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Erythrocyte sedimentation test

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THE ERYTHROCYTE SEDIMENTATION TEST

by

Olfert W. Topp

Senior Thesis
Presented to

The University of Nebraska,
College of Medicine,
Omaha, Nebraska, April 8, 1938.
CONTENTS

Title ............................. 1
Illustrations ....................... iii
Introduction ......................... iv
History .............................. 1
Technique ........................... 6
Speculative explanations of the phenomenon .......... 15
Clinical applications of the sedimentation test ......... 24
  Normal readings .................... 24
  Tuberculosis ........................ 27
  Syphilis ............................ 35
  Pneumonia and complications ...... 41
  Arthritis ........................... 42
  Rheumatic fever ..................... 49
  Cardiac lesions ..................... 55
  Malignant disease .................. 61
Obstetrics, gynecology, medical and surgical considerations ...... 64
  Anemia ............................. 79
  Diabetes mellitus and hyperthyroidism ............... 85
  Allergy and skin .................... 88
  Neuropsychiatry ........................ 89
Conclusion ............................ 93
Bibliography ........................ 94
<table>
<thead>
<tr>
<th>No.</th>
<th>Illlustration Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Bilateral tuberculosis</td>
<td>33</td>
</tr>
<tr>
<td>2</td>
<td>Tuberculosis: unilateral cavitation</td>
<td>34</td>
</tr>
<tr>
<td>3</td>
<td>Malarial therapy</td>
<td>38</td>
</tr>
<tr>
<td>4</td>
<td>Syphilis</td>
<td>40</td>
</tr>
<tr>
<td>5</td>
<td>Lobar pneumonia</td>
<td>43</td>
</tr>
<tr>
<td>6</td>
<td>Hypertrophic arthritis</td>
<td>50</td>
</tr>
<tr>
<td>7</td>
<td>Rheumatic fever with endocarditis and chorea</td>
<td>56</td>
</tr>
<tr>
<td>8</td>
<td>Pregnancy</td>
<td>67</td>
</tr>
<tr>
<td>9</td>
<td>Acute gangrenous appendix</td>
<td>71</td>
</tr>
<tr>
<td>10</td>
<td>Misc. readings</td>
<td>76</td>
</tr>
<tr>
<td>11</td>
<td>Chronic cholecystitis</td>
<td>80</td>
</tr>
</tbody>
</table>
INTRODUCTION

The writer's interest in the sedimentation test was first aroused during the summer of 1937 while engaged in laboratory work under Dr. S. Miles Bouton at the Hastings State Hospital. Sedimentation tests were done on patients as part of the routine blood examination. Much of the time was devoted to personal observation and study of the relationship of sedimentation curves to various mental and organic diseases.

The fundamental purpose of this paper is to explain and evaluate the use of the sedimentation test as a laboratory aid to diagnosis and prognosis. The discussions will not be limited merely to one phase of the subject, but the history, technique, causative factors, and clinical applications will be considered.
HISTORY

Many centuries ago, long before the advent of laboratory methods, Hippocrates and Galenus (Ostendorf, 1927) the founders of humoral pathology, called the whitish layer of fibrin, which appeared on top of venous blood after it had been exposed to the air for some time, "crusta phlogistica. They observed that it did not form in the blood of a healthy person, and the seriousness of the disease was judged from the size and consistency of this pseudo-membrane and they regarded it as the substance of the disease which had to be removed by frequent venesections. This theory has come to us through the middle ages to the end of the last half of the nineteenth century.

Musgrave in 1703 (Short, 1937) observed the accelerated sedimentation rate of erythrocytes in rheumatism, and referred to this as the "buffy coat".

Hewson in 1772 (Ostendorf) proved that the "buffy coat" was not the substance of the disease, but that the delayed coagulation of the plasma and the accelerated sedimentation of the erythrocytes was the cause of the dissociation.

John H. Hunter (1796) observed that during an inflammatory process the erythrocytes settled more quickly in their own plasma. He was the first to demonstrate that the red cells of the normal blood when placed in
infected blood settled with greater rapidity. It was his belief that the cause of this phenomenon was the fluctuation of the specific gravity of plasma and erythrocytes in the inflamed and non-inflamed blood.

Bellingeri in 1826 (Ostendorf) believed that the electricity of the blood was reduced in inflammatory conditions.

Johannes Müller in 1835 (Ostendorf) assumed an increase of the fibrinogen, and in 1836 Nasse believed that a qualitative change of the fibrinogen content of the blood was the cause which accelerated the settling of the erythrocytes.

John Ashhurst (1893) stated that the blood showed the following changes during inflammation: Due to the changes in the constitution of the blood in inflammation, its fashion of coagulation differs from normal blood. The clot forms more slowly than in health and is smaller and firmer in consistence. The slowness of coagulation and the increased cohesiveness of the erythrocytes permits the separation of the fibrin and the leukocytes to take place before the process of clotting is completed, and this gives rise to the peculiar appearance which is known as the "buffy coat".

Biernacki in 1897 (Morris, 1926) prevented coagulation by the addition of powdered oxalate and made his
determinations by placing the blood in specially con-
structed cylinders and measuring the degree of fall of
the erythrocytes at intervals of thirty minutes and one
hour. It was at this time that he reported a series of
sedimentation tests on seventy-five cases, including four
with pulmonary tuberculosis, and tabulated his compara-
tive results with defibrinated and oxalated blood. He
concluded that the test had clinical value; that the re-
sults were not dependent on corpuscular volume; and that
the sedimentation velocity was related to the content of
fibrinogen in the plasma and intimately related to con-
ditions of disordered oxidation. His work was confirmed
by his pupil, G. F. Muller of Berlin, Germany.

Marcano, in 1901, described perhaps the first micro-
method of blood sedimentation. (Morris)

Shortly after the time of Biernacki, under the in-
fluence of Virchow (Ostendorf) pathologic anatomy became
more important than hematology in humoral pathology,
but this era was of short duration.

Lattes (1925) found that Biffi had published a work
in 1904 in Lo Sperimentale. From a chapter headed "Blood
Sedimentation" he quotes as follows: "Agglutination of
erthrocytes cannot help having a great affect on sedi-
mentation, changing the physical conditions under which
the latter is affected. Erythrocytes collected in groups
sediment more rapidly and in shorter time than ones that are isolated or only collected in groups of few cells. The formation of the 'buffy coat' has been attributed by pathologists to the retarded coagulation of the blood under certain conditions. This explanation is not sufficient. It can be proved experimentally that a slight retardation in the coagulation of the blood is not sufficient to bring about the formation of a 'buffy coat'. But autoagglutination is a most important factor, and perhaps the principal one, in the formation of the 'buffy coat'."

Robin Fahraeus (1917) of Stockholm, while working in the Physiological Institute of the University of Kiel, rediscovered the phenomenon, and demonstrated that the formation of the "buffy coat" depends essentially on the speed of sedimentation of erythrocytes, and that this acceleration is due to the nummular agglutination of the red cells in clumps or rouleaux which sink more rapidly, due to the reduction of the surface of contact. Hober, his teacher, had shown that the red cells were electrically negative, and he therefore assumed that electrically positive substances in the plasma, due to adsorption, approach the surface of the negative erythrocytes, neutralizing them and rendering them unstable. Fahraeus stated that the phenomenon might be employed in the form of a test for pregnancy, as
there was a marked difference between the sedimentation of the red blood cells in parturient and non-parturient women. He also noted that rapid settling of erythrocytes occurred in infectious diseases and malignant tumors.

Linzenmeier, (Ostendorf) in 1920, another pupil of Hober, proved Fahraeus' theory experimentally. By adding electronegative adsorbents to blood with an accelerated sedimentation rate, the reading became normal. He demonstrated that defibrinated blood caused a delayed sedimentation rate, and in blood with much fibrinogen the settling of the red cells was accelerated. From this he concluded that the fibrinogen of the blood is the principal factor controlling the velocity of the erythrocyte sedimentation.

In the past twenty years there have been over three thousand articles written, dealing with the sedimentation reaction. The work has been largely divided into two groups, experimental and clinical. The first attempts to discover a specific cause for the sedimentation reaction, by experimenting with various factors influencing the velocity of the settling red blood cells. The second group has been trying to determine the value of the sedimentation test, as an aid in diagnosis and prognosis.
Many workers on the subject of red cell sedimentation have utilized much time in an attempt to standardize a definite method whereby the rate of sedimenting erythrocytes could be numerically expressed. Up to the present time there have been more than thirty methods devised, all of which are essentially the same and differ only in the amount of material used, the size of the tube, and the method of determining the reading and the collection of blood specimens. Herein the writer gives the principal methods used.

