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Reference

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Investigating the production of emotional facial expressions: a combined electroencephalographic (EEG) and electromyographic (EMG) approach

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Abstract
Facial expressions are part of emotional reactions. However, humans can voluntarily pose a specific emotional expression without having the corresponding underlying feeling, or voluntarily modify (e.g. reduce or enhance) their spontaneous expression in reaction to an emotional event. Few studies have attempted to distinguish these different processes at the level of the central nervous system (CNS), even though spontaneous and voluntary facial expressions are long thought to rely upon distinct neural circuitries. Here, we review the neural bases of spontaneous and voluntary facial expressions, report the results of a first study assessing the Bereitschaftspotential (BP) before voluntary smiles, and outline a combined EEG/EMG approach for investigating facial expressions at the level of the CNS.

1 Introduction
Facial expressions are part of emotional episodes [13; 33], and can be used from other individuals to infer the emotional state of the sender, as well as possible threats or resources in the environment. Besides humans, emotional facial expressions can be found in many animal species, and especially in primates [4]. However, most of us are, at different degrees of proficiency, able to voluntarily produce – even without experiencing the corresponding underlying emotion – facial expressions that resemble those typically triggered by emotions, so-called posed emotional facial expressions. Although even a greater number of types of facial expressions may exist, and everyday life expressions are likely to contain volitional as well as emotional elements, we will focus for the purpose of this paper upon the differences between spontaneous, emotional facial expressions on the one hand, and voluntary, posed facial expressions on the other hand.

2 Types of facial expressions
From a theoretical point of view, facial expressions can be either totally spontaneous, i.e. triggered by an emotional stimulus, and free of any form of (voluntary) control, or the opposite, i.e. produced on a 100% voluntary basis, without being influenced by any emotion whatsoever. Of course, this dichotomy is somewhat artificial, and naturally occurring facial expressions are more likely to be made out of blends of spontaneous and volitional behaviors. Nevertheless, brain damage studies and tracing studies in non-human primates, as well as neuroanatomic, neurophysiologic, and neuroimaging approaches carried out on human participants suggest that the dichotomy between spontaneous and voluntary facial expressions may also be true in terms of neural substrates. For example, the existence of a double dissociation between emotional and volitional facial paresis (see below) suggests that spontaneous and voluntary facial movements rely upon, at least partly, separate neural pathways. The distinction between spontaneous and voluntary facial expressions becomes even more complex when taking into account those facial expressions that are spontaneously triggered by an emotional event but are subsequently modified (e.g. diminished in amplitude or shortened in duration) by the person’s desire to display a certain feeling differently in a particular social context, or not to reveal it at all. This latter type of behavior falls under the broad category of emotion regulation strategies, the characteristics and neural bases of which have recently become of great scientific interest to the psychological and neuroscientific community [13; 27]. Another phenomenon that might be relevant to the topic of emotional facial expression is called facial mimicry and stands for the observation that people tend to unconsciously mimic the facial expressions they perceive in others, so that they slightly smile when seeing a smiling person, and frown when seeing an angry face [5].

Differences between spontaneous and posed emotional facial expressions have been investigated by several authors using mainly visual inspection [6]. Spontaneous emotional facial expressions are often conceptualized as the result of neuromotor programs inducing smooth and ballistic movements and thus show less irregularities (pauses and stepwise intensity changes), than expressions that are posed [7; 14]. Facial electromyography (EMG) consists of recording motor unit action potentials of the facial muscles by placing electrodes on the
surface of the skin. This type of measure is able to reveal muscle contractions that are too tiny to be spotted by eye. Unfortunately, it has rarely been used for comparing spontaneous and posed facial expressions. Furthermore, when EMG has been used for this purpose [e.g. 35], muscular activity was averaged over periods of several seconds, notwithstanding the fact that EMG’s high temporal resolution (allowing the precise determination of for example activity onset, apex, and offset) is one of its main assets.

Facial EMG has however been used frequently for studying unconscious facial mimicry in healthy subjects [1; 5], as well as in autistic patients [23]. In these studies participants typically show small contractions in their face of the same muscles they are seeing contracted in another face. Thus, observing smiling faces leads to greater EMG activity of one’s zygomaticus muscles, while observing angry facial expressions leads to increased contraction of one’s corrugator muscles.

In summary, few studies have looked into the distinction between spontaneous and voluntary facial expressions. In most cases, facial expression was analyzed via visual inspection of video recordings.

3 The neural bases of facial expressions

Most facial muscles contributing to facial expressions attach to the facial skin or fascia, a subcutaneous sheet of fibrous material, and are innervated by the 7th cranial nerve, the facial nerve [22; 31]. In contrast, the temporalis, masseter, internal and external pterygoid muscles are innervated by the 5th cranial nerve, also called the trigeminal nerve, attach to bone and ligament, and contribute to moving the mandibles [30]. The muscles moving the eyes, partly contributing to facial expressions, are innervated by the 3rd, 4th, and 6th cranial nerves [22]. In the remainder of the text, we will focus on the activity of the 40 or so muscles innervated by the facial nerve, as they play the main role in the production of emotional facial expressions.

