

## LEUCOCYTOSIS AFTER VIOLENT EXERCISE.

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The paper is based on a study of the blood of four of the contestants in the Boston Athletic Association's Marathon race of 1901. This is a road race of about twenty-five miles (40 kilometers), held each spring. The severity of the contest will be apparent when it is said that the winner — not included in my four — covered the distance in less than two and one-half hours. This is about ten miles an hour, about as fast as an ordinary man rides his bicycle for pleasure. In making the white counts and in collecting the blood I was assisted by Dr. W. H. McBain. The white counts were made with the Thoma-Zeiss apparatus. For the differentials one thousand white corpuscles were counted in each of the specimens collected after the race and five hundred in each of the normal ones collected before. Our results are shown in Table I.

The blood of these four cases before the race showed no abnormalities. The percentages of polymorphonuclear neutrophils may perhaps run a little high, but this is to be expected in active young men in the best possible physical condition.

After the race the blood was taken immediately, within five minutes from the actual finish. In every case a leucocytosis was found, varying from 14,400 to 22,200. The differential count showed that the increase was mainly in the polymorphonuclear neutrophils. The relation between the large and small mononuclear forms was changed, the proportion of large forms being increased. Eosinophiles were both relatively and absolutely diminished. In three of the four cases a few myelocytes were found. By myelocytes we mean mononuclear cells with neutrophilic granules, without reference to any particular theory as to their origin, and without intending to imply that they are or are not identical with the myelocytes of leukemia.

TABLE I. THE FIGURES IN PARENTHESES REFER TO THE ACTUAL NUMBERS OF CELLS PER CUBIC MILLIMETER.

NAME.	BEFORE RACE.						IMMEDIATELY AFTER RACE.						Remarks.	
	Interval.	Total Leucocytes.	Polymorph.	Small Mono-nuclear.	Large Mono-nuclear.	Eosinophile.	Myelocytes.	Total Leucocytes.	Polymorph.	Small Mono-nuclear.	Large Mono-nuclear.	Eosinophile.		Myelocytes.
H— . . . . .	3 days before	(9,800)	69% (6,762)	22.4% <sup>h</sup> (2,195)	8.0% <sup>h</sup> (784)	0.6% <sup>h</sup> (59)	0	(14,400)	88.5% (12,744)	7% (1,008)	4.4% (634)	0	0.1% (14)	
L— . . . . .	At start	(4,800)	72.8% (3,494)	18.2% <sup>h</sup> (874)	8.2% <sup>h</sup> (394)	0.8% <sup>h</sup> (38)	0	(16,200)	90.3% (14,629)	4.5% (729)	4.4% (713)	0	0.8% (130)	Many cells intermediate between polymorpho-nuclears and myelocytes.
M— . . . . .	2 days before	(5,800)	63.2% (3,666)	26.8% <sup>h</sup> (1,554)	8.2% <sup>h</sup> (476)	1.8% <sup>h</sup> (104)	0	(20,800)	83.8% (17,430)	7.8% (1,622)	8.2% (1,706)	0.2% (42)	0	
P— . . . . .	At start	(3,700)	72% (2,664)	14.6% (540)	10.6% (392)	2.6% (96)	0.2% (7)	(22,200)	86.1% (19,114)	6.6% (1,465)	7.1% (1,576)	0	0.2% (44)	Few cells intermediate between polymorpho-nuclears and myelocytes.
	3 days before	(8,200)	74% (6,068)	18.4% (1,509)	5.6% (459)	2.0% (164)	0							

The myelocytes were probably more numerous than the table would indicate, as we counted as polymorphonuclears a good many cells having neutrophilic granules, and single, but more or less indented nuclei. In fact one specimen showed numerous cells that appeared to be intermediate between polymorphonuclears and myelocytes, concerning whose classification there was considerable doubt. No abnormalities were noticed in the red corpuscles.

