with right hemisphere involvement, for example, was interpreted by spouses as a breakdown in interpersonal relations. The affective component of the patients' disabilities contributed most strongly to stress and misunderstanding within the family.

The patient who has had a moderate to severe head injury emerges from coma into a disoriented, confused state. In the early phases of recovery, patients remain sleepy and indifferent with fluctuating attentional processes and frequent mood changes without obvious reason. When stressed, the patient can pass over into somnolence, delirious states or coma. This state corresponds to Bleuler's (1951) "clouding of consciousness".

As the patient continues to recover, and becomes more attentive and lucid, memory deficits become noticeable. In addition, the whole personality appears to be affected. The patient frequently presents with illogical trains of thought, impoverishment of ideas in arguing a point with vague and generalized concepts, and an abnormal influence of mood and impulse on intellect. Patients may grasp only one detail or aspect of a situation neglecting other relevant features. There is frequently a tendency towards confabulation, perserveration, increased fatigability and labile emotionality. Emotional responsiveness can also become restricted, primarily because situations are not always readily understood. General apathy is frequently reported as well. Bleuler (1951) called this constellation of behavioral features the "amnestic syndrome". At this point in recovery, little is expected of the patient socially, and the primary focus is on physical recovery. However, once the patient is discharged, neuropsychological sequellae and personality traits become more important because they affect social and occupational integration.

Goldstein (1952) vividly described how the sequelae of brain injury can affect a patient's emotional reactions. He suggests that the brain-damaged individual has an "impairment of abstract attitude". Goldstein defines the latter as loss of the ability to take initiative, inability to shift mental set or to make a choice, inability to account for one's actions, inability to process simultaneously various aspects of a situation, loss of part-whole relationships or inability to grasp the essence of a whole, and inability to plan ahead ideationally. These patients are frequently in danger of being in a catastrophic condition since they have a constant difficulty in performing a variety of tasks. Goldstein gives, as an example, a patient who appears dazed, agitated, starts to fumble, becomes unfriendly, evasive or aggresive when he is not able to perform a task. Secondly, such patients may appear either emotionally dull or overexcited. Goldstein postulates that inappropriate emotional reactions in brain injured individuals are the result of an inability to grasp a situation in an integrated manner. Patients may grasp only one aspect and react to that alone.

These affects of brain damage on the personality illustrate the potential social problems of a neurologically impaired individual since interactions with others are certainly affected by catastrophic reactions or inappropriate behavior.

The behavioral sequalae of closed head injury and their relation to severity of injury have been studied in 70 patients who had sustained blunt trauma to the head (predominantly motor vehicle accidents) by Levin and Grossman (1978). Patients were divided into three grades of severity based on the duration of coma and the presence or absence of neurologic deficits. Grade 1 patients (mild) were concious on admission and throughout the hospital stay and presented with no neurologic deficits. Grade 2 patients (moderate) were comatose for 24 hours or less and may have had neurologic deficits. Grade 3 patients (severe) were in coma for over 24 hours and may have also manifested neurological signs. Patients were rated only after the phases of acute confusion and disorientation had subsided or had at least reached a stable level without further signs of improvement. To control for the possibility that duration of convalescence would affect the degree of residual psychicatric disturbance, behavioral ratings were adjusted for variation in the injury-to-test interval in an analysis of covariance. Behavioral ratings were carried out via the Brief Psychiatric Rating Scale (BPRS) of Overall and Gorham (1962). This is a practical and reliable test of 18 behavioral scales each of which is rated by the examiner on a scale ranging from 1 (no observable symptom) to 7 (most severe manifestation of symptom). In addition to individual scores for each symptom, a total score (sum of all individual scores) can be obtained and utilized as a global index of psychopathology. BPRS ratings in the sample of patients studied in this sample were based on a semistructured clinical interview with the patient, unobtrusive behavioral observations and the patient's behavior during neuropsychological testing. The three grades of severity did not differ from each other in terms of the patients' mean educational level. The mean ages for the three severity levels did differ, however, it was felt that this did not invalidate further comparisons since the behaviors studied are not known to vary within the age ranges observed in this study.

Maximal differentiation of the three grades of injury emerged along the scales measuring emotional withdrawal (i.e., isolation and lack of spontaneous interaction), conceptual disorganization (disconnected thought processes), motor retardation (slowed movements and speech), unusual thought content, blunted affect, excitement (increased reactivity, agitation) and disorientation (confusion, disorientation for person, place, time). Severely injured patients significantly differed from mild patients on all of these scales. Both blunted affect and excitement were differentiating factors seen in severely injured patients suggesting tht there was considerable variation in the form of affective disturbance seen after closed head injury. Moderately injured patients exhibited the above mentioned behavioral patterns to a lesser extent than severe patients but to a greater extent than mild patients. Patients with moderate injuries were noted to be particularly uncooperative and exhibited the greatest resistance towards the examiner: of all three groups, moderate patients exhibited the highest scores on the BPRS scales of Tension, Hostility and Uncooperativeness. Mildly injured patients exhibited no significant signs of behavioral disturbance other than axiety and somatic concern.

Within the group of severely injured patients, a subgroup of injuries involving the mesencephalic brain stem (6 patients) were compared with the other (15) severe patients. Evidence of less anxiety and depression in the brain stem patients was observed. There was also a mild trend towards greater thinking disturbance in these patients but it failed to reach statistical significance. ("Thinking disturbance" was derived as a composite of the BPRS scales of Conceptual Disorganization, Hallucination and Unusual Thought Conent).

Examining cases in terms of correlation between BPRS patterns and site of relatively greatest injury revealed no significant differences across hemispheres or within temporal, orbitofrontal or fronto-temporal regions as major sites of injury.

Hemispheric injury was inferred from the presence of a hemiparesis, unilateral hematoma, unilateral depressed skull fracture or contusion found on CT or arteriography. Focal injury in the temporal, orbitofrontal or frontotemporal regions was again inferred on the basis of CT, arteriography, or surgical findings in patients with depressed skull fracture or hematoma. The lack of correlation of behavior (as assessed by the BPRS) with any observable localized site of injury reflects the generally diffuse effects of closed head injury. Levin and Grossman (1978) are aware of this diffuse pattern of pathology and in fact state that one could not presume that any patient with closed injury sustained damage to only a single cerebral area. Their examination of frontal and temporal pathology was carried out only because of the particular vulverability of these areas to injury and because of their well-documented role in psychiatric disorders.

Patterns of personality changes observed in focal brain pathology have generally been extracted from studies of penetrating head injuries since, as mentioned above, damage in closed injuries is too widespread to reliably claim injury at an exclusive site. Even with penetrating injuries, edema and resulting circulatory disturbances usually affect areas beyond the immediately affected tissue (although to a much lesser extent than in closed injuries).

Nevertheless, studies of focal pathology have provided some insight into the organization of affective processes in the brain. The effects of both frontal lobe pathology and temporal lobe involvement will be considered.

Frontal lobe injuries produce marked personality changes which have been widely documented. They have been associated with a significant excess of psychiatric disturbance. The symptoms observed are remarkably invariant and cut across differences in premorbid personality. Generally the patient exhibits a lack of foresight, tact and concern, inability to judge the consequences of actions or to plan ahead and frequently assumes a euphoric disposition. Disinhibition is a common feature and behavior can become socially inappropriate to the point of becoming quite disabling with extensive lesions. With bilateral orbitofrontal lesions the above characteristics are especially severe with irresponsible and antisocial conduct frequently observed (Lishman, 1973). Lishman (1973) reports that orbital parts of the frontal lobes when injured are responsible mainly for the emotional changes (i.e., changes of mood and attitude - euphoria, facetiousness, irritability, apathy, attentional defects). Orbitofrontal patients frequently fail to maintain satisfactory relationships with others, lack perserverance, and tend to be aggressive and demanding. Increased libido is observed in these patients and criminality is not uncommon.

Nauta (1971) has postulated that the inappropriate behavior of the frontal lobe patient is due to poor integration of visceral hypothalamic responses with information from the environment provided via multiple cortical channels. The integration of external and internal information occurs in the frontal lobes according to Nauta, and this integration results in affective guides to behavior. It allows one to plan one's responses so that the outcome of an action is a socially acceptable one. The individual who has normal neuronal functioning always utilizes these "affective reference points" as Nauta calls them. The individual with frontal lesions or any disruption of frontal-hypothalamic circuitry loses his grasp on finely tuning his behavior. With frontal or midline fiber damage in trauma, a patient could, hypothetically misinterpret vocal intonation, due to either an inadequate visceral reaction to the stimulus at the hypothalamic level or inadequate reception of the stimulus at the auditory level, or inadequate integration in the frontal lobes of the auditory input with its visceral connotation.

Recent studies are confirming hypotheses regarding the importance of the frontal lobes in affective functioning. Regional cerebral blood flow studies in schizophrenics have shown reduced CBF in the frontal gray matter as compared to normals (Ariel et al, 1983). A study comparing left versus right frontal lesion patients indicated that left frontal patients were impaired on tasks of non-social cognition, right frontal patients were impaired on cognitive tasks with a social component and bilateral frontal patients were impaired on both tasks (Sorman, Wasserstein and Zapulla, 1983). It is expected that frontal lobe injury subsequent to trauma will disrupt the integration of affective information.

Temporal lobe injuries are reportedly associated with reduced control over aggressive impulses (Hooper, McGregor and Nathan, 1945). Injury affecting the periamygdaloid region is suspect in this behavior.

Sweet, Ervin and Mark (1969) have noted evidence linking violent behavior with pathology in medial temporal lobe structures. Hillbom (1951) has linked temporal lobe injuries to schizophreniform psychoses. In addition to the above features of temporal lobe disease, Baer et al (1982)have found epileptics with temporal lobe foci to be overly clinging, humorless, circumstantial, excessively paranoid and moralistic.

Although the temporal lobes are associated with affect, their relevance to this study lies in their role as corticon-cortical afferents to the frontal cortex (Nauta, 1971). Visual, auditory and somatic sensory systems reach the frontal cortex for processing via two temporal routes:

a) the anterior temporal projections via the uncinate bundle to a large portion of frontal cortex,

b) the middle and inferior temporal gyri projections via the inferior thalamic peduncle to the mediodorsal thalamic nucleus to the orbitofrontal areas.

To the extent that temporal-frontal pathways are disrupted, processing of affective information should be disturbed.

In summary, the head-injured patient is most vulnerable to injury in areas of the brain that integrate affective information and that are associated with the highest psychiatric hazard. In addition, he loses the overall ability to abstract which further compounds the problem, and places him at a social disadvantage.

V. The Neuropathology of Head Trauma

Severe head injuries result in multiple types of brain lesions and a diversity of secondary complications that further contribute to tissue damage. Langfitt and Genarelli (1982) report that there appear to be two principal causes of brain damage in head injury. One is the direct mechanical damage to neurons produced on impact (Adams et al, 1982) and referred to as the shearing of nerve fibers. This tears the neuron, especially the axon, and can be observed throughout the white matter in the hemispheres and brainstem in severe cases. The second cause of brain damage is an insufficient supply of oxygen to brain cells. Ischemia is generally caused by compression from a hematoma or raised intracranial hypertension from diffuse brain swelling. Ischemic damage is pernicious and Langfitt and Generalli (1982) speculate that it may already be irreversible at four hours.

Oppenheimer (1968) described diffuse microscopic lesions attributable to accelerations within the brain substance at the time of injury. They consisted of multiple capillary hemorrhages or microscopic disruptions of nervous tissue without hemorrhage. A diffuse proliferation of microglia was observed in these cases. Lesions were observed primarily in the corpus callosum, caudate nucleus, centrum ovale and in the upper brainstem (tegmentum and brachia conjunctiva). They were infrequent in the cerebral cortex. Most of Oppenheimer's cases were severe head injuries, dying from the effects of the brain injury combined with cerebral anoxia from chest troubles. The causes of the observed microscopic lesions were shearing of fibers as a result of impact, stretching and tearing of small blood vessels giving rise to hemorrhages of all sizes and stretching and tearing of groups of nerve fibers without hemorrhage.

Taylor (1967), employing an animal model, has discussed the neurologic sequalae due to a concussive blow to the head. He hypothesizes that the brain stem and cord are stretched as the neck is hyperflexed and rapidly decelerated. The cord is stretched and elongated but is fixed by the cranial nerves (especially V, VII, VIII) and other fibers. Consequently, this pull will be felt throughout the brainstem and forward through the long tracts to where the forebrain is fixed by the optic nerves, olfactory bulbs and carotid arteries. As a result of this stretching, fibers from the brain stem to the centrum ovale are actually fractured or snapped. Post-concussive symptomatology such as loss of consciousness, mass contraction of the limbs, cessation of respiration, rise in blood pressure, loss of corneal reflexes, dilated pupils, headache, vomiting, dizziness, confusion, and amnesia are all symptoms which could arise from damage to cranial nerve nuclei between the medulla (X) and the upper brain stem (III) as well as from the arousal center in the reticular formation, the pyramidal tracts and the vasomotor center. If damage is sought in the brain of the concussed animal this is the area in which it is mainly found. These lesions are present even after minor concussive blows to the head. Nerve cell counts, for example, in the lateral vestibular nucleus, are invariably reduced after a minor concussive blow and this effect is not a transitory one. Irregular thickening and fragmentation of myelin sheaths was also found in the areas described above.

Concussion is known to cause widespread lesions throughout the white matter. Taylor (1967) reports that maze running performance in animals one month post-concussion was impaired. Similarly, the animals had difficulty in learning new tasks.

Most patients with head injury are now considered to have a combination of white matter shearing lesions and cortical contusions. Ommaya and Gennarelli (1974) and Adams, Graham and Genarelli (1983) report that the prime location of contusions is in the temporal and frontal regions irrespective of the site of impact.

In addition to the above-mentioned lesions, raised intracranial pressure is a complicating factor in a high proportion of head injury cases. Severe contusions, for example, may lead to a significant occupation of space once edema and local extravasation of blood develop around them. Diffuse brain swelling may have the same effect. Both lead to increased pressure within the cranium with a resultant distortion of brain structure, midline shift or shifting of structures downward toward the brainstem. Raised intracranial pressure may impair perfusion of the cerebral cortex or produce dysfunction in the reticular activating system and other brainstem areas due to pressure and distortion leading to stenosis of blood vessels. For example, when the brain stem is shifted downwards, the basilar artery is not. The perforating branches from the basilar artery to the brainstem become stretched and narrowed and the blood supply to the brainstem is reduced with a resultant impairment in functioning (Jennett and Teasdale, 1981). This type of ischemic damage is significant, yet frequently goes unreported because it is not observable with the naked eye. Alternatively, hemorrhaging in the brainstem can occur secondary to compression (Adams et al, 1983).

Jennett and Teasdale (1981) report that hypoxic/ischemic damage can develop within a few hours post-trauma and devastate the cerebral cortex. It is observed only with proper microscopic examination. In their sample of 151 patients dying of head injury, 91% had some evidence of ischemic brain damage (83% had raised ICP). Forty-six percent of patients had lesions in the cortex, primarily in the boundary zones of adjacent major arterial territories. Seventy-nine percent of patients had lesions of this type in the basal ganglia.

