infarction. This opinion is based entirely on these negative experiments and may require modification as a result of further work.

ACCIDENTAL HAEMORRHAGE WITHOUT ALBUMINURIA.

It is said that accidental haemorrhage may occur without nephritis. At present I doubt very much whether this is so, and more evidence is necessary of the absence of nephritis in any given case than is commonly adduced. The mere absence of albumin in the urine is not sufficient evidence of the absence of nephritis, for it is known that acute nephritis may exist without any albuminuria, and examination of the urine in acute oxalate nephritis showed that albumin might be entirely absent even when the blood urea had reached a very high figure. In rabbit B 30 there was no albumin, though the blood urea had reached 128 mg. per cent. In addition to testing the urine for albumin use must in future be made of all the more modern methods of estimating kidney function, more especially the blood pressure, urea concentration test, and blood urea estimation, and where the kidney is available it must be examined histologically as far as possible throughout. Liver function tests, especially the phenol-tetrachlorophthalein excretion test, should also be carried out, for it is possible that a damaged liver may act in the same way as a damaged kidney.

It is also possible that many cases of so-called accidental haemorrhage may be really examples of low implantation of the placenta, and the greatest care must be taken in future to exclude these cases. A case which I treated about a year ago proves that well marked concealed haemorrhage may occur with placenta praevia, and even this possibility must be kept in mind. At least two other similar cases have been recorded—one by Eardley Holland⁸ and the other by Williamson.⁹

In future work an attempt must be made to ascertain the source and nature of the toxins at work, whether chemical or bacterial, and to trace the connexion which undoubtedly exists between accidental haemorrhage and eclampsia. This will involve a carefully co-ordinated and prolonged clinical and experimental study.

SUMMARY.

1. By setting up a chronic oxalate nephritis and then causing an acute exacerbation of the nephritis by introducing oxalates *plus* certain organisms it is possible to produce in pregnant rabbits accidental haemorrhage, both external and concealed, and placental infarction. This method is successful in 100 per cent. of cases.
2. Haemorrhage can be produced, though less easily, by setting up an acute oxalate nephritis and then introducing organisms.
3. A similar result can be obtained, also with less certainty, by producing an acute oxalate nephritis without subsequent introduction of organisms.
4. A similar result can be obtained by setting up a chronic nephritis and afterwards introducing organisms. The latter first set up an acute exacerbation, and haemorrhage occurs later. The result in this case is also less certain than by the first method.
5. In every case in which haemorrhage has so far been caused, an acute or subacute exacerbation of the nephritis, as indicated by a rise in the blood urea, has preceded the haemorrhage.
6. The coliform group of organisms seems to be the most potent in producing the acute nephritis that predisposes to haemorrhage and infarction.
7. An acute oxalate nephritis leads to marked retention of urea, but even when the urea concentration in the blood is at its highest point no albumin may be present in the urine. It is important to bear this in mind before excluding nephritis in accidental haemorrhage.
8. These experiments show that nephritis, acute or chronic, is an important predisposing factor in the production of accidental haemorrhage and placental infarction, and that toxæmia is the exciting cause. It is believed that organisms only act by setting up an acute exacerbation of the nephritis, and that they do not lead directly to

the haemorrhage, the latter being caused by endogenous poisons held up in the circulation by the acutely damaged kidney.

All the work in connexion with this research was carried out in the Research Laboratory of the Royal College of Physicians, Edinburgh, and to the Curator, Superintendent, and Laboratory Committee I am much indebted for the facilities placed at my disposal.

REFERENCES.