The Westergren (1926) method, a modification of the Fahraeus technique, is performed in a glass tube 300 mm. long and 2.5 mm. in bore, graduated downward from 0 to 200 mm. Blood is withdrawn from the antecubital vein and mixed with a small amount of potassium oxalate (Bouton 1937) or sodium citrate about .4cc. The blood is then sucked up into the tube to the zero mark and the lower end closed by setting the tube into a rubber stopper and the tube set perpendicular into a stand. The readings are taken after an hour. A plasma column of 3-12 mm. in one hour is considered normal.

The technique used by Linzenmeier (Waugh 1923) is described as follows: 0.8cc. of blood is drawn into a special graduated syringe containing 0.2 cc. of
a 5 per cent sodium citrate solution. The blood is then transferred to a small sedimentation tube about 6.5 cm. high and 0.5 mm. in diameter, with calibrations marked at the 1 cc. mark. The sedimentation tube is filled exactly to the 1 cc. mark with the citrated blood, and the line of separation, between the blood corpuscles and the plasma is noted as it reaches the various marks, that is 6, 12, 18 and 24 mm. below the 1 cc. mark. This is the sedimentation time.

Cutler (1926 and 1929) uses a short stout tube with an internal diameter of 11.2 mm. This test requires 0.5 cc. of 3 per cent sodium citrate and 4.5 cc. of blood. With this method, the readings are taken every five minutes for one hour and the observations are recorded on charts designed for that purpose. Bouton has also designed a chart, to be used in recording the readings obtained with the Westergren tube. This I feel is preferable to Cutler's chart, as there is a greater dispersal of indices in the Westergren tube, and thus a more readily measurable fluctuation of the readings, and the changes in the patient's condition can be more easily interpreted. The two charts will be illustrated in another part of the paper.

Wintrobe and Landsberg, (1935) recommend the following method as satisfactory and accurate:
1. Five cubic centimeters of venous blood is collected by means of a dry syringe and needle and mixed in a small bottle containing 4 mg. solid potassium oxalate and 6 mg. solid ammonium oxalate. This concentration of oxalate does not alter as compared with that of blood collected in heparin. Less than 1 cc. of blood is needed for the sedimentation test. The remainder can be used for other blood examinations. Other workers (Greisheimer, 1935) state potassium oxalate hastens the rate. Heparin increases the speed of sedimentation unless the amount of solvent is greater than the control amount.

2. Blood should be used within four hours after collection.

3. The hematocrit is filled to the 10 cm. mark with blood. The upper level of sedimenting corpuscles may be read at frequent intervals up to an hour. Dunn and Sharpe (1936) have devised a set of nomographic alignment charts to correct the volume of packed erythrocytes for the
amount of shrinkage that results from the use of an anticogulant. However, they state that if the ammonium and potassium oxalate mixture is used as recommended by Wintrobe then there is no shrinkage, and this correction chart is not necessary.

4. Since sedimentation rate increases with rising temperature, the sedimentation test should be carried out at a temperature not less than 22 degrees centigrade nor greater than 27 degrees centigrade. Within this range variations resulting from differences in temperature are small. Gordon and Cohen (1928) observed that varying temperatures caused fluctuations in the velocity of the settling erythrocytes, heat accelerating and cold slowing. Rourke and Plass, (1929) while experimenting with blood, took two specimens of the same blood and kept one tube in an incubator at 37 degrees centigrade and the other tube was kept at room temperature (19 to 23 degrees centigrade). They concluded that the rate of sedimentation was accelerated at the higher temperature. Walton (1933) and Yardumian (1937) substantiated
the above findings.

5. The hematocrit should be kept in an exact vertical position during the sedimentation of the blood corpuscles, for when the instrument stands at an angle of even 3 degrees from the vertical, significant acceleration of sedimentation takes place. Ponder (1925) demonstrated that if a 200 mm. tube deviated as little as 7.5 degrees, the sedimentation rate would almost double in a vertical tube.

6. After sedimentation rate has been determined, the hematocrit containing the blood should be centrifugalized and volume of packed red cells determined. The sedimentation rate may then be corrected for alterations due to anemia. These corrections will be discussed in this paper under the heading "Anemia".

Zwecker and Goodall (1925) collect 8 cc. of blood in a graduated centrifuge tube containing 2 cc. or 3 cc. of 3 per cent sodium citrate solution and allowed to stand for an hour and the cell column is then measured. Beaumont and Maycock (1935) modified this method; their sedimentation tube has a bore of 0.75 cm. and its
length is marked in ten divisions, each of 1 cm. Sodium citrate (3.8%) is placed in the tube up to the 2 mark. About 5 cc. of blood is drawn from a vein and placed in the tube containing sodium citrate to the 10 mark. The rate of sedimentation is read in cm. at the end of an hour by placing a millimeter rule along side of the tube. The column of supernatant fluid is not recorded. This method can be compared to others by subtracting result from 10 and then multiplying by ten, thus giving the supernatant fluid per cent. The normal is about 9.7 cm. of corpuscles.

Landau (1933) describes the micromethod of Linzenmeier-Raunot. The tube used is a capillary pipette of thick glass about 6 inches long, with a bore of 1.13 mm. and graduated from above downward in mm. from 1-100. The patients finger is pricked, after warming it in hot water, and eight drops of blood are sucked into a specially tested pipette; the blood is then mixed in a small test tube with two drops of 3.8 per cent solution of sodium citrate. The citrated blood is then sucked into the capillary pipette to the 1 mark, and the tube set in a water-bath at a temperature of 37 degrees centigrade. The reading of supernatant fluid is taken at the end of half an hour by some observers, and at the end of one hour by others. The
final reading is expressed as so many mm. of supernatant fluid per cent. Normal readings are between 0.5 and 4.0 per cent for men, and between 1.0 and 8.0 per cent for women after half an hour.

Harvey and Hamilton (1936) give details of a capillary tube technique which they maintain is easy to perform and repeat, and is a dependable measurement of the rate of sedimentation and blood volume. They use capillary vaccine lymph tubes of standard and uniform bore, graduated with India ink to 7 cm. With a tested pipette, one drop of citrate solution is placed in a watch-glass; this pipette is emptied and used to draw up blood, four drops of which are mixed with the citrate drop. The mixture is then quickly taken up into the capillary tube to the mark, and then the column of blood is given a tilt to bring it higher up the tube. Both ends of the tube are sealed with plasticine, and the tube is set in a vertical position. The time is recorded. The process is repeated with the operator's blood, and the time again recorded. This "simultaneous control" becomes unnecessary when standards have been established. The amount of settling in the tubes is read with a millimeter rule at the end of one hour; a settling of 7 mm. in a 7 cm. blood column would represent 10 per cent. The capillary tubes are next placed in a centri-
fuge tube with a plug of wool at the bottom and filled with water, and are then centrifuged to constant volume. The authors found that this was attained with two centrifugings at about 3,000 revolutions per minute. They also noted that preservation of the citrated blood seemed to slow the sedimentation rate, but add that this is of no significance if a "simultaneous control" has been performed with the operator's blood.

Others (Morris and Rubin, 1926) have felt the need of a micromethod. Although venous puncture is comparatively easy, there are a few cases in which successful venous puncture is very difficult, owing to veins which are deeply embedded in subcutaneous adipose tissue, or those veins which collapse spontaneously on puncture. Still another reason for developing the micromethod is for use on children and infants. For this reason the workers on sedimentation felt it would be feasible to devise a method whereby small portions of blood could be used, and could be obtained in the same manner as the blood used in blood counts.

Greisheimer, Treloar and Ryan (1934) studied the Cutler, Linzenmeier, and Westergren methods, which are to-day the three most commonly used methods. They compared the curves produced by each in 99 men and 102 women. Cutler and Westergren record the sedimentation
index as the length of clear plasma at the end of one hour, while Linzenmeier records the time for the blood cells to settle 18 mm. Through these experiments they found that the average sedimentation index for normals was reasonable concordant for all three methods, and that the measure of sedimentation is in part specific for the technique employed.
SPECULATIVE EXPLANATIONS OF THE PHENOMENON

Herein a brief review of the literature will be presented, dealing with the theoretical aspects of the phenomenon.

