Control and Function of Arm Swing in Human Walking and Running

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Control and function of arm swing in human walking and running

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SUMMARY

We investigated the control and function of arm swing in human walking and running to test the hypothesis that the arms act as passive mass dampers powered by movement of the lower body, rather than being actively driven by the shoulder muscles. We measured locomotor cost, deltoid muscle activity and kinematics in 10 healthy adult subjects while walking and running on a treadmill in three experimental conditions: control; no arms (arms folded across the chest); and arm weights (weights worn at the elbow). Decreasing and increasing the moment of inertia of the upper body in no arms and arm weights conditions, respectively, had corresponding effects on head yaw and on the phase differences between shoulder and pelvis rotation, consistent with the view of arms as mass dampers. Angular acceleration of the shoulders and arm increased with torsion of the trunk and shoulder, respectively, but angular acceleration of the shoulders was not inversely related to angular acceleration of the pelvis or arm. Restricting arm swing in no arms trials had no effect on locomotor cost. Anterior and posterior portions of the deltoid contracted simultaneously rather than firing alternately to drive the arm. These results support a passive arm swing hypothesis for upper body movement during human walking and running, in which the trunk and shoulders act primarily as elastic linkages between the pelvis, shoulder girdle and arms, the arms act as passive mass dampers which reduce torso and head rotation, and upper body movement is primarily powered by lower body movement.

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Key words: arm swing, walking, running, passive dynamics, tuned mass dampers.

INTRODUCTION

Arm swing is a distinctive readily apparent characteristic of human walking and running. Our arms tend to swing out of phase with our legs, the right arm swinging forward with the left leg and *vice versa*. Although it has long been established that the arms do not swing as simple, unrestrained pendulums (Elftman, 1939; Fernandez Ballesteros et al., 1965; Jackson et al., 1978; Hinrichs, 1987; Ohsato, 1993; Webb et al., 1994; Gutnik et al., 2005), the extent to which the shoulder muscles actively drive the arms, and the effect of arm swing on stability and economy during walking and running are poorly understood. In this paper, we examined the control of arm swing during walking and running, and investigated the effect of restricting arm swing on stability and metabolic cost.

In a seminal study examining the movements of the torso and arms during walking, Elftman suggested that the arms did not move as simple pendulums, but instead were driven by muscle activation in the shoulder (Elftman, 1939). Fernandez Ballesteros and colleagues expanded upon this work, using indwelling electrodes to measure muscle activity in the anterior, intermediate and posterior deltoid during walking, and confirmed that arm movement was accompanied by activity of the deltoid muscle, particularly during retraction (Fernandez Ballesteros et al., 1965). Retraction of the shoulder was associated with firing of the posterior deltoid and, to a lesser extent, protraction of the shoulder was associated with anterior deltoid activity (Fernandez Ballesteros et al., 1965). Further, Fernandez Ballesteros and colleagues showed that the shoulder muscles fire even when the arm is restrained during walking (Fernandez Ballesteros et al., 1965), suggesting that the neural control of arm swing may be controlled by a locomotor pattern generator, and is perhaps an evolutionary hold-over from a quadrupedal past, a view supported by other workers (e.g. Gray, 1944; Jackson et al., 1978).

Functionally, arm swing is often considered to be a mechanism for counteracting free vertical moments (i.e. torque about the body's vertical axis) imparted by the swinging legs. Elftman first proposed this mechanism for walking, showing that the angular acceleration of the arms was equal to that of the torso but in the opposing direction (Elftman, 1939). Hinrichs provided similar evidence for running, showing that the horizontal angular momentum of the upper and lower body were of equal magnitude and in opposing directions, resulting in a net angular momentum near zero for the entire body (Hinrichs, 1987; Hinrichs, 1990). More recently, Herr and Popovic (Herr and Popovic, 2008) showed that net angular momentum in all axes is kept remarkably close to zero during walking, and provided further evidence that arm moments serve to cancel lower limb moments about the body's vertical axis [figure 5C in Herr and Popovic (Herr and Popovic, 2008)]. These results are consistent with those of Li and colleagues, which showed that the free vertical moments produced by the stance limb during walking are higher when the arms are restrained from swinging (Li et al., 2001). Presumably, these greater vertical moments result from the absence of counteracting arm swing. It has also been suggested that restricting arm swing affects the metabolic cost of locomotion. Anderson and Pandy (Anderson and Pandy, 2001), in comparing their forward dynamics simulation of human walking with experimental data from human subjects, suggested that the high cost

of walking observed in their simulation resulted from the lack of arm swing in their model.

Together with data on muscle activity (Fernandez Ballesteros et al., 1965), these studies suggest that arm swing is largely driven by muscle activity in the shoulder, and serves an important role in maintaining stability during walking and especially during running. However, an alternative hypothesis is that arm swing is largely a passive response to the forces exerted on the torso by the swinging of the legs. According to this model, horizontal torques imparted on the pelvis by the swinging legs are transferred up the spinal column to the shoulder girdle, and then to the arms. Tonic or stabilizing muscle activity in the trunk and shoulder, along with ligamentous and other connective tissues, cause these elements to act as elastic elements or springs, an idea proposed by Fernandez Ballesteros and colleagues (Fernandez Ballesteros et al., 1965); the forces exerted by these anatomical 'springs' will increase in proportion to their angular displacement, or torsion. The direction of the torque transmitted through the trunk will alternate (clockwise/anti-clockwise) with each step as the legs swing in turn. The inertia of the torso and arms will tend to resist these forces, causing a time lag between movement in the pelvis and movement in the shoulder girdle. As a result, the shoulder girdle and arms will have the same oscillating frequency as the legs and pelvis, but will rotate out of phase with the legs.

Viewing arm swing as a passive, emergent property of human walking and running fits well with recent work demonstrating the self-stabilizing, 'passive-dynamic' nature of lower limb movement during walking (Collins et al., 2005). In fact, even simple physical models can develop human-like arm swing in response to leg swing (see supplementary material Movie 1). A passive model for arm swing would also have the advantage of being self-tuned, with greater leg accelerations leading to greater arm accelerations. Importantly, the effect of arm swing predicted by a passive model is similar to that suggested by active models, with the arms acting as mass dampers (see below), and angular acceleration of the upper body canceling horizontal angular accelerations by the swinging legs and maintaining whole-body net angular acceleration near zero. Lieberman and colleagues (Lieberman et al., 2007; Lieberman et al., 2008) have recently suggested that the arms act as mass dampers to minimize head pitch in the sagittal plane.

Here, we examined the control and function of arm swing in human walking and running. First, we tested the hypothesis that the arms act as mass dampers that decrease the amplitude of upper body rotation about the vertical axis. We then investigated the control of arm swing, testing predictions of the passive arm swing hypothesis against those of an active arm swing hypothesis, in which arm swing is driven by the shoulder muscles. We measured muscle activity, kinematics and oxygen consumption during walking and running in a sample of humans. The moment of inertia of the arms was decreased by asking subjects to run with arms folded across their chest, or increased by adding weights at the elbow. We expected arm swing in humans to behave as a mass-damped system, with changes in the moment of inertia of the arms leading to predictable changes in upper body rotation. Further, we predicted that the arms would act as passive mass dampers, with the energy for arm swing ultimately derived from movement of the lower body, and the trunk and shoulders acting as damped spring elements. Finally, to examine the effect of normal arm swing in maintaining stability, we examined the effect of restraining the arms on locomotor kinematics, footfall variability and the energetic cost of walking and running.

Modeling arms as mass dampers

In mechanical systems exposed to vibration or other external forces, several approaches can be used to decrease the amplitude of displacement of the principle mass (see Soong and Dargush, 1997). Systems for decreasing the amplitude of movement are generally termed energy dissipation systems, or dampers, and can be classed as passive or active. Passive dampers are those which impart no energy into the system, instead using the energy of the system to decrease movement of the principle mass (Symans and Constantinou, 1999). For example, frictional dampers convert energy in the system to heat, reducing energy and movement in the principle mass (see supplementary material Movie 1) (Soong and Dargush, 1997). Tuned mass dampers (Soong and Dargush, 1997) decrease movement of the principle mass by attaching an auxiliary mass using a damped spring (Fig. 1A). The effectiveness of passive tuned mass dampers is a complex function of the stiffness and damping constants of the damped spring by which they are attached but, generally, effectiveness is increased (i.e. movement of the principle mass is minimized) when the auxiliary mass is increased, and when the natural frequency of the auxiliary mass is below that of the principle mass (Soong and Dargush, 1997).

