GEOMETRICAL FACTORS INFLUENCING MUSCLE FORCE DEVELOPMENT

II. RADIAL FORCES

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ABSTRACT If the subfragment-2 (S2) portion of the myosin cross-bridge to actin does not lie parallel to the myofilament axes, then when a muscle fiber contracts, there will be a radial component to the cross-bridge force. When the subfragment-1 (S1) portion of the cross-bridge attaches to actin with its long axis projecting through the filament axis, the magnitute of the radial force depends upon the azimuthal location of the actin site, but when the attachment of the S1 to actin is slewed, as in the reconstruction of Moore et al. (*J. Mol. Biol.*, 1970, **50**:279–294), then for a single cross-bridge the radial component of the cross-bridge force is not quite so sensitive to actin site location and is ~ 0.1 the axial component. In both cases, the ratio of the radial to axial force decreases with decreasing filament separation. If the radial-axial force ratio for each cross-bridge is ~ 0.1, then at full overlap in a frog skeletal muscle fiber the radial component of the cross-bridge force accompanying full activation will exert a compressive pressure of $\sim 5 \times 10^{-3}$ atm. This would have little effect upon an intact muscle fiber where the volume constraints are likely osmotic, but it might produce a 1–2% change in filament spacing in a "skinned" muscle fiber from which the sarcolemma had been removed. These computations assume that the S2 link between the S1 head and the myosin filament does not support a bending moment or shear. If it does, then the radial component of the cross-bridge will be either greater or less, depending on the specific cross-bridge geometry.

INTRODUCTION

The previous paper (Schoenberg, 1980) dealt with the influence of geometrical factors upon axial muscle force, the force that would be recorded by a transducer attached to the end of the muscle. The cross-bridge model for contraction suggests that the force generated by the cross-bridge interacting with actin should have a radial, as well as an axial, component. (The only simple situation in which this is not so is the special case where the subfragment-2 [S2] portion of the cross-bridge lies parallel to the filament axes while the subfragment-1 [S1] lies perpendicularly.)

Radial forces act to compress or swell the myofilament lattice; the lattice spacing is determined by a balance of all the radial forces. In addition to any radial component of the cross-bridge force, radial forces may be exerted by van der Waals attractive forces, electrostatic repulsive forces (due to excess negative charge on both the actin and myosin filaments), forces, probably repulsive, related to hydration of the filaments, and possibly elastic forces arising from structures at the *M* and *Z* lines (Elliott, 1968; Elliott and Rome, 1969; Miller and Woodhead-Galloway, 1971; April et al., 1972). Also, the filament lattice is

known to respond to osmotic pressures (Matsubara and Elliott, 1972; April, 1975; April and Schreder, 1979; footnote 1).

In this paper we discuss those properties of the radial component of the cross-bridge force that may be derived from geometrical considerations. Assuming that the S2 portion of the cross-bridge does not support a bending moment or shear, we can estimate that, for a single cross-bridge, the radial component of the cross-bridge force might be on the order of one to two-tenths of the axial component. In this case, radial cross-bridge forces associated with the axial force might cause a filament spacing change as large as 1–2% upon complete activation of a skinned muscle fiber at rest length, but very little change in an intact fiber. Other possible assumptions are also discussed.

METHODS

The notation of this paper is like that of the previous paper. If we restrict our discussion to the ratio of the radial to axial force, f_r/f_x, and, as in Schoenberg (1980), assume that the S2 portion of the

