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The Role of Electromyography (EMG) in the Study of Anticipatory Postural Adjustments

William P. Berg¹ and Adam J. Strang² ¹Department of Kinesiology and Health, Miami University, Oxford, Ohio ²Consortium Research Fellows Program, Wright-Patterson AFB, Ohio USA

1. Introduction

Postural control and stability are critical to the execution of almost any voluntary movement. In fact, "posture is the foundation upon which movement rides" (Cram & Criswell, 2011, p. 182). Posture is actively controlled by the neuromuscular system in concert with somatosensory, visual and vestibular informational inputs. To maintain a desirable posture under a wide range of individual, task and environmental constraints, the central nervous system (CNS) minimizes deflections of the body from a desirable orientation through the use of postural adjustments that fall into two general categories, compensatory and anticipatory (Santos et al., 2010). *Compensatory postural adjustments* (CPAs) are feedback-based mechanisms that are initiated by sensory events following the loss of desirable posture (Alexandrov et al., 2005; Park et al., 2004). CPAs act to restore stability after a postural perturbation has occurred (Henry et al., 1998; Macpherson et al., 1989; Maki & McIlroy, 1996). For example, immediately after a trip, reflexive and/or consciously controlled CPAs could help prevent a fall.

The second category of adjustments, and the focus of this chapter, are *anticipatory postural adjustments* (APAs). APAs are feed-forward mechanisms elicited by expected postural disturbances that produce preemptive muscle responses that help maintain stability. For example, when reaching for an object such as a book on a shelf, muscles in the trunk and legs activate in advance of muscle activity and movement in the shoulder and arm (Aruin & Latash, 1995a). These contractions in trunk and leg muscles constitute APAs because they precede the principal (focal) movement of the arm¹.

APAs were first described in 1967 when Belen'kii et al. reported the existence of electrical activity in the sacrolumbar muscles of the trunk and upper leg prior to fast shoulder flexion in standing. The presumed functional roles of APAs are to predict the stability-perturbing forces to be generated by an imminent focal movement or external perturbation, and produce a preparatory muscular contraction (i.e., APA) to stabilize the body in advance of the perturbation (Aruin & Latash, 1995a; Bouisset & Zattara, 1987; Massion, 1992; Zattara & Bouisset, 1988). Research has demonstrated that APAs are tailored to specific characteristics

¹ There is a relationship between the anticipatory and compensatory components of postural control. Specifically, it has been proposed that the CNS attempts to optimize the use of APAs, when possible, in order to allow for a suitable "scaling-down" of compensatory activity (Santos et al., 2010).

of focal movements, such as the direction (Aruin & Latash, 1995a), load (Aruin & Latash, 1995b; Aruin et al., 2001), acceleration (Lee et al, 1987; Zattara & Bouisset, 1988), and velocity (Hodges & Richardson, 1999). APAs are also influenced by conditions such as Parkinson's disease (Aruin et al., 1996), stroke (Dickstein et al., 2004), low back injury (Hodges, 2001; Hodges et al., 2003; Hodges & Richardson, 1996; Hodges & Richardson, 1999), and muscle fatigue (Allison & Henry, 2002; Strang & Berg, 2007; Strang et al., 2008; Strang et al., 2009; Vuillerme et al., 2002). For example, fatigue in lower extremity, upper extremity, and trunk muscles has been shown to result in earlier APA onset during focal movements, compared to APA onset in the absence of fatigue (Allison & Henry, 2002; Kanekar et al., 2008; Strang & Berg, 2007; Strang et al., 2008; Strang & Berg, 2007; Strang et al., 2008; Strang et al., 2008; Strang et al., 2009; Vuillerme et al., 2008; Strang et al., 2008; Strang et al., 2009; Vuillerme et al., 2002). It has been speculated that this effect represents a functional attempt by the CNS to limit additional postural disturbance resulting from the presence of muscle fatigue, when force-producing capabilities of muscles are likely compromised (Allison & Henry, 2002; Kanekar et al., 2008; Strang & Berg, 2007; Vuillerme et al., 2007; Vuillerme et al., 2008; Strang et al., 2008; Strang et al., 2009; Kanekar et al., 2008; Strang & Berg, 2007; Vuillerme et al., 2008; Strang et al., 2009; Vuillerme et al., 2002; Kanekar et al., 2008; Strang & Berg, 2007; Vuillerme et al., 2009; Vuillerme et al., 2009; Kanekar et al., 2008; Strang & Berg, 2007; Vuillerme et al., 2002). Strang et al. (2009) confirmed that fatigue induced early APA onset is, indeed, a centrally mediated adaptation.

