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PLASMA CREATININE CLEARANCE IN THE DOG

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The concept of glomerular filtration rate (GFR) is sometimes difficult for the first year graduate student to comprehend. We have found with our students that by performing the following laboratory experiment they have a better understanding of the GFR. The purpose of this experiment is to determine the GFR which is in the dog equivalent to the renal plasma clearance of exogenous creatinine (1).

I. The renal plasma clearance, C, of a given substance, x, is equal to the virtual volume of plasma from which the substance is completely removed in one minute. If the substance is not destroyed intrarenally, its renal clearance must equal the amount excreted in the urine per minute, E_x, divided by the concentration of the substance in plasma, P_x, i.e.,

$$C_x = E_x/P_x. \qquad \text{(Eq. 1)}$$

E_x is equal to the product of the concentration of x in urine, U_x, in mg/ml and of the rate of urine flow, $\dot{V}$, expressed in ml/min., viz., $E_x = U_x\dot{V}$. After substitution, Equation 1 becomes

$$C_x = U_x\dot{V}/P_x. \qquad \text{(Eq. 2)}$$

The units of C_x are ml plasma/min; i.e. C_x is the virtual volume of plasma cleared completely of x per minute.

The concept of renal clearance as defined in Equation 2 has proven extremely useful, because it enables one to calculate the rate of glomerular filtration in an intact man or animal. If a substance is freely filterable at the glomerulus and is in addition neither reabsorbed from nor secreted into the renal tubular fluid by the tubular epithelium, the clearance of such a substance can be shown to be equal to the glomerular filtration rate as follows:

The quantity of any substance appearing in the urine per minute is equal to the quantity filtered at the glomerulus plus or minus the quantity secreted or reabsorbed by the tubule, accordingly,

$$U_x\dot{V} = P_x GFR \pm T_x \qquad \text{(Eq. 3)}$$

where GFR = glomerular filtration rate in ml/min and T_x = tubular contribution in mg/min.

In the case of a substance which is neither secreted nor reabsorbed, $T_x = 0$ and Equation 3 reduces to

$$U_x\dot{V} = P_x GFR \qquad \text{(Eq. 4)}$$

On rearrangement of Equation 4, we see that

$$GFR = U_x\dot{V}/P_x \qquad \text{(Eq. 5)}$$

If we compare Equations 2 and 5, it is seen that the clearance of x equals the glomerular filtration rate when tubular reabsorption or secretion of x is zero; i.e.,

$$GFR = C_x \qquad \text{(Eq. 6)}$$

when $T_x = 0$.

MATERIALS AND METHODS

Each group of students should have an anesthetized dog, surgical instruments, 10 ml syringe, 21 gauge hypodermic needle, tracheal cannula, polyethylene tubing (PE 260) for cannulating the ureters and femoral vein, bottle and tubing for the intravenous administration of 5% glucose solution, 20 ml syringe for injecting creatinine, test tubes, funnels, reagents for the determination of creatinine, and spectrophotometer.

1. The dog will be anesthetized with a solution of sodium pentobarbital (33 mg/kg body weight). Note dog's sex and weight.
2. Cannulate the trachea.
3. Cannulate a femoral vein and start an intravenous infusion of a solution containing 5% glucose at a rate of 4 drops per min. per kg body weight. Maintain the

infusion throughout the experiment to insure a satisfactory diuresis.

