"burning" character, "cutting," "stabbing," or "gnawing" like toothache. A pain of the intermittent "colicky" or "gripping" type does, however, admit of a more or less satisfactory explanation, for it indicates an unduly violent effort on the part of involuntary muscles to expel some foreign or excessively irritative material, or to overcome obstruction. We encounter this type of pain in chronic intestinal obstruction, and also in the presence of renal and biliary calculi. With this exception, however, the other characters of pain seem to be inexplicable; and from the fact that the same disease may cause different types of pain we are unable—at least at present—to attach much clinical importance to these varied manifestations.

When we come to consider pain from the point of view of degree or severity we are brought up against some very interesting problems. The first of these concerns the individual regarded as a collective organism. Some patients have a much more sensitive nervous system than others, so that what may be easily endured by one may be almost beyond toleration by another. These normal constitutional variations are always worthy of careful consideration. Then we have the effect of certain blood conditions which may have an augmentative action on the sensitiveness of the system to pain. In fever, for instance, the increased temperature may unduly excite those cells in the spinal cord or brain which are concerned in the production of pain; and these cells may also be similarly affected in certain anaemic conditions of the blood—possibly, from impoverishment, the result of deficient or defective nourishment, they are more susceptible to stimulation.

With regard to the distribution of pain, there are equally some very interesting problems to solve. To give an instance or two of what I particularly refer to: Why should pain in appendicitis sometimes be felt in the left iliac region? Why should stone in the kidney on one side give rise to pain on the other—a reno-renal reflex as it is termed? You will remember that, when discussing the causes of "referred" pain, I indicated how it was in all likelihood explained: that certain cells in the posterior cornua of the spinal cord, lying in close contact with other cells, when stimulated, transferred that stimulation to those other cells, and thereby caused an impression to be produced which accounted for the particular location of the pain. Now, these cells in the one cornua are connected by nerve filaments with similar cells in the other cornua; and if we assume that, for some unexplained reason, the cells in the opposite cornua are more sensitive to exciting influences than those on the side of the disease, we get the impression produced that the disease is on that particular side.

Let me instance a case that came under my observation many years ago—in the pre-x-ray days. The misfortune which followed the operation performed could not very well happen now when, by means of photography, we are so able to assure ourselves as to the existence or not of a stone. The patient referred to complained of pain in the right lumbar region, strongly suggestive of renal calculus. The kidney was cut down upon and explored, but no stone was found. The patient died four days after the operation from suppression of urine, and at the necropsy a stone was found in the left kidney.

It is but a further extension of the explanation that I have just given to understand how it is that pain may be widely diffused or distributed and not "referred" to any particular part or region. For if owing to undue excitability of all the cells in the spinal cornua more than those normally stimulated become involved, you can readily appreciate how much more extensively diffused must be the area of pain felt. Thus it sometimes happens that when these cells are in an unduly excitable condition, or when, perchance, the stimulus itself is of so acute a nature that it oversteps the limitations of even normal cells, the pain resulting from disease of an abdominal organ may not be limited to any particular part of the epigastric, umbilical, or hypogastric regions, but be more or less diffused over two or more of these regions.

I have not time to touch upon many other points which the study of abdominal pain presents, and this short hour has only permitted me to deal with the fringe of my subject. If I have done no more than to excite your interest I feel sure that that awakened interest will prove useful to you in the future pursuit of your profession.

THE PHYSIOLOGICAL COST OF MUSCULAR WORK.

BY

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AND

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In the article published in this JOURNAL (May 7th, 1921, page 669) Waller and De Decker record results giving the physiological cost of muscular work of colliers, shoemakers and others as measured by the exhalation of CO₂. We are carrying out experiments of a similar nature with Dr. E. Schuster's modification of Martin's bicycle ergometer—doing 13,000 kg.m. of work per hour—using both the method employed by the above observers and the standard Douglas bag method, in which both the CO₂ output and oxygen use are measured, and the heat loss calculated therefrom, as recommended by Cathcart.¹ As the results we have obtained with these two methods rarely agree, we consider that the possibility of limitation of Waller and De Decker's method should be emphasized before deductions other than the broad difference between light and heavy work are drawn from their results. We consider that a source of error is the short duration—thirty seconds—of the period for obtaining the sample of expired air. We have found that the volume of CO₂ expired while at rest or at work varies greatly during consecutive half-minute periods (Table I), and also that the CO₂ exhalation per second obtained in a thirty-second sample is rarely similar to that obtained per second during a four-minute period (Table II).

TABLE I.—Consecutive Half-minute Samples of Expired Air.
(Subject J.A.C., unaccustomed to use of mouthpiece.)

