IMITATION OF FACIAL MOVEMENTS IN BRAIN DAMAGED PATIENTS

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The notion of apraxia refers to the patient's inability to execute learned movements (Geschwind, 1975) particularly when a sequence of acts is required (Liepmann, 1980; Poeck and Lehmkuhl, 1980).

The disorder can be elicited by asking the patients to perform symbolic as well as non symbolic movements, on verbal command or imitation (De Renzi, Motti and Nichelli, 1980).

The specificity of the apraxic disorders was recently reconceptualized as the inability to produce a sequence of acts (Kimura and Archibald, 1974) or to control performances requiring rapid transition from one position to another (Kimura, 1982).

In a systematic study by De Renzi, Faglioni, Lodesani and Vecchi (1983) the apraxic patients failed both in imitating single postural movements of the hands as well as motor sequences, therefore these authors suggest that the central disorder deals with the ability to select a manual action from among a repertoire of motor patterns independently from the sequential characteristic.

Single case and quantitative studies (De Renzi et al., 1980) consistently supported Liepman's early notion that the gestures of both hands are controlled by a single hemisphere (generally the left) and that different areas of this hemisphere contribute to the planning and execution of the motor programs. Frontal (premotor) and parietal areas of the left hemisphere have been indicated to be involved in the control of hand and facial movements (Kolb and Milner, 1981; Archibald and Kimura, 1974; Kimura, 1982; De Renzi et al., 1983). In the case of a patient with a callosal lesion described by Geschwind and Kaplan (1962), the apraxic disorders included movements of the left leg together with movements of the left arm: nevertheless in the same patient axial and eye movements ("bend the head down", "stand", "show me the position of a boxer, "look up", "close your eyes") were generally correctly performed.

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To explain this dissociation, Geschwind (1975) suggested that while arms, legs and facial movements are predominantly controlled by the pyramidal system, axial muscles — such as the muscles of the neck, eyes, trunk — are predominantly controlled by the nonpyramidal system, which arise from multiple sites in the cortex. Following this reasoning the preservation of axial movements in apraxic patients can be attributed to the anatomic characteristics of the motor system that is not easily disconnected by central associative areas of the leading hemisphere.

The "mimic" or expressive muscles represent a special category of muscles and movements which has not been systematically considered in relation to the issue of the laterality of control.

In fact, when facial movements are considered, almost invariably this refers to 'oral movements' (Geschwind, 1975), e.g. such as movements involved in the standard testing of "oral apraxia" ("put out your tongue", "bite your teeth", "cough", "whistle" etc.). Although some tests of oral apraxia, might occasionally involve "mimic muscles", there has been no systematic investigation of the motor control of this class of movements and muscles in brain damaged patients. Thus, Nathan (1974) drew attention to the problem of facial apraxia describing four wounded cases without limb apraxia with left sided lesions: unfortunately, his clinical descriptions do not distinguish between responses to tasks similar to tests of oral apraxia, requests to intentionally produce an emotional expression or requests to move combinations of mimic muscles. Differently from axial and eye movements, the "mimic" movements are controlled by the pyramidal system and cortical lesions are known to produce, in rare cases, disordes of intentional emotional expressions, leaving the spontaneous emotional responses unmodified (Geschwind, 1975). Nevertheless without systematic studies it is not certain which neural system controls these particular facial movements.

The reason for focusing attention on facial movements is not simply a compulsive need to extend a classification to all possible actions. The neuropsychological literature provides a number of lines of evidence pointing to hemispheric specialization for the control of emotions. In this context the studies of facial movement and emotional and non emotional facial expressions are a very common object of study: the general idea was that asymmetries observed in the face of normal subjects or differential facial impairment in brain damaged patients can produce relevant information about the hemispheric control of emotions. The ideas proposed in the past fifteen years to explain this specialization can be reduced to three major theories.