Active damping with auxiliary masses is also an effective strategy for minimizing displacement of a principle mass. Active mass damping differs from passive damping in that the auxiliary mass is attached with an active controller, so that the auxiliary mass can impart energy into the system, effectively pushing or pulling on the principle mass to stabilize it (Symans and Constantinou, 1999). Notably, both active and passive mass dampers can be effective over a range of oscillation frequencies (Soong and Dargush, 1997; Symans and Constantinou, 1999).

In the body, the torso is the principle mass whose angular displacement must be controlled. The hypothesis that the arms act as mass dampers for the torso thus leads to three predictions. First, since the effectiveness of mass dampers generally increases with their mass (Soong and Dargush, 1997), decreasing the moment of inertia of the arms (the auxiliary mass) about the vertical (z) axis (Fig. 2) as in the no arms condition is expected to result in greater rotation of the torso (the principle mass). Conversely, increasing the moment of inertia of the arms in the arm weights condition is expected to decrease torso rotation. A second, related prediction is that these changes in torso rotation should result in similar changes in head yaw, since the head is modeled as a mass attached to the torso *via* a damped spring (Fig. 1). Third, changes in the moment of inertia of the upper body (i.e. in the no arms or arm weights conditions) are predicted to have measurable effects on the phase differences in the movement of the pelvis and shoulder girdle. Increasing the moment of inertia of the arms, and therefore the upper body moment of inertia, is expected to lengthen the lag between pelvic and shoulder rotation, while decreasing the upper body moment of inertia is expected to shorten the lag between pelvic and shoulder movement.

Passive arm swing model

The passive arm swing hypothesis proposes that the upper body behaves like a passive mass-damped system (Soong and Dargush, 1997; Symans and Constantinou, 1999), in which all energy in the system derives from the swinging legs, and the spinal column and shoulders act as damped springs (Fig. 1B). This hypothesis leads to the following predictions. First, angular acceleration of the upper torso is predicted to increase with angular displacement between the pelvis and shoulder girdle (Fig. 1B). That is, as torsion of the spinal column increases, the force exerted by this spring-like

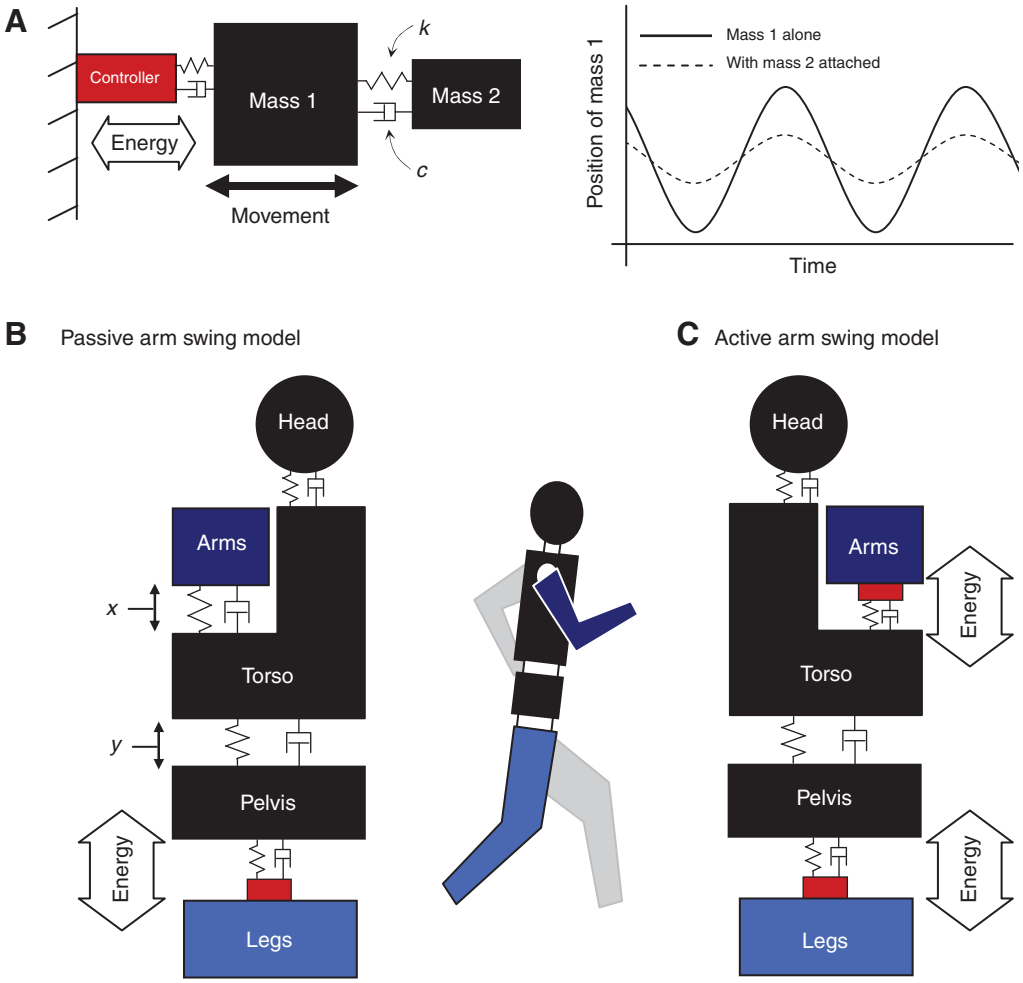

Fig. 1. Schematic diagram of passive and active arm swing hypotheses. (A) Simple mass damper (see Soong and Dargush, 1997). Oscillating forces applied by a controller (red element) to the principle Mass 1 will tend to move it (solid line in position plot); the attachment of an auxiliary Mass 2 using a damped spring can decrease the amplitude of movement of Mass 1 (dashed line in position plot); the effectiveness of the damping is a function of the spring stiffness *k* and damping constant *c*, and is proportional to Mass 2. (B) In the passive arm swing model, oscillating moments from the swinging legs tend to accelerate the pelvis and other body segments in turn; all energy in the system is generated by the legs. The arms act as an auxiliary mass which damps movement of the torso (and head). Shoulder and arm accelerations are predicted to increase with angular displacement of the trunk (*y*) and shoulder (*x*), respectively. (C) In the active arm swing model, energy into the system comes from both the swinging legs and the shoulder muscles driving the arms. Accelerations of the pelvis and torso are expected to be negatively correlated (i.e. in opposition). Since forces of the shoulder muscles will accelerate both the arm and torso masses, albeit in opposing directions, arm acceleration is predicted to be negatively correlated with shoulder acceleration. In both passive and active models, oscillation of the torso and head will increase if arms are removed. Note that these systems (B and C) are rotational in nature, but are rendered as linear systems here for clarity.

element will increase, resulting in greater acceleration of the shoulder girdle. Second, the angular acceleration of the arm is predicted to increase with angular displacement at the shoulder (i.e. the angle of the upper arm segment relative to vertical), just as the force generated by a spring increases with its displacement (Fig. 1B). In this way, rotation of the shoulders in the transverse plane is expected to result in arm swing in the sagittal plane. For example, as the shoulder girdle rotates and the right shoulder translates anteriorly, the arm will tend to remain in place following Newton's first law. Thus, the right arm will appear to swing posteriorly relative to the right shoulder, until the angular displacement of the shoulder is sufficient to swing the arm forward (protraction). As the right arm swings forward, the right shoulder will begin to translate posteriorly as the torso rotates with the next step, resulting again in angular displacement of the shoulder and acceleration of the arm. The shoulder musculature is expected to act as a spring-like element, translating angular displacement into torque. Thus a third prediction of the passive model is that the anterior and posterior portions of the deltoid will fire together, acting to stabilize the shoulder.

