bowel and has been tied to the tail thread at the starting point on the mesentery the anastomosis is complete and the forceps higher up is removed. The whole area of operation is now washed with sterile saline solution, especially the anastomosed portion of gut. The latter is then replaced in good position in the abdomen and this is closed with sutures. A pint of normal saline solution may be injected under the skin of the arm towards the axilla and five minims of solution of strychnine hypodermically. All other means for keeping up the patient's vitality, such as warmth and a rectal injection of brandy, are, of course, followed. And besides this I have long made it a practice to encourage such patients to spend all their waking hours in the sitting posture and only to lie down for sleep. This helps in preventing hypostatic pneumonia, as also probably does an occasional thorough rubbing over the back and bases of the lungs. In my oldest patient, aged 76 years, it has been mentioned that she was lifted out of bed to an armchair by the fireside on the sixth day with manifest advantage.

Since I began to employ this method of procedure in 1899 I have done seven enterectomies for gangrenous herniæ. Of these five have recovered. One of the fatal cases died from obstruction due to an old fibrous band pressing on the bowel after its return; the other case did well for several days and then slowly developed peritonitis, starting from the remains of the infected sac which I had unfortunately not drained and which having contained the cæcum was difficult to shut off by suture from the abdominal cavity. There was no failure of any kind at the seat of the anastomosis.⁸

These results, I feel, justify the hope that primary enterectomy may often take the place of the formation of an artificial anus in gangrenous hernia if the principle of *excising a large tract* of damaged gut above the constriction is followed for the reasons given above.

ON THE RELATION EXISTING BETWEEN URIC ACID EXCRETION AND THE BREAKING DOWN OF THE WHITE CORPUSCLES.¹

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THE important fact that uric acid can under certain conditions be experimentally produced from the white blood corpuscles was first observed by Horbaczewski.² Following his research the relation of uric acid excretion to leucocytosis has been worked at by many writers from the clinical standpoint; among these Kühnau³ and Richter⁴ in Germany, Douglas⁵ in this country, and Zarnier,⁶ Giajmis,⁷ and

⁸ Since the above was written I have successfully operated on an eighth case of gangrenous hernia and excised the necrosed portion, consisting of a strangulated Meckel's diverticulum.

¹ A thesis read at Cambridge for the degree of M.D.; also read before the Pathological Society of London.

² J. Horbaczewski: Untersuchungen über die Entstehung der Harnsäure im Säugethierorganismus. Wien Sitzungsbericht, Band 98, Abtheilung iii., S. 301. Also Sitzungsbericht der Königl. Akademie der Wissenschaften Math. Naturw. Cl., Wien, 1889, Band xxviii., pp. 301-318. Also Sitzungsbericht der Königl. Akademie der Wissenschaften, 1890; Band 48, 1891; Band c., 1892. Also Beiträge zur Kenntniss der Bildung von Harnsäure und der Xanthinbasen, sowie der Entstehung der Leucocyten im Säugethierorganismus, Monatschrift für Chemie, Band 12, pp. 221-275. c. confr. J. Horbaczewski, Zur Theorie der Harnsäurebildung im Säugethierorganismus, Wiesbaden, J. F. Bergmann, 1892.

³ W. Kühnau: Zeitschrift für Klinische Medizin, 1895, vol. xxviii., p. 534, Experimentelle und Klinische Untersuchungen über das Verhältniss der Harnsäureausscheidung zu der Leucocytose. Also W. Kühnau and F. Weiss: Weitere Mittheilungen zur Kenntniss der Harnsäureausscheidung bei Leucocytose und Hypoleucocytose, sowie zur Pathologie der Leukämie, Zeitschrift für Klinische Medizin, Berlin, 1897, Band xxxii., pp. 482-500.

⁴ P. F. Richter: Ueber Harnsäureausscheidung und Leucocytose, Zeitschrift für Klinische Medizin, Berlin, 1895, Band xxvii., pp. 290-311.

⁵ Carstairs C. Douglas: Some Observations on the Excretion of Uric Acid, with Special Reference to its Connexion with Leucocytosis, Edinburgh Medical and Surgical Journal, 1900, new series, vol. vii., pp. 32-42.

⁶ G. Zarnier: Rapporto tra Leucocytosi ed Eliminazione di Acido Urico, Gazzetta degli Ospedali, Milano, 1896, vol. xvii., pp. 676-679.

⁷ C. Giajmis: La Genesi dell' Acido Urico in Rapporto alle Sostanze Nucleiniche, Gazzetta degli Ospedali, Milano, 1898, vol. xix., pp. 545-547.