Compared to the role of APAs in self-induced stability perturbations - the focus of the discussion thus far - the role of APAs in externally-induced stability perturbations has received less attention. Externally-induced perturbations include those that can occur when catching an object or being knocked off balance while walking on a crowded sidewalk. There is evidence that APAs can occur in conjunction with catching an object, even though the stability perturbation is not self-induced (Eckerle et al., 2011; Kazennikov & Lipshits, 2010; Lacquaniti & Maioli, 1989a, 1989b; Shiratori & Latash, 2001). The function of APAs in catching is to predict the stability perturbing forces to be imposed by the object on the catcher, and to produce preparatory muscular activity in order to stabilize the limb/body in advance of the catch. It is also known that APAs occur in catching even when information about ball weight is unavailable (Kazennikov & Lipshits, 2010). In this case, the CNS appears to scale APA magnitude to afford the greatest chance of catching the ball, regardless of weight (Eckerle et al., 2011).

2. How are APAs studied?

Experiments intended to better understand APAs in self-induced stability perturbations require experimental tasks that when performed, reliably induce APAs. Many researchers have used rapid arm-raising/reaching tasks to produce postural perturbations in the anterior direction (Belen'kii et al., 1967; Benvenuti et al., 1997; Bouisset & Zattara, 1987; De Wolf et al., 1998; Friedli et al., 1984; Gantchev & Dimitrova, 1996; Latash et al., 1995; Shiratori & Latash, 2001; Slijper et al., 2002; Strang & Berg, 2007; Strang et al., 2008; Strang et al., 2009. In this task paradigm, research participants typically stand bipedally with arms at their sides, and respond to an auditory or visual stimulus by moving one or both arms forward and up in the saggital plane as quickly as possible using shoulder flexion exclusively (see Figure 1). This maneuver produces rapid forward-displacement of the center of mass (COM) and reliably elicits APAs in the lumbar paraspinal, abdominal, and hamstring muscles.

Experiments on the role of APAs in externally-induced stability perturbations have utilized moving platforms (Timmann & Horak, 2001), pendulums (Santos et al., 2010), and one-handed catching tasks (Eckerle et al., 2011; Kazennikov & Lipshits, 2010; Lacquaniti & Maioli, 1989a, 1989b; Shiratori & Latash, 2001). APAs are important in catching because the

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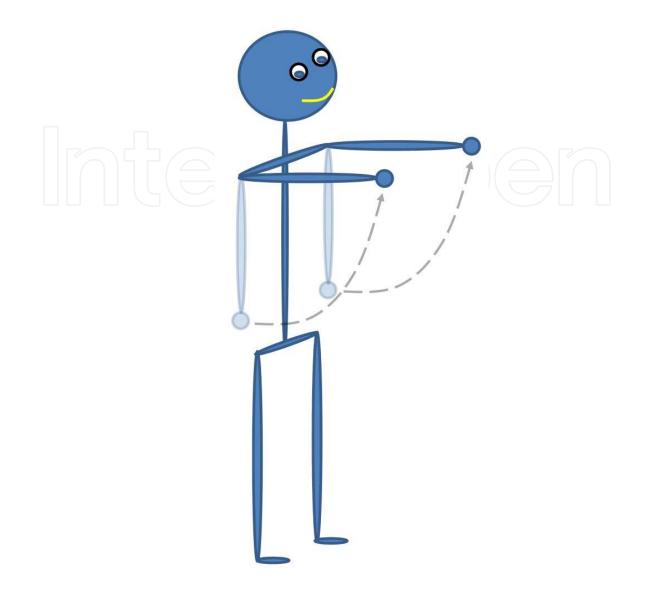