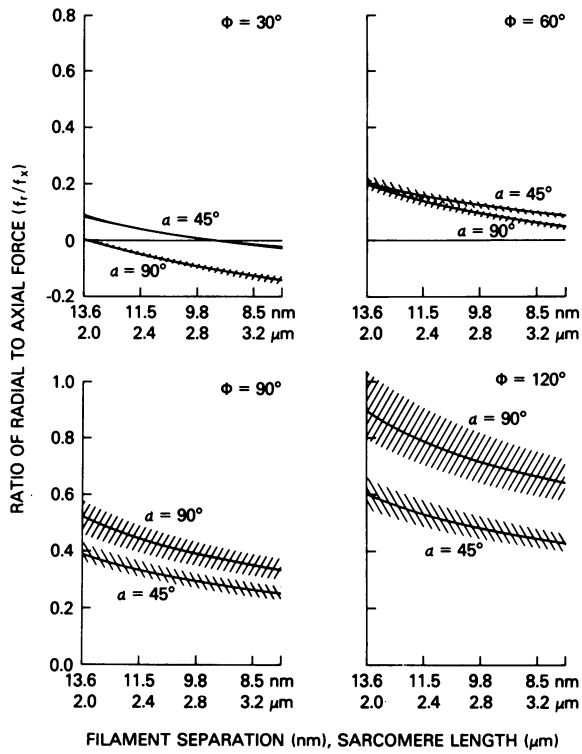

FIGURE 1 Ratio of the radial to axial components of the cross-bridge force (f_r/f_x) as a function of cross-bridge orientation, for attachment with $\gamma = 0°$ (S1 long axis projecting through actin filament axis). Values of azimuthal location of the actin site, ϕ, and axial tilt angle of the cross-bridge, α, are shown near each curve. Solid curves are for a cross-bridge attached at the axial location, $x' = 0$. (See Schoenberg [1980] for definition of x'). Hatched area shows range of variation of f_r/f_x for cross-bridges attached between $x' = \pm 4$ nm.

[1]Magid, A. Unpublished results.

cross-bridge does not transmit a bending moment or shear to the myosin filament (see Discussion), then the problem becomes purely geometric, independent of the site of cross-bridge elasticity or the mechanism of force generation. In this case the force acting upon the myosin filament is along the direction of the S2, and the ratio of radial to axial force, f_r/f_x, depends explicitly only upon the orientation of the S2 portion of the cross-bridge or implicitly upon the axial tilt of the S1 head, α, the azimuthal angle of the actin site, ϕ, the slew of the cross-bridge attachment, γ, the axial position of the cross-bridge attachment site, x, and the dimensions of the cross-bridge, filaments, etc. (These terms are more fully described in Schoenberg [1980].)

Referring to Fig. 1 *A* of Schoenberg (1980), it is seen that the force generated by the cross-bridge-actin interaction gets transmitted to the myosin filament through the S2 link, *AB*. This force in *AB*, F_{AB}, may be broken down into orthogonal components along *FB* and *AF*. Only the force along *AF*, F_{AF}, has a radial component. Now, $F_{AF} = F_{AB} \cdot \sin \angle ABF$, and it may be seen from Fig. 1 *B* of Schoenberg (1980) that the radial component of F_{AF}, i.e., the force along *OO'*, is $F_{AF} \cos (\angle AFD - \epsilon)$, where ϵ is the angle between *DF* and *OO'*. Therefore,

$$\begin{aligned} f_r &= F_{AF} \cos (\angle AFD - \epsilon) \\ &= F_{AB} \sin \angle ABF \cos (\angle AFD - \epsilon) \\ &= F_{AB} \sin \angle ABF (\cos \angle AFD \cos \epsilon + \sin \angle AFD \sin \epsilon). \end{aligned}$$

If we denote Q as the point where a perpendicular from A to *DF* intersects *DF*, then we have $\cos \angle AFD = \overline{FQ}/\overline{AF}$ and $\sin \angle AFD = \overline{AQ}/\overline{AF}$. Since $\sin \angle ABF = \overline{AF}/\overline{AB} = \overline{AF}/\ell_2$, we have

$$\begin{aligned} f_r &= F_{AB} \cdot (\overline{AF}/\ell_2) \cdot [(\overline{FQ}/\overline{AF}) \cos \epsilon + (\overline{AQ}/\overline{AF}) \sin \epsilon] \\ &= F_{AB} \cdot (\overline{FQ} \cos \epsilon + \overline{AQ} \sin \epsilon)/\ell_2. \end{aligned}$$

Now, $\overline{FQ} = Z' - \overline{AD} \cos \delta = Z' - \ell_1 \sin \alpha \cos \delta$ and $\overline{AQ} = \ell_1 \sin \alpha \sin \delta$, so that

$$\begin{aligned} f_r &= F_{AB} \cdot [(Z' - \ell_1 \sin \alpha \cos \delta) \cdot \cos \epsilon + \ell_1 \sin \alpha \sin \delta \sin \epsilon]/\ell_2 \\ &= F_{AB} \cdot [(Z' \cos \epsilon - \ell_1 \sin \alpha \cos (\delta + \epsilon)]/\ell_2. \end{aligned}$$