Ultimately, facial expressions take their origin in motor neurons of the nuclei of the facial nerves (the facial nuclei), located in the brainstem, in the ventrolateral region of the inferior pons [30]. Neural motor impulses from the facial nuclei reach the facial muscles via the fibers of the facial nerves leaving the skull from a hole called the stylomastoid foramen, just in front of each ear, and then divide into five major branches [30]. The anatomical organization of the motor neurons in the facial nucleus is not random, and contains a topographical representation of the facial nerve branches [24; 25]. The facial nucleus is similar across man, non-human primates, and lower animals (rabbits, dogs, cats), with the difference that it contains more neurons innervating muscles of the mouth and lower face in man, allowing for a high degree of fine and controlled movement as required for speech, and more neurons innervating the upper face and auricular muscles – that allow ear movements as part of the orienting response – in lower mammals [30].

3.1 Emotional and voluntary facial expressions

Interestingly, emotional and voluntary facial expressions seem to arise, at least partly, from different neural circuitries. This hypothesis stems mainly from the neurological observation of a double dissociation between emotional facial paresis (EFP) and voluntary facial paresis (VFP) [16]. EFP is a neurological disorder leading to the loss of the capacity to show emotional facial expressions (e.g. smiling to a joke), often on one side of the face, in the absence of any impairment of the ability to contract facial muscles voluntarily. EFP has been reported following lesions of the thalamus, the striatocapsular area, the frontal subcortical white matter, the insula, the medial frontal lobe including the supplementary motor area (SMA), or the dorsolateral pontine tegmentum area [16; 17; 25]. The more commonly observed VFP leads to difficulties in moving facial muscles of the lower face voluntarily, but does not affect a person’s emotional facial expressions. VFP is commonly observed with lesions of contralateral M1 and/or lateral premotor cortex (LPMC) or along the corticobulbar tract descending to the facial nucleus.

According to the mainstream conceptualization of the neural bases of facial expression [12; 29], voluntary movements of the lower-face muscles (e.g. allowing mouth movements) originate in contralateral cortical motor areas – the primary motor cortex (M1) and the LPMC, located on the lateral part of each hemisphere, just rostral to the central sulcus – and travel to the facial nucleus that is ipsilateral to the facial movement via the so-called direct pyramidal or corticobulbar tract. Voluntary movements of the upper face take their origin in the same cortical areas, but innervate both the ipsi and contralateral facial nuclei. The rather unilateral cortical innervation of the lower face and the bilateral control of the upper face may explain why most people can easily move only one corner of their mouth, while it is more challenging to move just one eyebrow. Moreover, a unilateral lesion of M1 and/or LPMC, caused for example by a middle cerebral artery infarction, affects most often muscles of the lower and contralateral part of the face, while sparing upper-face movements that can still originate in the healthy M1/LPMC [25; 30]. In the same view spontaneous facial expressions stem from subcortical structures including the basal ganglia, innervating both ipsi and contralateral facial nuclei via extrapyramidal tracts passing through the brainstem reticular formation. This hypothesis is
supported by the finding that decerebrated rats as well as human ancephalic newborns can produce some rudimental facial expressions in response to taste and tactile stimuli [9; 30]. Indeed, in addition to the direct pyramidal tract, cortical motor areas also project bilaterally onto the facial nuclei via interneurons of the reticular formation [30]. These indirect connections may allow for subcortical modulation of voluntary motor commands, particularly from the basal ganglia, hypothalamus, and periaqueductal gray [30; 36]. Interestingly, the indirect pathways influence mainly motor neurons of the facial nucleus that control the upper face [30].

However, as recent tracing studies in nonhuman primates suggest, at least five cortical motor areas project directly onto the facial nuclei [25]. These are the laterally lying M1 and LPMC, the more medially and rostrally situated SMA, as well as a rostral (CMCr) and a caudal motor cortex (CMCc) on the dorsal edge of the cingulate cortex. While M1, LPMC and CMCc project mainly to the contralateral facial nucleus via the direct corticobulbar tract, and here mainly to the motor neurons of the lower-face muscles (Figure 1), the SMA and CMCr synapse onto ipsi- and contralateral upper-face motor neurons of both facial nuclei [25]. Despite this pattern of main projections, all five cortical face representations actually innervate all subdivisions of the facial nucleus, and thus muscles of the upper as well as of the lower face, to some degree [24].

