Another View of Amobarbital Induced Speech and Language Disturbances

Stanley J. Ewanowski and Denise Cariski
University of Wisconsin Hospital and Clinics, Madison, Wisconsin

Jay Rosenbek
Wm. S. Middleton Memorial Veterans Hospital, Madison, Wisconsin

INTRODUCTION

For many years, the intracarotid injection of sodium amytal (amobarbital) has been used to determine hemispheric dominance for speech and language functioning. The rationale for using this pharmacological agent is that it induces a transient speech and language disturbance when injected into the dominant hemisphere. Most investigators using amobarbital have given only cursory descriptions of the specific speech and language disturbances that were induced. Very few have attempted to correlate the premedication performance on speech and language tests or measures with patient performance following the amobarbital injection(s).

The present investigation was designed to describe the specific speech, language, and other behaviors manifested following the injection of amobarbital into either the right or left cerebral hemisphere. In addition, an attempt was made to correlate the pre-medication clinical test results and/or interviews with the speech and language behaviors observed during the amobarbital testing.

SUBJECTS

The subjects were five (4 males, 1 female) neurosurgical patients with cerebral neoplasms. Four subjects were left-handed (WF, GM, EC, JO) and one subject (IH) wrote with his right hand but did all other activities with his left. A right hemisphere tumor was present in four of the subjects (WF, IH, GM, JO), and a left hemisphere tumor was present in one (EC). Subject JO had a recurrent right temporal lobe tumor for which he had undergone brain surgery 3 months prior to the amobarbital procedure. Selected patient data appear in Table 1.

Table 1. Descriptive data for age, sex, handedness, localization of neoplasm, and internal carotid artery injected for each subject.

Subject	Age	Sex	Handedness	Localization of neoplasm	Internal carotid artery injected
WF	57	Male	Left	Right temporo-occipital	Right
IH	45	Male	Mixed	Right frontal	Right
GM	58	Male	Left	Right temporal	Left
EC	59	Female	Left	Left frontal	*Left/Right
JO	57	Male	Left	Right temporal	*Right/Left

*Internal carotid artery injected first.

Three of the five subjects' communication behaviors (IH, EC, JO) were formally assessed prior to the amobarbital testing. Circumstances required that we interview two subjects (WF, GM). The Mayo Clinic Procedures for Language Evaluation were administered to IH and EC. JO was assessed via the Token Test, the Word Fluency Measure, a writing sample, and a Standard Speech Sample.

EC displayed no speech or language disturbances. IH displayed an aphasia of mild to moderate degree. JO had a mild aphasia. Neither of the two subjects who were interviewed (WF, GM) displayed aphasic disturbances. GM had an ataxic dysarthria which had appeared two days prior to the interview. His dysarthria was believed to have been pharmacologically induced.

METHODS

Each subject was injected with 100mg of amobarbital in 10ml of sterile water over a period of 3 to 4 seconds. Injection took place through a standard femorocerebral artery catheter positioned in the internal carotid artery. In two cases the right cerebral hemisphere was anesthetized (WF, IH), in one case the left cerebral hemisphere was anesthetized (GM), and in two cases bilateral hemispheric anesthetization took place (EC, JO). Side of injection is indicated in Table 1. Angiography was carried out in all cases prior to the amobarbital injections.

A three part behavioral protocol modeled after the Mayo Clinic Procedure (Blume, Grabow, Darley, and Aronson, 1973) was used to assess speech, language, and memory functions (see Table 2 for protocol). The entire assessment session was audiotaped and from these recordings typed transcripts were prepared. Three experienced speech-language pathologists (the authors) independently analyzed each subject's recordings and transcripts with respect to spontaneous speech as well as elicited speech and language behaviors. Each subject's performance was analyzed for dysarthric, apraxic, aphasic, and other deviant behavioral responses. Any interjudge differences were resolved through discussion.

RESULTS

Table 3 summarizes post-amobarbital behavior compared to pre-amobarbital behavior for each subject.