Jennett and Teasdale (1981) report on a series of of 151 autopsied patients studied by Adams and Graham. These were patients with nonmissile head injuries who reached the hospital alive and for whom surgical management was considered appropriate. Very early deaths were under-represented in this sample. Of these 151 patients, 91% had some evidence of ischemic lesions in similar cortical and basal ganglia areas but a high percentage of brainstem lesions were reported as well.

Bratzke (1983) studied brainstem lesions in twenty cases of blunt head injury whose survival times ranged from 43 days to 1 and a half years. The patients ages ranged from 7 to 60 years of age. Fifteen patients were younger than 40. All died of complications not directly related to trauma (primarily pneumonia). Marked brainstem atrophy was observed in this series of patients. In addition, expansion and deformation of the aqueduct and 4th ventricle was observed. Lesion quality indicated that they were due either to anoxia (secondary type) or direct damage against the edge of the tentorium when the head was flexed (primary type). The author claims that the types of lesions observed depend on the duration of the anoxic phase and the subsequent extent of reperfusion.

In head injury, more than one of the factors that are likely to reduce cerebral oxygenation are commonly found: raised ICP, hypoxemia, anemia, systemic hypotension, defective autoregulation. It does not take a marked abnormality in any one factor to affect oxygenation if more than one is contributing. Jennett and Teasdale (1981) speculate that the extensive hypoxic lesions found in cases of fatal head injuries occur in survivors but to a less severe degree and that they contribute significantly, as do other lesions discussed, to the neurologic and mental sequelae observed post-trauma in survivors. Their view of differences in degree but not type of lesion is shared by others. Symonds (1962) has postulated from clinical observations of concussed patients, that the difference between patients who remain unconscious for weeks rather than for minutes or hours could be in the quantity of brain damage and not in the location of the lesions. He suggested that varying grades of head injury should be recognized and that varying degrees of white matter damage, for example, accompany the different severities of injury. Pathological evidence supporting this view was provided by Oppenheimer (1968) who found similar types and distributions of lesions in both clinically severe cases dying from cerebral injury and in cases where cerebral injury was clinically trivial. The latter cases suffered from mild "concussion" lasting only a few minutes from which the patients quickly recovered. Cause of death was usually due to pneumonia or fat embolism. Oppenheimer's findings are congruent with results of animal studies of head trauma where both severe fatal injuries and minor concussive blows were found to produce lesions of similar distributions and with similar types of destruction to neurons:

fracturing of white fibers with thickening and fragmentation of myelin sheaths (Taylor, 1967). Animal models, as well as patients with head trauma who do not die of head injury but rather of unrelated causes, allow us to understand the pathology of trauma, and in some cases to relate this to behavioral outcome.

VI. Assessing Severity in Head Trauma

1. GCS

Variability in classifying the severity of head injury across national and international trauma centers interfered with interstudy comparisons of outcome from trauma. In particular, it was not clear whether differences in mortality in different series of head-injured patients were due to differences in management or to variability in the severity of patients studied (Langfitt and Genarelli, 1982). In response to this problem, a suggestion was made to standardize the definition of a serious head injury and consequently to standardize the criteria for admission to studies of head injured patients (Langfitt, 1978). The Glasgow Coma Scale (GCS) (Teasdale and Jennett, 1974) has been widely applied in head trauma studies and has been accepted as a prevalent measure of severity of injury and a basis for comparing patients across studies. The GCS is effective in predicting mortality (Wassertheil-Smoller et al, 1982), however, its relationship to long-term cognitive outcome is poor (Tabaddor, Mattis, Zazula, 1984, Levin et al, 1979). Nevertheless, for the purposes of comparability with other studies, the GCS was utilized in the classification of patients admitted to the Central Nervous System Trauma Center at Jacobi Hospital.

2. WAIS

The Wechsler Adult Intelligence Scale (WAIS) (Wechsler, 1955) is one of the most commonly used psychometric instruments. The Verbal and Performance I.Q. of the WAIS do not vary appreciably in a normal individual, however, large discrepancies may be seen in a patient with brain dysfunction (Strub and Black, 1981). Differences exceeding 25 points have been shown to be statistically significant (Field, 1960), while differences in 15 points have been shown to have clinical utility in identifying neurologic dysfunction (Black, 1974). Lower Performance I.Q.'s relative to Verbal I.Q.'s were typically displayed by patients with documented right hemisphere damage (Simpson and Vega, 1971; Reitan, 1955). However, bilateral damage following blunt head injury produces the same pattern of lower Performance I.Q. (Mandleberg and Brooks, 1975). Levin et al (1979) found that overall outcome in moderately and severely head-injured patients was more closely related to variation in Performance I.Q. as compared to Verbal I.Q. Furthermore, they found that Verbal I.Q. significantly correlated with educational level whereas Performance I.Q. did not. Mandleberg and Brooks (1975) attribute poorer Performance I.Q. in head trauma patients to the fact that Performance I.Q. subtests require the integration of complex functions including perception, learning, manual dexterity, speed of response and attention. Additive failures on any of these tasks will depress Performance I.Q. Verbal subtests are structurally simpler and, except for Arithmetic, are untimed. Jennett and Teasdale (1981) share the views of Mandleberg and Brooks, suggesting that Performance I.Q. subtests depend on the integration of a wider range of cerebral activities than do Verbal subtests. Motiviation, attention, speed of response and organizational ability are all reflected in PIQ and are the deficits commonly complained of by head trauma patients and their families. Performance I.Q. in the present study can be useful as an index of severity of injury.

VII. Neuropsychological Indices of Asymmetric Pathology

Asymmetry in the perception and production of intonation was suggested by the studies discussed in a previous section of this paper. Two neuropsychological tests commonly utilized in the lateralization of lesions will be applied in the present study as markers of left and right hemispheric functioning in the present series of patients.

The Purdue Pegboard (Science Research Associates 1948) is a test of manual dexterity (Costa et al, 1963; Vaughan and Costa, 1962). In a study of the lateralizing efficiency of this test, cutoff scores that were 70% accurate in predicting a lateralized lesion were developed (Costa et al, 1963). Slowing in one hand on bilateral slowing is seen with diffuse or bilateral brain damage. The Purdue was utilized in the present study as a lateralizing indicator of left or right hemispheric dysfunction and their relationship to tests of prosody.

In auditory dichotic testing, the auditory capacity of each ear is tested by using stimuli (such as digits or letters) delivered simultaneously through headphones by a dual track sound system (Kimura, 1967). The patient receives stimulus pairs, one to each ear, at the same time. Normally, the different stimuli received by each ear are both heard. When stimuli presented to an ear are poorly understood or not perceived at all, a lesion involving the auditory system on the contralateral side can be suspected (Lezak, 1976). Patients with lesions involving the temporal lobe or central auditory pathways may ignore auditory signals entering the ear opposite the side of lesion in the same way that unilateral neglect or extinction to double simultaneous stimulation in tactile (or visual) modes is observed on the side contralateral to a lesion (Oxbury and Oxbury, 1969). Simple auditory dichotic listening for letters was utilized in the present study as a lateralizing marker reflecting the degree of correlations of right versus left hemispheric auditory pathways and auditory association areas with the perception and production of prosody.

VIII. Assessing Social Maturity

The patients discussed above, who suffered from dysprosody had difficulties in social and occupational spheres because their affective expression was frequently misunderstood (Ross and Mesulam, 1979; Weintraub, Mesulam and Kramer, 1981). Although there is no direct evidence from the stroke and tumor patients discussed above, there is evidence that persons with receptive affective difficulty due to other neurologic or developmental syndromes (Asperger's syndrome, learning disabilities, for example) experience great difficulty in interpersonal relations and can have difficulty in adjusting to such occupational demands as interacting effectively with superiors or co-workers and maintaining employment (Weintraub and Mesulam, 1983; Wing, 1981). If affective perception and production is truly such an important component of social and occupational success, then head trauma patients who present with receptive or expressive affective disturbance should be less likely to adjust within these spheres than patients who do not exhibit such post-traumatic sequelae. The measurement of social competence and its relation to the perception and production of intonation would serve as a preliminary evaluation of this proposed relationship.

The Vineland Social Maturity Scale (Doll, 1935, 1953) was chosen to evaluate social competence in the present sample of patients. This scale was designed to gauge an individual's capacity for looking after oneself and for participating in activities which are associated with ultimate independence as adults. The test items within this scale are intended to represent maturation in the areas of "self-help, self-direction, locomotion, occupation, communication and social relations" (Doll, 1953). The underlying principle of this scale is that each of the above-mentioned items is viewed as representing social responsibility in some detailed performance that is an overt expression of that responsibility. The following are examples of questions that represent various areas of competence assessed by the scale:

self-help:	"Tells time to quarter hour"
self-direction:	"Is left to care for self or others"
locomotion:	"Goes to distant points alone"
occupation:	"Has a job or continues schooling"
communication:	"Makes telephone calls"
socialization:	"Assumes responsibility beyond own needs"

Patients who have difficulty with affective perception and production affecting social interaction should regress on a number of items within this scale if reports in the literature cited earlier are accurate.

METHODS

Subjects

The patient sample studied was comprised of 27 right-handed moderately and severely head injured adults enrolled in a comprehensive head trauma study conducted at Jacobi Hospital (1980-present). Criteria for inclusion of patients in this study include no history of hospitalization for drug or alcohol abuse, no prior head trauma, and no history of hospitalization for psychiatric disorder. Patient demographics are represented in Table I. Severity of injury is represented by Glasgow Coma Scale scores (GCS) (Teasdale and Jennett, 1974). GCS scores represent the lowest scores attained by the patient during the in-hospital stay and can reflect deteriorative processes not always evident on admission. Scores less than 8 represent the 'severe' category, while scores of 9 to 12 represent the 'moderate' category. Patients who present with GCS scores greater than 12 in this group are also categorized as 'moderate' on the basis of presence of mass lesion on CT or focal neurological signs. The mean GCS score for this patient population as well as the proportions of moderate and severe injuries is represented in Table I.

Table II presents the neuropsychological status of the patients. As a group, they present with a wide range of abilities extending from the severely defective to above average.

Table III presents the results of the Vineland Social Maturity Scale which was administered at the same time as the neuropsychological evaluation and tests of affective prosody. The 'Social Age' of the patients as a group is about half of their group chronological age. The

'Social Quotient' of the group is considerably lower than average, i.e., considerably lower than the 50th percentile for adults as described in the manual.

Thus, the patients as a group present with both intellectual deficits and lowered levels of social competence.

The control sample consisted of 10 adults with no history of head trauma, psychiatric disturbance, neurologic dysfunction or alcohol or substance abuse. Six were either relatives or close long-term friends of the patients described above. The remainder (n = 4) were Jacobi Hospital employees of similar socioeconomic and cultural background. Demographics of the control population are represented in Table I.

Patients and controls differ with respect to the proportion of males and females in each sample (Table I). Sex differences might be expected to contribut to differences in cores due to suggested differences in cerebral organization between males and females (Landsall, 1970, 1971; McGlone, 1976). T-tests indicated that no significant differences existed between male and female patients on perception of intonation, Verbal or Performance I.Q., age, education level, Social Quotient or time from injury (See Appendix I). In addition, no difference was observed for the Purdue Pegboard. Production of intonation differed signficantly in male and female patients. However, due to the above pattern, this may have occured by chance alone. (Furthermore, a lateralized test such as the Purdue should have reflected any real differences in performance due to different cerebral organization). Neither perception nor production of intonation differ between males and females in the control population.

Patients and controls also differ with respect to educational level (Table I). Education does not correlate significantly with perception or production of affect in the control population or the patient population. A subset of patients were closely matched to controls with respect to education, and scores on perception and production were compared to determine whether educational differences were accounting for differences in test scores. Patients and controls do not differ with respect to age (Table I).

Subject Selection

Patients were consecutively enrolled in the present study as they were admitted to Jacobi Hospital for head trauma, or as they kept their follow-up out-patient appointments at the Central Nervous System Trauma Center at Jacobi Hospital. Thus, the time from injury to evaluation varied from one week post-trauma to two years post-trauma. Controls were obtained whenever possible, from patient's families or friends or at the convenience of Jacobi Hospital personnel.

Test Administration

Patients were informed about the purpose of the study and asked to sign an informed consent form before proceeding. Affective tests of perception and production were embedded with a full neuropsychological evaluation which was part of the C.N.S. Trauma Center protocol. Tests of affect were administered early in the battery, following the administration of the first test, the Wechsler Adult Intelligence Scale. Patients were thus acclimatized to the testing situation when the affective tests were given. Controls were informed about the purpose of the study, and received a consent form. They received tests of perception and expression of affective prosody, however, no neuropsychologic tests were administered.

Test Description

A. Perception of Affective Prosody

The perception of affectively intoned stimuli was tested by having subjects listen to a tape comprised of 70 content-standard sentences presented in 5 intonational patterns. The subject was asked to listen to each sentence and identify the emotional quality in the speaker's voice from among the following choices: anger, anxiety, confidence, happiness, neutral. Subjects were asked to define the 5 states mentioned above before proceeding to insure that their verbal comprehension of these words was intact. All subjects wre capable of accurately defining the meaning of the five test words. The tape begins with a standardized set of instructions:

> "You are about to hear a series of vocal expresions of emotions. The verbal content of each statement will remain the same but the emotions expressed may differ. The emotions portrayed will be one of the five presented at the top of your answer sheet. A given emotion may be presented any number of times and in any order. Try to identify the emotions expressed. Please do not leave any blank spaces. If you are not sure, guess."

At this point, any further questions on the subjects' part were clarified and testing began. The score on this test consists of the number of correct identifications made by the subject.

B. Production of Affective Prosody

The expression of affectively intoned speech was tested by asking subjects to produce a content-standard sentence in one of four intonational patterns: happy, anxious, angry, or neutral. Twenty sentences were elicited from each subject (5 samples of each intonation presented randomly within the 20 trials). Sentences were recorded during the testing session for all subjects. Eighteen raters were recruited to evaluate the subjects' performance on the production task. The raters' ages ranged from 19 - 61 (X = 34.23) and educational level ranged from 13 - 21 (X = 15 years). Raters were asked to listen to the series of tapes and identify the speaker's intonation after each sentence. A multiple choice format was presented and the rater identified each expression as happy, anxious, angry, or neutral. Raters were not informed in advance that they were listening to patients and controls, and control tapes were randomly placed in between sets of patient tapes. Each patient and each control received a score ranging from 0 - 20 from each rater. (A score of 20 is one where all sentences identified by the rater match the intonation requested of the subject by the experimentor). The overall Production score was derived by taking the mean of the scores produced by each of the raters for that subject.

C. Conceptual Identification of Emotion

Subjects were presented with four words each of which represent an affective state: boredom, love, surprise, hope. Each experimental word was presented with a list of twelve statements descriptive of various affective states. The experimental words and descriptive statements were taken from Davitz (1970). This source is a compiled list of 556 words and phrases that had been used to describe emotion by a group of

50 students. Davitz presents frequency information on each descriptive statement. The form utilized in the present study was developed using six high frequency terms (50%) and six low frequency terms per word to be described. (Low frequency terms were either terms utilized with high frequency for other emotions, or terms utilized with very low frequency (e.g., 30%) for the word to be defined). Thus each experimental word was presented with statements of variable applicability to that word (see Appendix II).

The experimental words selected to be defined were chosen because they are more abstract, and it was felt that these words would well represent emotional conceptualization.

Subjects were asked to read the descriptive statements following each word and to check "yes" for those statements which applied to the word and "no" for those statements which did not apply. Subjects were asked to use their own judgment in answering each statement.

