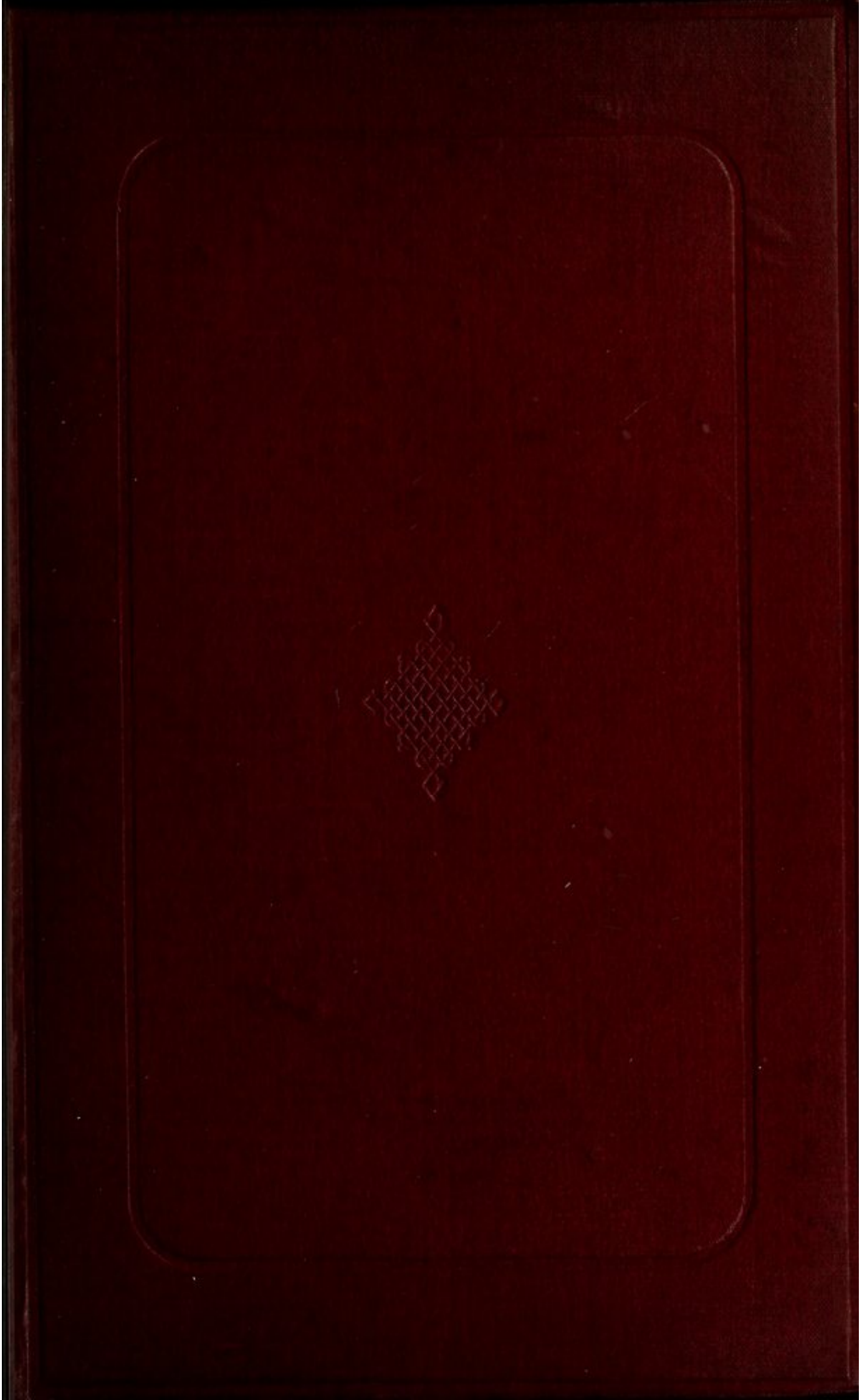
Physiological and clinical studies / by Alexander James.



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Physiological and Clinical Studies.

PHYSIOLOGICAL AND CLINICAL STUDIES.

BY

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PREFATORY NOTE.

The following Papers have been published from time to time in the medical journals. With some slight additions they are now collected in book form. The main object of the Author in thus presenting them to the Profession is to exemplify, by means of studies in which he has long felt a special interest, the aid which a consideration of the laws and phenomena of Physiology may render towards the elucidation of some complex problems in Practical Medicine.

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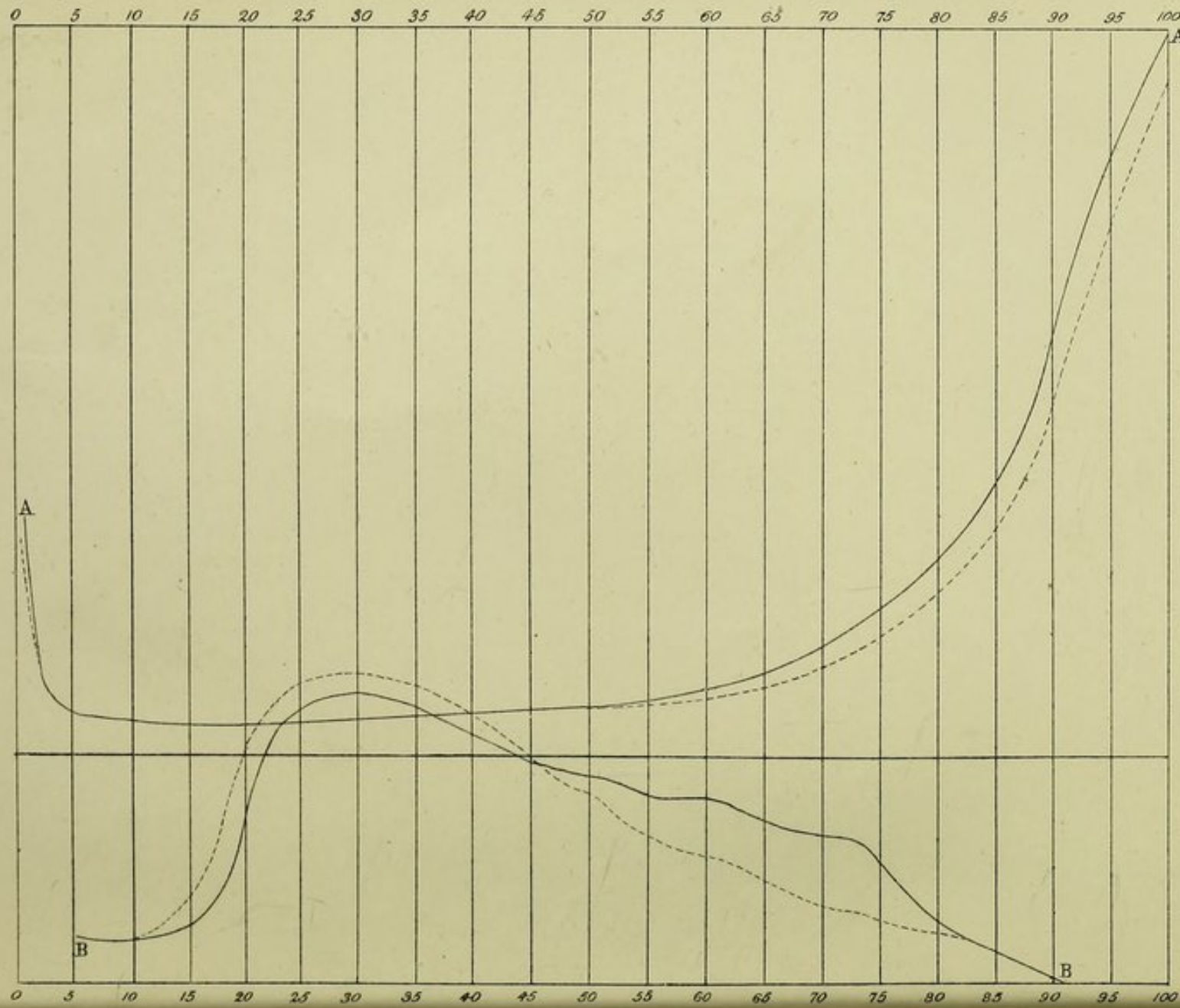


Fig. I.

A. *General Mortality.*

Males. ———

Females. - - - - - -

B. *Phthisis Mortality.*

Males, ———

Females, - - - - - -

N.B. *Curve A bears no proportionate relationship to curve B.*

TUBERCULAR DISEASE FROM THE PHYSIOLOGICAL STANDPOINT.

Of all the circumstances which lead to the development of the tendency to phthisis pulmonalis, age is unquestionably one of the most important. If we take a table of the general and one of the phthisis mortality, we get this remarkably exemplified. A glance at the curves (Fig I.) shows that while the former, the general mortality, is excessive at birth, rapidly diminishes till about the fifth year, more gradually till about the fifteenth, and then first slowly and then rapidly rises to the end; the latter, the phthisis mortality, shows during the early years of life a comparatively low rate, a rapid rise from about fifteen to twenty and twenty-five, and then a somewhat gradual decline.

This latter curve[1] represents the mortality from phthisis relative to the population living at different ages, and has been constructed from the Scottish Registrar General's Returns for the ten years 1876 to 1885 inclusive, these years being selected in order that the yearly average results might be compared with the population returns at different ages, as given in the Census for 1881. Whilst it does not altogether

[1] In the curve the phthisis mortality during the first five years of life is not represented, as I believe that errors of diagnosis are then specially common. In the table (I.) it is seen to be higher than during the next five years.

correspond with the results obtained by Hutchinson[1] from the English reports, which indicate the greatest liability to death from phthisis as occurring between the fortieth and fiftieth years; yet, like to these, this curve demonstrates that in the later years of life consumption is a much commoner cause of death than might otherwise have been supposed.

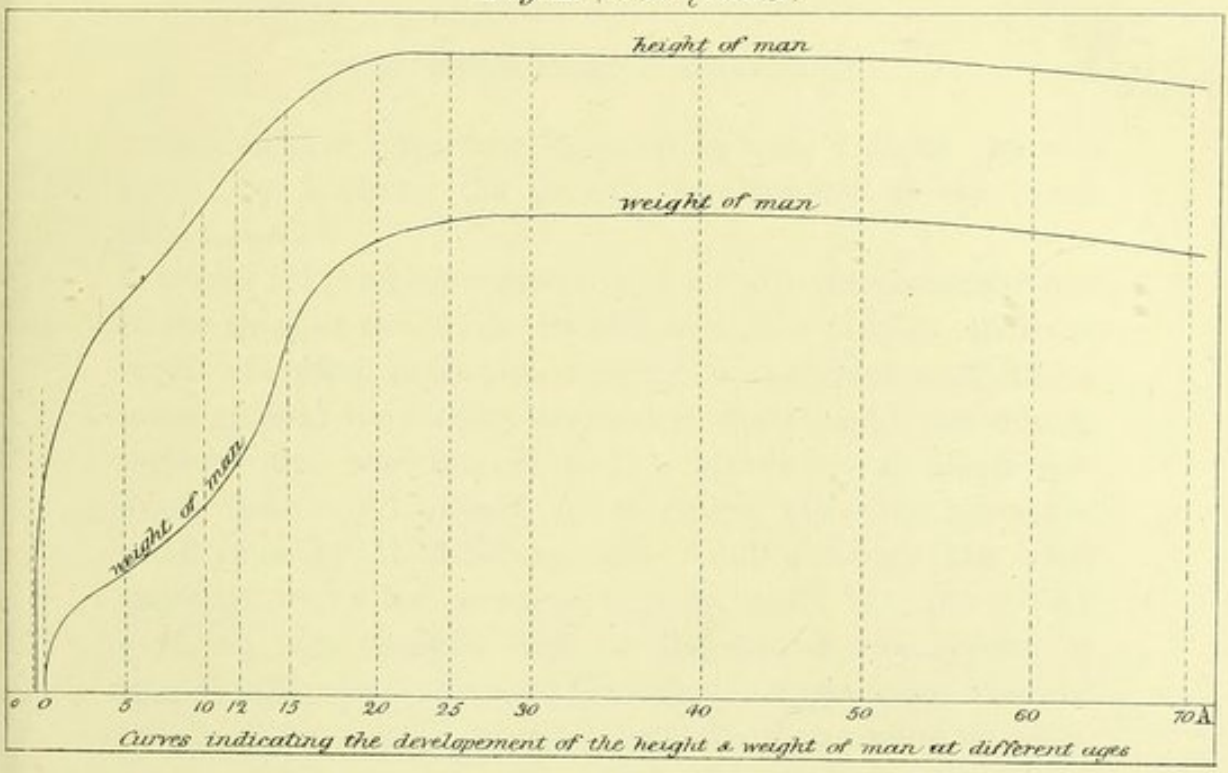
Whilst the first curve (*a*) illustrates what Addison has described in his "Vision of Mirza," viz., that the bridge over the tide of eternity has the concealed trap-doors laid thickly at the entrance, less so towards the middle, and close together towards the extremity; the other curve (*b*) demonstrates that with doors representing risks of death from phthisis pulmonalis alone, a very different distribution would be required to represent what occurs.

And now let us consider fully what the prevalence of phthisis at these ages means. It has been said, and the curve bears this out, that it is a disease which tends to occur at the most vigorous periods of life. What do we mean by the most vigorous period, or in what respects does the period between twenty and fifty differ from those above and below?

If we examine a curve (II.) showing the increase in height and weight of the body, we find that although in it there is a rapid rise about the periods of puberty and adolescence (best marked in weight), there is yet a marked difference between it and the curve of phthisis, in that while the former attains a maximum about the twentieth or twenty-first year, and continues as a straight line, the latter goes on rapidly increasing till about the twenty-fifth or thirtieth year, and then declines. This is important, in that it shows that phthisis is a disease which tends to affect the organism not so much when it is in the act of growing, as after growth has been completed.

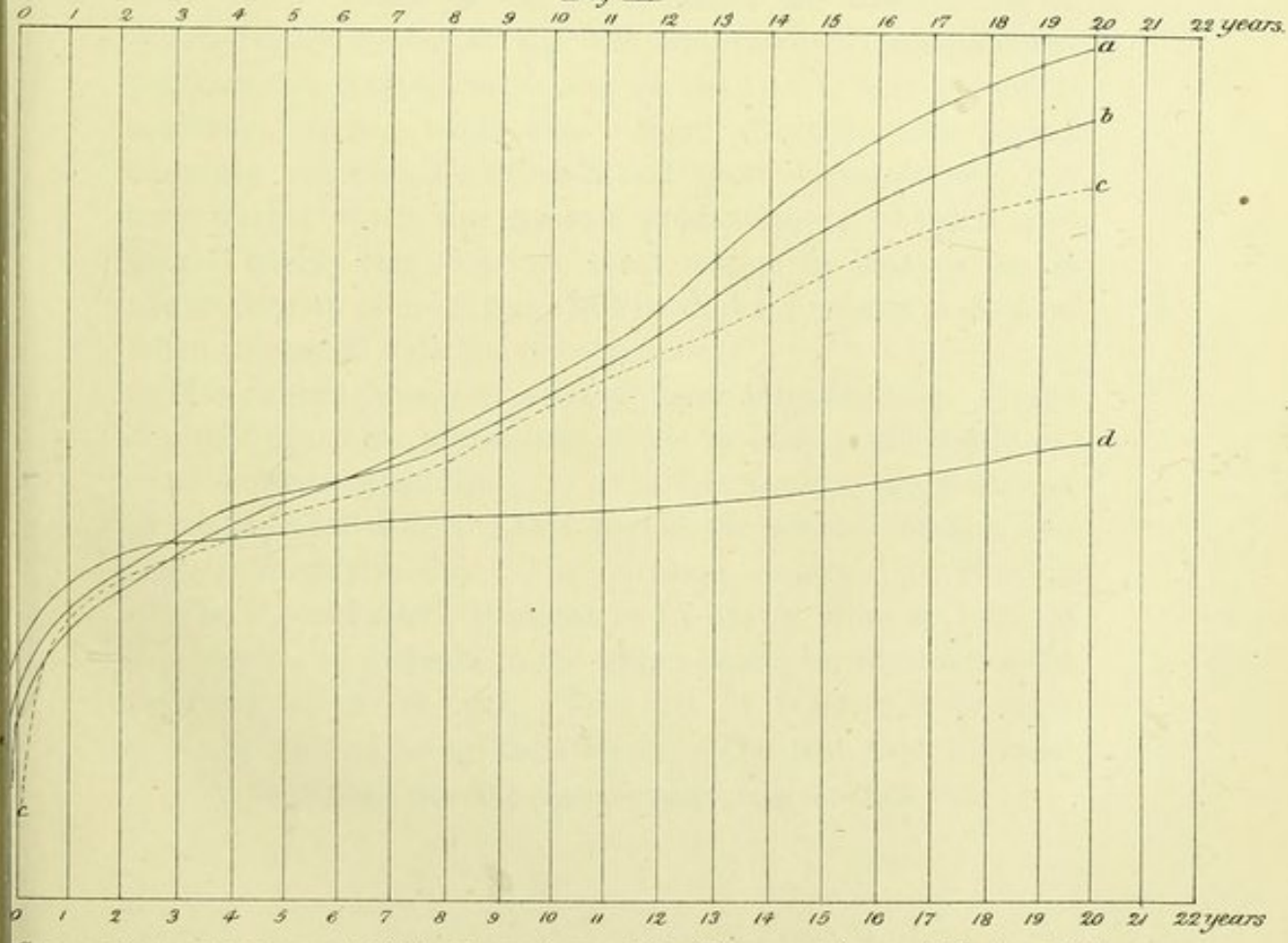
[1] *The Spirometer-Stethoscope and Scale Balance*, etc., pp. 43 and 46; and *The English Registrar General's Fifth Annual Report*. Compare also Sieveking's *Medical Adviser in Life Assurance*, 2nd edit., p. 93.

Fig II. (after Quetelet)



Curves indicating the developement of the height & weight of man at different ages

Fig III



Curves showing measurement of the circumference of the Thorax - Abdomen & Head at different ages

a. Thorax over the nipples. *c. Abdomen.*
b. do. at the 6th rib. *d. Head.*

But further important information can, I think, be obtained by studying the growth development of the lungs themselves.

Curve III.,[1] which represents at *a* and *b* the circumference of the chest at the sixth rib and over the nipples, shows a rapid rise after birth and during the early years of life, a more gradual one during boyhood a more rapid rise during puberty and adolescence, and a maximum at adult age. With this would closely correspond a curve to show the vital capacity at different ages; and although the vital capacity seems to increase slightly until the thirty-fifth year, we may consider that at the age of twenty-one, or thereabouts, the body and respiratory organs have attained their maximum bulk, and the latter their maximum development. That is to say, that whilst in the respiratory mechanism up to the age of twenty-one, or thereabouts, the income has been in excess of the expenditure, the assimilative or vegetative in excess of the disassimilative or animal functions, at this period a balancing of the two is struck, which continues during adult life. Now, although this period certainly represents the so-called most vigorous age, the time during which the greatest production of physical and mental energy can occur, we must remember that, as far as the storing up of energy is concerned, it represents a decline when compared with previous years.

This subject, however, requires closer consideration. Every one will, of course, admit that whilst in early years bodily income exceeds expenditure, in adult life the two are balanced in health, so that during this period the weight remains the same from day to day. The question, however, is, How is this balance struck? It might be by one or other or both of two ways,—by increase in the expenditure, by diminution of the income, or by both. The first of these will perhaps occur to some as being most likely. The fact that the real

[1] Constructed from the measurements given in Table VII.

work of life, be it physical or mental, is only begun after growth is complete, may lead us to suppose that in adult life the income remains the same, but that that energy which formerly went to increase the bulk and weight of the body is now expended in physical or mental work. But a closer consideration of the subject shows us that the increased capability for work during adult life is rather a question of development, is the result of our being then better able to direct energy. Although the total amount of carbonic acid and urea excreted daily is greater in adult life than in childhood, the amount relative to body weight is very much less, and shows that the molecular and chemical changes upon which life depends take place during adult years with greatly diminished activity.[1] But now, can the balancing be better explained by diminution of income? Scientific observation bears out what all have daily observed, that as animals advance from early to adult life, the amount of food required undergoes a great diminution relative to body weight,[2] and that this should be the case is evident from what we have noted as regards the greater relative expenditure of early years. In adult life, therefore, there is relatively less assimilative power and relatively less expenditure as compared to childhood, although *absolutely* both are increased. As to how the balance is struck, however, we cannot speak definitely, owing to want of sufficient data, but still some important conclusions can be drawn. Seeing that in both income and expenditure there is a gradual diminution relative to body weight as the animal passes from early to adult life, and remembering that in early life the relatively great expenditure is not only completely replaced by the income, but is actually exceeded in order to admit of growth, we must admit that in the striking of the balance of adult life the diminution of the

[1] Vierordt, "Physiologie des Kindesalters," in *Gerhardt's Handbuch der Kinderkrankheiten*, pp. 353–370 and 379.

[2] Vierordt, *ibid.*, pp. 403 and 413.

income is the more important item. Although the absolute amount of income and expenditure is greater in adult life than in childhood, we must remember that this relative diminution of income in adult life means a lessened formative power; it is betokened by the diminishing ratio of heart weight to body weight which occurs as the child grows older,[1] and as an illustration of its importance probably no better could be found than the rapid renewal of parts in certain young animals. As is well known, such animals as newts, etc., have the power of reforming limbs, tails, etc., after amputation when young, a power which rapidly diminishes as age advances. Of course, between early life on the one hand and adult life on the other there is no hard and fast line; but whilst we can recognise in the transition an increase in the absolute and diminution in the relative amounts of both income and expenditure, we must recognise also a greater relative diminution in the income, a greater relative diminution in the vegetative functions of the body.

But these considerations are important in connexion not only with the work of the body, but with the heat, and to this I would now direct attention. Heat, as we know, is constantly being produced, and we know also, as constantly being lost, mainly in man by radiation, conduction, and evaporation from the skin. A matter of interest, therefore, in connexion with our subject is to know the extent of surface of skin at different ages, inasmuch as from this we may be able to deduce some information as to the relative loss of energy to the body in the form of heat. Tables II. and III. show that the extent of skin surface to body weight is greatest at birth, and gradually diminishes as growth goes on, becoming least at and during adult life. Thus, then, although we must not lose sight of the fact that the loss of heat from the surface of the body can be modified by clothing, etc., we may say that as age advances less and less energy in the form of heat in proportion to body weight will be lost to the body

[1] Landois and Stirling's *Physiology*, 2nd edition, p. 80.

from the skin. Taken into consideration with the fact that four-fifths of the total energy of the body is expended as heat, and of this the great proportion in man by the skin, this gradual diminution in extent of skin surface in proportion to weight is specially interesting, as it shows that as years advance there occurs less and less requirement for heat production, *i.e.*, for expenditure. With this it is also specially interesting to notice that the apparatus for bringing about expenditure, *i.e.*, the lungs, become proportionally more extensive as age advances. As Vierordt has stated, the respiratory capacity in growing years increases more rapidly than the weight; and although that observer notes that the proportion of the respiratory capacity to body weight may not differ much at different ages,[1] the proportion of the respiratory capacity to extent of skin surface seems to become greater as age and size increase.[2] To this point we shall advert later on.[3] Meanwhile we must observe that the fact that with the much greater relative loss of heat which the large extent of surface in the young animal will entail, there is associated growth or increase in bulk, and the manifestations of physical or mental energy, great in proportion to the bulk of muscle or brain, is to be looked upon as further evidence that the period of adult age is, as compared with that which precedes it, one of decline in the vital assimilative power. Bearing further on this point, and of great practical importance in connexion with pulmonary phthisis, there are

[1] See also Table III., column 10.

[2] See Table III., column 9.

[3] This fact that the respiratory capacity bears an inverse proportion to the extent of skin surface may, of course, mean that whilst skin and lungs are together concerned in giving off heat, the proportionate amounts given off by such differs at different ages and in individuals of different heights. It is important, however, to observe that oxygen does not appear to be so necessary for assimilation. This can be concluded from the fact that new-born animals have a great power of resisting asphyxia, it being in intrauterine life that assimilation is most active.

certain further considerations which I propose now to consider in detail. These are that at the onset of adult life, *i.e.*, of the so-called most vigorous period of life, certain important physiological and pathological phenomena are to be observed. Those which I propose to discuss are the development of the reproductive functions and the disappearance in scrofulous subjects of enlarged cervical lymphatic glands.

