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Training-specific functional, neural, and hypertrophic adaptations to explosive- vs. sustained-contraction strength training

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Balshaw TG, Massey GJ, Maden-Wilkinson TM, Tillin NA, Folland JP. Training-specific functional, neural, and hypertrophic adaptations to explosive- vs. sustained-contraction strength training. *J Appl Physiol* 120: 1364–1373, 2016. First published April 7, 2016; doi:10.1152/japplphysiol.00091.2016.—Training specificity is considered important for strength training, although the functional and underpinning physiological adaptations to different types of training, including brief explosive contractions, are poorly understood. This study compared the effects of 12 wk of explosive-contraction (ECT, $n = 13$) vs. sustained-contraction (SCT, $n = 16$) strength training vs. control ($n = 14$) on the functional, neural, hypertrophic, and intrinsic contractile characteristics of healthy young men. Training involved 40 isometric knee extension repetitions (3 times/wk): contracting as fast and hard as possible for ~1 s (ECT) or gradually increasing to 75% of maximum voluntary torque (MVT) before holding for 3 s (SCT). Torque and electromyography during maximum and explosive contractions, torque during evoked octet contractions, and total quadriceps muscle volume ($QUADS_{VOL}$) were quantified pre and post training. MVT increased more after SCT than ECT [23 vs. 17%; effect size (ES) = 0.69], with similar increases in neural drive, but greater $QUADS_{VOL}$ changes after SCT (8.1 vs. 2.6%; ES = 0.74). ECT improved explosive torque at all time points (17–34%; $0.54 \leq ES \leq 0.76$) because of increased neural drive (17–28%), whereas only late-phase explosive torque (150 ms, 12%; ES = 1.48) and corresponding neural drive (18%) increased after SCT. Changes in evoked torque indicated slowing of the contractile properties of the muscle-tendon unit after both training interventions. These results showed training-specific functional changes that appeared to be due to distinct neural and hypertrophic adaptations. ECT produced a wider range of functional adaptations than SCT, and given the lesser demands of ECT, this type of training provides a highly efficient means of increasing function.

resistance exercise; neural drive; rate of torque development; maximum strength; contractile properties

NEW & NOTEWORTHY

Explosive-contraction strength training (ECT) denoted by brief contractions with high rate of torque development produced a wider range of functional adaptations than sustained-contraction strength training (SCT), with improvements in early- and late-phase explosive strength, as well as maximum strength. In contrast, SCT only improved maximum and late-phase explosive strength. The substantially lower loading duration of ECT (7% of SCT) makes this a less-demanding training modality compared with SCT, which may be preferentially tolerated by musculoskeletal patients.

MAXIMUM AND EXPLOSIVE STRENGTH are two components of skeletal muscle function that can be critical to the performance of human movement. Maximum strength is the greatest amount of force that can be generated, whereas explosive strength reflects the ability to increase force rapidly from a low or resting level (1, 20, 48). Muscle weakness, including low maximum and explosive strength, contributes to the functional limitations experienced by numerous patient groups (35, 42, 43), including osteoarthritis patients (26). Strength training is widely recommended for improving function of all adults (2a, 32), and the increases in explosive and/or maximum strength that occur following training may have profound benefits to mobility, locomotion, and quality of life of older individuals and patients (13, 25, 26, 34, 37, 44). While training specificity is widely considered important within the context of strength training (8, 12, 18, 21), the functional adaptations to different types of strength training are not well understood, reducing the efficacy of training guidance and prescription. Furthermore, the similarity or specificity of the underpinning neural and contractile adaptations to different training regimes has received relatively little attention.

Explosive-contraction strength training (ECT), emphasizing rapid torque development during short contractions, is a relatively nonfatiguing training modality that may be well tolerated by patient groups (i.e., osteoarthritis) who commonly report substantial fatigue (36, 38) and therefore may offer improved adherence within these populations. ECT has been found to produce significant increases in both maximum and explosive strength (48). In contrast, conventional strength training typically has a primary emphasis on training with sustained contractions (SCT) at high loads leading to pronounced fatigue (31) and may neglect rapid torque development. Our recent 4-wk intervention study contrasted ECT and SCT, finding distinct training-specific adaptations in functional capabilities and neural drive: maximum strength and corresponding electromyography (EMG) increased more after SCT, and early-phase explosive strength and EMG (≤100 ms) during the rising/explosive phase of contraction increased more following ECT (45). This demonstrated that at least in the initial stages of a training program, ECT and SCT produce distinct functional and neural adaptations. However, the efficacy of longer-term ECT for functional and neural adaptations remains unknown, and the contrasting influence of these training interventions on the intrinsic contractile properties and hypertrophy (volume) of skeletal muscle has yet to be investigated. A more comprehensive comparison of ECT and SCT may facilitate a greater

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understanding of the influence of training variables, particularly loading duration (high SCT vs. low ECT) and rate of torque development (RTD, high ECT vs. low SCT), on functional and physiological adaptations.

The purpose of this study was to investigate the efficacy of 12 wk of ECT and compare it to SCT and a control group (CON) by assessing the specificity of the functional changes (maximum and explosive strength), as well as the underpinning adaptations in neural drive, intrinsic contractile properties, and muscle volume after these interventions. We hypothesized that ECT and SCT would elicit distinct and specific functional changes [ECT > SCT for early-phase (≤100 ms) explosive strength; SCT > ECT for maximum strength], as a result of distinct neural and contractile adaptations.

MATERIALS AND METHODS

Participants

Forty-eight young, healthy, asymptomatic men who had not completed lower-body strength training for >18 mo and were not involved in systematic physical training were recruited and provided written informed consent prior to participation in this study, which was approved by the Loughborough University Ethical Advisory Committee. Following familiarization, participants were randomly assigned to ECT, SCT, or CON groups that were matched for maximum voluntary torque (MVT) and body mass. A total of five participants withdrew from the study (4 withdrew because of personal reasons, and 1 was excluded because of noncompliance). Forty-three participants (ECT, $n = 13$; SCT, $n = 16$; CON, $n = 14$) completed the study. Baseline recreational physical activity was assessed with the International Physical Activity Questionnaire [IPAQ, short format (14)].

Overview

Participants visited the laboratory for a familiarization session involving voluntary maximum and explosive as well as evoked twitch contractions to facilitate group allocation. Thereafter two duplicate laboratory measurement sessions were conducted both pre (sessions 7–10 days apart prior to the first training session) and post (2–3 days after the last training session and 2–3 days later) 12 wk of unilateral knee extensor strength training. Axial T1-weighted MRI scans of the thigh were also conducted pre (5 days prior to the first training session) and post (2–3 days after the final training session). Training and testing were completed with the same isometric apparatus. Training for the ECT and SCT groups involved unilateral isometric contractions of both legs 3 times a week for 12 wk (36 sessions in total), whereas CON participants attended only the measurement sessions and maintained their habitual activity. All participants were instructed to maintain their habitual physical activity and diet throughout the study. Laboratory testing sessions involved recordings of the dominant leg isometric knee extension torque and surface EMG of the superficial quadriceps muscles during voluntary maximum and explosive contractions, as well as evoked maximum twitch and octet contractions (via electrical stimulation of the femoral nerve). Measurement sessions were at a consistent time of day and started between 1200 and 1900.