Forty-eight statements per subject were available for analysis. For each statement, the percent of agreement was calculated separately for patients and controls. Thus for statement 1 under 'hope', 100% of the normals and 80% of the patients responded "yes" (see Figure I). Graphs for each of the four experimental words were plotted (Figures 1,2,3,4). The percent of agreement for each statement in the control group was plotted first. Patients were then analyzed according to the percent of agreement with the prevalent response in the normal group (yes or no).

D. Vineland Social Maturity Scale

Patients were administered this scale in order to gauge their level of social responsibility. The Vineland Social Maturity Scale (Doll, 1935, 1953) is composed of items that represent, in increasing difficulty, factors such as "self-help, self-direction, locomotion, occupation, communication and social relations" (Doll, 1965). These factors are represented by some detailed performance which the patient is able or not able to perform. The overall score should reflect the patient's ultimate level of independence. It is useful in this study since one needs to know a patient's level of adjustment and social status post-trauma. Furthermore, one would like to know the relation between receptive and expressive affective disturbance and the patient's overall level of functional competence.

E. Neuropsychological Battery

Each patient in the present study received a full neuropsychological battery as part of the C.N.S. Trauma Center protocol. Selected tests from this battery were correlated with the experimental affective tests and indices of brain pathology. The following tests were utilized for this purpose:

1. Wechsler Adult Intelligence Scale: (Wechsler, 1955)
    - Performance I.Q.
    - Verbal I.Q.
2. Purdue Pegboard (Science Research Associates, 1948)
3. Auditory Dichotic Listening (Kimura, 1967)

Pathological Data

CT scores were routinely obtained for most moderately and severely head injured patients. The CT scores utilized were preoperative scans using an EL-Scint 705 Scanner or a GE 8800. Only nonenhanced scans (no contrast agent) were used in the present study for the purpose of evaluation. Each CT scan was reviewed to determine the extent of ventricular shift, compression of brainstem cisterns, and the size and location of parencymal mass. Of the twenty-seven patients in this study, twenty-two had CT scans which were available for review.

HYPOTHESES

A. Major Hypotheses

1. Moderately and severely head-injured patients present with disorders of receptive and expressive affective intonation.
2. Disruption of perception and production of intonation need not necessarily be accompanied by disorders in affective concept formation.
3. Deficits in perception and production of intonation are reflected in social status post-trauma as measured by the Vineland Social Maturity Scale.
4. There are identifable pathological patterns in a head trauma population which increase the likelihood of deficits in the perception and production of intonation.
   a) Right hemisphere pathology contributes to deficits in both perception and production: right frontal lesions are associated with production deficit, right temporo-parietal lesions are associated with perceptual deficit.
   b) Brainstem compression (suggestive of secondary brainstem injury) is related to deficits in the perception of intonation.

B. Corollary Hypotheses

1. If the right hemisphere contributes to the perception and production of intonation then deficits in perception and production should correlate with scores on lateralized neuropsychological instruments, i.e., left Purdue Pegboard and auditory dichotic listening, left ear scores.

2. If production of intonation involves frontal cortical-striatal circuitry, then production scores should correlate with tests of anterior functioning such as the Purdue Pegboard.
3. If perception of intonation involves the central auditory pathways then perception scores should correlate with tests which reflect the integrity of those pathways such as auditory dichotic listening tasks.
4. If social status post-trauma as assessed by the Vineland Social Maturity Scale is related to the perception or production of intonation then social status should also be related to the pathology contributing to expressive or receptive disturbance.
5. If severity of injury correlates with deficits in perception and production of intonation, then perception and production scores should correlate with the WAIS Performance I.Q.
6. Since the role of the GCS in long-term cognitive outcome is not significant, GCS is not expected to show a relationship to outcome on the perception or production of intonation.

DATA ANALYSIS

Tests of normality were carried out for all numeric data. Normality was assessed using the W statistic (Shapiro and Wilk, 1975). Results are presented in Table IV.

Differences between patients and normals for perception and production scores were examined using a t-test for the differnces between two means for samples of unequal size (Guilford and Fruchter, 1978).

Potential differences between patients and normals on tests of affective concept formation were examined in the following way: the prevalent answer ("Yes" or "No") for each test item was summed across normal controls and represented as a percentage. The percentage of patients who responded with that prevalent answer for normals was then calculated. A percentage score for patients and normals was thus obtained for each of the 48 items on the affective conceptualization task (12 items each for boredom, love, hope and surprise). In order to quantify the degree of similarity (or difference) within a concept betwen patients and normals, Spearman correlation coefficients were utilized and tested for significance. The underlying measure for each individual was in terms of a binomial response ("Yes", "No"). The percentage distributions described above reflect the underlying binomial sums but it may not be reasonable to assume the degree of dispersion of an interval measure. Therefore, an ordinal measure was utilized.

In the instance where all patients and normals agreed completely on all items per concept, a plot of the percentage score for normals by the percentage score of patients for the 12 items would yield a straight line and a correlation coefficient of unity. Any other value would indicate how close the actual responses fall to the case of perfect agreement ($r = 1$). If patients and normals do not differ with respect to concept formation, Spearman correlations for each of the four affective words should be statistically significant (i.e., represent statistically significant agreement in the definition of affective concepts).

To evaluate the relationship of social status post-trauma to deficits in the perception and production of intonation, Pearson correlations were carried out between the Social Quotient (of the Vineland Social Maturity Scale) and perception and production scores.

The association of specific pathological indices to perception and production deficits was examined in the following ways: patients were divided into groups on the basis of presence or absence of a particular pathological variable (i.e., brainstem compressed vs. brainstem normal; presence of right lesion on CT vs. no right lesion on CT, etc.). Means for perception and production scores for the various groups were calculated and t-tests for the differences between two means were computed. Where t-tests indicated signficiant differences in scores for different pathological groups, a relationship was presumed to exist between pathology and the specific performance tested. Correlation coefficients were computed to assess the strength of this presumed relationship. Pearson product-moment correlations were carried out with all continuous variables. Where "number of lesions" was utilized as a variable, Spearman rank-order correlations were computed. Compression of brainstem cisterns was at times represented as dichotomous data where

0 = no compression and 1 = compression. There is no reason to assume that in a large population the degree of compression would not be normally distributed. In this situation, use of the biserial r is appropriate (Guilford and Fruchter, 1978) and the bilateral r was utilized for those instances in the present study.

To assess the relationship of production and perception scores to tests of lateralized neuropsychological functions, Performance I.Q., and GCS scores, Pearson product-moment correlations were employed.

RESULTS

Patients are poorer than normals in identifying affectively intoned speech and in producing affectively intoned speech. Differences between patients and controls reach significance as seen in Table V. Furthermore, patients whose educational levels closely match the controls' do not differ from the remainder of the patients in perception or production of vocal intonation (Table V). This indicates that educational level is not contributing to the poorer scores in the two patient groups.

Zero order correlations indicate that neither age, education nor time from injury are correlated with perception or production scores (Table VI). Perception and production of affectively intoned speech, however, correlate with one another ($r = .55$, $p < .01$) in patients.

Affective concept formation does not differ between patients and normals in the present study. Table VII presents Spearman correlation coefficients between patient and control responses for surprise, boredom, love and hope. Correlations are significant for all four words, indicating significant agreement between patients and normals in the definitions of affective concepts.

Figures 1 through 4 visually represent the extent of agreement within and between patients and controls in describing higher-order affective concepts. Congruence between patients and controls differed by more than 20% in only 8 of the 48 total items (17%).

The Social Quotient, derived from the Vineland Social Maturity Scale was examined in relation to the perception and production of intonation. The correlation of the Social Quotient with perception of intonation is significant while the correlation with production of intonation is not (see Table XVI).

The distribution of lesions for the 22 patients whose CT data was available are listed below. The pattern of pathlogy is as follows: frontal lesions occur with the highest frequency (11/22 or 50%). Five out of twenty-two patients (23%) presented with bilateral frontal lesions. Seven out of twenty-two (32%) presented with a temporal or temporo-parietal lesion. Occipital lesions were rare (4%). Eight patients (36%) had compressed brainstem cisterns on CT scans. Eleven patients (50%) showed midline shift. Four patients had no CT lesion with marked neuropsychological deficit, indicating diffuse cerebral injury. These patterns of pathology are presented in Table VIII.

Tables IX and X represent lesion distributions for average versus defective scores on perception and production of intonation. Visual inspection of this data indicates that patients with borderline/defective perception scores have, as a group, a high incidence of compressed brainstem cisterns while those with borderline/defective production scores have, as a group, a high incidence of right frontal lesions and midline shift. Patients with no CT lesions (i.e., diffuse injuries) can obtain either defective perception or production scores.

Patients were divided into those with presence of right hemispheric lesion on CT and those without in order to observe the effect of a right lesion on the perception and production of affectively intoned speech. Fifteen patients had a documented right cerebral lesion. Eleven of these fifteen (73%) had lesions in the right frontal area. T-tests indicated that the presence of a right lesion on CT (n = 15) was associated with a significantly lower mean production of intonation score. This relation did not hold for perception of intonation (see Table XI). Zero order correlations confirmed this finding, the number of right lesions on CT correlated with production scores ($p < .01$), but not with perception scores. The number of left lesions did not correlate with either perception or production.

To further explore the relationship between production scores and lesions within the right hemisphere, patients were divided into those with defective/borderline production scores and those whose scores were within the average range (Table XII). T-tests indicated that the group with borderline to defective production scores had a significantly lower mean score on the Purdue Pegboard, left hand (but not on Purdue right). The groups did not significantly differ on Dichotic Listening scores for the left ear. Results indicate that the presence of a right lesion and furthermore the presence of functional impairment on a right hemispheric anterior task (Purdue Pegboard, left hand) are associated with production deficits.

In order to explore the patterns of pathology observed in Table IX and their relation to perception of intonation, patients were divided into those with normal brainstem cisterns and those with compressed cisterns and the means for these two groups were compared across various dimensions (Table XIII). The two groups of patients did not differ in age or educational level. Patients with compressed brainstem cisterns had significantly lower perception scores than those whose cisterns were normal. This relation did not hold for production scores, which were not different between groups. Patients with compressed cisterns also performed more poorly on auditory dichotic listening in the left ear and on a test of fine motor coordination in the left hand. There is no such relation with right ear and right hand tasks on these tests. WAIS Performance I.Q. was significantly lower in the group of patients with compressed cisterns. WAIS Verbal I.Q. did not differ between the two groups of patients. Finally, patients with compressed cisterns have, as a group, greater midline shift than those with normal cisterns. Significant correlations confirmed the relations between brainstem compression and Dichotic Listening Left Ear, Purdue Pegboard Left Hand, Performance I.Q., shift size and perception of intonation.

The eight patients with compressed brainstem cisterns were further subdivided into those with cisterns absent (n = 3) and those with cisterns compressed (n = 5). On perception of vocal intonation, patients with cisterns absent (greatest pressure, worst clinical status) perform most poorly, $\bar{X} = 25.66 \pm 4.93$, those with cisterns compressed perform better, $\bar{X} = 32.20 \pm 9.67$, and those with normal cisterns perform best, $\bar{X} = 39.28 \pm 10.47$. See Tables XIV and XV. (The perception means between cisterns absent and compressed do not reach significance, however, mean perception scores for both cisterns compressed and cisterns absent differ significantly from the mean perception score in the normal cistern group.) The pattern observed for perception of intonation is closely followed by performance on dichotic listening in the left ear: cisterns absent group = worst scores, cisterns normal = best scores. Performance I.Q., Purdue Pegboard Left Hand and shift size do not, however, follow this pattern. Pearson correlations of brainstem status (absent, compressed, normal) with both perception and dichotic listening left ear were statistically signficiant (Table XV). Results indicate an interrelationship between brainstem integrity, right hemispheric auditory projections and the perception of intonation.

The Social Quotient derived from the Vineland Social Maturity Scale was examined in relation to brainstem compression and presence of right frontal pathology. This analysis was carried out it order to explore the question of which patients may be at greater risk for social-occupational disability.

T-tests (Table XVI) indicate that the mean Social Quotient score is significantly lower in patients whose brainstem cisterns were compressed or absent. (The correlation of the Social Quotient with perception of intonation is also significant as mentioned earlier.) The mean Social Quotient scores for patients who present with right frontal lesions on CT do not differ signficantly from those who do. (The correlation of the Social Quotient with production of intonation is also not significant as mentioned before). Correlations confirmed the patterns reported above, i.e., correlations of the Social Quotient with brainstem compression and with number of right lesions on CT were significant, while the correlation with number of left lesions was not (Table XVII). Results suggest that patients who have brainstem compression and associated perceptual intonational difficulties as part of their cluster of sequelae are more likely to have difficulty in social and occupational adjustment post-trauma.

Perception and production scores were correlated with Performance I.Q. to determine whether deficits are related to severity of injury.

Perception scores show a correlation of borderline significance with PIQ ($r = .36$, $p < .06$). In general, intonational difficulties are weakly related to severity of injury.

The role of lowest GCS in predicting outcome in perception and production of intonation was also examined. Table XVIII presents the mean GCS scores for patients divided into four categories: 1) borderline/defective Perception scores, 2) average/low average Perception scores, 3) borderline/defective Production scores, 4) average/low average Production scores. The means and standard deviations of GCS scores for all four groups are remarkably similar indicating that classification of patients based on GCS score is not sufficient to differentiate performance on the Production and Perception tests. T-tests between the groups show no statistically significant differences.

DISCUSSION

The results of the present study indicate that both perception and production of intonation may be impaired in moderately and severely head injured patients while conceptual identification of affect remains intact for the group as a whole. This indicates that the observed disorders of prosody are of a sensory and motor nature. Ross and Mesulam (1979) have reported similar findings. Heilman and Valenstein (1979) report that patients with right hemisphere damage do not necessarily lose the concept of what different emotions mean, citing a study where patients could not identify the same words presented with different tonal contours as "same" or "different" yet were able to clearly identify the intended emotions expressed in stories read without affective intonation. However, one would expect to see difficulties in concept formation in moderately and severely head-injured patients by virtue of the extent of impairment to the frontal lobes, mentioned earlier. An alternative explanation for the lack of observed differences between patients and normals may be that the present test served as an affective vocabulary test that tapped overlearned definitions of affective concepts. This may have rendered it less vulnerable to the effects of head trauma. The observed deficits in perception and production of intonation can be persistent sequelae and were, in some patients, observed as late as one or two years post-trauma. The fact that the perception and production of affectively intoned speech do not correlate with educational level implies that these tasks require a special integrative skill that is not readily available to the head trauma patient.

In the present sample of patients, production of intonation was related to the presence of right hemispheric lesions. Functionally, patients whose production scores fall in the borderline to defective range had significantly lower Purdue Pegboard Left Hand scores than those with normal production, implicating right frontal area involvement or interruption of fiber pathways to that area. Perception of intonation was related to brainstem compression. Compression of brainstem cisterns also coincided closely with performance on a dichotic listening task for letters in the left ear. This finding suggests that auditory pathways leading to the right hemisphere were interfered with in the brainstem group. Both the perception and production of intonation could be related in these patients to right hemispheric functioning and support the literature on prosodic deficits in CVA and tumor patients. In addition, dichotic listening studies in normals have suggested that the intact right hemisphere participates in the perception of prosody. A left ear advantage was reported for the perception of intonation in sentences filtered of linguistic content (Blumstein and Cooper, 1974). In this task, subjects were required to discriminate a statement from a question by intonational pattern. In dichotic studies where subjects were required to remember the grammatic structure of a sentence (when grammatic elements were indicated by prosody) a right ear advantage was reported (Zurif and Mendelsohn, 1972; Zurif and Sait, 1970). This latter task may be more "linguistic" thus involving the left hemisphere.