I. *Development of the Reproductive Functions.*—The connexion between innutrition and reproduction is well established, and the fact of the development of the reproductive functions about puberty, when, as we have seen, the excess of income over expenditure ceases, of course bears this out. In this connexion the great increase in the vital capacity about this period is interesting, as showing arrangements for promoting expenditure; and I would also here suggest that the enlargement of the respiratory passage, which leads in the male to the cracking of the voice, may have a similar explanation. Its association with the growth and development of the testicles is noteworthy; and I would here also point out that the fattening which occurs after removal of the reproductive organs in animals shows also the connexion between these functions and tissue oxidation.

But these points are of practical importance in connexion with phthisis. How menstruation, lactation, and pregnancy affect this disease I do not propose at present to discuss, but the relationship between reproduction and phthisis generally is interesting. Pollock (*Elements of Prognosis in Consumption*, p. 345), after discussing the injurious effects of marriages of consanguinity, says: "This quesion of large families is also of interest. Parents weakly from any inherited or personal disease are frequently very prolific, and become the propagators of constitutional and organic disorders. We have known as many as seventeen brothers and sisters die of consumption. As a matter of experience there is no doubt that the phthisical are prolific, and endless instances occur

of a mother conceiving when in an advanced stage of tubercular disease, and having a child which survives only a few months; nor is the generative power confined to the mother. Men in much advanced phthisis often marry and have children." Again, "Inter-marriages of individuals remote from each other in ancestry, in social condition, in constitution, and in what is called temperament, but above all the unions of persons of different race and country (if direct taint of disease be absent on both sides), are very generally free from resulting affections of constitutional origin in their children. It is perhaps true that marriages between individuals of different races are not very prolific; and if this be the case, it is another reason for the greater vigour of their offspring, for the members of large families are not generally so individually strong as those of small ones."

This question of large families in connexion with the tendency to disease is interesting, and I regret that I have at present no statistical information to offer in connexion with it. I give, however, the above quotation as an expression of opinion, and I think that it will be shared in by most observers. But, on the other hand, deficient health in individuals is often associated with sterility; and this requires notice, as it might appear to be an argument against what has been above stated. I think, however, it is not, because we have examples of the close connexion of the two, as opposed to healthy nutrition and reproduction; *e.g.*, Matthews Duncan pointed out that in women, as age advanced, barrenness and twinning were frequently associated.

II. *Enlarged External Lymphatic Glands.*—The connexion in scrofulous subjects of enlarged lymphatic glands with pulmonary phthisis is well known, and recognised to such an extent that it has been stated[1] that a scrofulous gland is tubercular, and the extirpation of such as a preventive to the absorption of tubercular matter, and consequent development

[1] Ruehle in Ziemssen's *Cyclopædia*, vol. v. p. 641.

of phthisis by auto-infection, has been proposed.[1] But all have observed the infrequency of enlarged cervical glands and pulmonary phthisis in the same subject, and a little consideration will, I think, explain this.[2] It shows that enlarged glands are associated with an earlier period of life than the phthisical—that, in fact, they are associated with the period when daily income is in excess of expenditure. As soon, however, as the income and expenditure of the organism are balanced, as they are when growth ceases and the respiratory functions reach a maximum, these glands diminish or disappear. This seems to be simply the result of the fact that tissue absorption then becomes more active, and the disappearance of these glands as the result of counter-irritation; or, as is frequently noticed on the supervention of a fever, an acute pneumonia or even of phthisis itself can be ascribed to this cause, and demonstrates the association of the period of adult life with an increased expenditure relative to income of bodily energy.[3] Here, again, I think some further suggestions may be made,—*e.g.*, looking upon those enlarged glands as tubercular foci, the supervention of tubercular at an earlier age than ordinary phthisis, may have an explanation in the fact that coincident with the disappearance of these glands there will be an increased risk of auto-infection with tubercular matter. Again, the risk of phthisis developing about that age in a scrofulous individual, as the result of his or her being somewhat out of health and feverish, can be readily understood by remembering that then resorption is very likely to occur.

Having seen that phthisis begins to become prevalent about puberty, and is most prevalent about the middle period of life, we have been considering in what respects

[1] Ruehle in Ziemssen's *Cyclopædia*, vol. v. pp. 605 and 642.

[2] Pollock, *Elements of Prognosis in Consumption*, p. 90.

[3] The diminution of lymphatic and blood glands with age is also referred to in Charcot's *Senile Diseases*, p. 28.

these years differ from the previous. A glance at the curve shows, however, that as age advances the tendency to phthisis gradually diminishes. Can we now get any information as to how this occurs? In the first place, we must remember that as age advances the lymphatic and blood glands gradually diminish in size, and that the liability to tubercle is lessened.[1] But there are other reasons. Seeing that in early years and in old age there is a comparative immunity from phthisis, can we recognise any similarity between these two as distinguishing them from the period of maturity? The first supposition which is likely to occur is probably a contradictory one to this, viz., that if maturity differs from early life, old age must differ still more. Let us, however, examine the matter more closely. We have seen that the period of maturity differs from that of the preceding years in the relative diminution of income, and as aided, perhaps, by the increase in the respiratory capacity by an (relative to income) increased expenditure. Let us now apply these considerations to the period of old age.

As age advances the respiratory capacity diminishes; as has been shown, it diminishes after thirty-five at the rate of about $1\frac{1}{2}$ cubic cm. per year. The cause of this we may suppose to be the gradual increase in the size of the pulmonary air cells, the occurrence, in fact, of an emphysema, which, although the lung is voluminous, renders less the quantity of air capable of being displaced.[2] This indicates a diminished expenditure of energy, and is associated with a diminution in the capability for physical and mental work. The extent of surface of the body, however, remaining the same, there is practically no difference in the amount of heat lost; owing, however, to the lessened production of heat by movement,

[1] Charcot, *Senile Diseases*, p. 28.

[2] We must remember that the size of the air cells will greatly influence respiratory activity. The small air cells of the child will allow of a large surface of blood and air being brought into contact.

etc., artificial means are used to maintain it. In this way, as age advances, there is in effect a diminution of expenditure, and to such an extent does this frequently occur, that a balance of a kind to the good takes place, indicated by the reappearance of fat.[1] With this lessened waste is further associated a diminution in the reproductive power[2] as indicated by the diminishing fecundity of marriages as age advances.

Further, we must remember that the lessened respiratory capacity in old age, to which we have referred, is frequently very considerable, a certain amount of emphysema being almost normal after fifty. Hence, although the expenditure is diminished, the lungs themselves must, in many cases, function actively, a condition analogous to that found in childhood, when, with a comparatively small vital capacity, there is proportionately great expenditure. In this way, then, old age has similarities to the early periods of life, but, of course, there are many important distinctions,—the main one being that, whilst in early life the balance to the good is the result of excess of income, in old age the reverse is the case, any balance to the good is the result of diminished expenditure. We must remember, also, that in old age assimilative power is also becoming impaired, and that the amount of this as compared with the expenditure varies greatly. In most individuals it is as we have stated, for a time at any rate, relatively in excess of waste, leading to fat accumulation. But such is not always the case. We find cases, on the one hand, in which the assimilative power relative to the expenditure diminishes rapidly as age advances, leading to feebleness, degeneration, and premature dissolution, whilst, on the other, the two processes, assimilation and disassimilation, may diminish *pari passu*, and very gradually, leading to an active, and, as regards nutrition and reproduction,

[1] Moderate accumulation of fat is normal in people over fifty. Traube quoted by Ebstein, on *Corpulence*, p. 5.

[2] Charcot, *Senile Diseases*, p. 28.

energetic old age. Of course these three varieties of the appearances presented by the decline of life are not always so clearly distinct as perhaps our description would lead us to expect, but that they indicate realities, every one will, I think, allow. Now it will be noticed that the first variety mostly resembles the period of youth, the third less so, and the second least of all.[1] The application of such considerations to phthisis is obvious,—in the first there is almost complete immunity from this disease, a state of affairs which cannot be said of the third, and still less of the second.

A general view, then, of the stages of youth, maturity, and decline in connexion with pulmonary phthisis leads us to connect that disease with the second of these, with the period when the income is just sufficient to meet the daily expenditure, *i.e.*, has (relative to the expenditure) sustained a diminution. This statement requires qualification. It may be argued, if pulmonary phthisis is apt to supervene at this period it should be apt to occur in any case where the quantity of food is insufficient. But does it? I think not. As has been remarked, "Good food and bad air are more hurtful than bad food and good air," and in cases of long standing inanition, as from œsophageal stenosis or in famine districts, pulmonary phthisis is rare.[2] How can we explain this? Simply by stating that what we now mean by assimilation is the vital power of transforming the nutritive material derived from the food into tissue, a power which the individual is born with, which, with greater or less variations, gradually diminishes as age goes on, and which is not necessarily interfered with in cases of inanition such as we have referred to. Pulmonary phthisis is apt to supervene when this power is diminished in vigour; at the periods when the reverse is the case there is comparative immunity.

[1] Compare Paget, *Surgical Pathology*, p. 82.

[2] This of course has been denied, but compare Ziemssen's *Cyclopædia*, vol. v. p. 488.

But, now, if phthisis is common at this period, we should expect to find, then, a prevalence of other diseases. Into the connexion of phthisis with such I shall not at present enter, but there is an affection connected with it which we may now appropriately discuss, viz., tubercle.

Tubercle occurs in man, affecting different organs and tissues—the brain and intestines as well as the lung—and it is apt to affect those at different ages,—in the case of the brain during infancy, and intestines during boyhood or girlhood. How does the view that pulmonary tubercle is apt to develop when assimilation has, relative to disassimilation, diminished, tally with the fact that it attacks those other localities in earlier years when increase in weight and bulk is rapidly taking place? Premising that pulmonary phthisis may not mean tubercle, we shall nevertheless find, I think, that the occurrence of tubercle during the early years of life is after all not conditioned differently from its occurrence in the lung.

Let us first take brain tubercle, the so-called tubercle of infancy and childhood. Examination of the Table IV., representing Rilliet and Barthez's 98 cases, shows it to be relatively uncommon in early infancy, but rising rapidly, to reach a maximum between the fourth and fifth year, and then gradually to decline. Undoubtedly, then, it may be said brain tubercle occurs at periods of energetic growth, but this does not invalidate our conclusion, for we have now to do with the brain growth, and the period of maximum growth of the brain is very different from that of other parts of the body.

Curve *d*, Fig. III. has been constructed from measurements of the heads of 500 individuals (males) between birth and the twentieth year, and it shows that the greatest growth of the brain takes place in the first months of life, and that after the second or third year the growth is comparatively very slow. Brain tubercle, therefore, cannot any more than pulmonary be associated with a period of life when assimilation is in excess.

Next, as regards mesenteric tubercle, the so-called tubercle

of boyhood. Table V., taken from Rilliet and Barthez's 215 cases, shows that it reaches its maximum between the sixth and tenth years, and these observers have stated that it is rare before the fourth year. By some observers it is said to occur earlier, and Widerhofer in Gerhardt's work makes its maximum between the fourth and fifth year. That abdominal tubercle should occur early and be somewhat widely scattered over the early years of life, we can understand for many reasons, more especially, perhaps, because then the risk of irritation from errors of diet is at its maximum, but there is, I think, no doubt that true abdominal phthisis is to be associated with a period of life later than that at which brain tubercle is apt to occur. All of us have had many cases of it during puberty and adolescence, which cannot be said of tubercular meningitis.

To ascertain at what period of life the digestive organs present their most active growth, various methods might be made use of, *e.g.*, we could get information by measuring the length, etc., of the stomach and intestinal canal at different ages from birth onwards. Or the proportionate size of the abdominal cavity at different ages can be made to afford data, and of this method the following from Liharzik, quoted by Vierordt in Gerhardt's work,[1] is an example—

	Birth.	21st Month.	7⅛ Yr.	Adult.
Belly, . .	100	160	240	260

This shows growth comparatively slow after the seventh year.

In order to obtain further information on this point, let us look again at Fig. III., curves *a*, *b*, and *c*, which show measurements of the circumference of the body at the sixth rib, over the nipples, and at the umbilicus. These measurements were made in order to ascertain the relationship between the digestive and respiratory functions at different ages. The curve shows that at birth the circumference at the sixth rib

[1] *Handbuch der Kinderkrankheiten*, vol. i. p. 272.

is greater than at the nipples, that about the fourth year they are equal, that afterwards the upper circumference of the chest proportionately increases, and that the circumference at the umbilicus, apparently greater than that at the nipples about the first or second year, does not increase to anything like the same extent at puberty and adolescence. All this seems to show that after the fourth or fifth year the growth of the digestive organs is exceeded in activity by that of the respiratory. The occurrence, therefore, of abdominal tubercles is, I believe, in accordance with the rule, that tubercle tends to develop in a tissue after the period of maximum growth, *i.e.*, when assimilative power begins to fail.

I am indebted further to Drs M'Bride and Caird for pointing out to me that a similar law may be applied as regards tubercle of the larynx and testes. In both these organs it occurs most frequently some time after puberty (about the twenty-second year), *i.e.*, after the period of maximum growth.

But closely connected with our subject, and of great practical importance in connexion with phthisis, is the *height* of the body. Remembering that, as Hutchinson has pointed out, the vital capacity increases in a direct ratio with the height, and that, as we have seen, phthisis prevails when the vital capacity is at its greatest, the influence of stature becomes peculiarly interesting. In 1586 cases (adult males) admitted into the Royal Infirmary, of which 237 were phthisical, I have obtained records of the height, and Table VI. shows the result. Of the phthisical cases, the greatest number (16·8 per cent.) were in height between 5 ft. 8 in. and 5 ft. 9 in., and 6·3 per cent. of these were over 6 ft. in height. Of the cases other than phthisis, the greatest number (18·5 per cent.) were between 5 ft. 6 in. and 5 ft. 7 in., and 4·2 per cent. were above 6 ft. I regret that the number of cases out of which this table has been made is so small, but I think that although phthisis is found at all statures, every one doing clinical work must have observed its prevalence among individuals above the

average height. I have been informed that many of the publicly exhibited giants fall victims to phthisis, and that in regiments of guards the proportion of phthisis is somewhat above that of ordinary regiments. But why should this be? Several suggestions might be put forward in explanation; the only one, however, which I shall mention now is that which can be obtained by considering together the vital capacity and the surface extent of the body. Remembering that for nutrition function of the part is necessary, one may suppose that as in a tall individual the extent of surface of body in proportion to weight is less than in a short one, there will be less proportionate loss of heat, and hence the respiratory activity need not be so active. The lungs, therefore, will not have such a demand made upon them, and will function less actively. But further, as we have seen in tall individuals, the lungs are specially large, the vital capacity being great, hence a second risk of deficient functional activity will occur.[1] The fact has again and again been observed, that although the weakly phthisical build is characteristic, we frequently find phthisis developing in individuals tall and of extra good physique. The explanation of this, I think, is obvious. Such individuals are fitted for an avocation requiring plenty of physical work in the open air; give them this and they remain healthy. Give them, on the other hand, indoor town life and sedentary work, they readily succumb to phthisis; and further, viewed in this light, we can regard with less regret the diminution in the stature of the inhabitants of the town and manufacturing districts of the British Isles, so much bewailed in military and other circles, for we cannot but acknowledge that it may, to some extent at least, mark a salutary process. We must look upon this, I think, simply as one of the many sacrifices which nature requires in her efforts to transform the struggle for existence from the physical to the intellectual.

[1] See Table III., columns 8 and 9.

But from the occurrence of phthisis preferably in tall individuals another deduction can, I think, be drawn, and that is an explanation of the view put forward by Laennec and many others, that phthisis does not, to a great extent, supervene as the result of "catching cold." While acknowledging the pernicious effect which such a theory would have if accepted and acted upon, I would submit that for this, as for all such extreme views, there is a certain foundation in fact. The evil effects of a chill on the body must, *ceteris paribus,* be most marked in those cases in which the surface extent bears the greatest proportion to the body weight, *i.e.*, in short individuals and in children, less in tall adults. The fact, however, that it is rather in the latter that phthisis is met with may be looked upon as denoting that it is something else than simple loss of heat which is the main determining cause of this disease.

From the point of view of nutrition and growth it may therefore be concluded—1. That tubercle tends to occur when the assimilative power fails, as indicated by its occurrence in the lungs, intestine, and brain at different ages, and that the development of the reproductive function, and the disappearance of enlarged cervical lymphatic glands indicate a lessened activity in the nutritive processes in adult life as compared with early years.

2. On the general principle of the connexion between supply and demand, we may suppose that this assimilative power is to a greater or less extent dependent on functional activity of the part. This seems borne out by the fact, that in tall people with large lungs and with proportionately less demand for metabolic activity (*i.e.*, less loss of heat) phthisis is specially common,[1] and also by the fact that as age advances the natural tendency to emphysema, by increas-

[1] Small animals have a more lively metabolism than old ones, they consume more proteid, and have generally less bodily fat. Landois and Stirling, p. 511, 2nd edition.

ing the functional activity of the lungs, seems to render them less liable to phthisis.

3. This assimilative power, though in part dependent on functional activity, is *innate* as regards the individual. Of this we have evidence in the different sizes to which individuals grow, the functional activity being the same, and in the varying proneness to phthisis in individuals, the surroundings being the same.

TABLE I.—*Mortality from All Causes and from Phthisis proportionate to Population living at Different Ages.*

Ages.	All Causes (per cent.)		Phthisis (per cent.)	
	Males.	Females.	Males.	Females.
Under 1	13·7	12·0	·11	·12
„ 2	6·1	5·9	·14	·14
„ 3	2·6	2·6	·08	·08
„ 4	1·7	1·7	·06	·05
„ 5	1·2	1·2	·05	·06
„ 10	·73	·72	·06	·07
„ 15	·45	·47	·08	·14
„ 20	·65	·66	·25	·34
„ 25	·80	·76	·36	·38
„ 30	·85	·88	·36	·40
„ 35	·95	·97	·34	·38
„ 40	1·1	1·0	·31	·33
„ 45	1·3	1·0	·27	·27
„ 50	1·7	1·3	·27	·22
„ 55	2·1	1·7	·24	·17
„ 60	2·9	2·3	·23	·16
„ 65	3·9	3·0	·19	·12
„ 70	5·6	4·5	·19	·10
„ 75	7·8	6·4	·12	·07
„ 80	11·9	10·1	·06	·06
„ 85	16·5	14·9	·04	·02
„ 90	28·2	24·5	·003	·02
„ 95	39·9	33·1	·002	·002
„ 100	44·0	43·4		
„ 105				

TABLE II.—*Proportion of Skin Surface to Body Weight at different Ages,* □ *cm. to* 1 *kilog.*

6	days,	=829.
6½	months,	=624.
6	years 8½ months,	=458.
14	years,	=354.
25	years,	=301.

—Meeh, quoted in Vierordt, *ibid.*, p. 288.

TABLE III.—*Proportion of Skin Surface to Body Weight and Vital Capacity, and of Body Weight to Vital Capacity, in six Individuals of different Ages and Statures.*

		Age.	Height.	Weight.	Skin Surface.	Vital Capacity.	Ratio—Weight to Skin Surface.	Ratio—Vital Capacity to Skin Surface.	Ratio—Weight to Vital Capacity.
		yrs.	ft. in.	st. lbs.	sq. in.	c. in.	lb. to sq. in.	c. in. to sq. in.	lb. to c. in.
1	Boy	7	3·6	3·8	1353	77·6	1 : 27·06	1 : 17·4	1 : 1·54
2	do.	11	4·3	5·4	1728	91·8	1 : 23·35	1 : 18·8[1]	1 : 1·22
3	do.	15	5·0½	7·10	2398	148·4	1 : 22·2	1 : 16·1	1 : 1·37
4	Man	28	5·5⅓	9·12	2768	216·0	1 : 20·07	1 : 12·8	1 : 1·59
5	do.	28	5·9¾	11·4	3024	247·0	1 : 19·1	1 : 12·2	1 : 1·56
6	do.	24	6·2½	15·5	3556	327·0	1 : 16·4	1 : 10·8	1 : 1·52

[1] This exception is probably due to the fact of the vital capacity being proportionately lower than in the cases of the other two boys.

TABLE IV.—*Cases of Tubercular Meningitis.* Rilliet and Barthez, vol. iii. p. 511.

	Paris.	Geneva.	Totals.
5 months,	0	1	1
9 „	0	1	1
1 yr. to 2½ yrs.,	4	13	17
3 „ 5½ „	11	23	34
6 „ 7½ „	7	16	23
8 „ 10 „	10	5	15
11 „ 15 „	4	3	7
			98

TABLE V.—*Case of Abdominal Tubercle.* Rilliet and Barthez, vol. iii. p. 841.

	Age.				
	1 yr. to 1½ yrs.	3 yrs. to 5½ yrs.	6 yrs. to 10½ yrs.	11 yrs. to 15½ yrs.	Totals.
Stomach, . .	4	11	5	1	21
Small Intestine,	25	34	49	26	134
Large Intestine,	9	17	21	13	60
Totals, . .	38	62	75	40	215

TABLE VI.—*Stature of Patients admitted into the Royal Infirmary* (1586 *male adults*).

	Phthisis (237 cases).	All other Diseases (1349 cases).
Ft. in.	Per cent.	Per cent.
5 2 and under,	2·1	3·0
5 3	1·6	2·3
5 4	5·9	6·5
5 5	8·8	10·9
5 6	11·8	18·5
5 7	13·9	17·0
5 8	16·8	13·1
5 9	14·7	10·2
5 10	12·6	8·4
5 11	5·0	4·0
6 0 and over,	6·3	4·2

For data for these results I have to thank Professors Grainger Stewart and Fraser, and Drs Muirhead, Brakenridge, and Wyllie.

TABLE VII.—*Measurements of the Circumference of the Thorax, Abdomen, and Head at different Ages* (500 *cases, males*).

	Thorax.		Abdominal Umbilicus.	Head.
	Over Nipples.	Over 6th Rib.		
	in.	in.	in.	in.
Birth	12·7	12·9	12·0	13·6
6 mos.	16·0	16·7	16·0	17·5
1 yr.	17·7	18·3	17·0	18·4
2 „	18·5	18·7	18·4	19·2
3 „	19·8	20·1	19·2	19·7
4 „	20·3	20·7	19·4	19·6
5 „	21·0	21·0	20·6	19·7
6 „	21·4	21·5	20·7	19·8
7 „	21·9	21·8	21·0	20·0
8 „	22·6	22·0	22·0	20·0
9 „	23·5	22·9	22·7	20·1
10 „	24·2	23·4	23·7	20·2
11 „	25·3	24·2	24·1	20·5
12 „	26·0	25·0	24·5	20·6
13 „	27·6	26·2	25·4	20·6
14 „	28·6	27·0	25·6	20·8
15 „	29·9	28·1	26·9	20·9
16 „	30·8	29·0	27·5	21·1
17 „	31·6	29·7	28·0	21·5
18 „	31·4	30·5	28·5	21·7
19 „	33·0	31·0	29·2	22·0
20 „	33·7	31·5	29·5	22·2

NUTRITION AND REPRODUCTION:

Considered Generally, and as Bearing on the Etiology and Treatment of Disease.

"In every part of the universe we observe means adjusted with the nicest artifice to the ends which they are intended to produce, and, in the mechanism of a plant or animal body, admire how everything is contrived for advancing the two great purposes of nature, the support of the individual and the propagation of the species." So said Adam Smith, and so says every one who has snatched some time from the performance of these functions and devoted it to the consideration of their nature.

In the following paper I propose to try if we cannot get some idea as to how these functions of nutrition and reproduction are brought about. I shall consider only general principles, but I trust that we may from these be able to draw some deductions of practical interest.

Let us, in the first place, define what we mean by nutrition. This, in the higher animal, comprehends the complicated processes of digestion, absorption, assimilation, excretion, etc., not to speak of the still more complicated processes concerned in the seeking and securing of food. In the lower animals and in plants the process is much more simple. In the amaiba, for example, it means the envelopment of a small piece of organic matter by the protoplasm mass of that

organism, and the gradual transformation, chemical and physical, of this minute piece of organic matter into the tissues of the amaiba. In the plant it means the taking in of certain chemical elements,—carbon, hydrogen, nitrogen, etc.,—and the building up of these into a tissue.