(1) The first one stress the role of the right hemisphere for processing emotional experience (Gainotti, 1972; Sackeim, Gur and Saucy, 1978). (2) The second suggests, in a variety of ways, the relative importance of the

right hemisphere for negative emotions (Sackeim and Gur, 1978; Reuter, Lorenz and Davidson, 1981), inaction-avoidance behavior (Davidson and Fox, 1982; Kinsbourne and Bemporad, 1984), while the left hemisphere controls positive emotions, action-approach behaviors.

Hager and Ekman (1985) correctly point out that neither of these theories explicitly predict the side of asymmetry in facial action. (3) The third suggests that asymmetries will only be for deliberate but not for spontaneous facial movements (Ekman, Hager and Friesen, 1981).

A systematic study, in a group of normal subjects, of 14 deliberate facial actions showed that approximately one third were more intensely performed on either the left or right side of the face, but this lateralization was inconsistent with any model of hemispheric specialization (Hager and Ekman, 1985).

The neuropsychological literature on apraxia and the inferences drawn from the studies on facial movement in relation to hemispheric specialization for emotions suggested the need to undertake a systematic investigation of the imitation of a representative set of facial movements in a population of unilateral brain damaged patients to answer the following questions:

(1) Is the imitation of facial actions more impaired by left or right brain lesions?

(2) Is the impairment in facial movements particularly connected with a specific location of the lesion in either hemisphere?

(2) Is the performance in facial imitation related to other forms of apraxia?

Material and Methods

Subjects

85 right handed people of both sexes were examined. 28 were control subjects; 24 were right and 33 left brain damaged patients. 18 of the left group were aphasics (A+) and 15 non aphasics (A−). The mean age of the control group was 54.4 (S.D. 10.95); that of the right lesioned group 57.43 (S.D. 14.30), and that of the left group 56.5 (S.D. 13.1). The etiology of the brain damaged patients was vascular in 50 cases and neoplastic in 7 cases.

No bilateral lesions or patients with previous history of stroke were included. In order to participate in the research the patient had to be able to understand the task requirements conveyed either by verbal instructions or by demonstrations. This prevented the examination of patients with severe comprehension disorders in the left group.

CT scan examination was available for about 2/3 of the brain damaged group. A representation of the lesion was reconstructed on a standard lateral diagram of the brain, using the technique described by Mazzocchi and Vignolo (1979).

Tests

Imitation of facial movements

The patient was given Ekman and Friesen's (1982) Requested Facial Action test (REFACT), while seated in a sound proof room facing a motion picture film screen. This test involves showing a patient a film which depicts 14 facial actions one at a time, and asking the patient to imitate each movement. These movements include 6 in the upper face and 8 in the lower face (see Table I). These 14 actions involve most of the muscular actions which are involved in emotional expression (Ekman e Friesen, 1978). The REFACT film shows a single actor (P.E.) performing each of the facial movements. Each movement is performed two or three times rapidly and then held on the actor's face for ten seconds. As each movement is shown the experimenter verbally describes the requested action. To aid imitation the patient is told of any mistakes he or she is making during the performance, and encouraged to continue to imitate the requested action: for example, "you are lowering not raising your eyebrow", "you are also raising your upper eyelid, just raise your eyebrow".

The patient's performance must be scored by someone who has learned how to distinguish the appearance produced by the action of each of the facial muscles (Ekman and Friesen's Facial Action Coding System, 1978). A four point scale is used to record the adequacy of the patient's performance of each of the 14 actions:

(1) accurate imitation: only the requested muscular action was performed
(2) accurate imitation with the addition of "irrelevant" muscular actions
(3) accurate imitation with the addition of "relevant" muscular actions (i.e., with movements of muscles which help the p. to move the requested muscle)
(4) the requested imitation was not performed.