Deficits in the perception and production of prosodic features, both propositional and emotional, have been identified not only in stroke and tumor patients but in several other distinct populations. Regardless of etiology and features specific to these various syndromes there is a common denominator of social awkwardness, problems in relating to others, and of lateralizing neurologic or neuropsychologic signs.

The pattern of abnormal behavior termed Asperger's syndrome (Wing, 1981) was first described in 1944. The condition becomes noticeable around the third year of life or later and is more common in males than females. The onset of walking is generally delayed in these children. In early development, there is a lack of the urge to communicate in babble, gesture, movement, smiling or laughter that is characteristic of the normal baby or toddler. Later language may be characterized by a difficulty in using pronouns. Stereotyped speech or the invention of words may be noted, the overall impression being that of a peculiar speech with abnormal content. Subtle verbal jokes may not be understood. Vocal intonation is usually monotnous or exaggerated. Gestures may be limited or clumsy and inappropriate to the accompanying speech. Comprehension of other people's expressions is poor and non-verbal signs may be ignored or misinterpreted. Social interaction is generally impaired since the person with Asperger's syndrome does not have the intuitive knowledge of how to adapt his approaches or responses to the signals given by others (the complex, constantly changing signs of voice, gesture, posture, movement, eye contact, proximity to others, that give informational cues). Asperger's syndrome tends to occur in fathers of those with the disorder and is believed to be genetically transmitted. It may also be found in children or adults with a history of pre-, peri-, or post-natal conditions (such as anoxia) which may have caused cerebral damage. Neuropsychological assessments have revealed that visuo-spatial abilities are impaired relative to expressive speech.

A similar, and possibly overlapping, syndrome is that of developmental learning disabilities described by Weintraub and Mesulam (1983). Persons who fall into this behavioral pattern are again characterized by shyness, social awkwardness, interpersonal difficulties, visuo-spatial disturbances and inadequate paralinguistic communicative abilities. These characteristics occurred together with neurologic evidence of right hemispheric dysfunction. Patients described by Weintraub and Mesulam were referred for evaluation due to either academic failure at the college level, inability to find employment, or behavioral disturbances. Many complained of chronic depression or extreme shyness. Most patients avoided eye contact and had awkward gestures accompanying speech. Speech prosody was described as "flat" or "atypical" and all patients examined (n=14) had difficulties with repetition, elicited production and perception of both affective and non-affective prosody. This behavioral syndrome begins early in life and most patients reported lifelong emotional and interpersonal problems. Furthermore, as with Asperger's syndrome, a prevalence of these phenomena was seen in the families of patients. The etiologies of the disorder in these patients consisted of either infantile hemiplegia affecting the left side of the body, perinatal stress, siezure disorder of early onset, or genetic predisposition due to family history. Neurologic findings in these patients generally indicated some form of motor abnormality on the left side of the body.

For example, on timed motor tasks, movements with the left hand were slower than with the right. Neuropsychologic assessments indicated that patients had higher Verbal than Performance I.Q.'s on the Wechsler Adult Intelligence Scale. Subtests of scores indicated a greater facility with verbal tasks such as Vocabulary and difficulty with Block Design and Oject Assembly. Verbal memory was almost always superior to non-verbal memory, which was generally moderately to severely impaired. The case reports in Weintraub and Mesulam's paper (1983) suggest that the cluster of abilities consisting of visuo-spatial skills, modulation of affect and paralinguistic communication, when dysfunctional, produces a real barrier to communicative competence and social adaptation. Bryan (1977) has studied learning disabled children and found that these children are less accurate than their normal peers in the perception of non- verbal communication as transmitted by tone of voice and body posture. He suggests that although the inadequate social adjustment of the learning disabled child is generally believed to be due to academic failure and subsequent lowered self-confidence, a significant contributing factor may be an insensitivity to social cues. Misinterpretations affect a child's subsequent response which in turn affects how a learning disabled child is perceived by peers and teachers. Head trauma patients have embedded within their post-traumatic sequelae the symptoms observed in learning disabilities suggestive of right hemispheric dysfunction, and right hemispheric stroke and tumor patients.

Patients with right hemispheric dysfunction, particularly those with developmental learning disabilities, are characterized by social disability. The present data indicates that those patients who have difficulty with perception of intonation and had brainstem compression have a poorer socio-occupational status post-trauma. It is important to note that brainstem compression is associated with greater overall brain pathology in head trauma patients. It correlates significantly with shift size and with the WAIS Performance I.Q. both of which reflect the severity of pathological changes subsequent to trauma. Performance I.Q. is dependent on the integration of a wide range of cerebral activities and is affected by such factors as motivation, attention, speed of performance and the ability to organize complex tasks over time (Jennett and Teasdale, 1981). Thus, Performance I.Q. in a head trauma patient is not a lateralizing test but a test of overall cerebral efficiency. Poorer socio-occupational status in these patients may be due to the fact that they have sustained a more serious injury. In spite of the association of brainstem compression with overall pathology, there is enough evidence to link brainstem compression to right hemispheric functions such as the dichotic left ear task and the Purdue left hand task. Table X indicated that patients with compressed cisterns had, as a group, significantly lower Purdue left hand and dichotic left ear scores than patients with normal cisterns.

One can view defective perception of intonation as only one symptom embedded within many in patients whose injuries are severe enough to cause brain swelling that compresses the brainstem. In such cases, most cerebral areas are affected. Compression of the brainstem can easily produce secondary ischemic damage. Tomlinson (1970) reports that downward displacement of the brainstem produces stretching and consequently haemorrhages that are midline, usually in the upper pons and throughout the midbrain and pontine tegmentum. Ischemia may subsequently occur. Axonal damage is frequently seen, particularly in the mid and rostral pons and the midbrain. Downward displacement produces marked elongation, distortion, and stretching of the midbrain and often of the thalamus and hypothalamus (Tomlinson, 1970). Therefore, fibers in the brainstem and hypothalamic areas (areas invloved in arousal and visceral, affective responses to the environment), are easily disrupted with brainstem compression. Taylor (1967) has indicated that damage to cranial nerve nuclei is common when the head is flexed during injury. In this case, conduction in the auditory nerve may be impaired. Furthermore, the cochlea possesses an extensive network of blood vesels that supply its oxygen requirement and the cochlear artery is derived from a branch of the basilar artery (Dirks, 1978). With brainstem compression and stenosis of the basilar artery, the cochlear blood supply may be reduced having an effect on auditory processing. Compression and displacement often include thalamic fibers as mentioned above. In such a case, auditory processing in the medial geniculate body may be disrupted. In summary, there is ample evidence that brainstem compression interrupts the functioning of fiber systems within the central auditory system and fiber systems critically involved in arousal and affective response.

Interruption of acoustic pathways due to brainstem compression however does not explain the lateralizing pattern observed, i.e., the selective relation of compressed brainstem cisterns to Purdue left hand and dichotic left ear tasks. An additional set of assumptions are needed to explain this phenomenon. One explanation may be that right cortical regions have particularly rich inter-connections to subcortical regions. This notion first emerged intuitively from knowledge of the participation of subcortical limbic pathways in emotion and subsequent research linking the cognitive functions of the right hemisphere with affective behavior. Recent studies have further specified this cortical-subcortical relationship. Robinson (1979) has demonstrated that right but not left hemispheric lesions produce hyperactive emotionality and alter brain levels of catecholamine neurotransmitters. Right hemispheric infarctions in rats led to significant depletions of norepinephrine in the lesioned cortex and brainstem (locus coeruleus) and significant depletions of dopamine in the substantia nigra. Left lesions had no effect on any brain region studied. Robinson concludes that an underlying anatomical or physiological asymmetry exists in the brain and that norepinephrine and dopamine-containing pathways are different on the two sides of the brain. Oke, Keller, Medford and Adams (1978) have shown that the distribution of norepinephrine in human thalamus is lateralized. The somatosensory input area of the right thalamus (VPL-VPM) has a higher concentration of this catecholamine. Association of the right hemisphere with autonomic processes have also been made. Davidson (1978) has reported that left-hand finger tapping can more accurately parallel heart beats than right-hand tapping.

The lateralization of norepinephrine systems is carried a step further by hypotheses that norepinephrine-mediated arousal may be particularly influential on the right hemisphere (Tucker, 1981). ECT therapy for depression is believed to increase brain norepinephrine as well as facilitate right hemisphere activation (Tucker, 1981, Kronfol et al., 1978). Norepinephrine-mediated arousal from tricyclic antidepressant medications was associated with a specific effect on the right hemisphere. Depressed children evaluated neuropsychologically before and after tricyclic treatment showed improvement in right but not left hemisphere cognitive performance (Brumback, Staton and Wilson, 1980). There indeed appears to be a special relationship between right hemispheric functioning and the distribution and supply of the noradrenergic system.

The importance of norepinehrine for right hemsipheric processing and the lateralization of the norepinephrine system allow for the development of a model of affective processing of information which involves the brainstem. There is evidence that axons from the <u>same</u> adrenergic neurons in the brainstem may be distributed to cerebral, hippocampal and cerebellar cortices as well as to the hypothalamic region (Kety, 1970). This pattern of distribution is of interest because a state of arousal by means of simultaneous adrenergic input to each could serve to reinforce, consolidate, or identify significant sensory patterns, their affective associations and the motor programs necessary in carrying out an appropriate response to the stimulus. This antatomical/neurochemical model can be applied to the perception of intonation. Noradrenergic activation in response to an affectively intoned sentence will simultaneously involve:

a) the hippocampus - where presumably the memory for the particular intonational contour and its affective associations were activated,

b) the hypothalamus - where the visceral reaction to the stimulus is processed,

c) the cerebellum - where any motor program required for responding to the stimulus is activated, and

d) the cerebral cortex (presumably the right cerebral cortex) - where the fusion of all these sensory and motor subcortical components takes place via syncretic conceptualization, i.e., visceral affective information is fused with cognitive information into a construct; complex nonverbal configurations are interpreted and their affective meaning is derived and recognized (Tucker, 1981).

Figure V illustrates schematically this noradranergic activation in response to an affective auditory stimulus. The significance of brainstem pathways is clearly illustrated in such a model. The importance of the brainstem is twofold:

1) in the sensory reception and processing of an auditory simulus

2) in the proper functioning of brainstem noradrenergic neurons so that disruption of this intricate arousal system does not take place.

This model, furthermore, makes the role of the brainstem inseparable from that of right hemisphere syncretic conceptualization. If one accepts this model, brainstem compression in a head trauma population interrupts right hemispheric processing at a <u>subcortical</u> level.

David Baer (1983) has developed a model of right intrahemispheric localization of function with affective processing. He has isolated two systems which are functionally and anatomically distinct: a dorsal emotional surveillance system and a ventral system of decoding specific emotional signals within a given modality. The ventral system involves the connections of the amygdala, hippocampus, hypothalamus and orbito-frontal cortex. Its distinctive feature is the retention of independent connections between individual cortical sensory systems (e.g., visual, auditory) and the limbic system. The decoding of emotional cues within a sensory modality such as vocal intonation or facial expression involves this sytem. The affective and autonomic response to such signals also occurs within this system. Baer states that the amygdala and hippocampus are essential for affective memory. This allows for the appropriate decoding (recognition) of an affective stimulus.

Baer's ventral system is similar to Nauta's (1971) anatomical and functional model of orbito-frontal connections to the limbic system. Nauta describes the frontal lobe as association cortex which integrates information from both the organism's internal environment and the external environment. The frontal lobe is a common end-point for neuronal systems that extend from the primary sensory regions of the cortex (auditory, visual, somatic) via various thalamic intermediaries. All three major sensoria represented by modality specific areas in neocortex find some form of re-representation in the frontal cortex. At the same time, the orbito-frontal area has substantial interconnections with the diencephalon (hypothalamus) and mesencephalic tegmentum as well as the amygdala and other limbic structures. There is reason to believe that afferent fiber systems to the orbito frontal areas convey information concerning the organism's internal milieu (visceral, emotional, motivational state) as mediated by telencephalic limbic structures. Given these anatomical and functional correlations, the frontal lobe (especially the orbito frontal area) can be viewed as a complex integrator of external sensory cognitive information and the organism's internal visceral, emotional state in response to that information. In a normally functioning individual, internal and external information are constantly integrated, i.e. internal visceral reactions are modulated by external sensory/cognitive information and vice versa so that behavior is appropriate to a given situation.

Baer's (1983) right-hemispheric dorsal system is described as an emotional orientation system. Anatomically, this system involves the interconnections of the inferior parietal lobe, cingulate gyrus and dorsolateral pre-frontal cortex as well as efferent pathways from the inferior parietal lobe to reticular activating nuclei and locus coeruleus. Functionally, it is involved in surveillance, orientation and emotional arousal. Malfunctioning of this system can lead to sensory inattention and emotional unconcern. The patient might not detect a threatening situation, for example, and cannot sustain attention, become appropriately emotionally concerned or initiate an adaptive response.

Both the dorsal and ventral right hemispheric systems have strong connections to brainstem areas. A patient who has sustained secondary ischemic damage due to brainstem compression could be handicapped in the affective sphere because he cannot correctly identify stimuli or integrate affective stimuli with the appropriate responses (ventral system) or because he is not properly orienting to affective stimuli (dorsal system). In the present sample of patients, the Social Quotient from the Vineland Social Maturity Scale was significantly lower in the brainstem group than in those with no brainstem compression. Similarly, the Social Quotient correlated significantly with a patient's ability to correctly identify vocal intonation and brainstem compression also correlated with the perception of intonation. These results are consonant with the theories discussed above. They support the hypothesized role of the right hemisphere in appropriate and adaptive social functioning. They also are congruous with the hypothesis of strong lateralized connections of brainstem areas to the right hemisphere.

The production of intonation, in the present sample, was significantly lower in patients with documented right lesions on CT scan than in those without right lesions. Seventy-three percent of the documented right lesions were frontal. Functionally, patients whose production scores were in the borderline to defection range had significantly lower scores on the Purdue Pegboard, left hand than patients with average production scores. The Purdue Pegboard has been found to be a useful and valid instrument for lateralization of lesion (Costa, et al, 1963). In addition, this lateralizing effect did not emerge on a task of functioning primarily in the temporal lobe (dichotic listening). The observed pattern suggests that production of intonation may be dependent on proper functioning within the anterior regions of the right hemisphere. This patterns supports Ross' (1979, 1981) work and the reports of Tucker, Watson and Heilman (1977) where CVA patients with evocative deficits showed CT lesions in the distribution of the right middle cerebral artery, in posterior frontal and parietal supra-Sylvian regions.

