Buccofacial Apraxia and the Expression of Emotion

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Apraxia is a disorder in executing "learned"[1] or "skilled" movement (excluding explicitly symbolic movements[2]) not accounted for by weakness, incoordination, sensory loss, incomprehension or inattention to commands.[3, 4] In a classic case, the patient carried out commands such as, "show me how you would use a hammer" with his right arm without hesitation.[1] However, when asked to perform the same actions with his left arm, the patient would either do nothing or make an obviously incorrect response. When given real objects, the patient showed no deficits with either arm. The fact that the right arm performed the actions normally rules out the possibility of incomprehension, inattention, or uncooperativeness; while the successful performance of the left arm with real objects rules out the possibility of weakness or incoordination. This particular pattern of deficits is now called *ideomotor apraxia*.

When patients have damage centered in the premotor area of the left frontal lobe, they may exhibit limb apraxia (LA) but may also show apraxia of the face, so-called *buccofacial apraxia* (BFA). These patients cannot pretend or imitate facial movements such as blowing out a match or sucking through a straw, but they usually perform normally when presented with a real match or straw. BFA, while fairly common, has been less studied than limb apraxia and has seldom been related to facial emotion expression.

Care must be taken to separate BFA from automato-voluntary dissociation (AVD) affecting the face. In AVD, corticospinal innervation of the face is im-

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paired, leading to loss of voluntary control of the face, while "automatic" emotional responses such as crying and smiling, presumably subserved by subcortical pathways, remain intact. This leads to an inability to voluntarily move the face or pose emotional faces while leaving spontaneous emotional facial movements and automatic facial movements intact. Bilateral lesions of the frontal operculum can lead to this syndrome.[5] The separation of neural circuits for voluntary and involuntary control of the face is supported by numerous lines of evidence including the existence of lesions that selectively impair either system and the decreased involuntary emotional facial displays with relative sparing of voluntary facial posing exhibited by patients with Parkinson's disease and other basal ganglia dysfunction.[3,6–9] Furthermore, patients whose severed facial nerves (VII) have been reattached to their accessory nerves (XI) recover voluntary, but not spontaneous, emotional control.[9] Because they fail on tests of praxis, patients with AVD are sometimes labeled as having BFA.[4,10] In contrast to those affected by BFA, AVD patients have paralysis of voluntary facial movement and thus lack any deficit specific to planning complex movement. Therefore, patients with AVD should not be considered apraxic.

To show that a patient has BFA, one must show that (1) the patient is not paralyzed for voluntary movements and (2) the patient has deficits specific to pretending and or imitating movements with the face. Merely observing that the patient cannot perform facial movements to command or imitation but does not exhibit total facial paralysis is insufficient to diagnose BFA, since casual observation cannot separate voluntary from involuntary movements. Asking the patient to perform buccofacial tasks with real objects such as sucking on a straw formally rules out AVD.

Most studies implicate the left frontal operculum and surrounding tissue to be important for BFA (refs. 3, 10–15; but see 16, 17). These areas include the premotor cortex for the face, an area known to be important for planning deliberate facial movement. Raade *et al*. explicitly investigated the relationship between limb apraxia and BFA in patients with single left-sided strokes.[11] While eight subjects exhibited both LA and BFA, three patients had BFA without LA, and three patients had LA without BFA. Furthermore, both the nature of errors and the lesion locations differed significantly between subjects with LA and BFA. The authors conclude that the underlying neural substrates of LA and BFA are at least partially distinct.

The relationship between BFA and emotion has rarely been studied. One study took 27 brain-damaged males and asked them to make 6 facial movements, first with a neutral command followed by an emotional command.[18] The neutral commands included "close one eye" and "put your tongue out," while the corresponding emotional commands were "close one eye like a wink" and "stick your tongue out like you are making a face at me." Commands were presented in a fixed order: 6 neutral commands, then 4 buccofacial tasks, followed by 6 emotional commands. Left brain–damaged patients

(LBD) exhibited BFA; all groups improved with emotional cueing, but the LBD group showed a significantly larger improvement.