Active arm swing model

The active arm swing hypothesis proposes that arm swing is an active mass damping mechanism in which the arms (an auxiliary mass) are driven by the shoulder muscles acting as controllers in order to decrease the amplitude of torso rotation (Fig. 1B). Since the arm and torso are attached at the shoulder, anterior acceleration of the arm in the sagittal plane will lead to posterior acceleration of the shoulder and torso following Newton's third law: protraction of the right arm will tend to accelerate the right shoulder posteriorly, while retraction of the left arm will force the left shoulder anteriorly, thereby translating sagittal plane accelerations of the arms into

transverse plane angular accelerations of the shoulders and torso. Thus the primary prediction of the active arm swing hypothesis is that increased anterior angular acceleration of the arm in the sagittal plane will result in increased posterior angular acceleration of the shoulder girdle in the transverse plane. Second, anterior and posterior deltoid fibers are expected to fire alternately, acting as agonists driving angular acceleration of the arm at the shoulder. Third, angular accelerations of the pelvis and shoulder girdle are predicted to be similar in magnitude but opposite in direction, as the upper body is driven to counteract vertical free moments produced by the swinging legs.

Stability and cost

To examine the function of arm swing in maintaining stability, we tested the effect of removing arm swing (no arms condition) on footfall variability and metabolic cost. If arm swing is critical for maintaining stability, then removing arm swing as in the no arms condition is expected create stability problems during walking and especially running, resulting in greater variability in footfall placement (Fig. 1B). Similarly, while the relationship between muscular work and metabolic cost is complex (Cavagna and Kaneko, 1977; Willems et al., 1995), if the muscular work is done to compensate for decreased stability in the no arms condition, the metabolic cost of locomotion in the no arms condition is expected to be greater relative to control walking and running (see Anderson and Pandy, 2001). In contrast, if the upper body acts as a passive mass-damped system, then stability and cost should remain unchanged in the no arms condition, with the energy imparted by the swinging legs dissipated through greater excursion of the pelvis, torso and head.

MATERIALS AND METHODS

Sample

Ten recreationally fit, healthy adult human subjects (six male, four female, mean ± s.d. body mass 61.9±14.1 kg) with no apparent gait abnormalities participated in this study. Subjects gave their informed consent prior to participation. Washington University approval was obtained prior to the study, and institutional guidelines were followed throughout. Subjects wore spandex shorts, t-shirts or tank tops, and their personal running shoes throughout the experiment.

Kinematics and muscle activity

Small (1 cm diameter) spherical reflective markers were adhered to the body using double-sided tape, and the position of these markers was tracked using an infrared camera system (Vicon®; Centennial, CO, USA) recording at 200 frames s^{-1}. Markers were placed on the following landmarks and locations: forehead (two markers), right and left acromia, right elbow, right wrist, right and left anterior superior iliac spines, right greater trochanter, right knee, right ankle (lateral malleolus), right and left heels, and right and left first toe (Fig. 1). All markers were adhered directly to the skin, except those for the toe and heel, which were adhered to the subjects' shoes.

Anterior and posterior deltoid activity was recorded using self-adhering surface electrodes (Ambu® Blue Sensor, Glen Burnie, MD, USA) and an electromyography (EMG) system (RunTech® Myopac Jr, Mission Viejo, CA, USA). Subjects wore a light (320 g) amplifier unit that transmitted conditioned EMG signals along a fiber optic cable to a receiver. Analog signals were then passed through the Vicon MX Control A/D board and recorded at 4000 Hz in Vicon Nexus software, simultaneously with the kinematic data. Electrode placement was determined by palpation and confirmed by having the subject flex anterior and posterior portions of the deltoid individually against resistance while the EMG signal was observed. Although other muscles may also serve as shoulder flexors and extensors (e.g. triceps, biceps, latissimus dorsi), we focused on the deltoids here, since they have been shown to be important in this role during walking (Fernandez Ballesteros et al., 1965). Additionally, other shoulder flexors serve multiple roles, such as elbow flexion and extension or arm rotation, making their action difficult to characterize.

After being fitted with the EMG sensors and reflective markers, subjects performed an arm pump trial, in which they stood in place and swung their arms back and forth as during normal running. Next, after warming up on the treadmill (Sole Fitness F85, Jonesboro, AR, USA), subjects performed a series of walking and running treadmill trials for a range of speeds and experimental conditions. In the control condition, subjects walked normally at three speeds (1.0, 1.5 and 2.0 m s^{-1}) and ran normally at three speeds (2.0, 2.5 and 3.0 m s^{-1}). In the arm weight condition, these walking and running speeds were repeated, while the subject wore a 1.2 kg 'ankle-weight' style weight on each arm, just proximal to the elbow. Finally, in the no arms condition, walking and running speeds were repeated again, with the subject instructed to keep their arms folded tightly across their chest. Note that the moment of inertia of the arms and upper body is increased in the arm weights condition, and decreased in the no arms condition, but the magnitude of change is likely to be different between conditions and among subjects.

Data analysis

Mean contact time (i.e. step duration), stride period and stride frequency were determined from five strides for each kinematic trial. Contact time was measured as the time between heel strike (the first kinematic frame showing heel–ground contact) and toe-off (the last kinematic frame showing foot–ground contact). Stride period was measured as the time between two consecutive right heel strikes.

Marker position data were filtered in Matlab® (MathWorks Inc., Natick, MA, USA) using a fourth-order, zero-lag Butterworth filter with a low-pass cut-off set at 10 Hz. Filtered data were then used to calculate angle, angular velocity (deg. s^{-1}) and angular acceleration (deg. s^{-2}) for different body segments. Angular displacements for the head, shoulder girdle and pelvis were calculated in the transverse plane using the two forehead markers, right and left acromia markers, and right and left anterior superior iliac spine markers, respectively (Fig. 1). For the right arm, the locations of the acromium, elbow and wrist markers were used to determine the location of the whole arm center of mass relative to the shoulder marker following Winter (Winter, 2005). This point mass was then used to determine the angular displacement of the arm relative to the shoulder joint in the sagittal plane.

EMG signals were band-pass filtered in Matlab® using a fourth-order, zero-lag Butterworth filter with cut-offs at 60 and 300 Hz. Filtered signals were then processed using Thexton's randomization method (Thexton, 1996). The signal was recitified and binned following Winter (Winter, 2005) using a 0.01 s reset integral. Thextonization requires a threshold, set at 1% of the maximum integrated signal. The number of times the signal rose above this threshold ('runs') was determined for each 8 s trial. The threshold was then raised by 0.5% of the maximum integrated signal and the number of runs was found. This process was repeated, each time raising the threshold by 0.5% of the maximum integrated signal, until the threshold was equal to the maximum magnitude. The signal was then randomized, and the threshold method was repeated on the randomized signal. The number of runs in the randomized signal was subtracted from the number of runs in the original signal, and

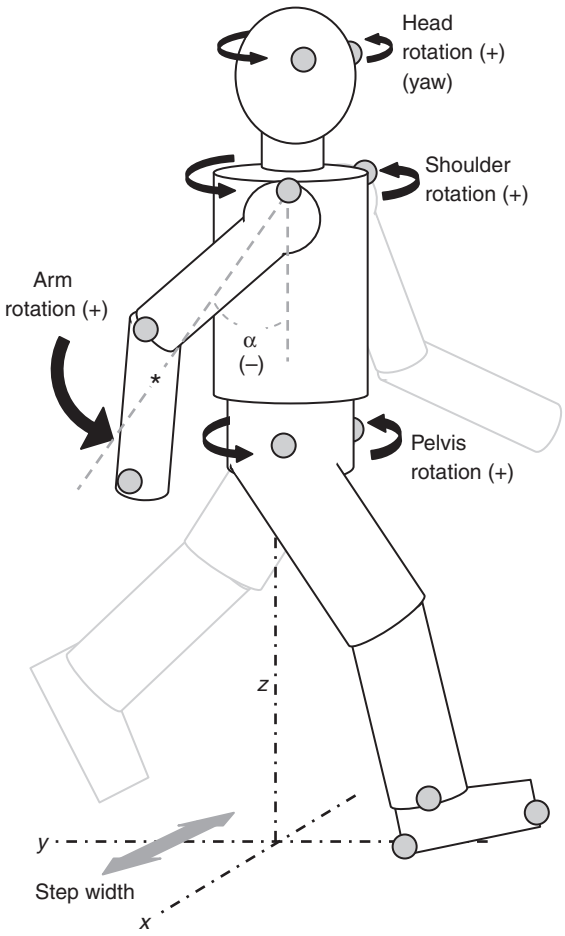

Fig. 2. Schematic diagram of the reference frame and kinematic variables. Rotation of the head, shoulders and pelvis in the transverse (*x*–*y*) plane about the vertical (*z*) axis was measured using reflective markers (gray circles) with reference to the *x*-axis; arrows indicate positive rotation. Trunk torsion was measured as the rotation of the pelvis relative to the shoulders. Arm rotation was measured in the sagittal (*y*–*z*) plane using the reconstructed arm center of mass (*) and shoulder relative to vertical; arrow indicates positive rotation. Angular displacement of the shoulder (α) was defined as negative when the arm was retracted (as shown), positive when protracted. Step width was measured as the difference in *x*-position of successive heel strikes.

the maximum difference was set as the threshold for the lowest muscle activity. All values below this threshold (e.g. values lower than random muscle activity) were removed from the original signal.

