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Child-adult differences in muscle strength and activation pattern during isometric elbow flexion and extension

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Abstract

Muscle strength and activation were compared in boys and men during maximal voluntary elbow flexion and extension contractions. Peak torque, peak rate of torque development ($d\tau/d\tau_{max}$), rate of muscle activation, and electromechanical delay (EMD) were measured in 15 boys (aged 9.7 \pm 1.6 years) and 16 men (aged 22.1 \pm 2.8 years). During flexion, peak torque was significantly lower in boys than in men (19.5 \pm 5.8 vs. 68.5 \pm 11.0 Nm, respectively; p < 0.05), even when controlling for upper-arm cross-sectional area (CSA), and peak electromyography activity. Boys also exhibited a lower normalized $d\tau/d\tau_{max}$ (7.2 ± 1.7 vs. 9.5 ± 1.6 (Nm·s⁻¹)·(Nm⁻¹), respectively; p < 0.05) and a significantly longer EMD (75.5 ± 28.4 vs. 47.6 ± 17.5 ms, respectively). The pattern was similar for extension, except that group differences in peak torque were no longer significant when normalized for CSA. These results suggest that children may be less able to recruit or fully utilize their higher-threshold motor units, resulting in lower dimensionally normalized maximal torque and rate of torque development.

Keywords

children; coactivation; EMG; maturation; neuromotor; strength

Introduction

Maximal muscle force is lower in children than in adults, even when size-normalized, for example, to body mass (De Ste Croix et al. 1999; Lambertz et al. 2003) or to muscle crosssectional area (CSA) (Grosset et al. 2008; Halin et al. 2003; Kanehisa et al. 1995a, 1995b; Lambertz et al. 2003; Seger and Thorstensson 2000; Wood et al. 2006). Thus, additional factors, such as neuromuscular activation or muscle composition, must account for the observed strength differences. Very few studies have examined muscle activation when investigating age-related muscle function differences.

Children's rate of force development $(d\tau/d\tau)$ also appears to be lower than that of adults (Asai and Aoki 1996; Belanger and McComas 1989; Grosset et al. 2005). As is the case with

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maximal force, explanations for children's lower $d\tau/d\tau$ may involve differences in muscle composition and in muscle activation. Other factors capable of affecting $d\tau/d\tau$ may include musculo-tendinous stiffness, excitation–contraction coupling, and muscle-fibre conduction velocity. These have been examined in children only to a limited extent, and with inconsistent results (Cornu and Goubel 2001; Grosset et al. 2005; Lambertz et al. 2003).

Muscle-fibre composition in children is believed to be similar to that of adults (Dubowitz 1965). Additionally, during twitch stimulation, contractile characteristics, such as contraction time and half-relaxation time, have been shown to be similar across age groups (Belanger and McComas 1989; Davies 1985, 1983; McComas et al. 1973; Paasuke et al. 2000), supporting the notion of a similar fibre composition in children and adults. Thus, differences in muscle composition, if any, do not seem sufficient to account for the observed child–adult strength differences.

An additional factor that could possibly affect measured strength is agonist–antagonist coactivation. While some studies demonstrate greater coactivation in children (Frost et al. 1997; Grosset et al. 2008; Lambertz et al. 2003), others do not (Bassa et al. 2005; Kellis and Unnithan 1999). All those studies were conducted only on the lower limbs, and their findings are, accordingly, limited.

Asmussen (1973) was one of the first to propose that child–adult strength differences could be related to neuro-motor maturation. Surprisingly, this topic has since been addressed only to a limited extent. Pediatric muscle-activation studies have focused on the lower extremities (Blimkie 1989; Paasuke et al. 2000; Grosset et al. 2008; Lambertz et al. 2003; Streckis et al. 2007). However, age-related strength changes in the upper extremities differ from those in the lower extremities (Parker et al. 1990), and strength changes in elbow flexors differ from those in elbow extensors (Kanehisa et al. 1995*a*). Moreover, while the lower extremities are commonly used for locomotion and posture control — tasks that do not normally require fast contractions — the upper extremities are more often used for reaching and grabbing, and may involve faster contractions and multiple coordinated steps of precise motor control (Hirschfeld 2007). Such functional differences could possibly manifest themselves as different muscular force–velocity relationships in the upper and lower extremities (Charteris 1999). Thus, muscle performance and activation of the lower extremity may not reflect the corresponding characteristics of the upper extremity.