A question now meets us which requires some little attention. Are we to distinguish between the animal and the plant as regards the conditions which lead to their nutrition? Are we, remembering that whilst in the plant the processes are mainly synthetic, and in the animal analytic, to suppose that the conditions which lead to their nutrition are not identical? Certainly not. We know, in the first place, that where the animal kingdom ends and where the vegetable begins there is no hard and fast line; we know that certain organisms, the *convoluta*, at one stage function like an animal, and at another like a vegetable; and we know that the process of building up, or synthesis, is not limited to the vegetable kingdom. As Foster says,[1] "Long ago, in opposition to the views of Dumas and his school, who taught that all construction of organic material, that all actual manufacture of protoplasm, or even of its organic constituents, was confined to vegetables and unknown in animals, Liebig showed that the butter present in the milk of a cow was much greater than could be accounted for by the scanty fat present in the grass or other fodder she consumed. He also argued, as an argument in the same direction, that the wax produced by bees is out of all proportion to the fat contained in their food, consisting, as this does, chiefly of sugar."

Finally, we know that during flowering in plants oxidative processes take place and carbonic acid is given off. We may therefore conclude that in animals and in plants synthetic and analytic processes are taking place, and that on the performance of those processes life depends.

But how, may we suppose, are these synthetic and analytic

[1] Foster's *Physiology*, fourth edition, p. 427.

processes, how in plants or animals is the constant building up and breaking down of their tissues, which constitute life, brought about ?

To get a general idea of this, let us look at the explanation given by Spencer as to how we may suppose a plant builds up its tissues. Speaking of the effect specially of the yellow rays on plants,[1] he says, "The atoms of several ponderable matters exist in combination, those that are combined having strong affinities, but having also affinities less strong for some of the surrounding atoms that are otherwise combined. The atoms, thus united, and thus mixed among others with which they are capable of uniting, are exposed to the undulations of a medium that is relatively so rare as to seem imponderable. Those undulations are of numerous kinds ; they differ greatly in their lengths, or in the frequency with which they recur at any given point ; and under the influence of undulations of a certain frequency some of these atoms are transferred from atoms for which they have a stronger affinity to atoms for which they have a weaker affinity. That is to say, particular orders of waves of a relatively imponderable matter remove particular atoms of ponderable matter from their attachments and carry them within reach of other attachments."

Probably this will be better understood from an example. The starch, sugar, albumen, etc., of plants is chemically composed of carbon, hydrogen, nitrogen, and oxygen, etc., and is formed out of CO_2, H_2O, NH_3, etc. Now, the unions of carbon with oxygen to form carbonic acid, of hydrogen with oxygen to form water, of nitrogen with hydrogen to form ammonia, are stable ones ; but we have to suppose that by the light undulations the molecules of carbon and oxygen in carbonic acid, of hydrogen and oxygen in water, and of nitrogen and hydrogen in ammonia, are made to vibrate at different rates, so that there occurs a separating of the carbon and oxygen of the carbonic acid, of the hydrogen and oxygen of the

[1] *Principles of Biology*, vol. i. p. 29.

water, and of the nitrogen and hydrogen of the ammonia, and that the dissociated molecules of carbon, hydrogen, nitrogen, are made to unite to form an organic substance, a substance by the breaking down of which into CO_2, H_2O, or NH_3, energy can be obtained.

So much by way of theory as to how the building up of tissue is brought about. We have now to consider the conditions which lead to a breaking down. We may here, I think suppose that, seeing that the molecules of carbon, hydrogen, nitrogen, etc., are in a more stable condition, when combined to form CO_2, H_2O, NH_3, than when associated to form starch, albumen, etc., there will always be a tendency for them to assume their more stable condition. We get an example of this in the fact that plants give off CO_2 in the dark,—that is to say, when the force which causes a building up, the light, is withdrawn, the plant tends to break down. But it is most important to remember that a breaking down will occur if the same solar radiations are allowed to impinge on a plant, but in excess. For example, if we allow these solar radiations to fall on the leaf of a living plant well provided with water, etc., we promote a building up of tissue, or deoxidation; but if we concentrate on a leaf these same rays by means of a lens, we cause a breaking down, or oxidation. Why this should be we need not consider, but it is important for the purpose of our paper to remember that such is the case. So much for the occurrence of synthetic and analytic processes in plants; let us now say a little about them in the case of animals.

To do this we must first of all form some ideas as regards the action of the nervous system. On this subject Mr Herbert Spencer says, "The simplest nerve-centre puts in relation not afferent and efferent fibres alone, but, through other fibres, commissural and centripetal connexions are made between it and other nerve-centres of the same grade and of a higher grade. Further, when such a nerve-centre is

excited through an afferent nerve, the disengaged molecular motion does not escape wholly along one or more efferent nerves, but that part of it propagated to higher centres there sets up supplementary changes. The diffusion does not stop here ; remoter parts are reached, and thus the disturbance of a single nerve fibre, if at all considerable, reverberates throughout the entire nervous system and affects all the functions controlled by it. Digging a pin into the foot may cause a convulsive contraction, not of the leg muscles only, but of many other muscles throughout the body. At the same time, it may alter the rate of pulsation and send waves of constriction along the arteries. The excreting structures of the skin may be so affected that a burst of perspiration results, and the actions going on throughout the alimentary canal may be changed. Such reverberations, which become conspicuous when the disturbances are decided, take place also when they are slight. A more vivid light, causing, as it does, stronger pulses of change through the optic nerve, increases the rate of respiration, and doubtless the other vital functions are simultaneously exalted. So that each nervous impression, beyond a direct response in the shape of increased action from one or more organs, calls forth an indirect response in the shape of increased action of the organism as a whole.

"Remembering that every instant the disturbance thus echoing throughout all passages of the nervous system is not solitary, but that there are many such disturbances, here arising from pressure, there from touch, in this place produced by sound and in that by light, at one part by muscular strain and at another by heat and cold, it will be manifest that, besides the few distinct waves of nervous change working their distinct effects, there are multitudinous indistinct waves, secondary and tertiary, travelling in all directions, working their indistinct effects.

"Since such reflected and re-reflected disturbances everywhere act as stimuli, we must regard the entire nervous

system as at all times discharging itself. Rightly to conceive nervous action, then, we must think of the conspicuous emissions of force from parts of the nervous system that are strongly disturbed as standing out from a vague background of inconspicuous emissions from the whole nervous system, which is slightly disturbed.

"To this general nervous disturbance, with its consequent general discharge, is probably due a certain general action of the motor organs. No muscles are ever in a state of absolute rest. What we distinguish as muscular motion is produced by the greater contraction of some muscles than of others. The others, however, are all slightly contracted. This pervading activity of the muscles is called their tonic state. And while we regard particular contractions as the results of particular nervous discharges, we have good reason for concluding that this universal contraction is the result of the universal nervous discharge. Of course, it is not the muscles alone on which this continuous centrifugal gush is expended. Through the intermediation of nerves connecting the cerebro-spinal system with the sympathetic system, the viscera receive their share of it. Hence the overflow of nervous energy which, without special solicitations, diffuses itself throughout the motor structures, giving elasticity to the step, and producing the concave bend of the back, the opened out shoulders, the raised head, etc., has for its simultaneous results an accelerated circulation, an invigorated digestion, and an exaltation of the vital processes at large."

But, besides supposing that this continuous centrifugal gush of nerve force produces this tonic contraction of the muscles, and, by its effects on the viscera, etc., produces that general feeling of well-being and desire for exertion which we all, I trust, frequently experience, we may, I think, be permitted to endeavour to obtain an idea—necessarily an obscure one—as to "how" it does this.

We know that when a nerve to a muscle is divided, not

only is there a disappearance of this tonic contraction, and not only does it become impossible for the nerve-centres to cause it to contract, but it becomes degenerated, its nutrition fails. We know, also, that if there is an increase in the amount of nerve force sent to a muscle from the nerve-centres it contracts, but, as the result of this, its substance breaks down into CO_2, urea, etc. Hence may we not conclude that, since the function of a nerve is to convey nerve force, its functional influence and its trophic influence over the parts to which it is distributed are not so separable as we may have supposed ?

That is to say, may we not suppose that just as the action of the solar rays on a leaf is, when in moderate amount, to produce a building up or deoxidation, and when in excess, as when concentrated by a lens, to produce a breaking down or oxidation, so the action of this nerve force is, when radiating out from the centres to the tissues in moderate amount, to produce an assimilation or building up, and when in extraordinary amount a disassimilation or breaking down ? Similarly, just as the tissues of a leaf, in the absence of light, break down, so do the tissues of an animal in the absence of nerve force.

Of course there are many important distinctions, and many arguments might be brought forward against this comparison. We must remember that the influence, in the higher animals, of the nervous system on nutrition, the so-called trophic influence, is not well understood. Although all admit that certain nerve lesions produce arthropathies, muscular degenerations, skin affections, bed-sores, sloughing, etc., many arguments might be adduced to show that tissue nutrition is independent of nerve supply. Such, however, do not, I think, affect the general question as to the trophic function of the nervous system in higher animals, inasmuch as, with respect to acknowledged functions of the nervous system, they can be more than paralleled, and an experiment by Goltz, I think, shows this. In December 1873 he divided the spinal cord in

the dorsal region of a puppy.[1] "In the following May the animal was in excellent health, and there was not the slightest indication that any functional connexion between the dorsal and lumbar portions of the spinal cord had been re-established. At the end of that month 'heat' came on, attended by all the ordinary phenomena, psychical as well as physical. Impregnation was effected, and the animal became gravid. The pregnancy, like the heat, was marked by the usual signs,—the mammary glands enlarged, and the usual mental accompaniments of the condition were present. Finally, one living and two dead puppies were born, the first without, the latter two with assistance. The mother, however, died soon afterwards from puerperal peritonitis. The post-mortem examination showed that there had been no regeneration of the divided spinal cord; the two portions were separated by more than a centimetre. In this case, the connexion between the ovary on one hand, and the mammary gland, brain, etc., on the other, must, if a nervous one, have been furnished by the abdominal sympathetic. We may, however, suppose that the nexus was a chemical one,—that the condition of the ovary and uterus effected a change in the blood, which in turn excited the mammary gland to increased action and produced special changes in the brain."

Be this the explanation or not, we may, I think, conclude that cases which seem exceptional are no more arguments against the trophic influence of the nervous system than are the phenomena exhibited by this dog to be looked upon as arguments against the spinal cord functioning as a conductor of nerve impulses.

But now the question is, Whence is derived this nerve force, which in ordinary amount, carried to a muscle or gland, has a trophic and constructive influence, and in increased amount has a functional and destructive one? Of course, the answer which occurs to us all is, that it is the result of

[1] Foster's *Physiology*, third edition, p. 618.

the combustion of the materials taken into the body as food, but we must consider this more definitely. In the first place, we may conclude that wherever chemical change is going on nerve energy is being produced. Now, chemical change is occurring in all the tissues of the body, but of these the muscles, the glands, specially the liver, and the nervous tissues, as the brain, are the principal. We can easily understand how nerve force, the result of molecular change in the brain and spinal cord, will radiate out towards the tissues; but how will the energy the result of molecular change in such a tissue as muscle be made use of? Some comparatively new observations help us here. Rumpf[1] showed that if in the frog the spinal cord was divided at two places, and the motor nerve roots leading to the separated portion divided, this portion became absorbed, and almost completely disappeared in about a week. This demonstrates that the muscles exercise a trophic influence backward on the nerve centres, and we may, I think, conclude that from other tissues in which molecular decomposition is active a similar centripetal flow of nerve force will be taking place. Hence we can, I think, form an idea as to how the energy taken into the body as food will be made use of.

But Rumpf made some further observations. He found that if a portion of the spinal cord were separated as before, and the sensory roots divided, a similar but less marked absorption of the portion of spinal cord resulted. This shows that from the surface of the body also a trophic influence is passing to the centres; and as there the animal body is usually losing energy in the form of heat, it suggests again the idea which I on a previous occasion brought before the Medico-Chirurgical Society of Edinburgh,[2] viz., that, as in plants, there is in animals an absorption of certain of the solar radiations which impinge upon

[1] *Pfluger's Archives*, 1881, p. 415.
[2] Vide *Edinburgh Medical Journal*, vol. xxvii. p. 340.

their surfaces. I do not propose again to discuss this question. I need only mention that the atrophy which occurs in the nerves and nerve-centres of special sense when the peripheral organ is removed, and such observations as those of Weir Mitchell in his article on "The Relations of Pain to Weather," may be looked upon as exemplifying it.

And now what are our conclusions regarding nutrition in man and the higher animals? These are—

1. That there is a flow of the nerve energy which produces it from the nerve-centres to the tissues.

2. That just as the sun's rays, when falling in moderate amount on a leaf, will cause a building up, and when in excessive amount a breaking down, so this flow of nerve force, when passing in moderate amount to muscles or glands, has a trophic or building up influence, and when in increased amount a functional or breaking down influence.

3. That this centrifugal flow of nerve force is brought about, at any rate to a considerable extent, by the fact that there is a centripetal flow of nerve force from the muscles and from the skin, and that the direction of the flow, centripetal or centrifugal, will depend on the amount of molecular change in the centres or peripheral organs.

Lastly, we have to remember that we have the power of diminishing or increasing the amount of nerve force which passes from the nerve-centres to the tissues, or *vice versa*. We can do this in one or other or all of three ways,—by diet, by drugs, or by acting directly on muscles or skin.

Let us now see if we cannot make use of these general points as regards nutrition in connexion with the symptoms and treatment of disease.

The first example which I propose to take is *chlorosis*. The general symptoms of a typical case are paleness of the surface, palpitation, breathlessness, loss of muscular power, digestive and other troubles, associated with—and that is most important—no loss of weight, but even a considerable

amount of *embonpoint*, although the amount of food taken is very small.

We can, I think, explain these symptoms by supposing that the amount of nerve force radiating outwards to the tissues is too small, and that we have in them a building up out of proportion to the breaking down, a vegetative rather than an animal process. When we look at a typically chlorotic girl with marked *embonpoint*, we cannot but admit that she represents a large store of potential energy, yet the indisposition to mental or physical exertion, the breathlessness, coldness of the extremities, etc., demonstrate that it cannot be made kinetic.

As regards the pathology of this affection I need say nothing beyond mentioning that Trousseau and some others regard it as a neurosis, and look upon the morbid condition of the blood as secondary to the nervous affection; but the treatment is important in connexion with our paper. The indications here are obvious enough. What we desire is that more nerve force be sent to the tissues, so as to produce a proper proportion between the breaking down and building up. We have plenty potential energy; we wish more kinetic. We can carry out this indication, in the first place, by diet. A nitrogenous diet, as we all know, increases tissue change, whilst a carbonaceous diminishes it. Of this no better examples can be had than the *Banting* treatment of adiposity, and the statement that it has been found that dogs fed on such a substance as Liebig's extract will die sooner than if they have no food whatever. A nitrogenous diet, therefore, is indicated in this affection, and we are mindful that to enable this to be made use of the administration of the peptic and tryptic ferments is often required.

Next, in order to favour molecular change, oxygen is necessary. We therefore insist on fresh air. Trousseau recommends the inhalation of oxygen, and we further, by the administration of iron and potash, endeavour to favour the

growth and development of the red corpuscles, the oxygen-carriers to the tissues. Of the drugs which favour molecular change, strychnine is an important one, and its value in the treatment of chlorosis is well known.

Lastly, by baths with salt and mustard added to the water, friction and massage, electricity, etc., we endeavour to increase the total amount of nerve force by adding to the quantity sent from the skin and muscles to the nerve-centres.

Of course there are many other points to be attended to in the treatment of a case of chlorosis; but, looking at it as being due to the deficient amount of nerve energy sent to the tissues, producing a construction out of proportion to the breaking down, we have probably considered the main indications.

Let us now consider a case in which the opposite condition prevails, in which the destructive is in excess of the constructive. Fevers might be looked upon as examples of this, but in a rapidly progressive phthisis we can appropriately study it. Here the fever, the high temperature, and rapid pulse denote increased tissue change, whilst the loss in weight and rapid emaciation, due to disappearance of fat all over the body, shows a destructive or animal process out of proportion to the constructive or vegetative. In the treatment we advise nourishing food of all kinds, but we do what we can to increase the amount of the food which we have seen diminishes tissue change, viz., the carbonaceous. Of such, cod-liver oil is the best example, and we favour its absorption by pancreatine, etc.

As regards drugs we can say little, as, from the many complications of phthisis, a correspondingly large number of drugs have been tried. As a rule, however, nerve sedatives are beneficial, as opium, hydrocyanic acid, quinine, etc. The hypophosphites are useful; they have a sedative effect on the nervous system, and the observation of Haubner, that cattle

fed exclusively on potatoes or upon roots very poor in phosphates fail to fatten, but that they do so rapidly if phosphate of calcium is given, shows that this drug must diminish tissue change.

But although we admit that in chlorosis the general treatment is to increase, and in phthisis to diminish, the amount of nerve force passing to the tissues, we must remember that frequently treatment which appears to be contradictory of this seems to be indicated. Anæmia and phthisis may co-exist, and we must remember, also, that whilst we have the breaking down, or animal, process taking place in connexion with one system of the body, we may have the building up, or vegetative, in another. For example, in phthisis we often find that with fever and rapid emaciation, a breaking down process, we may have an enormous accumulation of fat, a building up process, in the liver. Then, also, we know that in the apex catarrh which so often, if unheeded or undiagnosed, precedes phthisis, if, along with such treatment as Begbie's mixture, cod-liver oil, and the hypophosphites, treatment which diminishes tissue change, we endeavour to cause an increase of molecular change at the affected part, as we can do by a blister at the affected apex, we can readily promote recovery.

But arguments apparently stronger can be brought against our theory. Thus the secretion of milk, a highly carbonaceous, fatty substance, is increased by a nitrogenous and diminished by a carbonaceous diet.

Again, although, as is well known, there occurs both in men and animals a tendency to accumulate fat in advancing life, and although this may be ascribed to deficient vital energy, we must remember, also, that in infants this adiposity is well marked. Spencer mentions this, and explains it by stating that at neither of these periods is vigour the greatest and digestion the best. Again, Weir Mitchell, in his work on *Fat and Blood*, mentions that his patients under treatment

by massage and feeding generally gain fat and blood at the same time, yet he also draws attention to the fact that the *fat anæmics* are most difficult to treat, and that among men and animals an accumulation of fat has been observed as the result of bleeding.

Such considerations do not, I think, affect the general question that in anæmia the comparatively small amount of nerve force favours a building up process, and in phthisis the larger amount produces a breaking down; and as evidence, also, in favour of this, we must remember the opinion commonly entertained, and brought forward prominently by Trousseau, that the anæmic condition is favourable to phthisis remaining latent, and that in many cases the cure of the anæmia means the development of pulmonary tubercle.

Other examples of disease in the etiology or treatment of which such considerations might be of value could easily be got, *e.g.*, epilepsy, and the use in it of a non-nitrogenous diet might be thus explained. Let me suggest that in antero-lateral sclerosis the thoroughly well-nourished—and I might even say over-nourished—condition of the lower limbs may perhaps be explained by supposing that the nerve energy sent to the centres by the muscles and skin of those parts is prevented to a greater or less extent from nourishing the other parts, and consequently that these limbs, with an abundant supply of nourishment, get more than their normal share.

Reproduction.—In connexion with this, our first object is to ascertain what are the conditions which lead to it. It seems to have been pointed out first by Wolff, a German biologist, that there was a relation between fructification and innutrition. For example, trees are made to fruit while quite small by cutting their roots or putting them in pots, *i.e.*, by diminishing their nutrition; whilst by the opposite treatment, by increasing their nutrition, they develop large stems, branches, and leaves, but are late in flowering. With grain,

too much manure causes an excess of straw; too little causes the head to form too early.

This relationship between fructification and innutrition acquires corroboration from another phenomenon pointed out by Godron. This is, that when crossing takes place between animals of a different species there is a "destruction of the physiological equilibrium in favour of the organs conducive to the life of the individual, and at the expense of those conducive to the life of the species;" *e.g.*, the mule is stronger, more robust, more hardy than either of its parents the horse or the ass, but it is sterile; and in hybrid plants the stalks and leaves are always developed in an exaggerated manner as compared to the flowers. We may therefore, I think, accept this view of the relationship between reproduction and innutrition.

But now the question is, How can innutrition lead to reproduction? An example will, I think, explain this. Suppose we have a cell in a nutritive fluid. It grows by the absorption and assimilation of nutritive matter from the fluid in which it floats. It therefore increases in size, but, as it does so, what happens? We know that if we compare two bodies of similar shape but different size, their respective volumes vary as the cube of their diameters, whilst the proportion of their surfaces is as the square of their diameters; that is, that if we take our cell we find that as it increases in size its volume increases more rapidly than its surface. Now, of course, it is through its surface that it gets its nourishment; as it increases in size, therefore, it is getting less and less nourishment, because its surface is becoming less and less in proportion to its mass. Finally, a time comes when it cannot get a sufficient supply. What happens then? It simply breaks down into two or more pieces; but these small pieces, having a large surface in proportion to their masses, at once begin to grow, to form cells like the original one,—*i.e.*, when the nutrition of the original cell has failed, a reproduc-

tion takes place, and the death of the individual means the life of the race.

Having now seen the general principle that reproduction and innutrition are related, and having supposed how, in a general way, this may be explained, let me now endeavour to find any deductions of practical interest.

In the development of the impregnated egg we have, I think, a good example of it. The egg consists, as all typical cells do, of a wall, contents, nucleus, and nucleolus. It is a living animal structure, consequently it is undergoing chemical change, taking in oxygen and giving off carbonic acid, etc. These processes, however, in the unimpregnated condition, are taking place slowly. Suppose, now, that they are made to take place much more rapidly. What will happen will be, that a much larger supply of nourishment will be required, and the egg will accordingly divide and subdivide; *i.e.*, may we not suppose that the segmentation of the vitellus is the result of the molecular changes in the ovum being immensely increased in activity by the action of the spermatozoid?

Next, our theory may be of interest in connexion with Cohnheim's hypothesis as to the etiology of tumours.

In Zeigler's *Pathological Anatomy*, p. 250, it is stated, "We are not to refer the actual development of the tumour itself to the embryonic period, but are to attribute its appearance in later life to the persistence of germinal embryonic tissues in the otherwise mature organism. A tumour takes its rise in what we might call a belated rudiment, a focus of formative embryonic tissue which has not been utilized in elaborating the normal tissue of the part, and so has lingered on unchanged. Cohnheim, therefore, defines a tumour as an atypical new formation starting in a latent embryonic rudiment. The tumour germs, consisting as they do of embryonic cells, may be very small, and so escape observation. It is even conceivable, he thinks, that the germinal cells may be quite unrecognisable among the ordinary physiological elements of the part.

They may linger on for a long time inactive. It is only when they are favoured by the external conditions, such as the supply of nutriment and their relation to the surrounding tissues, that they begin to multiply and to form a tumour." This is Cohnheim's view; but is it not more probable that instead of there being in the adult a piece of embryonic tissue latent, there is taking place a continual transition process of simple cells into the various differentiated tissues, similar to what takes place in the embryo, only much more rapidly. In this transition process there will be stages in which the tissues formed exactly resemble the embryonic ones. Suppose that this is the case, and suppose that at one or other of these stages reproduction occurs at the expense of nutrition, we should have a mass of tissue more or less resembling the embryonic, *i.e.*, a tumour, produced. The traumatic origin of tumours would also be explained by supposing that the injury, by impairing the nutrition of the tissue, brought about reproduction.

Suppuration is, I think, another process which these general considerations help us to understand. Suppose that from any cause the cells which form the basis of all tissues have their nutrition seriously impaired, what will again happen will be, that instead of growing and developing into a tissue, reproduction will occur, and a large number of simple cells, pus cells, will be produced. We have here a process similar to that engaged in the production of tumours. The difference we may suppose to be due to reproduction taking place at an earlier stage in suppuration.

Lastly, our theory may give us some idea as to the conditions which lead to enlargement of the lymphatic or lacteal glands. In their passage towards the heart, the lymph and chyle have to pass through several of these. Now, the function of these glands is to elaborate out of the fluid passing through them the lymph and chyle corpuscles. We have here, therefore, an example of the formation and growth of

cells in a nutritive fluid. If, from any cause, the nutritive fluid is deficient qualitatively, what will happen will be, that instead of healthy lymph or chyle corpuscles, we shall have formed in these glands an excess of ill-nourished cells.

Such, then, are examples of processes on which our general considerations of reproduction may throw some light. As with nutrition, they could be multiplied.

In conclusion, I have only to state that, although well aware that there are multitudes of other phenomena to be taken into consideration, I trust that when we remember that nutrition and reproduction are the two great functions of nature, my applications of them will not be considered too wide.

DILATATION OF THE URETERS AND RENAL PELVES. HYDRONEPHROSIS.