Fig. 1. Illustration of a rapid bilateral arm-raising maneuver (adapted from Strang et al., 2008).

compliance of the catching limb/hand needs to closely match the properties of impulsive impact. The properties of impact will vary depending on the weight of the object to be caught and the height from which the object is dropped. If limb compliance too high, the catching arm/hand could yield upon contact; whereas if limb compliance is too low, the object could rebound (Lacquaniti & Maioli, 1989a). In either case, arm/hand yielding or ball rebounding, a successful catch is unlikely. The catching maneuver depicted in Figure 2 produces rapid forward-displacement of the COM and the reliably elicits APAs in the wrist flexors, bicep brachii, anterior deltoid and lumbar paraspinal muscles.

Research participants have been asked to perform APA-inducing tasks, such as those described above, under various conditions that are determined by the research question under investigation. For example, Strang and colleagues have examined APAs under conditions of muscular fatigue (Strang & Berg, 2007; Strang et al., 2008; Strang et al., 2009). As for dependent variables, much of what has been learned about APAs has been acquired

by recording electromyographic (EMG) signals from the muscles involved in both postural control and the focal movement (Rosenbaum, 2011). At any point in time, the surface EMG signal is a composite electrical sum of all active motor units in an observed muscle or muscle group (Robertson et al., 2004). The amplitude of the EMG signal is an indicator of the magnitude of muscle activity, and is produced by increases in the number of active motor units and the frequency of activation (Robertson et al., 2004). Naturally, EMG amplitude increases as the intensity of muscle contraction increases. However, the relationship between EMG amplitude and force frequently is nonlinear (Robertson et al., 2004).

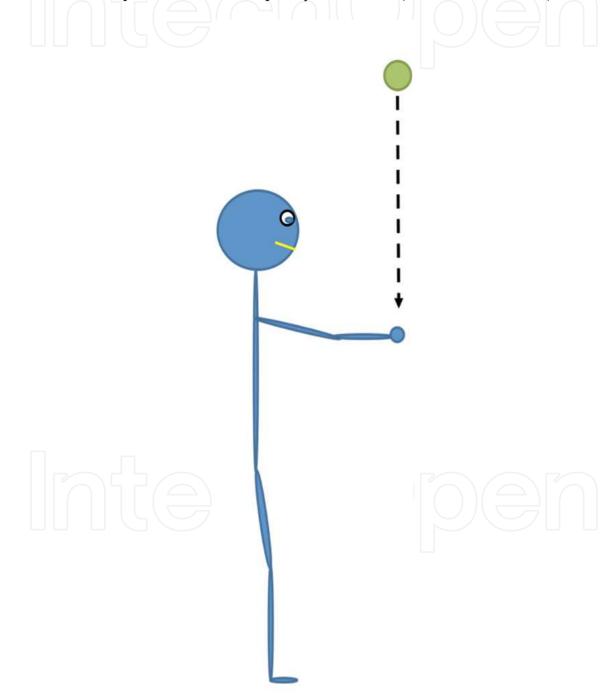


Fig. 2. Illustration of a one-handed catching task.

The purpose of EMG assessment is to document the activity of muscles under different conditions (Donaldson et al., 2003). As Basmajian (1967) stated, "electromyography is unique in revealing what a muscle actually does at any moment during movement and postures. Moreover, it reveals objectively the fine interplay or coordination of muscles: this is patently impossible by any other means" (p. 22). Regarding the study of APAs, EMG is commonly used to evaluate two fundamental properties of APAs, timing and magnitude.