Biffi in 1904, (Lattes 1925) concluded that autoagglutination was the most important factor, and perhaps the principal one, in the formation of the "buffy coat". Fahraeus, (1929) also believed that the sedimentation reaction was due to the autoagglutination of the red cells in clumps or rouleaux.

Gram in (1921) concluded that both fibrinogen and cell volume were the important factors in the sedimentation reaction. Starlinger in 1922 (Ostendorf) extended the electrophysical theory by a colloid-chemical one, assuming an increase of coarsely dispersed, readily flocculating globulins as the cause of a rapid sedimentation. Gram, however did not investigate the electrical factors but did determine the cell volume percentage and the temperature are directly proportional to the decreased sedimentation time in the normal.

Hober in 1922, (Ostendorf) summarizes these theories: The processes which accelerate the sedimentation reaction, change the globulin fraction of the blood quantitatively and qualitatively. The adsorption of
these changed globulins changes; (1) the electrophysical nature of the erythrocytes, because the isoelectric point at which the red cells precipitate is reached; (2) the degree of dispersion of the plasma, upon which the dissociation of the red cells from the plasma depends, and (3) the viscosity of the plasma, which controls the adhesiveness of the surface of the blood cells, and the single protein particles.

Waugh (1923) stated, that the viscosity of protein solutions is indirectly proportional to their electrical burdens; globulins having a very high, albumins a relatively low, viscosity. Therefore, he believed that if the globulin of the blood plasma is increased and the albumin is diminished, two changes take place which unite in aiding agglutination of the red cells, hence, the sedimentation of the red blood cells is accelerated. In the first change, the viscosity is raised and the more viscid protein particles cling more readily to the corpuscles, and the red cells to each other; and, in the second place, the electrical burden is diminished, the isoelectric point is approached and the corpuscles tend to aggregate.

Friedlander (1924) supports the experiments of Gram and found in all cases after he had defibrinated blood that it took from four to six times longer,
for the red cells to settle and from this he drew the following conclusions: That neither the red cells nor the viscosity of the plasma was changed by the process.

Fischl (1924) held that the erythrocyte sedimentation was due to the alteration of the albumin-globulin ratio, with an increase in globulin.

Meeker (1925) stated that the most acceptable theory is the hypothesis that a decrease in sedimentation time depends on a protein phenomenon, a disturbance of the normal albumin-globulin, fibrin ratio in the blood.

Schmitz (1926) also believed in the albumin-globulin theory.

Ostendorf (1927) observed that the change of the globulin fraction with all its attending phenomena, depends upon the products of the protein metabolism affected in the organism, by the disintegration of tissue, bacteria and toxins, which activates defensive ferments. The ferments again produce products of decomposition and their increase in the blood influences the plasma and the figured elements which enter into the sedimentation reaction.

Newham (1926) and Fahraeus in 1929, while doing separate investigation on the sedimentation reaction of blood, took into consideration the well-established law
of physicists known as "Stokes Law".

"Stokes Law" (Fahraeus 1929): The sedimentation velocity of the corpuscle in a suspension of globular elements in fluid is proportionate to the square of their radius.

Newham gives a detailed description of the law, according to which the speed of sedimentation, V, is directly proportionate to F, the force of gravity and inversely proportional to N, the viscosity of the dispersion medium, as well as to R, the radius of the particle in accordance with the formula $V = F/6 \pi NR$. He further states that one or more of these factors may be much altered in different disease conditions. Thus, in anaemia of the Addison type it is obvious that R, the radius of the sedimenting particle or red cell, is very considerably altered, and consequently F also will be affected, but in anaemias of the chlorotic type where the hemoglobin is much reduced and the plasma is increased, it is reasonable to expect the viscosity of that medium will be considerably altered, as also will the force of gravity, F, of the cells. Again, it is reasonable to suppose that the factors F, N, and R may in certain disease conditions, be altered in such proportions as to merely affect V very little.
Rosenthal (1928) noted a definitely rapid sedimentation rate in cases of tuberculosis, when the cholesterol was over 300 mg. He concluded that the sedimentation of the blood might be due to a shift in the cholesterol metabolism.

Westergren (1928) supports some of the above authorities by stating the chief cause of accelerated sedimentation rate is a rise in the globulin and fibrinogen content of the plasma proteins.

Reyner (1929) attempted to prove that a difference in surface tension of the blood plasma is responsible for the phenomenon. He increased the surface tension by adding formaldehyde to the blood specimen and maintained that a decrease in the rapidity of the red cell sedimentation occurred in direct proportion to the amount of formaldehyde added.

Greisheimer, Johnson and Ryan (1929) examined the blood of 200 women and 213 men, whose ages ranged from 7 to 94 years, and found that the sedimentation rate was accelerated along with an increased fibrin content in the blood. They also noted that the fibrin content was higher in women than in men, and maintained that this explained the reason for the increased velocity of blood sedimentation in women.

Cutler (1934) after an extensive study of the
subject, stated that the phenomenon depends upon the amount of cellular destruction in the body. As the blood circulates from part to part it carries away products of tissue destruction which alters its stability. In healthy persons as a result of wear and tear of every day life, a certain amount of tissue destruction always takes place, although this varies from day to day it remains within normal limits. Even this small amount of destruction is registered by sedimentation of red cells.

Kunz (1934) points out that the factors controlling the sedimentation reaction must be looked for in the plasma rather than in the blood cells. Hunt, (1929) Fahraeus and many others since the time of John Hunter have placed cells from a well individual into the plasma from a sick patient, and found that the normal cells in this plasma settled almost as rapidly as the sick patient's cells in the same plasma. Sasano, Ordway and Medlor (1936) thoroughly investigated this phase of the phenomenon by examining the blood of 200 patients, and concluded that the blood plasma was of greater importance and significance than the red cells in the sedimentation reaction. Two individuals belonging to the same blood group were selected for their experiment; one was normal and well and the other clinically ill. The cell volumes and sedimentation rate for each was
determined, and the plasma and cells of each separated. The cells of the normal individual was placed in the plasma of the ill patient, and vice versa. This was done after allowances had been made for cell volume. They found that when the cells of the normal blood was placed in the pathological serum there was an increased or accelerated sedimentation rate, and when normal plasma was mixed with the cells from the ill patient the sedimentation rate was normal.

Hirsh (1936) contends that although there are many factors which influence the red cell sedimentation rate the precise mechanism of the reaction is not yet known, but he believes that it depends on foreign protein; the rate in pregnancy depends upon fetal catabolism; in infection the rate is due to protein of bacteria; and in neoplasms or a disease such as coronary occlusion the accelerated sedimentation rate is due to the protein of necrotic tissue.

Yardumian (1937) after investigating the physico-chemical factors influencing the red cell sedimentation, brought out the following:

1. Factors which do not affect the sedimentation rate.
   a. Various anticoagulants.
   b. Various techniques.
c. Variations in temperature between 20 to 30 degrees centigrade.

d. Remixing and resedimenting.

e. Variations in chemical constituents, blood sugar, non-protein nitrogen, calcium, phosphorous, carbon dioxide combining power, cholesterol, plasma and cell chlorides, and albumin-globulin ratio.

f. Variations in the number of leukocytes and blood platelets.

2. Factors which affect the sedimentation rate.

a. Delay over two hours decreases the rate.

b. Variations in bore and length of the tube.

c. Extreme temperature below 20 degrees and above 30 degrees centigrade; cold slowing and heat accelerating.

d. Albumin and globulin content of the blood, high values accelerate the sedimentation rate.

e. Marked hyperglycemia of 200 mg. per cent slows the sedimentation rate.

3. Factors which markedly affect the sedimentation rate.

a. High fibrin content of the blood generally accelerates the rate.

b. High total lipoid content of the blood in certain cases generally accelerates the sedimentation rate.

c. The volume of packed red cells, the smaller the volume the more rapid the sedimentation rate.

d. Deviation of the tube from perpendicular.