TABLE I

Facial Actions Presented for Imitation

Muscles involved	Description of the action
Upper face	
Inner frontalis	Raises inner corner of brow
Outer frontalis	Raises outer corner of brow
Frontalis	Raises entire brow
Corrugator procerus	Lowers and pulls brow together
Orbicularis oculi, outer portion	Squints eyes, makes crowsfeet wrinkles
Orbicularis oculi, inner portion	Squints eyes, raises and straightens lower lid
Lower face	
Depressor labii inferioris	Pull lower lip down
Zygomatic major	Common smile
Mentalis	Chin raiser
Orbicularis oris	Lip pressor
Buccinator	Dimples cheeks
Risorius	Stretches lip corners straight to the side
Levator labii superioris	Raises upper lip
Levator labii superioris, alaeque nasi	Wrinkles nose

Asymmetries in the performance of any action were also noted. Past studies using REFACT has shown high inter-rater reability in scoring facial imitations on this four point scale (Ekman, Hager and Friesen, 1981: Hager and Ekman, 1985; Ekman, Roper and Hager, 1980).

Facial paralysis

Motor impairment of the inferior facial territory (VII nerve) was evaluated by a neurologist (C.C.) in terms of the intensity of the paralysis, ranging from score 0 (no impairment) to score 3 (complete paralysis).

Neuropsychological evaluation

Neuropsychological evaluation was given to all brain damaged patients includins a test of ideomotor apraxia (De Renzi et al., 1968), a test of constructive apraxia (Gainotti et al., 1977) and a test of oral apraxia (De Renzi, Piczuro and Vignolo, 1966). The latter test was scored twice: the first score refers to the performance on verbal command (from 0 to 10): the second to the responses on imitation (from 0 to 10).

RESULTS

The results of imitation of facial movements differed depending upon the kind of score that was considered.

When the evaluation was based on the number of correct imitations of the requested movement without any additional facial action (score 1), the task was very difficult for both the control and the brain damaged groups (20-25% correct). The performances were still quite poor even when the correct performance was considered to also include the addition of non-facilitating actions (score 2). With this criterion 30-35% of the requested actions were performed correctly by the three groups.

A large number of subjects, with or without brain damage, imitated the facial movements by producing other non requested actions which facilitate the response (raise the upper lip to facilitate wrinkling the nose).

When the score included any correct execution of the requested movement, irrespective of whether it had been produced alone or with the addition of facilitating and non facilitating actions (score 3), the performances of all three groups were close to 90% correct (Table II). This is true both for upper facial movements and for lower facial movements (Table II).

The results of the entire test were analyzed by three separate analyses of variance, each based on a different score.

When more restrictive criteria were used (score 1 and score 2) no

significant difference was found. Using the most permissive score (score 3) the three groups differed significantly ($F = 4.86$, $p<.009$).

Post hoc comparisons showed that the right brain damaged patients performed significantly worse than the control and left brain damaged groups.

Similar ANOVAS were performed for the group of 6 upper face and 8 lower face movements using the three scores.

No effect was found for both groups of movements when the score 1 or score 2 were used. For both upper and lower facial movements a significant difference between the groups was present only for the most permissive score (see Table II).

TABLE II

a: Imitation Facial Movements (maximum score = 14): Means and SD (in parenthesis)

		Score1		Score 2		Score 3	
Controls		3.17 (2.12)		5.17 (2.29)		13.06 (1.23)	
RBD		2.77 (2.22)		4.43 (2.12)		11.73 (1.76)	
LBD	A+	2.75 (1.89)	2.49 (1.94)	5.08 (2.17)	4.76 (1.76)	12.69 (1.28)	12.16 (1.38)
	A−		3.06 (1.85)		5.46 (2.61)		13.32 (0.81)
		F(2, 82)=.33		F(2, 82)=.66		F(2, 81)=4.86 p<.009	
			F(3, 81)=.38		F(3, 81)=.61		F(3, 81)=5.52 p<.002

b: Imitation of Superior Facial Movements (6 items)

		Score 1		Score 2		Score 3	
Controls		1.35 (1.22)		1.85 (1.35)		5.39 (0.87)	
RBD		1.04 (1.06)		1.30 (1.10)		4.73 (1.25)	
LBD	A+	1.27 (0.97)	1.11 (1.02)	1.69 (1.18)	1.38 (0.91)	5.30 (0.76)	5.16 (0.78)
	A−		1.46 (0.91)		2.06 (1.38)		5.46 (0.74)
		F(2, 81)=.61		F(2, 81)=1.48		F(2, 81)=3.77 p<.02	
			F(3, 80)=.67		F(3, 0)=1.8		F(3, 80)=2.32 p<.08

c: Imitation of Inferior Facial Movements (3 items)