- ¹ Gordon Ley: *Journ. Obstet. and Gynaecol. Brit. Empire*, xxviii, 69.
- ² J. Whitridge Williams: *Ibid.*, xxvii, No. 2, Summer, 1925, p. 253.
- ³ James Young: *Trans. Edin. Obstet. Soc.*, 1913-14, xxxix, 153.
- ⁴ Arthur H. Morse: *Surg., Gynecol. and Obstet.*, 1918, xxvi, 133.
- ⁵ J. Shaw Dunn, Albert Haworth, and Nora A. Jones: *Journ. of Path. and Bact.*, 1924, xxvii, pp. 299 and 377.
- ⁶ T. W. Eden: *Ibid.*, 1897, iv, p. 265.
- ⁷ J. Whitridge Williams: *Surg., Gynecol. and Obstet.*, 1915, xxi, p. 541.
- ⁸ Eardley Holland: *Proc. Roy. Soc. Med.*, vol. xiii, Part 3, 1920, Sect. Obstet. and Gynaecol., p. 132.
- ⁹ Herbert Williamson: *Ibid.*, p. 134.

An Address

ON

THEORY AND PRACTICE IN RELATION TO THE TREATMENT OF CANCER WITH LEAD.*

BY

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(With Special Plate.)

In the investigation of so complex a question as the nature and control of malignant disease it cannot be denied that the conversion of a working hypothesis, with which the investigator must start, into a definite and substantial generalization by the putting together of existing scientific facts and the elucidation of further evidence, is of vastly greater ultimate importance than the initial attempts at control based on the generalization enunciated. By this I mean that, provided a generalization concerning the true nature of malignant neoplasia can be established beyond question, some control of this disease—limited perhaps, as I shall explain later—must assuredly follow, if not at the first attempt, at any rate as the result of subsequent modifications and developments.

It has, therefore, been our chief endeavour to substantiate our hypothesis from every point of view. First let me state what our working hypothesis has been; afterwards I will bring forward evidence as to its correctness—evidence that before long will, we hope, enable us to affirm the generalization beyond criticism.

THEORY.

To us malignant neoplasia appears to be a reversion of the somatic cell to the early embryonic type which forms the trophoblast. We regard the chorionic epithelium as being a normally malignant tissue that comes under somatic control, as we have described in previous communications. It is malignant in that it is dependent on its own efforts to obtain nourishment for itself and, indirectly, for the growing embryo.

We believe that if it be possible for a cell to pass through all the phases of undifferentiation to a differentiated state with increasing specialization, as seen in the human embryo, there should be no difficulty in realizing that a differentiated cell is capable in certain circumstances of retracing the phases back to undifferentiation. This process is known as dedifferentiation, and it has been definitely shown to occur in normal differentiated cells grown *in vitro*.

* This is the full text of the address given by Professor Blair Bell in introducing the discussion on the treatment of cancer by lead at the Medical Society of London on March 22nd. A report of the discussion, including an abstract of Professor Blair Bell's address, was published in our columns on March 27th (p. 568). A report of his reply appeared on page 572.

Pathologists have long spoken of the *undifferentiated* cells seen in malignant neoplasia; but, strictly speaking, we should speak rather of the *dedifferentiated* cells. This dedifferentiation we hold to occur as the result of the action of the innumerable causes—mechanical, bacteriological, radiological, thermic, and the rest—all of which lead to a common, precancerous, condition of impaired vitality with starvation—possibly oxygen deficiency—in the cell. Whatever the actual metabolic disturbance produced may be, it is evident that the damaged cell, thrown on its own resources, must either recover, die, or develop abnormally in order to provide itself with nutriment. The last condition can only effectually be ensured if the injured cell revert to a trophoblastic type. This is, I believe, a rational explanation, and is what actually occurs. However, as I have said, such a hypothesis must be founded on accepted facts before it can become a generalization on which we can base our methods of control. We have, therefore, sought and found evidence in support of our hypothesis along the following lines: (a) Morphological. (b) Constitutional—(1) chemical constitution; (2) physico-chemical state. (c) Physiological. (d) Toxicological.

A comparative study on these lines with regard to the similarity of, and differences in, the structure and function of various tissues may be considered reasonably comprehensive. It is, indeed, a study so important and so extensive that some time must elapse before we can obtain an unequivocal and final proof from every point of view. I shall here indicate only our efforts along these lines, and the results obtained.

Morphological Evidence.