Only 2 studies have examined children's muscle strength and electromyography (EMG) activity in the upper extremities, and they reported only elbow flexion (Asai and Aoki 1996; Halin et al. 2003), with no data on extension or on co-activation. Additionally, one of these studies (Asai and Aoki 1996) provided no anthropometric data or any normalization for body size. These studies, therefore, do not elucidate whether children's lower relative strength of the upper extremity is due only to their smaller muscle mass or whether it is due to differential muscle activation or coactivation as well. Also, since the growth rates of muscular strength and rates of growth size of reciprocal muscle groups differ (Kanehisa et al. 1995*a*), it is unclear whether flexion and extension forces are similarly affected by maturation.

The purpose of this study was to compare muscle activation of prepubescent boys with that of men during maximal voluntary isometric contractions of both elbow flexors and extensors. It was hypothesized that size-normalized muscle strength would be lower in the boys and would be associated with less muscle activation.

Materials and methods

Subjects

Fifteen boys (age, 9.6 ± 1.6 years; body mass, 32.7 ± 6.9 kg; height, 1.37 ± 0.09 m) and 16 men (22.1 ± 2.8 years; 81.7 ± 7.2 kg; 1.80 ± 0.07 m) were recruited to participate in this study, which was approved by Brock University's Research Ethics Board (St. Catharines, Ont.). All participants were right-handed, healthy individuals with no upper-limb injuries. All were physically active, but no one was a competitive athlete or participated in a unilateral sport on a regular basis. Written informed consent was obtained from all participants and the children's parents prior to the study's onset.

Anthropometric measurements

Standing and seated heights were measured using a wall-mounted stadiometer (Length Boards, Ellard Instrumentation, Ltd., Monroe, Wash.). Body mass was measured on a digital scale (EKS Electronic Scales, Cedex, France). Skin-fold thickness was measured in triplicate, using skinfold calipers (RH15 916, Harpenden, England) at the biceps, triceps, subscapular, and suprailiac sites on the right side of the body. Body fat percentage was calculated using the equations of Slaughter et al. (1988). Upper-arm CSA was calculated using measures of upper-arm circumference, as well as biceps and triceps skinfold thickness, as described elsewhere (Gurney and Jelliffe 1973). The coefficient of variance of such measurements in another subset of subjects in our lab was 2.0%, with an intraclass correlation coefficient of r = 0.99, which is similar to values reported elsewhere (Knapik et al. 1996).

Experimental protocol

All subjects visited the laboratory on 2 occasions, at least 3 days apart. On the first visit, subjects were informed of the study's procedures, anthropometric measurements were taken, and questionnaires were filled out (facilitated by a researcher for child subjects). Physical activity levels were determined using the Godin-Shephard Leisure-Time Exercise Questionnaire (Godin and Shephard 1985). The children self-assessed their pubertal stage in accordance with secondary sex characteristics (pubic hair), as described by elsewhere (Tanner 1962; Duke et al. 1980). Then, the complete test protocol was practiced to familiarize the subjects with all test procedures. The actual test protocol was performed on the second visit.

The Biodex System 3 torque dynamometer (Biodex, Shirley, N.Y.) was used to measure torque during isometric contractions of the elbow flexors and extensors. An isometric task was chosen to minimize antagonist involvement (Calder and Gabriel 2007). Thus, child– adult torque differences would be mainly attributable to agonist activation. The subject sat upright in the dynamometer seat, with the right shoulder at 90° of flexion, resting his upper