Date.	Resting.			After working 1 hour on ergometer.		
	Sample.	Heat Loss.*	CO ₂ (c.cm. per sec.).	Sample.	Heat Loss.	CO ₂ (c.cm. per sec.).
April 1, 1921	1st	0.49	1.3	1st	4.76	15.7
	2nd	0.76	2.1	2nd	4.25	13.7
	3rd	1.10	4.1	3rd	4.00	12.6
April 4, 1921	1st	1.50	3.8	1st	4.49	13.1
	2nd	1.45	3.7	2nd	4.52	13.6
	3rd	1.24	3.3	3rd	4.00	13.8

* The heat loss in all tables is in terms of millicalories per square centimetre per second. Cathcart's method for calculation being used.
On April 4th J. A. C. had become more accustomed to the use of the mouthpiece.

TABLE II.—Half-minute and Four-minute Samples.
(No food taken. Subject C.P., accustomed to use of mouthpiece.)

Date		Half-minute Sample.		Four-minute Sample.	
		Heat Loss.	CO ₂ (c.cm. per sec.).	Heat Loss.	CO ₂ (c.cm. per sec.).
April 25, 1921	After 2 hours' work on ergometer	5.8	17.7	4.6	12.7
	After 3 hours' work on ergometer	6.3	19.2	—	11.0
	After 4 hours' work on ergometer	5.4	16.4	4.2	11.2
May 4, 1921	After 2 hours' work on ergometer	3.3	8.3	5.1	13.6
	After 4 hours' work on ergometer	4.8	12.7	4.9	13.2

Another factor on which these observers do not apparently place sufficient emphasis is the influence of food. Benedict and Carpenter have shown that the ingestion of all kinds of food in any amount results in an increment of metabolism.² We have obtained similar results. We think that there is a danger of a reader misinterpreting the charts and tables as presented by Waller and De Decker. In nearly all their cases the cost per hour of "practically maximal and constant" work appears to increase as the day proceeds. Johansson's curves of diurnal temperature

and CO₂ output of a normal individual living a normal life vary together. This, we suggest, is due to the influence of food, and that if this be excluded the CO₂ cost per hour of this constant work remains the same at any period.³ Benedict and Cathcart's well-known experiments on a trained bicyclist in the post-absorptive condition, show clearly that severe constant work requires the same amount of CO₂ exhalation per second at the end of three hours of work as during the first hour.⁴ Using a bicycle ergometer and four-minute samples of expired air, we have obtained similar results (Table II). It is only after taking food (Tables III and IV) that our subjects show a rise of CO₂ exhalation similar to the increase shown in Waller and De Decker's charts.

TABLE III.—*Showing Effect of Food whilst Resting.*
(Subject C.P., resting. Ten-minute samples of expired air.)

Date.		Heat Loss.	CO ₂ (c.cm. per sec.).
April 20, 1921	One hour after food ...	1.13	3.2
March 30 ..	" " " " ...	1.16	3.0
April 20, 1921	Two hours after food ...	1.43	3.7
March 30 ..	" " " " ...	1.47	4.0

TABLE IV.—*Showing Effect of Food whilst at Work.*
(Subjects C.P. and L.H., working on ergometer. Four-minute samples of expired air.)

	Heat Loss.			CO ₂ (c.cm. per sec.).		
	L.H.	C.P.	C.P.	L.H.	C.P.	C.P.
Two hours before food	3.3	4.9	4.23	12.7	12.3	11.8
One hour before food	3.5	4.8	3.88	11.9	12.0	11.1
Food taken.						
One hour after food ...	3.9	4.2	4.9	13.1	11.5	12.6
Two hours after food	4.2	5.1	4.86	14.9	14.3	14.7

It is well known that a fatigued or uncomfortable subject carries out a given amount of work at a greater cost than when normal, but it does not seem reasonable that the colliers and shoemakers under consideration are so unskilled and of such poor calibre as to show signs of fatigue after a couple of hours' constant work, when this work is of the degree specified.

REFERENCES.

¹ *Journal of the Royal Army Medical Corps*, November, 1918.
² Publication No. 261, Carnegie Institute, Washington, 1918. ³ Lusk, *Science of Nutrition*, Third Edition, p. iii. ⁴ *Muscular Work*, p. 66. The Carnegie Institute, Washington, 1913.

COMPARATIVE SYSTOLIC BLOOD-PRESSURE READINGS IN THE ARM AND LEG IN AORTIC INCOMPETENCE.