The histological evidence of the structure and mode of progression of malignant cells very strongly supports the view that these cells are of a *dedifferentiated* type, and that this dedifferentiation of the cell is comparable with its degree of malignancy. Moreover, I must call attention to the fact that in malignant neoplasia there is a tendency to syncytial arrangement on the part of the cells. Pathologists must often have noticed how true this is, not only in carcinoma, but also in sarcoma. Figs. 1 and 2 illustrate this phenomenon. In the section of carcinoma (Fig. 1) it will be observed that the syncytial tissue surrounds blood channels preparatory to perforation of them—a process exactly represented in the development of the early placenta (Fig. 3).

Again, while it is evident, as already stated, that in malignant cells developed from somatic tissues there is some degree of dedifferentiation, not only towards an earlier stage of development in the tissue concerned, but also towards the totally undifferentiated chorionic epithelium, yet there is an exception to this dedifferentiation in the development of malignant neoplasia in the case of chorion-epithelioma, which reproduces normal chorionic epithelium. In short, whereas benign neoplasia is the result of hyperplasia in normal tissues, malignant neoplasia is a process of dedifferentiation except in the case of chorion-epithelioma which represents hyperplasia of a normally malignant tissue—the chorionic epithelium.

Constitutional Evidence.

Chemical Constitution.—Although there are related secondary chemical differences between malignant cells and normal resting tissues, it is the lipin content, especially that of the phosphatides, and the phosphatide-cholesterol ratio that are interesting, for these substances are of primary importance in the constitution of the cell. Professor W. C. McC. Lewis, Dr. J. W. Corran, and Dr. M. Jowett have found that the phosphatide content and the phosphatide-cholesterol ratio are much higher in malignant than in normal tissues and benign tumours, and that these figures reach a maximum in the constitution of the chorionic epithelium (Table I). Further work is being done on this important subject.

Physico-chemical State.—With regard to the physico-chemical state of the cell membrane, it is obvious that permeability is a matter of prime importance; for, if a cell be in urgent need of nutriment, permeability may be a deciding factor in the continuance of vitality, as indeed

TABLE I.

Human Tissues Examined.	Water (per cent.).	Phosphatides (per cent. of dry weight).	Phosphatide-cholesterol Ratio.
Normal tissues	75.3	2.1	2.5
Innocent neoplasms	(80.9)	(1.5)	(2.8)
Malignant neoplasms	81.5	4.1	3.9
Chorionic villi	89.9	6.8	4.7

This table brings the details of our work on this subject up to date. The figures of innocent neoplasms, so far obtained, have been placed in parentheses, for we consider the number of our experiments in this respect too small. There is no doubt, however, that innocent neoplasms will fall into their proper place in this table.

it is an absolute requisite for rapid growth. Now it is interesting that the degree of permeability of the cell membrane is dependent on the phosphatide-cholesterol ratio: a high cholesterol content favours impermeability, whereas a low cholesterol content and a high phosphatide favour permeability, for this ratio is consistent with an oil in water type of emulsion.⁹

Physiological Evidence.

Many attempts have been made to discover differences between the metabolism of the malignant and that of the normal resting cell; but until recently little of importance had been established. Two years ago, however, Professor Warburg of Berlin commenced to publish a series of most valuable papers on the glucolytic power of various tissues.¹² The chief conclusion derived from his most ingenious experiments is that, whereas in normal resting tissues respiration is high and energy is produced exclusively by oxidation processes, malignant tissues have a lower respiration, and obtain a considerable proportion of their energy by glycolysis. Moreover, Warburg has shown that although a normal resting cell has a slight glucolytic power in the absence of oxygen, in aerobic conditions it does not perform glycolysis; malignant tissue, however, exerts its glucolytic power even in the presence of oxygen. So definite is Warburg's work on this main line that it is unlikely ever to be upset by subsequent researches; indeed, it has already been fully confirmed by Murphy and Hawkins of the Rockefeller Institute, New York,¹¹ and by others.⁸

Certain other investigations have naturally followed. Warburg has stated that benign tumours lie between malignant and resting tissues in regard to their glucolytic power. It is unfortunate, however, that this investigator should have chosen papilloma of the bladder as a representative of an innocent neoplasm, and there is little doubt that considerable difference would be found in the glucolytic power of this tumour and that of, say, a fibroma. This is a matter that is easily settled, and it in no way affects Warburg's general conclusion.