		Score 1		Score 2		Score 3	
Controls		1.75 (1.23)		3.25 (1.45)		7.67 (0.54)	
RBD		1.73 (1.45)		3.13 (1.35)		7.00 (1.12)	
LBD	A+	1.48 (1.37)	1.38 (1.24)	3.39 (1.43)	3.38 (1.24)	7.39 (0.89)	7.00 (1.02)
	A−		1.60 (1.54)		3.40 (1.68)		7.86 (0.35)
		F(2, 81)=.34		F(2, 81)=.23		F(2, 81)=4.16 p<.018	
			F(3, 80)=.30		F(3, 80)=.16		F(3, 80)=5.83 p<.018

The post hoc comparisons showed that the RBD group was significantly more impaired than both the control and LBD groups for the upper face movements. For the lower face movements the RBD group performed worse than the controls but not than LBD group.

In order to take into account the motor impairment present in some brain damaged patients in the inferior facial territory contralateral to the lesion, the comparison between LBD and RBD in imitating the facial actions was covaried for the score given to the degree of facial paralysis. The analysis of covariance did not introduce any significant correction in the results (LBD versus RBD: $F = 3.36$, $p < .07$).

When the left brain damaged group was divided into aphasic and non aphasic patients no substantial changes were obtained (see Table II).

When the responses 1 plus 2 plus 3 were considered correct the three analyses of variance for inferior, superior and the entire group of facial movements showed a significant effect; in all cases the right brain damaged (and not the aphasic group) showed a significant difference from the control group in post hoc comparisons.

Separate comparisons for each facial action showed that four actions (two of the upper and two of the lower face) were imitated more poorly by the RBD group and the controls when the most permissive scoring criterion was used: "pull the lower lips downwards", "stretch the lips horizontally", "raise the upper eyelids as high as possible", "tighten the lower eyelids" (see Table I for the muscles involved in each request). No effect of aphasia was found when the three groups were considered separately for any of the requested actions using any scoring criteria.

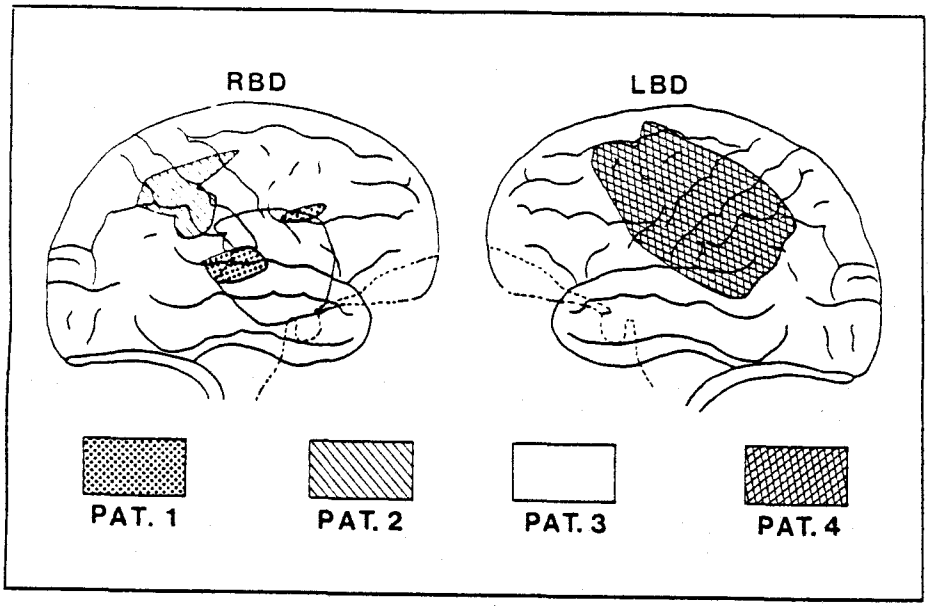
A separate analysis was performed with regard to the time required to produce a correct performance of the facial actions, independent of the obtained score (see Table III). A significant effect of time was found ($F = 3.07$, $p. < 03$) indicating that the RDB patients were significantly slower than controls and LBD patients in producing the requested action.