Metabolic cost of locomotion

After the kinematic trials described above, a subset (N=6, four male, two female, 70.2±15.9 kg) of subjects performed a set of metabolic trials in order to determine the effect of arm restraint on locomotor cost. For these trials, oxygen consumption was measured using the 'open-flow' method described previously (Fedak et al., 1981; Pontzer, 2007). Subjects wore a light mask through which air was pulled at 250 l min^{-1}. This air was sub-sampled continuously, scrubbed of water vapor and carbon dioxide, and analyzed for oxygen concentration using a paramagnetic analyzer (Sable Systems®, Las Vegas, NV, USA). Oxygen concentration was monitored in near-real time and recorded at 30 Hz in Vicon Nexus software. Oxygen concentration was then used to calculate the rate of oxygen consumption (ml O_2 s^{-1}) following Fedak et al. (Fedak et al., 1981); the system was calibrated daily and checked for leaks using a known flow rate of pure nitrogen.

The resting rate of oxygen consumption was first measured with the subjects standing on the treadmill. Next, the subjects performed two 1.5 m s^{-1} walking trials, and two 3.0 m s^{-1} running trials. In one walking trial and one running trial the subjects walked or ran normally, as in the control condition; in the other walking and running trial, they walked or ran with arms folded tightly across their chest as in the no arms condition. The order of conditions was varied, so that half of the subjects performed the control trials first, and half performed the no arms condition first. Each metabolic trial lasted at least 6 min, and mean oxygen concentration from the final 2 min of each trial was used to calculate the rate of oxygen consumption. Only trials in which oxygen consumption visibly reached a plateau (less than 10% change over the final 2 min) were used for analysis. For each subject, the resting rate of oxygen consumption was subtracted from the rate of consumption while walking or running in order to calculate a net cost of locomotion. This net cost was then divided by body mass and then by speed to give the mass-specific cost of transport (ml O_2 kg^{-1} m^{-1}) for each speed in each condition.

Hypothesis testing

Filtered kinematic and thextonized EMG data were used to examine predicted relationships. Segment velocities and accelerations were calculated using the finite differences method described in Winter (Winter, 2005). Predictions were considered to be supported if the correlation between two kinematic variables (e.g. shoulder displacement and arm acceleration) had a Pearson's R greater than 0.5 or less than –0.5, and in the predicted direction, following Cohen's index for a 'large' effect size (Cohen, 1992). This effect size (R=±0.5) recognizes the complexity of the multi-segment, multi-muscle system being analyzed, and anticipates variability within the system and between subjects. It should be noted that the conventional criterion for statistical significance, a P-value of <0.05 or <0.01, is inappropriate in determining the biological or biomechanical significance of these segment correlations due to the large number of data points generated by high-speed kinematic data. With a capture rate of 200 frames s^{-1}, three strides generate approximately 600 data points, because each frame produces a position, velocity and acceleration estimate for a given segment. With a sample of 600 points, even small correlations of R=±0.1 become significant at P<0.01; however, such small correlations indicate that only 1% of the variance in the dependent variable is explained by the independent variable. Therefore the criterion for a 'large' effect size (R=±0.5) (Cohen, 1992), while admittedly arbitrary, is preferable to a calculated P-value for these correlations.

To determine the effect of the no arms condition on locomotor cost, we compared the net mass-specific cost of locomotion in the control and no arms condition during walking and running for each subject using Student's one-tailed, paired t-test. Similarly, we used Student's one-tailed, paired t-test to compare mean contact times, stride frequencies, head yaw amplitude and footfall variability for each subject walking at 1.5 m s^{-1} and running at 3.0 m s^{-1} in each condition. Note that using a one-tailed test was deemed appropriate here, since the direction of the predicted difference is known *a priori*. We discuss the effect of using a one-tailed test below.

Lag time between shoulder and pelvis rotation and footfall variability were also compared between conditions using Student's paired t-tests. As pelvis and shoulder rotation occur with similar frequency but with different times of peak amplitude, we calculated the phase difference between pelvis and shoulder movement in order

to determine the effect of increasing or decreasing the moment of inertia of the upper body. The phase difference between peak pelvis rotation (t_{pelvis}) and peak shoulder rotation ($t_{shoulder}$) was calculated as phase difference=360 deg.×(|t_{pelvis}–$t_{shoulder}$|/stride period). The closest shoulder and pelvis peaks were compared, so that the maximum phase difference was 180 deg. To test for differences in footfall variability, the medio-lateral position of the heel at heel strike was recorded for eight consecutive steps at each speed (Fig. 1). The medio-lateral distance between successive steps, hereafter termed step width, was measured, and the coefficient of variation (a size-corrected measure of variance) was determined for each subject at each speed. Coefficients of variation (c.v.) were then compared using Student's paired *t*-test.

RESULTS

Kinematics

Kinematic analyses revealed correlated movements of the pelvis, shoulder and arm which support the hypothesis that the arms act as mass dampers, decreasing the amplitude of upper body rotation. Changing the moment of inertia of the arms (and hence the upper body) generally resulted in the predicted effects on the amplitude of upper body rotation (measured as shoulder rotation; Fig. 3A) and of the head (measured as the amplitude of head yaw; Fig. 3B), although this effect was stronger during running. For walking trials at 1.5 m s^{-1}, shoulder rotation was generally low, and there were no significant differences between no arms (mean±s.d. 8.6±1.9 deg.) and control (8.1±2.5 deg.) conditions (P=0.20), or between control and arm weights (9.1±3.4 deg.) conditions (P=0.33; Fig. 3A). In contrast, during running at 3.0 m s^{-1}, the amplitude of shoulder rotation was significantly greater (P<0.001) and changes in arm inertia had expected effects on shoulder rotation. Decreasing the moment of inertia of the arms in the no arms condition resulted in greater shoulder rotation (35.68 deg.) than in control trials (23.75 deg., P<0.01; Fig. 3A), while increasing the moment of inertia of the arms in the arm weights condition resulted in decreased shoulder rotation (17.48 deg.) compared with control trials, although this relationship barely met the significance criterion (P=0.049).

The amplitude of head rotation was significantly lower than that of the shoulder in all conditions, both walking and running (P<0.01 all comparisons; Fig. 3B). Still, changes in head yaw between conditions generally followed the pattern of shoulder rotation. During walking, head yaw was lowest in arm weights trials (5.1±1.7 deg.), slightly greater in control trials (5.2±1.1 deg.) and greatest in no arms trials (6.0±1.6 deg.). Significant differences were found between no arms and control trials (P=0.046), and between no arms and arm weights trials (P=0.01), but differences between control trials and arm weights (P=0.32) and no arms trials were not significant. During running, head yaw was lowest in arm weights trials (5.3±1.5 deg.), slightly greater in control trials (6.1±1.6 deg.) and greatest in no arms trials (11.3±3.4 deg.). The difference between no arms and both arm weights (P<0.01) and control trials (P<0.01) was significant, but the difference between control and arm weights conditions (P=0.26) was not. For all conditions, head yaw tended to be greater during running than during walking, but this difference was only significant for the no arms condition (P<0.01).