4. Expose the external jugular vein on one side. You will withdraw blood samples from this vessel for the determination of creatinine in plasma.
5. Prepare to inject the solution containing 8% creatinine (80 mg/ml) at the dosage of 200 mg creatinine per kg body weight. Inject one-half of the dose subcutaneously into each axilla. This has been found to require about 30 minutes for equilibration with extracellular fluid.
6. Make a midline incision 6-8 inches long in the lower abdominal wall to expose the urinary bladder. Grasp the bladder with a pair of hemostatic forceps and lift the bladder out of the abdominal cavity. Locate the ureters and place a ligature around each near its junction with the bladder. Cannulate each ureter with polyethylene tubing, directing the tip of the cannula toward the kidney. Secure the cannulae with ligatures. (It is suggested that you leave the bladder outside the abdominal cavity throughout the experiment. It has been found by experience that the act of returning the bladder to the cavity often displaces the tips of the cannulae sufficiently to obstruct the flow of urine through them.) Close the abdomen with hemostatic forceps and prepare to collect urine from the cannulae.
7. Allow 30-45 minutes for equilibration of plasma creatinine level. During this time, construct a data sheet and plan the remainder of the experiment. The rate of urine production should be at least 1 ml/min.
8. After step 7, commence collecting a 20-minute urine sample from both kidneys. Note the time. This is the first clearance period. (Allow the urine to drain continuously through the cannulae during the collection period, preferably directly into a graduated cylinder so that the rate of flow can be measured easily. With urine flow of 1 ml/min. or more, each clearance period should be 20 minutes. If the rate is less than this, you should increase the infusion rate.)
9. Using a syringe which has been rinsed with heparin (0.2-0.3 ml) to prevent clotting, collect 10 ml of blood exactly at the midpoint of each clearance period from the exposed external jugular vein. The time at which each blood sample is taken should be carefully noted. (Separate the plasma and cells from the blood by centrifugation and remove the plasma for subsequent use.)
10. At the end of the first clearance period, note the time and commence the second collection period immediately. For each urine sample, measure the volume accurately and calculate the average flow of urine in ml/min during the collection period; this value is $\dot{V}$ in Equations 2, 3, 4 and 5.
11. Continue the above general plan for at least three periods taking blood samples (accurately timed) at the middle of each period.
12. Creatinine concentration in plasma and urine is determined using the Jaffé reaction by the method of Folin and Wu (2) as given below:
    A. Add in sequence in a 125 ml Erlenmeyer flask, the following:
        (1) 24 ml of water.
        (2) 2 ml of plasma or urine (pipetted accurately with volumetric pipette).
        (3) 2 ml of 5% sodium tungstate dropwise with constant shaking.
        (4) 2 ml of 0.33N sulfuric acid.
        Note: The dilution is 1:15.
    B. Stopper, shake well and let stand for ten minutes, shaking at least once during this time. Filter the sample.
    C. Dilute each urine filtrate by taking 1 ml of filtrate and diluting to 50 ml. Do not dilute plasma filtrates.
    D. Color development:
        (1) Pipette accurately 5 ml of plasma filtrate or appropriately diluted urine into colorimeter tube.
        (2) For standard: 5 ml of standard creatinine solution containing 0.01 mg/ml.
        (3) For reagent blank: 5 ml of distilled water.
        (4) To each of above add:
            a. 2 ml of 0.04 M picric acid and mix by agitation
            b. 2 ml of 0.75 N NaOH and mix thoroughly.
        (5) Wait at least 20 minutes and read in colorimeter, set at 520 millimicrons, against the reagent blank set on 100% transmittance. Read the optical density (O.D.) of the plasma, urine and standard. The color is stable for at least 20 minutes but is sensitive to changes in temperature.
13. Calculation of data:
    A. Compute the concentration of creatinine in all samples from the readings obtained. Multiply by dilution factors so that creatinine concentration of original plasma and urine is expressed as mg/ml as shown below.
    B. Sample calculations of plasma and urine creatinine. Using the relationship:

$$\frac{\text{O.D. of plasma or urine}}{\text{O.D. of standard}} \times \text{concentration of standard (0.01 mg/ml)} \times \text{dilution (15 or 750)} = \text{concentration in plasma or urine (mg/ml)}$$

Calculate the concentration for each plasma and urine sample.

$$\text{Plasma: } \frac{0.301}{0.301} \times 0.01 \text{ mg/ml} \times 15 = 0.15 \text{ mg/ml}$$

$$\text{Urine: } \frac{0.372}{0.301} \times 0.01 \text{ mg/ml} \times 750 = 9.3 \text{ mg/ml}$$

    C. Compute the average urine flow for each period, $\dot{V}$.
    D. Calculation of clearance:

$$C_{cr} = \frac{U_{cr} \times \dot{V}}{P_{cr}}$$

U_{cr} = concentration of creatinine in urine (mg/ml.)

P_{cr} = concentration of creatinine in plasma at mid-point (mg/ml.)

$$C_{cr} = \frac{(9.3\ mg/ml)(1\ ml/min)}{0.15\ mg/ml} = 62.0\ ml/min.$$

E. Calculate clearance separately for each period. Take mean value for all periods.

RESULTS AND DISCUSSION

Typical results and calculations are given above and in Table I. Students are asked to hand in a table of their results

TABLE I. SAMPLE RESULTS FROM PLASMA CREATININE CLEARANCE IN THE DOG

Clearance Period	P_{Cr} mg/ml	U_{Cr} mg/ml	$\dot{V}$ ml/min	C_{Cr}(GFR) ml/min
I	0.15	9.30	1.0	62.0
II	0.13	5.80	1.3	58.0
III	0.12	5.83	1.3	63.2

showing their calculations along with the GFR and a few comments about the definition of renal clearance, its dimensions and information a clearance study may give concerning intrinsic renal mechanisms. This exercise has been very successful in our laboratory as well as being informative and relatively easy in execution.