Warm-up consisted of 3-5 three-second maximal voluntary contractions (MVC) of flexion and extension. Ten seconds separated successive contractions. Subjects were given explicit instructions to contract as hard and as fast as possible from a relaxed state to maximize torque and $d\tau/d\tau$. Online visual feedback of the dynamometer's force signal was available for the subjects on a computer screen. Visual feedback has been shown to be important for torque production (Kellis and Baltzopoulos 1996), especially in young children (Smits-Engelsman et al. 2003). Following 2–3 min of rest, the subject performed 2 sets of contractions, either flexion or extension, in a randomized order among subjects. Instructions were identical to those of the warm-up. Subjects completed 2 sets of 5 MVCs with 30-s rest periods between contractions and at least a 2-min break between sets. While a 30-s rest between contractions is arguably short, it was important for maintaining an appropriate level of concentration in the boys. Subsequent data analysis failed to reveal fatigue or other order effects in either the boys or the men. A minimum of 2 sets was required of each participant (totaling 10 MVCs each of flexion and extension). Additional sets were added, as needed, to reach 10 valid MVCs if some data were deemed unacceptable due to execution errors, deviations in EMG baseline, or abnormal torque or EMG amplitudes or tracing. Throughout each contraction, subjects were verbally encouraged to perform a maximal effort in both torque and contractile speed.

Electromyography

Surface EMG signals were collected from 2 sites: at the muscle-belly midsections of the biceps brachii, and at the lateral head of the triceps brachii. These were determined visually during a resisted static contraction. The bipolar surface electrodes (DE-2.1, DelSys Inc., Boston, Mass.) were placed in line with the muscle fibres, away from the estimated motor point (Delagi and Perotto 1980). The reference electrode was placed over the middle of the right clavicle. Electrode sites were prepared by shaving the skin, when necessary, and thoroughly rubbing the skin with alcohol. Raw EMG signals were amplified by a DelSys amplifier (10–500 Hz band-pass filter, Bagnoli-4 EMG System, Boston, Mass.), converted from analog to digital (DI-205-C, DATAQ Instruments, Akron, Ohio), and recorded at an acquisition rate of 1000 Hz (WinDaq Pro Data Acquisition, DATAQ Instruments).

Data analysis

Using MatLab (The MathWorks, Natick, Mass.), several variables were calculated separately for flexion and extension. Peak agonist and antagonist EMG amplitude were determined and averaged over the best 10 trials. Mean traces were created for torque, agonist EMG, and antagonist EMG. These traces were used to calculate $d\tau/d\tau$, rate of muscle activation (Q_{30}) of both muscle groups, and electromechanical delay (EMD). Peak rate of torque development ($d\tau/d\tau_{max}$) was calculated by taking the maximum of the first derivative of the

torque signal (Gabriel et al. 2001). Rate of muscle activation was defined by the area under the EMG curve of the linear envelope of the detected EMG signal during the first 30 ms (Gottlieb et al. 1989; Gabriel and Boucher 2000). EMD was defined as the time lapse between the onsets of EMG and torque generation, and was calculated in the agonist muscle. The time of $d\tau/d\tau_{max}$ was calculated as the time delay between the onset of torque generation and the occurrence of $d\tau/d\tau_{max}$. Coactivation was calculated as the ratio between the antagonist's EMG amplitude divided by its EMG amplitude as an agonist (i.e., for flexion: (triceps EMG amplitude in flexion)/(triceps EMG amplitude in extension); for extension: (biceps EMG amplitude in extension)/(biceps EMG amplitude in flexion)).

Statistical analysis

Data for both child and adult groups are presented as means \pm standard deviations. An unpaired 2-tailed Student's *t* test was used to compare the means between the 2 groups. Possible confounding factors (e.g., EMG signal amplitude) were entered as covariates in an analysis of covariance. Differences between groups were considered significant at p < 0.05. All analyses were performed using SPSS 16.0 (SPSS Inc., Chicago, Ill.).

Results

Subject characteristics appear in Table 1. While body size was significantly (p < 0.001) smaller in the boys, no differences were observed in body-fat percentage or in skinfold thickness over the biceps or triceps. Thus, the possible effect of subcutaneous fat on the EMG signal (De la Barrera and Milner 1994) was similar in the 2 groups.