The authors accept that their study design did not separate the effects of cueing from task order. However, they claim that the improved response of patients with BFA to emotional cueing is different from apraxic patients' known lack of improvement with repeated trials.[19] They also suggest that since the right hemisphere may have a "special role in emotion processing, it may [be] mediating the facilitation effect."[20] Given more recent evidence showing no particular connection between right or left hemisphere damage with deficits in spontaneous or voluntary expression of emotions,[21,22] this hypothesis seems unlikely. Furthermore, the authors note that context may have provided some sort of general facilitation. This seems probable given that the emotional commands contained within them the neutral commands; replacing "close one eye like a wink" with "wink your eye" would control for patients' possessing more information about the intended movement.

A more serious concern is the questionable relationship between commands such as "close one eye like a wink" and emotion. These commands could be taken as merely descriptive. Winking, for instance, can be considered emotional in the context of flirting and nonemotional in the context of having something caught in your eye. Performance for each item was not reported separately, preventing the evaluation of this hypothesis. These experimental limitations prevent strong conclusions from being drawn from this study, although the data suggest that emotional information can improve apraxic performance.

BFA is usually assessed by asking the patient to make simple single movements. However, most authors do not take into account the actual functional anatomy of the facial musculature when they define "single movements." For example, Mater and Kimura included "upper teeth on lower lip," while De Renzi *et al.* included "give a 'Bronx cheer' or 'raspberry' " as "single movements."[4,15] However, according to Ekman and Frieson's Facial Action Coding System (FACS, a system whereby all individual movements of the face can be objectively measured), each of these movements includes more than one action unit (AU, the simplest unit of facial movement).[23] Few studies of BFA have directly studied the relationship between patients' ability to make single AUs and to make these "simple movements."

One study of 57 stroke patients addressed the relationship between the ability to imitate 14 single AUs, including items such as raising the inner and outer eyebrows separately, and BFA. Subjects were shown videos of actors posing the various facial movements and were given verbal encouragement such as "you are lowering not raising your eyebrow."[24] Subjects were given three scores for making the movement: (1) absent all other movements, (2) associated with extraneous movements, and (3) associated with other facilitating movements. For all groups (LBN, RBN, and controls) the first two scores were low (20–25% and 30–35%), while the third score was high

(90%). Surprisingly, brain damage had no significant effect on either of the first two scores, while RBN showed a marginal but significant impairment in imitation with facilitation. Furthermore, deficits in AU imitation showed no relationship to BFA or other types of apraxia. However, only four individuals in the entire study showed deficits in imitation. This study suggests that the ability to imitate single facial movements (AUs) is more resistant to focal damage than the ability to pretend or imitate more complex movement combinations that may require learning. The authors conclude that the "control of facial mimic movements might be partly dependent on the pyramidal system, but in addition might have a diffuse and multiple representation in the nonpyramidal system, similarly to what has been proposed[1]... for the central axial and extrinsic eye movements."

If patients with BFA can perform single AUs, then why do they fail on tasks of BFA that require only a single AU? Several of the items in the test of BFA used in this study[24] such as "show how you would kiss someone" and "puff or blow," have corresponding single AUs (AUs 18 and 34, respectively). Unfortunately, this study[24] cannot address this issue since these AUs (18 and 34) were not evaluated during the AU imitation portion of the study. Furthermore, not all of the items on the test of BFA had corresponding AUs, and performance for each item was not reported separately. Further confusing the issue, Borod *et al.* used "pucker your lips like a kiss" for their emotional command 18, whereas the present study of Pizzamiglio *et al.*[24] used "show how you would kiss someone" as one of the items on the test of BFA.