The experiment has additional possibilities. If time permits the effects of a diuretic drug, an osmotic diuretic or saline loading can be examined. Renal acidification (e.g. effects of Diamox) or tubular transport (e.g. of PAH) can be studied.

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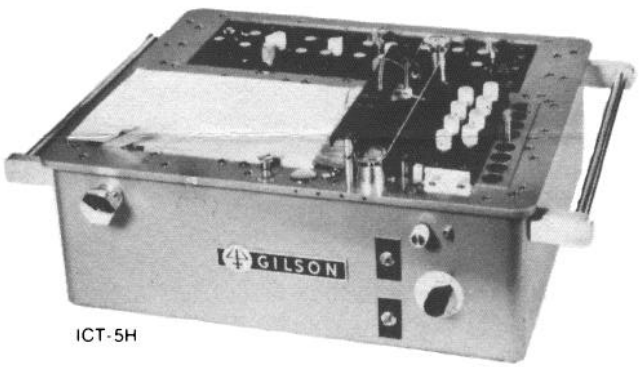

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COMPENSATORY HYPERTROPHY OF SKELETAL MUSCLE: CONTRACTILE CHARACTERISTICS

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INTRODUCTION

The understanding and study of muscle hypertrophy is of interest to those in a variety of academic disciplines and professions such as physicians, physical therapists, physical educators, physiologists and biochemists. The recent development of an animal model in which a rapid and extensive compensatory muscle growth can be induced has made it convenient to study the biochemical, morphological and contractile effects of hypertrophy. Such a model lends itself to student laboratory experiments. The changes that occur are of the magnitude that can be observed by students with minimal expertise. It also provides an opportunity for students to be exposed to small animal surgical technique and a useful protein assay. This experiment can serve as an extension of the frog muscle experiments used in many introductory physiology laboratory classes. Although the main purpose of this experiment is to study the contractile characteristics of normal and hypertrophied muscle, this model could be adapted for biochemical (e.g., amino acid uptake, DNA proliferation, etc.) or histological-histochemical student laboratory experiments.

MATERIALS AND METHODS

Animal Preparation – Most any species of rat may be used for this preparation. Male Sprague-Dawley albino rats with body weights of between 180-200 g have been successfully used by us. Compensatory hypertrophy of the plantaris muscle is induced by surgical removal of the synergistic gastrocnemius muscle. The plantaris muscle of the contralateral leg serves as the control. The animals are anesthetized with an intraperitoneal injection of 50-60 mg/kg of pentobarbital. Prepare one leg for surgery by shaving and scrubbing with alcohol. Make a midline skin incision from the popliteal fossa to the Achille's tendon (Fig. 1). Make another incision through the thin superficial biceps femoris muscle in order to observe the posterior aspect of the gastrocnemius muscle. Separate the medial and lateral heads of the gastrocnemius muscle beginning at the proximal end using a blunt probe. Free both heads, using blunt dissection, from the plantaris muscle which is imbedded mainly in the medial head of the gastrocnemius muscle. Transect the tendon of the lateral head first. Reflect it back and remove it *in toto* by transecting it near the origin (Fig. 2). Repeat the procedure with the medial gastrocnemius, making sure the tendon of the plantaris muscle remains intact. The surgical procedure generally results in only minimal bleeding. If considerable bleeding does occur, compress the area with a laboratory tissue or hemostat the specific region for a few minutes. Suture the skin incision with a 4-0 suture silk, using a continuous uninterrupted stitch (Fig. 3). Disinfect the surgical region. The contralateral leg can be sham-operated, but for a student laboratory it is not necessary. Return the animal to its cage for either 1 or 2 weeks. This will allow adequate time for a considerable amount of compensatory muscle growth (see below).