Interestingly, the cingulate cortex, which is typically been viewed as an important component of the limbic system, and which hosts the CMCr and CMCc on its dorsal edge, receives many projections from the prefrontal cortex and structures like the amygdala that are relevant for emotional processes. These connections might permit for one more way – besides their projections onto the reticular-formation interneurons of the indirect corticobulbar tract – of how emotional processes influence the motor system [15; 24; 26]. Reports of blunted emotional expression in the upper face following anterior cingulate cortex (ACC) damage [3] may thus be deriving from a disconnection of the CMCr with the limbic lobe and amygdala [24], or by the disruption of the ACC altogether, proposed to be the source of emotional facial movement by Damasio [3]. Importantly, Morecraft and colleagues [24; 25] suggest that the phenomenon of intact emotional lower-face movement after lesion of the contralateral M1 and LPMC may be attributable to sparing of the CMCc. In contrast, the traditional account outlined above rather supposes subcortical structures to be the basis of spared emotional lower-face movements after unilateral lesion of the corticobulbar tract. Finally, structures interconnected with the cortical motor areas, like the thalamus, the insula, and the temporal lobe, may also contribute to the generation and execution of voluntary and emotional facial expressions [24].

Recently, the topic of the brain activations underlying spontaneous and voluntary facial expressions has started being addressed with non-invasive neuroimaging techniques. For example, Iwase et al. [17] employed positron emission tomography (PET) and Wild et al. [37] used functional magnetic resonance imaging (fMRI) to investigate spontaneous smiling/laughter in response to amusing film clips and cartoons. Their results mostly confirm the hypotheses made on the basis of neurological and animal studies. For example, lateral somatomotor areas including M1 were found to be more activated during voluntary smiles. In contrast, spontaneous smiling led to activation of more rostral and medial cortical structures, such as the pre-SMA and the cingulate motor areas, as well as of subcortical structures including the basal ganglia and
amygdala. Somewhat surprising however may be the finding of greater SMA activation during voluntary compared to spontaneous smiling.

According to a large corpus of studies, the right cerebral hemisphere may be dominant for the perception of emotional stimuli [11]. In this line of thinking, the hypothesis can be made that the right hemisphere may also be at the source of the production of emotional facial expressions. This conception receives partly support by chimeric faces studies manipulating images with two right or two left sides of the face together. People tend to rate the resulting emotional expression as being more intense in the case of the composite picture containing two left halves of the original face [32]. However, this line of research is based on posed emotional facial expressions and thus, if ever, may only inform us about the hemispheric dominance concerning voluntary facial movements, which moreover are not generated strictly contralaterally, as especially the upper-face muscles are controlled from both the ipsi and contralateral hemispheres. Furthermore, the asymmetry found for posed emotional facial expressions is far less evident, or becomes totally absent, when facial expressions are generated spontaneously [8]. Finally, contrasting the hypothesis of a right-hemisphere dominance in the production of posed facial expressions, Gazzaniga and Smylie [12] propose — based on the study of split-brain patients — a model of the pattern of neural innervation, in which voluntary facial expressions are mainly carried out by the motor and premotor cortices of the left hemisphere.

Finally, two independent case studies of young epileptic patients with implanted electrode grids [10] and depth electrodes [20] may inform us about the role of cortical areas in the production of emotional facial expressions. The left prefrontal cortex comprising and neighboring the pre-SMA (rostral to the SMA), was electrically stimulated in these patients in an attempt to locate the seizure focus. Interestingly, smiling and laughter repeatedly accompanied stimulation of the left pre-SMA, while stimulation of adjacent contacts produced mainly speech arrest or motor movements of the extremities. These findings are in agreement with Iwase and colleagues’ [17, see above] finding of bilateral pre-SMA activation during spontaneous, humor-induced smiling in healthy participants. Importantly, smiling was produced by lower currents than laughter, suggesting “that smiling and laughter might involve similar mechanisms and are closely related phenomena on a single continuum” [10]. It remains unclear on the basis of these two studies, whether smiling and laughter induced through pre-SMA stimulation are accompanied by genuine feelings of merriment or mirth. Both patients reported such sensations, but for one of them they went together with discomfort, and appeared only after a first phase in which she felt her lip corners elevating in a forced smile [20]. Scalp EEG recorded in one of the patients during implicit processing of pictures depicting emotional facial expressions showed greater amplitudes starting at 150 ms after stimulus onset for happy facial expressions [20]. At first sight these findings are not obvious to interpret on the basis of the model sketched above. It may for example surprise that electrically stimulating the left pre-SMA leads to bilateral facial expressions including not only the upper, but also the lower face. This may partly be explained through cortico-cortical connections between the pre-SMA and other motor areas that project onto the facial nucleus. The pre-SMA has also been suggested to be more involved in the selection and preparation of movement, while the SMA proper would be more related to motor execution [28]. This higher-order role of the pre-SMA in motor functions may require the representation of muscles of the lower and upper face. Electrical stimulation of the pre-SMA may also have disrupted normal prefrontal inhibition of subcortical emotional centers. In line with this, Wild and colleagues [36, p. 2134] have proposed “a neural network in which frontal and temporal regions [...] would induce facial reactions and laughter mediated by dorsal brainstem regions. These reactions would be inhibited by the ventral brainstem, probably via frontal motor/premotor areas.” Lastly, it has to be kept in mind that these case studies can potentially inform us about the role of the left but not the right pre-SMA in smiling and laughter, as in both patients only the left SMA was stimulated.