Production of intonation, in this sample, was not related to brainstem compression. This suggests that evocative speech may be a process which is semi-independent of perception. In cases of adult acute onset of disability, such as trauma, the motor patterns accompanying affective speech have already been learned. Production may no longer require the complex acoustic analysis that must be required during development when such responses are first being learned. Production of intonation probably does require an intact corpus callosum. This is suggested by studies of aprosodic schizophrenics in whom transcallosal communication was shown to be impaired (Rosenthal and Bigalow, 1972; Dimond, et al., 1979; Gur, 1978). Production of prosody also requires adequate participation of subcortical structures such as the basal ganglia. Ross and Mesulam (1979) report that an aprosodic patient with right basal ganglia involvement did not recover intonational speech while a patient with a purely neocortical parietal lesion recovered completely. Furthermore, aprosody is a common feature of Parkinson's disease and this indicates that the striatum is involved in speech intonation (Scott and Caird, 1983). No direct measure of degree of basal ganglia disruption exists for the present sample. However inferences can be made from our knowledge about the pathology of head trauma. Oppenheimer (1968) studied lesions in 59 cases of head injury with clinical features ranging from mild concussion to decerebration and with survival times from 12 hours upwards. He found microscopic lesions not apparent to the naked eye. Lesion distributions tended to be in the corpus callosum (on one side of the midline), in the caudate nucleus, and in the upper brainstem (particularly tegmentum).

These lesions were infrequent in the cerebral cortex. The present sample of cases may have such basal ganglia and callosal lesions to varying degress. These may contribute in part to difficulties in production of intonation.

Production of intonation does not correlate with the Social Quotient. This indicates that patients with production deficits alone may be in a better position socially than those in whom brainstem compression disrupts emotional perception, attention and arousal.

Production of intonation does correlate significantly with perception of intonation. This correlation may reflect the fact that both functions are related to the extent of right hemispheric pathology. An alternative point is supplied by the motor theory of speech perception. This theory postulates that in order to perceive speech, the brain must determine how it would produce the same sounds. The concept arose of out research on similarities between sounds (i.e., consonants) perceived as identical by the brain. Sounds that were perceived as alike were produced in the same way (Liberman et al., 1967). What remained invariant was the way the throat, mouth, lips and tongue were controlled in the production of sound. Perhaps this relation between production and perception carries over into intonation.

Brainstem compression and the number of right lesions correlate significantly with the Social Quotient. Number of left lesions does not. If one accepts the hypotheses postulating an especially strong connection between subcortical pathways and the right hemisphere, one can regard correlations of brainstem compression and number of right lesions with the Social Quotient as indicative of a relation between right hemispheric integrity and social skills. Differences between the hemispheres in social and non-social information processing have already been reported. For example, patients with documented right frontal damage were depressed on tasks of social cognition but intact on non-social cognitive tasks. The reverse was true for left frontal patients (Sorman, Wasserstein and Zapulla, 1983). Social cognition involved tasks that required the patient to take on another person's perspective in a series of events without imposing their own motives (Chandler's Cartoons, 1973, Leehy's Pro-Social Behavior Task, 1979). The results of the present study suggest that a patient with right hemispheric pathology and brainstem pathology may be at a social disadvantage post-traumatically.

The fact that head trauma patients have difficulties in the perception and production of intonation should lead to systematic studies of affective difficulties within other modalities (such as visual). The pilot data from the present study already indicates that head trauma patients have difficulty in discriminating facial affective expression and do not utilize visual affective cues. Systematic examination of the multiple emotional functions of the right hemisphere, from emotional surveillance to affective identification and communication, is needed in head trauma patients in order that the prevalence and extent of their social handicap be identified. The prevalence of brainstem compression in severely injured patients places them at a greater risk for the interruption of critical affective pathways. This situation must affect functions beyond the perception of intonation, and these need to be identified. A better understanding of the aspects of affective behavior that are disrupted post-trauma would help explain at least some of the symptoms attributed vaguely to "personality" change following injury. Furthermore, if one considers the extent of non-affective cognitive deficit retained by the average severely injured patient, one begins to realize how devastating a combination of cognitive and affective sequelae must be. The importance of adequate social perception has, perhaps, most clearly been recognized within the realm of learning disabilities. Both parents and professionals have reported that social misperception appears to be the most debilitating deficit of all (Bader, 1975). The children with these problems are described as excessively dependent, insensitive to social situations, poor in judging people's emotions and attitudes and doing and saying inappropriate things (Lerner, 1976). Furthermore, parents and teachers who have dealt with these children have recognized that the problem pervades all social interactions and continues to create problems throughout adulthood (Minskoff, 1980). Social perception skills, in fact, become increasingly important in adulthood and have been identified as key factors in vocational and post-educational success (Edmonson, DeJung, and Leland, 1965). Finally, the learning disabled who have difficulties in circumscribed areas such as reading or math can find escape from these disabilities in a number of situations. It is, by contrast, almost impossible to find escape from difficulty in social perception (Minskoff, 1980). These statements can be readily applied to many head-injured patients.

The natural consequence of the identification of perceptual and evocative affective prosodic deficits in head trauma patients is the development of training techniques. Ideally these should help patients to emote verbally and to learn to recognize affective cues in speech (e.g., pitch, tempo, loudness, etc.). To the extent that patients have difficulty perceiving and producing affective cues in other modalities (visual, gestural), these may also be trained. Minskoff (1980) describes a training procedure developed for persons with social perception deficits. The training involves teaching students to discriminate critical visual and auditory social cues in the behavior of others and in themselves. Once the critical cue is discriminated, a verbal description of specific situations in which the cue occurs is presented to develop an understanding of the meaning of these cues. The next step involves teaching students to express specific motor and oral cues so that these are built into the students' response repertoires. The final stage involves training students to discriminate negative, non-verbal social cues in people with whom they interact, to relate the negative cues to specific responses they might have made and to then modify these inappropriate responses in future situations of a similar kind. The various techniques employed in this method involve role playing, verbal discussions and explanations and training in selective attention. Such a model can be easily applied to a head trauma population in a rehabilitative effort or to other neurological populations with similar disabilities.

The present study has identified deficits within the perception and production of affective prosody in head trauma patients. These findings suggest the need for quantifying difficulties in other aspects of perceptual and motor affective functions. An awareness of these deficits is important because they can have devastating consequences which are at least equal to those caused by deficits in language, memory, motor impairment and other already identified post-traumatic sequelae. Hopefully, the identification of difficulties in the area of affective processing of information and production will allow for the development of an affective therapy that will parallel other forms of cognitive therapy.

Table I

T-Tests and Demographic Characteristics of Patients and Non-Injured Controls

	Patients (N = 27)	Controls (N = 10)	t value
Age	$\bar{X}$ = 31.1 SD = 9.9	$\bar{X}$ = 29.3 SD = 5.6	.679 NS
Education (Years)	$\bar{X}$ = 11.5 SD = 1.8	$\bar{X}$ = 13.8 SD = 1.7	-3.5 $p < .0001$
Sex			
Male	78%	60%	
Female	22%	40%	
Ethnicity			
Black	33%	30%	
Hispanic	26%	30%	
White	41%	40%	
Patient GCS Scores	$\bar{X}$ = 9.40 SD = 3.75 Moderate = 56% Severe = 44%		

Table II

Neuropsychologic Tests (Patients)
N = 27

	$\bar{X}$	SD	Range
WAIS			
PIQ	87.7	14.6	64 - 126
VIQ	93.0	13.3	69 - 135
Purdue R	10.82	2.74	6 - 16
Purdue L	11.67	2.05	6 - 14.5

Table III

Social Maturity (Patients)

	$\bar{X}$	SD
Social Age (N = 27)	16.3	4.5
Social Quotient (N = 27) (105 = 50th percentile, adult score for life ages > 25 years)	57.7	29.8
Chronological Age (N = 27)	31.1	9.9

Table IV

Results of Tests on Normality of Distributions

	Patients	Controls
Perception	Normal	Normal
Production	Normal	Normal
Age	Normal	Normal
Education	Not Normal	Not Normal
VIQ	Normal	------
PIQ	Normal	------
Purdue R	Normal	------
Purdue L	Normal	------
Social Maturity	Not Normal	------
Time From Injury	Not Normal	------
Number Right Lesions	Not Normal	------
Number Left Lesions	Not Normal	------
Shift Size	Not Normal	------
Size of Largest Right Lesion	Not Normal	------
Size of Largest Left Lesion	Not Normal	------

Table V

Means and Standard Deviations of Affective Prosody Tests

	Patients (N = 27)	Controls (N = 10)	t value
Perception	$\bar{X}$ = 37.3 SD = 10.2	$\bar{X}$ = 45.3 SD = 7.8	-2.56 ($p < .01$)
Production	$\bar{X}$ = 12.5 SD = 3.4	$\bar{X}$ = 14.9 SD = 2.9	-2.16 ($p < .04$)

Comparison of Patients and Normals Matched for Educational Level

	Normals (N = 10)	Patients Matched for Education (N = 10)	Patients (Remaining) (N = 17)
Perception	$\bar{X}$ = 45.3 SD = 7.8	$\bar{X}$ = 37.2 SD = 10.5	X = 37.3 SD = 10.3
Production	$\bar{X}$ = 14.9 SD = 2.9	$\bar{X}$ = 12.1 SD = 4.2	X = 12.7 SD = 3.0

Table VI

Zero Order Correlations Between Neuropscyh Outcome, Tests of Affective Perception, Production and Age, Education, Time From Injury (N = 27) (r)

	Perception	Production	PIQ	VIQ
Age	-.20	-.07	.06	.19
Education	.26	.26	.42 ($p < .03$)	.65 ($p < .0002$)
Time From Injury	.17	-.03	.08	-.11

Perception and Production: $r = .55$ $p < .01$

Table VII

Conceptual Identification of Emotion

Intercorrelations on All Questionnaire Items
Spearman Correlation Coefficients N = 12

		Normals			
		Surprise	Boredom	Love	Hope
Patients	Surprise	0.60291*	0.28446	0.08464	0.24962
		0.0380**	0.3702	0.7937	0.4340
	Boredom	0.35496	0.67569	0.20617	0.28419
		0.2575	0.0159	0.5203	0.3707
	Love	-0.22068	-0.28118	0.60699	0.03599
		0.4907	0.3760	0.0364	0.9116
	Hope	-0.34769	-0.23836	-0.17944	0.77373
		0.2681	0.4556	0.5768	0.0031

*r values
**p values

Table VIII

Patient Pathology

Patient	*R Extra Paren-chymal	L Extra Paren-chymal	R Fron-tal	L Fron-tal	R Temporo-Parietal	L Temporo-Parietal	R Occip-ital	L Occip-ital	**Brain Stem	Shift Size (mm)
1	13.5	0	0	0	0	0	0	0	N	4
2	0	0	0	0	0	0	0	0	C	0
3	15.0	0	0	0	0	0	0	0	N	0
4	3.0	0	0	0	0	0	0	0	N	0
5	0	0	0	0	0	0	0	0	N	0
6	0	0	30.0	0	0	0	0	0	C	24
7	0	0	9.0	0	105.0	75.0	0	0	C	15
8	0	0	18.0	12.0	0	0	0	0	N	0
9	0	0	60.0	60.0	45.0	0	0	0	C	0
10	0	0	6.0	30.0	0	111.0	0	0	C	9
11	0	0	45.0	45.0	0	0	0	0	N	6
12	0	0	45.0	30.0	0	0	0	0	C	3
13	0	0	30.0	0	60.0	0	0	0	N	2
14	0	0	0	0	0	0	0	0	N	0
15	0	0	34.0	0	0	0	0	0	N	0
16	30.0	0	0	0	0	0	0	0	C	9
17	0	0	0	0	0	0	0	0	N	0
18	0	0	0	45.0	60.0	60.0	0	0	N	0
19	0	0	0	0	0	60.0	0	0	N	3
20	0	0	21.0	0	0	0	0	0	N	0
21	0	12.0	0	0	0	9.0	0	0	N	3
22	0	0	0	0	0	0	0	24	C	2

*Lesions in mm

**Brain Stem: C=Compressed, N=Normal

Table IX

Lesion Distribution - Perception

Patient	R *Extra	L Extra	R Frontal	L Frontal	R T-P	L T-P	B Stem Compres-sion	Mid-line Shift
Perception: Borderline/Defective (N = 8)								
1	+	-	-	-	-	-	-	-
2	-	-	-	-	-	-	-	-
3	-	-	+	-	-	-	+	+
4	-	-	+	-	+	+	+	+
5	-	-	+	+	+	-	+	-
6	-	-	+	+	-	-	+	+
7	+	-	-	-	-	-	+	+
8	-	-	-	-	-	-	+	+
	25%	0%	50%	25%	25%	13%	75%	63%
Perception: Average/Low Average (N = 14)								
9	+	-	-	-	-	-	-	+
10	-	-	-	-	-	-	+	-
11	+	-	-	-	-	-	-	-
12	-	-	+	+	-	-	-	-
13	-	-	+	+	-	+	+	+
14	-	-	+	+	-	-	-	+
15	-	-	+	-	+	-	-	+
16	-	-	-	-	-	-	-	-
17	-	-	+	-	-	-	-	-
18	-	-	-	-	-	-	-	-
19	-	-	-	+	+	+	-	-
20	-	-	-	-	-	+	-	+
21	-	-	+	-	-	-	-	-
22	-	+	-	-	-	+	-	+
	14%	7%	43%	23%	14%	29%	14%	43%

*Extra = Extraparenchymal Lesion

Table X

Lesion Distribution - Production

Production: Borderline/Defective (N = 8)

Patient	R *Extra	L Extra	R Frontal	L Frontal	R T-P	L T-P	B Stem Compres-sion	Mid-line Shift
1	+	-	-	-	-	-	-	+
2	+	-	-	-	-	-	-	-
3	-	-	-	-	-	-	-	-
4	-	-	+	-	-	-	+	+
5	-	-	+	+	+	-	+	-
6	-	-	+	+	-	+	+	+
7	-	-	+	+	-	-	-	+
8	-	-	+	-	+	-	-	+
	25%	0%	63%	35%	25%	13%	38%	63%

Production: Average/Low Average (N = 14)

Patient	R *Extra	L Extra	R Frontal	L Frontal	R T-P	L T-P	B Stem Compres-sion	Mid-line Shift
9	-	-	-	-	-	-	+	-
10	+	-	-	-	-	-	-	-
11	-	-	+	-	+	+	+	+
12	-	-	+	+	-	-	-	-
13	-	-	+	+	-	-	+	+
14	-	-	-	-	-	-	-	-
15	-	-	+	-	-	-	-	-
16	+	-	-	-	-	-	+	+
17	-	-	-	-	-	-	-	-
18	-	-	-	+	+	+	-	-
19	-	-	-	-	-	+	-	+
20	-	-	+	-	-	-	-	-
21	-	+	-	-	-	+	-	+
22	-	-	-	-	-	-	+	+
	14%	7%	36%	21%	14%	29%	36%	43%

*Extra = Extraparenchymal Lesion

Table XI

Means and Standard Deviations of Production and Perception Scores in Patients with Documented Right Lesions on CT Versus Patients Without Documented Right Lesions

	No Right Lesions on CT (N = 7)	(11 or 73% = Frontal) Presence of Right Lesion on CT (N = 15)	
Production Scores	$\bar{X}$ = 14.55 SD = 2.93	$\bar{X}$ = 11.10 SD = 3.20	t = 2.41 $p < .05$
Perception Scores	$\bar{X}$ = 38.28 SD = 12.24	$\bar{X}$ = 34.67 SD = 10.10	NS

Zero Order Correlations:

Production With Number of Right Lesions on CT: r = -.49, $p < .01$
Perception With Number of Right Lesions on CT: r = -.29, NS

Table XII

Means and Standard Deviations of Neuropsychologic Functions in Patients with Defective versus Average Production Scores

Patients N = 27

	Production Scores Borderline/Defective (N = 8)	Production Scores Average/Low Average (N - 19)	
Purdue Left	$\bar{X}$ = 10.5 SD = 2.20	$\bar{X}$ = 12.35 SD = 2.09	t = 2.06 $p < .05$
Purdue Right	$\bar{X}$ = 9.81 SD = 2.50	$\bar{X}$ = 11.19 SD = 2.67	NS
Dichotic Left Ear	$\bar{X}$ = 26.00 SD = 4.84	$\bar{X}$ = 22.42 SD = 10.34	NS

Table XIII

Means and Standard Deviations of Demographic, Pathologic, and Neuropsychologic Variables in Patients with Compressed versus Normal Brainstem Cisterns

Patients N = 22

	B Stem Cisterns Normal (N = 14)		B Stem Cisterns Compressed (N = 8)		t tests
	$\bar{X}$	S.D.	$\bar{X}$	S.D.	p value
Age	29.57	9.26	34.12	5.51	NS
Education	11.28	1.32	11.12	1.36	NS
PIQ	88.92	11.79	77.63	11.39	$p < .05$
VIQ	92.35	12.82	89.50	9.18	NS
Purdue R	10.96	3.00	10.18	1.99	NS
Purdue L	12.00	1.61	10.25	2.37	$p < .01$
Perception	39.28	10.47	29.75	8.48	$p < .05$
Production	12.86	3.86	11.19	2.62	NS
Shift Size	1.28	1.97	7.75	8.41	$p < .01$
Dichotic L	25.80	8.40	16.80	8.10	$p < .02$
Dichotic R	37.10	9.70	37.60	16.60	NS

Zero Order Correlations: (N = 22)

Brain Stem with Perception: $r = .55$, $p < .05$
Brain Stem with PIQ: $r = .55$, $p < .05$
Brain Stem with Shift Size: $r = -.46$, $p < .05$
Brain Stem with Dichotic L: $r = -.52$, $p < .02$
Brain Stem with Purdue L: $r = -.36$, NS

Table XIV

Patients (N = 8) Perception Scores

Brain Stem Cisterns Absent (N = 3)	Brain Stem Cisterns Compressed (N = 5)
$\bar{X}$ = 25.66	$\bar{X}$ = 32.20
SD = 4.93	SD = 9.67

t = 1.08 NS

Patients (N = 8) Production Scores

Brain Stem Cisterns Absent (N = 3)	Brain Stem Cisterns Compressed (N = 5)
$\bar{X}$ = 11.86	$\bar{X}$ = 11.87
SD = 4.13	SD = 2.61

NS

Zero Order Correlations: Biserial r's

Brain Stem with Perception: $r = .55$, $p < .05$

Brain Stem with Production: $r = .29$, NS

Table XV

Means and Standard Deviations of Neuropsychological and Pathological Indices in Patients with Absent versus Compressed versus Normal Cisterns

Brain Stem Cisterns	Absent (N=3)	Compressed (N=5)	Normal (N=14)
Perception	$\bar{X}$ = 25.66 SD = 4.93	$\bar{X}$ = 32.20 SD = 9.67	$\bar{X}$ = 39.28 SD = 10.47
Dichotic Left Ear	$\bar{X}$ = 12.6 SD = 9.86	$\bar{X}$ = 19.4 SD = 6.8	$\bar{X}$ = 25.80 SD = 8.40
PIQ	$\bar{X}$ = 77.66 SD = 13.05	$\bar{X}$ = 77.60 SD = 11.90	$\bar{X}$ = 88.92 SD = 11.79
Purdue Left	$\bar{X}$ = 10.5 SD = 1.0	$\bar{X}$ = 10.1 SD = 3.04	$\bar{X}$ = 12.00 SD = 1.61
Shift Size	$\bar{X}$ = 3.66 SD = 4.72	$\bar{X}$ = 10.20 SD = 9.62	$\bar{X}$ = 1.28 SD = 1.97

Correlations, Pearson:

Brain Stem with Perception: $r = -.48$, $p < .05$

Brain Stem with Production: $r = -.52$, $p < .02$

Table XVI

Mean Social Quotient Values in Patients with Compressed Cisterns, Normal Cisterns, Right Frontal Lesions and No Right Frontal Lesions

Brain Stem Compressed or Absent (N = 8)	Brain Stem Normal (N = 14)	t value
$\bar{X}$ = 40.25	$\bar{X}$ = 62.64	2.09
SD = 13.64	SD = 29.66	($p < .05$)

Correlation of Social Quotient with Perception of Intonation = .51 ($p < .01$)

Presence of (R) Frontal Lesion (N = 10)	No (R) Frontal Lesion (N = 12)	t value
$\bar{X}$ = 50.90	$\bar{X}$ = 57.50	.56
SD = 31.00	SD = 24.00	NS

Correlation of Social Quotient with Production of Intonation = .24 (NS)

Table XVII

Correlations: Social Quotient with Number of Lesions in Right and Left Hemispheres and Brain Stem Compression

	No. (R) Lesions	No. (L) Lesions	Brain Stem Compression
Social Quotient	-.43*	.16	-.42*

(*$p < .05$)

Table XVIII

Lowest GCS Scores Attained by Patients Post-Trauma and Perception & Production Scores

Production		Perception	
Borderline/ Defective (N = 8)	$\overline{X}$ = 10.00, SD = 2.97	Borderline/ Defective (N = 8)	$\overline{X}$ = 10.62, SD = 3.96
Average/ Low Avarage (N = 14)	$\overline{X}$ = 9.78, SD = 2.97	Average/ Low Average (N = 14)	$\overline{X}$ = 9.42, SD = 3.22

Figure 1

RESPONSES TO THE IDENTIFICATION OF FACIAL COMPONENTS OF EMOTION

Moderately & Severely Injured Patients

Error Category I

"Person number 1 and person number 3 feel alike because their boss probably gave them a raise. Person number 2 is happy probably but not because of a raise."

"This girl looks like she doesn't belong with the other two. She must feel different. The other two feel alike because they have money."

"Both these people have a definite sense of humor which they are not using at the moment. Both seem to have good basic humorful personalities. The third one does not."

Error Category II

"These two people feel alike because they are both holding microphones."

"This man feels different because he's a priest - can tell by his collar."

Error Category III

"This mother and daughter don't look happy. Yeah, they do look happy but it's phony, it's a put-on."

Mild Head Trauma Patient

"Person 1 & 2 are feeling alike. You can tell by their smiles that they must feel happy. The third person looks depressed by the way her lips are. When you're upset you make a certain face and she looks that way."

"One persons looks like she's crying and the other one also looks sad. The little boy looks happy. You can tell by the expression in their eyes. One woman's eyes are tearing and the other one's are half-closed while the child's have a happy expression."