Perhaps the discrepancy in behavioral deficits is due to significant differences in the procedure for eliciting the facial movements. For the test of individual AUs, the patients saw a video and were given verbal encouragement. For the test of BFA, patients were given a verbal command and then, if necessary, shown the proper movement. The verbal encouragement in particular could have given the patients the extra information necessary for successful facial posing. This methodological concern could be addressed by systematically altering the procedures used to elicit identical facial movements. This would allow the exact deficit in BFA and its relation to the ability to make single movements to be ascertained. For example, for the pucker (AU 18), a patient could be asked to "pucker your lips" as a simple anatomical command. Then the patient could be asked to "show how you would kiss someone." This command should activate stored, possibly learned, motor programs. Finally, the patient could be asked to "show how you would suck on a straw" and then given a real straw and asked to repeat the movement. These commands would presumably engage a stored motor program involving tool use. By using this research methodology, the target motor output would be held constant while the method for eliciting the movement is systematically varied. This would allow the specific nature of the deficit in BFA to be investigated (TABLE 1).

TABLE 1. Facial action units

Action Unit	Anatomical Command	Nonemotional Motor Program	Nonemotional Motor Program Utilizing Tools	Emotional Motor Program
1+2	Raise your eyebrows.	Make your forehead wrinkle.		Move your eyebrows as if you are surprised.
4	Lower your eyebrows.	Move your eyebrows as if you are concentrating.		
	Bring your eyebrows together.	Move your eyebrows as if the sun is in your eyes.		Move your eyebrows as if you are angry.
5	Raise your eyelids.			Move your eyelids as if you are surprised.
	Open your eyes wide.			
7	Tighten your eye-lids without closing your eyes.	Move your eyelids as if the sun is in your eyes.		Move your eyelids as if you are angry.
		Squint.		
		Move your eyelids as if you are trying to see something far away.		
9	Wrinkle your nose.	Move your nose as if you have smelled something bad.		Move your nose as if you are disgusted.
12	Pull the corner of your lips up.			Move your lips as if you are happy.
15	Pull the corner of your lips down.			Move your lips as if you are sad.
17	Push your lower lip up.			Pout.
18	Purse your lips.	Show how you would kiss someone.	Show how you would suck through a straw.	
	Pucker your lips.	Show how you would whistle.	Show how you would blow out a match.	
	Make your lips as small as you can in the shape of an "O" and push them slightly forward.			
27+25	Lower your jaw and part your lips.	Pretend to say "Ah."		Move your mouth as if you are surprised.
	Open your mouth.	Yawn as if sleepy.		

The same patients from the Pizzamiglio *et al.* study[24] were asked to pose the six commonly accepted basic emotions, and their attempts were both FACS coded and judged by naïve observers.[21] No differences were found between controls and brain-damaged patients in the appropriateness of their posed facial displays. Furthermore, while no groups showed deficits in posing, roughly half of the LBDs had BFA; no correlation was found between the presence of BFA and the ability to perform facial expression. These data suggest that BFA and emotional face posing do not share a neural substrate.

BFA is a common neurological disorder that affects the ability to make some voluntary movements with the face to command or imitation. Involuntary facial movements such as smiling at a joke or coughing, imitation of individual facial movements, and voluntary emotional facial displays are apparently spared in BFA. The definitions of these categories of facial movements have not always been carefully delineated, and much confusion exists as to how to differentiate among them. A promising avenue of future research is to address the salient features of these categories and how they relate to BFA. While the current data remain incomplete, available evidence suggests that there are at least three distinct neural circuits innervating the face. These include a subcortical circuit subserving spontaneous emotional expression, a presumably multiply represented circuit subserving voluntary emotional expression, and a circuit that contains the left frontal cortical lobe that subserves complex and/or learned movements, which is disrupted in BFA. Knowledge of these separate circuits allows for more precise investigation and classification of patients with facial movement dysfunction and direct testing of this schema. This will allow a deeper understanding of the functional neuroanatomy of the neural circuits innervating the face.

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