Robots Should Have Brakes, That Work Like Muscle
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In our quest to engineer human levels of performance in robots, I and others in robotics have been trying to imitate the macroscopic behavior of muscle. We have superficially simulated the springlike properties of muscles and their role in control [2] using force controlled electric motors and hydraulics. We have followed a design path of ever higher performance force and torque sensing and lower latency velocity and torque feedback control, resulting in ever more expensive robots. Perhaps we have focused on the wrong aspect of muscle. This talk focuses on muscle damping, and how imitating it may help us build less expensive robots with a more human-like touch.

Current torque-controlled humanoid robots are now successfully walking largely because torque control is finally good enough: achieved with high quality expensive and delicate sensors, and an order of magnitude less feedback latency than biological systems. However, it is notoriously difficult to damp out oscillations in torque controlled robots, and especially robots with series elastic actuators (for example, M2 [10]). Motors with low gear ratios use a lot of energy to hold a position or resist a push. Electric motors produce torque that is proportional to current, and the power used is the current multiplied by the power supply voltage. Hydraulic motors can hold a position with no energy cost by closing valves. However, resisting a pull while moving has an energy cost of the fluid flow multiplied by the supply pressure. In addition, force-controlled hydraulic systems used on humanoid robots typically have significant continual energy loss through internal leakage, which is necessary to keep friction in pistons low. It is clear that robotics has been pursuing a mistaken design paradigm. Because our abstractions about muscle were wrong, we missed a better design approach: a set of actuators at each joint: inaccurate but repeatable unidirectional motors and physical brakes, similar to the actuation of automobiles.

Muscle is a modal actuator that behaves quite differently in its shortening, isometric, and lengthening modes. How muscle behaves mechanically and chemically is determined by the direction and amount of relative movement of the thin (actin) and thick (myosin) filaments, in addition to activation. Shortening muscle can be viewed primarily as a force source that uses metabolic energy and fatigues. Lengthening muscle largely acts as a brake or energy sink that does not use much metabolic energy and does not fatigue as rapidly. Muscle under all conditions including isometric has short range stiffness for stretches (but not releases) that does not use metabolic energy.

There is ample psychophysical and physiological evidence that muscles are at least twice as strong, use much less energy, are stiffer, and fatigue much less when producing a force during lengthening as compared to shortening [6]. In humans, the energy demand of eccentric and isometric work was negligible compared to concentric work, and only the concentric work significantly increased with force level, in a study using ^{31}P magnetic resonance spectroscopy (MRS) to measure energy flow (the ratio of ATP-related phosphates Pi to PCr) [9],

There have been phenomena such as residual force enhancement in fiber-level studies of muscle stretch that are not explained by the crossbridge mechanochemical cycle involving the use of ATP to detach crossbridges. It is becoming increasingly clear that there are additional molecular mechanisms that make muscle lengthening quite different from shortening. It has been hypothesized that 1) crossbridges do not need ATP to detach when the muscle is lengthening, but instead detach when stretched beyond a mechanical limit [4]. 2) Once detached, these crossbridges rapidly reattach [8, 4]. 3) In this situation attachment of the 2nd myosin head is facilitated [3, 7].

Additional complementary mechanisms that may also enhance eccentric work have been proposed involving titin, a molecule that links the thick fibers to the Z line of the sarcomere. Titin may wind or

cross link with the actin and/or myosin filaments.

Although we have not quantitatively simulated a model of these effects, we can make some qualitative predictions of macroscopic effects of these molecular mechanisms: 1) Isometric stiffness and the force necessary to initiate movement is higher after lengthening vs. shortening for the same activation level. A way to increase isometric strength is to allow a small slip beforehand. 2) There is hysteresis in a single muscle between lengthening and shortening, as the forces and stiffnesses generated by the same activation are higher for lengthening. 3) Because a muscle has unidirectional damping, and the antagonist muscle of an agonist/antagonist pair also contributes damping, oscillations and any physical shock waves due to impact are quickly damped out. Muscle is an efficient shock absorber. 4) Because the damping is a direct effect of velocity, neural delays do not contribute to oscillations or instabilities. Neural damping feedback gains can be much larger than reflex latencies and muscle activation time constants would predict. 5) In trying to reduce metabolic cost, concentric work should be minimized in favor of isometric work and eccentric work. 6) Muscles acting isometrically or as brakes is an important part of behavior [1, 5] A better understanding of how muscles work will lead to higher performance robots. One day we may be able to engineer molecular motors. In the meantime, we can focus on simulating the muscle properties and behavior described above with a combination of simple inexpensive actuators: unidirectional motors (no backlash issues) and variable brakes (greatly reducing stability issues and the need for high quality torque and velocity feedback).

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