Training

After a brief warm-up of submaximum contractions of both legs, participants completed 4 sets of 10 unilateral isometric knee extensor contractions of each leg, with sets alternating between dominant and nondominant legs until 4 sets per leg had been completed. Each set took 60 s with 2 min between successive sets on the same leg. ECT involved short, explosive contractions with participants instructed to perform each contraction "as fast and hard as possible" up to ≥80% MVT for ~1 s and then relax for 5 s between repetitions (Fig. 1*A*). A computer monitor displayed RTD (10-ms time epoch) to provide biofeedback of explosive performance, with a cursor indicating the highest peak RTD achieved throughout the session; participants were encouraged to achieve a higher peak RTD with each subsequent contraction. The torque-time curve was also shown: first with a horizontal cursor at 80%MVT (target force) to ensure sufficiently forceful contractions and, second, on a sensitive scale highlighting baseline torque in order to observe and correct any pre-tension or countermovement.

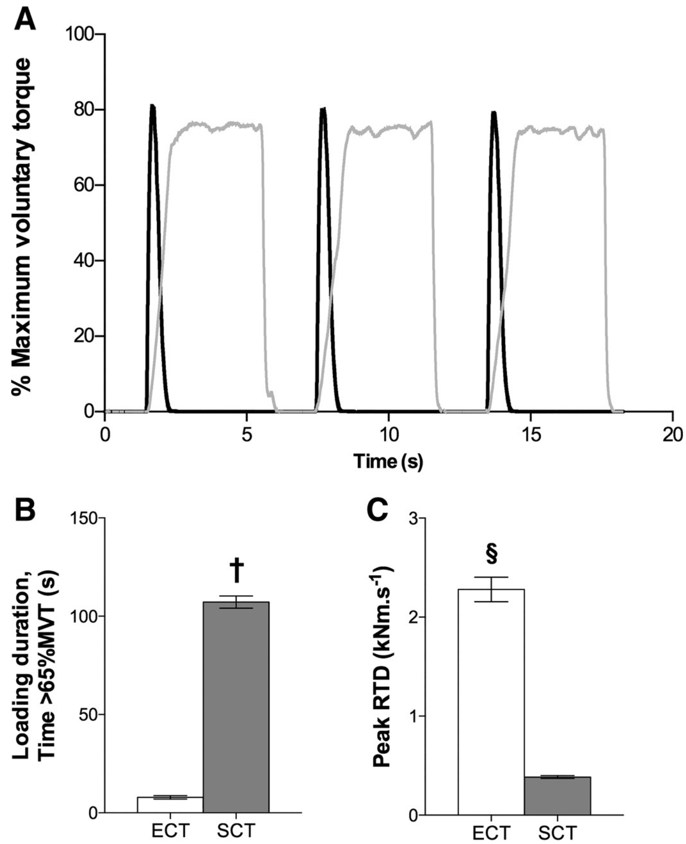

Fig. 1. *A*: example torque-time curves recorded during three isometric knee extension contractions for two participants performing either explosive-contraction strength training (ECT; black line) or sustained-contraction strength training (SCT; grey line). *B*: loading duration per training session measured by time >65% maximum voluntary torque (MVT) for ECT vs. SCT. *C*: peak rate of torque development (RTD, 50-ms epoch) during training contractions for ECT vs. SCT. Symbols indicate differences between training groups as determined from unpaired *t*-tests and are denoted as follows: †greater than ECT, §greater than SCT. Data are means ± SE.

SCT involved sustained contractions at 75% MVT, with 2-s rest between contractions. To control the RTD, these participants were presented with a target torque trace 2 s before every contraction and instructed to match this target, which increased torque linearly from rest to 75% MVT over 1 s before holding a plateau at 75%MVT for a further 3 s. All training participants (ECT and SCT) performed three maximum voluntary isometric contractions (MVCs, see below) at the start of each training week to reestablish MVT and prescribe training torques. Torque data from the first training session of *weeks 1, 6*, and *12* were analyzed for all training participants (i.e., ECT and SCT) to quantify peak loading magnitude (peak torque, mean of all repetitions), loading rate (peak RTD, 50-ms epoch, mean of all repetitions), and loading duration (defined as time >65%MVT per session).

Force and EMG Recording

Measurement and training sessions were completed in a rigid custom-made isometric dynamometer with knee and hip angles of 115° and 126° (180° = full extension), respectively. Adjustable straps were tightly fastened across the pelvis and shoulders to prevent extraneous movement. An ankle strap (35-mm-width reinforced canvas webbing) was placed ~15% of tibial length (distance from lateral malleolus to knee joint space), above the medial malleolus, and positioned perpendicular to the tibia and in series with a calibrated S-beam strain gauge (Force Logic, Swallowfield, UK). The analog force signal from the strain gauge was amplified (×370) and sampled at 2,000 Hz using an external analog-to-digital (A/D) converter (Micro 1401; CED, Cambridge, UK) and recorded with Spike 2 computer software (CED, Cambridge, UK). In offline analysis, force data were low-pass filtered at 500 Hz using a fourth-order zero-lag Butterworth filter (33), gravity corrected by subtracting baseline force, and multiplied by lever length, the distance from the knee joint space to the center of the ankle strap, to calculate torque values.

Surface EMG was recorded from the superficial quadriceps muscles [rectus femoris (RF), vastus lateralis (VL), vastus medialis (VM)] using a wireless EMG system (Trigno; Delsys, Boston, MA). Following skin preparation (shaving, abrading, and cleansing with 70% ethanol), single differential Trigno Standard EMG sensors (Delsys, Boston, MA), each with a fixed 1-cm interelectrode distance, were attached at six separate sites over the superficial quadriceps muscles at set percentages of thigh length above the superior border of the patella (RF 65 and 55%; VL 60 and 55%; VM 35 and 30%) and parallel to the presumed orientation of the underlying fibers. EMG signals were amplified at source (×300; 20- to 450-Hz bandwidth) before further amplification (overall effective gain, ×909) and sampled at 2,000 Hz via the same A/D converter and computer software as the force signal, to enable data synchronization. In offline analysis, EMG signals were corrected for the 48-ms delay inherent in the Trigno EMG system and band-pass filtered (6–500 Hz) using a fourth-order zero-lag Butterworth filter.

Pre and Post Measurement Sessions

Following a brief warm-up of the dominant leg [3 s contractions at 50% (×3), 75% (×3), and 90% (×1) of perceived maximum], measurements were completed in the following order.

Maximum voluntary contractions. Participants performed 3–4 MVCs and were instructed to "push as hard as possible" for 3–5 s and rest for ≥30 s between efforts. A torque-time curve with a horizontal cursor indicating the greatest torque obtained within that session was displayed for biofeedback, and verbal encouragement was provided during all MVCs. Knee extensor MVT was the greatest instantaneous torque achieved during any MVC or explosive contraction during that measurement session. Root-mean-square (RMS) EMG for a 500-ms epoch at MVT (250 ms either side) was calculated for each electrode site before averaging across the six sites to provide a whole quadriceps measurement ($QEMG_{MVT}$). In addition, RMS EMG at MVT was normalized to maximum M-wave (M_{MAX}) area (see below) from the corresponding EMG electrode site and then averaged across all quadriceps EMG sites.