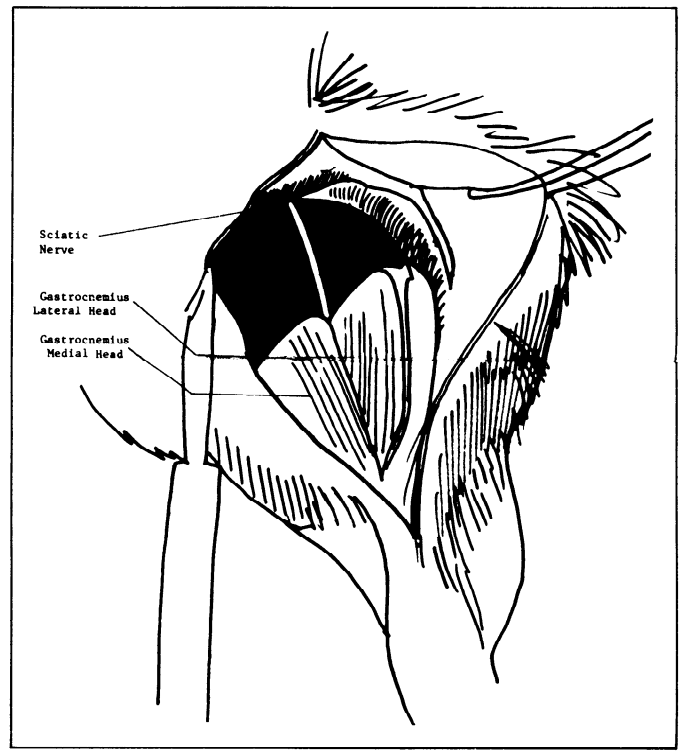

Fig. 1. Illustration of the surgical approach and view of the medial and lateral heads of the gastrocnemius muscle and sciatic nerve.

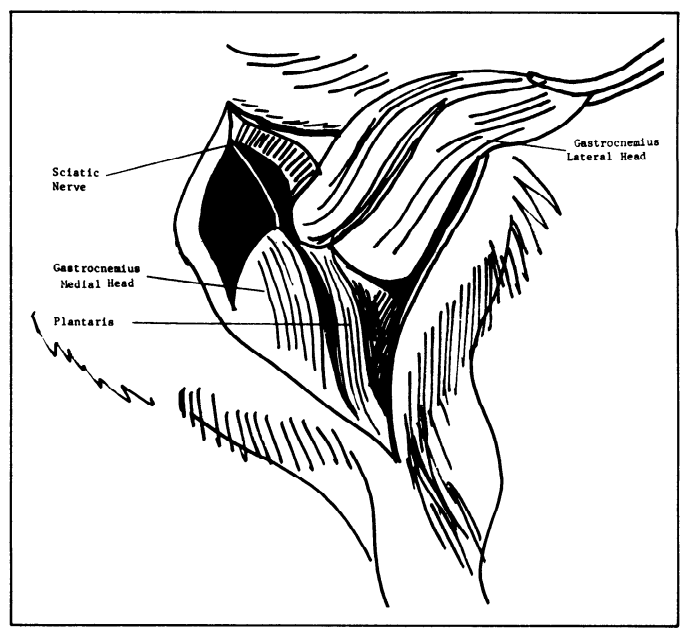

Fig. 2. A view of the plantaris muscle following transection of the tendon and reflecting back of the lateral head of the gastrocnemius muscle.