What has, however, interested us is the question of the position of the chorionic epithelium in respect of this function. Warburg himself states that the anaerobic glucolytic ability of this tissue is even greater than that of malignant tissues, but that it does not perform glycolysis aerobically. There is no doubt that his work in regard to the chorion is open to the possibility of experimental error, in that he examined this structure after removal from the uterus with very young rat embryos, and may, therefore, have left much of the chorionic epithelium in the uterus. Murphy and Hawkins appear to have investigated this matter a little more definitely, for they chose the placenta of rats at about half-term. Figures given by them do not express quite such a high glucolytic power as that obtained in the case of the Flexner-Jobling tumour, but in this case we may make the criticism that the chorionic epithelium at mid-term has ceased to perform its true malignant activities, and therefore would record a lower figure than at an earlier stage of development. However, these workers commit themselves to the definite statement that, as regards the placenta, the type of metabolism is exactly the same in respect of glycolysis as that of frankly malignant tissue (Table II). I have little doubt

TABLE II.—*Glucolytic Power of Normal Resting Tissues, and of Innocent and Malignant Tissues.*

	Respiration.	Anaerobic Glucolysis.	Aerobic Glucolysis.	Ratio Aerobic Glucolysis-Respiration.
Normal resting tissues ...	++	+	—	—
Differentiating embryonic tissues	++	++	—	—
Benign neoplasms* ...	+	+	+	+
Malignant neoplasms ...	+	++	++	++
Chorionic villi ...	+	+++	++	++

* Papillomata of bladder and adenoids (Warburg).

† "The type of metabolism was similar to that of frank malignant tissue." Murphy and Hawkins (1925).

that when even more careful experimental work is done on the subject this statement will be amply confirmed. Meanwhile, although so far the evidence obtained is confirmatory of our views concerning the essential similarity of the chorionic epithelium and malignant neoplasia, a final decision in regard to the glucolytic power of the different tissues awaits further experimental demonstration. Professor Lewis and his associates are now engaged on this work.

Toxicological Evidence.

Although it goes without saying that the toxicological affinities of a cell must be closely bound up with its chemistry and physical state, we feel that it is necessary, from a practical point of view, that the resemblance in this respect between malignant and chorionic tissues should be made manifest; for, obviously, this is the information on which we have based our methods in the medical treatment of cancer. We have shown,⁴ then, that the chorionic epithelium is singularly sensitive to the action of lead, and that it is possible readily to produce coagulation necrosis of the epithelium of the chorion in the rabbit (Figs. 4 and 5). Lead appears to have the same action on the cells of a malignant neoplasm when it is able to reach them immediately and in sufficient quantity (Figs. 6, 7, 8, 9, and 10). When the effect is gradual fibrosis occurs around the atrophic inactive cancer cells, this being almost a natural form of cure (Figs. 11 and 12).

I need hardly again refer to the obvious inference that the biological hormone that arrests the invasive powers of the chorionic epithelium in normal circumstances should also have a similar effect on malignant neoplasia; but we ourselves have, so far, been unable to identify this substance. If our hypothesis be correct, and have reached the stage of generalization, there is plenty of work for others along this line; and, indeed, it is not impossible that other inorganic and organic substances may be found that will exert the same effect as lead.

In concluding these remarks relating to the toxicology of lead I should like to reaffirm my belief in the fact that sufferers with lead poisoning—who if males may be infertile and if women may abort during pregnancy—do not suffer with cancer.⁵ I do not say such a thing is impossible; for obvious reasons it is not; but there appears to be no doubt that carefully corrected statistics do suggest that the two diseases are not compatible.

TREATMENT OF PATIENTS.

Before discussing our work in regard to the treatment of malignant disease I wish to invite your attention to the impossible position that has developed in connexion with the estimation of the value of any form of treatment of this malady in the human subject.