The foregoing material has presented many theories
and speculative opinions as to the cause of the erythro-
cyte sedimentation reaction. It has not been definitely
established, however, that any one factor is entirely
responsible for the phenomenon. Therefore, it would
seem logical to assume that the sedimentation reaction
of the red blood cells depends on a disturbance of the
physicochemical balance due to constitutional changes
of the blood during a disease process. The part that
each factor plays in this phenomenon would perhaps be
governed by the area involved by the disease process,
the type of process, and the extent to which it has
developed.
The remainder of this paper will be devoted to the value of the sedimentation test as a laboratory aid in medicine and surgery.

In order to avoid confusion to the reader, only the figures for the Cutler, Westergren and Linzenmeier techniques will be used. Where results are found, the method employed will be mentioned.

The sedimentation test has a three-fold value, (Bannick, 1937) namely:

1. To indicate the presence of disease.
2. To indicate the intensity and progress of the disease.
3. To aid in differential diagnosis.

NORMAL READINGS

Cutler (1926) gives a normal range from 0 to 8 mm. in men and 0 to 10 mm. in women. Westergren (1926) gives his normal as 8 to 12 mm. I have found there is very little consistency in the normal readings for the Linzenmeier technique. Waugh (1923) employing Linzenmeier technique, observed that fully developed males in health gave a sinking time of 1,200 to 1,400 minutes to reach the 24 mm. line. Women during their
sexual life have a shorter time, 800 to 1,000 minutes, except during menstruation when it falls to about 600 minutes. He also noted that the sinking time in the new-born up to the fifth week is 1,500 minutes and from then on there is a marked drop or shortening of the time up to the fourteenth year, and the readings during this period may be as low as 100 minutes.

Friedlander (1924) uses the Linzenmeier method and records the time it takes the red cells to fall to the 18 mm. mark, which he reports as 1,000 to 1,200 in healthy males and 600 to 1,000 in females.

These wide fluctuation in the Linzenmeier technique will not be found in the Cutler and Westergren methods, and the writer believes the latter two are easier to interpret.

Ellenberg (1934) carried out a series of tests on babies varying from two days to 10 days, using the Linzenmeier method, and found the normal range of the new-born between 7 and 23 hours with an average sedimentation speed for the entire new-born period being about 15 hours. There was also a tendency for the sedimentation reaction to become less prolonged as the infant grows older.

The following table, which has been worked out, takes into consideration the affect of various pathologic conditions on the sedimentation rate: (Cutler, 1932)
Diseases with abnormal sedimentation rates:

1. Chronic infectious diseases, such as tuberculosis and syphilis.
2. Acute infectious diseases, such as pneumonia, septicemia, acute endocarditis, and exanthemata and acute bronchitis.
3. Malignancy.
4. Localized suppurations, such as pelvic inflammatory disease, suppurative mastoiditis, suppurative sinusitis, empyema of the gallbladder, bronchiectasis.
5. Acute intoxications, such as lead and arsenic poisoning.
6. Certain endocrine disturbances, such as thyroid toxicosis.

Diseases influencing the sedimentation rate very little; if at all:

1. Simple catarrhal inflammations, such as acute catarrhal appendicitis, simple rhinitis and colitis.
2. Chronic ulcerations of small extent, such as gastric or duodenal ulcer.

Diseases not influencing the sedimentation rate:

1. Functional diseases, such as the various neuroses, and neurasthenia.
2. Certain nervous diseases such as dementia praecox.
3. Focal infections, such as abscessed teeth, diseased tonsils and chronic sinusitis.
4. Metabolic diseases, such as uncomplicated diabetes and essential hypertension.
5. Allergic diseases, such as asthma and hay fever.
6. Most skin diseases.

7. Simple growths, such as fibroma, lipoma and fibromyoma.

8. Simple cysts.

9. Chronic valvular diseases of the heart.

Cutler has neglected to mention anemia and into which category it falls. This will be given consideration under a separate heading.

Discussion of the various diseases and the value of the sedimentation test in each will follow:

**TUBERCULOSIS**

One of the first uses of the sedimentation test was to determine the activity of a tubercular lesion. The opinions and conclusions of various workers will herein be given:

Morris (1924) observed a definite acceleration in the speed of sedimentation of the red cells in active tuberculosis. He also found that in the presence of definite symptoms of activity that the rate was always rapid, and in those conditions which were believed to be quiescent the settling of the red cells was found within normal limits. However, he found normal readings frequently in women. At this time he concluded that the test was of little value in the diagnosis of tuberculosis. In a paper (Morris and Rubin, 1926)
which was published two years later, while working in conjunction with Rubin he repudiated his previous statement by saying, "the test has been found especially useful in tuberculosis as an indication of the degree of activity present."

Westergren (1928) after thoroughly investigating the use of the sedimentation test in tuberculosis, was also of the opinion that it could be used as a criterion to determine the activity of the disease.

Two years later a report came out (Cutler 1930) that the human equation could now be eliminated from the diagnosis of activity in tuberculosis, which in the past was entirely based on clinical judgement, and by using the sedimentation test the three following questions can be answered: 1. How much constitutional disturbance is the tuberculosis producing? 2. Is it active? 3. Does it require special treatment?

It was in this same year that Banyai and Anderson (1930) brought out their report on 2,000 cases of tuberculosis, and found that the activity of the disease has a direct influence on the velocity of the sedimentation test in the majority of instances. In these cases 7.35 per cent showing evidence of active tuberculosis resulted in a normal curve. Nevertheless, they believed that the value of the test lay in observing
the course of the disease, and could be used as a sensitive indicator of complication of the development of new lesions and as a criterion as to the treatment. They suggested that it might be of value in discovering new foci of infection in the lungs, as many times these are not discovered by routine diagnosis. Other complications like intestinal or renal tuberculosis are often unsuspected, and an accelerated sedimentation rate might aid in directing the attention to the source of disturbance. Although the test cannot estimate the extent of the lesion a rapid increasing rate for any length of time indicates an unfavorable prognosis.

Roche (1932) ran sedimentation tests on 244 tubercular patients, using the Westergren technique. His normals in healthy males were 4 to 15 mm. in one hour and in healthy women 6 to 15 mm. in one hour, but he regarded these figures as slightly increased, and placed the readings in three groups: 1. Normal, 4 to 6 mm.; 2. Medium increase, 15 to 30 mm.; and 3. marked increase, 30 to 50 mm. He regarded his low rates as a sign of good prognosis, although he found in emaciated patients where the disease was markedly advanced a relatively lower reading was almost invariably constant. This he explained on a basis of a diminished fibrinogen and a lower concentration of serum globulin in the
blood plasma. He goes further to state that prior to
the onset of relapses, such as a rising temperature or
other signs of increased activity as hemoptysis or pleu-
risy, the patient's temperature, resting pulse rate, and
general condition and appearance, and even his exercise
tolerance, may have been quite satisfactory. But with-
out any special means to prevent occurrences of this
type these are apt to be considered unavoidable develop-
ments of the disease. He is of the opinion that in num-
erous patients such an occurrence could be avoided if,
in the presence of normal temperatures and pulse rates,
their activities are limited until the sedimentation
rate has fallen within the slightly increased group.

Stainsby (1934) brought out that in tuberculosis,
however, secondary infection of the bronchi by organ-
isms other than tubercle bacilli takes place, especially
in advanced cases. He finds that the sedimentation rate
may be of considerable diagnostic and prognostic value
if one will keep in mind the question as to how much of
the increased rate is due to tubercle bacilli and how
much is caused by the secondary invaders.

"In detecting early tuberculous infection, the sed-
imentation test is sometimes of value."

"The sedimentation test is also of help in follow-
ing the course of the disease in any particular patient,
and is of considerable value when patients are being treated at regular intervals by pneumothorax. When used in conjunction with sputum examinations and temperature records, a satisfactory picture of the course of the disease is obtained. In the ambulatory treatment of tuberculosis by pneumothorax, the sedimentation index is more reliable than the temperature for the obvious reason that the temperature varies considerably during the course of a day, while the sedimentation index remains constant.

"A normal sedimentation test at the time when a patient with tuberculosis is considered to have reached an arrested or inactive stage of the disease affords assurance to the physician that his impression is correct. A high index, on the other hand, does not indicate activity of the disease, for secondary infection in the bronchi may be responsible."

Lesser and Goldberger (1935) studied 50 cases of pulmonary tuberculosis and found that the height of the sedimentation reading is an accurate indication of the extent and activity of the pulmonary pathology. Similarly in tuberculous peritonitis, high sedimentation readings were obtained.