TABLE III

Latency (in sec.) of the Groups of Subjects to Achieve the Best Imitation of the Facial Movements (14 items): Means and S.D. (in parenthesis)

Controls	2.54 (.46)	
RBD	3.07 (.88)	
	A+	2.57 (.78)
LBD	2.52 (.63)	
	A−	2.46 (.42)
$F(2, 81) = 7.71$	$F(3, 81) = 3.08$	
$p < .001$	$p < .03$	

RBD = right brain damaged; LBD = left brain damaged;
A+ = aphasic; A- = non aphasic



Further analysis of each individual action unit showed that only the latency to produce "nose wrinkling" was significantly longer for RBD and aphasic group.

In order to find out whether it is possible to identify a subsgroup of patients with a form of apraxia for mimic movements, a cut-off score to separate normal and pathological performances was computed.

Considering the correct responses according to the more restrictive criteria (score 1 and 2), it was not possible to devise a useful cut-off score since many control subjects scored very low. Using the score 3, the worst performance of controls was 10 out of 14 correct imitations. Three RBD and one LBD patients scored below the cut-off score of 10 (9.8, 7 and 9 respectively). The lesions of the four patients are reported in Figure 1. The marginal difference between the performances of RBD, LBD and controls is quite remarkable as compared to the substantial discrepancies between normal and pathological performances described in any form of apraxia.

Relation with other forms of apraxia

The performances of BD patients in the facial imitation task and oral, ideomotor and constructive apraxias were compared by means of chi^2.

TABLE IV

Contingency Table for Presence/Absence of Different Forms of Apraxia and the Performance on the Task of Facial Imitation

	Imitation of facial movements			
	Cut-off	above	below	
Oral apraxia =58	(9) above	40	3	$chi^2=0.001$
	below	14	1	$p<.45$
				$C=.005$
Ideomotor apraxia N=36	(12) above	27	4	$chi^2=0.75$
	below	5	0	$p<.40$
				$C=.140$
Constructional apraxia N=44	(66) above	34	4	$chi^2=0.69$
	below	6	0	$p<.41$
				$C=.124$

The BD patients below the cut-off score in the test of oral apraxia (two or more errors in imitating oral movements) were 14: all of them were LBD patients. Table IV shows that only one patient below the cut-off in the test of facial movement imitation (score 3) also has oral apraxia.

The dissociation between the two task of imitation of oral and facial movements points to a substantial independence of the clinical occurrence of these symptoms.

Similarly the analyses in Table IV point to the independence between ideomotor apraxia, constructive apraxia and the imitation of facial movements.

Localization and size of the lesion

The CT score was available for 40 BD patients (17 RBD), 15 LBD A+ and 8 LBD A−).

Two types of analysis were based on the lateral reconstruction of the lesion: one relates to the size and one to the location of the lesion in different lobes.

The size of the lesions was evaluated as large, medium and small: the relative distribution to the left and right hemispheres are represented in Table V.

There was no difference between the size of the lesion in the two hemispheric groups.

The relationship with the size of the lesion shows that, independently of the three kind of score used, large and medium size lesions tend to produce lower performances then small lesions, but in no case were the

TABLE V

Size of the Lesion in RBD and LBD

		Size			
		small	medium	large	
RBD		0	8	9	
LBD	A+	3	5	7	
		5	9	9	$chi^2=4.25$ ($p=.12$)
	A−	2	4	2	$C=.31$

contingency coefficient significant.