3. Using EMG to ascertain the timing of APAs

Frequently, movement scientists are interested in determining when muscle electrical activity begins and/or ends, and this is especially true when studying APAs. Judging whether or not muscle activity qualifies as anticipatory requires a comparison of the onset of muscle activity to other temporal events, such as movement initiation or the activation onset of other muscles. The criteria used to identify muscle activity as anticipatory varies considerably in the literature. In most studies, however, muscle activity is considered anticipatory only when it occurs within a specified period of time - referred to as the APA timing-window (Allison, 2003). In studies on APAs resulting from self-induced postural perturbations, the APA timingwindow has been established in reference to one of two events; 1) the onset of the focal movement (Aruin & Latash, 1995; Bleuse et al., 2006; Girolami et al., 2010; Mochizuki et al., 2004; Schmitz et al., 2002; van Dieën & de Looze, 1999), or 2) the onset of muscle activity in the prime mover (agonist) of the focal movement (De Wolf et al., 1998; Fujiwara et al., 2003; Jacobs et al., 2009; Kanekar et al., 2008; Lee et al., 2009; Mannion et al., 2008; Mezaour et al., 2010; Morris & Allison, 2006; Strang et al., 2007; Strang et al., 2008; Tsao & Hodges, 2007; Tsao & Hodges, 2008; van der Fits et al., 1999; van der Heide et al., 2003; Yiou et al., 2009). A common example of the latter reference event is the onset of anterior deltoid muscle activity during forward arm-raising (e.g., Fujiwara et al., 2003; Gantchev et al., 1996; Morrison & Allison, 2006; Nana-Ibrahim et al., 2008; Strang & Berg, 2007; Tsao & Hodges, 2007; Vuillerme et al., 2002). In either case, the reference event is referred to as time zero (T_0), and the APA timing-window is defined relative to T_0^2 . It is generally assumed that muscle activity occurring just before or very shortly after T_0 must be pre-programmed, and therefore qualifies as anticipatory - although there may be problems with this assumption, which will be addressed later in the chapter. In contrast, studies of externally-induced postural perturbations usually define T_0 as the onset of the perturbation (Eckerle et al., 2011; Kazennikov & Lipshits, 2010; Lacquaniti & Maioli, 1989a, 1989b; Li & Aruin, 2007; Santos & Aruin, 2008; Santos et al., 2010; Shiratori & Latash, 2001; Timmann & Horak, 1998).

APA researchers have used a variety of techniques for detecting the onsets of focal movements and externally-induced perturbations including accelerometers (Girolami et al., 2010), video/motion capture (Bleuse et al., 2006), force transducers (van Dieën & de Looze, 1999) switch mats (Strang et al., 2009), and photoelectric techniques (Eckerle et al., 2011). The only requirement is that these devices possess high levels of temporal resolution, accuracy and reliability, and that they be able to interface with an EMG recording system.

As for identifying the onset of muscle activity in a prime mover and/or postural control muscles, APA researchers typically use one of two general techniques. The first involves visually inspecting the EMG signal to identify the earliest detectable change in EMG activity

² One study, however, established the APA timing-window using both the onset of the focal movement and the onset of muscle activity in the prime mover of the focal movement (Strang et al., 2009).

beyond baseline (e.g., Allison & Henry, 2002; De Wolf et al., 1998; Hodges et al., 2001; Hodges & Richardson, 1996, 1998; Mannion et al., 2008; Mezaour et al., 2010; Strang & Berg, 2007; Strang et al., 2008; Strang et al., 2009; Tsao & Hodges, 2007, 2008). Although visual inspection of the EMG signal is subjective, this technique has been shown to be reliable (Hodges & Bui, 1996). Visual inspection requires that EMG readers have adequate training and experience, and that satisfactory inter and intra-reader reliability is achieved prior to the beginning of data reduction and analysis.

Alternative computer-based techniques utilize statistical algorithms to determine muscle EMG onset (e.g., Jacobs et al., 2009; Kanekar et al., 2008; van der Fits et al., 1999; van der Heide et al., 2003). The most common automated EMG onset detection technique is the standard deviation (SD) method (Hodges & Bui, 1996), also broadly referred to as Shewhart protocols (Allison & Eastwood, 2002; Stokes et al., 2000). Shewhart protocols use different thresholds as multiples of average baseline EMG amplitude during a target window, for example, two SD (Hodges & Richardson, 1996) and three or more SD (Di Fabio, 1987; Happee & Van der Helm, 1995; Hodges & Bui, 1996; Stokes et al., 2000; Studenski et al., 1991) above baseline EMG amplitude have been reported. The advantages of computer-based methods are increased objectivity, faster data reduction, and a reduced requirement for EMG reader experience. Regardless of the onset identification technique employed, a EMG sampling rate of at least 1000 Hz should be used to achieve satisfactory temporal resolution.