Merritt (1937) stated that the sedimentation rate is an especially valuable aid in determining the activity
of tuberculosis. In July of the same year Biern (1937) reported that the sedimentation test serves as a guide to re-infection and an inactive process shows a normal sedimentation rate.

It is the opinion of the writer that the sedimentation curve is a more delicate criterion of activity in tuberculosis, than either the temperature chart or the pulse rate. No case of tuberculosis should be allowed to exercise until the rate is normal and has remained so for quite some length of time.

The writer feels that a paper of this kind would not be complete without some charts to demonstrate how the sedimentation curves can be graphically plotted. The curves on the graph illustrate clearly the severity of the disease, and the reaction to treatment.

Illustration No. 1 is a record of the sedimentation curves of a man with a far-advanced bilateral pulmonary tuberculosis in whom any type of pulmonary compression was out of the question, because of the anatomical distribution of the lesions. Fifteen months of bed rest brought about quiescence of the pathological process, and a corresponding decrease in sedimentation rate. (Ringer, 1934).

Illustration No. 2 is from the records of a patient with a unilateral cavitation who, after several months
CUTLER BLOOD SEDIMENTATION TEST

Name
Address

Date
Diagnosis Bilateral T.B.

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Illust. 1
CUTLER BLOOD SEDIMENTATION TEST

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Illust. 2
of bed rest with no improvement, was given artificial pneumothorax, but, as so often happens, the cavity was held open by adhesions. An internal pneumolysis was successfully done, and the clinical improvement was gradual but steady, as shown in the graph. The patient had a remaining plural effusion, but was clinically well and entirely symptom free.

SYPHILIS

There has been very little written about the use of the sedimentation test in syphilis. However, it is the consensus of opinion that the rate has a definite relationship to the activity of the disease.

Rubin (1926) found in the majority of patients that the sedimentation rate in syphilis accelerates with the degree of activity.

Schattenberg (1932) found approximately 40 per cent of patients with positive serology showed accelerated sedimentation rates, and in all of these he found some form of open lesion or some form of syphilitic process. Those whose blood gave a positive serological test without an accelerated sedimentation rate appeared to have latent syphilis, or showed no activity or outward appearance of the disease.

Merritt (1937) supports these views by his findings
that the sedimentation rate in syphilis seems to correspond to the activity, and in latent syphilis a normal rate is found.

Fremming (1937) while investigating the sedimentation rate of 53 cases of cerebrospinal lues found a normal rate in only 20 per cent. These were mainly classed as paretics. The rate was definitely accelerated in 50 per cent of the cases. A lower rate and marked changes in spinal fluid points to a more superficial form of cerebral syphilis and constitutes a favorable prognostic sign, while a lower sedimentation rate and slight spinal fluid changes indicates an older and more chronic process with very little possibility of remission after treatment with malaria. He also found that patients under malarial treatment give a marked rise in sedimentation reaction which occurs in different degrees. In some cases the rate falls after the fourth or fifth attack of fever in spite of continued rise in temperature. It is not known whether this is due to hepatic injury with consequent lowering of fibrin or not.

The following sedimentation graphs (Bouton) clearly illustrate the varying degrees with which the sedimentation may react after treatment with malaria: (Illust. 3)

"This patient, E.W., a muscular 41 year old white male, was admitted to the Springfield State Hospital,
Maryland, on February 4, 1935, with general paresis.

"His present illness began about two years ago with confusion, memory defect and grandiose delusions.

"At the hospital he was kept in bed. He was restless and disturbed and occasionally needed restraint. He was definitely clouded, but his physical condition was fairly good.

"The patient received malaria on February 26, but showed only a slight reaction with subsidence, so that he was reinoculated on March 29. There was no definite reaction.

"On April 5, he began to have convulsions, and died on April 8, exhibiting marked meningeal symptoms."

The charts may also be used to show the increased rate resulting from meningeal symptoms. Westergren (1928) demonstrated that meningitic forms of cerebrospinal syphilis show slight or moderately high rates. He also added that luetic aortitis, as well as gummatous lues, is almost always associated with a moderately high sedimentation rate.

I might add at this time that Newham (1926) found that the sedimentation rate in malaria was between 25 and 30 mm. with the Westergren technique.

The effect of chemotherapy on the sedimentation rate has not as yet been discussed in this paper. The
SEDIMENTATION CHART

Name: E. W.  
Date: 2-26-35

Sed. I: 76  
Time: 1:00 p.m.

MINUTES

<table>
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RBC: 4,970,000  
HB: 95  
CI: .95

WBC: 4,800  
Polys: 66  
Lymphs: 24

Before inoculation with malaria

Bnds: 10  
Yg. F:  
Myel:  
Monos:  
Eos:  
Bas:  

Illust. 3
SEDIMENTATION CHART

Name  E.W.  Date  2-28-35

Sed. I.  25  Time  1:30 p.m.

MINUTES  0  10  20  30  40  50  60  70  80  90

MM.  5

10

15

20

25

30

35

40

45

50

55

60

65

70

75

80

85

90

95

100

105

110

115

120

125

130

RBC  4,800,000  HB  87  CI .90

WBC  10,600  Polys  87  Lymphs  7

Malaria inoculated  February 26

Bnds.  4  Yg. F.  1

Mycel.  Monos.  1

Eos.  Bas.

(Continued)
SEDIMENTATION CHART

Name     E·W. Date 3-2-35

Sed. I. 63 Time 12:45 p.m.

MINUTES 0 10 20 30 40 50 60 70 80 90

MM. 5

10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100 105 110 115 120 125 130

RBC 4,780,000 HB 90 CI .95

WBC 13,800 Polys 72 Lymphs 14

No parasites found in the blood

Bnds 14 Yg. F.

Myel Monos.

Eos Bas.

 Illust. 3 (cont)
SEDIMENTATION CHART

Name: E.W.  Date: 3-7-35

Sed. I: 90  Time: 1:00 p.m.

MINUTES  0  10  20  30  40  50  60  70  80  90

MM.  5

RBC  4,210,000  HB  50  CL  .97
WBC  16,200  Polys  81  Lymphs  8
Bnds.  9  Yg. F.  

No parasites found in blood

Myel.  Monos.  2
Eos.  Bas.

Illust. 3 (cont)
SEDIMENTATION CHART

Name: E.W.  Date: 3-13-35

Sed. I: 36  Time: 10:00 a.m.

MINUTES  0  10  20  30  40  50  60  70  80  90

MM.  5  10  15  20  25  30  35  40  45  50  55  60  65  70  75  80  85  90  95  100  105  110  115  130

RBC. 4,370,000  HB. 76  Cl. .89

WBC. 6,200  Polys. 57  Lymphs. 28

Bnds. 13  Yg. F.

First chill March 11.  Parasites found in blood

Myel.  Monos. 2

Eos.  Bas.

Illust. 3 (cont)
SEDIMENTATION CHART

Name: E.W.  Date: 3-19-35

Sed. I. 57  Time: 10:00 a.m.

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<td>35</td>
<td>40</td>
<td>45</td>
<td>50</td>
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RBC 3,330,000  HB 72  CI 1.09
WBC 10,700  Polys 70  Lymphs 24
Bnds 5  Yg. F._______

Parasites found in the blood
Myel. Monos. 1
Eos. Bas._______

Illust. 3 (cont)
SEDIMENTATION CHART

Name: E.W.  Date: 3-23-35

Sed. I: 80  Time: 10:00 a.m.

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RBC 3,540,000  HB 52  CI .74
WBC 13,600  Polys 59  Lymphs 16

No satisfactory reaction since March 19

RBC 3,540,000  HB 52  Cl .74
WBC 13,600  Polys 59  Lymphs 16

No satisfactory reaction since March 19

Bnds 21  Yg. F. 4
Myel.  Monos.
Eos.  Bas.

Illust. 3 (cont)
SEDIMENTATION CHART

Name: E.W.  Date: 3-30-35

Sed. I. 102  Time: 10:00 a.m.

MINUTES  0  10  20  30  40  50  60  70  80  90

MM.  5  10  15  20  25  30  35  40  45  50

RBC  3,490,000  HB  60  CI  87

WBC  11,600  Polys  59  Lymphs  27

Bnds  13  Yg. F.  1

Reinoculated March 29  Myel  Monos

Eos  Bas

Illust. 3 (cont)
SEDIMENTATION CHART

Name: E.W.          Date: 4-4-35

Sed. I: 87          Time: 10:15 a.m.