Dysarthria. A rating of post-amobarbital dysarthria was possible in only four of the five subjects, because one subject, GM, exhibited dysarthria prior to the study. He will be discussed separately. Of these four subjects, all displayed dysarthria regardless of the hemisphere injected with amobarbital. Dysarthria was classifiable as ataxic in three of the four subjects. One subject's dysarthria (WF) was mild and we could not classify it. Severity of dysarthria showed both intersubject and intrasubject variability. There was noticeable waxing and waning of the dysarthria from one moment to another. In one case (EC) the dysarthria also varied in relationship to the hemisphere that was injected. EC's dysarthria was more pronounced when the left hemisphere received the barbiturate than the right. Onset of dysarthria following injection ranged from 3-20 seconds. It cleared in all cases by the end of the procedure. Subject GM was mildly dysarthric prior to the amobarbital injection. His dysarthria was probably secondary to pharmocological agents, and it exhibited no change following the barbiturization.

Table 2. Language and memory assessment protocol.*

	Time (minutes)	Task	Example
Section 1	3	Shown number and asked to remember it	3
	2	Shown color and asked to remember it	Red
	1/2	Requested to	Raise arms, wiggle fingers, and begin counting
		Injection of amobarbital	
Section 2	When testable	Requested to	Continue counting
		Follow spoken commands	Stick out tongue, wiggle tongue, and blow
		Read printed words	House, tree, and sleep
	1-1/2	Identify designs	Cross and clock
		Repeat spoken words	Snowman, gingerbread, artillery, impossibility, and please sit down
	2	Exhibit limb strength	Raise arms and grip strength
	2-1/2—3	Describe pictures	Boy, truck, and dog
		Define words	Robin and machine
	3-1/2	Recall number and color: spontaneous or multiple-choice	3, red
	4	Exhibit limb strength	Raise arms, grip strength
	At periodic intervals thereafter		Limb strength tested until normal for patient
Section 3	12	Recall of number and color and/or events of procedure	Spontaneous or multiple-choice

*After Blume et al.. 1973.

Table 3. Comparison of pre-amobarbital communication behavior with post-amobarbital communication and/or other behavior for each subject.

Subject	Pre-amobarbital communication behavior	Post-amobarbital communication behavior		Post-amobarbital other behavior	
		Left injection	Right injection	Left injection	Right injection
WF	No aphasia	(Not injected)	Mild aphasia Dysarthria	(Not injected)	No symptoms
IH	Mild-moderate aphasia	(Not injected)	Severe aphasia Ataxic dysarthria	(Not injected)	Unresponsiveness
GM	No aphasia Ataxic dysarthria	Aphasia Ataxic dysarthria	(Not injected)	Unresponsiveness	(Not injected)
EC	No aphasia	Mild aphasia Ataxic dysarthria Apraxia Stuttering	Severe aphasia Ataxic dysarthria Apraxia Stuttering	No symptoms	Unresponsiveness Confusion Euphorical-maniacal reaction
JO	Mild aphasia	Mild aphasia Ataxic dysarthria	Severe aphasia Ataxic dysarthria	Unresponsiveness Agitation	Confusion

Apraxia. In only one case (EC) was verbal apraxia observed. This occurred when each hemisphere was injected, and was greater for the right than the left.

Aphasia. Three of the subjects did not exhibit aphasia prior to the barbiturization, and two did. Of the three subjects who were not aphasic pre-injection (WF, GM, EC), two of them (GM, EC) became grossly aphasic when the suspected dominant hemisphere was injected. In both cases, a tumor was present contralateral to the suspected dominant hemisphere and side which received the amobarbital. EC also displayed mild aphasic symptoms when her nondominant hemisphere was injected. The third subject (WF) displayed subtle aphasic symptoms when his (suspected) nondominant hemisphere was injected. His (suspected) dominant hemisphere was not injected. Of the two subjects who exhibited some degree of aphasia pre-injection (IH, JO), one of them (IH) was mildly to moderately aphasic from the tumor in his right hemisphere. He became grossly aphasic following right sided injection. His left hemisphere was not anesthetized. JO's aphasia increased in severity when both hemispheres were perfused, but he became grossly aphasic following left sided perfusion. Although all were aphasic post-amobarbital, they displayed a variety of language disturbances while under the influence of the drug, including the following: verbal paraphasias in one case (EC), reading errors in three cases (WF, GM, JO), and jargon in two cases (EC, JO). Verbal perseveration in either counting or discourse was present in four subjects (IH, GM, EC, JO). Confused counting was present in three subjects (WF, IH, GM). (Sound and syllable repetitions were not classified as verbal perseverations.) Only subject EC exhibited the sound and syllable repetitions which some investigators have referred to as cortical or neurogenic stuttering (Canter, 1971). This behavior resulted from amobarbital being injected into each of the hemispheres.