Lag time between pelvis and shoulder rotation increased with greater moment of inertia of the upper body as predicted (Fig. 3C), with the greatest phase differences between pelvis and shoulder rotation during arm weights trials for both walking (157.6±22.1 deg.) and running (74.1±35.8 deg.). Phase differences in control trials were slightly lower (walking 149.2±41.5 deg., running 93.9±60.9 deg.) but these differences were not significant (walking P=0.10, running P=0.46). Phase differences were smallest in the no arms trials (walking 93.9±60.9 deg., running 33.0±23.3 deg.), significantly smaller than control trials for both gaits (walking P=0.01, running P<0.01), and significantly smaller than arm weights trials during running (running P<0.01, walking comparison approach significance at P=0.055). For each condition, phase differences were significantly greater during walking (P<0.01, walking *versus* running trials, all comparisons).

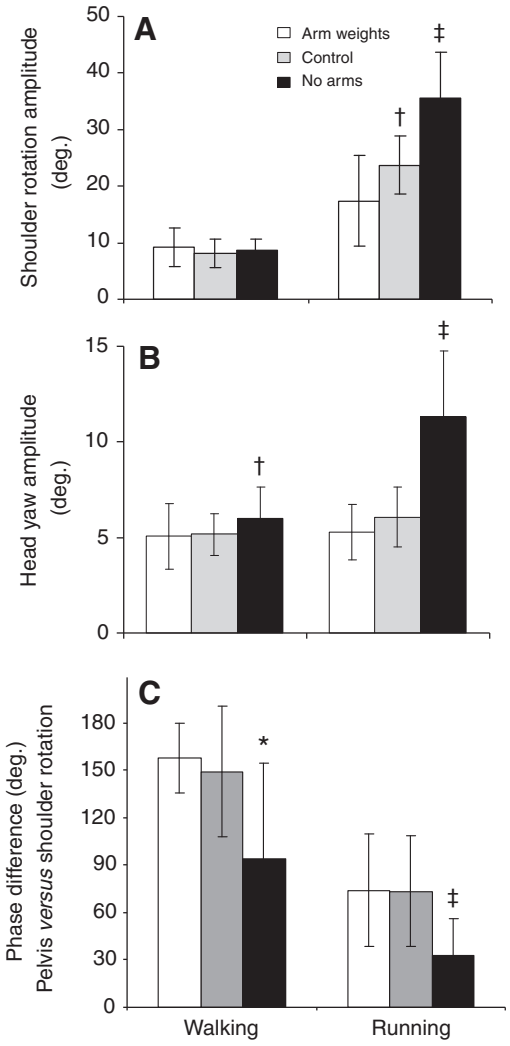

Fig. 3. Mean ± s.d. (A) shoulder rotation, (B) head yaw and (C) phase differences between peak shoulder rotation and peak pelvis rotation. *Significant difference compared with control trials (P<0.05). †Significant difference compared with arm weights trials (P<0.05). ‡Significant difference compared with both arm weights and control trials (P<0.05).

Passive arm swing predictions were strongly supported by kinematic results. During walking and running, angular acceleration of the shoulders in the transverse plane was consistently, positively correlated with torsion of the spinal column, measured as the difference in angle between the shoulder and pelvis in the transverse plane (mean Pearson's R=0.59; Table 1; Fig. 4). Similarly, for both walking and running trials, angular acceleration of the arm in the sagittal plane was strongly correlated with angular displacement of the shoulder, with greater retraction associated with greater anterior

Table 1. Correlations (Pearson's *R*) between body segments during normal walking and running

	Passive arm swing						Active arm swing					
	Pelvis–shoulder angle *versus* shoulder acceleration			Arm angle *versus* arm acceleration			Pelvis acceleration *versus* shoulder acceleration			Arm acceleration *versus* shoulder acceleration		
Speed/gait	Mean	s.d.	(Min., max.)	Mean	s.d.	(Min., max.)	Mean	s.d.	(Min., max.)	Mean	s.d.	(Min., max.)
1.0 m s^{-1} walk	0.51	0.113	(0.387, 0.736)	–0.76	0.135	(–0.889, –0.574)	0.01	0.119	(–0.118, 0.266)	0.04	0.217	(–0.229, 0.365)
1.5 m s^{-1} walk	0.41	0.151	(0.241, 0.667)	–0.85	0.087	(–0.955, –0.672)	0.05	0.092	(–0.100, 0.184)	0.13	0.269	(–0.286, 0.472)
2.0 m s^{-1} walk	0.41	0.197	(0.170, 0.701)	–0.89	0.065	(–0.952, –0.758)	–0.07	0.228	(–0.417, 0.249)	0.20	0.311	(–0.155, 0.667)
2.0 m s^{-1} run	0.69	0.185	(0.266, 0.824)	–0.84	0.048	(–0.889, –0.773)	–0.09	0.233	(–0.385, 0.224)	0.39	0.164	(0.148, 0.584)
2.5 m s^{-1} run	0.75	0.114	(0.562, 0.887)	–0.84	0.045	(–0.895, 0.776)	0.01	0.259	(–0.365, 0.303)	0.38	0.143	(0.190, 0.598)
3.0 m s^{-1} run	0.75	0.080	(0.589, 0.835)	–0.85	0.048	(–0.913, –0.778)	0.02	0.263	(–0.431, 0.313)	0.40	0.141	(0.202, 0.645)
Walking	0.45	0.160	(0.170, 0.736)	–0.83	0.112	(–0.955, –0.574)	0.00	0.154	(–0.417, 0.266)	0.14	0.274	(–0.286, 0.667)
Running	0.74	0.133	(0.266, 0.918)	–0.84	0.044	(–0.913, –0.773)	–0.01	0.238	(–0.431, 0.313)	0.42	0.159	(0.148, 0.711)
All	0.59	0.206	(0.170, 0.918)	–0.84	0.085	(–0.955, –0.574)	–0.00	0.197	(–0.431, 0.313)	0.27	0.264	(–0.286, 0.711)

acceleration (mean Pearson's R=0.59; Table 1; Fig. 4). These results are consistent with the passive arm swing prediction that the spinal column and shoulder effectively act as springs, with greater displacement leading to greater acceleration.

Active arm swing predictions were generally not supported by kinematic analyses. Angular accelerations of the pelvis and shoulder were not correlated (mean Pearson's R=0.00; Table 1; Fig. 4). Further, while arm acceleration was weakly correlated with the angular acceleration of the shoulder (mean Pearson's R=0.27; Table 1; Fig. 4), the positive direction of correlation was opposite to that of the active arm swing hypothesis, which predicts that anterior acceleration of the arm will lead to posterior acceleration of the ipsilateral shoulder.

Comparing walking and running (Table 1), it is evident that Pearson's correlations between shoulder acceleration and both arm acceleration and spinal torsion are greater during running. The significance of this change and the underlying mechanism are unclear. In both cases, the greater ground forces encountered during running may lead to greater stabilizing muscle activity, and therefore a stronger linkage (i.e. a stiffer 'spring') between the pelvis, shoulder and arm. Stiffer 'springs' may also be necessitated by the greater stride frequencies used in running, since stiffer springs would increase the natural frequencies for the body segments involved. For example, a stiffer 'spring' in the shoulder will increase the natural frequency of the swinging arm. Finally, the greater angular excursions seen in running (Fig. 3A,B) may lead to a stronger correlation of movement between segments.

Muscle activity

Patterns of muscle firing were generally consistent with predictions of the passive arm swing hypothesis, although some alternating activity in the anterior and posterior deltoid was observed. When compared with the clear alternating pattern of anterior and posterior deltoid activity seen in the arm pump trials (Fig. 5A), firing of these muscles during both walking and running was largely simultaneous. This suggests that the deltoid is acting to stabilize the shoulder as predicted by the passive arm swing hypothesis, rather than to drive it anteriorly or posteriorly as predicted by the active arm swing hypothesis. However, some alternating activity was observed, particularly in walking trials (Fig. 5B), indicating that the deltoid does drive arm swing at least occasionally for some individuals. During running, firing of the anterior and posterior portions of the deltoid was almost exclusively co-contraction (Fig. 5C).