Reale⁸ in Italy may be mentioned. If it be true that uric acid is formed from the white corpuscles, the fact is to be explained by its production from the nuclein contained in these cells, either as a result of their destruction or in consequence of their metabolism. The conclusions arrived at by the above-mentioned observers have, when definite, been by no means in agreement. Kühnau, in an extensive and careful research, showed that, following the crisis of lobar pneumonia, at which period there is an increased destruction of the white cells, there is an increase in the daily amount of uric acid excreted. Making observations in various other diseases he found that following a decrease in the leucocyte count there is in most cases a corresponding increase in the daily amount of excretion of uric acid, an increase which he considers is caused by the increase in destruction of the white blood corpuscles. He further showed that in leucocythæmia, in which condition the number of white cells in the blood is largely increased, the daily excretion of uric acid is also markedly increased.

With regard to his observations showing a rise in the amount of excretion of uric acid following a fall in the leucocyte curve, I would point out that the latter may be referable not to an increased destruction of leucocytes but simply to a diminished production. As regards his researches in leucocythæmia, Milroy and Malcolm⁹ have shown that in medullary as well as in lymphatic leukæmia, and Hale White and Hopkins¹⁰ have demonstrated that in medullary leukæmia, the daily amount of excretion of phosphoric acid is diminished, so that the increased amount of uric acid excreted in these diseases can hardly be ascribed to an increase in the breaking down of the white corpuscles. The other observers mentioned (with the exception of Richter who lays stress on the importance of making observations extending over a considerable period of time) contented themselves with making in each case at most two or three observations on consecutive days of the numbers of white cells in the blood and comparing these with the amounts of uric acid excreted on the same days. Now inasmuch as the physiological amount of the daily excretion of uric acid varies for different individuals within wide limits (between 0.2 gramme and 1.5 grammes) it is obvious that the amount excreted in a particular individual under pathological conditions may be very largely increased without this amount exceeding these limits. It would seem to follow from this that observations of the ratio between the average amount of the daily excretion of uric acid and the average number of white corpuscles in the blood in a particular case are of less value than consecutive observations in the same case, showing alterations in the one side by side with alterations in the other. It must be confessed that, apart from Horbaczewski's experimental work, the only convincing observations as to uric acid being formed from the white blood corpuscles are those of Kühnau on pneumonia.

My object in making the observations which are recorded below has been to determine in pathological conditions the times in each case observed at which an increased destruction of white cells is taking place, and to notice whether this is followed by an increase in the amount of the daily excretion of uric acid. With this end in view daily observations were made of the number of white corpuscles in the blood for many consecutive days, whilst corresponding daily estimations of the total amount of uric acid excreted in the urine in the 24 hours were also performed. It is fairly obvious that in any condition such as advanced pulmonary tuberculosis, in which there is a leucocytosis for a considerable period of time, there must be associated with this (always assuming that the leucocytes concerned are not specially long lived forms) an increase in the amount of the daily destruction of the white cells. When I refer below to increased destruction (as evidenced by certain alterations in the differential count to be presently explained) it must be understood that I mean an increase in the amount of destruction of the white cells over and above what has just previously been taking place (i.e., a relative increase in the breaking down of the white blood corpuscles). In order to prove that a rise

⁸ E. Reale: Il Ricambio della Nucleina in Rapporto alla Genesi dell' Acido Urico ed alla Patologia delle Affezioni Uricemiche, Clinica Medica, Pisa, 1898, vol. iv., pp. 169-171, 185, 225.

⁹ The Metabolism of the Nucleins under Physiological and Pathological Conditions, by T. H. Milroy, M.D., B.Sc., assistant to the professor of physiology, Edinburgh University, and J. Malcolm, M.B., Journal of Physiology, Supplement, 1898-99, also 1899-1900.

¹⁰ Hale White and Hopkins: On the Excretion of Phosphorus and Nitrogen in Leukæmia, Journal of Physiology, 1899.

in the amount of uric acid excreted is due to an increase in the destruction of the white cells, it is necessary to show a corresponding rise in the amount of phosphoric acid excreted; accordingly, besides daily observations of the amount of uric acid excreted, estimations of the daily amount of excretion of phosphoric acid were also made. As Milroy and Malcolm say, "it is hardly likely that there is a marked retention of the phosphoric acid in the organism and if there be not, then the absence of the rise in the phosphorus excretion negatives the view that the alloxur bodies are derived from the direct breaking down of the nucleins."