OBSTRUCTION to the escape of the contents of a hollow viscus, or of the secretion of a gland, causes dilatation, and this pathological process in the case of the kidney gives rise to the conditions of cystic kidney, hydronephrosis, and pyonephrosis, or surgical kidney. In these affections the position of the obstruction is different. In the former the small tubules are obstructed, causing a formation of cysts, varying in size and number, in the substance of the organ; in the latter the obstruction is lower down in the urinary tract, causing a dilatation, varying in degree, of the ureter and pelvis of the kidney.

It is this latter condition, and more especially hydronephrosis, that I now propose to discuss. The causes of it are very numerous. In his work on *Urinary and Renal Diseases*, Dr Roberts has collected particulars of many cases of this disease. In nearly all of them distinct mechanical obstructions in the urinary tract could easily be demonstrated, such as congenital imperforate ureter or urethra, or ureter rendered imperforate by the pressure of an abnormal renal artery, by cicatrization after ulceration, by twisting, etc. He notes, however, that cases have been reported where no obstruction could be discovered, and which seem therefore to be mechani-

cally inexplicable.[1] The following case would belong, I think, to this latter class. I shall now describe it, and shall also propose a mechanical explanation, which I venture to hope may seem sufficient.

J. G., æt. 8 years, admitted to the Liverpool Infirmary for Children, 21st January 1875, suffering from incontinence of urine. His mother states that he was quite healthy till 3 years old, when as the result of an injury he had *phymosis*. After this had been pricked by a doctor, the swelling got better, and he seemed nothing the worse. He had then no incontinence. Shortly after this, however, he seems to have had a feverish attack, which, although not severe, weakened him very much. At this time, *i.e.*, about four years before his admission, the incontinence began. It has continued, and his mother states that he has never regained strength, and has grown very slightly since. On admission the boy looked thin and weakly, and not more than five or six years of age. The prepuce was contracted and almost completely adherent to the glans. He had no pain, but great incontinence of urine. There was increased frequency of micturition, often three or four times in the hour—only a few drops of urine were passed at a time, and the bladder was never distended. The urine seemed over the normal in amount, but the exact quantity passed in the 24 hours could not be ascertained. It was pale in colour, sp. gr. 1010, deposited a slight amount of pus and renal cells on standing, and the supernatant liquid showed traces of albumen.

Circumcision was performed on the 3rd February, and repeated more thoroughly in the beginning of March. Slight temporary improvement followed both operations, but in April the boy was as ill as ever. He was getting gradually weaker till the 16th April, when, after a slight attack of diarrhœa, he suddenly became insensible, respirations rapid (48 per minute) and panting, pulse 116, temp. 97·4°. He

[1] *Urinary and Renal Diseases*, Dr Roberts, 3d edition, p. 491.

became comatose, and died after remaining so for about 12 hours. On post-mortem the body was found healthy, with the following exceptions:—

The bladder was somewhat contracted, and its walls thickened. Its inner surface showed no signs of cystitis; there was no pus formation, but great roughness, owing to the hypertrophied muscular fasciculi projecting rib-like into the interior, and forming small diverticula in many places. The cavity of the bladder when distended was smaller than natural, the walls about a quarter-inch thick. There was no stricture in the urethra, and the openings of the ureters through the bladder-wall were normally patent. Both ureters were greatly dilated in their entire length. Both kidneys were hydronephrotic; on section they showed the dilated pelvic chambers, replacing to a greater or less extent the normal pyramidal portions of the kidneys. The cortical portions were also encroached upon in several places. The capsules were slightly adherent. The renal sacculi and ureters contained urine, and there was a small sediment (consisting of pus and renal cells) in one of the dilated chambers of the right kidney. Microscopically its appearance resembled in parts that of the cirrhotic kidney. There was a great excess of fibrous tissue in the cortical portion, and the tubules, etc., were correspondingly obliterated. As this condition was manifestly the result of compression, it is interesting when considered in connexion with Dr Johnson's theory that the fibrous appearance is simply the result of the obliteration and shrinking of the tubes and intertubular capillaries. Before considering the pathogeny of this case, I may mention that it is one of three similar cases, which I had the opportunity of seeing at the Liverpool Children's Infirmary, during a period of 14 months. In those two cases also, the cause of the urinary irritation was the condition of the prepuce. They were both thin boys, looking much under their ages. There was greatly increased frequency of micturition; the urine was pale, of

low sp. gr., and containing albumen proportionate to the amount of deposit of pus cells. Both died, after manifesting the same marked uræmic symptoms. In only one, however, was I able to verify my diagnosis by a post-mortem.

In these cases, then, as the specimen shows, there is no apparent cause for the damming up of the urine which has dilated the ureters and pelves, but I think when we consider the symptoms during life, we have explanation sufficient.

For about four years the boy has been having almost constant trouble with his urine. He has had greatly increased frequency of micturition, amounting often to forty or fifty times a day, and he always wet the bed at night. This necessitated equally frequent contractions of the bladder, and as, to permit the urine to flow into the bladder, a relaxed condition of that organ is necessary, by every contraction the entrance of urine into the bladder was prevented, and damming up and consequent dilatation of the ureters and pelves resulted. That this is the pathogenesis in these cases, I have been led to believe on the following considerations :—

1*st*, The damming up must have occurred at the points of entrance of the ureters into the bladder, because the ureters are dilated, whilst the bladder is not so, but if anything contracted and thickened.

2*nd*, The tracts of the ureters through the bladder-wall are normally patent.

3*rd*, Contraction of the bladder is quite capable of preventing the entrance of urine into it, as the pressure put into action for the expulsion of urine is greater than a pressure which, applied to the ureter, will not only dam up its contents, but cause consequent complete suppression of urine ; and when we remember the oblique course of the ureters through the bladder-wall, we can the more readily understand how this will take place. The expulsive force of the bladder, including, of course, the abdominal pressure (voluntary and involuntary), varies in different individuals ; but from obser-

vation and experiment, I think that in calling it 2, 3, or 4 in hg. I am not overstating, while it has been found that a pressure of 7–10 mm. hg.[1] (·4 in hg.), will greatly impair the secretion of urine, and a pressure of 40 mm. hg. (1·6 in hg.) will completely suppress the secretion of both its solid and fluid constituents.

It would be interesting to know the contractile power possessed by the bladder itself, but I have not been able to get an exact estimate of it. I find, however, that Donders[2] says that the detrusor urinæ is of itself able to overcome the resistance of the sphincter vesicæ. He has more than once observed in dogs in which the abdominal cavities were opened, that when an electric current was applied to the distended bladder, it contracted rapidly and equally, so that the urine was driven out in a strong stream.

4th, A similar condition of the ureters and renal pelves is met with in old cases of stone in the bladder—"Surgical kidney," so called. If in these cases the stone has acted as the obstruction by preventing the escape of urine through the urethra, we find the bladder dilated also to a greater or less extent. It is often, however, contracted and thickened, and here the irritation of the stone has caused increased frequency of micturition, correspondingly frequent contractions of the bladder, and hypertrophy of its walls, and this tends to dam up the urine in the ureters. But the pyelitis which usually accompanies this affection has probably the great share in causing the dilatation, as by its occurrence the lining membrane of the ureters and renal pelves is swollen, and by the purulent secretion their contents are increased in amount and consistence. Perhaps, also, the power of resistance to distension possessed by the ureters and renal pelves may be diminished by the inflammation, and so the process of dilatation be further accelerated.

[1] Wundt, *Physiologie d. Menschen*, 3 Auflage, p. 434.
[2] Donders, *Physiologie des Menschen*, p. 484, footnote.

5th, I have had many opportunities of studying the urinary symptoms in children affected with incontinence of urine, so called. Those cases were mostly due to a hypersensitiveness of the bladder, owing to the reflex irritation of an enlarged or adherent prepuce. In all the severe cases I noted that as long as the increased frequency of micturition lasted, the urine passed was (as far as could be ascertained) not markedly increased in amount, its colour was pale, and sp. gr. low. When a cure was effected, and the irritability of the bladder had disappeared, the urine became normal in its characters, thus:—

C., æt. 11, admitted for incontinence due to adherent prepuce. Urine pale, sp. gr. 1006, no albumen. Circumcision was performed, and cure resulted. On leaving the hospital the urine was of amber colour, sp. gr. 1027.

D., æt. 7, urine before the operation pale, sp. gr. 1007; afterwards amber, sp. gr. 1022.

J., æt. 8, was another patient, in whom the urinary symptoms seemed due to cold. When first seen, there was greatly increased frequency of micturition, although the total amount of urine passed in the twenty-four hours seemed not increased; it was pale in colour, and sp. gr. 1007, no albumen. After being kept quiet in bed for a week, the symptoms passed off, the urine became of a dark amber colour, sp. gr. 1025.

I have notes of similar cases, but it is not necessary to give them here. The following case is, however, more than usually interesting:—

W. M., æt. 2 years, affected with incontinence, etc., very severely. Urine pale, sp. gr. 1006. He was circumcised October 1874. Immediate and complete relief followed, and the sp. gr. of the urine rose to 1020. I had opportunities of watching this case for more than a year. He had frequent temporary relapses, and at these times the urine always became pale in colour and diminished in sp. gr., whilst as long as he was well the urine was normal in all its characters.

The pale colour and low sp. gr. of the urine met with in such cases can, I think, be easily explained. The urine being dammed up at the point of junction of the ureters and bladder, there results an increase of pressure of the urine in the ureters, renal pelves, and uriniferous tubules, and consequent impairment of its secretion and diminution of the transudation of the urea salts, etc., which go to make up the colour and specific gravity. If the secretion of urine were a process of osmose alone, we could explain this occurrence on the theory that it depended on the difference of pressure in the bloodvessels and urinary tubules; for on trying the effect of pressure on osmose through a membrane, it will be found that with a solution of a certain specific gravity and water of sp. gr. 1000, an increase of pressure on the solution will impede the entrance of water into it, and will increase the transudation of the salt into the water, and so add to its specific gravity; whilst a contrary pressure will increase the transudation of water into it, and diminish the transudation of the salt into the water, and so diminish its specific gravity. In the kidney, of course, the conditions are not the same; we have the transudation of the watery portion taking place into the Malpighian capsules, and of the urea, etc., from the capillaries into the tubules; but we are warranted in concluding that similar results will take place from pressure. Thus Hermann[1] found that urine secreted under a positive pressure in the ureter was less in quantity, and contained a smaller proportion of urea than when secreted under normal conditions.

Haidenhain[2] ligatured one ureter in a rabbit, and 24 hours afterwards injected into the circulation a solution of indigo. On killing the animal it was found that in the kidney of which the ureter had been tied, the amount of colouring matter which had passed into the tubules was less than in that of the opposite side, of which the ureter remained patent.

[1] See Wundt's *Physiologie*, p. 434. [2] Pfluger, *Archiv* ix. 1, p. 10.

It has also been observed clinically, that in cases of blocking up of the ureter by a calculus, any urine which escapes, and which has of course been secreted under pressure, is pale, contains little urea, and is of low sp. gr.[1]

I regret that I have been unable to give more precise information as to the quantity of urine in the cases which I have mentioned, but the impossibility of doing so is readily understood on considering the age of the patients and the nature of the affection. I mention this, as I believe that although there may be an impairment of the secretion of urine at first, by the damming up of the ureters owing to frequent micturition, I believe also that this damming up, if it last long enough to cause structural change in the renal pelves, tubules, etc., and from the fact of its being not constant but intermittent, may result in an increase of the total amount of fluid secreted; and I think we are free to consider that this increase in the total amount of fluid associated with the continuing, and probably from this same cause increasing irritability of the bladder, cannot but result in augmenting the process of dilatation.

The object of this paper then is to suggest—

1*st*, That increase in the frequency of micturition is capable of causing a greater or less damming up of the urine in the ureters, renal pelves, and tubules, and consequent pale colour and diminution in the sp. gr. of the urine.

2*nd*, That this damming up, if continued, will in time cause dilatation of the ureters and renal pelves, and a more or less hydronephrotic condition of the kidneys.

[1] Dr Roberts, p. 27.

THE

PHYSICS OF THE BLADDER AND URETERS.

WHEN a healthy adult bladder contains urine to the amount of 20 oz., it may be said to be full, and any increase of this quantity must necessarily cause a corresponding distension of the bladder walls. This must in turn cause an increase of pressure in the bladder, and, consequently, in the ureters, *i.e.*, the urine will be secreted under a counter-pressure in the ureters and renal tubules.

For instance, in one of the first bladders which I tested,[1] and which I mention as one which may be considered healthy, I found that with a head of water of 3 feet it rapidly distended till it held 20 oz., after which it took in water so slowly as to be almost imperceptible. I then gradually increased the height to 8 feet, when it burst, after having taken in 10 oz. more, in all 30 oz. Thus, then, after 20 oz. a greater pressure would seem to be required to make the bladder hold more.

Now, as an increase of the pressure in the urinary tubules has for its result a gradual diminution in the specific gravity and amount of the secretion, and as, if continued till it amounts to 2·4 in. hg., it will cause complete suppression, we should expect that, in cases where from any cause the bladder is prevented from emptying itself for a considerable period, symptoms would occur showing that

[1] In all the experiments referred to the bladders were removed from the body.

the secretion of urine was being checked, or, at any rate, we should expect to find the specific gravity of the secreted urine lowered.

In practice, however, we find that both these suppositions are, as a rule, wrong. Retention of urine almost never leads to suppression, and although I have observed many cases in which complete retention had existed for hours, and in which by catheterism quantities of urine varying from 30 to 50 and 60 oz. have been drawn off at one time, I have never had the slightest reason to believe that there was in these cases any diminution in the specific gravity of the secretion. It is, however, necessary to mention here that the maintenance of a high specific gravity in these cases may be explained by supposing that a certain amount of absorption takes place by the bladder walls. In this way, although the urine sent down by the kidneys might be of low specific gravity owing to counter-pressure, yet, having reached the bladder, an absorption of the watery part would occur, and so the specific gravity of the urine in the bladder be kept high. But when we remember the very large quantities of urine which are removed from previously healthy bladders in cases of retention, we may, I think, conclude that this cannot take place to any great extent.

The physical properties of the bladder during life, therefore, as seen in practice, would seem to be different from those manifested by it after death, as seen by the above experiment, but this difference can be demonstrated perhaps even better in the case of the ureters.

If the pressure necessary to burst one of these be tried, it will be found to amount to about 5 ft. hg., the ureter distending to about $\frac{1}{4}$ or $\frac{3}{8}$ in. in diameter before giving way. Yet cases occur in practice where the ureters are found dilated to $\frac{1}{2}$, $\frac{3}{4}$, or 1 in. in diameter, this distension having been caused by an internal pressure not exceeding 2·4 in. hg., as one exceeding this would have caused complete suppres-

sion of urine. The object of this paper is to endeavour to reconcile this seeming disagreement in physics, and as much as possible by physical means. There are, of course, vital elements to be taken into consideration, viz., the functions of nutrition and growth, and the importance of these is also more obviously seen in the case of the ureters than in that of the bladder. Thus, in hydronephrosis there is, in addition to dilatation, an evidently great increase of the substance of the ureter walls, and we may assume that the same process occurs in the bladder in cases of retention. No doubt only to a comparatively very slight extent, but yet so much that we need not expect by experiment to be able to distend a perfectly normal bladder till it holds 60 oz. of water, nor a ureter till it is 1 in. in diameter, in the same way as we need not expect by simple distension to be able to dilate a non-pregnant uterus till it presents the form and size of one at the full time.

In all these cases, then, the vital functions of nutrition and growth must be considered; but, in this paper, we shall confine ourselves to the discussion of the purely physical causes, and the physical properties of the tissues, which for this purpose we must consider are those manifested by them when subjected to a distending force, viz., cohesion and distensibility.

On these points many experiments have been made. Perhaps the earliest are those of Hales,[1] and as they are of interest in connexion with the subject of this paper, I shall shortly mention some of them. By means of a mercurial manometer he found that the carotid artery of a spaniel dog required a pressure of 5·42 atmospheres (80 lbs. per sq. in.) to burst it.

In a mare he found the carotid so strong that he could not burst it with his apparatus.

The jugular vein of a horse bore a pressure of 148 ft. water (65 lbs. per sq. in.) and did not burst.

[1] Stephen Hales, *Statical Essays*, London, 1740.

Hales also experimented on the tenacity of the periosteum, and noted Musschenbroek's experiments on the strength of silk threads.

He further tried the strength of the walls of the stomach of a dog, finding that it burst with a pressure of 36 in. of water. All his experiments are, however, very elementary, and he has neglected to take into consideration many important physical points.

Among other investigations those of Valentin and Wertheim must be noted. They tested the cohesion of the different tissues of the body, and the following represents the average results:—

Muscles	·06	Tendons	5·3
Arteries	·14	Bone (after Wertheim)	8·0
Veins	·23		
Nerves	·98	Bone (after Bevan)	37·9
Hair	9·9		

More lately Matthews Duncan and Poppel tested the cohesion of the membranes, and from this were enabled to form estimates of the power of labour. Still more lately, Symington has tested the cohesion of the sciatic nerve in connexion with the operation of nerve-stretching.

From all these experiments much valuable information has been derived, but in none of them has sufficient consideration been paid to one very important subject, the neglect of which assuredly leads to wrong conclusions, and the proper estimation of which it is the object of this paper to show. The point is one which is of necessity well known to engineers, and to explain it I quote from an essay on the "Strength and Qualities of Wood and Metals," by Professor Macquorn Rankine.[1] Speaking of the cohesion of an iron bar, he says, "If the pull upon a bar be gradually and continually increased, a limit is at length reached at which

[1] *Cyclopædia of Machine and Hand Tools*, part ii. p. 6.

the material ceases to be able to resist the stress, and then the bar is torn asunder. The utmost pull per sq. in. of cross section which a bar of given material sustains just before being torn asunder is called the tenacity, ultimate tenacity, or tearing force of the material, and sometimes the direct cohesion.

"The proof tenacity or limit of the pull per sq. in. of section which a bar of a given material can bear without injury is indicated in experimenting especially with ductile materials, either by an increase in the extensibility of the material when a pull exceeding that limit is applied, or by an increase of extension at each time that such pull is reapplied after having been removed,—showing that the stress, though not sufficient to tear the bar asunder at once, would ultimately do so if its application were often enough repeated," or what amounts to the same thing, if its application were long enough continued.

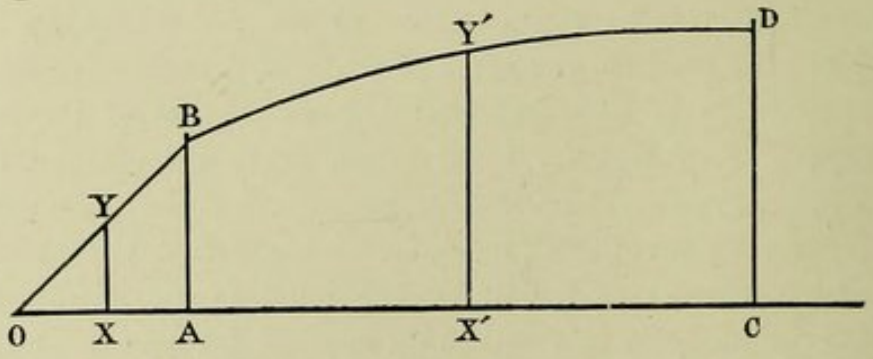
Thus, then, engineers, in estimating the strength of materials, not only know what is the force which will burst a given material at once, but also that there is a smaller force which, if it be continuously or intermittently applied, will in time produce the same effect.

Further, it is manifest that the estimation of the latter force, the *proof tenacity*, is of much more importance than that of the former, the *ultimate tenacity*, for the reason that in testing say a boiler or receiver, we know that the pressure to which in working it will be subjected is one which will be continuously or intermittently applied for weeks, months, or years.

It is also equally evident that in the animal body, in calculating the amount of pressure put into action from the effects produced, the knowledge of the proof tenacity is by far the more important. The pressure in the bloodvessels, gland-ducts, stomach and intestines, uterus in pregnancy and parturition, bladder, etc., are all pressures which are being

continuously or intermittently applied for a longer or shorter period of time. The estimation of the proof as distinguished from the ultimate tenacity is, then, for this reason of equally great importance in physiological as in engineering physics; but when we consider the difference in the ratios of the proof to the ultimate tenacity in the different materials, we must conclude that for this further reason the importance, as far as forming correct conclusions is concerned, is far greater in the physiological. This is simply because in the latter the difference between the proof and ultimate tenacity is far greater. The effect of strains, not exceeding and exceeding the proof tenacity, on cohesion and distensibility, can be best understood by the following diagram from Rankine.[1]

Let O represent the position of the right-hand end of a horizontal bar in its free condition, the other end being fixed somewhere to the left of the figure. When a gradually increasing stretching-load is applied to the bar, let its right-hand end move from O to C, so that a scale of distances, or



abscissæ, measured from O towards C, shall represent the successive elongations of the bar. Let *ordinates* at right angles to O C represent, according to a suitable scale, the loads required to produce these elongations, so that X Y represents the load which produces the elongation, O X, X′ Y′

[1] *Cyclopædia*, part ii. p. 8.

the load which produces the elongation O X′, etc. Let A B be the proof load, and X Y a load within the proof load, and let X′ Y′ and C D be loads exceeding the proof load. It will now be noticed that when the load does not exceed the proof load the elongation bears a nearly constant proportion to the load, so that the line from O to B is nearly straight; but when the load exceeds the proof, the elongation increases more rapidly than the load in a continually increasing proportion till the bar is torn asunder; hence from B to D the line presents a curve concave towards the axis of abscissæ or scale of elongations O C. Having now understood what is ultimate tenacity and proof tenacity, and what are the effects of loads exceeding the proof, we must next consider what is the proportion of the proof to the ultimate tenacity. On this point Rankine says:—"The proof tenacity, generally speaking, approaches nearest to the ultimate tenacity in the stiffest and strongest materials." Conversely, we conclude that in the softer and weaker materials the proof tenacity will be proportionally smaller than the ultimate, and as the animal tissues with which we have to deal in this paper come under the latter class of materials, we can readily understand the importance of considering the proof as distinguished from the ultimate tenacity. In fact, it may be said that, speaking in the strict physiological or pathological sense, the knowledge of the ultimate tenacity of any of the tissues is of no importance whatever. In actual treatment it is of the greatest importance; the surgeon should know the ultimate tenacity of the sciatic nerve while he is stretching it for sciatica; the accoucheur should know that of the child's neck when he is engaged with a breech presentation; and in a medico-legal sense it is often an important point, but when we have to deal with forces exercised within the body, its importance is almost *nil*.

To obtain data upon which to form conclusions as to the

proportion between the proof and ultimate tenacity in animal tissues, I performed the following experiments:—

The tissues tested were the intestines of sheep, and the results obtained show—1*st*, The ultimate tenacity or bursting weight; 2*nd*, The length of time which $\frac{2}{3}$ of this takes to cause rupture; 3*rd*, The length of time which $\frac{7}{12}$ takes; and 4*th*, The time taken by $\frac{1}{2}$. The method employed was to fix equal lengths of intestines (12 in.) to wooden cores, by means of which the various weights could be conveniently applied.

The ultimate tenacities and the various fractions of them, $\frac{2}{3}$, $\frac{7}{12}$, and $\frac{1}{2}$, were obtained in the following way:—For each experiment three feet of intestine was taken and divided into three equal portions. By means of a spring weigher, the weights required to burst the two outer portions were obtained, and the average of these two was taken as representing the ultimate tenacity of the middle portion. To the middle portions the various fractions of these weights were applied, and the time which elasped between the application of the weights and rupture of the gut noted.

The greatest possible care was taken to prevent the pieces drying, and in every case the experiments were made within eighteen hours of the death of the animal.

The results of fifty such experiments are given in Table I. on the following page.

This table shows some very interesting points: *First*, The great variations in the amount of the bursting weight in different portions of intestines, the extremes being 20 oz. and 60 oz. The strength of the gut seems to be owing to the peritoneal coat and not to the muscular, and I came to this conclusion from observing the manner of breaking, and also from having in other experiment weighed the portions before testing them. As the weight depends upon the muscular coat rather than on the serous, if the strength of the gut were due to the former the heaviest portions would always be the strongest. By experiment, however, I found that

although this was for the most part the case, yet the results were not such as to enable me to calculate from the weight alone what the tenacity should be. In all cases further I found that the upper and middle portions of the intestine were stronger than the lower.