Figure 2

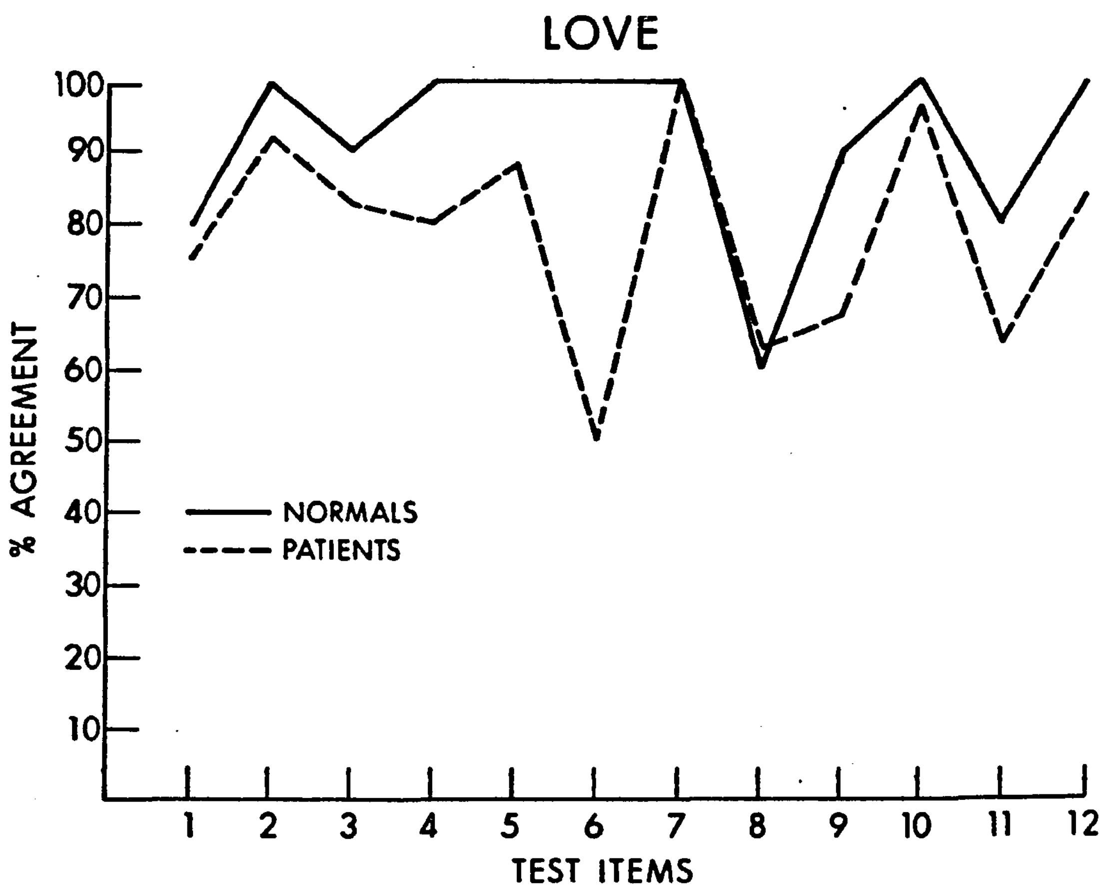

LOVE
% AGREEMENT
100
90
80
70
60
50
40
30
20
10
NORMALS
PATIENTS
1
2
3
4
5
6
7
8
9
10
11
12
TEST ITEMS

Figure 3

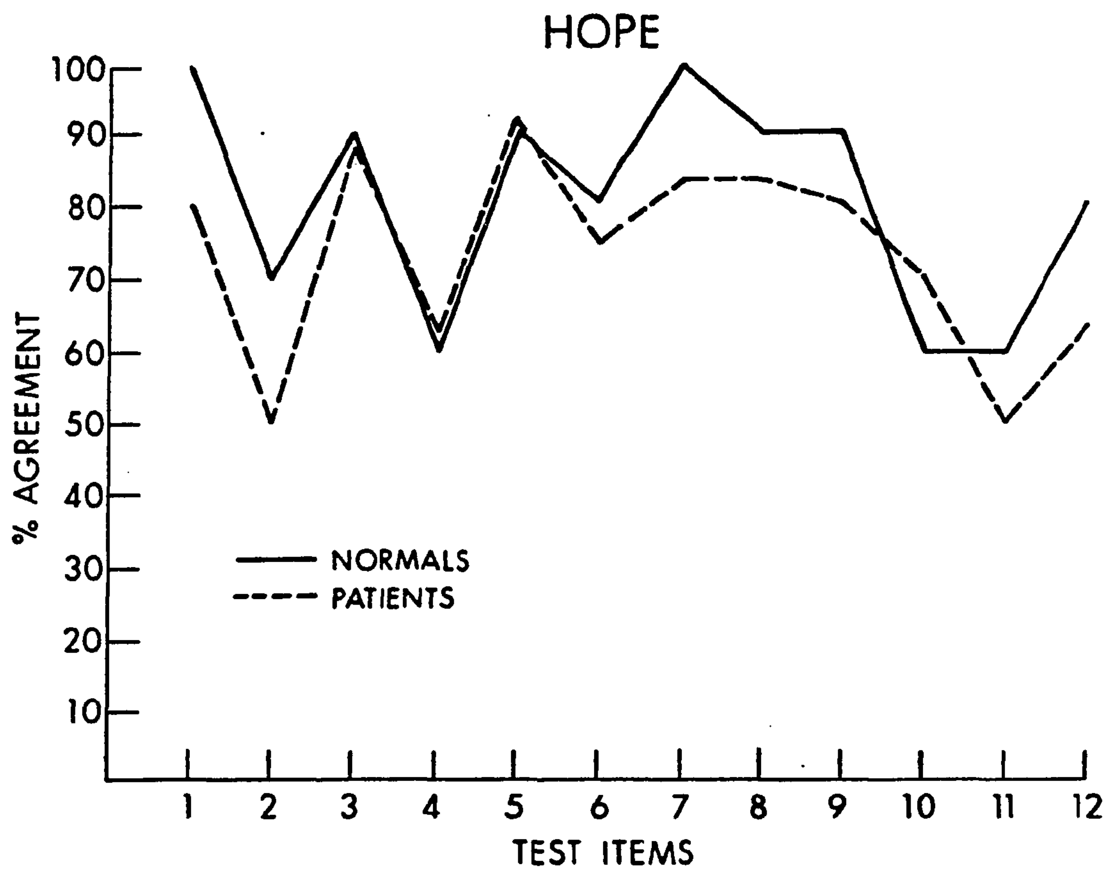

HOPE
100
90
80
70
60
50
40
30
20
10
% AGREEMENT
NORMALS
PATIENTS
1
2
3
4
5
6
7
8
9
10
11
12
TEST ITEMS

Figure 4

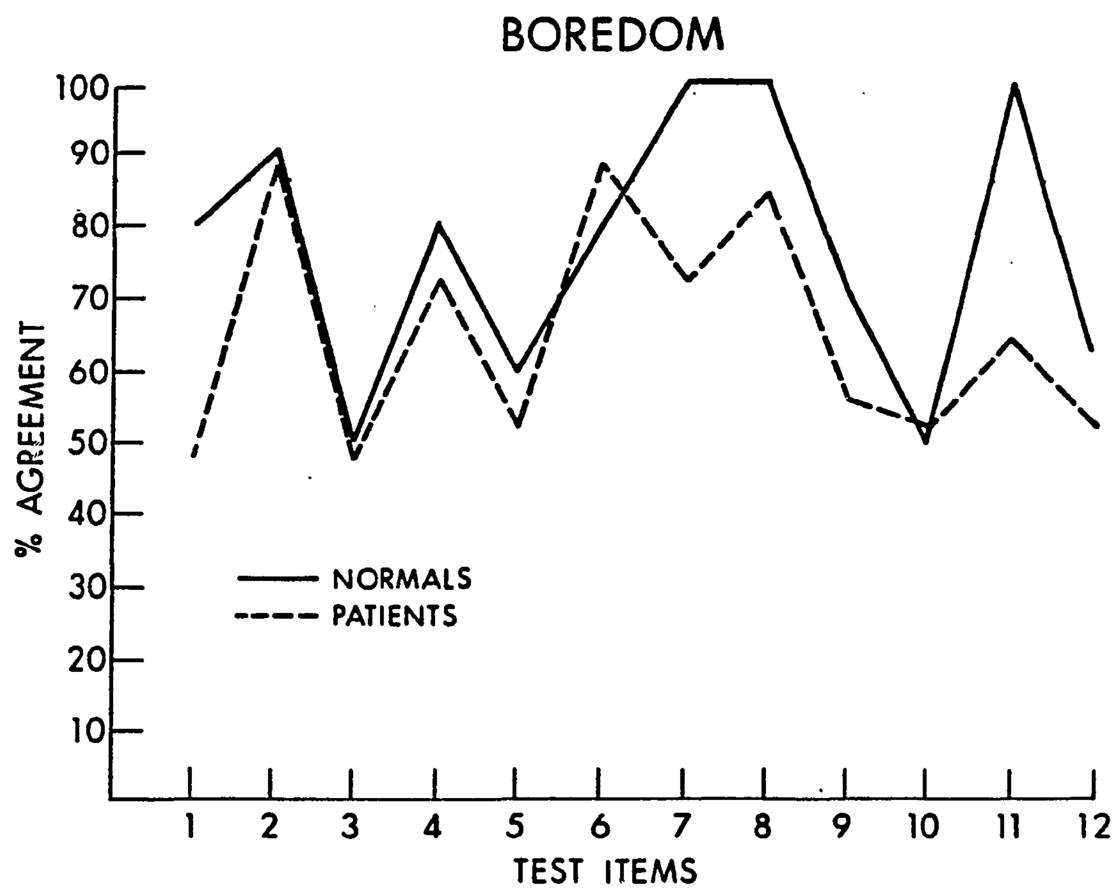

BOREDOM
% AGREEMENT
100
90
80
70
60
50
40
30
20
10
NORMALS
PATIENTS
1
2
3
4
5
6
7
8
9
10
11
12
TEST ITEMS

Figure 5

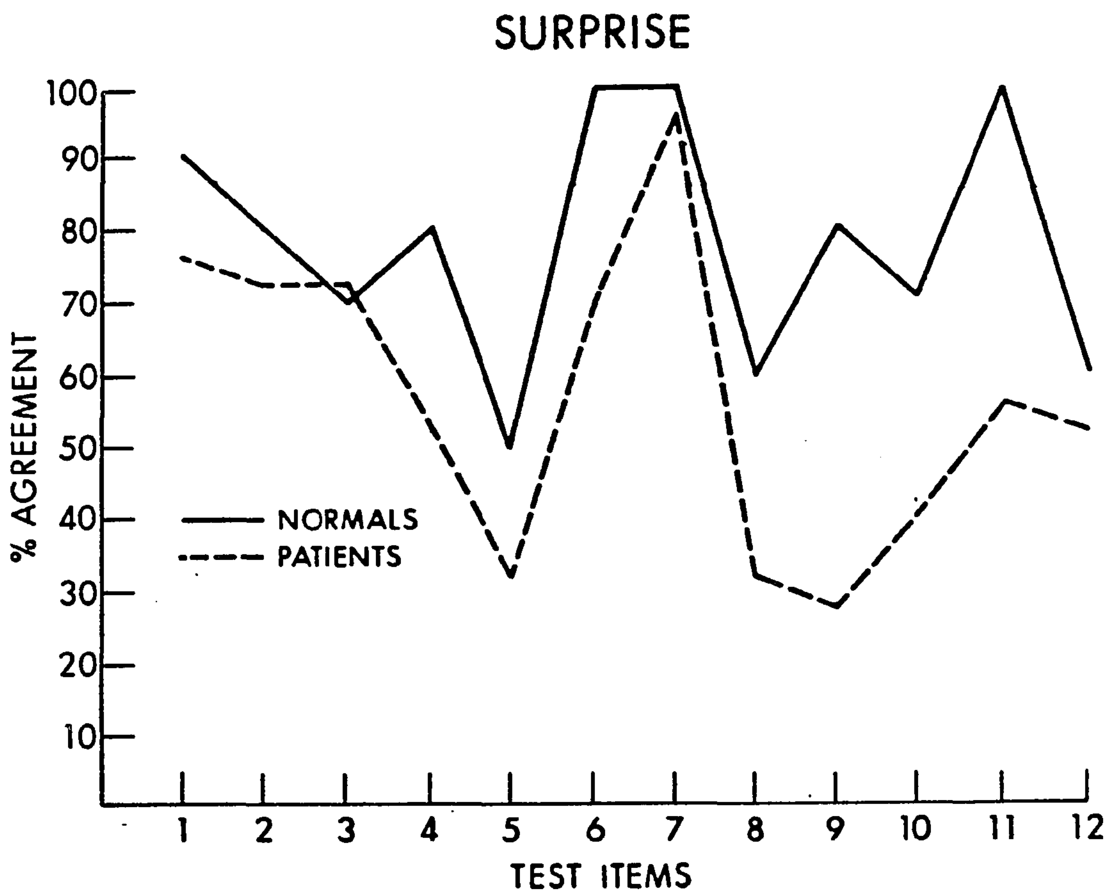
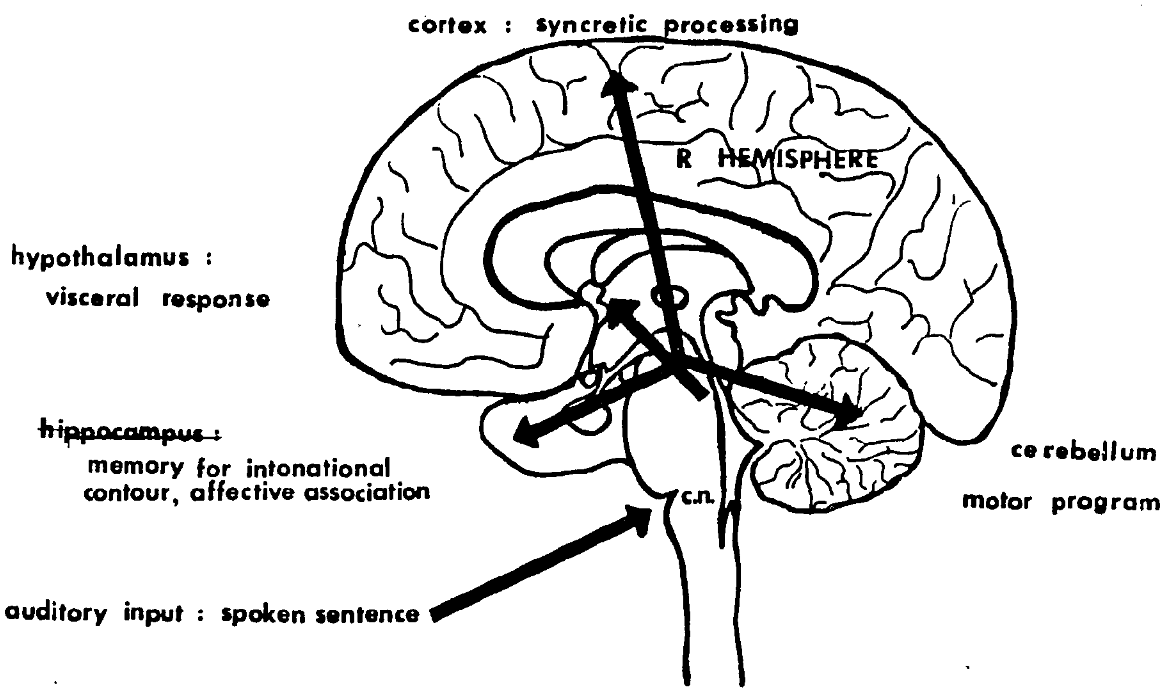

SURPRISE
100
90
80
70
60
50
40
30
20
10
% AGREEMENT
NORMALS
PATIENTS
1
2
3
4
5
6
7
8
9
10
11
12
TEST ITEMS

Figure 6

Axonal Projections of a Noradrenergic Brainstem Neuron and the Processing of Affectively Intoned Speech



Appendix I (a)

Perception and Production of Intonation:
Males Versus Females

PATIENTS

Perception

Males (N=21) X = 37, SD = 9.55

Females (N=6) X = 33, SD = 18.50

t = .710 NS

Production

Males (N=21) X = 12, SD = 3.17

Females (N=6) X = 16, SD = 3.19

t = 2.50 p .02

CONTROLS

Perception

Males (N=6) X = 42, SD = 6.09

Females (N=4) X = 50, SD = 8.01

t = 1.86 NS

Production

Males (N=6) X = 14, SD = 3.39

Females (N=4) X = 16, SD = 1.92

t = 1.37 NS

Appendix I (b)

Differences Between Males and Females (t tests)

CONTROLS (N=10)

	t	p value
Production	1.36	.21 NS
Perception	1.85	.10 NS
Education	1.08	.31 NS
Age	.35	.73 NS

PATIENTS (N=27)

	t	p value
Production	2.98	.006
Perception	.74	.46 NS
Education	.66	.53 NS
Age	.84	.41 NS
PIQ	1.27	.21 NS
VIQ	.43	.68 NS
Purdue Left Hand	.21	.83 NS
Purdue Right Hand	.85	.40 NS
Shift Size	-1.77	.09 NS
Social Quotient	.20	.84 NS
Time From Injury	.05	.95 NS

Appendix II

Conceptual Identification of Emotion Questionnaire

THIS QUESTIONNAIRE IS COMPRISED OF A SERIES OF BODILY STATES OR EMOTIONS WITH DESCRIPTIVE STATEMENTS FOR EACH. PLEASE ANSWER "YES" or "NO" FOR EACH STATEMENT DEPENDING ON WHETHER YOU FEEL THAT IT APPLIES OR DOES NOT APPLY TO THE STATE OR EMOTION ABOVE. PLEASE ANSWER ALL QUESTIONS. IF A PARTICULAR STATEMENT IS AMBIGUOUS, CHOOSE THE ANSWER THAT YOU FEEL IS MOST APPROPRIATE.

HOPE:_____ there is a warm excitement

_____ I'm wound up inside

_____ the world seems basically good and beautiful

_____ I sweat

_____ there is a sense of anticipation, waiting for something to happen

_____ I feel choked up

_____ the future seems bright

_____ I begin to think what I can do to change the situation

_____ I'm excited in a calm way

_____ men are essentially kind

_____ I keep thinking about what happened over and over again

_____ I wish I could go back in time

BOREDOM:

_____ tears come to my eyes

_____ I feel tired, sleepy

_____ not caring about anything that goes on around me

_____ I'm optimistic abut the future, the future seems bright

_____ there is a sense of belonging with others

_____ my body seems to slow down

_____ my blood pressure goes up, blood seems to rush through my body

_____ there is a very strong sense that time seems to slow down, to drag

_____ there is a sense of trust and appreciation of another person

_____ it's as if I'm suffocating or smothering

_____ my whole body is tense

_____ everything seems unimportant

LOVE:_____ there is a sense of regret

_____ a sense of increased clarity and understanding of the world

_____ I keep thinking how lucky I am

_____ there is a feeling of warmth all over

_____ my fists are clenched

_____ there is a sense that I have no control over the situation

_____ I want to be tender and gentle with another person

_____ there is a heaviness in my chest

_____ there is a quickening of heartbeat

_____ I want to help, to protect, please another person

_____ there is a muscular rigidity

_____ my speech gets softer

SURPRISE:

_____ a sense of being more alive

_____ my face and mouth are tight, tense, hard

_____ I'm breathless

_____ there is an inner ache you can't locate

_____ everything, breathing, moving, thinking seems easier

_____ my pulse quickens

_____ I can't believe what's happening is true

_____ I feel safe and secure

_____ I feel wide awake

_____ everything is going right for me

_____ I want to strike out, explode, but I hold back, control myself

_____ there is a sense of disbelief

Bibliography

Adams, J.H., Graham, D.I., Gennarelli, T.A. Head injury in man and experimental animals: neuropathology. Acta Neurochirurgica, Suppl. 32, 1983, 15-30.

Adams, J.H., Graham, D.I., Murray, L.S., Scott, G. Diffuse axonal injury due to nonmissile head injury in humans: an analysis of 45 cases. Annals of Neurology, 1982, 12, 557-563.

Alpert, M., and Martz, M.J. Cognitive views of schizophrenia in light of recent studies of brain asymmetry. In C. Shagass, S. Gershon, and A.J. Friedhoff (Eds.), Psychopathology and brain dysfunction. New York: Raven Press, 1977.

Ariel, R.N., Golden, C.J., Berg, R.A., et al. Regional cerebral blood flow in schizophrenics - Tests using the Xenon XE-133 inhalation method. Arch. G. Psych., 1983, 40, 258-263.

Bader, B.W. Social perception and learning disabilities. Des Moines, Iowa: Moon Lithographing & Engraving, 1975.

Barr, D.F. Auditory perceptual disorders. Springfield: Charles C. Thomas, 1972.

Bear, D.M. Hemispheric specialization and the neurology of emotion. Archives of Neurology, 1983, 40, 195-202.

Bear, D., Levin, K., Blumer, S., et al. Interictal behavior in hospitalized temporal lobe epileptics: relationship to idiopathic psychiatric syndromes. J. Neurol., Neurosurg., Psychiatry, 1982, 45/6, 481-488.

Bergman, M., Hirsch, S., Najenson, T. Tests of auditory and management of patients with cerebral cranial injury. Scandinavian Journal of Rehabilitation Medicine, 1977, 9, 173-177.

Black, F.W. WISC verbal performance discrepancies or indications of neurological dysfunction in pediatric patients. J. Clin. Psychol., 1974, 30, 165.

Bleuler, M. Psychiatry of cerebral diseases. Br. Med. J., 1951, 1233-1238.

Blumstein, S., and Cooper, W.E. Hemispheric processing of intonation contours. Cortex, 1974, 10, 146-158.

Bond, M.R. Assessment of psychosocial outcome of severe head injury. In Outcome of Severe CNS Damage, Ciba Found. Symp. 34 (1975).

Bond, M.R., and Brooks, D.N. Understanding the process of recovery as a basis for the investigation of rehabilitation for the brain injured. Scandinavian Journal of Rehabilitation Medicine, 1976, 8, 127-133.

Bond, M.R., Brooks, D.N., and McKinlay, W. Brudens imposed on the relatives of those with severe brain damage due to injury. Acta Neurochirurgica, 1979, Suppl. 28, 124-125.

Borod, J.C., Koff, E., Perlman, M., and Nicholas, M. Channels of emotional expression in patients with focal lesions. Paper presented at the meeting of the International Neuropsychological Society, February, 1984.

Bratzke, H. Brain-stem injury and long survival - a forensic analysis. Acta Neurochirurgica, Suppl. 32, 109-114 (1983).

Brumback, R.A., Staton, R.D., and Wilson, H. Neuropsychological study of children during and after remission of endogenous depressive episodes. Perceptual and Motor Skills, 1980, 50, 1163-1167.

Bryan, T.M. Learning disabled childrens' comprehension of nonverbal communication. Journal of Learning Disabilities, 1977, 10, 501-506.

Chandler, M. Egocentrism and anti-social behavior: the assessment and training of social persepctive skills. Developmental Psychology, 1973, 9, 326-332.

Chusid, J.G. Correlative neuroanatomy and functional neurology. California: Lange Medical Publications, 1982.

Costa, L.D., Vaughan, H.S., Levita, E., Farber, N. Purdue pegboard as a predictor of the presence and laterality of cerebral lesions. Journal of Consulting Psychology, 1963, 27 (2), 133-137.

Critchley, M., and Henson, R.A. (Eds). Music and the brain. London: William Heinemann Medical Books Limited, 1977.