Now an increased destruction of the white cells means a diminution in the number of the old cells, the young cells either not diminishing at all, or at any rate not to a corresponding degree. Inasmuch as the polymorphonuclear neutrophiles—in this article I have adopted the classification of Cabot¹¹—form by far the larger proportion of the white blood corpuscles in a healthy individual and are those concerned in ordinary leucocytosis, we will first consider them. Ehrlich's¹² view that the myelocyte and polymorphonuclear neutrophile represent different stages in the life history of one variety of cell, the myelocyte being the young cell, is generally accepted as correct. It is further probable that the small lymphocyte is quite distinct from these, being from first to last a small lymphocyte and nothing else, and that the large lymphocyte is, like the myelocyte, a young form of the polymorphonuclear neutrophile about the origin of which, however, we know less than in the case of the myelocyte. In examining a stained blood film from a case in which there is, or has recently been, a leucocytosis, forms intermediate in appearance between the myelocyte and the polymorphonuclear neutrophile are to be seen in varying numbers. The nucleus of one of these cells stains evenly and lightly; it is circular or oval or sometimes bilobed as if about to undergo division; moreover, at times not only the nucleus but the whole cell body is bilobed: the granules of the cell are similar to those of the polymorphonuclear neutrophile. Since all transition stages between the myelocyte and the polymorphonuclear neutrophile are represented by these cells it is clear that they are intermediate in age between these two varieties or are, in other words, simply young polymorphonuclear neutrophiles. Now if it be found as the result of observations made on two successive days that the polymorphonuclear neutrophiles are diminished in absolute number, the "young cells" (including the myelocytes) not being diminished or, at any rate, not being diminished to a corresponding degree, it may be assumed that a destruction of this form of white corpuscle has taken place. If, on the other hand, these show that whilst there has been a diminution in the absolute number of polymorphonuclear neutrophiles the number of the "young cells" is also markedly lessened, an increase in destruction of this variety of cell cannot be assumed, but rather a diminished production. In order to determine the absolute numbers of each variety of white corpuscle stained films were prepared and differential counts were made from specimens taken at the same time as those specimens from which the simple blood counts were made. By means of these differential counts one is thus able to obtain confirmatory evidence of the breaking down of the white blood corpuscle. As regards the small lymphocytes, inasmuch as we are unable to distinguish the young from the old forms of these an increased destruction of this variety of cell can only be inferred when there is a fall in the number thereof followed by a rise in the amount of excretion of phosphoric acid.

From an examination of the results it will be seen that in 17 out of 20 instances in which an increased excretion of phosphoric acid follows a fall in the white blood corpuscle curve, pointing to an increased destruction of white corpuscles, there is associated with this increase a corresponding increase in the amount of uric acid excreted. In 13 instances there is confirmatory evidence of increased destruction of the white cells obtained from the differential counts, whilst in three instances this confirmation, owing to the absence of differential counts, could not be obtained. In five instances the examination of the differential counts gave a directly opposite interpretation to the study of the white

corpuscle and phosphoric acid curves, the one pointing to increased destruction and the other not. Most of the observations were on cases of advanced pulmonary tuberculosis; in these we may fairly assume, as Haig¹³ has pointed out, that there is no retention of uric acid within the body. If we carefully examine the curves we shall see that in those cases in which there are great and sudden variations in the number of white cells, there are also great and sudden variations in the amounts of uric acid and phosphoric acid excreted. This tends to confirm the theory that uric acid is derived from the white corpuscles (compare, for instance, Cases 1 and 5, on the one hand, with the earlier observations on Cases 10 and 11 on the other). It will also be observed that in the case of the three children (Cases 1, 2, and 3) there is, relatively to the body-weight, more uric acid and phosphoric acid excreted than in the case of the adults. This may be explained by assuming a more rapid destruction of the white cells in the former case than in the latter. This is confirmed by reference to the curves, when we find that the rise of uric acid and phosphoric acid follows more closely on the fall of the white corpuscle curve than in the case of the adults.

As regards methods, the white blood corpuscle counts were made by the Thoma Zeiss hæmacytometer, the diluting solution used being a $\frac{1}{3}$ per cent. solution of acetic acid in water coloured by gentian violet; the differential counts were made from films fixed by heat and stained by a modification of Ehrlich's triacid stain. The uric acid was estimated by Hopkins's method (by Folin's modification in Case 9). 100 cubic centimetres of urine were used for each estimation and the total quantity passed in the 24 hours being measured the total quantity of uric acid passed in the 24 hours was thus calculated. The phosphoric acid was estimated by means of the volumetric method with uranium nitrate, 50 cubic centimetres of urine being used for each estimation and the total quantity passed in the 24 hours being calculated as in the case of the uric acid. I append tables (Tables I. to XI.) and curves of the results obtained in the different cases. The observations were made on 11 patients and the results include upwards of 100 estimations of uric acid. The numbers in the columns headed "uric acid" and " P_2O_5 " (phosphoric acid) are the amounts (in grammes) of these substances respectively passed in the urine in the 24 hours. The columns headed "W.B.C." show the number of white blood corpuscles per cubic millimetre. The columns under the headings "young cells" give the percentage of cells having the characters intermediate between those of myelocytes and polymorphonuclear neutrophiles as described above. Owing to unavoidable accidents the continuity of the observations was unfortunately broken in places and in the short description of the results which I shall now give only those differential counts will be discussed which have a definite bearing on the subject of this paper.