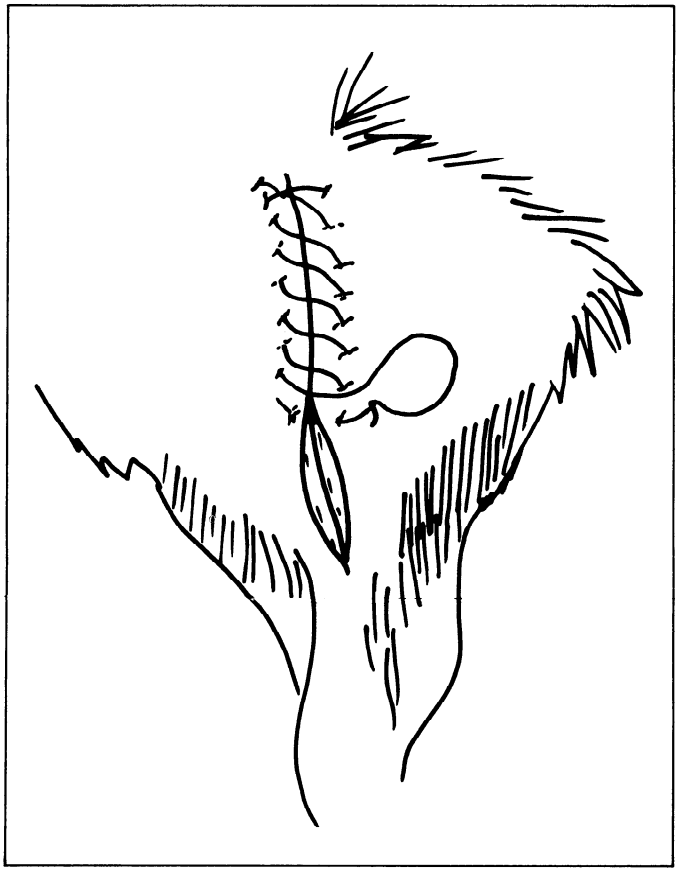

Fig. 3. Illustration of skin suture using a continuous uninterrupted stitch.

Surgical Equipment – Each surgical group should be supplied with the following: 1 pair small scissors, 1 blunt probe, 2 forceps, 1 scalpel handle, 1 No. 10 surgical blade, 4-0 surgical silk, disinfectant, surgical board with limb clamps, 1 curved cutting suture needle, 1 hemostat, 1 rat, 1 syringe (1 ml) and Nembutal.

Muscle Preparation and Contractile Characteristics – At a designated time post-surgery (i.e., 1-2 weeks) anesthetize the animal as described above. Excise *in toto* either the hypertrophied or control plantaris muscle. Set up the muscle preparation for determining contractile characteristics using an isometric transducer, stimulator and recorder as illustrated in Fig. 4. This procedure is described in most introductory physiology laboratory manuals. Keep the preparation moistened with a Tyrode solution. The muscle not being used at the time should be left intact until needed and the animal kept alive. Ligature the dissected leg if there is undue bleeding.

Determine the following contractile properties for both the control and hypertrophied muscle:

a. Latent period – The time (msec) lapse between the application of the stimulus and the commencement of tension development.
b. Twitch contraction time – The time (msec) required from the commencement of tension development until peak twitch tension is attained.
c. Relaxation time – The time (msec) period for tension to decay to the resting value.
d. Maximum rate of twitch tension development – Maximal contraction velocity as determined from the slope of the recording (g/msec).
e. Twitch isometric force – The force (g) developed during the twitch response.
f. Time-to-peak tension – The time (msec) required for the muscle to attain maximal tension development when stimulated with a voltage and frequency resulting in maximal fused tetany.
g. Tetanic isometric force – The force (g) developed at maximum fused tetany.

Before removing the muscle from the set-up, determine the cross-sectional area through the belly of the muscle at resting length (l_o). This can be accomplished by determining the circumference using a piece of suture silk or calipers.

Muscle Protein Determination – After removing the muscle from the set-up, blot with a laboratory tissue and determine the muscle wet weight (mg). Mince the muscle with scissors and scalpel blade and place it in a 15-20 ml tissue grinder. Dilute the muscle 1:20 (w/v) with distilled water and homogenize. Sample 0.1 ml of the homogenate and dilute to 10 ml with water (1:2000 w/v). Use 0.5 ml of this solution for the protein determination. Using the Lowry method (Lowry et al., *J. Biol. Chem.* 193: 265, 1951), as summarized below, determine the muscle protein content.

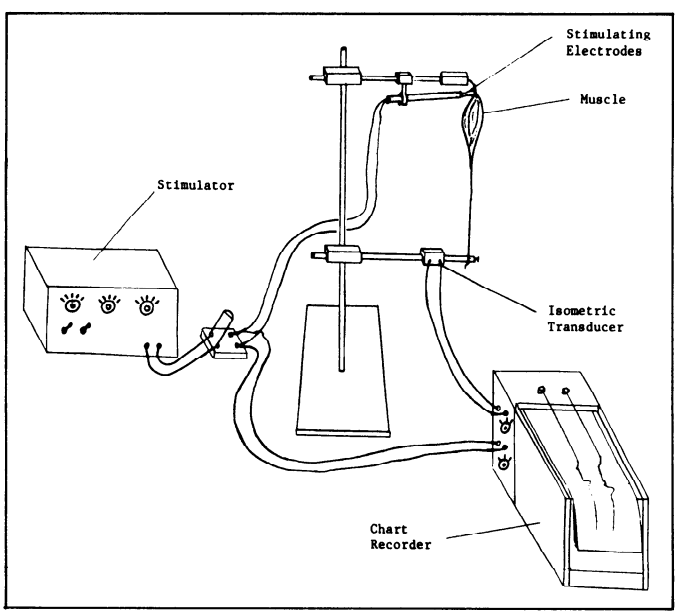

Fig. 4. A typical set-up for determining muscle contractile properties.