MINUTES    0  10  20  30  40  50  60  70  80  90

MM.         5

RBC 3,410,000   HB 70   CI 1.03

WBC 12,600      Polys 70   Lymphs 17

Died April 8

Bnds 10   Yg. F. 1
Myel. Monos. 2
Eos. Bas.

Illust. 3 (cont)
following chart (Hakansson, 1928) shows the result following treatment with arsphenamine and mercury.

"The primary stage with chancre on penis and inguinal adenitis; Kahn 4+. Curve A, December 30, 1926: No treatment and has had no fever up to this time. The diagonal curved line, with a rate of 23.5 mm. indicates considerable activity, and confirms the assumption even at this early stage the infection has spread throughout the body. Curve B, January 12, 1927: Had received three intravenous injections of arsphenamine and three of mercury. Curve C, January 25, 1927: One more injection of mercury and arsphenamine. Curve D, April 26, 1927: Further treatment, two of arsphenamine and five of mercury; Kahn 4+." (Illust. 4)

The sedimentation rate in this case indicates that the tissue destruction has been checked. Although the rate has returned to normal, one must not assume that the patient is cured; the patient's serology must be negative before treatment can be stopped.

Hakansson observed the sedimentation rate after treatment in syphilis of long standing, and discovered that there is first an acceleration of sedimentation rate, probably indicating activity of dormant lesions. This increased velocity may be in the nature of a Herxheimer reaction, and as more treatment is given the
**CUTLER BLOOD SEDIMENTATION TEST**

<table>
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**TIME IN MINUTES**

- **Sed. in MM.**
  - 0 5 10 15 20 25 30 35 40 45 50 55 60

**GRAPH**

- Illustration 4
sedimentation rate will be brought back to normal.

PNEUMONIA AND COMPLICATIONS

Gallagher (1934) while studying such illnesses as bronchopneumonia found the test to be an aid particularly in those individuals whose cough persists and whose convalescence is slow. In those patients a steady decrease in the sedimentation rate gives one assurance that the trouble is gradually subsiding, or a continued acceleration will compel one to investigate more carefully for some complication.

Lesser and Goldberger (1935) in taking five weekly readings in each of 60 cases revealed a definite prognosis in the course of the disease. The acute full-blown case of pneumonia, regardless of type, showed an average reading of 80 to 100 mm. per hour with the Westergren technique, and with beginning resolutions and convalescence the study of the patients showed a gradual decrease in sedimentation readings. With the onset of complications, such as pleurisy, empyema or pericarditis, a sudden sharp acceleration in sedimentation rate was noted.

Nungester, (1937) experimenting with type three strain of pneumonoccus, prepared a specific polysaccharide, and found that this material accelerated the
sedimentation rate, and followed this up by saying that an abnormally high rate is found in the disease.

Illustration No. 5 (Hakansson) is a graphic representation of four sedimentation tests taken from a case of typical lobar pneumonia which terminated by crisis and the convalescence was without complications. "Graph A, taken January 9, 1927. Onset 5 days prior. Consolidation of right lower lobe; temperature 104.6 degrees; white blood count, 18,000. B, January 12, 1927, crisis began 12 hours ago; temperature 99.2 degrees; white blood count, 9,000. C, January 17, 1927, temperature normal for 2 days; lungs clear. D, February 12, 1927, recovered."

In these graphs one can readily note a parallel between the sedimentation rate and the other clinical findings upon which the condition and progress of the patient is usually based.

**ARTHRTIS**

Boots (1930) has written an excellent article explaining the formation of the various classifications of arthritis, which is quoted in part: "The study of chronic multiple arthritis has demonstrated the existence of two separate and distinct disease processes which are conveniently grouped under the general title. The first of these represents a multiple arthritis with
**CUTLER BLOOD SEDIMENTATION TEST**

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<td>Vertical Curve</td>
<td>Moderately or markedly active</td>
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**Diagnosis:** Lobar pneumonia

**Date:** January 2, 1927

**Time in Minutes**

| Sed. Time in MM | 0 | 5 | 10 | 15 | 20 | 25 | 30 | 35 | 40 | 45 | 50 | 55 | 60 |
|-----------------|--|--|--|--|--|--|--|--|--|--|--|--|--|--|
| Sed. Index      | 0 |   |   | 10| 20| 30| 40| 50|    | 60|    |

**Illust. 5**
additional evidence of a generalized tissue response to an infectious process; the second, a degenerative process involving the joint structures and appearing at the time of life when other degenerative changes are prone to occur. The existence of these two clinical entities is clearly recognized, but the obscurity surrounding their etiology, has precluded the adoption of an adequate and generally accepted terminology.

"The terminology most generally accepted in America is either one of the following:

   I  Chronic infectious arthritis
   II Degenerative Arthritis

   I  Atrophic arthritis
   II Hyperthrophic arthritis

"The classification adopted by the British Ministry of Health is as follows:

   I  Rheumatoid arthritis
   II Osteoarthritis"

Stainsby, (1933) while working in conjunction with Nicholls, after running sedimentation tests on 507 patients with atrophic arthritis found the test to be a reliable criterion in gauging the activity of an arthritic process, and would indicate any change in the clinical picture. Stainsby (1934) mentions the fact
that the temperature, although not elevated, and the leukocytic count within normal limits, the patient's symptoms are not a reliable index of the activity or progress of the disease, as they are readily influenced by climatic conditions, rest and exercise. He goes further by adding that even specialists in this field are many times deceived, as some of the most severe cases have few clinical signs such as swelling and tenderness. Three questions are asked by him which can be answered by the sedimentation test: "Is the disease active? How active is it? Is the patient improving or getting worse under the treatment administered to him?"

He classifies the results of treatments as follows: A sedimentation rate near normal means the case is inactive; if the rate is above normal, the disease is active; a decreasing rate indicates a favorable response to treatment.

Another investigator (Weiss, 1931) while studying 150 cases of atrophic arthritis found the sedimentation rate to be accelerated in this disease, and it did not return to normal with the disappearance of the arthritic symptoms. Three to five per cent of his cases did not show a normal rate for three to seven weeks after the patient was free of clinical symptoms. He points out that in his patients he did not find the severity of
the joint inversely proportional to the sedimentation rate.

Kling (1932) conducted a series of very interesting experiments by removing the synovial fluid from arthritic joints and placing red blood cells in this fluid. He did a sedimentation test with this mixture, and compared the readings with a sedimentation test of the true blood of the same patient. In synovial fluids from an infected joint there is an increased fibrinogen. Those which are not infected, are non-inflammatory, have a low protein content and have a low comparative sedimentation index and a low viscosity. This was also the case in transudates. On the other hand, it was found that fluids with a high content of mucin showed a low comparative sedimentation index but a high velocity. He demonstrated that the severity of the infection of the joint is indicated by the sedimentation curve in the synovial fluid. The general reaction is found in the blood curve. Using a comparative sedimentation test in monarticular arthritis, a high increase in the blood sedimentation rate is found and a low rate in the synovial fluid, which indicates a focus of infection outside of the joint as the etiological factor. The same may be used in polyarthritis. Kling is of the opinion that this may be a valuable
the joint inversely proportional to the sedimentation rate.

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aid in differentiating the atrophic type from the hypertrophic type of chronic arthritis, and that the underlying process can more accurately be diagnosed by the comparative sedimentation test.

Various authors (Merritt 1927, Oppel 1933, Keefer 1934, Stainsby 1934, Hirsh 1936, Lautmann 1937) agree with the following statements:

1. Active cases of atrophic arthritis usually give accelerated sedimentation readings of 30 mm. or over in one hour (Westergren technique).

2. A relationship usually exists between the activity and the extent of the disease.

3. Increase in symptoms almost without fail causes an acceleration in sedimentation rate, and remissions a retardation.

4. In arrested cases the sedimentation rate is inclined to remain normal.

Short, Dienes and Bauer (1937) comparing the Vernes' resorcinal test, Schilling count and streptococcus agglutination and sedimentation rate in 49 patients to determine the degree of activity of the disease in atrophic arthritis. They found the sedimentation rate positive in 92.2 per cent of the cases, the Schilling count in 87 per cent, and the remaining in 50 per cent.
Oppel (1933) and Keefer (1934) agree that patients with gonococcal arthritis invariably have an increased sedimentation rate. They find the examination is of no specific diagnostic value in these cases, but both are of the opinion that it is of great value in showing the clinical course of the active infection.