From the previous paper $f_x = F_{AB} \cdot (x + \ell_1 \cos \alpha)/\ell_2$, so that

$$f_r/f_x = [Z' \cos \epsilon - \ell_1 \sin \alpha \cos(\delta + \epsilon)]/(x + \ell_1 \cos \alpha) \qquad (1)$$

RESULTS

The Dependence of the Radial-Axial Force Ratio of a Single Cross-Bridge upon Geometry

Eq. 1 relates the ratio of the radial to axial components of the force of a single cross-bridge to the parameters of the cross-bridge geometry. Fig. 1 shows this relationship for the case where the S1 attaches to actin with its long axis projecting through the filament axis ($\gamma = 0°$) and Fig. 2 shows the case where the long axis of the S1 is parallel to the tangent at the actin site ($\gamma = 90°$). (See Fig. 2 of Schoenberg [1980] for an illustration of these two cases.) In each figure the upper abscissa is filament separation and the lower abscissa is the sarcomere length at which the separation exists in an intact frog skeletal muscle fiber. The radial-axial force ratio depends somewhat upon the longitudinal separation of the cross-bridge origin and the actin site (x), so that the solid lines in Figs. 1 and 2 have been drawn for a cross-bridge attached at $x' = 0$, while the hatched regions indicate the range of variation of the force ratio

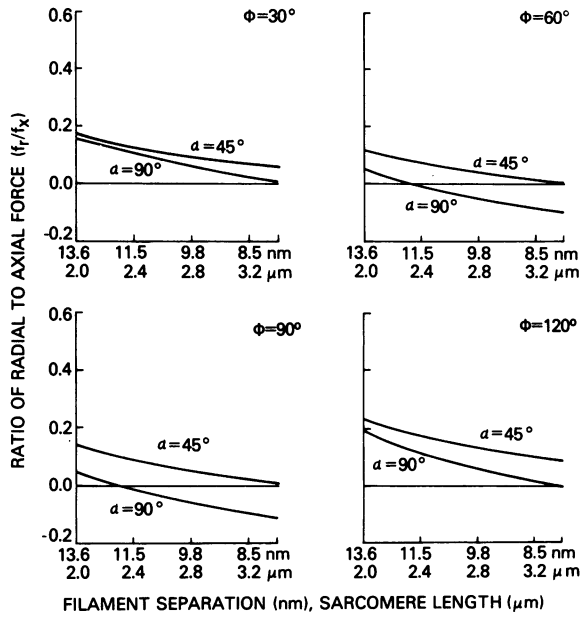

FIGURE 2 Same as Fig. 1, except attachment with $\gamma = 90°$ (long axis of S1 parallel to the tangent at the actin site). The variation in the force ratio for x' between ± 4 nm (not shown) is the same order of magnitude as for $\phi = 30°$ in Fig. 1.

for cross-bridges attached between $x' = \pm$ 4nm. The appropriate values for axial tilt, α, and azimuthal position of the actin site, ϕ, are shown next to each curve.

Two general conclusions may be drawn. First, there is a gentle decrease in the ratio of radial to axial force with decreasing filament separation. Second, when the attachment of S1 to actin occurs with $\gamma = 90°$, cross-bridges at actin sites at different azimuthal locations behave much more similarly than when $\gamma = 0°$. (The reason for this is discussed in the preceding paper.) Additionally, it is interesting to note that in certain configurations the radial component of the cross-bridge force may actually be in the direction of lattice expansion rather than compression.

Estimation of the Magnitude of the Overall Radial Force in a Contracting Muscle

Radial forces act to swell or compress the myofilament lattice. As such they may be evaluated in terms of a radial pressure acting upon the circumference of the myofilament lattice, or, since pressure is an isotropic property, we may calculate it from the forces acting on any surface. The hexagonal arrangement of the myofilament lattice of frog skeletal muscle is shown in Fig. 3 *A*. The larger circles represent the location of the myosin filaments and the smaller ones the actin filaments. An instructive surface to consider is one that encloses a single myosin filament, including its cross-bridges. We may draw the required volume, hexagonal in cross-section, with a length of one half-sarcomere, as shown in cross-section in Fig. 3 *B*. It is clear that this volume contains all the cross-bridges of the enclosed myosin half-filament and that an entire muscle fiber may be reconstructed from a repeat of similar volumes, so that the