TABLE I.—Intestine of Sheep.[1]

No.	$\frac{2}{3}$.		$\frac{7}{12}$.		$\frac{1}{2}$.			
	Bursting Weight.	Time.	Bursting Weight.	Time.	Bursting Weight.	Time.		
	Ounces.	Minutes.	Ounces.	Minutes.	Ounces.	H.	M.	
1	21	$6\frac{3}{4}$	42	$5\frac{1}{2}$	31	3	30	
2	36	10	42	14	26	7	30	
3	42	1	44	10	24			More than 10 hours.
4	36	14	48	16	24	0	20	
5	47	15	52	49	23	1	30	
6	48	$5\frac{1}{2}$	55	81	44	7	0	
7	56	20	56	14	45	1	30	
8	48	11	54	8	43			More than 10 hours.
9	40	3	49	55	40	2	35	
10	36	$6\frac{1}{2}$	49	100	35	3	0	
11	40	20	44	25				
12	52	$\frac{1}{2}$	42	4				
13	52	5	46	$23\frac{1}{2}$				
14	48	$1\frac{1}{2}$	44	5				
15	54	$1\frac{1}{2}$	44	120				
16	60	1	46	25				
17	60	2	54	180				
18	36	$\frac{1}{4}$	54	13				
19	30	8	28	25				
20	30	$\frac{1}{2}$	30	13				
Aver. in min.....		$6\frac{1}{4}$ (nearly).		41 (nearly).		202 (nearly).		

Secondly, The lengths of time which the different fractions of the bursting weights take to cause rupture. These also vary considerably, with $\frac{2}{3}$ from 15 sec. to 20 min., and with $\frac{7}{12}$ from 4 min. to 180 min. With $\frac{1}{2}$ the shortest time was 20 min., and the longest of the eight which burst was 7 hours. The other two did not burst within 10 hours, but by that time both of them had almost given way; and had I been able to completely prevent drying taking place for an hour or two longer, I am sure that rupture would have occurred.

[1] For further details on this subject of tenacity, see p. 70.

The cause of these great variations is not very clear, but they are probably in great part due to the estimate of the bursting weights being so uncertain.

The average lengths of time are—

For $\frac{2}{3}$ bursting weight nearly $6\frac{3}{4}$ minutes.
" $\frac{7}{12}$ " " 41 "
" $\frac{1}{2}$ " " 202 "

But does half the bursting weight represent the proof tenacity of the intestine? We may, I think, conclude that it does not. One-half will cause rupture in say four hours, but less would probably have the same effect if a longer time were given. To determine what is the proof tenacity further experiments are necessary; but from these few which I have tried we can, I think, obtain some interesting information as regards the physics of the bladder, ureters, and other tissues.

In order, however, to understand these, we must bear in mind another important consideration. This is, that in the case of the bladder, ureters, etc., the force tending to their rupture is exercised against all points of the inner surface of their walls; and, consequently, the greater the extent of this surface the greater will be the amount of force to which it is subjected, and the less will be the power of the walls to resist a force tending to their distension or rupture. In practice, the strength is measured by the radius or diameter—if this be doubled the strength will be halved. Thus with equal strength of walls, a hollow sphere, the superficial extent of the inner surface of which is 25 sq. in., and the diameter of which is 2·8 in., will be able to resist twice the internal pressure which can be borne by one having an internal superficial area of 100 sq. in. and a diameter of 5·6 in.

Similarly any hollow organ, as a bladder, ureter, uterus, stomach, etc., will oppose a greater resistance to distension or rupture when the contained distending fluid is small in amount than when it is large.

For example, let us take three bladders A, B, and C, containing 20 oz., 40 oz., and 60 oz. respectively. Supposing that they are all equally spherical, the diameter of A will be 4·04 in.; of B, 5·19 in.; and of C, 5·83 in. Thus, B will be $\frac{1}{4}$ and C $\frac{1}{2}$ weaker than A; or, in other words, if A can just stand a pressure of 15 lbs. per sq. in., B will only be able to stand about 12 lbs. and C only about 10 lbs. per sq. in. This is assuming that in A, B, and C the walls are equally strong and able to resist a distending force, which is, however, not the case in the animal body. On the contrary, we may conclude that the more stretched the walls of a bladder become the less will they be able to resist a further stretching, and so as a bladder becomes more and more distended it loses strength, not only by becoming larger, but also by its walls becoming intrinsically weaker. Thus, then, in studying the physics of such a structure as the bladder, we must always bear in mind two physical facts, *first*, that it is the proof and not the ultimate tenacity with which we have to deal; *second*, that as such structures are becoming distended they are losing strength, from their increase in size and also from their walls becoming weaker.

Further, we must always bear in mind the dependence which the tenacity has upon time. A comparatively slight pressure acting for a long time will have the same effect as a greater pressure acting for a short time.

Let us now apply these facts to the physics of the bladder, ureters, etc., and let us take the ureters first.

The ultimate tenacity of the ureters is, as we have seen, about 5 feet hg., and the greatest pressure which can be exercised by the urinary secretion is about 2·4 in. hg. Supposing now that, as regards the proportion between the proof and ultimate tenacity, the ureter resembles the intestine, and allowing for the weakening which would result from distension, let us assume that in four hours $2\frac{1}{2}$ feet hg. would be sufficient to cause rupture. This would, however, be about

twelve times greater than the greatest secretion pressure in the ureters; but further, if a ureter be completely obstructed, the amount of urine secreted in four hours would be far more than sufficient to distend the entire urinary tract above the obstructed portion. In this way the secreting process would be arrested, and the secreting tissue would become destroyed. Hence, in cases of complete obstruction of the ureters, long before such a comparatively slight force as that of the renal secretion will have had time to act so as to cause distension or rupture, the kidney structures will have become disorganized and destroyed. It is otherwise, however, when the obstruction is not complete. Here the secreting pressure will probably never be so high as 2·4 in. hg.; but, at the same time, as there will be no arrest of the secretion, it will continue acting not for hours only, as in complete obstruction, but for weeks, months, or years. It will thus cause distension; and, as we have seen, the more this occurs the less will be the power of resistance to further distending force.

Hence dilatation of the ureters can never occur to any extent as the result of complete obstruction. The time allowed to the distending force to act is so short, that long before it can have had any effect in causing distension the secreting tissues will have been destroyed. On the other hand, we can understand how readily it will occur when the obstruction is incomplete, as here the pressure, though not so great, acts for a long period of time. We need not discuss the possibility of rupture of the ureter. Let us now apply the same two physical considerations to the case of the bladder. In the first place, then, we have our distending force the same as in the case of the ureter, *i.e.*, not exceeding 2·4 in. hg.; but, in the second place, inasmuch as the size of the bladder is very much greater than that of the ureter, the effects of this pressure will be correspondingly increased. For instance, in a case of completely obstructed ureter, we may, I think, safely say that the resulting distension will not

exceed ½ in. in diameter, whilst, as we have seen bladders containing 20, 40, or 60 oz. urine will probably have diameters of about 4, 5, and 6 in. respectively. Thus they will have correspondingly less power to resist the distending force. Further, the quantity of urine secreted in a given time is so great as to preclude the idea that a corresponding increase of the substance of the bladder walls—as occurs, for example, in the uterus, *pari passu*, with its dilatation, during pregnancy —could occur.

Thus, then, we may with reason suppose that a bladder, if the exit of urine be completely prevented, is very likely to burst, and that, too, in a comparatively short period of time.

To obtain some more precise information on this point, I have, during the past few months, performed experiments on many bladders, both human and animal. My results, probably owing to the fact that there are great variations in the size and strength of this organ in different, otherwise similarly, constituted individuals, both human and animal, are somewhat indefinite. Such as they are, however, they entirely corroborate the above conclusion.

The points which I have endeavoured to determine are—first, Can a pressure not exceeding that of 2·4 in. hg. burst a bladder, the exit of water per urethram being prevented? and, secondly, What is the proportion between the amount of pressure, on the one hand, and the time occupied in bursting, along with the amount of distension, on the other?

The method employed was to distend the bladder with water, raising or lowering the head of water according to the pressure required. As, however, I found that in this way there was a certain amount of oozing through the walls, and as, when the head of water was small, this took place to such an extent as to effectually prevent the bladders becoming further distended, I, after some trials and failures, fell upon a plan which, though it has one great disadvantage, yet gives fairly trustworthy results. This was to distend the bladders

indirectly by means of a large-sized india-rubber balloon. Such a balloon when collapsed can be easily introduced into the bladders of sheep after they have been first slightly distended with a few ounces of water. This previous distension I found to be absolutely necessary, as the bladders were all tested within a short time after death, and were, when I obtained them, so firmly and completely contracted as to render introduction of the balloon impossible.

The great disadvantage of the balloon is, of course, that it does not give us the exact amount of pressure which the bladder has to bear, as a certain amount of pressure is taken up in distending its own walls. Hence the results must not be taken as absolutely correct, although comparatively they may be regarded as being so.

I have selected the following sixteen cases as being the most trustworthy and characteristic. The first eight were taken from one lot of sheep, and the second eight from another, the former being of rather larger size. In all cases the experiments were made within a few hours after death.

TABLE II.—Bladders of Sheep.

No.	Head of Water in Feet.	Time.	Amount of Water at Bursting.		No.	Head of Water in Feet.	Time.	Amount of Water at Bursting.	
		Minutes.	Ounces.				Minutes.	Ounces.	
1	9	2¼	31	First Set.	9	5	4	38	Second Set.
2	9	1 m. 55 s.	34		10	5	5	40	
3	8	4	33		11	4½	5	50	
4	8	2	36		12	4½	4½	60	
5	7	4	48		13	4	18	48	
6	7	2½	45		14	4	20	65	
7	6	5	65		15	3½	75	70	
8	6	5	50		16	3½	80	50	

The results show that the greater the pressure the more rapidly will the bladder burst, and the smaller will be the amount of water required; while the less the pressure the longer is the time before rupture, and the greater the amount

of water held. The lowest amount of pressure is represented by a head of water of 3½ ft. This is about 1½ ft. higher than the urinary secretion, and would seem to give us no grounds for the belief that such a pressure could cause rupture; but, on the other hand, we must remember that the balloon itself will take up some of the pressure, and that the time was not very great. Further, I have frequently succeeded in bursting bladders, both of the sheep and pig, with the balloon and only 3 ft. head of water in periods varying from 4 to 40 hours.

Of human bladders I have tested twenty, but as the individuals from whom they were derived differed from one another in age, sex, and pathologically, the results obtained are very discordant. The strongest was probably that of a boy aged 13 years. With a head of water of 6 ft. it held for 72 hours, when the experiment was stopped, and it was then found to contain 16 oz. water. This bladder was afterwards caused to burst almost at once by a head of water of 10 ft., the amount of water being a little over 16 oz. Several of them I succeeded in bursting with a pressure of 4½ ft. and 5 ft. water (balloon included) in periods varying from 20 minutes to 48 hours.

The last one I treated was specially interesting. It was that of a particularly well-developed and muscular man aged 34 years, who had been killed by a gunpowder explosion. The bladder was very capacious, being distended when obtained, and with the balloon and 4½ ft. head of water, it took in 50 ounces very rapidly. It was then left in a vessel of water (so as to prevent drying), and allowed to distend. It took in water rapidly at first, and afterwards more slowly, and burst in 60 hours, having taken in 18 ounces more water—in all, 68 ounces. I then, by means of a mercurial manometer, made an estimate of the amount of pressure taken up by the india-rubber balloon, and calculated that in this experiment it must have amounted to 2½ in. hg. As the total head was 4½ ft. water, we may conclude that

this bladder burst in 60 hours with a pressure of about 1 ft. 9 in. water, *i.e.*, about 12 in. below that of the urinary secretion. We need not here discuss whether the bladder tissue is weaker during life than after death; probably it is so. It is necessary, however, to remark that, as regards the manner of bursting, the human bladders differ from those of the sheep. In the former the rupture occurs usually near the neck, and always at a part uncovered by peritoneum; in the latter it takes place near the fundus, and through the peritoneum. In the human subject the peritoneum is proportionately much stronger.

These considerations will, I think, enable us to understand that a bladder may rupture if the exit of urine be completely prevented, and further, how the maintenance of a high specific gravity of the retained urine may be explained. But we must always bear in mind that we can never in experiment fulfil all the conditions existing in the living body. The circumstances are very different, but I feel convinced that, as far as the subject of this paper is concerned, the difference is one which will cut both ways. On the one hand, in the human body the bladder is to some extent bound down by the tough peritoneum, and when full further distension is prevented below by the bony pelvis, and impeded above by the muscular abdominal walls. Hence any of the numerous conditions which increase the abdominal pressure will have the effect of checking distension, and, while tending to prevent rupture of the bladder, will correspondingly aid the occurrence of suppression of urine.

On the other hand, as the experiments of Wertheim, Caspar, and others show, we must remember that the direct cohesion of the fibrous tissues is greater after death than during life; and from my own observations, I feel sure that at the same time their proof tenacity is higher and extensibility less.

Clinically, some cases of retention have been reported as ending by rupture of the bladder, and others by suppression

of urine, but I have not been able to obtain particulars sufficient to enable me to form an opinion as to the cause of these different methods of termination.

Further, the consideration of these two physical points—the proof tenacity, and the weakening which results from increased capacity—enables us also to understand many other processes which occur in the body,—how, for example, in the circulatory system a slight increase of the blood pressure, or a slight weakening of the cardiac or vascular walls, may cause gradually-increasing dilatation of the heart or vessels, or aneurisms, which enlarge and burst. The second point further shows us the value of the clot in the case of the latter.

Similarly they explain how readily a very slight pyloric obstruction will cause dilatation of the stomach, how cystic tumours and abscesses distend and burst, how the treatment of continuous extension acts, etc., etc.

Of the dynamics of the ureters I know absolutely nothing, and as regards this in the case of the bladder, I have very little information. Such as it is, however, is rather interesting.

The greatest expulsive power of the bladder, including the abdominal pressure, voluntary and involuntary, I found to be in the case of a healthy adult, aged 26 years, 4 to 4½ in. hg. The position, erect or lying on the side or back, made little or no difference.

In a female, aged 35, affected with diabetes insipidus, it was found to amount to 38 in. water. In three cases of paraplegia which I tested carefully as here, I could eliminate the abdominal pressure, obtaining the expulsive force of the bladder itself, I found it to amount to 20, 24, and 30 in. respectively. In all cases the rise of the columns of hg. or water was the same, whatever the amount of the urine contained, showing that the muscular fibres of the walls of the bladder, like those of other hollow muscular organs, as the

uterus, though they lose force in becoming shorter, yet compensate for this by acting on a fluid contained within a sphere of smaller radius. This fact I had many opportunities of observing in the third of the paraplegic cases just mentioned. The bladder was somewhat irritable, and I had to wash it out at intervals. While doing so I frequently found that after expelling its contents it would remain so firmly contracted round the catheter for 30 seconds or longer at a time as not to admit a single drop of water even with a head of 3 ft. This is specially interesting when considered in connexion with the paper on "Hydronephrosis,"[1] in which I endeavoured to show that the increased frequency of micturition which occurs in irritable bladders will cause dilatation of the ureters and renal pelvis by damming up the secretion in the ureters.

In this paper, then, I would draw attention to the following considerations:—

1. The great importance of the *proof* as distinguished from the *ultimate* tenacity.

2. That the secretion pressure of the urine (2·4 in. hg.) is not sufficient to cause any marked dilatation of the ureter in cases of sudden complete obstruction, but is so where the obstruction is partial or gradual.

3. That the secretion pressure of the urine is sufficient to burst the bladder in cases of sudden complete obstruction.

4. That the contractile force of the bladder is greater than that of the secretion pressure of the urine.

In conclusion, I have to express my thanks to Mr Scott Lang for the most valuable assistance I have received from him whilst writing this paper.

[1] Page 41.

NOTE ON THE TENACITY OF TISSUE.

As a subject for investigation the purely physical properties of the tissues have always been specially attractive, and consequently our knowledge of them is very considerable. As the methods of investigation have become more plentiful and precise this knowledge has correspondingly increased in amount, and at the same time become more exact.

Of these properties that of the cohesion or tenacity of tissue has been, for long, very well known to us. By the work of Wertheim, Weber, Valentin, Matthews Duncan, etc., we have been made well acquainted with it, and from this knowledge much valuable information, as regards the processes which take place in the animal body in health and disease, has been gained.

On examining the work of these different observers it will be found, however, that their results determine, for the most part, the *ultimate tenacity* rather than the *proof tenacity*, of the various tissues experimented upon. The distinction between these terms may be thus explained: By *ultimate tenacity* we understand the utmost pull which a portion of tissue of known dimensions sustains just before being torn asunder. By *proof tenacity* we mean the pull which such a portion can sustain without injury, or that pull, any excess of which, though not sufficient to tear it asunder at once, would ultimately do so if its application were often enough repeated or long enough continued.

Thus, while as regards the ultimate tenacity, we have only to consider the effects of the pull or weight, in estimating the proof tenacity, we must add to those the effects of time.

That the latter is the most important, in a physiological or pathological sense, is very evident, the arterial and venous urinary and other secretion pressures, all causing strains which have to be borne by the various tissues, continuously or intermittently for prolonged periods.

Whilst making some investigations into the tensile strength of the ureter and urinary bladder in the human subject and in animals, the results of which have been given at page 57, I was unable to find, on examining the literature of this subject, any experiments in which the effects of time were taken into full consideration. To procure information on this point, I made a series of observations on the small intestines and bladders of sheep and on the ureters and bladder in the human subject, and so obtained data on which to form conclusions as regards the amount of force necessary to cause dilatation and rupture of these structures. The experiments on the intestines of sheep which I then made, although warranting the conclusions I drew from them, were not sufficiently numerous to give thoroughly accurate results. I have since repeated and extended them, and the results are given in the Table (p. 70).

The tissue tested, viz., the small intestine of the sheep, was selected solely on account of its being most convenient for experimental manipulation.

The results obtained show that strains or weights applied to this tissue, less than the ultimate tenacity or bursting weight, in the proportion of $\frac{2}{3}$, $\frac{7}{12}$, and $\frac{1}{2}$, will cause rupture in certain definite periods of time.

The portions of intestine, each 1 foot in length, were tested by being fixed to wooden cores, by which means the weights could be conveniently applied.

On attempting to form an estimate of the average ultimate tenacity, I found that this could only be obtained approximately. This was owing to the fact that the strength of the gut varies at different parts. Thus with a piece of intestine 10 feet in length the bursting weights of the several successive portions, each 1 foot in length, presented the following variations in ounces,—40, 50, 55, 53, 50, 60, 55, 59, 52, and 49 respectively. In order to obtain as accurate an estimate as possible, I therefore proceeded in the following way.

For each experiment 3 feet of intestine was taken and divided into three equal portions. By means of a spring weigher the weights required to rupture the two outer portions were obtained, and the average of these two was taken as representing the ultimate tenacity of the middle portion. To these middle portions the various fractions of these weights were applied, and the time which elapsed between their application and the rupture of the gut noted.

As will be seen by a reference to the 10 bursting weights given above, the estimates of the ultimate tenacities obtained in this way are very uncertain. For instance, while in the first three the estimated tenacity would be $47\frac{1}{2}$, the real one was 50; in the second three the estimated would be $56\frac{1}{2}$ and real 50; in the last three the estimated 54 and real 52.

By making our experiments sufficiently numerous, however, we can, I think, obtain fairly trustworthy results; while, at the same time, we have explained for us the occurrence of the very great variations as to time which the Table shows.

The greatest care was taken to have the condition of the portions the same as regards moisture, etc., and in every case the experiments were made within 18 hours of the death of the animal.

Table of Experiments on Proof Tenacity.

$\frac{3}{4}^2$			$\frac{7}{12}$			$\frac{1}{2}$			
Number of Experiment.	Bursting Weights (Estimated).	Time.	Number of Experiment.	Bursting Weights (Estimated).	Time.	Number of Experiment.	Bursting Weights (Estimated).	Time.	
	oz.	min.		oz.	min.		oz.	h. min.	
1	36	¼	51	42	5½	101	23	1·30	
2	30	½	52	42	14	102	24	·20	
3	52	½	53	44	10	103	37	3·30	
4	42	1	54	48	16	104	26	7·30	
5	40	3	55	52	49	105	24		Did not burst in 14 hrs.
6	54	1½	56	55	81	106	44	7·0	
7	60	1	57	56	14	107	45	1·30	
8	60	2	58	54	8	108	43		,, ,, ,,
9	52	5	59	49	55	109	40	2·35	
10	21	6¾	60	49	100	110	35	3·0	
11	36	10	61	44	25	111	37	12·8	
12	36	14	62	42	4	112	40	4·5	
13	47	15	63	46	23½	113	44	7·23	
14	48	5½	64	44	5	114	54	5·30	
15	56	20	65	44	120	115	56	1·47	
16	48	11	66	46	25	116	54	·58	
17	36	6½	67	54	180	117	47	1·10	
18	40	20	68	54	13	118	52	·15	
19	30	8	69	28	25	119	48		,, ,, ,,
20	48	1½	70	30	13	120	48	1·31	
21	26	1½	71	56	31	121	46	3·30	
22	29	51	72	58	7	122	44		,, ,, ,,
23	33	3	73	53	6	123	48	3·27	
24	36	¼	74	47	120	124	60	·17	
25	37	¼	75	58	14	125	56	1·15	
26	37	9½	76	64	51	126	56		,, ,, ,,
27	35	1¼	77	69	11	127	58	4·3	
28	36	3¼	78	70	6½	128	60	3·4	
29	36	8	79	68	12	129	30	·41	
30	33	¾	80	63	7	130	30	11·12	
31	37	1	81	41	16½	131	34	2·53	
32	39	2	82	39	78	132	32		,, ,, ,,
33	39	50	83	37	102	133	39	·46	
34	48	¾	84	42	56	134	28	5·40	
35	39	9	85	60	8	135	26		,, ,, ,,
36	32	1	86	51	11½	136	30	·25	
57	27	27½	87	61	53	137	33	1·52	
38	28	23½	88	65	18	138	32	7·14	
39	27	22½	89	61	33	139	32	1·48	
40	27	7½	90	62	68	140	34	2·3	
41	24	6½	91	63	150	141	34	·53	
42	26	¼	92	67	51	142	34	2·42	
43	25	6½	93	70	36	143	44		,, ,, ,,
44	48	¼	94	62	22	144	52	·17	
45	45	7¼	95	59	110	145	34	·18	
46	58	¼	96	53	144	146	44	·27	
47	52	¼	97	52	38	147	42	2·0	
48	48	¼	98	55	34	148	44	9·5	
49	52	1	99	53	86	149	45		,, ,, ,,
50	52	36	100	53	45	150	41	8 9	
Average,		8 nearly.	Average,		44 nearly.	Average,		247 nearly.	

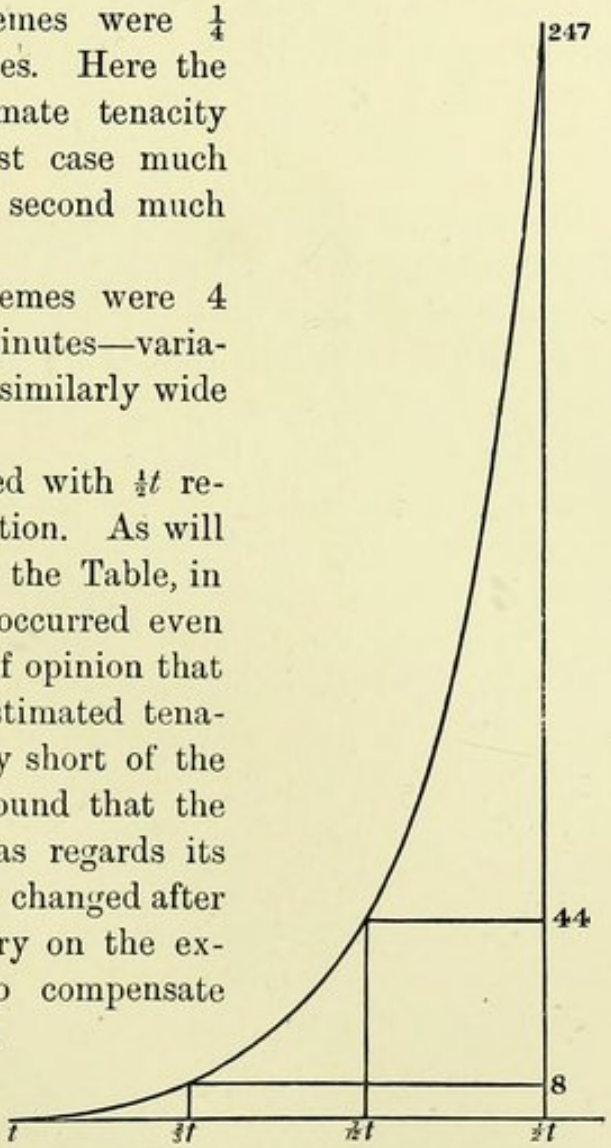
We thus see that with the *ultimate tenacity* or bursting weight t,

$\frac{2}{3}t$	will cause rupture in about	8	minutes
$\frac{7}{12}t$	„	44	„
$\frac{1}{2}t$	„	247	„

With $\frac{2}{3}t$ the extremes were $\frac{1}{4}$ minute and 51 minutes. Here the estimate of the ultimate tenacity had been in the first case much too high, and in the second much too low.