The relationship which exists between bacterial activities and chronic arthritis still requires considerable study. Regardless of the type of arthritis, some time during the disease evidence of bacterial invasion can usually be found. In the atrophic arthritis bacteria are usually believed to be the outstanding etiological factor. On the other hand, hypertrophic arthritis is usually regarded as due to senility, wear and tear and metabolic disturbances. (Lautmann, 1937).

Westergren (1928) finds no increase in hypertrophic arthritis, osteochondritis and Perthes' disease. He also finds that the sedimentation rate is often increased before the temperature, and its chief importance lies in its ability as an indicator of the intensity of the disease. Others (Dawson 1929, Oppel 1933, Merritt 1937) are in agreement with Westergren on the following:

1. In cases of hypertrophic arthritis the sedimentation rate as a rule is slightly elevated but rarely attains a greater value than 30 mm. in one hour.
Boots (1929, 1930) observed that all cases of non-articular rheumatism such as myositis, fibrositis, neuritis, showed normal or only slightly elevated sedimentation rates.

The following chart is an illustration of the patient, Mrs. O.E.J., who entered the Clarkson Memorial Hospital October 14, 1937, suffering from chronic arthritis, which at times would assume an almost acute form. The patient was diagnosed by the committee on arthritis as a very active form of hypertrophic arthritis. This patient received seven treatments in Kettering hypertherm. (Illust. 6)

RHEUMATIC FEVER

Rheumatic fever has a variety of names: acute rheumatic fever, acute articular rheumatism, acute rheumatism, and polyarthritis rheumatica. The etiology of the disease is probably infectious, and seems to be closely related to an invasion of the body by hemolytic streptococci, and is characterized by febrile and toxic states. It also invades the synovial cavities and joints, and may produce in the cardiovascular system and joints numerous disseminated focal inflammatory lesions. Rest, relief of pain, well-regulated diet, and convalescence, are the chief therapeutic in-
### CUTLER BLOOD SEDIMENTATION TEST

**Name:** O. E. J.  
**Address:**  
**Date:** October 14, 1937  
**Diagnosis:** Hypertrrophic Arthritis  

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**Illust 6.**
dications. It has been stated that during the period of convalescence extreme caution must be exercised, and that the rate of recovery is roughly proportional to the length of the illness. Before the use of the sedimentation test the patient was required to remain in bed until free from fever and other signs of infection for two or three weeks without the influence of salicylates. The white blood count must have returned to normal, the heart rate must be fairly slow, and the patient must have regained his normal weight. (Cecil, Third Edition).

There exists a close relationship between the clinical findings and the sedimentation rate, and almost every variation in the curves will show a corresponding clinical change during the disease. The most practical use of the test lies in its value as an indicator of the patient's progress, and will decide for the physician the course of treatment to follow. (Payne, 1932).

Lesser and Goldberger (1935) studied 40 cases and averaged five readings to the case. During acute clinical stages, the average readings were 90 to 100 mm. per hour (Westergren technique). They took weekly readings of the cases which gave a prognostic indication as to the course in these febrile conditions. For example, with a regression of joint symptoms, a normal white count
and temperature, the sedimentation rate was definitely decreased. On the other hand, with an increase in cardiac or joint symptoms, the rate persistently remained above normal. Other investigators, (Mossell and Duckett, 1936) after studying 178 cases, came to the same conclusions as Lesser and Goldberger, but went further and compared the white count to the sedimentation test as indicators of the course of the disease, and found a close relationship between the two.

British investigators (Bach and Hill, 1932) stressed that in a clinical picture of active juvenile rheumatism the sedimentation rate was decidedly increased, or when it is a quiescent rheumatism or rheumatic heart disease without any signs of activity the rate was within the range of normality. In children with a sedimentation rate of 11 to 21 (Westergren technique) or over without the usual clinical signs of activity, the active disease should be suspected. Merritt (1937) concurs by stating that in order to prevent a convalescent juvenile patient from getting out of bed too soon, the sedimentation rate should be the deciding factor in establishing whether the child should remain in bed or not. Stainsby (1934) has a similar opinion, that a case should not be considered inactive until the leukocyte count, temperature chart, and sedimentation tests are normal.
White (1937) is in agreement that there is an almost direct correlation between the activity of the disease and the sedimentation rate, and that this may be the only indicator as to the extent of the infection.

Others (Hench, 1934) have confirmed the work of Payne, and Bach and Hill on the value of the test in determining the difference between an improvement due to the abating of the disease or a false one due to such antipyretic drugs as salicylates.

An article on this subject (Hench) is quoted in part: "The rate is elevated in rheumatic fever and rheumatic carditis; it varies with the activity of the disease and affords one of the most delicate gauges of activity. It is normal in cases of uncomplicated chorea. However, it is accelerated in cases of chorea with carditis, and changes occur even before carditis is distinguishable. Because uncomplicated chorea is associated with a normal rate, Elgehammer suggested that it may not be a true rheumatic lesion, and Warner suggested that chorea was not an infective process although associated carditis is. According to Struthers and Bacal and to Perry the rate returns to normal or goes to less than normal with the onset of cardiac failure with edema, a sign of bad impart. Elgehammer stated that when the rate increases after removal of foci of infection he suspects
reactivation of the infection."

Lyon (1935) states the prevention of disability from rheumatic heart disease lies in the early detection of the presence of the rheumatic infection and of its subsequent periods of activity. The heart in childhood is especially susceptible to rheumatic infection. If the infection in its early stages goes unrecognized and untreated, cardiac incompetency may develop.

The following chart, illustration No. 7, is a graphic presentation of the sedimentation curves, showing the progress of a patient during and following treatment: C.T., a white male, age 12, entered the Clarkson Memorial Hospital April 21, 1937, and was diagnosed as having rheumatic fever with rheumatic endocarditis and associated chorea. On the day of admittance the patient received three hours of fever therapy. He received five treatments, totaling fifteen hours, at temperatures of 104 to 109 degrees F. The last treatment was May 1, 1937. The treatments were well tolerated, and there were no complications. During treatment the patient showed very little change in sedimentation rate, but had remarked of subjective improvement. During treatment there was a marked improvement in objective findings in the knees and ankles. On dismissal the patient had no involuntary movements; the heart had only a slight systolic murmur after exercise.
with occasional rhythm changes.

CARDIAC LESIONS

The first condition to be discussed will be coronary occlusion, also known as coronary thrombosis and cardiac infarction. Angina pectoris, which is also called stenocardia and breast pang, will also be taken into consideration.

Coronary occlusion is defined as an obstruction, usually acute, of one of the branches of the coronary arteries resulting in an infarction and death of the heart muscle supplied by the occluded vessel. (Cecil).

Angina pectoris is not defined as a disease in the true sense of the word, but as a clinical syndrome which is characterized by paroxysmal attacks of pectoral or precordial pain, syncope, and a sensation of intense anxiety. (Cecil).

Riseman (1937) pointed out that the sedimentation test is frequently a help in differentiating between coronary thrombosis and severe paroxysms of angina pectoris. He finds this especially true when the patient is seen for the first time several days following an attack, and under such adverse conditions it may be impossible to ascertain whether the fever, white count, or changes in the electrocardiogram have taken place.
CUTLER BLOOD SEDIMENTATION TEST

**Name**: C. T.  
**Address**: Clarkson Hospital  
**Date**: April 21, 1937  
**Diagnosis**: Rheumatic fever with endocarditis & chorea

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**TIME IN MINUTES**

- **Sedimentation Index**:  
  - **0**: 0  
  - **10**: 10-30  
  - **20**: 10-12  
  - **30**: 6-29  
  - **40**: 9-28  
  - **50**: 10-7  
  - **60**: 20  
  - **70**: 6-1  
  - **80**: 4-23  
  - **90**: 4-27  
  - **100**: 5-1  
  - **110**: 4-29  
  - **120**: 4-21

**Illust. 7**
A persistently normal or only slightly accelerated sedimentation rate between the fourth and twelfth day after the attack is strong evidence that there has been no myocardial infarction. Conversely, a marked acceleration in rate, especially if it shows progressive changes from day to day, may be a distinct aid in establishing the diagnosis of coronary occlusion. They believe the test to be of little value in foretelling the outcome of the disease, but found that the period during which the sedimentation rate is most rapid corresponded to the period when the maximal myocardial softening occurs. The period of recovery and healing was accompanied by a retarded rate. The mortality of the patients who were discharged from the hospital with accelerated sedimentation rates more than doubled those discharged with low rates.