4 Studying facial expressions with EEG

Modern neuroimaging techniques have recently been applied to discern brain activity accompanying emotional and voluntary facial expressions [17; 37]. Although highly interesting findings have so been obtained, their utilization has yet remained scarce, most likely due to artefact problems involved with participants’ facial movements during data acquisition.

Here, we propose the use of a combined EEG/facial EMG approach for investigating differences between spontaneous and posed facial expressions of emotion by focusing onto neurophysiological patterns preceding the onset of facial expressions.

The Bereitschaftspotential (BP) is a slow negative cortical potential developing up to two seconds before the onset of a movement and originating probably in contralateral to movement SMA and M1/LPMC [34]. Although typically shown to precede voluntary movements of the limbs, the BP may also occur prior to posed smiles, as recently suggested. Korb, Grandjean, and Scherer [19] asked healthy right-handed participants to
repeatedly perform five types of movements in a self-paced, voluntary manner: elevations of the right or of the left index-finger, a right or left-sided unilateral smile (consisting of pulling one mouth corner upwards and backwards), and a bilateral smile. Movements were preceded by a BP with a distinct distribution over the scalp, which at time of movement onset had a negative maximum over the contralateral hemisphere for finger movements, and a more bilateral, symmetrical, and somewhat weaker topography for bilateral smiles (see Figure 2).

Figure 2: BP (N = 16) at central electrodes C1, C2, and Cz preceding voluntary, self-paced smiling. Red line at time 0 marks EMG-onset as recorded over the zygomaticus muscles (smaller graphs). Adapted from [19].

The BP is mainly thought to reflect motor preparation and thus to precede only the execution, or at least the imagination, of voluntary movements [2; 34]. However, Keller and Heckhausen [18] reported similar albeit weaker BPs starting approximately 500 ms before spontaneous, unconscious movements of the hand and fingers. Others have suggested the existence of a BP before spontaneous movements too [21]. It remains thus an empirical question awaiting for an answer whether – after having shown a BP before posed smiles – (1) spontaneous (emotional, not preplanned) smiles and other facial expressions are preceded by a BP, and what its characteristic features would be, and (2) what occurs to the BP when the person voluntarily refrains from showing a felt emotion in her face, thus voluntarily regulating her spontaneously triggered facial expressions. We are currently attempting to address these points experimentally.

It has to be kept in mind, that when analyzing the EEG signal preceding different facial expressions the risk of major confounds that may lead to erroneous interpretations exists, as potential differences in brain activation patterns may be due to the activation of partly different facial muscles (for example, the orbicularis oculi is more often activated in spontaneous than voluntary smiling), or to different contraction intensities of the same muscles (e.g. spontaneous smiles may be of weaker or greater intensity than voluntary ones). A further limitation of this approach is that the same movement has to be repeated several times, and the neurophysiological data averaged over several trials, in order to get a reliable signal.

5 Conclusion

We have distinguished between spontaneous emotional facial expressions that are triggered by an emotion, and voluntarily posed facial expressions that can resemble spontaneous ones but are not accompanied by the respective underlying emotion. This distinction is most likely to be too strict and rare in everyday’s life facial expressions, but makes nevertheless sense on the basis of a neurological double dissociation between central paresis affecting selectively the production of voluntary or spontaneous facial movements. Further, we described the neural bases of spontaneous and voluntary facial expressions in humans based on a review of the literature. Here, the most consistent finding/hypothesis is that “the M1 is not necessary for emotional facial expression” [17, p.759]. Also, the role of cortical motor areas in the production of emotional facial expressions may have been underestimated in the past. Finally, we have presented the results of a study showing the existence of a BP before posed smiles [19], and discussed how future EEG/facial EMG studies may address the question of exactly what neurophysiological activities precede voluntary vs. spontaneous facial expressions.

This line of research is just at its beginning and, as the neuroscientific study of the production of facial expressions is a thorny endeavor and most studies in the past focused rather on the perception of facial expressions, many questions remain. For example, one can ask whether smiling and laughing result from different degrees of activation in common neural structures, or whether they rely on different mechanisms. Another point worth investigation is related to the communalities and/or differences between emotional facial expressions and the phenomenon of spontaneous, unconscious facial mimicry.

References