CASE 1.—The patient was a boy, aged ten years. His weight was 3 stones 13 pounds. He had lymphadenoma with intermittent temperature. On examining the curves and tables it will be seen that there are two rises in the phosphoric acid curve corresponding with two rises in the uric acid curve following falls in the white corpuscle curve; also rises in the phosphoric acid and uric acid curves

FIG. 1.

corresponding with a fall in the white corpuscle curve. From a study of the differential counts (Table I.) we find evidence of breaking down (increased destruction) of the white cells between March 15th and 16th, 22nd and 23rd,

¹¹ Cabot: Clinical Examination of the Blood.

¹² Ehrlich and Lazarus: Histology of the Blood, translated by W. Myers, Chap. ii.

¹³ Haig: Uric Acid as a Factor in the Causation of Disease.

TABLE I.—Case 1.

Date.	P ₂ O ₅ in grammes.	Uric acid in grammes.	W.B.C.	Differential counts.					
				Polymorpho-nuclear neutrophiles.	Lympho-cytes.		"Young cells."	Myelocytes.	Eosinophiles.
					Large.	Small.			
				Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
March 13th	—	—	22280	—	—	—	—	—	—
" 14th	—	—	17250	75.19	14.07	4.20	3.75	1.49	1.29
" 15th	0.42	0.18	13200	72.72	18.19	3.86	3.40	0.71	1.10
" 16th	0.25	0.21	11800	71.38	19.02	3.10	4.37	0.54	1.55
" 17th	0.94	0.36	—	—	—	—	—	—	—
" 18th	0.62	0.33	—	—	—	—	—	—	—
" 19th	0.52	0.21	12900	78.46	14.73	3.15	2.43	0.14	1.05
" 20th	0.76	0.30	13500	74.82	13.54	5.19	4.20	0.75	1.48
" 21st	1.47	0.40	—	—	—	—	—	—	—
" 22nd	0.62	0.21	21330	79.38	5.63	6.76	6.44	0.48	1.28
" 23rd	0.31	0.08	14500	74.47	14.01	7.53	2.71	0.62	0.62
" 24th	1.01	0.47	13950	68.89	10.84	13.07	5.90	0.31	0.95
" 25th	0.47	0.15	—	—	—	—	—	—	—
" 26th	0.85	0.22	18640	65.91	13.81	16.78	3.14	0.18	0.18
" 27th	—	0.21	12150	75.66	8.05	13.85	0.93	0.0	1.49
" 28th	—	0.094	13600	66.08	12.65	14.93	3.86	0.0	2.46
April 16th	0.59	0.19	11930	—	—	—	—	—	—
" 17th	0.80	0.25	6260	71.23	7.79	16.63	0.0	0.0	4.33
" 18th	1.02	0.29	5630	71.38	5.20	14.14	5.04	0.0	4.22

and finally April 16th and 17th, such evidence being, as I stated above, a decrease in the absolute number of polymorphonuclear neutrophiles, whilst the number of the myelocytes and young cells remains the same or even increases. These three occasions correspond with the three above-mentioned falls in the white corpuscle curve.

CASE 2.—The patient was a boy, aged seven years. He weighed 2 stones 10 pounds. He had chronic pulmonary tuberculosis and pyuria. There are seen to be marked rises of the phosphoric acid and uric acid curves (on Sept. 27th and 29th) respectively following falls in the white

FIG. 2.

corpuscle curve. Following the last fall in the white corpuscle curve is a rise in the phosphoric acid curve but not in the uric acid curve. From the differential counts (Table II.) there is evidence of breaking down of the white corpuscles between Sept. 26th and 27th, also between Sept. 30th and Oct. 1st, or in two out of the three instances in which a study of the three curves points to increased destruction of the white cells. Between Sept. 28th and 29th no differential counts were made.

CASE 3.—The patient was a male, aged seven years. His weight was 3 stones. He was suffering from chronic pulmonary tuberculosis. Here we find two rises in the phosphoric acid and uric acid curves immediately following falls in the white corpuscle curve. From examination of the differential counts (Table III.) there is evidence of increased destruction between Oct. 11th and 12th corresponding to one of these two falls. Between Oct. 16th and 17th, corresponding to

TABLE II.—Case 2.