In commenting upon these results we must bear in mind that leucocytosis may be of two sorts:

(1.) An increase in all the forms of white corpuscles so that their relative numbers are unchanged — the “physiological leucocytosis” such as is seen during digestion or after parturition.

(2.) Leucocytosis involving only or mainly the polymorphonuclears, the so-called “inflammatory” type, seen in many infections and toxic conditions.

According to Cabot<sup>1</sup> violent exercise gives a leucocytosis of the first type, usually explained by concentration of the blood from vaso-motor contraction and increase of blood-pressure. Shultz<sup>2</sup> holds that the increase is due to greater rapidity of circulation carrying corpuscles into the general arterial system that had been at rest in the greater abdominal veins. According to the same author, the number varies up to 13,600, which was his highest figure. Obviously our cases do not come under this heading. In each of the four runners the polymorphonuclear cells are increased out of proportion to the other forms, and again the degree is far above Shultz's. Moreover in our cases after the race the blood-pressure was decreased. The exertion had gone far beyond physiological limits and our results certainly show that where this is the case we may get a considerable leucocytosis of the inflammatory type.

A close correspondence exists between our results and those obtained by F. G. Burrows<sup>3</sup> in a study of the leucocytosis associated with convulsions. As in our cases, he found a leucocytosis conforming both in degree and in preponderance of the polymorphonuclear forms to the inflammatory

type. As in our cases he also found relative and absolute decrease of the eosinophiles and the appearance of a few myelocytes. He found reasons for supposing that the leucocytosis was the result of a double cause — first a moderate increase due purely to the muscular work of the convulsions and added to this a leucocytosis of an inflammatory or toxic nature. Where both causes acted together a higher leucocytosis would result than from the toxic cause alone, but the percentage of polymorphonuclears would be less than where the increase was purely inflammatory. From the study of a case of general paralysis with violent frenzy, but no convulsions, and of a healthy young athlete after a short violent run, he infers that muscular work alone is not capable of producing leucocytosis of the inflammatory type. Our figures, however, certainly prove that this inference was not justifiable; muscular work alone, if sufficiently violent and prolonged, can produce leucocytosis of the inflammatory type.

The question then arises, May not the increase of white corpuscles in our cases be due to a double cause: muscular work acting mechanically and producing a physiological leucocytosis plus a toxemia acting chemically to produce an inflammatory leucocytosis? Looking at our figures once more it will be noticed that the two highest white counts are in the cases showing the lowest percentage of polymorphonuclears. This is what we should expect if the lower counts were due to increase of the polymorphonuclears alone or mainly, while the higher were made up of the same thing plus a further increase involving all the forms about alike.

Since the toxic portion of the leucocytosis involves only the polymorphonuclear neutrophiles, let us assume that the increase in total mononuclears (large and small) indicates the degree of increase of all forms alike. In the case of H—— the total of mononuclears just before the race was twelve hundred and sixty-eight, and after the race sixteen hundred and forty-two. It is a simple arithmetical problem to find that if all forms of leucocytes were increased in this ratio the leucocytes at the finish would number sixty-two hundred and fif-

teen, an increase ("physiological" in type) of fourteen hundred and fifteen ( $= 6,215 - 4,800$ ). The actual count was fourteen thousand four hundred, so that eighty-one hundred and eighty-five ( $= 14,400 - 6,215$ ) more polymorphonuclears must have been added, over and above the general increase. In other words, the fourteen thousand four hundred leucocytes found at the finish consisted of the original forty-eight hundred, an additional fourteen hundred and fifteen of physiological type, and eighty-one hundred and eighty-five more from toxic causes. I use the count made at the start. By using the same method on all four cases we made up the accompanying chart (Table II). In L——'s case there is an absolute *loss* of mononuclears, assumed to occur "all along the line."

TABLE II.

