Substantial evidence has accumulated over the past 35 years suggesting deficient intersentential cohesion in the narrative discourse of many individuals with traumatic brain injury (TBI). Since Mentis and Prutting (1987) reported significantly fewer cohesive ties in the narratives of brain-injured versus normal speakers, a number of reports have replicated and expanded upon these findings (e.g., Coelho, Liles, & Duffy, 1995; Davis & Coelho, 2004; Hartley & Jensen, 1991; Liles, Coelho, Duffy, & Zalagans, 1989; Marini et al., 2011). At the same time, several studies have not found evidence of deficient intersentential cohesion in these adults (Coelho, 2002; Glosser & Deser, 1991; Hough & Barrow, 2003). It can be said then that the narratives produced by at least some speakers with TBI tend to show less adequate intersentential cohesion than that observed in normal speakers (Cannizzaro & Coelho, 2012; Coelho, 2007).

Variable microlinguistic impairment has also been observed in narrative discourse following TBI. Sentences produced by speakers with TBI have been reported to include more lexical and syntactic errors, increased mazes, more frequent pausing, and greater reductions in content when compared to normal speakers (Ellis & Peach, 2009; Glosser & Deser, 1991; Hartley & Jensen, 1991; Peach, 2013; Peach & Schaude, 1986; Stout, Yorkston, & Pimenthal, 2000). Such narratives also tend to be less efficient (i.e., lengthier and containing more words per maze) (Erlich, 1988; Hartley & Jensen, 1991; Stout et al., 2000) and less complex (Coelho, Grela, Corso, Gamble, & Feinn, 2005; Peach, Shapiro, Rubin, & Schaude, 1990) than those produced by normal speakers. Nonetheless, other studies have not found few, if any, such microlinguistic disturbances in speakers with TBI (Hough & Barrow, 2003; Marini et al., 2011).

It has been suggested that brain injury produces a dissociation between the macrolinguistic and microlinguistic components of narrative production (Glosser & Deser, 1991; Hough & Barrow, 2003) and thus, that the processing of discourse and the processing of sentences are based on different cognitive devices (Consentino, Adornetti, & Ferretti, 2013). Alternatively, it might be that the macrolinguistic structure of narratives produced by brain injured speakers is affected by their microlinguistic impairments (see, e.g., Boyle, 2011; Christiansen, 1995). Given the variable patterns that have been observed in the narratives of speakers with TBI, it may be that narrative production following TBI is the result of an effortful interaction between macrolinguistic and microlinguistic processes rather than dissociation of separate cognitive mechanisms. However, few studies exist that attempt to establish the connections between these different levels of language. As a result, there is little information available to describe how specific changes at either level may influence the processing of narrative discourse. Armstrong (2000) has suggested that cohesion analysis is one of the few methods which directly attempts to link the macro- and microlinguistic aspects of discourse. This study therefore investigated the relationship between intersentential cohesion and microlinguistic impairments in discourse produced by speakers with severe traumatic brain injury (TBI).

Method

Participants

Fifteen non-aphasic individuals approximately six months post severe traumatic brain injury (TBI) participated in this study. All participants were native English speakers and had attained at least 11 years of formal education. Medical records were obtained for each TBI

participant. All patients were screened prior to admission to the study to rule out the presence of hearing loss sufficient to interfere with conversation or a history of significant alcohol, drug, or psychiatric involvement (e.g., depression); the screening results were verified subsequently through review of the participants' medical records. The demographic and clinical characteristics of the participants are presented in Table 1.

All TBI participants presented a history of hospital admission with a diagnosis of traumatic brain injury and a documented period of coma greater than 24 hours. None of these participants had a history of previous head injury or other neurological involvement.

All of the participants demonstrated the typical pattern of diffuse axonal injury associated with traumatic brain injury. Participants presented no complicating medical conditions resulting in diffuse brain damage (e.g., anoxia) other than that attributable to the head injury. Focal injuries, when present, were limited to cerebral contusions and/or subdural hematomas. Medical diagnoses were established using information obtained from CT scans, angiography, electroencephalography (EEG), and neurological examinations. In order to rule out significant aphasia, the oral language subtests of the Western Aphasia Battery (WAB) were administered using standard procedures and an aphasia quotient (AQ) was calculated for each participant (scores appear in Table 1). No TBI participant exhibited dysarthria or apraxia of speech (AOS) sufficient to affect the results of this study as determined by two certified and licensed speech-language pathologists.

Procedure

Narratives consisting of WAB *Picnic Scene* descriptions were audio recorded and analyzed. The narratives were elicited using the standard instructions for this task and were transcribed orthographically. The audio samples were uploaded into *Praat* (v.5.3.04) (Boersma & Weenink, 2010), displayed on a computer monitor, and compared to the language transcriptions. Pauses of greater than 200 ms (Goldman-Eisler, 1968; Kirsner, Dunn, Hird, Parkin, & Clark, 2002) were identified within sentences.