As expected, in absolute terms, men were significantly stronger than boys in both flexion $(68.5 \pm 11.0 \text{ vs. } 19.5 \pm 5.8 \text{ Nm}$, respectively; p < 0.001) and extension $(55.0 \pm 10.1 \text{ vs. } 18.4 \pm 5.7 \text{ Nm}$, respectively; p < 0.001). Normalized to body mass, peak torque remained significantly higher in the men in flexion $(0.84 \pm 0.15 \text{ vs. } 0.60 \pm 0.15 \text{ Nm} \cdot \text{kg}^{-1}$, respectively; p < 0.001), and not significantly higher in extension $(0.68 \pm 0.14 \text{ vs. } 0.58 \pm 0.20 \text{ Nm} \cdot \text{kg}^{-1}$, respectively; p = 0.01). Similar findings were obtained when peak torque was normalized for upper-arm CSA (Fig. 1). Peak torque was moderately correlated with peak EMG amplitude (r = 0.48, p = 0.01 and r = 0.36, p = 0.05 for flexion and extension, respectively). Controlling for EMG amplitude (as a covariate) did not change the pattern of results. That is, child–adult differences in body-size-normalized peak flexion torque remained significantly lower in boys than in men, while no such difference could detected in extension peak torque.

Men exhibited significantly higher absolute $d\tau/d\tau_{max}$ values than boys in both flexion and extension (652 ± 154 vs. 141 ± 53 and 445 ± 113 vs. 113 ± 32 Nm·s⁻¹, respectively; p < 0.001). This difference persisted in both contractions when $d\tau/d\tau_{max}$ was normalized for peak torque (Fig. 2). Normalized $d\tau/d\tau_{max}$ was moderately correlated with Q_{30} (r = 0.40, p = 0.02) for flexion, but not for extension. Controlling for Q_{30} as a covariate, $d\tau/d\tau_{max}$ remained lower in boys than in men, although the difference did not reach significance in flexion (p = 0.08). The time from torque onset to $d\tau/d\tau_{max}$ was significantly longer in boys during flexion (71.0 ± 29.1 vs. 51.2 ± 9.7 ms, respectively; p = 0.015), but not during extension (58.3 ± 31.7 vs. 58.8 ± 23.7 ms, respectively; p = 0.96).

Flexion Q_{30} was significantly lower in the boys than in the men (0.097 ± 0.080 vs. 0.366 ± 0.168 mV·s, respectively; p < 0.001). A similar pattern was observed during extension, but the difference did not reach statistical significance (0.070 ± 0.046 vs. 0.082 ± 0.047 mV·s, respectively; p = 0.46). Q_{30} was moderately correlated with agonist EMG amplitude during flexion (r = 0.55, p = 0.01), but not during extension. Controlling for root mean squares amplitude as a covariate, the boys' Q_{30} values remained lower than the men's.

Agonist EMD was significantly shorter in men than in boys in both flexion (p = 0.002) and extension (p = 0.002) (Fig. 3).

The coactivation index was not significantly different between boys and men $(0.59 \pm 0.44 \text{ vs.} 0.44 \pm 0.27 \text{ in flexion}, p = 0.26; \text{ and } 0.09 \pm 0.06 \text{ vs.} 0.12 \pm 0.07 \text{ in extension}, p = 0.41, respectively). It should be noted that during extension, antagonist activation could not be detected in 7 of the men or in 6 of the boys.$

Discussion

This study examined maximal voluntary isometric torque and rate of torque development, along with agonist and antagonist EMG activity, during elbow flexion and extension in prepubertal boys and men. The main findings are the lower peak torque and $d\tau/d\tau_{max}$ in boys than in men, not only in absolute terms, but also when corrected for body size and controlled for EMG activity. The elbow-extension peak torque, corrected for arm CSA, was no longer different between groups, while $d\tau/d\tau_{max}$ remained lower in boys, even after correcting for body size and controlling for EMG activity. The EMD was consistently longer in the boys, during both flexion and extension.