Date.	P ₂ O ₅ in grammes.	Uric acid in grammes.	W.B.C.	Differential counts.					
				Polymorpho-nuclear neutrophiles.	Lympho-cytes.		"Young cells."	Myelocytes.	Eosinophiles.
					Large.	Small.			
				Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Sept. 24th	1.37	0.30	9690	56.24	10.26	27.48	1.42	0.0	4.58
" 25th	1.18	0.17	9700	62.43	7.85	24.00	1.71	0.0	4.00
" 26th	0.89	0.088	11560	55.54	12.39	25.65	2.33	0.14	3.93
" 27th	1.56	0.24	9200	53.38	4.769	34.00	1.69	2.30	3.84
" 28th	1.18	0.12	14600	66.24	10.58	19.89	0.91	0.0	2.37
" 29th	1.90	0.28	—	—	—	—	—	—	—
" 30th	1.85	0.13	12200	57.34	7.48	30.36	1.55	0.0	3.25
Oct. 1st	1.08	0.23	11000	60.70	7.44	26.78	1.74	0.0	3.33
" 2nd	1.35	0.22	7100	54.59	7.08	31.56	1.28	0.48	4.99

the other white corpuscle fall, there is no evidence of increased destruction from the differential counts, except, perhaps, in regard to the small lymphocytes.

FIG. 3.

TABLE III.—Case 3.

Date.	P ₂ O ₅ in grammes.	Uric acid in grammes.	W.B.C.	Differential counts.					
				Polymorpho-nuclear neutrophiles.	Lympho-cytes.		"Young cells."	Myelocytes.	Eosinophiles.
					Large.	Small.			
				Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Oct. 8th	—	—	16460	—	—	—	—	—	—
" 9th	—	—	16830	49.93	8.37	37.96	1.94	0.0	1.79
" 10th	1.7012	0.30	15150	51.67	8.29	38.11	0.63	0.0	1.27
" 11th	0.87	0.15	14400	53.34	8.47	35.36	1.33	0.0	1.48
" 12th	0.75	0.12	14000	45.78	8.44	43.18	2.04	0.0	0.54
" 13th	1.01	0.094	—	—	—	—	—	—	—
" 14th	1.57	0.25	12430	60.74	3.23	33.67	0.44	0.0	1.91
" 15th	—	—	11250	50.60	9.07	37.90	2.01	0.0	0.40
" 16th	0.79	0.13	17400	55.73	5.57	35.98	1.43	0.0	1.27
" 17th	0.72	0.11	17000	67.44	2.54	27.25	0.69	0.0	2.07
" 18th	1.29	0.25	15130	61.82	9.23	27.83	0.0	0.0	1.11
" 19th	—	—	12530	57.14	6.10	33.92	1.78	0.0	1.04
" 20th	1.06	0.15	—	—	—	—	—	—	—
" 21st	0.81	0.17	16700	60.00	6.40	36.62	2.03	0.0	0.93
" 22nd	1.13	0.20	17500	60.19	6.47	30.42	1.13	0.16	1.61

CASE 4.—The patient was a male, aged 20 years. He was suffering from caseous pulmonary tuberculosis. He died on Feb. 16th. In this case we find marked rises in the phosphoric acid and uric acid curves following one fall in the

FIG. 4.

white corpuscle curve (between Feb. 9th and 11th). Unfortunately, no differential counts were made between Feb. 9th and 11th. (Table IV.)

TABLE IV.—Case 4.

Date.	P ₂ O ₅ in grammes.	Uric acid in grammes.	W.B.C.	Differential counts.					
				Polymorpho-nuclear neutrophiles.	Lympho-cytes.		"Young cells."	Myelocytes.	Eosinophiles.
					Large.	Small.			
				Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Feb. 5th	—	—	15200	79.75	4.63	8.96	6.02	0.15	0.46
" 6th	1.70	0.77	13600	85.78	4.47	7.18	2.07	0.0	0.48
" 7th	1.93	0.67	13400	77.21	5.84	10.54	5.41	0.99	0.0
" 8th	1.44	0.76	10850	80.91	4.56	10.64	3.54	0.17	0.17
" 9th	—	—	17380	75.48	2.45	12.25	9.10	0.17	0.52
" 10th	—	—	—	—	—	—	—	—	—
" 11th	—	—	12050	—	—	—	—	—	—
" 12th	1.70	0.65	21500	85.52	4.25	8.51	1.53	0.17	0.10
" 13th	2.30	1.42	17500	84.21	5.08	8.42	1.93	0.35	0.0
" 14th	1.34	0.67	9600	87.46	4.12	5.44	2.80	0.16	0.0
" 15th	1.38	0.56	17000	86.05	3.26	7.78	2.71	0.18	0.0

CASE 5.—The patient was a male, aged 21 years, suffering from caseous pulmonary tuberculosis. He died on June 30th. On examining the table (Table V.) and curves we see two well-marked rises in the phosphoric acid and uric acid curves following falls in the white corpuscle curve, also a slight rise

FIG. 5.

of each following a fall in the white corpuscle curve. In only one instance is confirmatory evidence of increased destruction to be obtained from the differential counts—viz., between May 16th and 17th. Between May 20th and 21st

and 22nd and 23rd there is no confirmatory evidence of increased destruction.