Language of Confusion. In addition to the more classical types of motor speech and language deviations, two of the subjects (EC, JO) exhibited behaviors that might better be classified as the language of confusion. For example, JO in the middle of counting, began to talk about golf balls. EC commented about dropping a cigarette even though she had not been smoking. Confusion appeared shortly after amobarbital injection in the case of JO, but only after several minutes in EC's case.

Other Behaviors. Behavioral changes other than speech and language also were observed following amobarbital injections. Four subjects (IH, GM, EC, JO) showed total unresponsiveness when the dominant hemisphere was injected. (One subjects, WF, did not have his dominant hemisphere injected.) The duration of the unresponsive period varied from 5 to 11 minutes. One of the four, JO only, became agitated. He began to groan, wail, and thrash about. Only one subject, EC, exhibited a "euphorical-maniacal" reaction (Rossi and Rosadini, 1967). None of the five subjects displayed recall or memory disturbances after the barbiturization effects wore off.

DISCUSSION

As in other amobarbital studies, the perfusion of the drug into the dominant cerebral hemisphere disrupted speech and language functioning. An interesting finding in the present investigation was the presence of dysarthria in four of the subjects, regardless of the hemisphere that was perfused. As you recall, the fifth subject exhibited dysarthria pre-injection. Previous studies (Gordon and Bogen, 1974; Werman, Christoff, and Anderson,

1959) have reported the presence of dysarthria with barbiturization, but it has not been a consistent finding, nor has the type of dysarthria been identified. Gordon and Bogen (1974) reported dysarthria only following right carotid injection. The present investigators classified the amobarbital induced dysarthria in three subjects as being of the ataxic type (Darley, Aronson, and Brown, 1975). Such a classification would lead one to speculate that the cortico-cerebellar connections or the basal ganglia were affected by the barbiturization. Three of our subjects displayed severity of dysarthria greater than would be expected with unilateral cerebral involvement. The early onset of the dysarthria makes crossfilling a questionable cause in the cases where it appeared when the nondominant hemisphere received the amobarbital. It may be that pharmacologically induced dysarthria is not the same as neuropathologically acquired dysarthria. Subject GM's dysarthria, which was present prior to the study, was probably chemically produced. Perhaps his dysarthria did not change in degree following injection because of a pharmacological saturation level.

Even though all subjects were dysarthric and aphasic, only one subject, EC, displayed apraxia of speech. This apraxia was present only during the production of longer and less frequently occurring words. The characteristics of her apraxia were compatible with those described in the literature (Darley, Aronson, and Brown, 1975; Rosenbek, 1978). Injection of amobarbital to either of the two hemispheres resulted in this speech disturbance. The appearance of apraxia following hemispheric barbiturization has not been cited elsewhere. Perhaps its presence may typically be masked by a predominating aphasia.

The one patient who displayed apraxic errors (EC) also displayed cortical stuttering. She denied any history of stuttering but reported its appearance and resolution a few weeks prior to this procedure. This behavior was produced regardless of the injected hemisphere, but was more prominent when the right side was injected. Stuttering has not been reported as a sequelae to barbiturate perfusion. On the other hand, a study by Lussenhop, Boggs, LaBrowit, and Walle (1973) found that speech fluency in chronic adult stutterers showed either no change or improvement following the injection of amobarbital into either hemisphere.