The relevance of the location of the lesion was assessed by comparing the performance on the facial imitation task of the patients with specific lobe involvement with that of all patients without a lesion in that lobe (i.e. patients with a parietal lobe vs. all patients without a parietal lobe impairment). Although the number of patients with single lobe involvement was small for any definitive conclusion, none of these analyses pointed to any specific location producing greater impairment in the task.

It is concluded that the size of the lesion might play a marginal role in affecting BD performance on the facial imitation task, while there are no indications that the specific location of the lesion to be more important for this task. Finally the lesion location for the four patients below the worst controls are shown in Figure 1.

The variety of the locations does not suggest any priviledged locus for a form of facial apraxia.

DISCUSSION

The most clear result is that an apparently simple task, such as imitating facial mimic actions, is in fact difficult to be performed by both normal and brain damaged subjects.

Within the brain damaged group, the presence of a lesion in the left or right hemisphere does not produce any asymmetrical results in imitating facial movements, contrary to what is consistently observed for oral and limb movements even if of a very simple degree of complexity or for deliberate facial expressions (Ekman et al., 1981).

The difficulty in producing mimic movements in isolation cannot be attributed to the difficulty in understanding the task. Since aphasic patients did not have greater problems than other BD to perform the task.

At first glance, the imitation of single movements of the face may seem as a more unusual task than the other ones requested for testing different forms of apraxia. However, commands such as "raise the eye brows" or "pull up your lips" are not more unusual than "touch your nose with the tip of your tongue" or "close fist, thumb sideways on table, open hand, slop down on table". In any case, this does not account for the lack of asymmetry for any of the large set of movements included, some of which are certainly "easier" and less uncommon (wrinkle your nose) than others (raise just the outer part of the eye brows upward).

It is important to notice that this conclusion extends to a large variety of isolated mimic movements (about half of all possible movements) and equally applies to actions produced by muscles which receive bilateral (upper face) as well as contralateral (lower part) innervation.

Another relevant objection to the conclusion is that, accepting only correct imitation of a single movement without any additional one, kept the average performance of all groups of subjects, including the normal controls, very poor, so that the lack of differences between groups might be due to a floor effect.

Nevertheless if one accepts as correct, the imitation of a single movement even when it is produced together with non facilitating movements, the means of the different groups approached 35% correct responses, and the lack of between group differences was still evident.

The pattern of results considerably changed when a more permissive score was accepted, not penalizing subjects who added facilitating movements. In this case the task becomes relatively easy for all groups and, in different analysis, a numerically small but significant difference emerged, pointing to a poorer performance by the RBD patients.

The first comment on these results is that, in spite of the fact that several individual facial movements can be spontaneously produced in isolation, it seems difficult to segregate and evoke them intentionally.

The difficulty in producing fine, isolated movements of the face should be greater for the bilaterally innervated muscles (superior face) than the contralaterally innervated ones (inferior face). The muscles that manipulate the lips receive a much greater contingent of fibers than the corrugator or the frontalis (Rinn. 1984). In spite of these anatomical and functional considerations, the present data do not show any difference in the use of facilitating movements of either part of the face. This result remains true even when the produced action units are treated one by one. Data collected on normal subjects, both adults and children (Hager and Ekman, 1985, and Ekman, Roper and Hager, 1980) confirm the absence of this relation.

A second point is that while normal controls and LBD can easily pose many facial movements in combination with other frequently associated

actions (raise the eye brows together with opening the eyes), the RBD patients are not as able to make use of such facilitating actions.

There was a tendency for the right lesion to be greater then the left hemisphere lesions, which did not reach statistical significance. In addition, the size of the lesion does not correlate with the performance in imitating facial movements. It cannot therefore be concluded that the size of the lesion is entirely responsible for the poorer capacity the RBD patients to make use of facilitating strategies in imitating facial movements, although it might reasonably contribute with some other factor which did not emerge in the present study.

Another interesting point concerns the strong dissociation between the imitation of mimic movements and other forms of apraxia. It seems clear that the control of mimic facial movements is quite independent from the control of oral movements. The high frequency of oral apraxia with left lesions contrasts with the symmetrical results in facial imitation using a strict criterion or a tendency toward right hemisphere control accepting a more permissive scoring criterion. A similar lack of relation was also evident for ideomotor and constructive apraxia.