TABLE V.—Case 5.

Date.	P ₂ O ₅ in grammes.	Uric acid in grammes.	W.B.C.	Differential counts.					
				Polymorpho-nuclear neutrophiles.	Lympho-cytes.		"Young cells."	Myelocytes.	Eosinophiles.
					Large.	Small.			
				Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
May 16th	1.49	0.49	15700	83.93	6.15	3.96	4.16	1.38	0.40
" 17th	0.89	0.38	13580	79.51	6.20	7.51	5.07	0.75	0.94
" 18th	1.82	0.62	14100	80.07	5.71	6.23	6.75	0.17	0.69 (also basophiles 0.35)
" 19th	—	—	—	—	—	—	—	—	—
" 20th	2.74	1.04	18300	81.44	5.30	8.90	2.08	0.57	1.51 (also basophiles 19)
" 21st	1.77	0.59	7900	85.74	3.61	6.22	2.61	0.20	1.60
" 22nd	1.08	0.49	14000	78.92	5.23	8.24	6.02	0.31	1.27
" 23rd	1.69	0.89	12570	81.62	6.35	9.62	1.20	0.17	1.03
" 24th	3.62	1.05	20270	80.25	6.87	4.93	5.99	1.23	0.71
" 25th	—	—	—	—	—	—	—	—	—
June 25th	1.22	0.85	18500	90.71	3.48	2.65	1.16	1.99	0.0
" 26th	2.40	0.74	22800	85.55	1.82	2.82	6.64	3.15	0.0
" 27th	2.44	0.87	23800	87.07	1.30	3.60	4.41	3.60	0.0
" 28th	2.66	0.77	29250	88.87	4.17	2.26	2.60	2.08	0.0
" 29th	1.42	0.34	29500	—	—	—	—	—	—

CASE 6.—The patient was a female, aged 21 years, suffering from caseous and miliary pulmonary tuberculosis. Unfortunately, no estimations of phosphoric acid and no continuous differential counts were made in this case. It may be noted, however, that after each fall in the white corpuscle

FIG. 6.

The marks + x represent readings of uric acid.

curve there is a rise in the uric acid curve, the most marked rise (that between Jan. 13th and 15th) occurring after the longest and most marked fall in the blood corpuscle curve. The low white corpuscle counts in this case should be noted. (Table VI.)

TABLE VI.—Case 6.

Date.	Uric acid in grammes.	W.B.C.	Date.	Uric acid in grammes.	W.B.C.
Jan. 4th	0.29	3600	Jan. 11th	0.71	4111
" 5th	0.47	3166	" 12th	0.55	3400
" 6th	—	—	" 13th	—	—
" 7th	0.49	2400	" 14th	0.30	1600
" 8th	0.33	4300	" 15th	—	2500
" 9th	0.49	—	" 16th	0.70	1750
" 10th	0.37	4600			

CASE 7.—The patient was a female, aged 36 years, suffering from caseous and miliary pulmonary tuberculosis. Here we see that after the fall in the white blood count between

Oct. 28th and 29th there are marked rises in the curves of phosphoric acid and uric acid. No confirmatory evidence of

FIG. 7.

increased destruction is obtainable from the differential counts between Oct. 28th and 29th. (Table VII.)

TABLE VII.—Case 7.

Date.	P ₂ O ₅ in grammes.	Uric acid in grammes.	W. B. C.	Differential counts.					
				Polymorpho- nuclear neutrophils.	Lympho- cytes.		"Young cells."	Myelocytes.	Eosinophiles.
					Large.	Small.			
				Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Oct. 28th	1.97	0.53	11700	70.54	6.84	19.42	1.75	0.0	1.43
29th	1.11	0.31	9300	73.28	4.92	19.70	0.59	0.0	1.49
30th	1.93	0.44	12100	60.86	4.46	29.58	4.16	0.15	0.77
31st	2.18	0.30	10130	68.30	8.92	17.26	2.52	0.59	2.38
Nov. 1st	—	—	—	—	—	—	—	—	—

CASE 8.—The patient was a male, aged 21 years, suffering from caseous pulmonary tuberculosis. Here there is nothing in confirmation of the theory that uric acid is produced by the destruction of the white blood corpuscles. There are two rises in the phosphoric acid curve following falls in the blood corpuscle curve between Sept. 10th and 11th and Sept. 16th and 17th respectively, but neither of them corresponds with a rise in the uric acid curve. From the differential counts we find that there is evidence of increased

FIG. 8.