Overlaying the angular velocity and acceleration of the shoulder in the sagittal plane on EMG activity (Fig. 6), it appears that many, perhaps most, of the deltoid contractions are eccentric, with the anterior deltoid firing while the arm moves posteriorly, and the posterior deltoid firing while the arm moves anteriorly. These eccentric contractions are consistent with the view of the shoulders as spring-like linkages. Further, while contraction of the anterior or posterior deltoid is typically associated with predictable accelerations at the shoulder, there are also periods in which arm acceleration and deltoid activity are in opposition, with anterior acceleration of the arm associated with posterior deltoid activity (Fig. 6A), even when the lag time between activation and force production are considered. Similarly, periods of arm acceleration are also seen when the deltoid muscles are quiet (Fig. 6B). These patterns suggest that forces, in addition to those from the deltoid, are acting on the arm. These results are consistent with the mass damping hypothesis, in which forces acting on the arms are primarily derived from the legs *via* the trunk.

Gait characteristics

Stride period during no arms trials (walking 1.05±0.06 s, running 0.74±0.04 s) was similar to that in control (walking 1.05±0.07 s, running 0.75±0.04 s) and arm weights trials (walking 1.04±0.05 s, running 0.76±0.08 s). These differences were not significant for walking or running (P>0.05) with the exception of the control–no arms comparison for running (P<0.01). However, this difference was small (0.01 s, or 1.3%) and probably biomechanically unimportant. Contact times during no arms trials (walking 0.68±0.05 s, running 0.30±0.03 s), control trials (walking 0.68±0.05 s, running 0.30±0.03 s) and arm weights trials (walking 0.68±0.05 s, running 0.32±0.03 s) were similar (P>0.05 all comparisons).

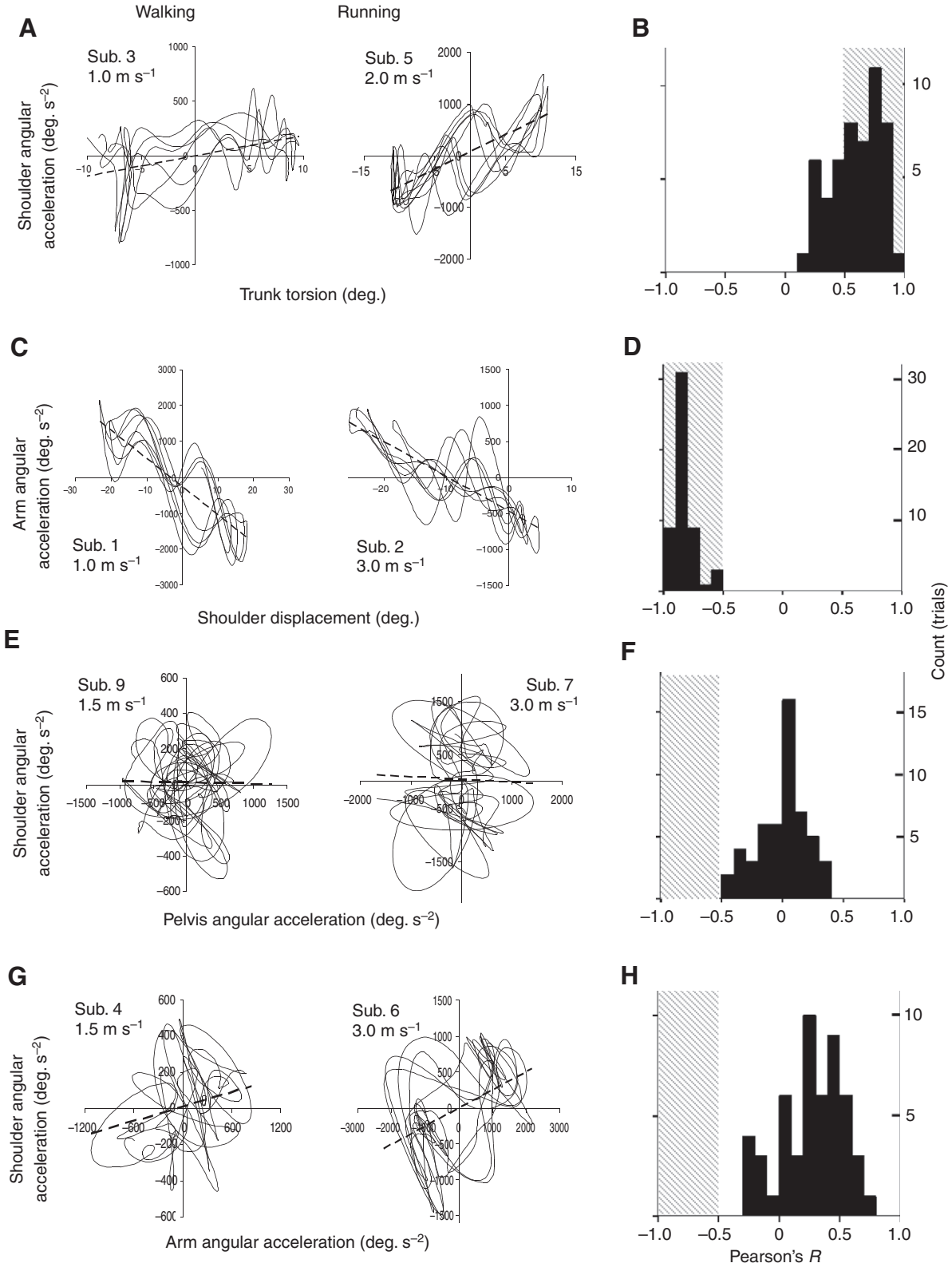

Fig. 4. Kinematic results. (A–D) Predictions of the passive arm swing hypothesis (see Fig. 2B); (E–H) active arm swing predictions (see Fig. 2C). Plots are representative results for walking and running and list the subject (Sub.) and speed shown. Histograms are Pearson's *R*-values for all speeds and subjects, walking and running combined. Hatched areas in histograms indicate predicted values for passive (B,D) or active (F,H) hypotheses.

Footfall variation and metabolic cost

During walking at 1.5 m s^{-1}, variation in step width during no arms trials (mean c.v. 0.053±0.026) was greater than for control trials (0.044±0.021) although this difference was only marginally significant (P=0.039). There was no difference between control and arm weights (0.056±0.013) conditions, or between no arms and arm