More interesting at this point is to consider the difficulty with any of the criteria used, to find patients with severe impairments in imitating mimic movements in a relatively large population of BD with a variety of lesions of different size and location.

One possibility is that this is due to the lack of sensitivity of the task, too difficult when strict criteria are used and too easy when more flexible criteria are adopted.

However it may be observed that other studies have been able to identify pathological performances in different praxic tasks, which had performance levels similar to that of our flexible criterion.

For example, studying finger and hand movements imitation, De Renzi et al. (1980) and De Renzi et al. (1983) developed tasks which were very easy for normal controls (from 77% to 95% correct). Even with tasks of difficulty comparable with the present one these A.A. were able to find approximately 60 to 70% LBD aphasic and 43% LBD respectively performing below the cutting score. Some of their patients did show markedly impaired performance, clinically identifiable as defective in comparison with the controls. Similarly Kimura (1982) in a task of single oral movement imitation with a high overall percent correct in the LBD group, was still able to identify significantly impaired performance in the frontal lesioned group.

In the present study only four cases below the cut off score were identified, with the more permissive criterion; in all cases their scores were marginally different from the worst control's performance and no relation was evident with either side, size or locus of the lesion. Therefore it seems

reasonable to conclude that there is insufficent evidence to postulate a form of apraxia for the facial mimic movements.

Recollecting the different aspects that have been discussed, the following conclusions can be drawn.

(1) Focal brain lesions of either hemisphere do not produce differences in imitating isolated facial movements: moreover the capacity of all BD patients is indistinguishable from that of normal controls in segregating individual bilateral contraction of the mimic muscles on imitation.

Controls and LBD improve their performance in this task using facilitating movements when requested to intentionally reproduce "mimic" muscle contractions: RBD patients seem to be slightly less efficient in making use of these facilitation. In addition RBD patients are slower in producing appropriate imitation of facial movements.

(2) The imitation of facial movements in BD patients shows no relation with the performance in other tasks of oral and limb apraxia. This conclusion is particularly evident considering the double dissociation between the poorer performance given to the facial imitation and oral apraxic task. In addition it was very difficult to identify in the present population clearly pathological performance on imitation of facial movements; this finding contrasts with the greater occurrence of clinically evident deficits of oral, ideomotor and constructive apraxia in the same population of brain damaged patients.

(3) The task of mimic facial imitation is not selectively impaired by focal cortical and subcortical lesions in different location on both sides of the brain: the size of the lesion is not correlated with the degree of impairment. This set of data suggests that, differently from limbs and oral movements, the control of the facial mimic movements might be partly dependent upon the pyramidal system, but in addition might have a diffuse and multiple representation in the nonpyramidal system, similarly to what was hypothesized by Geschwind (1975) for the central axial and extrinsic eye movements.

This suggestion would account for the rare clinical descriptions of this selective impairment in patients with hemispheric focal lesions. The present findings are also relevant to the question of whether asymmetries in the production of facial expressions should be attributed to differential involvement of the two hemispheres in the direction of facial expressions. The results were not consistent with the view that the right hemisphere is more involved in the production of all facial muscular movements (Sackeim, Gur and Saucy 1978), but instead were congruent with Hager and Ekman's (1985) findings that asymmetries in expression can not be attributed to cerebral asymmetry in production.

ABSTRACT

The imitation of a large repertoire of upper and lower facial actions was requested from a group of left, right brain damaged and control subjects in order to explore: (1) if a left or right hemispheric focal lesion produces a similar pattern of impairment on this task; (2) if the impairment is associated with oral apraxia.

The results show that left and right brain damaged patients score significantly lower than controls but the two pathologic groups do not differ from each other.

The imitation of facial movements is not related in any way to oral apraxia and no specific localization of the lesion seems responsible for the impairment in this task.

It is suggested that the control of facial movements might have a multiple representation in either hemisphere.

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