With $\frac{7}{12}t$ the extremes were 4 minutes and 180 minutes—variations evidently due to similarly wide estimates.

The average obtained with $\frac{1}{2}t$ requires further explanation. As will be seen by reference to the Table, in nine cases no rupture occurred even after 14 hours. I am of opinion that in each of these the estimated tenacities were considerably short of the real ones, and as I found that the condition of the gut as regards its cohesion almost always changed after a time,[1] I did not carry on the experiments longer. To compensate for those which did not burst, I have deleted nine others which gave way in correspondingly short



t = ultimate tenacity or bursting weight, 8, 44, and 247 = time in minutes.

[1] Wertheim found an *increase* in the tensile strength of the fibrous tissues for some days after death.—*Annales de Chimie et de Physique*, tome xxi. p. 385.

periods,[1] as in these we may conclude that the estimated tenacities were too high.

The average, 247, is thus obtained from only 32 experiments. It agrees, however, fairly well with the result obtained theoretically. Thus, with the weights for the abscissa and the time in minutes for the ordinates, if we regard the curve (p. 71) obtained as a logarithmic one, we get

$$\frac{(44)^2}{8} = 242.$$

The increase obtained by experiment, viz., 247, is doubtless due to the increase in the tensile strength of tissue which occurs for some time after death.[2] This same consideration also led me to believe that experiments with less than $\frac{1}{2}t$ would not afford trustworthy results. I therefore did not carry the investigation further.

[1] Marked in italics.

[2] Wertheim, *ibid.*

TRANSUDATIONS AND EXUDATIONS.

In the following paper I propose to give the results of some inquiries which I have made on the composition of various transudations and exudations. The transudations were the pleural, peritoneal, cutaneous, and cerebral; the exudations were the contents of abscesses, inflamed bursæ, and inflamed joints. In these inquiries my object has been to discover how much the composition of these fluids is influenced by simple physical processes, more especially by filtration. The action of this in the case of the transudations is obvious, they being simply *filtrates*. The exudations, on the other hand, have not such a simple explanation, but with them also I hope to demonstrate the importance of taking this process into consideration.

Transudations.

Analyses of these occurring in one individual, and due to a general cause, as heart or kidney disease, have been made by several observers. Schmidt[1] gives (page 146) in 1000 grammes transudation—

	Pleura.	Peritoneum.	Cerebral ventricles.	Sub-cutaneous.
Albumen	28·5	11·3	8·0	3·6

He also notes (page 145) that the proportion of the mineral constituents remains nearly constant, about 8 p.m. Hoppe-

[1] "Characteristik der Epidemischen Cholera," 1850.

Seyler[1] (page 257) gives, in a case of general dropsy, in a patient suffering from Bright's disease and bladder catarrh, the following in 1000 parts transudation—

	Pleura.	Peritoneum.	Subcutaneous.
Total solids . . .	42·41	32·32	17·83

He also notes that the proportion of salts remains constant, about 7 or 8 p.m.

Lehmann,[2] in the dropsical transudations in the dead body of a drunkard with granular liver, found in 1000 parts transudation—

	Pleura.	Peritoneum.	Cerebral ventricles.
Albumen	18·52	10·44	5·64

and makes the same observation as regards the proportion of salts. Similar investigations have been made by many others,[3] — Heller, Scherer, Simon, etc.,—and their results, though not obtained from fluids effused under identical conditions, coincide in the main with those given above. I do not, however, give them, because the fluids which I wish here to consider are those which occur under conditions precisely identical, *i.e.*, which are transuded in one individual, simultaneously, and due to a general cause.

On examining these results we find, first, that the amount of mineral matter remains nearly constant in all cases; secondly, that the amount of organic matter presents great variations in individual cases; thirdly, that in the different transudations a distinct relationship can be detected as regards the proportion of organic matter, the pleural being richest in it, the subcutaneous poorest, the peritoneal occupying an intermediate position. Further, from the observations

[1] Virchow's *Archiv*, vol. ix.

[2] *Physiological Chemistry* (Cavendish Society), vol. ii. page 316.

[3] Becquerel and Rodier differ from others in stating that the peritoneal fluid contains the largest proportion of albumen (Speer's Translation, page 479).

of Schmidt, etc., we may conclude that the fluid of the cerebral ventricles has always a larger proportion of solid residue than the subcutaneous transudation. This relationship in the proportion of organic matter has been expressed by Schmidt in the following rule:—"The quantity of albumen (equal to total solids) contained in a transudation is dependent on the system of capillaries through which the transudation flows."[1] What I now propose to do is to show that, in order to find an explanation for this relationship, we will obtain considerable information by studying the physical conditions existing simultaneously in connexion with the various capillary systems. In the first place, then, if a portion of a fluid holding organic matter and salts in solution is made to filter through a membrane, what changes does it undergo?

On this point Hermann says,[2]—"True solutions pass through the medium unchanged. Viscid fluids, such as solutions of albumen, starch, and gum, filter less perfectly, a portion only of their contained substances, in amount varying with the pressure, being able to percolate. If, indeed, the pressure is very slight, the whole is retained on the filter. Under slight pressure, therefore, the blood loses, by filtration, water, salts, sugar, etc., which form true solutions, while under a greater pressure it is deprived of more or less of its albumen, fibrinogen, etc."

Thus, then, we see that the amount of organic matter passing through varies directly as the pressure—*i.e.*, that with a high pressure a large quantity of organic matter, and with a low pressure a small quantity, will filter through. These considerations, then, explain clearly why the proportion of salts remains constant; and from them also we might readily conclude that the difference in the proportions of solid constituents effused by the different capillary systems

[1] Schmidt's *Epidem. Cholera*, and Lehmann, page 315.

[2] Professor Gamgee's Translation (second edition), page 122.

is due to the blood in these capillaries being subjected to different degrees of pressure.

That this occurs, and how this is effected, is what I have to demonstrate. Before doing so, however, let me mention a few other considerations.

We might suppose, for example, that one reason why the fluids transuded into the pleura, peritoneum, and cerebral ventricles were thicker (if I may use the term) than that effused into the subcutaneous tissue is, that in the case of the former the fluids are mixtures of transudation from the capillaries and secretion from the serous membrane, whereas in the latter it is a transudation alone. This, however, although it must be taken into consideration, will not explain why the peritoneal fluid is thicker than that of the cerebral ventricles, and the pleural thicker than either. Again, we might suppose that the blood varies in composition in the different capillary systems, more especially, for example, in the case of those of the abdominal cavity, where we should expect that the performance of the function of absorption is likely to bring this about. Or, again, it may be that the pressure varies in the different capillaries, that the capillaries vary as regards the permeability of their walls, or that the absorbent vessels may be more numerous, and the absorbent function more active, in connexion with one of the systems than another. Further, we must remember the connexion of dropsies with nervous lesions.[1] All these are possible agents in producing the variations in the chemical composition of the fluids which we are discussing, and, as such, must be taken into consideration. I trust, however, that I may succeed in demonstrating that, in the physical conditions operating in the different cavities, we have by far the most important factors.

[1] Lehmann says, page 318, "that the transudation will be richer in albumen in proportion to the slowness with which the blood passes through the capillaries."

Let us, therefore, neglecting the special secretory action of the serous membranes, suppose that we have the following alike conditions existing in the various capillary systems, viz., same composition of contained blood, equal permeability of capillary walls, equal activity of absorbents. Let us further suppose that the blood pressure in the interior of the capillaries is the same, and allow that the fluid transuded into the subcutaneous tissues (which, as we have seen, contains the smallest proportion of solid residue) is the result of this pressure alone. Why is it that transudations into the pleura contain organic matter in so much greater abundance? To understand this, we must remember that the lung, with its contained air-cells, blood-capillaries, etc., is an elastic structure, and that it is in the thorax always more or less stretched. The amount of this stretching, or, in other words, the collapsing power of the lung, varies, of course, according to the amount of air contained in it.

Hutchinson [1] estimates it in two cases, as follows:—

I.—(*a.*)	Containing breathing volume of air .	8·2 in.	water.
(*b.*)	„ vital capacity „ .	17	„
II.—(*a.*)	Containing breathing volume of air .	10	„
(*b.*)	„ vital capacity „ .	20	„

Donders' estimate is 7·5 mm. hg. (less than 4 in. water) at the end of expiration, and 9 mm. hg. (about 4½ in. water) at the end of inspiration, and 30 to 50 mm. hg. (about 15 to 21 in. water) after a forced inspiration.

We must further remember the very great force which the inspiratory muscles are capable of exerting, the force varying, as Hutchinson has shown, from 1·7 to 7 in. hg. Arguing theoretically, therefore, from the enormous aspiration which the thorax can produce, we might easily imagine that, in cases of increased pressure in the lung capillaries, or still more in cases of obstruction to the entrance of air into the lungs, we should have the transudation of fluid into the

[1] *Cyclopædia of Anatomy and Physiology*—"Thorax."

pleura correspondingly favoured. To ascertain this point clearly, however, I have tested it experimentally, and the following I give as one of my most successful results:[1]—

The lungs and trachea of a freshly killed cat were taken, washed, and emptied as much as possible of blood. To the pulmonary artery a tube was connected by a canula, this tube communicating with a reservoir of water. The pulmonary veins were ligatured, and it was so arranged that the lungs could be placed under the receiver of an air-pump, the trachea communicating with the outer air by a tube. Water with a head of 13 in. was then passed through in quantities of 100 cc. at the time, and the results noted.

The First 100 cc. passed into lungs in 45 min.,	and of it there transuded into air-passages	8 cc.;	transuded through pleura,	50 cc.;	the rest in the lungs.		
The Second in 20 min.,	"	19 "	"	50 "	"	"	
The Third in 10 min.,	"	35 "	"	18 "	"	"	
The Fourth in 5 min., (Artificial respiration by air-pump.)	"	35 "	"	15 "	"	"	
The Fifth 100 cc. passed into lungs in 51 min., (Artificial respiration stopped.)	"	5 "	"	90 "	"	"	
The Sixth 100 cc. passed into lungs in 12 min., (*Trachea tied*; artificial resp. applied.[2])	"	65 "	"	25 "	"	"	
The Seventh 100 cc. passed into lungs in 10 min.,	"	— "	"	30 "	lungs extremely œdematous; trachea and bronchi distended with fluid.		

From these experiments we obtain precise information on two very important points: first, that where no hindrance exists to the entrance of air into the air-cells of the lungs, the inspiratory act will favour the transudation of fluid from

[1] *Id est*, In which I consider that the natural conditions were most closely simulated. An interval of about two hours occurred between the second and third quantities (see Table), as I was unfortunately called away at the time.

[2] In applying artificial respiration, care was taken to simulate the positive pressure of expiration as well as the negative one of inspiration; the lungs, however, were constantly distended, *i.e.*, scarcely diminished in size at expiration.

the lung capillaries into the pleural cavity; and, secondly, that when the air-passages are obstructed, this same inspiratory force is, for the most part at least, expended in causing this fluid to transude into the air-cells. In this latter case there will, however, be transudation into the pleura from the capillaries of the costal parietes. Clinically, therefore, we should expect that, in cases of Bright's disease, or of obstruction to the circulation alone, as in heart affections, *hydrothorax* will result, whilst in cases where the entrance of air into the air-cells is impeded, as in bronchitis, pressure on trachea by tumours, croup, etc., *œdema of the lungs* is, in addition, most likely to occur.

But it is necessary to examine the mechanism by which, as we have seen, hydrothorax is produced a little more minutely. Is the aspiration produced by the inspiratory act sufficiently powerful to cause the transudation into the pleura of a fluid much thicker than that transuded into the subcutaneous tissue by intra-capillary pressure alone?

If the fluids which we have examined had been from cases with obstructed air-passages, we might have had little doubt as regards this, the inspiratory power amounting, as we have seen, to from 1·5 to 7 in. hg. But the fluids which we are considering were taken from cases (Bright's disease, etc.) where we may assume that there existed no marked obstruction to the entrance of air into the lungs.

The aspiration power, therefore, by which transudation into the pleura is produced will depend on the elasticity of the lungs alone, and the estimates of this, as we have seen, never exceed 20 in. water. Further, we must remember that, as fluid collects in the pleura, the lungs will correspondingly retract, and so this power will be gradually reduced in force. The results of Homolle[1] also show that this aspiration power is inconsiderable in amount. In his cases of pleuritic

[1] *Revue Mensuelle de Médecine et de Chirurgie*, February 1879: "De la tension intrathoracique dans les épanchements pleuraux."

effusion, the intrathoracic tension was tested by a manometer, and the greatest negative tension obtained was (Case IV.) 38 mm. hg. in inspiration, and 33 mm. hg. in expiration.[1] This is about 19 and 16 in. water, *i.e.*, less than the collapsing force of the lungs, as estimated at the end of the fullest possible inspiration. This aspiration power, therefore, is never very great; and it will be noted in the Table (fifth quantity) that transudation took a long time to occur. On the other hand, we have to remember, however, that the intra-capillary pressure must be very small; and it is with this that we must compare it when we wish to judge of its effects. What the intra-capillary pressure is we have no precise means of discovering, but when we remember the smallness of the venous pressures, and that the sectional area of the systemic capillaries may be estimated as being 800 times that of the aorta,[2] we can, I think, admit that this aspiration, though equalling no more than 10 or 12 in. water, will have the effect of causing the transudation of a somewhat larger relative quantity of organic matter.

On this subject further experimentation seems desirable, as the results hitherto obtained have not, as far as I am aware, been very definite. Dybkowsky[3] endeavoured to cause an increase in the pleural fluid by ligaturing the aorta

[1] The greatest positive pressure which Homolle obtained was (Case II.) 15 mm. hg. at the end of expiration. From these data we can readily understand how, unless in cases of paralysis of the intercostal muscles, as in empyema, "bulging of the intercostal spaces" is so rarely seen.

[2] Of course, I do not propose this as an estimate of the capillary pressure. Küss estimates the systemic capillary pressure as $\frac{12}{100}$ of the atmosphere, about 36 or 48 in. water. The pulmonary capillary pressure would be considerably less. — *Manual of Physiology* (Dr Amory's translation), page 105. By experiment (N. v. Kreiss) it has been estimated, in the capillaries of the hand, at 38 mm. hg.—Landois' *Physiologie*, page 173.

[3] Ludwig's *Arbeiten*, 1866: "Ueber Aufsaugung und Absonderung der Pleurawand."

below the diaphragm, and by causing paralysis of the thoracic vaso-motor nerves. On killing the animal after a time no fluid was found in the pleura.

He found, however, that fluid introduced into the intercostal tissue always made its way into the pleural cavity.[1]

Apart from this, however, there is another reason why pleural fluid should be thicker than that found in the subcutaneous tissue. Hoppe-Seyler has shown that, in cases of ascites due to cirrhotic liver, the more the fluid increases in amount, the more concentrated does it become.

The explanation of this is, that by the lymphatics and peripheral branches of the systemic veins absorption is constantly going on, and as the water is the constituent of the transudation which is absorbed in most abundance, it (the transudation) is consequently constantly becoming more concentrated. Thus, then, concentration will vary directly with the rapidity of absorption, and, as in the chest the negative pressure of inspiration is immediately followed by the less negative or slightly positive (in forced expiration) pressure of expiration, we can easily understand how this process will be favoured.

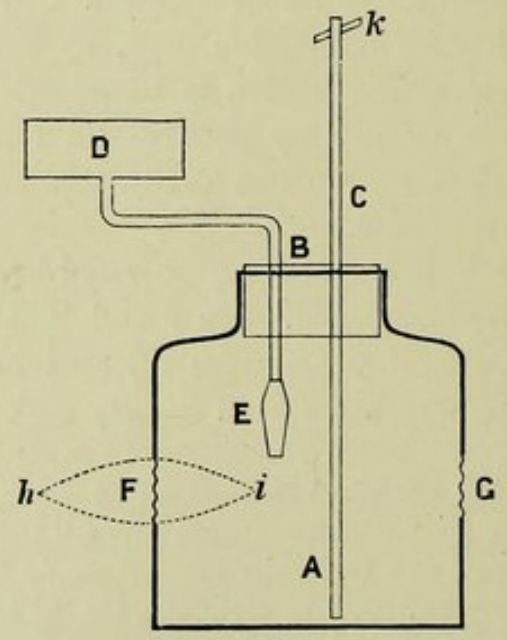
This hypothesis is directly corroborated by Dybkowsky in the article before mentioned. He found that with solutions of Berlin blue, etc., if these were injected into the pleural cavity of a living dog, absorption rapidly took place, but that

[1] I am indebted to my friend Dr Graham Brown for affording me an opportunity of the perusal of Runeberg's paper, "Ueber die Filtration von Eiweislösungen durch thierische Membranen." Runeberg's results are contrary to those indicated by Hermann, etc., as he found that with increased pressure the amount of albumen which filtered through was not materially increased. How the respiratory movements will favour the transudation of albumen, however, may be imagined from his conclusion No. 8—"Der albumingehalt des Filtrats ist bei demselben Druckgrad beträchtlich grösser, wenn die Membran einige zeit vorher vom Druck befreit war, als wenn sie unter Einwirkung des Drucks gestanden hat."

this did not occur if the animal were killed immediately after the injection. He also found that the rapidity of absorption increased with the activity of the respiratory movements. The transudation into the pleura, therefore, has a larger proportionate amount of solid residue than that of the subcutaneous tissue, as the result of the respiratory acts.

Abdominal Transudation.—This, as we have seen, is more concentrated than the subcutaneous, and less so than the pleural. When we look for an explanation of this, we shall, I think, discover that we have in the abdomen processes at work similar in kind to those which we have studied in the thorax, but less in degree.

We can best understand this by the diagram. A is a



jar into the stopper of which two tubes B and C are fitted; B communicates above with a reservoir of water D, and below with a membrane E, through which water will readily filter; C is an open tube extending to within half

an inch of the bottom of the jar A; F is an india-rubber diaphragm which can be moved at will towards *h* or *i;* G is also a diaphragm which can be made rigid or flexible. Supposing now that the diaphragm F is placed as at *i*, and water poured into the reservoir D, it will filter into the jar A, and this filtration will continue till we have in tube C a column of water equal in height to level of that in reservoir D.

If, now, the diaphragm G be perfectly flexible, any movement of the diaphragm F to *h* will cause no alteration of the level of the column of water in tube C. If, on the other hand, this same movement be made when the diaphragm G is rigid, a diminution of pressure will be produced in the jar, and so the level of the column of water in C lowered. But if this lowering be prevented by clamping the tube C at *k*, the negative pressure thus produced will be expended in aspirating water through the filter into the jar A, so that when the diaphragm F is again placed at *i*, the clamp *k* having been previously removed, the column of water in C will rise higher than before. The extent to which this increase in rise will occur will depend, first, on the amount of force with which, and distance to which the diaphragm F can be moved from *i* towards *h*, and back again, and, secondly, on the rigidity of the diaphragm G.

Supposing now that the jar A represents the abdominal cavity, the filter E is a capillary vessel containing blood at an abnormally high pressure, F is the diaphragm, and G the more or less flexible anterior abdominal wall, we can readily understand how the expiratory ascent of the diaphragm, due to the elastic recoil of the lungs, will cause "aspiration" in the abdominal cavity, and how this aspiration may be diminished or completely abolished, either, in the first place, by anything which interferes with the movement of the diaphragm, as emphysema, etc., or, secondly, which renders the anterior abdominal wall more flaccid, as repeated preg-

nancies.[1] Of course, it will also be abolished in cases of large effusion.

In this aspiration, therefore, we have to recognise one reason for the greater concentration of the abdominal as compared with the subcutaneous transudation; and when we compare the cause of this aspiration in the abdominal cavity with that which produces it in the pleural, we can further understand why its concentration should not be so great as in the latter.

This abdominal aspiration I have endeavoured to estimate experimentally, and the simplest way of explaining the method which I followed is to say that I substituted the abdominal cavity of a rabbit for the *jar* figured in the diagram. This I did by fixing two metal tubes through the anterior abdominal wall, one tube communicating with a reservoir of water raised about two inches above the level of the animal's body, and the other communicating with a straight tube as at C. Having inflated the lungs so as to represent full inspiration, I allowed water to flow into the abdomen till it rose in the upright tube to the same level as that in the reservoir. Having then clamped this tube, I allowed the air to escape from the lungs. About three drachms of water at once ran into the abdominal cavity.[2]

In abdominal transudations we have also to recognise, as

[1] Compare Matthews Duncan on the "Retentive Power of the Abdomen"—*Researches on Obstetrics*, page 309.

[2] These considerations seem to show that this abdominal aspiration will occur during expiration alone. In connexion with it, however, it is interesting to note that Professor Mosso, of Turin, in a paper—"Ueber die Gegenseitigen Beziehungen der Banch und Brustathmung"—in the *Archiv für Anatomie und Physiologie* (His, Braune, and Du Bois-Reymond) for 1878, found that during sleep there was, coincident with the first part of the inspiratory act, a drawing in of the abdominal walls. This will represent a tendency to an aspiratory act. Matthews Duncan considers that the retentive power exists during both inspiration and expiration.

in the pleural, the existence of conditions favouring absorption. Here, again, we have a similar alternation of tension —the negative or positive tension of expiration followed by the less negative or more positive one of inspiration. Indeed, at first sight we might say that the conditions for absorption in the abdominal cavity are more favourable than in the thoracic, as the pressure in ascites often amounts to 6 in. or 7 in. water (Hoppe-Seyler[1] mentions one of 25 mm. h.; Stone[2] gives as an average 5 in. to 7 in. of water), whilst in pleural transudation the pressure is negative.[3] But although great pressure will promote absorption, the experiments of Dybkowsky, previously referred to, demonstrate that an alternation of negative and positive tension is still more favourable.

It is important, also, to bear in mind that we have, in ascites from cirrhotic liver, conditions differing somewhat from those present when the fluid is due to renal or cardiac disease alone. In the latter class of cases, transudation will occur from both the portal and systemic vessels; in the former it will occur from the portal vessels alone, the (blood) pressure in the systemic capillaries and absorbents remaining normal, or, perhaps, being even slightly diminished. This will certainly have the effect of greatly promoting the absorption of the watery part of the transudation. We must further remember that, in cirrhosis of the liver, the greater the pressure in the portal system the more rapidly will anastomoses form between its branches and those of the systemic circulation,[4] and when this is established, not only will the fluid already transuded be still more rapidly absorbed, but by the free passage of portal blood into the systemic

[1] Virchow's *Archiv*, ix., page 248.

[2] *Lancet*, 30th August 1879.

[3] See *Pneumono-Dynamics*, by G. M. Garland, M.D. New York.

[4] For details as to this anastomosis, see Frerichs *On the Liver*, vol. ii. p. 39 (New Sydenham Society's Translations).

vessels further transudation will be completely prevented. This is in the main the explanation of the immense improvement which one occasionally sees in cases of cirrhosis of the liver with ascites. Thus I have under observation a man who, within two years, has been tapped nineteen times for this malady, each time about 500 oz. being taken off. Although his liver is gradually diminishing in size, he is now not only completely recovered from his ascites, but is able to do easy work.

The *cerebral transudation* occupies, as regards concentration, a place between the abdominal and subcutaneous. About it I have little more to say than that, when we remember the unyielding osseous case which contains the brain, and the almost complete incompressibility alike of blood, ventricular fluid, and cerebral substance (the changes in volume which this last undergoes being insignificant, even with a pressure of 180 mm. hg.), we fail to find the existence of conditions similar in any way to those which we have noted in the thorax and abdomen.

The fluid found in the cerebral ventricles in Bright's disease, etc., is probably an increase of secretion rather than a transudation. Schmidt says, "Die krankhafte Transudation durch die Hirncapillaren ist als reine quantitive Steigerung der normalen (Cerebrospinalflussigkeit) zu betrachten;" and in connexion with this he points out that, whilst the proportion of salts is the same as that found in the other transudations, yet when these salts are examined, it is found that there is in them an increase in the relative proportions of potassium and phosphates to sodium and chlorides.