As originally conceived by Wada and his colleagues, the amobarbital test was employed for determining speech and language dominance, and most early publications report merely that patients either were or were not aphasic following injection. If our experience is typical, it would perhaps be fairer to say that patients are likely to be initially speechless and unresponsive after injection of either hemisphere. They tend to remain so longer after the dominant hemisphere has been anesthetized. We did not feel that the unresponsiveness was due to an excessive dosage of amobarbital, in view of the fact that our subjects received only 100mg of the drug. Language performance across subjects was variable. This may partly be due to the hereogeneity of our population, and the fact that all of our subjects already had brain pathology (all had tumors). Because the disruptive effect of the drug was so complete and seemed to clear so rapidly (within minutes), it was difficult for us readily to identify outright traditional aphasic symptoms. There were exceptions, as when two subjects (EC, JO) had periods of extended jargon. Unfortunately, what we judged to be aphasic performance was more frequently seen in reading. Reading errors are difficult to interpret because they may occur for so many different reasons besides aphasia.

Wada and Rasmussen (1960) described an initial brief period of confusion following barbiturate injection, but they did not define the confusional state nor did they indicate how often this occurs. JO's confusion about golf balls developed almost immediately subsequent to the injection and lasted only a few seconds. On the other hand, EC's confusion, marked by irrelevance and confabulation, developed about 6 minutes post-injection and lasted about 4 minutes. The language of confusion as described in the literature (Darley, 1969; Wertz, 1978) is said to be indicative of diffuse or bilateral involvement. Since EC's onset of confusion was late, it is speculated that the presence of the tumor on the side contralateral to the injection coupled with the injection to the uninvolved side resulted in bilateral cerebral involvement.

The finding that all four of the patients who had their dominant hemispheres injected became unresponsive to auditory and visual stimuli is compatible with the reports of Serafetinides, Hoare, and Driver (1964, 1965) who reported loss of consciousness following dominant side injection. Others, however, (Rosadini and Rossi, 1967; Rossi and Rosadini, 1967; Fedio and Weinberg, 1971) have not found this effect and instead suggest that impaired consciousness is the result of either inadvertent cross-filling of the barbiturate, or the creation of bilateral suppression due to preexisting damage on the uninjected side. In three of our four subjects, the injected hemisphere which caused the unresponsiveness was contralateral to the hemisphere containing the neoplasm.

Mood and/or emotional changes followed barbiturization in two of our subjects were similar to what Rossi and Rosadini (1967) reported. These emotional reactions included a "depressive-catastrophic" reaction, usually following left or dominant hemisphere injection, and a "euphorical-maniacal" reaction, usually following right or nondominant hemisphere injection. EC displayed a euphoric reaction following barbiturization of the dominant (right) hemisphere. Milner (in Rossi and Rosadini, 1967, p. 182) commented that the most euphoric subjects typically have frontal-lobe lesions (which EC had). Our other subject, JO, became agitated soon after injection of his left, more dominant hemisphere. Since his reaction was nonverbal, it cannot be classically described as a depressive-catastrophic state.

Based on pre-barbiturization clinical test results and/or interviews, we attempted to predict the speech and language lateralization resulting from amobarbital testing. In all five cases, our clinical findings correlated positively with the amobarbital test results for dominant hemisphere functioning. We had predicted right hemisphere dominance in two subjects, (IH, EC), left hemisphere dominance in two subjects (WF, GM), and bilateral representation in one subject (JO). An unanticipated finding was the additional utilization of the nondominant hemisphere by two subjects (WF, EC) in language processing.

Amobarbital testing continues to be a useful test of hemispheric dominance. If the protocol were expanded to include more naming, spontaneous speech, and auditory comprehension, a number of interesting questions about speech and language function and dominance might get at least partial answers. If there are surgical reasons for doing amobarbital testing, it is logical to do it with an eye also toward answering theoretical questions.

REFERENCES

Blume, W.T., Grabow, J.D., Darley, F.L., and Aronson, A.E. Intracarotid amobarbital test of language and memory before temporal lobectomy for seizure control. Neurology, 23, 812-819, 1973.

Canter, G.J. Observations on neurogenic stuttering: A contribution to differential diagnosis. British Journal of Disorders of Communication, 6, 139-143, 1971.