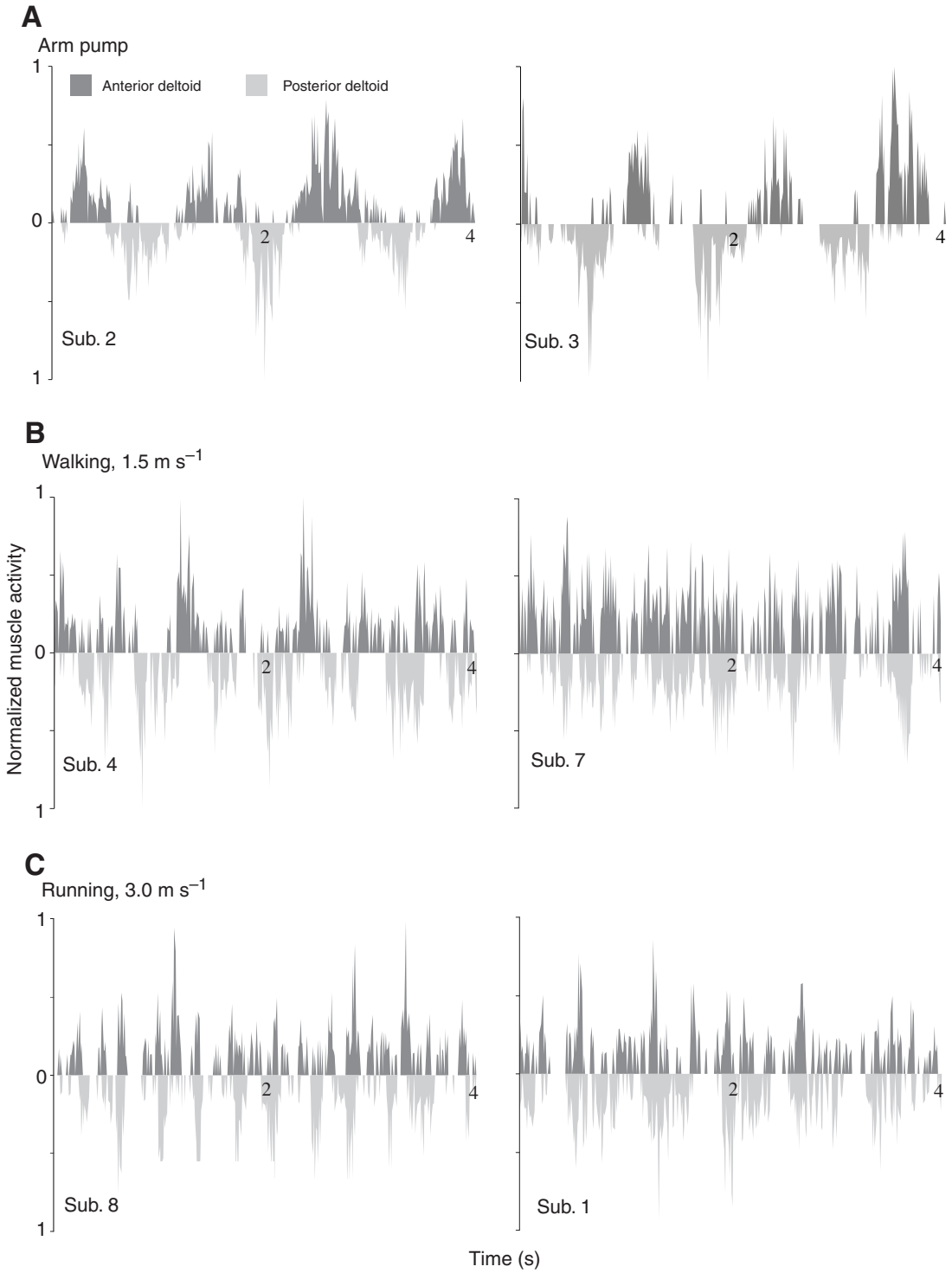

Fig. 5. Representative anterior and posterior deltoid activity for (A) arm pump, (B) 1.5 m s^{-1} walking, and (C) 3.0 m s^{-1} running trials. EMG data have been processed as described in the text and normalized to the maximum activation within a trial. The subject from whom data were obtained is listed.

weights conditions (P>0.10 both comparisons; Fig. 7A). During running at 3.0 m s^{-1}, there were no differences between no arms (0.059±0.020), control (0.053±0.018) and arm weights trials (0.048±0.017; Fig. 7A).

Restricting arm swing in the no arms condition had no effect on the mass-specific energetic cost of transport (ml O_2 kg^{-1} m^{-1}). Locomotor costs during no arms trials (walking 0.13±0.03 ml O_2 kg^{-1} m^{-1}, running 0.21±0.04 ml O_2 kg^{-1} m^{-1}) and control trials (walking 0.12±0.02 ml O_2 kg^{-1} m^{-1}, running 0.21±0.04 ml O_2 kg^{-1} m^{-1}) were similar (walking P=0.10, running P=0.14; Fig. 7B).

DISCUSSION

Arms as mass dampers

Our results support the hypothesis that the arms act as mass dampers during human walking and running, although the evidence is clearest for running. In running trials, the amplitude of shoulder

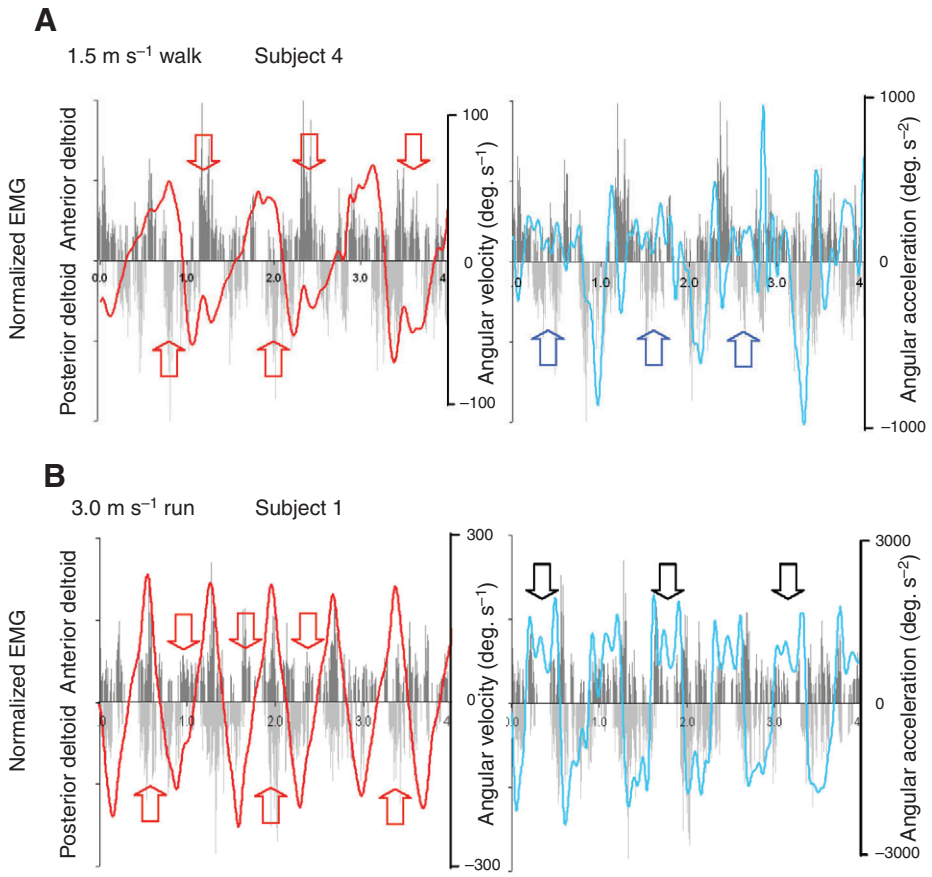

Fig. 6. Representative angular velocity (red line) and angular acceleration (blue line) for the arm at the shoulder, overlaid on normalized anterior and posterior deltoid activity, during (A) walking at 1.5 m s^{-1} and (B) running at 3.0 m s^{-1}. Deltoid activity is processed and shown as in Fig. 5. Periods of apparent eccentric contraction are indicated (red arrows), as are periods in which shoulder acceleration is in opposition to prevailing muscle activity (blue arrows) or occurs without substantial deltoid activity (black arrows). Not all such periods are indicated.

rotation clearly increased when the moment of inertia of arms decreased (Fig. 3A), just as movement the principle mass of a mass-damped system should increase with a decrease in the auxiliary mass (Fig. 1A) (Soong and Dargush, 1997). While this relationship was not observed for walking (Fig. 1A), this does not mean that a mass-damper view of the arms should be rejected; the effectiveness of a mass damper and the effect of changing its inertial properties depend upon the magnitude and frequency of the external forces acting on the system (Soong and Dargush, 1997). The magnitude and frequency of forces from the lower body may simply be too low during walking to elicit a significant change in torso movement with the manipulations of arm inertial properties used here. The magnitude of head yaw was less than that of the shoulders, but changes in head yaw across experimental conditions generally followed the pattern of shoulder movement, supporting the view of the head as a mass attached *via* a damped spring. Finally, phase lag between the lower body and upper body decreased when the moment of inertia of the arms was decreased during both walking and running (Fig. 1C), as predicted for a mass-damped system. The view of the arms as mass dampers is consistent with previous work (e.g. Hinrichs, 1987; Hinrichs, 1990; Li et al., 2001; Herr and Popovic, 2008) indicating that angular acceleration in the upper and lower body tend to cancel, resulting in near-zero net moments about the vertical axis. However, while the results of this study fit predictions of a mass-damper model, the tests here are certainly not exhaustive, and future work might test other predictions of a mass-damper hypothesis in order to determine whether this model alone is sufficient for explaining upper body movement, particularly during walking.

Passive *versus* active arm swing

The passive arm swing hypothesis proposes that upper body movement is driven by movement in the legs and pelvis, with force transferred to the shoulders and arms *via* spring-like elements (ligaments and muscles) in the spine and shoulder. This differs from an active arm swing hypothesis, which proposes that upper body movement is driven primarily by swinging the arms using the shoulder muscles. As predicted by the passive arm swing hypothesis, angular acceleration of the shoulders was correlated with increased trunk torsion, and arm acceleration was strongly correlated with angular displacement of the shoulder (Fig. 4). In contrast, angular acceleration of the shoulders and pelvis were not inversely correlated, nor was shoulder acceleration inversely correlated with arm acceleration, as predicted by the active arm swing hypothesis (Fig. 4). EMG recordings of the anterior and posterior deltoid suggest that, while these muscles may play a limited role in driving arm swing, they act primarily to stabilize the shoulder through co-contraction or eccentric contractions (Figs 5 and 6). Taken together, the kinematic and EMG results support the passive arm swing hypothesis.