Reagents

1. 3% Na_2CO_3 in 0.1 N NaOH
2. Alkaline copper reagent:
   Add 1 ml of 2% $CuSO_4 \cdot 5H_2O$
   Add 1 ml of 4% Na-tartrate
   (Add both to the same 50 ml flask)
   Dilute to 50 ml volume with reagent # 1 (prepare twice weekly)
3. Folin (phenol) reagent
   Dilute 1 part of phenol reagent with 2 parts H_2O just before use.
4. Standards: 50, 100,300 micrograms proteins (albumin) per ml.

Procedure:

1. Add 0.5 ml of standards and experimental samples to appropriately labelled test tubes. Add 0.5 ml H_2O to the tube designated as the blank.
2. Add 5.0 ml of alkaline copper reagent to each tube.
3. Mix well and let stand for 10 minutes or longer.
4. Add 0.5 ml of the dilute phenol reagent and mix rapidly (i.e. within 2-3 sec.).
5. Let stand for 30 minutes.
6. Read O.D. at 660 nm.

Calculation:

Protein Concentration (mg protein/g muscle wet weight) =

$$\frac{\text{O.D. unknown}}{\text{O.D. known}} \times \text{concentration of known (mg/ml)} \times \text{dilution (2000)}$$

Materials for Protein Determination – Each group of students should have available the following items: 1 test tube rack; 12 test tubes; 1 (20 ml) tissue grinder; graduated pipettes (1, 0.1 ml; 1, 1.0 ml; and 6, 1/2 ml pipettes); spectrophotometer and cuvettes; and reagents listed above for protein analysis.

RESULTS

Students should calculate the data and organize it in tabular form similar to that in Table 1. The contractile characteristics of normal and hypertrophied muscle should be compared. The references provided will direct the student in the understanding and discussion of the findings.

TABLE 1. CHARACTERISTICS OF CONTROL AND HYPERTROPHIED MUSCLE

Muscle Characteristics	Control vs Hypertrophied Plantaris muscle Δ %
Muscle wet weight (mg)	+ 40–60
Muscle cross sectional area (mm^2)	+ 40–60
Muscle protein content (mg/total muscle)	+ 35–50
Muscle protein concentration (mg protein/g muscle wet weight)	– 15–(–20)
Latent periods (msec)	+ 5–10
Twitch contraction time (msec)	+ 10–20
Relaxation time (msec)	+ 10–20
Twitch isometric force (g)	+ 20–40
Time-to-peak tension (msec)	+ 20–30
Tetanic isometric force (g)	+ 30–50
Twitch force/tetanic force ratio	NC
Maximal tetanic force/cross sectional area (g/mm^2)	– 15–(–20)
Maximal tetanic force/mg protein (g force/mg protein)	NC
Maximal rate of tension development ($g/sec/mm^2$)	– 15–(–30)

DISCUSSION

This laboratory experiment will require 2-3 laboratory periods to complete. In the first laboratory session, the surgical procedure can easily be accomplished. Students may also have time to become familiar with the Lowry protein assay by determining the protein concentration of prepared standards. The reagents should be prepared in quantity by the laboratory instructor if the experiment is to be completed in 2 sessions, otherwise the students should prepare them. In the next week's laboratory session the contractile properties, the protein determination on the muscles can be accomplished. Some instructors may prefer to take 3 laboratory sessions for the experiment. The first session can be used to accomplish the surgical procedure, the second week's session for becoming familiar with the protein assay and equipment for muscle set-up and the third week's session for determining the contractile properties and the muscle protein content.

The surgical removal of the synergistic gastrocnemius results in a rapid and dramatic increase in the wet weight of the ipsilateral plantaris muscle. We (4) have observed a 45% increase in the wet weight of the plantaris muscle of normal animals within 16 hours post-surgery. Compensatory muscle growth continues up to approximately 30 days and becomes completed within 30-40 days post-surgery. Upon completion the hypertrophied muscle has a 80-100% greater mass than the control muscle. The time course of the response is shown in Fig. 5. The protein concentration is significantly lower (15-18%) in the hypertrophied versus the control muscle for up to 15 days and then returns to normal. Possible reasons for the reduction in protein content have been discussed elsewhere (4). A considerable amount of investigation has been accomplished in relation to the biochemical correlates associated with the acute phase of compensatory hypertrophy (2). The references listed will provide adequate detailed information for the student.