BY

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THE higher systolic reading by the blood-pressure instrument in the leg compared to that in the arm, when both are at the level of the heart, appears to have been so generally accepted as a characteristic and even diagnostic sign in aortic incompetence that it would seem to serve a useful purpose briefly to review the subject, so as to consider whether or not this conclusion is a sound one, and, if not, what are the fallacies which underlie it.

Leonard Hill, with the co-operation of Martin Flack and W. Holtzmann,¹ first made the observation that in all cases of aortic regurgitation there is a marked difference between the reading of the systolic arterial pressure in the arm and in the leg, that in the leg being the higher, the observation being taken in recumbency. These observers further state that "when such patients are lying quiet in bed this difference is a diagnostic sign of aortic regurgitation." They found that the difference of pressure is not abolished by the method of oscillating the pressure up and down near the obliteration pressure. The explanation which they give of the difference is that the "big systolic waves are conducted better

down the leg arteries, these being slightly thicker and more contracted and rigid." In a later paper Hill and Rowlands² brought forward additional cases illustrating the same phenomenon. H. A. Hare came to similar conclusions to those of Hill.³ H. D. Rolleston⁴ confirmed the observation, and among other conclusions found that the difference between the maximum systolic blood pressure in the arm and leg is most marked in uncomplicated and compensated aortic regurgitation, but "is much less marked in aortic regurgitation when the compensation is failing, and in cases of combined aortic regurgitation and mitral disease"; further, that "in recent aortic regurgitation before compensation is established the difference is slight or may be absent." J. R. Murray⁵ published records showing similar differences. J. Heitz⁶ recorded observations on 51 cases concerning this phenomenon, and whilst finding it in the majority of cases, disagrees with Hill as to its constancy. He, moreover, draws attention to the important fact that the difference is to be found in other classes of cases. He says: "L'élévation de pression dans les artères tibiales peut être constatée chez la plupart des sujets atteints d'insuffisance aortique" . . . but "elle peut manquer dans un certain nombre de cas." Again: "L'élévation de la pression systolique aux membres inférieurs peut se constater avec une certaine fréquence chez des sujets hypertendus et non atteints d'insuffisance aortique." He has also found it in abdominal aortitis. He concludes that the sign "ne présente donc pas de valeur diagnostique absolue."

Such a difference of pressure readings may certainly occur in other conditions than aortic incompetence. Thus I have observed it in high-pressure cases associated with arterio-sclerosis,⁷ and, according to Clifford Allbutt, this observation was first made by Volkmann.⁸ Further, MacWilliam has found the same difference in some patients with sclerotic arteries.

The conclusions drawn from my own observations are as follows:

1. Although higher systolic readings in the leg than in the arm appear to occur in the majority of cases of aortic incompetence, there is a large minority in which they do not obtain.

2. There is no evidence that such cases, when they do exist, are the result of the actual aortic incompetence rather than of the commonly associated conditions of hypertonic contraction or hypertrophy of the muscular coat, with or without accompanying sclerotic changes, conditions which also occur independently of aortic incompetence.

3. In support of the arterial explanation of such differences is the fact that the large majority of cases of the series in which the arteries were thickened showed the difference, being a much larger proportion than obtained in the total number of cases of the series (all of which had aortic incompetence); and further, that in the adult cases of aortic incompetence, nearly all of which had thick arteries, the phenomenon was more than twice as common as in the children, in none of whom were the arteries thickened.

4. If due to the aortic incompetence it would seem reasonable to suppose that the difference of reading should show some relationship to the degree of incompetence, which for practical purposes may be considered as measured by the pulse pressure. No parallelism, however, exists between these arm-leg differences and the corresponding degrees of pulse pressure.

5. In the few cases in which this was tried, in the majority the difference of pressure was abolished by repeated recompression, a fact decidedly in favour of the arterial explanation. (In cases of a higher leg reading than that in the arm in arterial disease it has been frequently found that the difference is abolished by such recompression.)

Method of Observation and Apparatus.

This paper being based on observations which were spread over many years, different instruments were used, but these were one or other of the modifications of the Riva-Rocci method—namely, Stanton's instrument, Oliver's compressed air haemomanometer, Martin's instrument, and that of French.

The armlets used were at least 12 cm. breadth, except in a few of the children, in whom an armlet of 10 cm. was used for the arm observations. When not otherwise stated, the armlet for the lower extremity was placed on the calf. In the few cases in which the thigh was used for compression a specially broad armlet (18 cm. broad) was used. All readings were taken on reducing the pressure after obliteration of the pulse. Unless otherwise stated, the tactile method was the one adopted. In the readings by this method the radial artery was used for the upper extremity, the dorsalis pedis or posterior tibial for the