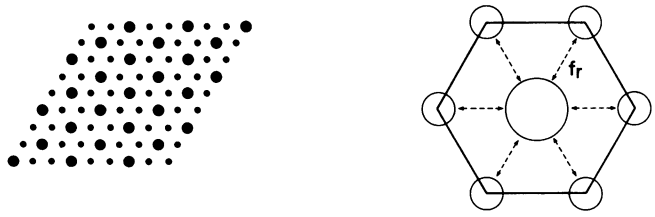

FIGURE 3 (*A*) Filament lattice structure of a frog skeletal muscle fiber. The large circles connote myosins and the smaller circles actin. (*B*) Myosin filament (large circle) with its nearest neighboring actins. Hexagon shows cross-section of a volume that encloses the central myosin filament and that extends over one half-sarcomere, thereby including all the cross-bridges from that half-myosin filament. The direction of the radial force component of the cross-bridge force, f_r, is shown by the dotted arrows.

forces and pressures acting upon the muscle fiber may be derived from the forces and pressures acting upon our volume.

Within our volume, using our present notation, each cross-bridge exerts a force, f_r, in the radial direction (along the arrows in Fig. 3 *B*), and a force, f_x, in the longitudinal (axial) direction (perpendicular to the arrows in Fig. 3 *B*). The radial forces act at 60° to the surfaces of our volume. As such, the radial force, f_r, from each cross-bridge may be resolved vectorially into two forces, each of magnitude $f_r\sqrt{3}/3$, and each acting normal to a surface. The total normal radial force per unit surface area, or in other words, the radial pressure, P_r, is therefore, $N\,(2f_r\sqrt{3}/3)/(6d_{AM} \cdot 0.5\,S)$, where N is the number of cross-bridges per filament per half-sarcomere, d_{AM} is the center-to-center distance between the actin and myosin filaments, and S is the sarcomere length, with $6d_{AM} \cdot 0.5\,S$ being the surface area around the circumference of our surface. (For the hexagonal lattice of the frog, d_{AA}, the actin-to-actin separation, is equal to d_{AM}.)

The axial force per unit cross-sectional area, P_x, is simply $Nf_x/(1.5\ \sqrt{3}\,d_{AM}^2)$, where $1.5\ \sqrt{3}\,d_{AM}^2$ is the cross-sectional area of our volume. Therefore we may write $P_r/P_x = [(N2f_r\sqrt{3}/3)/(3\,d_{AM}S)]/[(Nf_x)/(1.5\ \sqrt{3}\,d_{AM}^2)]$ or

$$P_r = P_x \cdot (f_r/f_x) \cdot (d_{AM}/S). \tag{2}$$

Eq. 2 is in a form suitable for the estimation of the radial pressure exerted by contracting cross-bridges. Since the cross-section of a frog fiber is ~ 80% filament lattice, P_x for a fully activated fiber is roughly on the order of the recorded muscle force per cross-sectional area, which is about 4 Mdyn/cm^2. Referring to Figs. 1 and 2, we can take $f_r/f_x \sim 0.1$ as an order of magnitude. For a frog skeletal fiber $d_{AM} \sim 25$ nm and $S \sim 2\ \mu$m, which yields the result $P_r \sim 5$ Kdyn/cm$^2 \sim 5 \times 10^{-3}$ atm.

Stiffness of Various Muscle Preparations

To estimate the effect of the radial component of the cross-bridge force upon the myofilament lattice, we need to know the stiffness of the lattice, that is, the responsivity of the lattice to pressure perturbations.

INTACT FIBERS A prevailing theory of the control of the myofilament lattice in the intact fiber is that of Matsubara and Elliott (1972). According to this theory, the volume of the intact fiber is governed by osmotic constraints of the type described by Boyle and Conway (1941), and the filament lattice simply swells to occupy the available space. Since the sarcolemma of a frog fiber does not resist radial pressure at sarcomere lengths between 2 and 2.5 μm (from Table II of Rapoport [1973]), this implies that there is no significant hydrostatic pressure gradient across the sarcolemma, so the osmotic pressure will be approximately the same on both sides of the membrane. The osmotic pressure of frog Ringer's solution is calculated to be on the order of 250 mOsm or $\sim$ 6 atm.

From the Boyle-van't Hoff pressure-volume relationship, a 1% change in fiber volume will produce a 1% osmotic pressure change or, conversely, a pressure of 6×10^{-2} atm will change the fiber volume by 1%. This pressure is at least one order of magnitude greater than the pressure generated by the radial component of the cross-bridge force, suggesting that the latter would have little effect upon either lattice or fiber volume in an intact fiber.