Exudations.

Those which I have studied are the contents of abscesses, inflamed bursæ, and inflamed joints, and the results I have summarized in the Table.

In each case I have estimated the following points:—Duration of the affection; amount of fluid; its tension; specific gravity; per cent. of solids; per cent. of ash. In the last column I have added explanatory remarks, etc. Before we examine these in detail, let me describe the means by which the *tensions* were obtained, these being the only data, of the means of estimating which a detailed account is necessary. This was done by introducing into the abscess cavity the largest-sized trocar and canula of a Potain's aspirator. To the branch tube of the canula a piece of glass tubing of small calibre, and containing a few drops of carbolic solution, was connected. On withdrawing the trocar and closing the tap, the fluid rose in the tube, and, care being taken to keep this perpendicular, the height to which the column of water rose was measured off, and so the pressure was obtained.

I am well aware that this is not at all an accurate method, and that the smaller the collection of fluid, the tension of which is to be tested, the greater will be the deviation from the real tension. On the other hand, I am not aware of any means by which absolutely accurate results could be obtained in all cases,[1] and I believe that by the simple method which I have employed, and by making due allowances, we can obtain results sufficiently trustworthy for our purpose, *i.e.*, we can say whether a pressure is high or low. Another source of error is, that the rise in the tube may be prevented by flakes of lymph, or impeded by the viscidity or thickness of the fluid. This must also be noted and allowed for.

Let us now examine our results, and endeavour to find if we can detect any relationship between them.

First, we might expect that the tension will vary directly with the acuteness of the process, and *vice versâ*. A reference to the Table will show that, although a relationship of this kind does exist, yet it is not very well marked. For this

[1] *E.g.*, How could the tension be obtained in a *whitlow?*

there are obviously various reasons, *e.g.*, one abscess may be situated in lax tissues, and another in dense; a chronic abscess may have become acute, or an acute chronic, etc.

Secondly, according to the hydrostatic law, the tension in small abscesses, etc., should be greater than in large ones, in order that their distension should occur. Here, also, the relationship is not well marked, owing to the fact that the abscesses have not the same degree of acuteness, and that they are not in tissues of the same density.

Thirdly, can we trace a connexion between the tension and the composition of the fluid? On this point more definite results are, I think, obtainable. As regards the mineral matter, a glance shows that its proportion remains fairly constant—about ·7 per cent. This is, of course, just what we might expect. The organic matter, however, requires more detailed consideration. What is the effect of pressure on a secretion, supposing that pus is such?

We know in the case of the kidney that, if to the secretion of urine a counter-pressure be applied, as occurs, for example, in the case of a calculus in the ureter, the secreted urine becomes more and more watery. We should, therefore, on this supposition, expect that with high tensions the contained fluids will be correspondingly deficient in solids.

To obtain information on this point from the Table we must, however, be sure that we are dealing with fluids identical in character; and that these will vary, if from different sources, is evident. Of this Nos. 6, 14, 18, and 20 are perhaps the best examples. These fluids are thin, being made up to a large extent of slightly altered bursal or joint secretion. On the other hand, pus derived from abscesses containing portions of sloughed tissue will be comparatively thick in composition. Bearing this in mind, and looking, therefore, at what we may call the true abscesses, we cannot, I think, fail to discover that the opposite of what we might have expected holds—viz., that high tensions are associated

TABLE.

Those within brackets are abscesses which were re-tapped.

	Duration	Quantity.	Tension.	Sp. gr.	Solids.	Ash.	
			inch		per cent.		
[1	3½ mo.	4½ oz.	7	1024·8	9·76	·6	Abscess of back; laudable pus.
2	9 days	2 ,,	5½	1024·3	8·5	...	Pus on standing twenty-four hours, one-fourth liq puris.
[3	6 ,,	1½ ,,	5	1023·5	8·1	...	Pus on standing twenty-four hours, one-half liq puris.
4	6 ,,	25 ,,	15	1030·5	11·24	·7	Abscess of thigh; laudable pus.
5	2½ mo.	10 ,,	5½	1029·2	9·8	·7	Abscess on gluteal region
6	3 wks.	2 ,,	7	1018·2	6·3	·7	Inflamed bursa over elbow; clear yellow fluid.
7	3 ,,	1 ,,	6	1024·9	9·3	...	Strumous abscess of neck.
8	1 mo. (?)	10 ,,	3	1034·0	11·8	·84	Abscess of shoulder-joint; bone; had been opened one year previously.
9	10 wks.	12 ,,	6	1025·0	9·5	·7	Abscess on hip after scarlatina.
[10	6 ,,	1 dr.	7½	...	10·3	·7	Abscess on back.
11	5 days	3 ,,	...	1022·5	7·5	·7	Clear yellow fluid.
12	5 ,,	2¼ ,,	3	...	7·0	·6	Clear yellow fluid.
[13	11 ,,	3½ ,,	3½	...	...	...	Turbid fluid, depositing pus corpuscles.
14	2 wks.	2 oz.	10	1022	7·2	·6	Inflamed knee-joint; fluid glairy, and slight deposit of pus corpuscles on standing.
[15	2 ,,	10 drs.	8*	1035·3	13·5	·8	Abscess on lower part of chest; pus very viscid, clotted in tube.*
[16	5 days	1 oz.	12	1033·5	12·3	·7	Pus not so viscid.
[17	5 ,,	6 drs.	10½	1033·5	12·07	...	Pus not so viscid.
18	7 ,,	3 ,,	9	...	7·9	·6	House-maid's knee; yellow fluid; slight deposit of pus corpuscles on standing.
19	1 mo.	1 oz.	5½*	1031·3	10·0	...	Abscess of glands of neck; skin reddened, and *slight oozing.**
20	1 year	1 dr.	0	...	6·0	...	Knee-joint; gelatinous degeneration; small amount of fluid.
21	6 days	3 drs.	5*	1033	12·5	·7	Abscess of thigh; pus thick, clotted in tube.*
22	2 wks.	9 ,,	5	1025	9·4	·7	Abscess on upper arm; fluid, laudable pus.
23	4 ,,	15 oz.	6	1026·4	9·6	·7	Abscess of thigh; fluid, laudable pus.
24	3 mo.	4 ,,	3	1022·0	7·8	·7	Abscess over tibia; pus thin, containing flaky matter.

with thick pus. Thus in Case 4 a tension of 15 in. is associated with pus containing 11·24 per cent. solid residue; in Case 10 a tension of 7½ in. (certainly too low an estimate, there being only 1 oz. of pus), with 10·35 solids; in Case 15 a tension of 8 in. (too low, the abscess being small and the pus extremely viscid), with 13·5 per cent. solids; Cases 16 and 19, retappings of the last, where, with tensions of 11 in. and 10½ in. (the pus being much less viscid), there was 12·3 and 12·07 per cent. solids; in Case 19, with a tension of 5½ in., there was 10 per cent. solids, but here there had been some oozing, which indicated that a higher tension had existed; in Case 21, with 12·5 solids per cent., the fluid rose only 5 in., but here the quantity was very small, only 3 drachms, and the pus was found afterwards to have clotted in the tube.

How does this occur? On the supposition that pus is a secretion, the explanation would be that with high tension, although the pus may be more watery when secreted, yet a correspondingly large proportion of the water is squeezed out, leaving the corpuscles and thickened liquor puris; and it might be further suggested, that the watery portion thus made to filter out is an important factor in the production of the œdema, which can often be detected like a ring surrounding an abscess-cavity. But it may be argued, we cannot assume that the process of pus-formation in the case of abscesses is similar to that of a normal secretion. In the latter we have a distinct apparatus for the purpose,—viz., a membrane having on the one side secreting-cells, and on the other bloodvessels,—and the secretion is the result of the elaborating action of the cells on the blood. In abscesses, on the other hand, the pus-formation and increase in size are mainly due to the breaking down of the surrounding tissues, and consequent addition of pus corpuscle and leucocytes.

On this hypothesis the association of thick pus with high tension may be explained by supposing that, in cases of high tension, the tissues immediately surrounding the abscess will

be devoid of serum, etc., as the direct result of pressure, and consequently that when they break down there will result a pus containing a very small comparative amount of liquor puris.

When we reflect, moreover, on what physiology has demonstrated to us in connexion with the influence of the nervous system on secretion, *e.g.*, the watery "chorda" saliva and the thick viscid fluid which results from sympathetic stimulation, we might be tempted to theorize further. As my subject, however, is the consideration of physical processes, and not that of pus-formation, I shall refrain.

But since high tension is associated with thick pus, does the converse hold? Is thin fluid associated with low tension? A reference to the Table will show that—excluding Cases 6, 14, and 18 for reasons before mentioned, and Case 8, for a reason to be given immediately—this association does exist. The reason why in Case 8 there was found thick pus with low tension appears to me to be that, although there has been no active secretion of pus to produce a high tension, yet, owing to the long duration of the abscess, a proportionately great absorption of its fluid part has occurred.[1]

The effects of repeated tappings are also very interesting. As observations 1, 2, and 3, 10, 11, and 12, 14, 15, and 16 show, there is diminution alike in the quantities, tensions, and amounts of solid constituents. How does this occur? The explanation seems to be, that the pus having been drawn off, there occurs an effusion of serum more or less mingled with pus corpuscles into the cavity. At the same time this cavity is rapidly contracting, so that by this process, and by the collection of serous fluid, etc., there is in a very short time a considerable amount of tension established. This tension will of itself act as an irritant; it will cause an increased secretion of pus corpuscles, which, by increasing

[1] This will explain the association of thick pus with low tension in so-called "cold" abscesses.

still further the tension, will squeeze out the watery portion, leaving within a fluid with a larger proportion of corpuscles. Thus in Cases 2 and 3, 11 and 12, 14 and 15, we find an ever-decreasing tension and proportion of solids; but in 13, where a longer period was allowed to elapse than between the previous tappings, we find the tension higher, and (although the fluid obtained was too small in quantity to admit of more accurate analysis) containing a much larger proportion of corpuscles.

We may further be allowed to suppose that this same process of secretion of pus corpuscles, consequent increased tension and squeezing out of the watery portion, will go far to explain how the fluid found in inflamed joints, bursæ, pleurisy, etc., becomes in certain circumstances transformed into pus.

Remembering that tension is an irritant, I cannot refrain from suggesting that in this fact we have a simple explanation of what otherwise seems puzzling. For instance, how is it that we sometimes find abscesses to form, increase rapidly in size, and then, after no treatment, or perhaps only the application of a poultice, or allowing a few drops of pus to escape, gradually to diminish and disappear?

From the above considerations the following may be regarded as a description of the process:—Suppose that, as the result of a bruise, a small portion of tissue in the deeper subcutaneous layer loses its vitality, it will at once act as an irritant, causing the collection around it of leucocytes or pus corpuscles. By this, tension will be produced, the irritation of which will of itself greatly increase the formation of pus. While this is going on, however, the portion of sloughed tissue will be breaking down into *débris*, so that as an irritant it will be gradually becoming less powerful, until it ceases altogether to act as such. The tension, however, will still continue to stimulate pus-formation, but it will be remembered that the larger the abscess becomes, the less

(according to the hydrostatic law) is the pressure required to distend it. Gradually, therefore, the irritation of tension will be diminishing, the secretion of pus corpuscles will therefore become less and less until the absorption of the fluid part of the secretion, which always occurs to a greater or less extent, is sufficient to abolish tension entirely. In this way no further formation of pus will occur, and absorption going on, the abscess will disappear. It will be easily understood how the escape of a few drops of pus, at the time when the tension is reaching its minimum, will hasten the process.

In connexion, further, with the relationship between the tension and the composition of the fluid, which I have been endeavouring to demonstrate, the following considerations are of interest:—

In the fluid of *acute hydrocephalus* there is always a larger proportion of solids than in that of the chronic disease.

In the *liquor amnii* there is a larger proportion of solid constituents in the early than in the later months of pregnancy.

In such tumours as *wens* the contents are much more solid when the tumours are small than when they are large.

Conclusions.

1. The pleural and peritoneal transudations have a large proportionate amount of solid constituents, because in these cavities there exist physical conditions which favour (1), the transudation of fluid into them; and (2), the reabsorption of the watery part of the fluid so transuded.

2. In the pleural cavity the above conditions are most favourable.

3. When, as the result of an abnormal composition of the blood or of increased pressure in the vessels, there occurs transudation of fluid through the lung capillaries into the lung tissue, the *inspiratory act* will favour the passage of

the fluid so transuded into the pleural cavity when the air-passages are free (hydrothorax), but into the air-passages themselves when the entrance of air into the lungs is prevented (œdema of the lungs).

4. Cerebral transudation should be regarded rather as an increased secretion (Schmidt).

5. In exudations there is a relationship between the tension and the composition of the fluid; the higher the tension, the thicker the fluid.

6. This is probably due to two conditions—(1), that in high tension a much larger proportionate amount of the watery part of the fluid is driven out into the surrounding tissues; and (2), that the tension itself acts as an irritant.

7. In abscesses of long standing the association of low tension with thick pus may be explained as being due to absorption of the watery part of the pus.

TENDON REFLEX AND CLONUS PHENOMENA.

THROUGH the kindness of Dr G. W. Balfour, I have been enabled to investigate this subject in the case of a patient, Kellie, æt. 30, in the Royal Infirmary. He has the well-marked symptoms of lateral sclerosis, *i.e.*, loss of voluntary power over the muscles of the legs, which are, however, very well nourished, and when he attempts to perform any movement with them he fails almost entirely, owing to the spasms which occur. There is, in addition, some loss of the cutaneous sensibility about the feet and ankles, and this, along with the lightning pains which he complains of, leads us to believe that other parts of the cord are involved. As a rule, the cutaneous reflexes are diminished, and the deep increased. It is the latter and the clonus which I now wish to discuss.

The ankle clonus is well marked in both legs (especially the left), either on sudden passive tension, or on tapping the muscle or tendon after gradual passive tension. In the left leg the "front tap" contraction can usually be easily induced. By means of tambours, one placed in connexion with the foot, and the other with a revolving cylinder, I have obtained tracings of the clonus, and by the chronograph I find the contractions to occur at the rate of about 6·8 per second. The movement is, as usual, perfectly uniform. By

grasping the foot, and suddenly pressing it inwards or outwards, a lateral clonus, due to clonic spasm of the peronei or tibialis posticus, can be induced. The "toe clonus" I have not been able to excite.

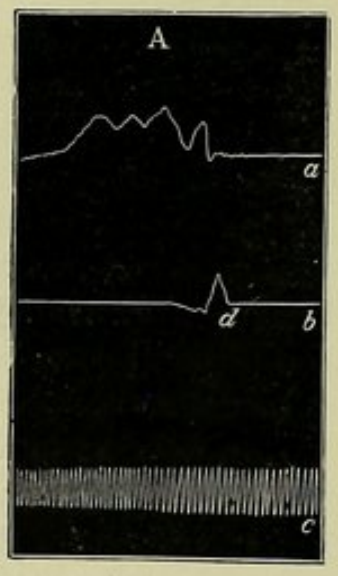
On getting the patient to stand erect, it is found that, in a certain position of the leg, as regards the trunk (the significance of which will be denoted by-and-by), a clonus of the gluteal muscles can be induced on stimulating them by sudden pressure. Its tracing shows it to be quite uniform, the time being about nine contractions per second.

I have also been able to obtain tracings of the "knee clonus," but this I propose to discuss in a more detailed manner. In his article on "Tendon Reflex Phenomena," in the *Medico-Chirurgical Transactions* for 1879, Dr Gowers notes that he has met with this knee clonus in two cases, in which the patellar tendon reflex was very well marked. It differed from the ankle clonus in both cases, being slower in time, only about two and a half contractions occurring per second. Dr Gowers explains this difference between the intervals of contraction in the knee and ankle clonus by regarding the former as a truly reflex, and the latter as a direct contraction, and in support of this theory he notes that the intervals between the application of the stimulus and the contraction of the muscles are different. As regards the knee reflex, he has found that the interval between the tap on the patella and the contraction of the quadriceps is on the average ·10 or ·11 second, whilst in the ankle the interval between the tap on the muscle, or front tap, and the contraction of the gastrocnemius, he found to be only about ·035 or ·04 seconds. Thus he says, "The proportion between the frequency of the ankle clonus and the frequency of the knee clonus is nearly as four to ten, and the proportion between the interval which intervenes between a tap on the Achilles tendon and the contraction of the gastrocnemius, is to the interval between the tap on the patellar tendon and

the contraction of the thigh muscles also as four to ten." He therefore concludes that, in the case of the knee clonus, the several contractions are of true reflex origin, that "the sudden tension of the quadriceps by the weight of the extended leg on the fibres after the cessation of one contraction probably constitutes the afferent stimulus for the next," whilst in the case of the ankle clonus, he concludes that the clonic contractions are the result of direct stimulation, the muscle being in a state of extreme irritability, developed in a reflex manner as the result of the passive flexion of the ankle-joint.

In the cases which I have been studying, I have never been able to induce knee clonus by a stimulus applied to the muscle or tendon, the limb being in the position in which the patellar tendon reflex is usually induced, *i.e.*, the patient sitting, and the legs hanging at right angles to the thigh; but what suggested itself to me was to place the quadriceps muscle under the same conditions as is the gastrocnemius when clonus can be induced in it, *i.e.*, in a state of passive tension. On strongly flexing the leg on the thigh, and so stretching the quadriceps, a tap on the tendon or muscle, however, produces neither reflex nor clonus, but I found that, with the leg slightly flexed (about 25° to the line of thigh), a tap on the tendon usually produced a well-marked clonic spasm. This, however, although quite distinct, never lasted long. The greatest number of contractions I estimated to have been about twelve; usually they did not exceed four or five. I was able to obtain several tracings of this clonus, of which A is a specimen. The upper line (*a*) shows the clonus, the middle (*b*) the instant at which the tendon was tapped, and the lower (*c*) is the chronograph tracing. The clonus tracing was obtained by placing the button of the receiving tambour in contact with the quadriceps muscle or tendon, close to the knee-joint, and is the only one of the three for the comprehension of which more detailed description is necessary. It will be noticed that in

it there is at first a slight fall, and that this is followed by a rise, the first of four distinct undulations. The primary



a, Tracing of knee clonus; *b* at *d* marks where the tendon was tapped; *c*, Chronograph tracing 50 per second.

Tracing of patellar tendon reflex, where leg hanging over edge of bed, to show initial slight fall. The writing needle was flexed to the leg about three inches above the ankle.

slight fall I believe to be due to the fact that, at the moment of contraction of the quadriceps, there is not only a raising of the leg on the knee-joint, but also a slight downward movement of the distal end of the thigh. The extent of this downward movement will, for physical reasons, be greater when the leg is slightly flexed, as regards the thigh, than when it is at right angles, as obtains when the patient is sitting with his legs hanging over the edge of a bed or table, but even under these latter circumstances, a tracing of the knee reflex shows an initial slight fall (*see* B). The rise and its undulations are, of course, caused by the clonic contractions, the muscle not undergoing complete relaxations during the intervals. On studying this tracing with reference to the periods of stimulation and the chronograph vibrations, we find that the contractions occur at the rate of about seven per second. This is slightly more frequent than the ankle clonus, and is quite different from the knee clonus

as described by Gowers. We further find that the interval between the tap on the patellar tendon and the contraction of the quadriceps is about ·025 second. This is about the same as Gowers has found in the ankle reflex, but is different from what he has found in the case of the knee. The conclusion is that (in this patient at any rate) the knee clonus is much the same in mode of production as the ankle, and probably also the hip, although in the latter I have not been able to estimate the interval which elapses between the application of the stimulus and the occurrence of the contraction.

But several other important considerations at once present themselves. Why is it that in lateral sclerosis this clonus can be so readily elicited in connexion with the different muscles? Gowers explains the action of a stimulus in producing it by considering that the "passive extension leads to a reflex irritability, a hair-trigger susceptibility to local influences, an incipient contraction excited to developed contraction with extreme readiness, and that this irritability is at its maximum after the cessation of a previous contraction."[1]

But have we grounds for considering that the passive extension is the cause of this reflex irritability? In the case of the ankle it might seem to apply. Here it is found that clonus, readily induced when the muscle is tense, can be stopped at once on relaxing it. But if tension of muscle were the sole cause, clonus should be most readily induced when the tension is greatest, as, for example, on pushing up the foot with the leg perfectly straight. In this case, however, as in others which I have observed, and as noted by Gowers (*Spinal Diseases*, p. 23), it occurs most readily when the leg is slightly flexed.

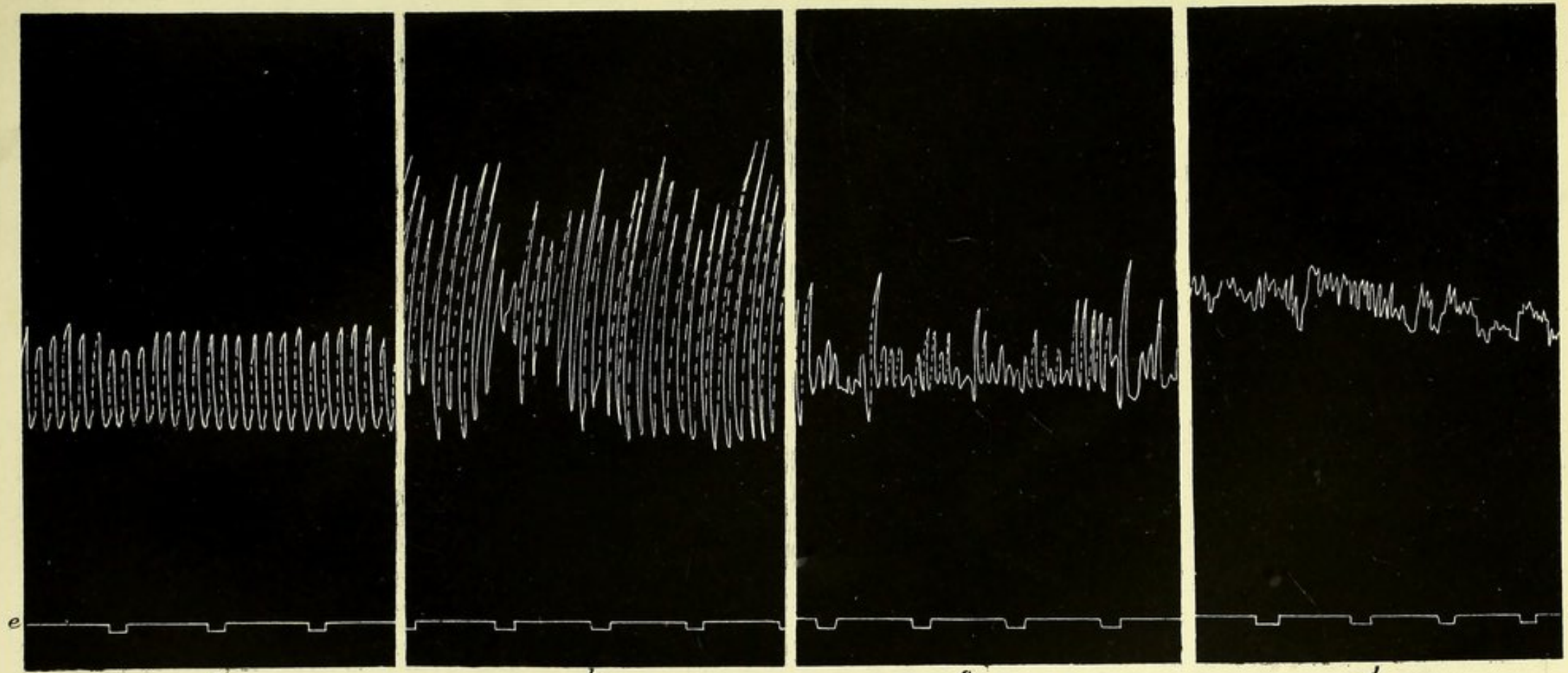
In the case of the knee, it will certainly not apply, for here the clonus can be induced only when the leg is but slightly flexed, and when, consequently, the quadriceps is rather relaxed than tense.

[1] *Medico-Chirurgical Transactions*, 1879, p. 300.