Normalizing peak torque for lean upper-arm CSA (Fig. 1) greatly reduced peak torque age differences, but the normalized torque remained lower in the boys than in the men. This was clearly apparent in flexion, but not in extension. Anthropometrically derived CSA, as determined in this study, is limited in its accuracy and resolution. Nevertheless, our results agree with previous studies, which also demonstrated a lower CSA-normalized torque in children than in adults in elbow flexion, as well as in other muscle groups, using anthropometry or magnetic resonance imaging (Kanehisa et al. 1995b; Seger and Thorstensson 2000; Davies 1985; Grosset et al. 2008; Halin et al. 2003; Tonson et al. 2008). It should be noted that Tonson et al. (2008) recently suggested that the appropriate body-size parameter for normalizing muscle strength should be muscle volume (determined by magnetic resonance imaging). The authors showed that when using the volume parameter, no child-adult strength difference could be detected. However, Bamman et al. (2000) demonstrated that muscle CSA was a better parameter than volume (both determined by magnetic resonance imaging), since strength better correlated with CSA and regressed to the origin. Furthermore, the study did not find physiological CSA to be more precise than the anatomically determined CSA, leading the authors to recommend anatomical CSA for estimating specific tension in vivo. Thus, using various indices, body size does not appear to fully explain the child-adult differences in maximal flexion strength. It is interesting to note that, similar to the current study's findings, Kanehisa et al. (1995a) reported an age-related increase in CSA-normalized elbow-flexor, but not in elbow-extensor, strength in 7- to 9- vs.

16- to 18-year-old boys. Therefore, as suggested by Kanehisa et al. (1995*a*), it is possible that strength development in reciprocal muscle groups may not follow the same pattern during growth and maturation.

Age-group differences in size-normalized-flexion peak torque were still apparent after controlling for EMG activity (root mean squares peak amplitude). Our results agree with previously reported age-related differences in peak torque, relative to EMG amplitude, in the lower extremities (plantar flexion) (Grosset et al. 2008; Lambertz et al. 2003; Moritani et al. 1989), and extend them to the upper extremities. These data suggest that other factors, such as possible differences in moment arm or muscle composition, may also partly explain age-related differences in elbow-flexion strength.

The lower absolute $d\tau/d\tau_{max}$ observed in the boys is partly explained by the dependence of $d\tau/d\tau$ on peak torque. Thus, normalizing $d\tau/d\tau$ to peak torque can be useful in searching for other factors that might determine $d\tau/d\tau$ (Holtermann et al. 2007). Surprisingly, only 1 previous study normalized children's rate of force development to peak force; it reported lower values for elbow flexion in 6-year-old boys, compared with men (Asai and Aoki 1996). Our findings (Fig. 2) correspond to those of Asai and Aoki (1996), and complement them with elbow-extension data. Thus, children's lower rate of force development is a persistent finding, independent of their lower maximal strength.

The rate of force development depends on the rate of muscle activation (Corcos et al. 1989). To our knowledge, no previous study has related children's rate of force development to any index of muscle activation. In both flexion and extension, when Q_{30} was taken into account, $d\tau/d\tau_{max}$, absolute or corrected for peak torque, remained lower in boys. These results suggest that factors other than muscle size and muscle activation may be involved in determining the rate of force development, as is the case for peak torque (see below).

EMD reflects muscle-tendon stiffness, excitation-contraction coupling, and muscle-fibre conduction velocity (Cavanagh and Komi 1979; Halin et al. 2003). EMD in the current study was consistently longer in boys than in men during flexion and extension (Fig. 3), as was the time to maximal rate of torque development. An age-related decrease in EMD has previously been reported in maximal elbow flexion and in plantar-flexion twitch contraction (Asai and Aoki 1996; Grosset et al. 2005). Using different types of muscle contractions, Cavanagh and Komi (1979) demonstrated that, in adults, it is mainly the series-elastic component (muscle-tendon stiffness), and not the excitation-contraction coupling, that determines EMD. Indeed, lower musculo-tendinous stiffness has been reported in 7-to 10year-old boys compared with adults during plantar flexion (Lambertz et al. 2003). However, Cornu and Goubel (2001) could not show these differences during elbow flexion. Moreover, in a recent study (Grosset et al. 2009), musculo-tendinous stiffness changes could account only for <20% of the variance in EMD changes. Thus, it is unlikely that the boys' longer EMD in the current study is solely due to their more compliant muscle-tendon complex. More likely is the proposition that factors such as lower muscle activation and lower musclefibre conduction velocity in boys (Halin et al. 2003) are also significant determinants of EMD. Further study is needed to elucidate this issue.