In comparing the applicability of visual inspection and computer-based muscle EMG onset detection techniques in the context of APAs, using computer-based methods is especially difficult because the detection algorithms need to be sensitive to very small increments beyond the baseline signal (Allison, 2003). The reason for this is that APA onset is followed immediately by significantly larger amplitude compensatory responses (Allison, 2003). Moreover, the SD method can be ineffective for identifying APA onset when there is significant noise and/or electro-cardiac (ECG) artifact, or when EMG activity increases slowly (Allison, 2003; Hodges & Bui, 1996). These issues may partly explain why visual detection methods are often preferred for detecting APA onset (Allison, 2003). Moreover, it is not uncommon for an automated method for identification of APA onset to be accompanied by a visual check of each trace against the computer-derived value to ensure that the onset is meaningful (Adkin et al., 2002).

Smoothing is typically done prior to analyzing EMG signals to reduce baseline noise or ECG artifact. However, excessive smoothing of the data results in a loss of information and inaccurate identification of muscle onset (Gabel & Brandt, 1994; Halbertsma & DeBoer, 1981; Soderberg & Cook, 1984). On the other hand, insufficient data smoothing results in delayed identification of muscle onset (Hodges & Bui, 1996). The low pass cut-off frequency that most accurately and consistently determined the muscle onset in a study by Hodges & Bui (1996) was 50 Hz. Other studies have incorporated filtration similar to this value (Di Fabio, 1987; Nashner et al., 1983).

To increase the likelihood of detecting genuine APA onsets, the only postural muscle onsets that qualify as anticipatory are those that occur within the defined APA timing-window. The use of the APA timing-window is based on physiological limits and expectations of response latencies (Allison, 2003). Muscle activity onsets that occur too long after T_0 are more likely to represent compensatory rather than anticipatory activity. Likewise, muscle activity that occurs too far in advance of T_0 may not be directly related to the focal movement.

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There is tremendous variability in the literature regarding the definition of the APA timingwindow. First, and as mentioned earlier, studies on self-induced APAs often differ as to how T_0 is defined, i.e., some researchers define focal movement onset as T_0 and some define the onset of EMG activity in the prime mover as T_0 . The impact of the discrepancy in defining T_0 as different motor events has not been discussed in the literature. However, this issue is important because for any given focal movement, these two events are unlikely to occur simultaneously. In fact, in most focal movements, there is a delay between the onset of EMG activity in the prime mover and the actual initiation of movement. The delay is referred to as motor reaction-time and is attributed to the contractile properties of muscle and spread of electrical activity across the muscle that is required to generate enough force to start a focal movement (Fischman, 1984; Weiss, 1965). Strang & Berg (2007) and Strang and colleagues (2009) showed that the delay in typical unilateral and bilateral arm-raising maneuvers was 70 ms and 140 ms, respectively³. From this, it is not only apparent that a significant delay exists between agonist EMG onset and movement initiation, but also that this lag is influenced by the nature of the focal task itself.

Finally, not only does the referent for establishment of the APA timing-window differ across studies, the establishment of the beginning and end of APA timing-windows also varies tremendously. Studies of self-induced APAs using onset of the focal movement as T_0 have employed APA timing-windows ranging from the 200 ms prior to T_0 (Mochizuki et al., 2004) to $T_0 \pm 50$ ms (Schmitz et al., 2002). $T_0 + 50$ ms is the most commonly used termination point for the APA timing-window. The rationale is that since a latency of $T_0 + 50$ ms marks the threshold for the monosynaptic reflex, i.e., the earliest any form of feedback can be used to produce movement adjustments (Aruin & Latash, 1995a, 1995b), $T_0 + 50$ ms reflects the latest point at which one can safely assume that only feed-forward activity (APA) is reflected in the EMG signal.