The public and a large body of the medical profession are searching the heavens for what they call a "cure for cancer," and apparently they imagine that some day there will be a means of curing every case of cancer, no matter how advanced the disease may be or what organs may be involved. One moment's consideration should convince anyone familiar with cancer in the human subject of the futility of such an idea.

It goes without saying, of course, that whatever treatment is adopted this is most likely to be effectual when applied early. Nevertheless, many varieties of malignant

disease present so few or such trivial symptoms in their beginnings that they escape recognition by the patient, and it may even be impossible for the doctor to diagnose the condition. I am inclined to think that the difficulties of early recognition will not be completely overcome unless some day a specific reaction which will show the presence or absence of a few cancer cells in the body be discovered—*pace* the interferometer—and every person from the day of birth to that of death be compelled to undergo examination once a month. Even so, pregnancy will probably vitiate the test.

Meanwhile, although much may be done in the way of education and on preventive lines, such as I have suggested elsewhere,⁶ it must definitely be understood that the public will often fail to apply for treatment early, and also that surgery will continue to fail in a large proportion of cases—estimated, I am told, by American authorities to be over 90 per cent.

If we add to these difficulties the situation often created by the involvement of important organs, in which the cure of the disease may mean the death of the patient from other causes, we may indeed express wonder that anyone can expect an infallible cure for all cases of cancer. Yet this attitude of mind appears to be universal in civilized communities, and every attempt to advance the treatment of malignant disease is judged by a false standard, which is based on hope and not on fact.

No method of treatment can be appraised by comparison with an ideal—always impossible of attainment—but rather by *properly standardized comparison with other methods of treatment*, and it is by this latter method that we wish our own work to be judged. By "standardized comparison" I mean especially that methods of treatment in exactly similar cases and circumstances must be compared. A new method is handicapped from the start: the successful treatment of hopeless cases, in which all other methods have failed, is demanded; and if this result be accomplished the method is not accorded the credit due to it. Still, a method of treatment should be given the chance of starting on the same mark as other methods that are in vogue, including surgery.

Moreover, as I have said, an initial attempt to treat patients by a method based on a generalization must not be too closely regarded as a test of that method. We have looked upon the results obtained so far with critical eyes, and have considered the beneficial effects of lead upon malignant growths as important chiefly because they afford confirmatory evidence of the correctness of our views. It would indeed be ridiculous to expect any method in the treatment of cancer to bridge over the enormous clinical difficulties at the first attempt.

It is, however, fortunate that our application of theory to practice has at one step given fairly satisfactory results; yet there is danger in this. I do not doubt that any preparation of lead may give certain results, and that eventually one preparation will be found to exhibit more definitely than others specific toxic effects in regard to malignant neoplasia, and less towards normal resting tissues. Still, neither I nor the other members of our clinical staff have any doubt concerning the possibility of further modification and improvement in the matter of treatment, for if our generalization is accepted a wide field of research is thrown open.

With regard to the treatment of malignant disease with lead, I have little to add to what has already been published. There are, however, some points I am glad to have the opportunity of emphasizing.

In the first place I would say that there can be little doubt that the use of colloidal lead in the prevention of recurrence after operation is a matter so important that every case subjected to operation for cancer, whether the disease be believed to be totally eradicated or not, should be treated as if the patient still had the disease. Owing to the pressure of work associated with the treatment of existing disease, mostly of a recurrent nature, we ourselves have not had the opportunity of making comparative studies in this matter. Nevertheless, the present is the time when this should be done, for if the method were to be further improved it might be considered unjustifiable for any surgeon to operate without subsequently treating

the patient with whatever preparation might then be in use. I hope, therefore, that some arrangement will be found possible whereby surgeons will be able to treat two series of cases of the same type of disease by operation, administering lead subsequently to every alternate case. Obviously this would not lead to conclusions that could be considered statistically accurate, because the character and malignancy of the cases would vary, and no one man would be likely to have a sufficient number of cases from which to make a true statistical survey. Still, I think any scientific surgeon would be able to come to a more or less satisfactory conclusion in his own mind from such a study.