Explosive voluntary contractions. Participants completed 10 explosive voluntary contractions. They were instructed to perform each contraction "as fast and hard as possible" for ~1 s, in order to exceed 80% MVT, and then relax for ≥15 s between contractions. Contractions with a change in baseline torque (pre-tension or countermovement) of >0.34 N·m in the 300 ms prior to contraction onset were discarded. The three best contractions (highest torque at 100 ms) were analyzed in detail for torque and EMG. Voluntary explosive torque was measured at 50, 100, and 150 ms from contraction onset (T_{50}, T_{100}, and T_{150}), before averaging across the three contractions. Explosive torque was also expressed relative to MVT to assess if explosive and maximum strength changed proportionally.

RMS EMG of each of the quadriceps sensor sites was measured over three time periods: 0–50, 0–100, and 0–150 ms from EMG onset of the first agonist muscle to be activated (see below), before averaging to produce overall quadriceps measurements ($QEMG_{0-50}$, $QEMG_{0-100}$, $QEMG_{0-150}$) for the three best contractions. RMS EMG values from each sensor were also normalized to both EMG_{MVT} and M_{MAX} area for that site before averaging. To decide whether to report absolute RMS EMG or RMS EMG normalized to M_{MAX}, the intraparticipant reproducibility of EMG_{MVT} for both EMG measures was assessed over the 12-wk intervention for CON (see below), and the most reproducible measure was used. The ratio of Voluntary T_{50}/Octet T_{50} (see below) was used as an additional measure of volitional neural efficacy during the voluntary explosive contractions.

During offline analysis, all torque and EMG onsets were identified manually by visual identification by one trained investigator using a systematic approach (46, 49) considered to be more valid than automated methods (49). Briefly, torque and EMG signals were initially viewed on an *x*-axis scale of 300 ms prior to the contraction and *y*-axis scales of 0.68 N·m (torque) or 0.05 mV (EMG) (46, 49) before zooming in to determine the instant of the last peak or trough before the signal deflected away from the envelope of the baseline noise.

Evoked twitch and octet contractions. A constant-current variable-voltage stimulator (DS7AH; Digitimer, Welwyn Garden City, UK), cathode probe (1-cm diameter; Electro-Medical Supplies, Wantage, UK), and anode electrode (7 × 10-cm carbon rubber electrode; Electro-Medical Supplies, Wantage, UK) were used to electrically stimulate the femoral nerve. The cathode and anode were coated with electrode gel and securely taped to the skin over the femoral nerve in the femoral triangle and over the greater trochanter, respectively. Cathode location was determined by delivering single electrical impulses (square wave pulses of 0.2-ms duration, ≥12 s apart) to identify the position that elicited the greatest submaximum twitch response. The current intensity was progressively increased until plateaus in peak twitch force and peak-to-peak M-wave amplitude were reached. Then three supramaximal twitch and M_{MAX} responses were evoked (15 s apart) at a higher current (≥50%) to ensure supramaximal stimulation. The following variables were averaged across the three supramaximal twitch contractions: peak twitch torque (Twitch Peak T), absolute torque (Twitch T_{50}) and torque expressed relative to Twitch Peak T (Relative Twitch T_{50}) at 50 ms after contraction onset, time from contraction onset to peak twitch torque (Twitch TPT), and the cumulative M_{MAX} area from EMG onset to the point where the signal returned to baseline for each of the six EMG sites.

During the second pre and first post measurement sessions only, octet contractions (8 impulses at 300 Hz) were evoked at progressive currents (≥15 s apart) until a plateau in the amplitudes of peak torque and peak RTD were achieved. Then, three discrete pulse trains (≥15 s apart) were delivered with a higher current (≥20% to ensure supramaximal stimulation) to evoke maximum octet contractions. Peak torque (Octet Peak T), absolute torque (Octet T_{50}) and torque expressed relative to Octet Peak T (Relative Octet T_{50}) at 50 ms after contraction onset, and time from contraction onset to Octet Peak T (Octet TPT) were averaged across the three maximum octet contractions. Because of the discomfort caused by the octet contractions a total of seven participants across the three groups were unable to tolerate this measurement.

Muscle Volume

A 1.5-T MRI scan of the dominant leg was made in the supine position at a knee joint angle of ~163° using a receiver eight-channel whole-body coil (Signa HDxt; GE). T1-weighted axial slices (5 mm thick, 0-mm gap) were acquired from the anterior superior iliac spine to the knee joint space in two overlapping blocks. Oil-filled capsules placed on the lateral side of the participants' thigh allowed alignment of the blocks during analysis. MR images were analyzed by two investigators using Osirix software (version 6.0; Pixmeo, Geneva, Switzerland). Pre and post scans of each participant were analyzed by the same investigator. The quadriceps (RF, VL, VM, and vastus intermedius) muscles were manually outlined in every third image (i.e., every 15 mm) starting from the most proximal image in which the muscle appeared. The volume of each muscle was calculated using cubic spline interpolation (GraphPad Prism 6; GraphPad Software). Total quadriceps volume ($QUADS_{VOL}$) was the sum of the individual muscle volumes. Inter- and intrarater reliability for $QUADS_{VOL}$ calculated from the repeated analysis of five MRI scans was 1.2 and 0.4%, respectively. Data from one participant were excluded because of excessive movement artifacts.

Data Analysis and Statistics

All data were anonymized prior to analysis. Reproducibility of the measurements over the 12-wk intervention period was calculated for CON (pre vs. post) as within-participant coefficient of variation [CV_W; (SD/mean) × 100]. MVT and $QEMG_{MVT}$ measurements from the duplicate test sessions were averaged to produce criterion pre and post values for statistical analysis; unless the CV_W for the MVT was ≥10% (calculated from duplicate test sessions), in which case the lowest MVT value and corresponding $QEMG_{MVT}$ were discarded. Mean T_{50}, T_{100}, and T_{150} and corresponding QEMG ($QEMG_{0-50}$, $QEMG_{0-100}$, and $QEMG_{0-150}$) from the duplicate test sessions were used as criterion pre and post values for statistical analysis, unless the CV_W (calculated from duplicate test sessions at the given time point) for T_{50} was ≥20%, in which case a weighted mean for all three explosive torque time points and corresponding QEMG measures was used.

All statistical analyses were performed using SPSS Version 22.0 (IBM, Armonk, NY). Data are reported as means ± SD; apart from within figures, where data are means ± SE for presentation purposes. One-way ANOVAs were conducted on all pretest variables to assess whether baseline differences existed between groups. Unpaired *t*-tests were used to assess differences in training variables (loading rate, duration, and magnitude) between ECT and SCT. Within-group changes were evaluated with paired *t*-tests. Comparison of between-group adaptations to the intervention were assessed with repeated-measures analysis of covariance [ANCOVA; group (ECT vs. SCT vs. CON) × time (pre vs. post)], with corresponding pre training values used as covariates. When group × time interaction effects displayed $P < 0.05$, then post hoc tests were conducted and included the calculation of effect size (ES) and least significant differences (LSD) of absolute changes (pre to post) between groups (i.e., ECT vs. SCT, ECT vs. CON, and SCT vs. CON). ES for absolute change data was calculated as previously detailed for between-subject study designs (30) and classified as follows: <0.20 = "trivial," 0.20–0.50 = "small," 0.50–0.80 = "moderate," or >0.80 = "large." LSD post hoc tests were produced from one-way ANCOVAs and were corrected for multiple comparisons (5). We considered there to be good evidence of between-group differences if both ES > 0.50 and LSD post hoc $P < 0.10$.