What next suggests itself is, that this so-called "irritability" of the muscles may be in some way connected with the co-ordinated movements which the limbs have to perform. For example, in walking, in making the step from one foot to the other, we first cause contraction of the gastrocnemius of the rearmost leg, by which the heel is raised. Then, the leg being slightly flexed on the thigh, we contract the quadriceps, and so bring forward the foot; then, having placed the foot in front, we find, as we begin to bear on it, that the gluteal muscles contract. Now, it will be found in the case under consideration that it is precisely when the limb is placed in the positions in which it is when any of these muscles are contracting that clonus of them can be induced.

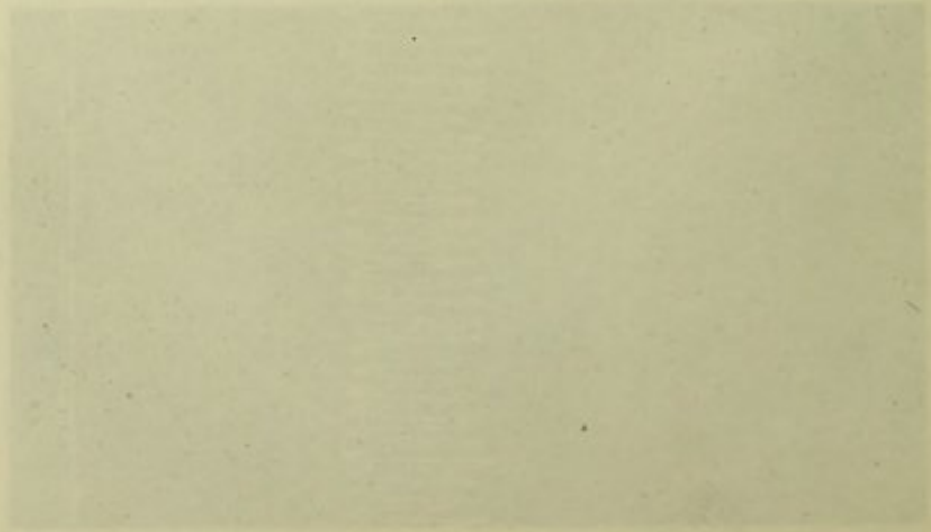
Further, that this "irritability" is specially manifested at certain definite positions of the limb, rather than due to tension of the muscles concerned, can be shown by experiment on ourselves. On testing this, we find that clonus of the gastrocnemius can be most readily (if not only) induced when sitting on the edge of a chair with the foot resting on the ball; and we find also that, as regards the knee, we can most readily induce it with the leg slightly flexed on the thigh, and the toes drawn up. Clonus of the muscles of the upper arm can be best induced voluntarily when the arm is semiflexed. I have obtained tracings of these from six healthy adults, and in three of these I was also able to obtain tracings of a "head clonus." The numbers of contractions per second of these I give in the following Table:—

	Ankle.	Knee.	Arm.	Head.
A.	7·4 per sec.	8·4 per sec.	9·5 per sec.	
B.	8·0 „	7·3 „	8·8 „	
C.	6·2 „	7·0 „	7·5 „	12·4 per sec.
D.	6·0 „	7·6 „	9·5 „	13·0 „
E.	6·7 „	6·5 „	8·4 „	
F.	7·0 „	8·0 „	10·5 „	13·0 „



Specimen tracings of clonus from a healthy adult, (F page 100)
a — ankle. b — knee. c — elbow. d — head.
e — time in seconds.



I have also endeavoured to discover if the number of contractions can be varied at will, but have concluded that if this occurs at all, it can only be to a very slight extent. Thus, in the arm clonus of A. (my own case), I found that by the greatest effort I could produce 10 contractions per second, whilst in ordinary circumstances 9·5 is the number. In the ankle clonus of B., I found that, on his being asked to increase the number of contractions, there was really a diminution from about 8·6 to 8, whilst in C. there was, in similar circumstances, an increase from 6·2 to 6·5 per second.

And now, having so far considered the phenomena of clonus, can we say any more as regards its production in health and disease? The facts—(1), that it occurs most markedly in certain definite positions of the limbs; (2), that in these positions it can be set agoing voluntarily, and that when so, it seems in most individuals to continue for a time, to a certain extent, independently of the will; (3), that the rate of contraction cannot be materially altered by the will; and (4), that in lateral sclerosis it can be set agoing by peripheral stimulation when the limb is placed in one or other of certain definite positions—suggest to us, I think, some ideas as regards the nature of its production.

We know that the nervous axis is formed of cells and of fibres, afferent, efferent, and those connecting cells, and we know that on peripheral irritation afferent impulses are carried along certain tracts, and cause stimulation of certain cells, and that by these, impulses are generated which pass along definite efferent tracts. We further know that for the proper performance of any movement, the muscles employed must be co-ordinated, *i.e.*, when some, singly or in groups, are contracting, others must be relaxing, and that these must alternate as regards contraction and relaxation. All this implies that, in the nerve centres at one time, one set of cells must be stimulated, at another time another set, that the resulting motor impulses must travel now along one tract of

fibres, now along another. When, therefore, we consider the complicated movements which we daily unconsciously perform, and the even more complex processes in the nerve centres, etc., which these necessitate, we can, I think, more easily understand how clonus occurs, and why most markedly or solely in certain definite positions. Thus, in ankle clonus, we may suppose that, with the gastrocnemius somewhat contracted, so as to raise the heel, and the leg somewhat flexed on the thigh, the effect of a stimulus applied to the muscle will, like a wave, be carried to cells in the cord, and that the impulse there generated will be carried from these back again to the muscle as a stimulus to contraction, *without loss by escape along other tracts to other cells or muscles.* And we are the more inclined to believe this when we remember that, in the act of walking, impulses must pass along certain definite tracts without escape thousands of times every day. Similarly with the knee clonus, arm clonus, etc.

Conversely, we may suppose that the fact of its being difficult or impossible to excite clonus in muscles when the limbs are not in such positions, even although the muscle concerned may be in a state of greater passive tension than before, is due either to increased resistance in the appropriate nerve channels, or to the escape or overflow of the impulse to others.

I therefore would suggest that the position of the limb, as affecting through the muscle the nerve tracts and centres in the cord, is, rather than tension of muscle, the important item in the production of clonus.

But how is it that if, in a case of lateral sclerosis, we place the limb in a certain position, and apply a certain stimulus peripherally, we excite a clonus which will last a longer or shorter time, and which is beyond the control of the will, whilst in health, in like circumstances, this either does not occur at all, or if it does, only to a very limited extent?

Before we attempt to discuss what may be said in answer to this query, let us see if there is nothing else of interest to be derived from a study of these tracings of healthy clonus.

On the justifiable assumption, that for each contraction there must be the passage of an impulse from the muscle to the centre in the cord and back to the muscle, we should expect to find that in the clonus of muscles distant from the cord, the intervals between the contractions would be greater than in those in closer proximity to it, as in the former a greater length of nerve has to be traversed. A reference to the notes of the tracings does, I think, bear this out. In the head clonus, in the cases in which it could be obtained, the number of contractions per second was the greatest, and the arm clonus was next in number to this. In three out of the five cases, the knee clonus was more rapid than the ankle. In the two cases in which the knee clonus was the slower, very great difficulty was experienced in producing it as compared with the ankle clonus, and this I believe to be the explanation of their being exceptional.

I further endeavoured to obtain data on which to form an estimate of the distances which the nerve impulses must travel in the case of the different muscles, and I found that in a man 5 feet 11 inches in height the distance from the 12th D. V. to the middle of the leg was 42 inches, and to the middle of the thigh 27 inches. From the 5th C. V. to the middle of the upper arm, the distance was about 20 inches. Seeing, then, that the distances to be travelled are so unequal, and that the differences between the intervals of contraction are often comparatively slight, we may conclude that the great proportion of the time is taken up at the peripheral and central terminations of the nerves, and not in their course, and this further leads us to expect such exceptions as the two cases of knee clonus before mentioned.

Note further that in the case of lateral sclerosis, which formed the subject of these notes, the ankle clonus

was 6·8, the knee 7, and the hip 9 contractions per second.

Can any relation be traced between the clonus of health or disease and the normal muscle contraction? The latter is, as evidenced by the muscle tone, caused by successive stimuli, and, according to Helmholtz and others, these number 19·5 per second. As to this being the same for all muscles, there is, I think, some room for doubt. I have examined and compared the "muscle tone" in the corresponding muscles (biceps) of boys and men, and in different muscles (masseter, biceps, gastrocnemius) in individual cases, and have concluded that the farther distant they are from their nerve-centres in the cord, the lower is the pitch of the tone resulting from their contraction.[1] Should I be correct in supposing that this, like the clonus, is explained by the differences in the distance along which the nerve impulses have to travel, the connexion between clonus and normal muscle contraction is evident. Thus in A. the arm clonus was 9·5, whilst the normal stimuli to the upper arm follow one another at (as nearly as necessary for our argument) double that rate. We have only to suppose, then, that the difference between normal bracing of the muscles of the upper arm and clonus is, that in the former the stimuli pass simultaneously to biceps and triceps muscles, whilst in the latter they pass alternately. Similarly with the thigh and gastrocnemius, the fewer contractions per second of the clonus corresponding with the lower-pitched muscle tone.

And now let us return to the query which we left unanswered at page 102. Why is it that in lateral sclerosis clonus can, under certain conditions, be readily induced by

[1] It might be said that the size of the muscle had to do with the different pitch of the muscle tones. It may be an argument against this to mention that the toe clonus described by Dr Gowers (*Med. Chir. Trans.*, vol. lxii. p. 288) was about the same in rate as that of the gastrocnemius. Compare Herbert Spencer's paper on the *Physiology of Laughter*, and also his *First Principles*, 3rd edition, p. 238.

peripheral stimulation, and will go on independently of, and to a great extent in direct opposition to, the will, whilst in health this does not occur? The theory that it is due to an irritability of the reflex mechanisms in the cord, though it does not give us much information, is conceivable enough, for we may suppose that just as sclerosis of the posterior columns manifests itself to the cells in the brain by lightning pains, etc., so in lateral sclerosis the condition of the cells in the anterior cornua cannot be one of continued quiescence. But there are other considerations. It is well known that when the brain of a frog is removed, reflex action is developed to a much greater extent than in the entire animal; and many interesting experiments might be mentioned to show that stimulation of the optic lobe will restrain or inhibit this function. It has, therefore, been concluded that in the optic lobes in the frog, and there or thereabout in other animals, there is an inhibitory centre. This theory, however, although useful to us in many ways in the present state of our knowledge, can never be regarded as an exact explanation of the phenomena. Apart from the experimental evidence against it, *e.g.*, that stimulation of any afferent nerve will, in the absence of cerebrum, optic lobes, and medulla, cause inhibition of reflex action, we must remember that, for general reasons, an increase of reflex action, on removal of the brain, is to be expected. In the entire frog, the effects of a peripheral stimulus are carried in part to the motor centres in the cord, and in part to the brain; in the former resulting in motion, in the latter in consciousness. In the decapitated frog, on the other hand, the nerve channels leading to the brain are cut, and hence the effects of the peripheral stimulus are manifested as motion alone. This suggests a very simple method by which what may be called the "mechanical equivalent" of consciousness may be obtained. To do so we have only to connect a muscle—say the gastrocnemius of a frog—with a given weight, and to apply

a stimulus of a given strength, so as to produce reflex contraction of that muscle. The difference in the height to which the weight is raised before and after decapitation will, *cæteris paribus*, yield this equivalent. I am, I trust, sufficiently acquainted with physiological experiments to know the value of the term *cæteris paribus*, and I shall not at present discuss this subject, but in support of these ideas, as regards inhibition or inhibitory centres, I take the liberty of quoting the following from George Henry Lewes's *Physiology of Common Life*, vol. ii. p. 201:—

"What is the process of control? Every action is a response to a sensitive stimulus. Muscles are moved by motor nerves which issue from nerve-centres; these nerve-centres are excited by impressions carried there, either by sensory nerves going from a sensitive surface, or by impressions communicated from some other centre. A stimulus applied to the skin excites a sensation, which, being reflected on a muscle, excites a contraction. But, instead of the sensation exciting a muscle, it may be reflected on some nerve-centre, and excite a reflex feeling. This secondary or reflex sensation may either play upon a muscle or upon some other centre, and *this* will excite an action. Thus it is that the same external stimulus may issue in very different actions. We decapitate a frog, and half an hour after prick or pinch its leg; the frog hops, or suddenly draws up its leg. We now prick or pinch an uninjured frog in the same way, and we mostly (not always) observe that its leg is motionless; it does not hop away, it only lowers its head, and perhaps closes its eyes; a second pinch makes it hop away. In the decapitated frog the action was reflex; the stimulus transmitted from the skin to the spinal cord was directly answered by a contraction of the leg. In the uninjured frog the stimulus was also transmitted to the spinal cord, but from thence it ran upwards to the brain, exciting a reflex feeling of alarm, but, though alarmed, the animal was not forced

into any definite course of action to secure escape, and while thus hesitating, a second prick came, and the urgency of the sensation then caused it to hop away. This hopping was reflex, but it was indirectly so; it was prompted by a reflex feeling, which, in turn, had been excited by the original sensation."

But will this increase in the reflex function of the cord, which occurs when the brain is removed, on the above theory as to the nature of its production, account for the increased tendon reflex and clonus of lateral sclerosis? On the theory that they depend on the results of peripheral stimulation being kept within certain channels, it will, I think, go far towards doing so, for in this disease their passage towards the brain will be more or less blocked. The argument that the fibres of the lateral columns have a centrifugal and not centripetal function, and hence that their being sclerosed will not prevent the access to the brain of the effects of any peripheral stimulation, is to my mind hardly valid, inasmuch as a consideration of the evidence will, I think, show that the conduction of centrifugal impulses is not the only function of motor nerve fibres. The consideration of the muscular sense leads us to believe that they will, in addition, conduct centripetally; at any rate it will, I presume, be admitted that this theory is not more hypothetical than one which supposes them to conduct inhibitory impulses from a centre in the brain above to nerve cells in the cord below.

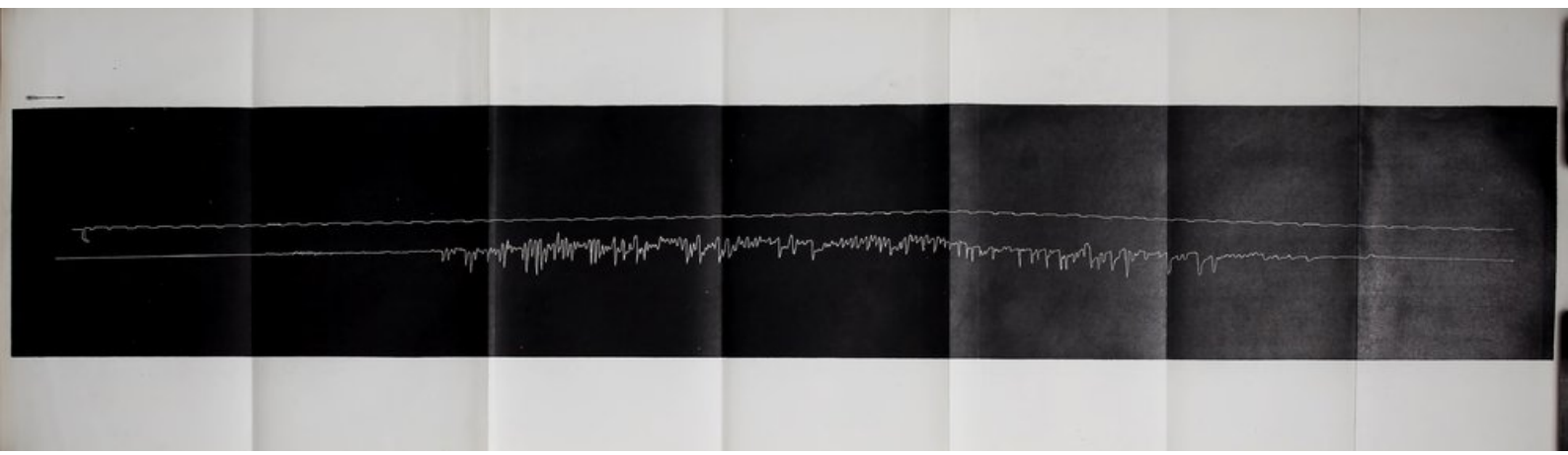
ANKLE CLONUS IN RELATION TO THE HEIGHT OF THE INDIVIDUAL.

WHILST working at the subject of clonus, I have found, as previously stated,[1] that it varies in time considerably in different muscles. Thus the knee clonus I have found to be, for the most part, more rapid than the ankle, the elbow more than the knee, and the head most rapid of all. The elbow and head clonus I have timed in healthy individuals, in most of whom they can readily be induced by a little practice. The knee and ankle clonus I have studied in diseased as well as in healthy cases.

I have suggested that the cause of the differences in the rapidity of those clonic contractions lies in the differences in the distance of the muscles concerned from their nerve centres in the spinal cord,—the muscles concerned in the production of ankle clonus being furthest distant from their nerve centres, those engaged in the production of the head clonus being the least. On the assumption that clonus is a reflex phenomenon, and that the time required for a reflex act in the cord and for the latent period of muscle is always the same, the greater the length of the nerve the slower will be the clonus.

But now it must be noticed that clonic contractions need not be reflex, and that they may vary very much as regards rapidity in the same muscle; *e.g.*, in epilepsy the stage of tonic contraction is followed by one of clonic, and the time of occurrence of these latter varies apparently indefinitely. This is very well seen in a tracing which I have obtained through the kindness of Dr Affleck from a patient in the Royal

[1] See page 100.



Infirmary. The case is one of Jacksonian epilepsy affecting the left arm, and the tracings were obtained by means of Marey's tambours, the receiving one being applied to the extensor surface of the forearm. As can be readily understood, a good tracing of muscular contraction is not easily obtainable in a case of this kind. The movements of the limb, at times even violent, cannot readily be followed. Still, the one which I show is sufficient for our purpose. In it we recognise that at first the line is almost straight, denoting *tonic contraction;* then it presents *clonic,* the rapidity being about 8 or 9 per second; then more violent, but at the same time less rapid, clonic contractions appear, and we can here and there notice that between the violent and slower clonic contractions there are less violent but more rapid ones. Finally, we can notice these large contractions becoming smaller, and the intervals between them lengthening, till they cease altogether.

Again, in the gastrocnemius of the frog we can easily get an example of clonic contractions varying as regards rapidity. If we connect this muscle with a weighted lever, we can, by stimulating it, either directly through the sciatic nerve, or reflexly, produce clonus if we use an interrupted current in strength slightly less than what is required to produce tonic contraction.

Here the explanation seems to be, that just as water made to flow through a tube will fall in a continuous stream when the head is great, and will fall in drops at longer and longer intervals as the head falls, so will a stimulus applied to the spinal cord, nerve, or to muscle directly produce tonic contraction when the irritability is great, and clonic contractions with intervals becoming longer and longer as the irritability becomes exhausted.

It will thus be seen that the rate of clonic contractions may vary in the same muscle. Into the production of the clonus to which this paper applies other elements probably enter;

it may, however, be represented by the first clonus on the tracing, *i.e.*, the most rapid which can be induced in a given muscle.

If now our theory is correct,—*i.e.*, if the difference in the rapidity of the ankle, knee, elbow, and head clonus depends on the difference in the distance of the muscles concerned from their nerve centre in the cord,—we should expect to find differences in the rapidity of clonus in individuals of different heights; and as the height of the individual depends mainly on the length of his legs, we should expect to find this difference best marked in the ankle clonus. That this should be the case is fortunate, inasmuch as this clonus is the one which can be most readily induced by healthy individuals, and consequently is the one which can be most readily timed.

During the last few months I have, therefore, timed the ankle clonus in a number of individuals, endeavouring to limit the difference between them, as far as possible, to one of height alone. Thus, with few exceptions, the individuals experimented on were students of medicine or young graduates, the average age about two or three and twenty years, and all in perfect health. The exceptions were females, patients in the Infirmary, although, as far as their nervous and muscular systems were concerned, in good health. The results given in the Table bear out, I think, the theory which I have suggested; but before considering them in detail, let us devote a few minutes to the consideration of what results we should theoretically expect.

In the first place, then, let us suppose (speaking roughly) that the distance between the muscles concerned in the production of ankle clonus and their spinal centre is 1 yd. in individuals a little over 6 ft. in height, whilst in individuals a little over 5 ft. in height let us suppose it to be 2 ft. Let us further suppose that the nerve impulse travels at the rate of 120 ft. per sec., that the latent period of muscle contraction is $\frac{1}{50}$ sec., and that the time required for a reflex act is $\frac{10}{100}$

sec.[1] Then $\frac{6}{120}$ sec. + $\frac{1}{50}$ sec. + $\frac{1}{10}$ sec. = $\frac{17}{100}$ sec., and $\frac{4}{120}$ sec. + $\frac{1}{50}$ sec. + $\frac{1}{10}$ sec. = $\frac{23}{150}$ sec.

In this way, if our data are correct, the rapidity of the ankle clonus in an individual 6 ft. in height should be $\frac{100}{17}$ = 5·8 per sec., whilst that of one about 5 ft. in height should be $\frac{150}{23}$ = 6·5 per sec. The difference is in this way very slight; and when we reflect on the many and diverse ways in which the rapidity of nerve impulses, the time required for reflex action, and the latent period of muscle contraction, may be influenced, we might reasonably enough expect that our investigations should yield results too discordant to be of any value. Let us, however, examine them (Table, page 112).

In individuals varying in height between 6 ft. and 6 ft. 4 in. (10 cases) the average clonus was 5·84 per sec., the lowest being 5, and the highest 6·75 per sec.

In individuals between 5 ft. 6 in. and 5 ft. 11 in. (29 cases) the average was 6·48 per sec.; the lowest being 5·7, and the highest 7·4 per sec.

In individuals between 5 ft. and 5 ft. 5 in. (9 cases, of which 2 were females) the average was 6 91 per sec.; the lowest being 6·7, and the highest 7·2 per sec.

In 2 cases, females, of 4 ft. 11 in. in height, the clonus was 7 8 and 7·3 per sec.; and in one child, suffering from paralysis, in the Children's Hospital, in height about 4 ft. 6 in., the clonus was 7·8 per sec.

A study of these results in this way shows, I think, that the theory with which we started is in the main correct, *i.e.*, that *cæteris paribus*, the rapidity of the clonus bears an inverse proportion to the height of the individual. That there should be great differences in individuals of the same height is, of course, to be expected. We know that the rate of cerebral processes varies, as evidenced, perhaps best of all, by the fact that the "cerebral equivalent" requires to be obtained

[1] Of course I am aware that different estimates of these periods have been made. These are, however, sufficient for our purpose.

for men engaged in astronomical work, and we may be certain that the "spinal equivalent" will present corresponding variations. To have discussed this point would have required a much greater number of observations. I have no hesitation, however, in saying that individuals of the so-called nervous temperament have a rapid clonus as compared with those who appear phlegmatic; and it appears to me, also, that men who take part in athletic sports have a more rapid clonus than their physically less active neighbours. The influence of sex I can say almost nothing about, but I feel sure that as age advances the clonus tends to become less rapid.

TABLE.

No. of Cases.	Height.	Time of Clonus.	No. of Cases.	Height.	Time of Clonus.
	Ft. In.	Per sec.		Ft. In.	Per sec.
1.	6 4	6·0	25.	5 8	7·0
2.	6 2	6·25	26.	5 8	7·4
3.	6 2	5·0	27.	5 8	6·4
4.	6 1½	5·5	28.	5 8	6·33
5.	6 1	5·2	29.	5 8	7·0
6.	6 1	5·7	30.	5 7½	6·75
7.	6 0	5·3	31.	5 7	6·25
8.	6 0	6·25	32.	5 7	7·5
9.	6 0	6·53	33.	5 7	6·7
10.	6 0	6·75	34.	5 7	6·25
11.	5 11	6·85	35.	5 7	6·82
12.	5 11	6·66	36.	5 6	6·0
13.	5 11	6·37	37.	5 6	7·0
14.	5 11	6·6	38.	5 6	6·2
15.	5 11	6·4	39.	5 6	6·1
16.	5 11	6·0	40.	5 5½	6·75
17.	5 10½	6·2	41.	5 5	6·7
18.	5 10	6·0	42.	5 5	6·8
19.	5 10	6·2	43.	5 5	7·0
20.	5 10	6·83	44.	5 4	7·0
21.	5 10	6·0	45.	5 2	7·25
22.	5 9½	6·83	46. *f.*	5 2	7·2
23.	5 9	6·0	47. *f.*	5 2	7·2
24.	5 9	5·7	48.	5 1	6·75

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