Secondly, with regard to the treatment of existing disease, it must never be forgotten that the action of lead is probably quantitative, and that satisfactory results naturally depend on the lead reaching the malignant tumour in sufficient quantity without at the same time poisoning the patient. Consequently nothing that can be done to lessen the quantity of lead necessary should be omitted; all large accessible growths should be removed, and in suitable cases x rays may be employed in conjunction with intravenous injections of lead.

I must, too, refer to the extraordinary differences in the susceptibility to the toxic effects of lead observed in patients. It is, however, rare to find a patient with a large growth affected to any considerable extent; it appears that the lead is attracted to the tumour.

It may be thought by some that we have exaggerated the dangers connected with the treatment of cancer with lead. In answer to this we would point out that we have had a considerable experience, and have come to realize that to ensure good results it is absolutely necessary to give the patient as heavy doses and as much lead as can be tolerated.

Results.

I shall say nothing here about the bad effects that may be produced, for, I hope, my colleague Dr. Cunningham will refer to this matter.*

The fact that we have undoubtedly had disasters should be sufficient warning to those who intend to carry out this treatment, possibly with products of unknown toxicity. We hope in due course to be able to establish an organization for those who wish to learn our methods, and to carry out the treatment on proper lines. When, if ever, the history of our endeavours and arrangements comes to be unimpassionately considered, I think it will be found that in no direction could we have acted in any other way than we have done, if we were to protect the public from danger and, at the same time, bring our work to a position of comparative security.

In conclusion I wish to say a few words concerning the general results that we have obtained. I am glad to learn that those best qualified by actual experience of our clinical work to know regard the clinical paper recently published⁷ as being a cautious and moderate statement; for, if we look at our results, as shown in Table III, for the first five

TABLE III.—Fate of Patients: November 9th, 1920, to November 9th, 1925.

	Cases.
1. Admitted, but died before treatment could be commenced	20
2. Died before treatment could be completed... ..	50
3. Died of intercurrent affections	3
4. Died after treatment (including two deaths from acute nephritis, the result of lead poisoning)	106
5. Died as a result of extensive destruction of growth by lead	4
6. Too recent for results to be estimated	14
7. Complete treatment refused, but patients are living normal lives	9
8. Disease completely arrested	10
9. Believed cured, and treatment stopped	31
	227

years, and care to make an estimate of the percentage of successful cases after excluding groups 1, 2, 3, 5, and 6, or after allowing for a proper percentage of the probable failures that would have occurred in these groups, we find there is left an extraordinary percentage of successes in a class of case that has hitherto never been successfully

treated. When we were putting together this paper my collaborators agreed with me that we should make no attempt to work out this percentage, and I will not do so now, for we have never failed to realize the enormous difficulties still to be overcome, and we are exerting our best endeavours to deal with these before we can allow ourselves any peace of mind.

At present we feel there is sure to be great uncertainty and variation in the results that will be obtained by others. For ourselves I think I may truthfully say that we soon forget our successful cases, whereas every failure is a constant source of worry to us. In such circumstances it is not possible for any human being to form a true estimate of his own work, or to share the optimism which onlookers may justifiably have. Had the work been that of others, no doubt we ourselves should have all been extremely impressed with the results obtained; but the heavy responsibility that rests on us for raising a single hope over this age-long problem is sufficient to weigh down any personal satisfaction accruing from our efforts.

To-night, for the first time, I have suggested that the working hypothesis with which I started in the year 1908, or thereabouts, has now practically reached the position of a generalization concerning the true nature of malignant neoplasia, and that, if this be accepted, a real and substantial foundation has been established from which future researches in regard to treatment may proceed. So far we have explored one line only, but with success that has been, I think, beyond expectation.

REFERENCES.