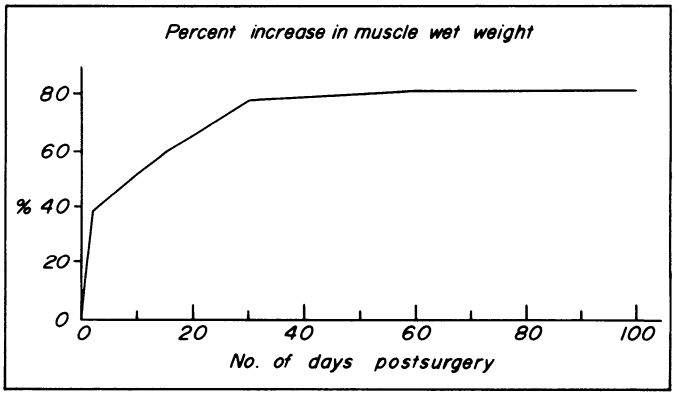

Fig. 5. Graph showing the time course of compensatory muscle growth using the described procedure.

The acute phase of compensatory hypertrophy is associated with contractile changes (e.g., prolongation of contraction time and reduced tension development per unit mass) (6). Possible explanations for these changes are: (a) reduced protein concentration, (b) increase in the relative amount of connective tissue, and (c) reduced specific activity of the enzyme myosin ATPase (3,4,8). Following chronic compensatory hypertrophy (approximately 50 days) there is a commensurate increase in tetanic force and muscle hypertrophy resulting in a similar tetanic force per unit cross sectional area and per unit wet weight for the control and hypertrophy muscle. Maximal speed of contraction of the hypertrophied muscle remained slow even after a long-term overload (1). The increase in contraction time has been suggested to result from an increase in the number of slow-twitch fibers (5) and an increase in angle of muscle fibers in the compensated muscle (1). The student should be aware that there are differences between the acute and chronic effects of hypertrophy and that the data obtained from acute hypertrophy cannot necessarily

be extrapolated to long-term consequences.

We wish to thank Mr. Peter Herrmann for assistance in preparing this manuscript.

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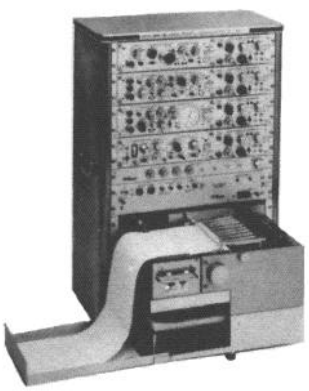

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BOOK REVIEW

Psychological Aspects and Physiological Correlates of Work and Fatigue (Simonson and Weiser, Contributors and Editors). Charles C. Thomas, 1976.

This extension of the original volume *Physiology of Work Capacity and Fatigue* published in 1971 comprises one of the last publications with which Ernst Simonson was associated before his untimely death. Throughout his career Simonson was interested in the psychological and physiological, including neurological, correlates of the identifiable aspects of the fatigue process. In the introduction, he states – "It is obvious that separation of physiological and psychological aspects of work and fatigue is to a large degree arbitrary." The various contributing authors have, in large measure, tried to avoid separation. Thus, the two volumes are indeed complementary if not as integrative as a single volume might have been. It is noteworthy that a 3rd volume on the clinical aspects of the fatigue process, although conceived, will never appear unless one of Simonson's colleagues takes on the editorial task as Philip C. Weiser did for completion of the current 2nd volume. Of particular value in the *Psychological Aspects and Physiological Correlates of Work and Fatigue,* are the 5 contributions from German authors all of whom have worked extensively on fatigue but whose work may not be well known to American investigators as judged by literature citations.

The volume is divided into 7 sections and 15 chapters plus an introduction and an epilogue. The sections are respectively: Biophysical Models and Physiological Background, Motor Aspects, Sensory Aspects: Vision, Aspects of Central Processing, Aging, Introspective Aspects of Work and Fatigue, and Closing Comments. In general, fatigue is treated as decreased work or other capacity whether that capacity is in terms of fine manipulative skills or gross motor performance. Monotony and boredom while important psychological inputs to one's perception of fatigue, are not dealt with in detail.