SKINNED FIBERS When the sarcolemma is removed from an intact fiber, the fiber and filament lattice swell (April et al., 1972; Matsubara and Elliott, 1972). According to the theory of Matsubara and Elliott, this follows because of the removal of the osmotic constraint upon fiber volume. As the filament spacing is now controlled by different factors than in the intact fiber, the stiffness of the myofilament lattice in this preparation, (its responsiveness to radial pressures) may be different from in the intact fiber.

The response of the filament lattice in the skinned fiber to radial pressure may be estimated from experiments that impose an osmotic pressure upon the fiber and measure the filament spacing change. Operationally, the osmotic pressure is created by adding a high molecular weight polymer to the bathing fluid of the fiber. Godt and Maughan (1977), who added high molecular weight polyvinylpyrrolidone (PVP K40) to a skinned frog fiber, found that if they assumed that PVP did not enter the fiber volume, then an osmotic pressure in the milliosmolar range changed the fiber diameter $\sim$ 10%. A. Magid, under similar experimental conditions, has measured filament spacing changes the same order of magnitude.[1] Since a pressure of 1 mOsm, which is equivalent to 24×10^{-3} atm, has about a 10% effect upon filament spacing, we might expect the radial component of the cross-bridge force, which, by our previous calculations is on the order of 5×10^{-3} atm in a fully-activated fiber, to produce about a 2% effect.

It should be mentioned that Millman and his colleagues (Millman and Racey, 1977; Millman and Wakabayashi, 1979) found virtually no change in the filament spacing of relaxed skinned frog or rabbit fibers, even upon addition of concentrations of PVP K40 as high as 0.4 g/ml. The reason for this discrepancy is not clear. If the filament lattice is stiffer than measured by Godt and Maughan and A. Magid, then the radial component of the cross-bridge force would have correspondingly less effect upon filament spacing.

DISCUSSION

Figs. 1 and 2 show the behavior of the cross-bridge radial forces for two types of cross-bridge attachment to actin. It appears to be a general property that the ratio of radial to axial force decreases with decreasing filament spacing. However, the magnitude of this ratio at a given filament separation depends upon the geometry and specific mode of attachment of S1 to

actin. When the S1 head attaches to actin with its long axis projecting through the filament axis (Fig. 2 *a*, Schoenberg [1980]), the radial-axial force ratio depends greatly upon the azimuthal location of the actin site (Fig. 1). When the attachment of the S1 is slewed, with its long axis parallel to the tangent at the actin site (Fig. 2 *c*, Schoenberg [1980]), cross-bridges at each of the sites behave more similarly. Most of the information concerning the mode of attachment of S1 to actin comes from the three-dimensional reconstruction of Moore et al., (1970). Their findings suggest that S1 more likely attaches to actin slewed, with γ at least 60°. The effective slew in their reconstruction is, for purposes of force vector analysis, probably greater than 60° due to the curvature of S1 they report. As discussed above and in Schoenberg (1980), this type of configuration produces a greater similarity in the behavior of cross-bridges attached to actin sites at different azimuthal position. (Although the computations in this and the previous paper were for cross-bridges originating from the myosin at an azimuthal angle of 0°, unpublished computations show this conclusion to be more or less valid for angles of cross-bridge origin, $0 \leq \theta \leq 60°$.)

If the mode of attachment is indeed similar to that deduced by Moore et al., or as in Fig. 2 *c* of Schoenberg (1980), then our computations derive the radial component of the cross-bridge force for a single cross-bridge to be on the order of one to two-tenths of the axial component. This result rests heavily upon the assumption that the cross-bridge does not transmit bending moments or shear to the myosin filament. This would be the case if the S2 portion of the cross-bridge behaved as a spring, as in the illustrated example of Huxley and Simmons (1971), or if the S1-S2 and LMM-S2 junctional regions functioned as flexible swivel joints, as suggested by Huxley (1969). The assumption that the S1-S2 junction functions as a swivel joint is compatible with the experimental findings of Mendelson et al. (1973), who used polarization anisotropy decay to measure the segmental flexibility of S1 and heavy meromyosin. On the other hand, Harvey and Cheung (1977), using the same technique, concluded that the LMM-S2 junction, while capable of showing flexibility, might not be flexible at physiological pH. If the S2 link between the S1 head and the myosin filament can support bending moments or shear, then the ratio of the radial to axial components of the cross-bridge force may be very significantly increased or decreased relative to the values reported here, depending upon the specific cross-bridge geometry.