Studies of self-induced APAs using the onset of EMG activity in the prime mover as T_0 have utilized APA timing-windows ranging from 300 ms prior to T_0 to 50 ms after T_0 (Mezaour et al., 2010), to 100 ms proceeding T_0 to 200 ms after T_0 (De Wolf et al., 1998). The lack of standardization across studies in defining the APA timing-window means that postural muscle onsets that would be described as anticipatory in some studies would not be described similarly in other studies. As will be addressed in the next section, this methodological variation also impacts the measurement of APA magnitude. In studies of externally-induced APAs, where there has at least been agreement that external perturbation onset represents T_0 , vastly different APA timing-windows have been employed as well, ranging from the 500 ms prior to T_0 (Kazennikov & Lipshits, 2010), to 100 ms before T_0 to $T_0 + 50$ ms (Aruin & Latash, 1995).

As an example of a study that focused on APA timing, Strang and Berg (2007) examined the fatigue-induced adaptive timing changes of APAs. APAs of 30 research participants were recorded before (baseline) and after (post-test) conditions of either rest (control group, n = 15) or fatigue (fatigue group, n = 15). Muscle fatigue was generated using a dead-lift exercise performed to exhaustion. Results showed that fatigue had no effect on postural stability during the focal movement, and yet caused earlier APA onsets in three of the six muscles evaluated. In spite of early APA activation, the APA EMG integrals of two of the three postural control muscles which exhibited fatigue-induced early APA onsets (T9 and L4).

³ Average motor reaction-times estimated from data collapsed across conditions shown in Table 1, p. 56 (Strang & Berg, 2007), and Table 3, p. 250 (Strang et al., 2009).

contralateral paraspinals) did not differ between baseline and post-test measures. The findings suggested that early APA onset may enhance postural stability by permitting a longer duration APA which can counteract fatigue-induced decreases in the force-producing capability of muscles that contribute to postural stability.

4. Using EMG to ascertain the magnitude of APAs

Researchers have relied on measures such as EMG integral (Aruin & Latash, 1995a, 1995b; Fujiwara et al., 2003; Strang & Berg, 2007), EMG Root Mean Square (RMS) amplitude (Lee et al., 2009; Tsao & Hodges, 2007), and EMG mean amplitude (van Dieën & de Looze, 1999; Van der Fits et al., 1999; van der Heide et al., 2003) to quantify the magnitude of muscle activity associated with APAs. In the following section, the nuances in the computations, interpretations, and applications of these measures are reviewed, along with a brief discussion of the EMG-force relationship within the context of APAs.

4.1 EMG integral

The most popular measure of APA magnitude has been the EMG integral, EMG_{Integral}

(Adkin et al., 2002; Aruin & Latash, 1995a, 1995b; Aruin et al., 1996; Aruin et al., 1998; De Wolf et al., 1998; Fujiwara et al., 2003; Girolami et al., 2010; Mochizuki et al., 2004; Nana-Ibrahim et al., 2008; Santos & Aruin, 2008; Strang & Berg, 2007; Xiaoyan & Aruin, 2007). This measure follows the basic formula, $\int EMG_{Integral} = \int EMG_{APA} - \int EMG_{Baseline}$ where

 $\int EMG_{APA}$ and $\int EMG_{Baseline}$ are the sum of rectified EMG amplitudes across timing-

windows of equal duration. There are two noteworthy features regarding the computation of EMG integral for APAs, which are, 1) the EMG integral is an estimate of the total EMG activity associated with an APA relative to baseline EMG, and 2) estimates of the EMG integral require equivalent duration APA timing- and baseline timing-windows.

With regard to the first feature, there are both practical and theoretical reasons why EMG integrals for APAs are estimated relative to baseline EMG, rather than a more standard comparator, e.g., EMG obtained during maximal voluntary isometric contraction (MVIC). First, MVICs are difficult to obtain for lumbar and trunk muscles where APAs are often observed⁴. Second, APAs elicit EMG activity that is so small compared to that produced during voluntary isometric contractions⁵, that relative comparisons would likely result in floor effects for integral estimates. Finally, while an APA can generally be considered a form of sub-maximal isometric muscle contraction, unlike MVICs, which are typically obtained during prolonged contraction following the slow development of muscle tension (Disselhorst-Klug et al., 2009), an APA exhibits a rapid onset, is involuntary, and take place over an extremely brief period of time (less than 150ms). These attributes of APAs make it difficult to estimate the force produced by APAs from EMG, since it is known that during rapid isometric contraction of relaxed muscles that force is not generated during the first 50-100ms of activation - this is because the muscle must first build intrinsic tension

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⁴ See McGill (1991) for a review of the difficulty in obtaining MVICs from lumbar and trunk muscles.