RESULTS

Group Characteristics at Baseline

At baseline no differences (ANOVA, P ≥ 0.767) were observed between groups for habitual physical activity (IPAQ: ECT 2,047 ± 1,081; SCT 2,135 ± 1,230; CON 2,321 ± 1,614 metabolic equivalent min/wk), age (ECT 25 ± 2; SCT 25 ± 2; CON 25 ± 3 yr), body mass (ECT 70 ± 10; SCT 71 ± 9; CON 72 ± 7 kg), or height (ECT 1.74 ± 0.07; SCT 1.75 ± 0.08, CON 1.76 ± 0.06 m). Similarly, no baseline differences were detected for functional, neural, intrinsic contractile properties, or muscle volume.

Reproducibility of Torque and EMG Measurements

The reproducibility of pre and post measures for the CON group over the 12-wk period was excellent for MVT, T_{100}, and T_{150} (CV_W 2.9, 4.4, and 4.9%, respectively), but poor for T_{50} (CV_W 15.7%). Absolute EMG_{MVT} (9.8%) had better CV_W than EMG_{MVT} normalized to M_{MAX} area (14.7%), and therefore absolute EMG data are presented. Twitch (Twitch T_{50}, Twitch Peak T, Relative Twitch T_{50}, Twitch TPT) and octet ($n = 11$, Octet T_{50}, Octet Peak T, Relative Octet T_{50}, Octet TPT) variables displayed excellent to good CV_W values (1.8–6.1%).

Training Quantification for ECT vs. SCT

Loading duration, quantified as time >65%MVT per session, was greater for SCT than for ECT (unpaired *t*-test $P < 0.001$; Fig. 1*B*). Conversely, ECT involved ~6-fold greater RTD per repetition than SCT (unpaired *t*-test $P < 0.001$; Fig. 1*C*). Peak loading magnitude was also slightly greater for ECT than for SCT (81 ± 4 vs. 75 ± 2%MVT; unpaired *t*-test $P < 0.001$).

Table 1. *Maximum voluntary torque (MVT) and explosive torque (absolute and relative to MVT) pre and post explosive-contraction strength training, sustained-contraction strength training, and control interventions, and explosive torque production expressed relative to MVT*

	ECT		SCT		CON		
	Pre	Post	Pre	Post	Pre	Post	ANCOVA Interaction (*P* Value)
Absolute (N·m)							
MVT	232 ± 27	272 ± 37‡	239 ± 48	295 ± 46‡	257 ± 49	259 ± 57	<0.001
T_{50}	43 ± 20	57 ± 23*	47 ± 21	47 ± 19	39 ± 19	42 ± 19	0.058
T_{100}	132 ± 25	155 ± 29†	138 ± 28	145 ± 22	138 ± 26	141 ± 27	0.036
T_{150}	177 ± 27	210 ± 35‡	182 ± 34	204 ± 25‡	192 ± 31	193 ± 35	<0.001
Relative (%MVT)							
T_{50}	18 ± 8	21 ± 7	20 ± 8	16 ± 7*	16 ± 7	16 ± 6	0.055
T_{100}	57 ± 8	57 ± 7	59 ± 10	50 ± 7†	55 ± 9	55 ± 9	0.007
T_{150}	76 ± 6	77 ± 6	77 ± 9	70 ± 7†	75 ± 8	75 ± 7	0.004

Data are means ± SD. Within-group effects of training were determined from paired *t*-tests and are denoted by. *$P < 0.05$, †$P < 0.01$, or ‡$P < 0.001$. ANCOVA interaction effects of time (pre vs. post) × group (ECT vs. SCT vs. CON) are reported. Post hoc comparisons of between-group changes are shown in Figs. 2 and 3. ECT, explosive-contraction strength training ($n = 13$); SCT, sustained-contraction strength training ($n = 16$); CON, control ($n = 14$); T, explosive torque (at 50-ms intervals from torque onset).

Voluntary Torque

MVT increased after ECT and SCT (both paired *t*-test $P < 0.001$), but not following CON ($P = 0.739$; Tables 1 and 4). The absolute increase in MVT was greater than CON for both ECT and SCT (both ES $\geq$ 2.06 "large," LSD $P < 0.001$) and 38% larger after SCT than ECT (ES = 0.69 "moderate," $P = 0.052$; Fig. 2).

Explosive torque increased at T_{50}, T_{100}, and T_{150} after ECT (paired *t*-test $P = 0.047$, $P = 0.008$, and $P < 0.001$, respectively; Tables 1 and 4). Whereas there were no changes in explosive torque after CON (paired *t*-test $0.420 \leq P \leq 0.847$), and only T_{150} increased following SCT ($P < 0.001$) with no change in T_{50} or T_{100} ($0.140 \leq P \leq 0.939$). Group comparisons revealed that ECT produced greater increases in explosive torque than SCT after 100 ms, but not after 150 ms (T_{100}: ES = 0.72 "moderate," LSD $P = 0.092$; T_{150}: ES = 0.54 "moderate," $P = 0.145$), and larger increases than CON from 100 ms onward (T_{100}: ES = 0.98 "large," $P = 0.042$; T_{150}: ES = 1.59 "large," $P < 0.001$). SCT resulted in greater increases than CON only at T_{150} (ES = 1.48 "large," LSD $P = 0.008$).

Relative explosive torque (%MVT), at all time points, decreased following SCT (paired *t*-test $0.004 \leq P \leq 0.032$; Table 1), but remained unchanged after ECT and CON ($0.344 \leq P \leq 0.984$). The decrease in relative explosive torque after SCT was greater than ECT (T_{100}: ES = 0.88 "large," $P = 0.015$; T_{150}: ES = 0.91 "large," $P = 0.006$) and CON (T_{100}: ES = 1.15 "large," $P = 0.016$; T_{150}: ES = 0.99 "large," $P = 0.022$; Fig. 3). Changes in relative explosive torque did not differ between ECT and CON (T_{100}: ES = 0.11 "trivial," $P = 0.844$; T_{150}: ES = 0.12 "trivial," $P = 0.547$).