Additional support for the passive arm swing model comes from the metabolic comparisons of control and no arms conditions. As noted above, upper body movement during running increases in the no arms condition by approximately 50% compared with control trials (Fig. 1A). If upper body movement is actively driven by trunk and arm musculature as in the active arm swing model, the larger displacements of the torso should require a corresponding increase in oxygen consumption. Instead, energy use is similar to that in the control condition, indicating that greater movement of the torso in

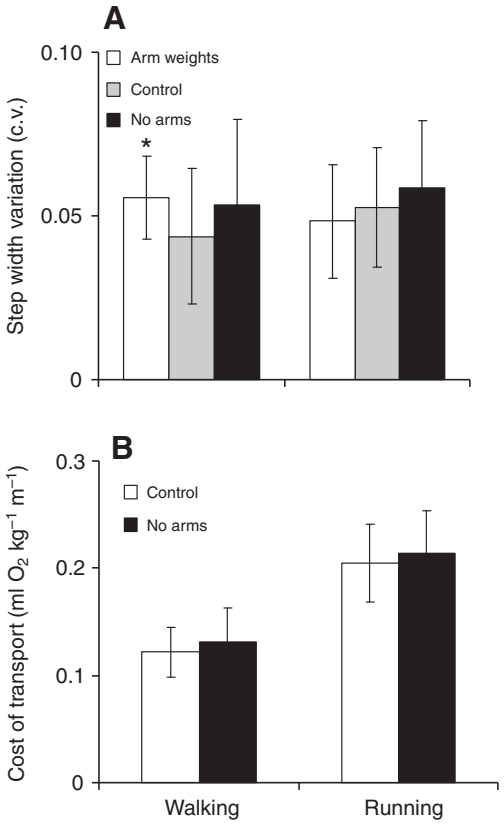

Fig. 7. Mean ± s.d. values for walking (1.5 m s^{-1}) and running (3.0 m s^{-1}) for (A) step width variation and (B) locomotor cost. *Significant difference compared with control trials (P<0.05).

the no arms trials results from the decreased inertia of the upper body, not an increase in muscle activity.

Further tests of the passive mass damping model

While our results support the hypothesis that the upper body behaves as a passive system, limitations in our methods must be considered. Perhaps most critically, our analysis of muscle activity is limited to surface EMG of the deltoids, and further data are needed to determine whether muscles and other connective tissues in the back and shoulder performed mechanical work or acted as springs. Our analyses suggest these linkages behave like springs, but the possibility that muscles are performing work while mimicking purely elastic behavior cannot be ruled out using our methods; such 'pseudo-elastic' muscle activity has been suggested before for the leg muscles during terrestrial locomotion (Ruina et al., 2005). Even if the muscular linkages involved do act as springs, without performing positive mechanical work, it is important to note that such isometric or eccentric muscle contraction incurs a metabolic cost. Thus, arm swing may be 'passive' in the mechanical sense, with energy for movement being derived ultimately from leg swing, and yet be 'active' in the metabolic sense, requiring metabolic energy for muscle activation.

It is also important to note that mass-damped systems can respond in complex ways to changes in the oscillation frequency, spring and damping constants, and relative masses of the segments (Soong and Dargush, 1997). Our simple five-segment model essentially treats these variables as constant across conditions, but this assumption is difficult to test and not addressed here. More sophisticated models, in concert with more in-depth measurements of muscle activity, may provide a more comprehensive test of the mass damping model for upper body mechanics. Specifically, expanding current forward dynamics models of human walking (e.g. Anderson and Pandy, 2001) to include full musculoskeletal treatment of the trunk and arms will provide a means of examining the interaction between upper and lower body movement.

Both the active arm swing and passive arm swing hypotheses predict that net moments about the body's vertical axis will be kept near zero for steady-state walking and running, and thus net-moment analyses are not able to distinguish between these two mechanisms. Our passive arm swing hypothesis differs primarily in that the power for arm swing is ultimately derived from the swing legs. As such, future work might examine non-steady-state locomotion in which lower limb energy changes, such as with the increase in stride frequency associated with increased walking speed. Active arm swing models would predict these changes to be immediately matched by corresponding changes in upper body movement, whereas a passive model would predict a measurable lag time of at least one step (i.e. one oscillation of the pelvis in the transverse plane) for the increased energy in the legs to be transferred to the upper body.

By highlighting the importance of spring-like mechanisms in the trunk and shoulder, our work builds upon that of Fernandez Ballesteros and colleagues (Fernandez Ballesteros et al., 1965), which suggested that elastic mechanisms in the shoulder are critical to normal arm swing. This view of arm swing as an emergent property of human walking also fits well with recent passive-dynamic models of lower limb mechanics for human walking (Collins et al., 2005). As with passive-dynamic lower limb movement, passive spring-driven arm swing mechanics proposed here are inherently self-tuning without requiring extensive feed-forward neurological control. Passive-dynamic walkers which include upper body segments connected to the lower body through elastic elements would provide a further test of the passive arm swing hypothesis, and perhaps refine current models for upper body movement in humans.

The role of arm swing in walking and running

With the exception of a small, mechanically negligible decrease in stride frequency during no arms running and a small but statistically significant increase in footfall variability during no arms walking, restricting arm swing or adding weights to the arms had no effect on the lower limb kinematics or footfall variability measured here, nor did restricting arm swing affect walking or running cost (Fig. 7B). These results provide further support for the idea that upper body movement is inherently self-tuned, producing stable walking and running even when upper body inertial properties are modified. However, as a consequence of this self-tuning, upper body kinematics were significantly affected by restricting arm swing, with shoulder rotation and head yaw increasing substantially in no arms running trials (Fig. 3A,B). These results, as well as the relative isolation of the head from the larger rotations experienced by the shoulders, support Bramble and Lieberman's (Bramble and Lieberman, 2004) hypothesis that the derived configuration of the human upper body in which humans have low, wide shoulders that are mostly decoupled from the head are exaptive for walking, and are especially important for limiting head yaw and improving visual stability during running.

The importance of normal arm swing in reducing head yaw in humans raises the question of how cursorially adapted birds

dampen upper body oscillations, and how bipedal dinosaurs met this mechanical challenge. While researchers have examined head stabilization in the sagittal plane in birds (e.g. Katzir et al., 2001; Troje and Frost, 2000; Necker, 2007), stability in the transverse plane warrants investigation. Three potential mechanisms are immediately apparent. First, the horizontally oriented trunks of these bipeds will serve to increase the moment of inertia about the vertical axis and decrease angular excursions. Second, the long, relatively thin neck of some avian cursors (e.g. ostriches) might act as a filter for oscillations of the torso, limiting transverse head movements. Third, the long, relatively massive tails of dinosaurs might provide adequate mass damping of the torso. Indeed, passive mass damping might be a widespread phenomenon in terrestrial animals. For example, in kangaroos, movement of the tail in the sagittal plane acts to dampen pitching of the trunk during hopping (Alexander and Vernon, 1975); the long tendons in the kangaroo tail suggest an elastic linkage between the trunk and tail, as would be expected for a passively damped system.

The anatomical model used here greatly simplifies upper body anatomy, reducing the multi-segment, multi-muscle, upper body to a five-segment system with simple damped spring linkages. Still, the evidence for a passive mass damping model as a predictor of the relative movements of the pelvis, shoulders and arms suggests that the passive arm swing hypothesis tested here may provide valuable insight into the mechanics and control of upper body movement during human walking and running. Future work might integrate a more sophisticated, multi-segment anatomical model (e.g. Herr and Popovic, 2008) with a focus on the mechanisms driving upper body movement. The implication that upper body movement is a self-tuned, self-stabilizing phenomenon may inform future analyses of human gait, and may be useful in biomimetic and prosthetic engineering.

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