Assuming the radial-axial force ratio to be on the order of 0.1, our analysis suggests that an increased radial pressure of $\sim 5 \times 10^{-3}$ atm accompanies full activation at sarcomere length 2.0 μm. Comparison of this pressure with the osmotic pressure in the intact fiber, as in the Results section, suggests that if the major constraint on the filament lattice is osmotic (Matsubara and Elliott, 1972), then the radial cross-bridge forces should have little effect in an intact fiber. This agrees with experimental x-ray findings that show no significant changes in filament spacing associated with activation of the intact fiber (Elliott et al., 1967; Huxley and Brown, 1967).

In skinned fibers, if it is correct to compute the osmotic pressure exerted by a PVP K40 solution from the membrane osmometric data of Vink (1971), as Godt and Maughan (1977) have done, and PVP K40 does not penetrate the myofilament lattice, then our computations, taking the stiffness of the skinned fiber lattice from Godt and Maughan's (1977) and Magid's[1] results, calculate a 1–2% change in filament spacing upon development of full tetanic tension in a frog muscle at rest length. This is smaller than the 15% change in filament

spacing measured by Shapiro et al. (1979) upon calcium activation of a skinned rabbit preparation, and also smaller than either the 11% change recorded by April and Schreder (1979) upon induction of low-tension rigor in a skinned crayfish fiber, or the 10% change measured by Maughan and Godt[2] upon induction of rigor in a skinned frog fiber. Perhaps the assumptions we have made underestimate the radial cross-bridge force, or possibly other factors, for example, changes in filament charge, also play a role.

At first glance it seems difficult to account for the entire effect measured by Shapiro et al. (1979) in terms of radial cross-bridge forces, especially if the total axial force produced by their rabbit preparation at 4°C is at all reduced relative to that of the frog. One way out of the dilemma is to assume that the total lattice compression that accompanies calcium activation or rigor induction has two components, one independent of axial force and one that behaves as described here. This might be tested in the rigor preparation, where the compression generated by the change in state at zero tension could be separated from any additional compression induced by pulling on the fiber to generate tension.

Another interesting effect is that reported by E. W. April at the 1979 Biophysical Society meeting.[3] He reported that under most conditions the myofilament lattice of a skinned crayfish fiber shrinks when the fiber goes into rigor, but when the lattice has already been very greatly shrunken using PVP, it actually expands upon rigor induction. Goldman et al.[4] have found similar results using skinned frog fibers. It is interesting to note (Figs. 1 and 2) that it is precisely under conditions of small filament separation that the radial component of the cross-bridge force may sometimes be expansive. If the lattice expansion upon induction of rigor is indeed due to the radial component of the cross-bridge force, then slightly stretching the fiber to increase the axial force should actually cause a further expansion of the filament lattice.

In 1968, Elliott suggested that both electrostatic and van der Waals forces might extend over interfilamentary distances and be responsible for the force balance in muscle. The radial component of the cross-bridge force is smaller in magnitude than the original estimates of either of these forces (Elliott, 1968). However, it is the difference between balancing forces and how much that difference changes with filament spacing that is important. For example, even if the balancing forces are as large as 10^{-3} dyn/μm filament length at equilibrium, the force imbalance created by a 1% change in filament separation might be no more than 10^{-6} or 10^{-5} dyn/μm (for example, see Fig. 6 of Elliott, 1968). 10^{-6}–10^{-7} dyn/μm is the range of the radial component of the cross-bridge force, assuming reasonable estimates of the number of cross-bridges and filaments and assuming the radial component for each cross-bridge averages out to about one-tenth the axial component. In summary, then, with our simplifying assumptions, the radial component of the cross-bridge force is computed to be an order of magnitude such that its significance cannot be of a priori ruled out.

I am greatly indebted to Dr. Dave Maughan for stimulating my interest in radial forces and for discussing with me much of his unpublished results. I would also like to thank Doctors Geoffrey Naylor and Paul Shapiro for many helpful discussions.

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[2]Maughan, D. W., and R. E. Godt. Personal communication.

[3]April, E. W. Personal communication.

[4]Goldman, Y. E., I. Matsubara, and R. M. Simmons. Personal communication.

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