	H——.	L——.	M——.	P——.
Loss of weight during race .....	5½lbs.	4½lbs.	4lbs.	2½lbs.
Physiological leucocytosis .....	+1,415	-1,680	+9,512	+4,470
Toxic leucocytosis ....	+8,185	+12,080	+7,588	+9,530
Original number of leucocytes .....	4,800	5,800	3,700	8,200
Total leucocytes.....	14,400	16,200	20,800	22,200

The value of the table is greatly decreased by several facts. In two cases we had to use counts made several days before the race. Again the change in relative numbers of large and small mononuclears throws doubt on the propriety of using the total mononuclears as indicators of a like increase in all forms of leucocytes. Finally it is perhaps more than questionable whether we have a right to assume sufficient mathematical exactness in these blood changes to give such a table even approximate accuracy.

It will be noticed that the physiological element varies greatly, but that the toxic part is fairly constant, strikingly so if we omit the case of L—, which shows a puzzling decrease of mononuclears.

These results become clear and harmonize with those of Burrows and of Shultz, if we assume that there are three stages in the blood changes due to severe, prolonged, exhausting work. (1) A stage where there is a simple physiological leucocytosis — an increase “all along the line.” That this exists Shultz and others have demonstrated. (2) A stage where, in addition to this, there is also an increase of the inflammatory type, probably due to toxic causes. That such a double cause may exist Burrows has shown. The count is here highest of all. (3) A final stage where, owing to extreme exhaustion, the physiological increase has disappeared, leaving only the toxic. Here the total is less high than in the second stage, but the proportion of polymorphonuclears is higher. That the physiological leucocytosis in convulsions is temporary, and may subside while its cause continues, Burrows has shown. It is probable that in our cases also the physiological leucocytosis is temporary.

Of our four cases all had passed the first stage. M— and P—, with high total white corpuscles and relatively low percentages of polymorphonuclears, were in the second stage, while H— and especially L—, with low totals and relatively higher polymorphonuclears, were in the third. Incidentally it may be said that of the four men M— was in the best, and L— in the poorest, condition at the finish.

We are aware that the results here stated depend upon the manipulation of figures, perhaps to an unjustifiable extent, for the changes do not occur with mathematical precision. Too few cases were studied to justify final conclusions. Excitement and exposure to cold probably have a hand in the results. It is probable that the count is affected by concentration of the blood from sweating, though all drank water during the run. All the men lost weight, but the physiological increase of leucocytes was not, as the table shows, in proportion to this. The subject demands further study.

The disappearance of the eosinophiles has been observed by others in leucocytosis from various diseases. As Dr. Cabot has pointed out to the writer, its importance here is that it makes the comparison with the leucocytosis of diseased conditions closer. Mechanical changes pure and simple might increase the absolute numbers of the different forms of leucocytes differently, but would hardly account for an absolute *decrease* in one form alone. The change in proportionate numbers of the small and large mononuclears has also been previously noted.

The occurrence of myelocytes has been noticed both by Burrows<sup>3</sup> and by Capps<sup>4</sup> in convulsions. The latter found, in a general paralytic, who had had an apoplectiform attack and was dying with extreme cyanosis and dyspnea, twelve and six-tenths per cent. of mononuclear neutrophils, besides a number of cells showing every gradation between such and the ordinary polymorphonuclears. His description of this blood would apply to one of our cases. Such observations are of importance in connection with the theory that myelocytes give rise to polymorphonuclears by changes in the shape of the nucleus.

CONCLUSIONS. — Violent, prolonged, exhausting work produces a leucocytosis.

This leucocytosis is made up principally by an increase in the polymorphonuclear cells, but the other forms may also be considerably increased in numbers.

More than one cause acts to produce the leucocytosis — probably a temporary, mechanical cause, and a toxic cause, more slow to develop, but lasting as long as the exercise continues.

#### REFERENCES.

1. Cabot. Clinical Examination of the Blood, 1897. Pages 87 and 92.
2. Shultz. Deut. Arch. f. klin. Med., 1893. Page 234.
3. Burrows. Amer. Journ. Medical Sciences. May, 1899.
4. Capps. Amer. Journ. Med. Sciences. June, 1896.