Damasio, A.R., Damasio, H., Rizzo, M., et al. Aphasia with nonhemorrhagic lesions in the basal ganglia and internal capsule. Archives of Neurology, 1982, 39, 15-20.

Davidson, R.J. Hemispheric specialization for affective processes in normals: behavioral and electrophysiological studies. Presented at the meeting of the Society for Biological Psychiatry, Atlanta, Georgia, May, 1978.

Davitz, J.R. A dictionary and grammar of emotion. In M. Arnold (Ed.), Feelings and emotions: The Loyola Symposium. New York: Academic Press, 1970.

Denckla, M.B. The neuropsychology of social-emotional learning disabilities. Archives of Neurology, 1983, 40, 461-462.

Dikmen, S., and Reitan, R. Emotional sequelae of head injury. Annals of Neurology, 1977, 2, 492-494.

Dimond, S.J., Scammell, R.E., Pryce, I.G., Huws, D., and Gray, C. Callosal transfer and left-hand anomia in schizophrenia. Biological Psychiatry, 1979, 14, 735-739.

Dirks, D.D. Effects of hearing impairment on the auditory system. In E. C. Carterette, and M.P. Friedman (Eds.), Handbook of Perception, 567-608. New York: Academic Press, 1978.

Doll, E.A. A genetic scale of social maturity. The American Journal of Orthopsychiatry, 1935, 5, 180-188.

Doll, E.A. The measurement of social competence: a manual for the Vineland Social Maturity Scale. Minneapolis Educational Test Bureau, 1953.

Doll, E.A. Vineland Social Maturity Scale: Condensed manual of directions (1965 edition). Minnesota: American Guidance Service, Inc., 1965.

Eccles, J.C. The understanding of the brain. New York: McGraw-Hill, 1973.

Edmonson, B., DeJung, J.E., Leland, H. Social perception (non-verbal communication) training of retarded adolescents. Mental Retardation, 1965, 3, 7-9.

Fahy, T.J., Irving, M.H., and Millac, P. Severe head injuries. A six year follow-up. Lancet, 1967, 2, 475-479.

Field, J. Two types of tables for use with Wechsler's Intelligence Scales. J. Clin. Psych., 1960, 16, 3.

Gelmers, H.J. Non-paralytic motor disturbances and speech disorders: the role of the supplementary motor area. Journal of Neurology. Neurosurgery and Psychiatry, 1983, 46, 1052-1054.

Gelmers, H.J. Regional cerebral blood floow correlates of speech in the dominant and in the non-dominant hemisphere. J. Neurol. Sci., (in press).

Goldstein, K. The effect of brain damage on the personality. Psychiatry, 1952, 15, 245-260.

Graham, D.I., and Adams, J.H. Ischaemic brain damage in fatal head injuries. Lancet, 1971, 265-266.

Guilford, J.P., and Fruchter, B. Fundamental statistics in psychology and eduction. U.S.: McGraw-Hill, Inc., 1978.

Heilman, K.M., and Satz, P. (Eds.), Neuropsychology of human emotion. New York: Guilford Press, 1983.

Heilman, K.M., Scholes, R., and Watson, R.T. Auditory affective agnosia. Disturbed comprehension of affective speech. Journal of Neurology, Neurosurgery and Psychiatry, 1975, 38, 69-72.

Heilman, K.M., and Valenstein, E. (Eds.), Clinical neuropsychology. New York: Oxford University Press, 1979.

Hillbom, E. Schizophrenia-like psychoses after brain trauma. Acta Psychiatrica Scandinavia, Suppl. 60, 1951, 36-47.

Hooper, R.S., McGregor, J.M., and Nathan P.W. Explosive rage following head injury. Journal of Mental Science, 1945, 91, 458-471.

Jennett, B. The problem of mild head injury. The Practioner, 1978, 221, 77-82.

Jennett, B. Prognosis after head injury. In P.J. Vinken, and G.W. Bruym (Eds.), Handbook of clinical neurology Vol. 24: Injuries of the brain and skull, 669-681. Amsterdam: North-Holland Publishing Co. 1976.

Jennett, B., and Teasdale, G. (Eds.), Management of head injuries. Philadephia: F.A. Davis Co., 1981.

Kety, S.S. The biogenic amines in the central nervous system: their possible roles in arousal, emotion and learning. In F.O. Schmitt (Ed.), The neurosciences. New York: Rockefeller University Press, 1970.

Kimura, D. Functional asymmetry of the brain in dichotic listening. Cortex, 1967, 3, 163-178.

Kronfol, Z., Hamsher, K. des., Digre, K., and Waziri, R. Depression and hemispheric functions: changes associated with unilateral ECT. British Journal of Psychiatry, 1978, 132, 560-567.

Langfitt, T.W., Gennarelli, T.A. Can the outcome from head injury be improved? J. Neurosurg., 1982, 56, 19-25.

Lansdell, H.C. Relation of extent of temporal removals to closure and visuomotor factors. Perceptual and Motor Skills, 1970, 31, 491-498.

Lansdell, H.C. A general intellectual factor affected by temporal lobe dysfunction. Journal of Clinical Psychology, 1971, 27, 182-184.

Larsen, B., Skinhj, E., Lassen, N.A. Variations in regional cortical blood flow in the right and left hemispheres during automatic speech. Brain, 1978, 101, 193-209.

Leahy, R.L. Development of conceptions of prosocial behavior: information affecting rewards given for altruism and kindness. Developmental Psychology, 1979, 15, 34-37.

Lerner, J.W. Children with learning disabilities, ed. 2, Boston: Houghton Mifflin Company, 1976.

Levin, H.S., and Grossman, R.G. Behavioral sequelae of closed head injury. Archives of Neurology, 1978, 35, 720-727.

Levin, H.S., Grossman, R.G., and Kelly, P.J. Aphasic disorder in patients with closed head injury. Journal of Neurology, Neurosurgery and Psychiatry, 1976, 39, 1062-1070.

Levin, H.S., Grossman, R.G., ROSE, J.E., Teasdale, G. Long-term neuropsychological outcome of closed head injury. J. Neurosurg., 1979, 50, 412-422.

Levin, H.S., Madison, C.F., Bailey, C.B., Meyers, C.A., Eisenberg, H.M., Guinto, F.C. Mutism after closed head injury. Archives of Neurology, 1983, 40, 601-606.

Lezak, M. Neuropsychological Assessment, New York: Oxford University Press, 1976.

Lishman, W.A. The psychiatric sequelae of head injury: a review. Psychological Medicine, 1973, 3, 304-318.

Lundholm, J., Jespen, B.N., and Thornval, G. The late neurological, psychological, and social aspects of severe traumatic coma. Scandinavian Journal of Rehabilitation Medicine, 1975, 7, 97-100.

Luria, A.R. Traumatic Aphasia. Netherlands: Mouton & Company, N.V., 1970.

Malmberg, B. Prosodic phenomena. In B. Malmberg (ed.), Manual of Phonetics. Amsterdam: North Holland Publishing Company, 1968.

Mandleberg, I.A., and Brooks, D.N. Cognitive recovery after severe head injury: 1. Serial testing on the Wechsler Adult Intelligence Scale. Journal of Neurology, Neurosurgery, and Psychiatry, 1975, 38, 1121-1126.

Marsden, C.D. The mysterious motor function of the basal ganglia: The Robert Wartenberg lecture. Neurology, 1982, 32, 514-539.

Minskoff, E.H. A teaching approach for developing nonverbal communication skills in students with social perception deficits. International Journal of Learning Disabilities, 1980, 13, 118-124.

Mitchell, D.E., and Adams, J.H. Primary focal impact damage to the brainstem in blunt head injuries - does it exist? Lancet, 1973, August, 215-218.

McGlone, J. Functional brain asymmetry studied in patients with lateralized lesions. Paper presented at the International Neuropsychological Society Meeting, Toronto, 1976.

McKinlay, W.W., Brooks, D.N., Bond, M.R., Martinage, D.P., and Marshall, M.M. The short-term outcome of severe blunt head injury as reported by relatives of the injured persons. Journal of Neurology, Neurosurgery and Psychiatry, 1981, 44, 527-533.

Monrad-Kohn, G.H. The third element of Speech Prosody and Its Disorders. In L. Halpern (ed.), Problems of Dynamic Neurology. Jerusalem: Jerusalem Post Press, 1963.

Nauta, W.J.H. The problem of the frontal lobe: a reinterpretation. Journal of Psychiatric Research, 1971, 8, 167-187.

Oddy, M., Humphrey, M., and Uttley, D. Subjective impairment and social recovery after closed head injury. Journal of Neurology, Neurosurgery and Psychiatry, 1978, 41, 611-616.

Oke, A., Keller, R., Mefford, I., and Adams, R.N. Lateralization of norepinephrine in human thalamus. Science, 1978, 200, 1411-1413.

Ommaya, A.K., and Gennarelli, T.A. Cerebral concussion and traumatic unconsciousness. Brain, 1974, 97, 633-654.

Oppenheimer, D.R. Microscopic lesions in the brain following head injury. Journal of Neurology, Neurosurgery and Psychiatry, 1968, 31, 299-306.

Outcome of Severe Damage to the Central Nervous System. Ciba Foundation Symposium 34 (new series). Elsevier: Excerpta Medica, 1975.

Overall, J.E., and Gorham, D.R. The brief psychiatric rating scale. Psychological Report, 1962, 10, 799-812.

Oxbury, J.M., and Oxbury, S.M. Effects of temporal lobectomy on the report of dichotically presented digits. Cortex, 1969, 5, 3-14.

Pallay, S.L., Chute, P., Zapulla, R.A., and Rosen, J.J. The relationship between brainstem compression and time compressed speech perception. Paper presented at the meeting of the International Neuropsychological Society, February, 1984.

Penfield, W., and Jasper, H. Epilepsy and the functional anatomy of the human brain. Boston: Little Brown, 1954, 88-108.

Penny, J.B., Jr., and Young, A.B. Speculations on the functional anatomy of basal ganglia disorders. Annu. Rev. Neurosci., 1983, 6, 73-94.

Reitan, R. Certain differential effects of left and right cerebral lesions in human adults. Journal of Comparative and Physiological Psychology, 1955, 48, 474-477.

Robinson, R.G. Differential behavioral and biochemical effects of right and left hemispheric cerebral infarction in the rat. Science, 1979, 205, 707-710.

Rosenthal, R., and Bigelow, L.B. Quantitative brain measurements in chronic schizophrenia. British Journal of Psychiatry, 1972, 121, 259-264.

Rosenthal, M., Griffith, E.R., Bond, M.R., and Miller, J.D. (Eds.), Rehabilitation of the head injured adult. Philadelphia: F.A. Davis Co., 1983.

Ross, E., The aprosodias. Functional-anatomic organization of the affective components of language in the right hemisphere. Archives of Neurology, 1981, 38, 561-569.

Ross, E.D., and Mesulam, M.M. Dominant language functions of the right hemisphere? Prosody and emotion gesturing. Archives of Neurology, 1979, 36, 144-148.

Science Research Associates. SRA Examiner's Manual for the Purdue Pegboard. Chicago Science Research Assoiates, 1948.

Scott, S., and Caird, F.I. Speech therapy for Parkinson's disease. Journal of Neurology, Neurosurgery, and Psychiatry, 1983, 46, 140-144.

Shapiro, S.S., and Wilk, M.P. An analysis of variance test for normality. Biometrika, 1965, 52, 591-611.

Simpson, C.D., and Vega, A. Unilateral brain damage and patterns of age-corrected WAIS subtest scores. Journal of Clinical Psychology, 1971, 27, 204-208.

Sorman, P., Wasserstein, J., and Zapulla, R. Cerebral correlates of cognitive regression and parallels with schizophrenic thought processes. Presented at the International Neuropsychological Society Conference, Mexico City, 1983.

Springer, S.P., and Deutsch, G. Left Brain, Right Brain. San Francisco: W.H. Freeman and Co., 1981.

Strub, R.L., and Black, F.W. Organic brain syndromes. Philadelphia: F.A. Davis Company, 1981.

Sweet, W.H., Ervin, F., and Mark, V.H. The relationship of violent behavior to focal cerebral disease. In S. Garattini and E.B. Sigg (eds.), Aggressive Behavior, Proceedings for the International Symposium on the Biology of Aggressive Behavior, Amsterdam: Excerpta Medica, 1969.

Symonds, C.P. Concussion and its sequelae. Lancet, 1962, i, 1-5.

Tabaddor, K., Danziger, A., and Wisoff, H. Estimation of intracranial pressure by CT scan in closed head trauma. Surgical Neurology, 1982, 18, 212-215.

Tabaddor, K., Mattis, S., Zazula, T. Cognitive sequelae and recovery course after moderate and severe head injury. Neurosurgery, 1984, 14, 701-708.

Taylor, A.R. Post-concussional sequelae. British Medical Journal, 1967, 3, 67-71.

Teasdale, G., and Jennett, B. Assessment of coma and impaired consciousness. Lancet, 1974, II: 81-84.

Thomsen, I.V. The patient with severe head injury and his family. Scandinavian Journal of Rehabilitation Medicine, 1974, 6, 180-183.

Tomlinson, B.E. Brainstem lesions after head injury. Journal of Clinical Pathology, 1970, 23, Suppl. No. 4, 154-165.

Tucker, D.M. Lateral brain function, emotion, and conceptualization. Psychological Bulletin, 1981, 89, No. 1, 19-46.

Tucker, D.M., Watson, R.T., Heilman, K.M. Discrimination and evocation of affectively intoned speech in patients with right parietal disease. Neurology, 1977, 27, 947-950.

Vaughan, H.G., Jr., and Costa L.D. Performance of patients with lateralized cerebral lesions. II: Sensory and motor tests. Journal Nerv. Ment. Dis., 1962, 134, 237-244.

Walker, A.E. Long term evaluation of the social and family adjustment to head injuries. Scandinavian Journal of Rehabilitation Medicine, 1972, 4, 5-8.

Wassertheil-Smoller, S., Tabaddor, K., Feiner, C., Shulman, K. Factors affecting short-term outcome of head-trauma patients. Neuroepidemiology, 1982, 1, 154-166.

Wechsler, D. Manual for the Wechsler Adult Intelligence Scale. New York: The Psychological Corp., 1955.

Weintraub, S., and Mesulam, M.M. Developmental learning disabilities of the right hemisphere. Emotional interpersonal and cognitive components. Archives of Neurology, 1983, 40, 463-468.

Weintraub, S., Mesulan, M.M., and Kramer, L. Disturbances in prosody. A right hemisphere contribution to language. Archives of Neurology, 1981, 38 (12), 742-744.

Werner, H. The concept of development from a comparative and organismic point of view. In D. B. Harris (Ed.), The concept of development. Minneapolis: University of Minnesota Press, 1957.

Wing, L. Asperger's syndrome: a clinical account. Psychological Medicine, 1981, 11, 115-129.

Zazula, T., Tabaddor, K., Chaudhari, A.B., Sharpless, N., Mattis, S. Neurotransmitter disturbance and neuropsychological status in severely head injured patients. (in prep).

Zurif, E.B., and Mendelsohn, M. Hemispheric specialization for the perception of speech sounds. Perception and Psychophysics, 1972, 11, 329-332.

Zurif, E.B., and Sait, P.E. The role of syntax in dichotic listening. Neuropsychologica, 1970, 8, 239-244.