We suggest that the boys' longer EMD is partly explained by their lesser recruitment or utilization of the faster, higher-threshold motor units. Two studies have examined muscle strength and activation during elbow flexion in children and adults (Asai and Aoki 1996; Halin et al. 2003). In both studies, it is argued that children involve fewer type-II fibres during maximal voluntary contractions than do adults. Halin et al. (2003) reported that during 30 s of isometric maximal voluntary elbow flexion, force decrement was lower and the decline in the EMG mean power frequency was slower in boys than in men. Additionally, during the 30-s contraction, there was no change in the boys' muscle-fibre conduction velocity, while the men's conduction velocity decreased. Based on these findings, the authors suggest that the boys' lower maximal force and fatigability were due to lower involvement of type-II fibres. Asai and Aoki (1996) reported that increased elbowflexor pretension resulted in a decreased rate of isometric force development in men but not in 6-year-old boys. The authors argue that the difference between children and adults in response to pretension may be explained by children's lower reliance on type-II muscle-fibre recruitment (Asai and Aoki 1996). This contention is supported by our findings, where lower recruitment or utilization of type-II muscle fibres could explain the observed lower peak torque and $d\tau/d\tau_{max}$, as well as the longer EMD, in the boys. This differential recruitment or utilization of higher-threshold motor units is also consistent with the lower size-normalized anaerobic power (Falk and Bar-Or 1993) and the faster recovery from intense short-term exercise (Falk and Dotan 2006; Hebestreit et al. 1993) observed in children.

Systematic group differences in coactivation could not be discerned in the current study. In fact, the EMG amplitude of the antagonist muscle was consistently low, or was nonexistent, in both groups. This finding upholds the choice of isometric testing as a methodological means of isolating group differences in muscle function. Thus, in isometric contraction, coactivation does not appear to be a substantial contributor to the child–adult differences observed in measured force or power generation. Therefore, our findings of consistently lower elbow-flexion peak torque and rate of torque development in boys provide further support to the notion of lower agonist activation in children during maximal force generation.

In conclusion, the results of the current study suggest that, during maximal voluntary isometric elbow flexion, children are less able to recruit or utilize their higher-threshold motor units, resulting in lower maximal force and rate of force development. Further research is needed to better understand children's force generation and motor-unit recruitment patterns in maximal, as well as submaximal, isometric and dynamic contractions of reciprocal muscle groups. The possibility of affecting the rate and magnitude of age-related changes should be investigated in training studies.

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Peak torque during flexion and extension, corrected for size (upper-arm cross-sectional area (CSA)). *, p < 0.05.





Peak rate of torque development during flexion and extension normalized for peak torque. *, p < 0.05.





The delay between electromyography onset of the agonist and force onset in flexion and extension. *, p < 0.05.

Table 1

Subject characteristics (data are means \pm SD).

Variables	Men (<i>n</i> = 16)	Boys (<i>n</i> = 15)
Age (years)*	22.1±2.8	9.6±1.6
Body mass (kg)*	81.7±7.2	32.8±6.9
Height (cm)*	180.9±7.2	137.5±8.7
Body fat (%)	18.3±5.5	16.4±6.2
Lean body mass $(kg)^*$	68.7±5.5	27.1±4.6
Biceps skinfold (mm)	5.0±1.8	4.9±2.1
Triceps skinfold (mm)	10.4±3.2	10.6±4.0
Upper-arm CSA (cm ²)*	69.4±9.0	24.6±4.3
Years from PHV	na	-3.6±1.0

Note: CSA, cross-sectional area; na, not applicable; PHV, peak height velocity.

* Significant difference between groups, p < 0.05.