Neural Drive

$QEMG_{MVT}$ increased, or had a tendency to increase, after SCT (paired *t*-test $P < 0.001$) and ECT ($P = 0.099$), but not CON ($P = 0.130$; Tables 2 and 4). The increase in $QEMG_{MVT}$ was greater than CON for both ECT (ES = 0.87 "large," LSD $P = 0.018$) and SCT (ES = 2.30 "large," $P < 0.001$) but was not different between ECT and SCT (ES = 0.36 "small," $P = 0.370$; Fig. 2). $QEMG_{0-50}$, $QEMG_{0-100}$, and $QEMG_{0-150}$ increased or had a tendency to increase after ECT (paired *t*-test $P = 0.089$, $P = 0.048$, and $P = 0.003$, respectively; Table 2). There were no changes in explosive QEMG measurements after CON, and only $QEMG_{0-150}$ increased after SCT (paired *t*-test $P = 0.009$; Table 2). Group comparisons showed ECT to increase explosive neural drive by more than CON at all time points ($QEMG_{0-50}$: ES = 0.85 "large," LSD $P = 0.036$; $QEMG_{0-100}$: ES = 1.07 "large," $P = 0.018$; $QEMG_{0-150}$: ES = 1.57 "large," $P < 0.001$; Fig. 3) and by more than SCT for $QEMG_{0-150}$ (ES = 0.58 "moderate," $P = 0.061$) but not earlier periods ($QEMG_{0-50}$: ES = 0.58 "moderate," $P = 0.101$; $QEMG_{0-100}$: ES = 0.46 "small," $P = 0.254$; Fig. 3). SCT increased $QEMG_{0-150}$ more than CON (ES = 1.20 "large," LSD $P = 0.021$), but this was not the case for earlier periods ($0.30 \leq$ ES ≤ 0.61 "small" to "moderate," $0.154 \leq P \leq 0.463$).

Relative explosive neural drive (as $\%QEMG_{MVT}$) for all time periods decreased after SCT (paired *t*-test $0.001 \leq P \leq 0.004$), but not following ECT or CON ($P \geq 0.395$; Table 2). After SCT the decreases in relative $QEMG_{0-100}$ were greater than ECT (ES = 0.59 "moderate," LSD $P = 0.086$) and CON (ES = 0.99 "large," $P = 0.045$), as was $QEMG_{0-150}$ vs. ECT (ES = 0.62 "moderate," $P = 0.066$).

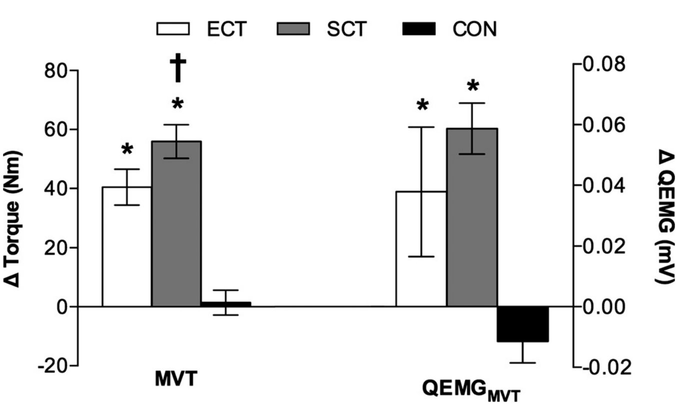

Fig. 2. Changes in maximum voluntary torque (MVT) and quadriceps EMG RMS amplitude at MVT ($QEMG_{MVT}$) during isometric knee extensions after explosive-contraction strength training (ECT), sustained-contraction strength training (SCT), and control (CON) interventions. Symbols indicate differences in the magnitude of pre to post changes where post hoc tests displayed both effect size >0.50 and least significant difference $P < 0.10$: *greater than CON, †greater than ECT. Data are means ± SE.

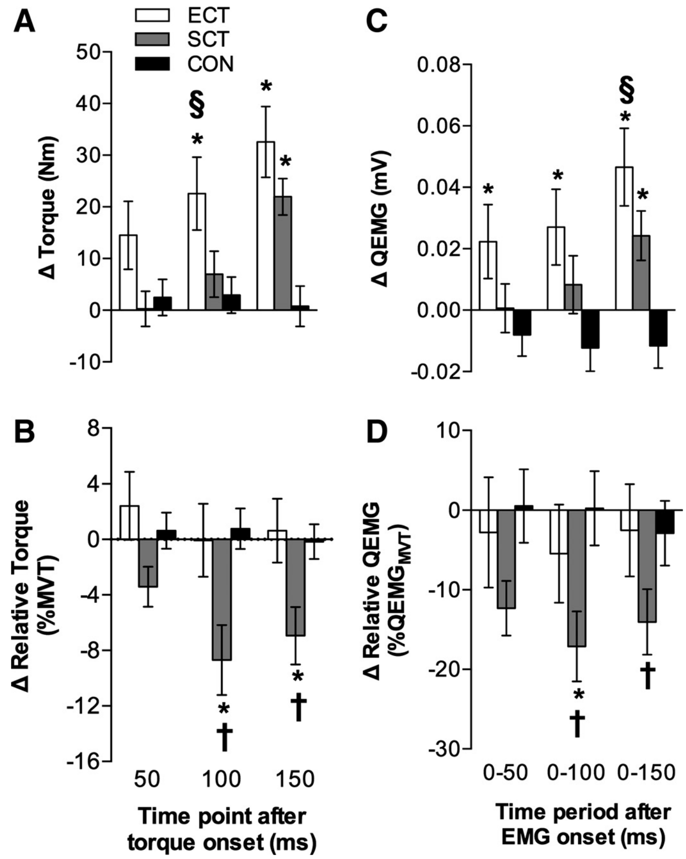

Fig. 3. Changes in torque (*A*), relative torque (%MVT) (*B*), quadriceps EMG RMS amplitude (*C*), and relative explosive quadriceps EMG RMS amplitude ($\%QEMG_{MVT}$) (*D*) during explosive isometric knee extensions after explosive-contraction strength training (ECT), sustained-contraction strength training (SCT), and control (CON) interventions. Symbols indicate differences in the magnitude of pre to post changes where post hoc tests displayed both effect size >0.50 and least significant difference $P < 0.10$: *different from CON, †different from ECT, §different from SCT. Data are means ± SE.

Table 2. *EMG recorded at maximum voluntary torque (EMG_{MVT}) and during explosive contractions (absolute and relative to EMG^{MVT}) pre and post explosive-contraction strength training, sustained-contraction strength training, and control interventions*

	ECT		SCT		CON		
	Pre	Post	Pre	Post	Pre	Post	ANCOVA Interaction (*P* Value)
Absolute (mV)							
EMG_{MVT}	0.21 ± 0.08	0.25 ± 0.10§	0.18 ± 0.07	0.23 ± 0.08‡	0.19 ± 0.07	0.17 ± 0.06	0.001
EMG_{0-50}	0.10 ± 0.06	0.12 ± 0.07§	0.08 ± 0.05	0.08 ± 0.05	0.08 ± 0.05	0.07 ± 0.04	0.033
EMG_{0-100}	0.16 ± 0.07	0.18 ± 0.08*	0.13 ± 0.05	0.13 ± 0.06	0.13 ± 0.06	0.12 ± 0.05	0.022
EMG_{0-150}	0.16 ± 0.07	0.21 ± 0.08†	0.14 ± 0.05	0.16 ± 0.06†	0.15 ± 0.06	0.14 ± 0.05	<0.001
Relative (%EMG_{MVT})							
EMG_{0-50}	49.2 ± 22.8	46.5 ± 16.6	46.6 ± 21.2	34.3 ± 14.4†	41.2 ± 17.2	41.8 ± 20.6	0.102
EMG_{0-100}	78.2 ± 17.6	72.7 ± 16.1	75.3 ± 23.2	58.1 ± 17.3†	71.8 ± 16.1	72.0 ± 23.6	0.031
EMG_{0-150}	83.6 ± 15.9	81.1 ± 13.0	81.2 ± 19.9	67.2 ± 15.9†	79.5 ± 15.1	76.7 ± 18.2	0.048

Data are means ± SD. Within-group effects of training were determined from paired *t*-tests and are denoted by *$P < 0.05$, †$P < 0.01$, ‡$P < 0.001$, or §$P \leq 0.10$. ANCOVA time (pre vs. post) × group (ECT vs. SCT vs. CON) interaction effects are also reported. Post hoc comparisons of between-group changes are shown in Figs. 2 and 3. ECT, explosive-contraction strength training (n = 13); SCT, sustained-contraction strength training (n = 16); CON, control (n = 14); EMG_{0-50}, EMG_{0-100}, and EMG_{0-150}, explosive contractions over three time periods from EMG onset (0-50, 0-100, and 0-150 ms).

