given with each point). From this graph it is concluded that the number of sarcomeres along the long axis of this muscle increases only initially, that is, until the animal attains a body-weight of approximately 10 g. I have shown in earlier investigations[2,8] that embryonic differentiation of the biceps branchii of the mouse is not complete until the animal is approximately 8–10 g. Therefore this initial period of increase in the number of sarcomeres along the length of the muscle represents the completion of differentiation. After differentiation is complete then it would seem that the number of sarcomeres along the long axis of the muscle is fixed and no further increase takes place. My findings do in fact agree with Aronson's, who found that the post-embryonic elongation of striated muscle is due to the increase in length of each sarcomere and not due to the increase in the number of sarcomeres along the length of the muscle.

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G. Goldspink *

Department of Zoology,
Trinity College,
Dublin.

* Present address: Department of Zoology, University of Hull.

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HÆMATOLOGY

Plasma Angiotensinase Activity in Human Hypertension

Angiotensin is the pressure-active component of the renal pressor system and is suspected of playing a part in cases of human hypertension accompanied by primary or secondary renal lesions. The angiotensin-level results on one hand from its rate of synthesis by renin, on the other from its uptake by tissues and its inactivation by 'angiotensinase'. Angiotensinase activity results from a peptidasic enzyme system which has not yet been shown to be specific.

Blood or plasma angiotensinase activity has been measured with methods varying widely in principle; the level in hypertensive patients has been reported to be decreased[1], normal[2], and elevated[3–5]. These contradictory results led us to apply to 52 hypertensive patients a previously described method[6] based on: (*a*) the incubation of angiotensin with untreated plasma under standard conditions of time, temperature, *p*H, buffer and substrate concentration; (*b*) the assay of residual pressor activity in a rat preparation. Since the principle was strictly physiological, the results may have more significance than those obtained by measuring the degradation of a chemical or radioactive element of the angiotensin molecule. The degree of inaccuracy inherent to bioassays was reduced to 15 per cent by duplicating all assays, multiplying injections to each rat, using normal plasma as an extra control, and discarding less-than-ideal animal preparations.

Table 1. Mean Plasma Angiotensinase of 25 Normals and 52 Hypertensives. Normal Range is 80–120 per cent

Diagnosis	No. of patients	Mean ± *S.E.M.*	*P*
Normal	25	100 ± 2	
Essential hypertension	27	105 ± 4	> 0·05
Renal artery stenosis	14	109 ± 6	> 0·05
Malignant hypertension	11	115 ± 8	> 0·05

From the results shown in Table 1, it appears that the increase observed in the three hypertensive groups was not statistically significant according to the '*t*' test. No correlation was found between the angiotensinase-level and the blood-pressure reading at the time of sampling. Blood was obtained from one essential hypertensive patient every 3 h for 30 consecutive h to learn about the stability of the plasma enzyme-level; for an average value of 105 per cent, the standard deviation of the 11 samples was ± 7; considering the error due to technique, these fluctuations were of a minor degree. Two patients with renal artery stenosis in whom renal surgery was beneficial were examined before and after the operation: the post-operative plasma angiotensinase of one remained elevated, whereas it reverted to normal in the other.

These results confirm those of Klaus *et al.*[2], who observed a plasma angiotensinase increase of only 6 per cent in a group of 65 essential hypertensives. Those and the present results are not compatible with the production of a specific angiotensinase as an adaptive mechanism to prevent chronic hyper-angiotensinæmia.

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P. Biron *
R. Landesman
J. C. Hunt

The Rockefeller Institute, New York,
The New York Hospital, and
The Mayo Clinic.

* Present address: Department of Pharmacology, University of Montreal, Canada.

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Solubility of Fibrin Clots in Solutions of Heparin

Heparin has been shown to retard both the beginning and the general course of polymerization of purified fibrinogen[1]. Evidence will be presented that this anticoagulant, in solutions of low ionic strength, in addition to inhibiting the polymerization of fibrinogen, is able to depolymerize and thus dissolve preformed clots.

(*A*) *Effect of heparin on the polymerization of activated fibrinogen.* Saline solutions of four different preparations of bovine fibrinogen (34–54 mg/ml.) isolated by the method of Laki[2] and of two preparations of human fibrinogen (64 and 46 mg/ml.) obtained by ammonium sulphate precipitation of fraction I0 of Blombäck and Blombäck[3] (kindly supplied by Dr. A. M. Fisher of the Connaught Laboratories of Toronto) were mixed 1 : 1 with 2 M sodium bromide, *p*H 5·3. To these mixtures a thrombin solution of 100 N.I.H. units/ml. was added to a final concentration of 10 units/ml. and the samples kept at room temperature (24° C) for 3 h. One-tenth ml. from each mixture was then gently added to 9·9 ml. of each of several heparin solutions varying in concentration from 10–100 for human or 100–500 μg/ml. for bovine fibrinogen samples in 15 mm × 100 mm tubes. A small clot-like structure appeared immediately after the addition. The tubes were then stoppered and attached horizontally on a Kahn shaker with their long axis parallel to the motion of the machine. They were