⁵ See Figure 5, p. 402, of van Dieën & de Looze (1999) to view an example of how little EMG activity is associated with APAs relative to that produced during even sub-maximal voluntary isometric contractions of lumbar muscles.

(Disselhorst-Klug et al., 2009). Though speculative, this dilemma is why most researchers have refrained from attempting to draw direct inference about the force produced by APAs from APA magnitude measures.

For the most part, the APA timing-windows used for computing EMG integrals have been similar to those used for detecting APA onsets, and thus have been wildly diverse. What is unique in the case of EMG integral calculation is the requirement of an equivalent-duration baseline time-window, which has typically been set at some pre-established time period prior to T_0 (e.g., T_0 -800 ms to -650 ms; Strang & Berg, 2007) where muscles are assumed to be in a relaxed state⁶. It has also been common to obtain estimates of EMG baseline integrals in smaller timing-windows, e.g., 50 ms, and then multiply the integral estimate by a common factor, e.g., 2, to achieve equivalent-duration baseline integrals to that of the predefined APA timing-window (Aruin & Shiratori, 2003; Shiratori & Aruin, 2004).

Finally, it is worth noting that there is a practical concern for studies that compute the EMG integral while also defining T_0 as the beginning of the focal movement. APA onset in such cases could precede the established timing-window set for integral estimates. Put another way, it is possible that in some, or perhaps many cases, when T_0 is defined as focal movement initiation, that EMG activity indicative of an APA may not be included in the computation of the EMG integral. To illustrate this point, we re-examined data presented by Strang et al., (2009) that included reports of EMG onset of the prime mover (anterior deltoid), EMG APA onset in selected postural muscles (low back and hamstring), and focal movement initiation (reaction time). From these data it was discovered that if T_0 had been defined as focal movement initiation, as opposed to the onset of the prime mover as was the case, APA onsets would have preceded the established APA timing-window (T_0 -100ms to +50ms) by an average of 50ms. This means that a significant amount of EMG activity that potentially represented an APA would not have been included in EMG integral calculations had that measure been derived.

4.2 Normalizing the EMG integral

EMG integrals for APAs are not typically reported in standard units of voltage [e.g., millivolt/s, see Fujiwara et al., (2003)], but instead as normalized values. This is done to account for large within and between-subject variability typically seen in APA response across muscles. To accomplish this, two normalization techniques have been common. The first normalizes EMG integrals *within-trial* relative to baseline EMG,

$$\int EMG_{Integral} = \frac{\int EMG_{APA} - \int EMG_{Baseline}}{\int EMG_{Baseline}} \text{ where } \int EMG_{Integral} \text{ greater than 1 indicates increased}$$

APA activity in targeted muscles, and $\int EMG_{Integral}$ less than 1 indicates inhibition of APA activity (Aruin & Latash, 1995b; 1996; Latash et al., 1995; Mochizuki et al, 2004; Morris & Allison, 2006; Santos & Aruin, 2008; Strang & Berg, 2007; Xiaoyan & Aruin, 2007). This approach is satisfactory for within-subject designs because it accounts for changes in baseline EMG magnitude and noise within-trial and across conditions for individual

⁶ Note that this statement is not true for studies in which the perturbation caused by the focal

movement comes in the form of force unloading (e.g., dropping a held mass; Aruin & Latash, 1995b; Schmitz et al., 2002; Shiratori & Aruin, 2007). In these cases, baseline activity reflects low-level isometric contraction of APA muscles.