Changes in relative explosive $QEMG_{0-100}$ and $QEMG_{0-150}$ did not differ between ECT and CON (0.02 ≤ ES ≤ 0.29, LSD 0.623 ≤ P ≤ 0.697).

Voluntary T_{50}/Octet T_{50} ratio appeared to increase after ECT (n = 12; pre 42 ± 20% vs. post 53 ± 19%) but did not reach statistical significance for the within-group change (paired *t*-test P = 0.122) or group × time interaction effect (ANCOVA, P = 0.107). No changes in the Voluntary T_{50}/Octet T_{50} ratio occurred after SCT (n = 14; pre 47 ± 15% vs. post 46 ± 19%; paired *t*-test P = 0.772) or CON (n = 11; pre 40 ± 18% vs. post 40 ± 17%; P = 0.816).

Intrinsic Contractile Properties and Muscle Size

Both training groups increased Octet Peak T (paired *t*-test ECT P = 0.001, SCT P = 0.015) and Octet TPT (ECT P = 0.017, SCT P < 0.001), with no change after CON (0.689 ≤ P ≤ 0.986; Table 3). Increases in Octet TPT were greater after SCT than CON (ES = 1.35 "large," LSD P = 0.009), but not for other comparisons (P ≥ 0.132, 0.42 ≤ ES ≤ 0.74 "small" to "large"). No changes in Octet T_{50} occurred after ECT, SCT, or CON (paired *t*-test 0.489 ≤ P ≤ 0.857), although Relative Octet T_{50} decreased after ECT and SCT (both paired *t*-test P = 0.001), but not CON (P = 0.638; Table 3). There was no ANCOVA interaction effect for Octet T_{50} (Table 3); however, the decreases in Relative Octet T_{50} after both ECT (ES = 1.36 "large," LSD P = 0.086) and SCT (ES = 1.37 "large," P = 0.003) were greater than CON, but these changes were similar after ECT and SCT (ES = 0.25 "small," P = 0.209; Fig. 4).

Twitch Peak T was unchanged in all three groups (paired *t*-test 0.127 ≤ P ≤ 0.821), although Twitch TPT was longer after both training interventions (0.009 ≤ P ≤ 0.047; Table 3), but not CON (P = 0.132). No changes in Twitch T_{50} occurred after ECT, SCT, or CON (paired *t*-test 0.489 ≤ P ≤ 0.857). Relative Twitch T_{50} decreased after SCT and ECT (paired *t*-test 0.008 ≤ P ≤0.032), but not CON (P = 0.919; Table 3).

$QUADS_{VOL}$ increased 8.1% after SCT from 1,820 ± 274 to 1,967 ± 316 cm^3 (n = 15; paired *t*-test P = 0.001), but not following ECT (n = 13; 1,770 ± 252 to 1,816 ± 286 cm^3; P = 0.247) or CON (n = 14; 1,891 ± 272 to 1,906 ± 261 cm^3; P = 0.550; Table 4). There was a group × time interaction effect for $QUADS_{VOL}$ (ANCOVA, P = 0.018), with the change in $QUADS_{VOL}$ after SCT being greater than that following CON (ES = 1.15 "large," LSD P = 0.021) and

Table 3. *Intrinsic contractile properties assessed by evoked torque production during octet and twitch contractions pre and post explosive-contraction strength training, sustained-contraction strength training, and control interventions*

	ECT		SCT		CON		
	Pre	Post	Pre	Post	Pre	Post	ANCOVA Interaction (*P* Value)
Octet							
Octet T_{50}, N·m	101 ± 12	105 ± 15	107 ± 14	106 ± 13	108 ± 14	109 ± 16	0.365
Octet Peak T, N·m	159 ± 20	174 ± 23†	171 ± 23	183 ± 24*	177 ± 26	177 ± 26	0.077
Relative Octet T_{50}, %	64 ± 5	60 ± 4†	63 ± 3	58 ± 3†	61 ± 2	61 ± 3	0.006
Octet TPT, ms	121 ± 7	127 ± 7*	121 ± 6	130 ± 6‡	123 ± 6	124 ± 5	0.010
Twitch							
Twitch T_{50}, N·m	37 ± 8	38 ± 11	39 ± 9	40 ± 8	43 ± 12	43 ± 10	0.865
Twitch Peak T, N·m	43 ± 9	45 ± 12	47 ± 11	50 ± 10	52 ± 14	52 ± 12	0.535
Relative Twitch T_{50}, %	86 ± 6	83 ± 6*	83 ± 5	81 ± 4†	82 ± 5	82 ± 3	0.157
Twitch TPT, ms	73 ± 8	76 ± 7*	73 ± 5	77 ± 4†	78 ± 4	76 ± 3	0.101

Data are means ± SD. Within-group effects of training were determined from paired *t*-tests and are denoted by *$P < 0.05$, †$P < 0.01$, or ‡$P < 0.001$. ANCOVA interaction effects of time (pre vs. post) × group (ECT vs. SCT vs. CON) are reported. Relative octet and twitch measures are expressed as percentage of peak torque during these contractions. Participant numbers are as follows. Octet variables: ECT, n = 12; SCT, n = 14; CON, n = 11. Twitch variables: ECT, n = 13; SCT, n = 16; CON, n = 14. ECT, explosive-contraction strength training; SCT, sustained-contraction strength training; CON, control.

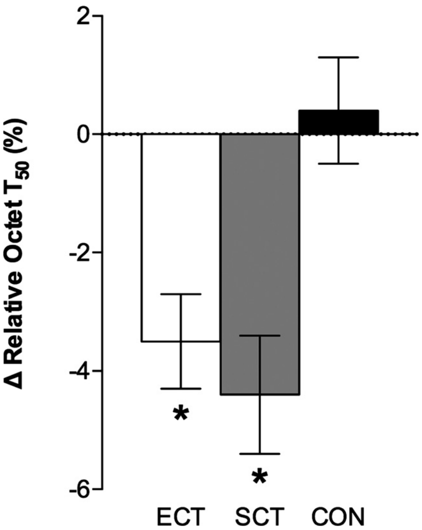

Fig. 4. Pre to post changes in Relative Octet T_{50} (the ratio between octet torque 50 ms after contraction onset and octet peak torque) after explosive-contraction strength training (ECT, n = 12), sustained-contraction strength training (SCT, n = 14), and control (CON, n = 11) interventions. Symbols indicate differences in the magnitude of pre to post changes where post hoc tests displayed both effect size >0.50 and least significant difference $P < 0.10$: *different from CON. Data are means ± SE.