The transcripts were entered into *Systematic Analysis of Language Transcripts* (SALT, v. 9.0) (Miller & Iglesias, 2008), segmented into utterances using standard procedures (SALT Guide version 9, 2008), and analyzed. The intersentential cohesive ties produced by each speaker were identified and judged for their adequacy (complete, cohesive errors). Three types of microlinguistic events were identified and coded within the transcripts: a) pauses, b) mazes (i.e., word and nonwords fillers, repetitions, and revisions), and c) grammatical errors (e.g., subject-verb disagreement, omissions). Instances of immediate co-occurrence between complete and error cohesive ties and microlinguistic impairment were tallied and analyzed with regard to the relative frequency of microlinguistic failures associated with each of these types of ties.

Results and Conclusions

The mean number of cohesive ties (complete and error) produced by these speakers was 11.8 (range = 5-28) (Table 2). The mean cohesive adequacy of the narratives was 69% (range = 0-100%). All but one of the speakers produced at least one error tie. Participants produced significantly more complete cohesive ties (69%) than error ties (31%) (t=3.00, p = .01).

In general, more cohesive ties were produced without microlinguistic impairment than with microlinguistic impairment by these speakers (t=3.47, p=.004). However, nearly one-third (30%) of the total number of cohesive ties was associated with some degree of microlinguistic impairment (r = .74, p = .002). When analyzed for cohesive adequacy, no differences emerged between the instances of microlinguistic impairment associated with complete ties (26%) than with error ties (19%) (p = .36). When the individual patterns displayed by these speakers are examined, significantly more speakers demonstrated microlinguistic impairment during production of cohesive ties than not (p = .007) (Table 3).

These results will be discussed with regard to a processing account of discourse planning. That is, utilization of resources required for establishing adequate cohesion in discourse following TBI appear to negatively affect microlinguistic processing for sentence production. These findings are consistent with recent work that microlinguistic deficits following TBI are related to difficulties with the recruitment and control of attention required for discourse planning.

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	TBI			
	Mean	(SD)	(Range)	
Age	26.1	(6.0)	(19-36)	
Education (years)	13.5	(2.0)	(11-18)	
Time post injury (months)	6.3	(0.5)	(6-7)	
Coma duration (days)	4.2	(3.4)	(1-11)	
Length of Hospitalization (days)	42.6	(19.1)	(16-79)	
Western Aphasia Battery - Aphasia Quotient	96.0	(1.9)	(91.8-98.6)	

Table 1. Demographic and clinical characteristics of participant group with traumatic brain injury (TBI).

Table 2. Discourse measures for speakers with TBI.

Participants	Ties (Complete % Co + Error) Ade	% Cohesive Adequacy	Types of Microlinguistic Impairment by Cohesive Adequacy Pauses Mazes					Total Impairment/ Cohesive Adequacy		
			Complete Ties	Error Ties	Complete Ties	Error Ties	Complete Ties	Error Ties	Complete Ties	Error Ties
1	7	100	0	0	0	0	0	0	0	0
2	16	94	4	0	1	0	0	0	5	0
3	6	67	1	0	0	0	0	0	1	0
4	6	67	0	1	Ő	0 0	Ő	Ő	0	1
5	5	0	0	0	ů 0	ů 0	Ő	0 0	ů 0	0
6	5	60	1	1	0	0	0	0	1	1
7	28	68	8	1	1	2	0	0	9	3
8	15	73	5	3	2	1	0	0	7	4
9	6	33	0	0	0	1	0	0	0	1
10	9	78	1	0	0	0	0	0	1	0
11	18	78	2	0	2	0	0	0	4	0
12	19	84	4	0	2	0	1	0	7	0
13	20	95	1	0	0	0	0	0	1	0
14	8	75	0	0	1	0	2	0	3	0
15	9	67	2	0	0	0	1	1	3	1
TOTAL	177	69 ^a	29	6	9	4	4	1	42	11

^aMean

Table 3. Cohesion patterns with or without accompanying microlinguistic deficits in speakers with TBI.

Cohesion/deficit ^a patterns among speakers	Subjects	
Cohesive Ties with associated microlinguistic deficits Complete ties with Deficits and Error ties with No Deficits Complete ties with Deficits and Error Ties with Deficits Complete Ties with No Deficits and Error Ties with	7 4 2	
Deficits Cohesive Ties with no associated microlinguistic		
Complete Ties only with No Deficits Error Ties only with No Deficits	1 1	

^aDeficits include any combination of pauses, mazes, and/or grammatical errors