participants. For between-subject designs, EMG integrals haven been normalized *within-subject* according to, $\int EMG_{Integral} = \frac{\int EMG_{APA} - \int EMG_{Baseline}}{\int EMG_{APA}^{Max} - \int EMG_{Baseline}^{Max}}$, where $\int EMG_{APA}^{Max}$ is the

highest APA integral value obtained across trials and condition and $\int EMG_{Baseline}^{Max}$ is the corresponding baseline EMG for that trial (Aruin, 2001; Girolami et al., 2010; Kanekar et al., 2008; Nana-Ibrahim et al., 2008; Schmitz et al., 2002; Shiratori & Latash, 2000; Shiratori & Aruin, 2007; Slipper & Latash, 2000; Xiaoyan & Aruin 2007). This approach restricts $\int EMG_{Integral}$ to a range of -1 to 1, where positive values indicate increased muscle activation in APA muscles and negative values corresponded to APA inhibition (Shiratori & Latash, 2000).

4.3 EMG mean amplitude and RMS

Because EMG mean amplitude is typically estimated after EMG amplitudes are rectified (van Dieën & de Looze, 1999), RMS and mean amplitude of EMG provide a similar quality of information, i.e., the central tendency of EMG amplitude response, despite subtle differences in mathematical computation. Also, it is worth mentioning that as long as the sampling rate of EMG is fixed, which was the case for every study reviewed for this chapter, EMG mean amplitude and EMG integral measures are equivalent for equal duration APA timing-windows. Finally, similar to EMG integral estimation, calculations of RMS and mean amplitude require an established APA timing-window. Researchers electing to use these alternative measures have mostly used timing-windows consistent with those described earlier (van Dieën & de Looze, 1999). The exceptions are two studies that calculated EMG mean amplitudes in adjacent epochs of 25ms (Lee et al., 2009) and 10ms (Tsao & Hodges, 2007) around T_0 to achieve higher resolution for description of changes in APA activity.

4.4 Limitations and caution for use of APA magnitude measures

Based on the information provide above, it is apparent that the accuracy of any estimation of APA magnitude is dependent on the reliability of EMG amplitude responses. However, there is ample reason to question the reliability of EMG when factors such as warm-up exercise, increased skin temperature (independent of exercise), and peripheral muscle fatigue are present. For example, peripheral muscle fatigue has been associated with both increases (Krogh-Lund, 1993) and decreases (Maton & Gamet, 1989) in EMG amplitude, compared to non-fatigued muscles. Bell (1993) demonstrated that increases in skin surface temperature, induced by blowing warm air directly onto the skin, elicited a decrease in EMG amplitude - thought to be caused by changes in fluid distribution within the muscle and/or nerve conduction velocity. Finally, Stewart and colleagues (2003) found that warmup exercise caused a decrease in EMG amplitude during voluntary isometric contractions thought to be caused by a rise in intramuscular temperature. Based on this, researchers seeking to examine the effects of muscle fatigue on APAs (Kanekar et al., 2008; Mezaour et al., 2010; Morris & Allison, 2006; Strang & Berg, 2007; Yiou et al., 2009), as well as those studies where experimental procedures might elicit changes in skin and/or muscle temperature following repeated experimental trials, should consider these effects when attempting to employ APA magnitude measures.

5. Conclusion

EMG has been, and will continue to be, an important tool for advancing understanding of anticipatory postural control. As this chapter has shown, the application of EMG in the study of APAs requires unique experimental methodologies, processing techniques, and statistical computations that accommodate both the behavioral constraints of the phenomenon within its observed context (e.g., self-induced or external perturbation) and the specific research question under examination. However, there is clearly more work needed that is focused on developing standard processing techniques (e.g., determination of the reference event for T_0 and the size of the APA timing-window) for the use of EMG in the study of APAs.

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This second of two volumes on EMG (Electromyography) covers a wide range of clinical applications, as a complement to the methods discussed in volume 1. Topics range from gait and vibration analysis, through posture and falls prevention, to biofeedback in the treatment of neurologic swallowing impairment. The volume includes sections on back care, sports and performance medicine, gynecology/urology and orofacial function. Authors describe the procedures for their experimental studies with detailed and clear illustrations and references to the literature. The limitations of SEMG measures and methods for careful analysis are discussed. This broad compilation of articles discussing the use of EMG in both clinical and research applications demonstrates the utility of the method as a tool in a wide variety of disciplines and clinical fields.

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