~3-fold greater than after ECT (ES = 0.74 "moderate," P = 0.074; Fig. 5). Increases in $QUADS_{VOL}$ after ECT were not greater than CON (ES = 0.27 "small," LSD P = 0.552).

DISCUSSION

This study compared the specificity of functional adaptations to 12 wk of ECT vs. SCT and assessed underpinning neural, contractile, and hypertrophic adaptations contributing to these functional changes. MVT increased after both SCT and ECT, but these changes were greater after SCT (+23 vs. +17%). Increases in EMG_{MVT} were similar following SCT and ECT, while greater increases in $QUADS_{VOL}$ (+8.1 vs. +2.6%) suggest muscle size rather than neural drive explained the greater improvement in MVT after SCT than ECT. Improvements in early-phase explosive torque production (≤100 ms) only occurred after ECT (+17–34%), were greater than after SCT (at 100 ms), and appeared to be due to increased early-phase neural drive. ECT and SCT both improved explosive strength at 150 ms (+18% vs. +12%) with corresponding increases in neural drive likely explaining the enhancement in late-phase explosive torque production. Octet Peak T increased after training, but there were no changes in the intrinsic contractile explosive capability (Twitch and Octet T_{50}) as the time course of the evoked response (Octet and Twitch TPT as well as Relative Octet and Twitch T_{50}) decreased after both SCT and ECT, indicating a likely slowing of the muscle's contractile properties after both training interventions. Overall, the results support our hypothesis of distinct and specific functional changes (ECT > SCT for early-phase explosive strength; SCT > ECT for maximum strength), and these appeared to be due to distinct neural and hypertrophic, but not intrinsic contractile, adaptations.

Both ECT and SCT increased maximum strength, and by more than CON, but with greater increases after SCT (+23 vs. +17%). Maximum strength has been reported to increase by varying extents following both SCT (+11–36%) (1, 4, 9, 24, 40) and ECT (+7–25%) (7, 16, 48), yet this study is the first to directly compare the magnitude of maximum strength improvements after prolonged training with these different approaches. Loading duration (also referred to as time under tension) and loading magnitude have been suggested to be important training stimuli for maximum strength adaptation (15). Maximum strength improvements after ECT were ~70% of those after SCT, despite ECT involving only 7% of the loading duration (time >65%MVT) and thus considerably less effort and fatigue. In contrast, the loading magnitude of the two interventions in the current study were physiologically, if not statistically, quite similar (ECT 81% vs. SCT 75%). Overall, this provides evidence that loading magnitude rather than loading duration accounts for the majority of the maximum strength improvement following the first 12 wk of SCT and is the primary training stimulus. In this case, brief explosive contractions up to a high loading magnitude appear to be an efficient means of increasing maximum strength without the requirement for sustained muscular contractions. Furthermore, if loading magnitude is the primary stimulus for maximum strength gains, then it is possible that even higher loading magnitudes than those employed in the current study (i.e., >95%MVT), which may be achievable during very short contractions, could provide an even greater stimulus for enhancing maximum strength. The importance of loading magnitude for maximal strength gains may have application for optimizing training prescription of athletes and patient populations, in particular for patient groups where more sustained contractions may be problematic because of fatigue.

Neural drive at MVT increased more after both SCT and ECT than CON. Numerous previous studies have found neural drive at MVT (assessed with EMG) to increase after SCT interventions (24, 47); however, the current study is the first to

Table 4. *Summary of within-group changes from pre to post training in functional, neural, hypertrophic, and intrinsic contractile properties after explosive-contraction strength training, sustained-contraction strength training, and control interventions*

	ECT	SCT	CON
	Functional		
MVT, N·m	↑ +17%	↑ +23%	↔
Explosive T_{50}, N·m	↑ +34%	↔	↔
Explosive T_{100}, N·m	↑ +17%	↔	↔
Explosive T_{150}, N·m	↑ +18%	↑ +12%	↔
	Neural drive		
EMG_{MVT}, mV	↑ +18%	↑ +33%	↔
$EMG_{0–50}$, mV	↑ +23%	↔	↔
$EMG_{0–100}$, mV	↑ +17%	↔	↔
$EMG_{0–150}$, mV	↑ +28%	↑ +18%	↔
	Hypertrophy		
$QUADS_{VOL}$, cm^3	↔	↑ +8%	↔
	Contractile properties		
Octet Peak T, N·m	↑ +9%	↑ +7%	↔
Octet TPT, ms	↑ +5%	↑ +7%	↔
Twitch TPT, ms	↑ +4%	↑ +5%	↔

The directions of the changes are shown by ↑ or ↓ with the percentage change in the group mean also shown. Nonsignificant changes are indicated by ↔. ECT, explosive-contraction strength training; SCT, sustained-contraction strength training; CON, control.

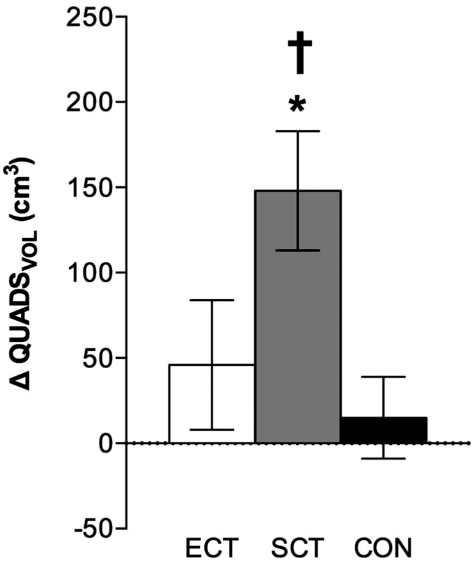

Fig. 5. Pre to post changes in total quadriceps muscle volume ($QUADS_{VOL}$) after explosive-contraction strength training (ECT, $n = 13$), sustained-contraction strength training (SCT, $n = 15$), and control (CON, $n = 14$) interventions. Symbols indicate differences in the magnitude of pre to post changes where post hoc tests displayed both effect size >0.50 and least significant difference $P < 0.10$: *greater than CON, †greater than ECT. Data are means ± SE.

show that short-duration explosive contractions can produce increases in neural drive at MVT, and this likely explained the efficacy of ECT for increasing MVT. In fact, there was no difference between ECT and SCT for this neural adaptation (EMG_{MVT}), indicating that loading magnitude rather than loading duration is the primary stimulus for increasing neural drive at MVT. Previous evidence suggests that increased motor unit firing frequency explains enhanced neural drive at MVT after training (27, 28), and this likely accounts for the improvement of both groups in the current study. In contrast, ECT did not stimulate an increase in muscle volume, and therefore, while ECT appears to be effective at enhancing neural aspects of maximum strength, it is relatively ineffective at stimulating hypertrophy. Whereas SCT did induce an increase in muscle volume, that was ~3-fold greater than after ECT (+8.1 vs. +2.6%). Thus hypertrophy was sensitive to loading duration, and this adaptation appears to explain the larger improvements in maximum strength for SCT vs. ECT. In this case, for longer-term training goals that are primarily reliant on hypertrophic, rather than neural, adaptations, loading duration may become the key training variable. These findings may have relevance for athletic and patient groups where increasing muscle volume is a primary training goal.

