slow loss of angular momentum of the solar type. By using the upper envelope of the observed distribution of rotation speed with spectral type and comparing it with the angular momentum-mass distribution for upper-main-sequence stars he claims that the rapid braking ceased abruptly at a rotation speed that was the same for all stars of the same spectral type and was about 2–2.5 times the present maximum observed values.

Some models of solar-wind braking do show a rapid initial braking¹⁴ if the magnetic flux is proportional to the angular velocity, but the timescale is a few times 10^8 years, whereas the giant stars must be braked within a few times 10^6 years. Since there is no direct evidence for a phase of very rapid braking for main-sequence stars, and there are many theoretical uncertainties about the approach to the main sequence, any clear picture of the early rotational history of main-sequence stars must await more data on very young main-sequence stars. For the

moment, the observations are qualitatively consistent with a solar-type loss of angular momentum, with the production of more effective winds by the deeper convective envelopes in cooler stars, and the loss of much of the angular momentum in the pre-main-sequence phase¹⁵. □

Robert Connon Smith is Lecturer in Astronomy in the Astronomy Centre, University of Sussex, Falmer, Brighton BN1 9QH.

1. Gray, D.F. *Astrophys. J.* **251**, 155 (1981).
2. Gray, D.F. *Astrophys. J.* **254**, 162 (1982).
3. Gray, D.F. *Astrophys. J.* **262**, 682 (1982).
4. Skumanich, A. *Astrophys. J.* **171**, 565 (1972).
5. Gray, D.F. *Astrophys. J.* **261**, 259 (1982).
6. Carroll, J.A. *Mon. Not. R. astr. Soc.* **93**, 478 (1933).
7. Gray, D.F. *The Observation and Analysis of Stellar Photospheres* (Wiley, New York, 1975).
8. Endal, A.S. & Sofia, S. *Astrophys. J.* **245**, 671 (1981).
9. Walter, F.M. & Bowyer, S. *Astrophys. J.* **245**, 671 (1981).
10. Svalgaard, L. & Wilcox, J.M. *A. Rev. Astr. Astrophys.* **16**, 429 (1978).
11. Durney, B.R., Mihalas, D. & Robinson, R.D. *Publ. astr. Soc. Pacif.* **93**, 537 (1981).
12. Smith, M.A. *Astrophys. J. Lett.* **242**, L115 (1980).
13. Soderblom, D.R. *Astrophys. J.* **263**, 239 (1982).
14. Belcher, J.W. & MacGregor, K.B. *Astrophys. J.* **210**, 498 (1976).
15. Vogel, S.N. & Kuhi, L.V. *Astrophys. J.* **245**, 960 (1981).
16. Fukuda, J. *Publ. astr. Soc. Pacif.* **94**, 271 (1982).

Biophysics

Muscle crossbridges in disarray

from Robert A. Mendelson

MUSCLE contraction has been thought to occur by the asynchronous rocking of myosin crossbridges between the parallel arrays of thick myosin filaments and thin actin filaments to produce a longitudinal translation between the two. This has been presumed, largely on the basis of X-ray diffraction and electron microscopy, to occur between the 45° orientation seen in rigor muscle and the relaxed orientation, in which nearly all the crossbridges have been thought to be roughly normal to the fibre axis with their distal ends towards the thin-filament-binding site. In this issue of *Nature*, however, Poulsen and Lowy¹ report that a significant fraction of myosin crossbridges in relaxed vertebrate muscle scatter X rays as if they were in solution rather than locked into the thick filament lattice.

The main-line technique for the study of filament movements, X-ray diffraction from living fibres, has been focused on the changes in the reflections arising from thin (mostly actin) and thick (mostly myosin) filaments. Isometrically contracting fibres have failed to show any new reflections or any significant change in intensity on the actin-based layer lines². Myosin reflections, except the 14.3 nm meridional, only decrease in intensity. Although the interpretation of these patterns is unclear, it appears that at least azimuthal disordering is required. The difficulties in interpretation of thick filament patterns and their changes may be relieved in the future by the work of Poulsen and Lowy, who observe that the diffuse scattering seen in relaxed fibres originates largely from randomized

myosin crossbridges (S1) rather than other proteins, and thus diffuse scattering data must also be considered in models of crossbridge action.