Darley, F.L. Aphasia: Input and output disturbances in speech and language processing. Presented in dual session on aphasia to the American Speech and Hearing Association, Chicago, Illinois, 1969.

Darley, F.L., Aronson, A.E., and Brown, J.R. Motor Speech Disorders. Philadelphia: W.B. Saunders Company, 1975.

Fedio, P. and Weinberg, L.K. Dysnomia and impairment of verbal memory following intracarotid injection of sodium amytal. Brain Research, 31, 159-168, 1971.

Gordon, H.W. and Bogen, J.E. Hemispheric lateralization of singing after intracarotid sodium amylobarbitone. Journal of Neurology, Neurosurgery, and Psychiatry, 37, 727-738, 1974.

Lussenhop, A.J., Boggs, J.S., LaBorwit, L.J., and Walle, E.L. Cerebral dominance in stutterers determined by Wada testing. Neurology, 23, 1190-1192, 1973.

Rosadini, G. and Rossi, G.F. On the suggested cerebral dominance for consciousness. Brain, 90, 101-112, 1967.

Rosenbek, J.C. Treating apraxia of speech. In D.F. Johns (Ed.), Clinical Management of Neurogenic Communicative Disorders. Boston: Little, Brown and Company, 1978.

Rossi, G.F. and Rosadini, G. Experimental analysis of cerebral dominance in man. In C.H. Millikan and F.L. Darley (Eds.), Brain Mechanisms Underlying Speech and Language. New York: Grune and Stratton, 1967.

Serafetinides, E.A., Hoare, R.D. and Driver, M.V. A modification of the intracarotid amylobarbitone test: findings about speech and consciousness. Lancet, 1, 249-250, 1964.

Serafetinides, E.A., Hoare, R.D. and Drive, M.V. Intracarotid sodium amylobarbitone and cerebral dominance for speech and consciousness, Brain, 88, 107-130, 1965.

Wada, J. and Rasmussen, T. Intracarotid injection of sodium amytal for the lateralization of cerebral speech dominance. Journal of Neurosurgery, 17, 266-282, 1960.

Werman, R., Christoff, N. and Anderson, P.J. Neurological changes with intracarotid amytal and megimide in man. Journal of Neurology, Neurosurgery, and Psychiatry, 22, 333-337, 1959.

Wertz, T. Neuropathologies of speech and language: An introduction to patient management. In D.F. Johns (Ed.), Clinical Management of Neurogenic Communicative Disorders. Boston: Little, Brown and Company, 1978.

DISCUSSION

Q: I may have missed this, but did you include EEG recordings in your protocol?

A: No, EEG was not part of this protocol. However, we are aware that other investigators have included simultaneous EEG recordings in their studies. We intend to include EEG in future studies.

Q: Section 2 of the protocol is begun when the patient is testable. If you didn't use EEG, how did you determine when the patient was ready to be tested?

A: It was obvious when the patients were not testable, because their eyes were closed and they were snoring. We attempted to arouse them continuously, and once each one became aroused, testing was initiated.

Q: I find it hard to believe that these patients displayed an ataxic dysarthria. How certain were you of this diagnosis?

A: Of the four patients that became dysarthric following injection, three of them displayed an obvious ataxic dysarthria. The fourth one was mildly dysarthric, and we could not classify his dysarthria. We unanimously agreed on the type of dysarthria.

Q: Did you include diadochokinetic speech testing to determine the type of dysarthria?

A: No, we don't feel that rapid alternate motion rates alone allow for a differential diagnosis of the dysarthrias, particularly in the case of ataxic dysarthria.

Q: Were there any motor signs present in these patients?

A: Yes, each patient displayed a hemiplegia contralateral to the side of injection. In addition, those patients who responded to the oral motor command "stick out your tongue" had tongue deviation from midline to the side of weakness.

Q: Since the speech tasks in the protocol were limited to word and phrase repetition, do you feel these were an adequate sample to type the dysarthria?

A: In addition to the repetition tasks in the protocol, each patient produced some spontaneous speech. So their spontaneous as well as their elicited speech was used to judge and classify the type of dysarthria.