Early-phase (first 100 ms) explosive strength increased more after ECT than SCT. In contrast, later-phase explosive strength (T_{150}) was enhanced after both types of training. The improvements in T_{50} and T_{100} following ECT in the current study are consistent with our previous observation that early-phase explosive strength adaptations were highly specific to 4 wk of ECT vs. SCT (45) and demonstrate this to also be the case with more prolonged (12 wk) training. The loading rate (peak RTD) during the short explosive contractions of ECT was almost sixfold greater than SCT, and therefore high loading rates, rather than loading magnitudes (similar for ECT and SCT) or duration (greater for SCT), appear to be critical for enhancing early-phase explosive strength. Previous investigations of ECT have consistently reported improvements in explosive strength (7, 16, 22, 23, 45, 48). In contrast, training regimes similar to SCT in the current study have demonstrated both enhanced (1, 9, 13, 15a, 29, 44) and unchanged (10, 40, 47) explosive strength. The inconsistent changes in explosive strength in these studies may be partly explained by the variable training instructions provided [e.g., an explosive component (13, 40, 44); no explosive component (9, 15a, 47); or unclear (1, 29)]. In our laboratory, we have consistently found no increase in early-phase explosive strength after 4 wk (47) and now 12 wk of isometric SCT, as well as 3 and 12 wk of dynamic SCT with isoinertial lifting and lowering (10, 19). Therefore, for early-phase explosive strength gains, a specific explosive component to the training, involving contractions starting from a low/resting level and performing the rising phase of contraction at a high rate, appears to be important.

Neural drive during the early phase of explosive contractions increased only after ECT (EMG_{0-50} and EMG_{0-100}; Table 2), and these changes were greater than for CON, but not SCT. The Voluntary T_{50}/Octet T_{50} ratio, which provides an alternate measure of early-phase neural drive, increased from 42 to 53% after ECT, but this was not statistically significant because of the large variability in response between participants. Qualitatively, however, the group-level Voluntary T_{50}/Octet T_{50} ratio response was notably larger after ECT (+26%) than SCT (−2%) or CON (0%). Later-phase neural drive (EMG_{0-150}) was increased after both types of training (Table 2). Overall, the current study shows that neural adaptations during the early phase of explosive contraction that are specific to ECT, which had previously only been documented for a 4-wk training period (45), are still present following a more prolonged intervention. Improvements in early-phase explosive torque production (T_{50} and T_{100}) occurred after ECT without increases in muscle size or early-phase intrinsic contractile capacity for explosive torque production (Octet and Twitch T_{50}), supporting the importance of neural drive adaptations for the enhancement of early-phase explosive strength following training.

Explosive torque and EMG expressed relative to corresponding maximum force and EMG were unchanged with ECT but decreased with SCT (Tables 1–2 and Fig. 3, *B* and *D*); highlighting further the comprehensive adaptations to ECT (i.e., proportional increases in both explosive and maximum torques and corresponding neural drive) but not SCT (i.e., increases in only maximum torque and neural drive). These changes after ECT partly oppose our previous findings of a greater proportion of maximum strength and EMG being expressed during explosive contractions after 4 wk of ECT (45), which may be explained by the apparent slowing of the contractile properties and/or greater changes in MVT, and neural drive at MVT, after ECT in the current study. Neurologically, increases in instantaneous motor unit discharge rates and the number of motor units able to produce high discharge rates during explosive contractions and a degree of transfer of these adaptations to maximum contractions may explain the increases in explosive (early and late phase) and maximum neural drive after ECT (17, 50). In contrast, the low loading rates (385 $N \cdot m \cdot s^{-1}$) but high loading magnitudes (75% MVT) with SCT may have only stimulated adaptations in discharge rate during the production of larger torques (i.e., the late phase of explosive torque production and the plateau phase of contraction) (27, 28).

Overall, ECT denoted by brief contractions with high RTD produced a wider range of functional adaptations than SCT, with improvements in early- and late-phase explosive strength, as well as maximum strength (Table 4). In contrast, SCT only improved maximum strength and late-phase explosive strength (Table 4). The substantially lower loading duration of ECT (7% of SCT) makes this a less-demanding training modality compared with SCT, which may be preferentially tolerated by musculoskeletal patients and older adults. Future research should *1*) investigate whether ECT may be preferentially tolerated by musculoskeletal patients and older adults and *2*) also evaluate the efficacy of ECT, and underpinning neuromuscular adaptations, in an isoinertial dynamic training model that is more widely accessible.

The within-group increase in Octet Peak T following both ECT and SCT demonstrated an increase in the maximum contractile capacity of the muscle-tendon unit, although between-group differences were not detected. In contrast, Twitch Peak T was unresponsive to training even after SCT that induced hypertrophic adaptations. Changes in the time course of evoked responses (Octet and Twitch TPT as well as Relative Octet and Twitch T_{50}) indicated an overall slowing of the contractile properties of the muscle-tendon unit after both types of training. This apparent slowing of the intrinsic contractile properties is likely due to decreased expression of myosin heavy-chain type IIX fibers after training (2, 3, 11). For SCT, the slower contractile properties may explain why, during the early phase of explosive voluntary contraction, relative torque decreased and absolute torque remained unchanged, despite increases in maximum strength. After ECT the slower contractile properties may explain why relative explosive torque remained unchanged despite improved neural drive and why the increases in absolute explosive torque were more modest than might have been expected on the basis of our previous 4-wk training study (45), when presumably any potentially negative morphological changes would have been more limited. Furthermore, even after the brief explosive contractions of ECT, the intrinsic contractile properties of the muscle were slowed, which might suggest that these changes may be unavoidable with strength training of previously untrained individuals.

In conclusion, functional, neural, and hypertrophic adaptations showed marked training specificity. ECT produced wide-ranging functional adaptations with increases in early- and late-phase explosive and maximum strength due to neural adaptations, and the very low loading duration of ECT (7% of SCT) makes this a substantially less demanding training modality that may be preferentially tolerated by musculoskeletal patients and older adults. SCT produced a greater improvement in maximum strength, but no improvements in early-phase explosive strength. The similar changes in neural drive at MVT after ECT and SCT (despite a lesser gain in MVT following ECT) indicate that this adaptation is largely dependent on loading magnitude. In contrast, the ~3-fold greater hypertrophy after SCT than ECT indicates that this adaptation is dependent on loading duration. Improvements in early-phase explosive torque production (≤100 ms) appear to rely on a high RTD to induce specific neural adaptations. Finally, an apparent slowing of the intrinsic contractile properties of the muscle-tendon unit after both types of training likely compromises improvements in explosive strength.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

T.G.B., G.J.M., T.M.M.-W., N.A.T., and J.P.F. conception and design of research; T.G.B., G.J.M., and T.M.M.-W. performed experiments; T.G.B. and T.M.M.-W. analyzed data; T.G.B., N.A.T., and J.P.F. interpreted results of experiments; T.G.B. prepared figures; T.G.B., N.A.T., and J.P.F. drafted manuscript; T.G.B., G.J.M., T.M.M.-W., N.A.T., and J.P.F. edited and revised manuscript; T.G.B., G.J.M., T.M.M.-W., N.A.T., and J.P.F. approved final version of manuscript.

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