Poulsen and Lowy find that the shape of the diffuse portion of the scattering curve is circularly symmetric and its intensity profile matches that from isolated S1-solution scattering data. This match occurs within an angular range whose lower bound is large enough to eliminate particle-particle interference and excluded-volume effects, and whose upper bound minimizes the influence of S2 cross-sectional scatter on the scattering curve. When a relaxed fibre is stretched to a length where thick and thin filaments no longer overlap, there is a slight increase in disordered intensity. However, if non-overlapping muscle is then put into rigor a further increase in intensity of the disordered fraction occurs and almost no reflections can be seen. The authors take this situation, in which the thick filament structure is almost certainly perturbed, as being the base-line fully disordered intensity.

Modelling the decrease in the diffuse scattering curve at small angles relative to S1 solution data, the authors find that an 8 nm root-mean-square displacement (in every direction) is required. However, the layer-line intensity does not fall off rapidly enough with increasing angle to make such single-state crossbridge models tenable. In a model in which one fraction is completely ordered, the second fraction should have 9 nm displacement. The second fraction would be about 45 per cent in stretched muscle, and extrapolation of the authors'

data indicates it would be about 30 per cent in fully overlapped muscle.

On passing into rigor at full overlap, the diffuse intensity drops by a factor of about 1.8 and the pattern becomes compressed diagonally. This remaining intensity arises from crossbridges occupying actin sites randomly and thus creating substitution disorder. Modelling studies show that the observed compression can only be created by a crossbridge having a bend of about 45°.

How do these studies correlate with those using different methods? Fluorescence depolarization³ and saturation transfer electron paramagnetic resonance⁴ solution experiments, in which dipolar probes are rigidly attached to a single fast-reacting S1 thiol, indicate that brownian rotational movement of S1 moieties is much greater when myosin is in monomeric (soluble) form than when part of a synthetic or native thick filament. Presumably this large decrease in mobility is caused either by steric hindrance of the partner head or by the thick filament backbone, or both.

These methods have so far failed to detect a significant component of free or nearly free S1 motion in thick filaments of myofibrils. On the other hand, experiments aimed at measuring the average orientation of these probes in oriented muscle fibres⁵ have been unable to detect any ordering. Thus, probe results give a picture of scrambled heads with little mobility, presumably because at least a part of S1 is near the thick filament backbone. Since it is known that glycerinated rabbit *psaos* fibres have less order than living frog muscle, the lack of order found in these experiments perhaps should not be compared with the X-ray results. It is however unclear whether the model of the disordered fraction used by Poulsen and Lowy is consistent with the probe results. Fortunately, further probe⁶ and X-ray⁷ work is under way to study this question.

The current X-ray results raise fresh questions about the functional significance, if any, of the relaxed crossbridge order. For example, must myosin heads be ordered or disordered for attachment to the thin filament upon activation? Are myosin heads briefly adopting one of these states in active muscle? Whatever the significance of these results it is clear that simultaneous studies of both crossbridge order and orientation will be useful, if not obligatory, for unravelling the binding and micromechanism of crossbridges in contracting muscle. □

Robert A. Mendelson is at the Department of Biochemistry and Biophysics, University of California, San Francisco, California.

1. Poulsen, F.R. & Lowy, J. *Nature* **303**, 146 (1983).
2. Huxley, H.E. *et al. J. molec. Biol.* **158**, 637 (1982).
3. Mendelson, R.A. & Cheung, P. *Science* **194**, 190 (1976).
4. Thomas, D., *et al. Biophys. J.* **32**, 873 (1980).
5. Thomas, D. & Cooke, R. *Biophys. J.* **32**, 891 (1980).
6. Wilson, M.G.A. & Mendelson, R.A. *J. Muscle Res. Cell Motil.* **3**, 464 (1983).
7. Kress, M. *J. Muscle Res. Cell Motil.* **3**, 465 (1983).