Those primarily interested in the physiological rather than the psychological aspects of fatigue will find the following chapters quite informative: P.C. Weiser, Interrelationships of the Motor and Metabolic Support Systems During Work and Fatigue; O.H. Schmitt, Biophysical Models for Studying Work and Fatigue; T.A. Easton, Reflexes and Fatigue: New Directions; K.C. Hayes, Reaction Times, Reflex Times and Fatigue; W. Rohmert, Motor Coordination; and S.H. Bartley and E. Simonson, Use of Visual Methods for Measurements of General Fatigue.

A chapter by S.H. Bartley entitled What Do We Call Fatigue reveals the general dilemma of using a single word–fatigue–to describe a wide variety of events varying from incapacitation to motivational change and from cellular deterioration to disorganization in gross motor function. To quote from H. Schaeffer in the Epilogue–"The science of fatigue has to do with both spheres: the rational world and the world of subconscious processes, which are probably more decisive for the general course of things than rationality. Both spheres, of course, can be explored only in a scientific way, and this is the very appeal of such a science of fatigue." Aside from its general interest and the fact that an attempt is made to maintain continuity of perspective in psychosomatic or psychophysiological terms, the book is quite useful for not only are a variety of hypotheses presented but the review of the relevant literature is generally excellent and the author and

subject indexes well prepared. It should remain an important reference volume for years to come.

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BOOK REVIEW

Structure of the Autonomic Nervous System by G. Gabella. Halsted Press, New York, pp. 214, 1976.

The book entitled "Structure of the Autonomic Nervous System," by G. Gabella, gives detailed descriptions of almost all aspects of the structure. The entire book is a kind of encyclopedia on this subject, and the literature cited covers from very old papers of the 19th century to the current ones. Description is in a narrative style, and some sections contain rather many paragraphs, each of which extends from one half to over one page. This causes some trouble when readers try to find specific information under a subheading, which sometimes covers a very broad topic, as exemplified by "Ganglia of the sympathetic chain." In general, subheadings are not always appropriate, being at times too broad or too narrow. The book would be much easier to use if more appropriate subheadings with sub-subheadings for each major topic were used.

Most parts of the book give a detailed account of the structure of the tissue in question, such as the total number of cells in a ganglion, its change with growth, electron microscopic observations of neurons, glial cells, synapses, and subcellular organelles. Enzymes such as cholinesterases and catecholamines as examined by flourescence techniques are also described. However, with the exception of a few cases such as those on page 112, the implication of histological data to electrophysiological activity is not always described. This makes the book less attractive to those whose prime interest lies in physiology rather than anatomy. Addition of more illustrations would be of immense help for general readers, who are not always anatomists.

The book is divided into ten chapters. Chapter 1 is for introduction. Chapter 2 gives a detailed account of the structure of the sympathetic ganglia, including chromaffin cells, the ganglia of non-mammalian animals such as amphibians, reptiles and birds, preganglionic and postganglionic fibers, and afferent fibers. Chapter 3 is devoted to the structure of the adrenergic fibers and includes a brief description of release, uptake, synthesis and inactivation of the transmitter as related to the structure. Structural changes during development are the main topic of chapter 4, which covers the effects of nerve growth factor to those of denervation. The cultured sympathetic ganglion is also discussed. Chapter 5 briefly describes the paravisceral ganglia. Chapter 6 deals with the ciliary ganglion and contains the interesting electrophysiological behavior of the ciliary ganglion of birds. The intramural ganglia are described in chapter 7, and the vagus nerve in chapter 8. Chapter 9 gives a brief account of the autonomic efferent neurons in the central nervous system, and chapter 10 briefly summarizes the innervation of a variety of organs. All references are collected at the end of the book and are quite comprehensive.

In spite of some drawbacks pointed out above, this book contains highly useful information concerning every structural aspect of the autonomic nervous system. This is not the type of book which can be recommended as a text for freshman graduate or medical students, but is a useful reference, especially for research workers who are interested in the autonomic nervous system.

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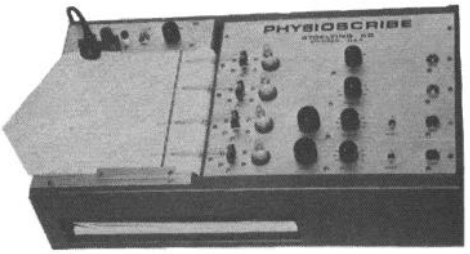

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