- ¹ Bell, W. Blair: *Lancet*, 1922, ii, 1005.
- ² Idem, *ibid.*, 1924, i, 267.
- ³ Idem, *Public Health*, 1924, xxxvii, 217.
- ⁴ Bell, W. Blair, Hendry, R. A., and Annett, H. E.: *Journ. Obstet. and Gynaecol. Brit. Empire*, 1925, xxxii, 1.
- ⁵ Bell, W. Blair, Williams, W. R., and Cunningham, L.: *Lancet*, 1925, ii, 793.
- ⁶ Bell, W. Blair: *Ibid.*, 1925, ii, 1003.
- ⁷ Bell, W. Blair, Woolfenden, H. F., Williams, W. R., Cunningham, L., Herd, S. B., and Adami, J. G.: *Ibid.*, 1925, i, 537.
- ⁸ Cori, C. E., and Cori, G. T.: *Journ. Biol. Chem.*, 1925, lxx, 397.
- ⁹ Corran, J. W., and Lewis, W. C. McC.: *Biochem. Journ.*, 1924, xviii, 6.
- ¹⁰ Lewis, W. C. McC., and Corran, J. W.: *Ibid.*, 1924, xviii, 6.
- ¹¹ Murphy, J. B., and Hawkins, J. A.: *Journ. Gen. Physiol.*, 1925, viii, 115.
- ¹² Warburg, O.: *Journ. Cancer Research*, 1925, ix, 148.

POSTURE AS A FACTOR IN HEALTH AND DISEASE.

BY

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THE subject of the function of the body from the standpoint of mechanics seems to have received from the medical profession in Britain less attention than it deserves. The mechanics of the heart have been the subject of much study, and so have those of the digestive tract; yet it is often forgotten that both the circulatory and alimentary systems can be most seriously interfered with by errors in the locomotory system, more particularly in the bones and muscles which support the body cavities.

This question of bodily mechanics has been made the subject of elaborate study from its anatomical, physiological, and surgical aspects by Dr. Goldthwait of Boston, and he has kindly placed his material at my disposal. I hope that the following sketch of the work already done may stimulate interest in the subject on this side of the Atlantic, and induce practitioners here to continue the study on the material at their disposal.

The examination of recruits during the war showed what a large proportion of the civilized population are using their bodies incorrectly, and the improved physique of men from sedentary occupations after military training showed how readily such errors can be corrected by fresh air, good food, and systematic exercise. Similarly a consideration of the vast proportion of cases of minor ailments which come under the care of the general practitioner will show how large a part faulty mechanics play in their causation, and how important it is that such errors should be treated at their first onset, or, better still, forestalled.

* BRITISH MEDICAL JOURNAL, March 27th, p. 569.

FIG. 1.—Section of carcinoma of breast, showing syncytial arrangement of cancer cells. ($\times 480$.)

FIG. 2.—Section of sarcoma of ovary, showing syncytial arrangement of malignant cells. ($\times 550$.)

FIG. 3.—Section of early placenta showing foetal ectoderm surrounding blood spaces. ($\times 400$.)

FIG. 4.—Coagulation necrosis, produced by lead, in the chorionic epithelium of the rabbit. It will be observed that the adjacent maternal tissues (above and below) are unaffected. ($\times 120$.)

FIG. 5.—Coagulation necrosis, produced by lead, in the chorionic epithelium of the rabbit. The maternal tissues below and between the advancing columns of foetal ectoderm are unaffected. ($\times 225$.)

FIG. 6.—Section of post-cricoid carcinoma before treatment. ($\times 170$.)

FIG. 7.—Section of post-cricoid carcinoma shown in Fig. 6 after one massive dose. ($\times 170$.)

FIG. 8.—Section of carcinoma of oesophagus before treatment. ($\times 140$.)

FIG. 9.—Section of cancer of oesophagus shown in Fig. 8 after a single dose of lead, showing almost complete necrosis. ($\times 140$.)

FIG. 10.—Section of gland in case of carcinoma of oesophagus shown in Fig. 8 after a single dose of lead. There is almost complete necrosis. ($\times 140$.)

FIG. 11.—Section of carcinoma of pancreas after considerable treatment, showing fibrosis and atrophy of cancer cells. ($\times 80$.)

FIG. 12.—Section of carcinoma of pancreas shown in Fig. 11 under higher magnification. ($\times 240$.)