The marks x represent readings of uric and phosphoric acid.

destruction of the white cells between Sept. 10th and 11th, 12th and 13th, and finally 16th and 17th. (Table VIII.) The first and last of these three occasions correspond then to the two periods of increased destruction as shown by the white corpuscle and phosphoric acid curves; in the second case, however, there is no succeeding rise in the phosphoric acid curve and therefore no other evidence of increased destruction.

TABLE VIII.—Case 8.

Date.	P ₂ O ₅ in grammes.	Uric acid in grammes.	W. B. C.	Differential counts.					
				Polymorpho- nuclear neutrophils.	Lympho- cytes.		"Young cells."	Myelocytes.	Eosinophiles.
					Large.	Small.			
				Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Sept. 10th	2.65	0.28	12830	81.11	3.22	10.44	3.99	0.76	0.46
11th	—	—	10600	76.43	6.56	10.77	4.37	0.67	1.17
12th	1.22	0.37	11900	80.95	5.39	8.73	2.38	0.16	2.38
13th	2.09	0.35	10300	80.20	3.49	6.84	7.42	0.43	1.60
14th	1.62	0.24	15500	82.82	4.42	8.33	1.87	0.17	2.38
15th	1.18	0.07	—	—	—	—	—	—	—
16th	1.18	0.30	13400	83.12	5.34	5.62	3.89	0.14	1.87
17th	1.17	0.43	12000	81.95	3.41	7.31	3.90	0.32	3.08
18th	1.24	0.41	13150	84.87	3.81	8.81	0.39	0.0	2.11
19th	—	—	—	—	—	—	—	—	—

CASE 9.—The patient was a male, aged 40 years, suffering from chronic pulmonary tuberculosis. In this case it is probable that a rise in the phosphoric acid and uric acid curves occurred soon after the 31st inst., following a slight

FIG. 9.

The marks x represent readings of uric and phosphoric acid.

fall in the white corpuscle curve. There is evidence of increased destruction from the differential counts between August 29th and 30th. (Table IX.) Here there is little evidence either for or against the theory.

TABLE IX —Case 9.

Date.	P ₂ O ₅ in grammes.	Uric acid in grammes.	W. B. C.	Differential counts.					
				Polymorpho- nuclear neutrophils.	Lympho- cytes.		"Young cells."	Myelocytes.	Eosinophiles.
					Large.	Small.			
				Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
August 26th	0.75	0.27	8750	62.55	5.92	28.12	2.01	0.0	1.38
27th	—	—	8550	63.82	8.68	24.89	0.57	0.0	2.02
28th	0.86	0.35	7400	64.99	3.34	25.41	2.73	0.30	3.19
29th	0.69	0.22	7830	67.00	7.96	21.41	1.73	0.14	1.73
30th	0.46	0.064	7600	63.94	3.91	27.55	2.89	0.0	1.70
31st	—	—	7150	61.72	6.43	28.21	1.32	0.49	1.81
Sept. 3rd	1.51	0.86	7750	57.83	8.02	27.13	4.45	0.13	2.42
4th	0.83	0.25	6500	64.73	5.64	25.23	2.19	0.0	2.19
5th	—	—	—	—	—	—	—	—	—

CASE 10.—The patient was a male, aged 62 years, suffering from chronic pulmonary tuberculosis, bronchitis, and emphysema. Here we see two rises in the phosphoric acid and uric acid curves, slight ones on Nov. 13th and very marked ones on Nov. 16th, following two falls in the white corpuscle curves. From a study of the differential counts we obtain confirmatory evidence of increased destruction of the white

corpuscles between Nov. 12th and 13th, corresponding to the first of these occasions. Corresponding to the second there

FIG. 10.

is no confirmatory evidence, seeing that no differential count was made either on Nov. 14th or 15th. (Table X.)

TABLE X.—Case 10.

Date.	P ₂ O ₅ in grammes.	Uric acid in grammes.	W.B.C.	Differential counts.						
				Polymorpho-nuclear neutrophiles.	Lympho-cytes.		"Young cells."	Myelocytes.	Eosinophiles.	Basophiles.
					Large.	Small.				
				Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Nov. 6th	2.43	0.17	8700	65.74	6.06	22.95	1.80	0.16	2.78	0.49
" 7th	—	—	8830	54.96	6.2	28.89	5.38	0.0	3.96	0.56
" 8th	—	—	8150	—	—	—	—	—	—	—
" 9th	—	—	6550	63.91	5.15	25.15	2.06	0.21	3.29	0.21
" 10th	—	—	—	—	—	—	—	—	—	—
" 11th	2.41	0.29	8700	66.62	5.07	22.76	2.15	0.0	2.77	0.61
" 12th	2.48	0.30	8820	64.64	4.70	24.73	2.73	0.0	2.88	0.30
" 13th	2.42	0.35	7020	62.37	5.30	26.67	2.47	0.0	2.82	0.35
" 14th	2.50	0.39	11450	—	—	—	—	—	—	—
" 15th	1.94	0.24	9050	—	—	—	—	—	—	—
" 16th	1.12	0.15	8360	66.36	3.69	24.58	2.40	0.0	2.58	0.36
" 17th	2.94	0.36	—	—	—	—	—	—	—	—

CASE 11.—The patient was a female, aged 42 years. She was suffering probably from a new growth of the mediastinum. She died on June 22nd. In this case there are three

FIG. 11.

The mark + represents a reading of phosphoric acid.

corresponding rises in the phosphoric acid and uric acid curves following respectively three falls in the white blood corpuscle curve. There is, corresponding to these three occasions, confirmatory evidence of increased destruction of the white blood corpuscles between June 11th and 12th, 14th and 15th, and finally between the 19th and 20th. (Table XI.)

To Dr. Eustace Smith and to my colleagues at the City of London Hospital for Diseases of the Chest, Victoria Park, Dr. G. A. Heron, Dr. E. C. Beale, Dr. H. Sainsbury, Dr.

TABLE XI.—Case 11.

Date.	P ₂ O ₅ in grammes.	Uric acid in grammes.	W.B.C.	Differential counts.						
				Polymorpho-nuclear neutrophiles.	Lympho-cytes.		"Young cells."	Myelocytes.	Eosinophiles.	
					Large.	Small.				
				Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
June 11th	—	—	12700	51.97	11.21	31.43	0.79	0.15	4.42	
" 12th	0.64	0.27	7300	58.81	7.05	30.79	1.48	0.0	1.85	
" 13th	0.79	—	8300	54.24	5.59	34.06	1.35	0.0	4.74	
" 14th	0.34	0.17	9900	55.89	11.77	26.36	2.81	0.0	3.15	
" 15th	0.63	0.38	9760	52.04	16.83	23.97	2.89	0.17	4.08	
" 16th	0.49	0.17	—	—	—	—	—	—	—	
	(mean of two days)	(mean of two days)								
" 17th	0.49	0.17	8160	64.24	4.23	24.40	1.69	0.34	5.08	
	(mean of two days)	(mean of two days)								
" 18th	0.93	0.29	11050	67.35	5.33	25.27	0.15	0.0	1.88	
" 19th	—	—	18300	68.12	12.07	14.97	3.22	0.16	1.45	
" 20th	1.59	0.54	10700	70.33	12.12	11.80	3.82	1.27	0.48	
" 21st	1.70	0.24	11600	69.32	10.41	15.72	2.65	1.13	0.76	
" 22nd	2.11	0.39	—	—	—	—	—	—	—	

T. Glover Lyon, and Sir Hugh Beevor, I am indebted for permission to use the results obtained from cases under their care.

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A FORM OF REMOVEABLE DEEP SUTURE.

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ALTHOUGH an aseptic buried suture should remain buried and never be heard of further, still it does sometimes happen that such a suture will, after a time, make its presence known by causing the formation of a pustule to be followed by a sinus which will not heal until the offending stitch has been removed. This may happen after the completely aseptic healing of the original wound and even after every precaution has been taken in the preparation of the suture material. This reappearance of the suture is always a cause of annoyance to the patient and of heart-searching to the surgeon and, if a continuous suture has been used, as for instance, in the peritoneal suture after abdominal section, may necessitate the performance of an operation involving the opening up of the whole original wound. In order to get over this difficulty it is necessary to devise some form of suturing in which the deep suture, although practically buried, may still be removed at any period after the healing of the wound. Again, after many operations, such as those involving suturing at the bottom of cavities, such as in the vagina for vesico-vaginal fistulæ or in colporrhaphy, the patient has to undergo what may cause her almost more suffering than the original operation when the time comes for removing the stitches; and here again the use of an easily removeable suture would be of immense advantage both to the surgeon and to the patient.

It is evident that to be capable of removal at a period subsequent to complete inclosure in a healed wound, any suture of more than a single point must be a continuous one and the form of continuous suture most easily removed by a single application of force would seem to be that which is commonly known as the ordinary machine chain stitch and in order to make use of this it is only necessary to employ a form of needle which will take the entering and returning thread through the same hole. This, then, was the first form