In the past the gauge for determining convalescence has been the absence of an increased white count, the return of the blood pressure to normal or thereabout, and the disappearance of abnormalities in the electrocardiogram, but this was not necessarily a proof that the lesion had healed and that the patient was ready to assume a normal mode of living. In the event the patient gives a history of angina pectoris which has occurred several days prior to his examination, one should be
suspicious of a recent coronary artery thrombosis. The temperature and blood count are usually normal in such cases, and the electrocardiogram is indefinite. In cases of this type the sedimentation test is a valuable aid in differentiating between coronary thrombosis and angina pectoris. (Biern, 1937).

Hoffman (1936) does not entirely agree with Biern, as he feels that the degree of accelerated rate is no indication of the severity of the injury, and although a return of the rate to normal usually means healing of the infarction, this is not invariably true.

Shookhoff, Douglas and Rabinowitz (1936) using the Linzenmeier technique, made a study of twenty-nine cases of acute coronary thrombosis. All diagnoses were confirmed by electrocardiogram. Nine patients died, post-mortem was obtained on five of the patients, which confirmed the diagnoses. The sedimentation rates were below 60 minutes except one the fifth day, and the one case was abnormal after the end of the first week. In two cases the temperature and leukocyte count were normal throughout, while the sedimentation rates were abnormal. The majority of patients showed a fever and leukocytosis the first few days; during this time the sedimentation rate was normal. Toward the end of the first week the temperature and leukocyte count returned to normal while
the sedimentation rate was definitely abnormal. This enables one to gauge the rapidity of healing. Hoffman agrees with Shookhoff that in the majority of cases the leukocyte count and the temperature rises before the sedimentation rate. Therefore, in the first few days following the occlusion its diagnostic importance does not rank with the temperature and white cell count. Its real value lies in the fact that it usually persists for a much longer period than either of the above mentioned factors, which are often quite brief in duration and are occasionally absent.

Wood (1936) studied 22 cases of congestive heart failure, 21 of which were treated with salyrgan, and the sedimentation rate was measured at intervals after admission. He noted in all instances except one that the rate in congestive heart failure was slower than when the cardiac efficiency had been restored. In some instances the rates were fast. These were diagnosed as rheumatic carditis. Wood gives a clinical picture of a girl, age 16, who had rheumatic carditis. The initial sedimentation rate was 96 mm. in one hour (Westergren technique). Three weeks later it returned to 18 mm. in one hour. At this time this was believed to show improvement. A week later signs of congestive failure appeared. The body weight increased as edema accumulated and the sedimenta-
tion rate slowed to 10 mm. in one hour. The patient was at this time critically ill. She showed a sudden rally, and when all signs of cardiac failure had disappeared the sedimentation rate was found to be 89 mm. in one hour, almost its initial level. Immediately following a steady improvement took place; she had lost her edema and showed a natural gain in body weight, and the sedimentation rate was finally 15 mm. in one hour, showing a true improvement. It must be noted that the sedimentation rate had slowed before there was any evidence (clinical) of congestive heart failure.

In addition to the above, the sedimentation test may be an aid in differentiating between old rheumatic valvular disease, atherosclerosis, and syphilitic aortitis. It is normal in the first two, and increased in the last. Rapid rates may indicate a guarded prognosis in cases of syphilitic aortitis. He agrees with Hakannson (1928) that antiluetic treatment decreases the rate of blood sedimentation, and may be used in following the progress of the case.

Malignant hypertension is associated with an increased sedimentation rate which may be due to the degree of renal impairment. Angina pectoris is associated with a normal sedimentation rate.

The writer believes that as long as the sedimentation
rate remains accelerated there is an absorption of products of heart muscle degeneration, and that healing is completed when the rate has returned to normal. The observations of Wood that the sedimentation rate decreases with the onset of myocardial failure must also be kept in mind.

Goldbloom and Libin (1935) attempt to explain the cause of most sedimentation rate retardations in congestive heart failure by the fact that the circulating blood volume is increased in polycythemia, essential hypertension, left ventricular failure, chronic disease of the coronary arteries, and mitral valve disease.

MALIGNANT DISEASE

The value of the sedimentation rate as a diagnostic and prognostic test of neoplasms has been studied by many investigators. It is the consensus of opinion that the test possesses considerable value if the results are correlated with the balance of the clinical picture. However, one must always keep in mind the limitations of the test, and above all remember that the test is non-specific.

Rubin (1927) studied the sedimentation reaction in 127 patients with diseases of neoplastic origin. In the study of carcinoma of the breast, all sedimentation read-
ings were high. The sedimentation rate was found to accelerate with the growth of the tumor, and was even higher when weakness, anemia, loss of weight, body pains and occasionally fever were present due to toxic action of malignant cells. All cases in which metastasis had taken place, the readings were found exceedingly high. There was no correlation between temperature and sedimentation rate. Similar results were obtained with carcinoma of the esophagus, of the rectum, and of the stomach. It was mentioned that there might be a low sedimentation rate in cancer of the esophagus due to an increased blood concentration which seems to develop in these cases. It was observed that cachectic individuals tend to have a retarded rate. The test was found to have little value in differential diagnosis. Repeated tests, however, were found to be valuable in prognosis and the severity of the disease was in proportion to the accelerated rate. Rosenthall (1928) in the study of 18 cases of new growth, accompanied by a complete or non-obstructive jaundice, all diagnoses were confirmed by autopsy, X-ray, or biopsy during operation, and the sedimentation rate was accelerated in all but two cases, and one of these had polycythemia. His results agree with Rubin.

Nitschmann (1927) does not agree with Rubin on the
use of the test in differentiating between benign and malignant tumors. However, he feels that this is a method of differentiating between benign and malignant tumors of the female genital organs.

Reichel (1936) and other investigators (Rubin 1926-27, Biern 1934, Miller 1935, Hirsh 1936, Merritt 1937) are of the same opinion, that the acceleration which is brought about by neoplasia is evidently not a reaction to the tumor as such but likely to the product of cell deaths, for both malignant and benign growths are accompanied by increased rates once they have become necrotic or infected. Small tumors, accordingly, are apt to be without influence, acceleration being an indication of tissue destruction.

Reichel limits his material to tumors which have been proved malignant by operation or autopsy, and found the rate increased in 90 per cent of 76 patients with such neoplasms at one site or another. Therefore, in a doubtful diagnosis, a normal rate strongly suggests the absence of malignant disease.

Biern (1934) agrees with Reichel (1936) and Schiller (Biern 1934, Hirsh 1936) that the sedimentation rate accelerates after irradiation, which is probably due to the decomposition of the tumor with a resulting increase of the amount of foreign protein in
the blood. Schiller and Reichel find that after radical operation the sedimentation rate should return to normal within four to eight weeks. It will be noted that a sedimentation increase is found after any surgical intervention, which continues until all inflammatory exudate, necrotic material or blood has been absorbed, and a rate that does not return to normal within six months after the removal of a tumor is a sign either of incomplete extirpation or a secondary recurrence. With the extension of the neoplasm by metastasis the sedimentation rate gradually accelerates and this generally occurs before there is any clinical evidence of a recurrence. This may be used as a guide by the radiologist as an index to therapy.

The foregoing evidence clearly points out that the diagnostic value of the sedimentation test can be of only secondary importance. The primary value lies in its use as a gauge of the patient's clinical condition and the severity of the disease. Repeated tests are very valuable in determining the prognosis.

OBSTETRICS, GYNECOLOGY, MEDICAL AND SURGICAL CONSIDERATIONS

Since Fahraeus (1929) ran sedimentation tests on the blood of pregnant women in an attempt to diagnose
pregnancy, much work has been done on the subject, but it is now generally agreed that the test is non-specific in pregnancy but merely indicates the destructive metamorphosis of the fetus.

Many agree (Biern, 1934, Lesser 1935, Grodinski 1933, Falta 1927, Ringer 1934) that from the second to the fourth month of pregnancy there is a low graded acceleration in sedimentation rate up to term. Illustration 8 shows the effect of pregnancy on the sedimentation rate. (Ringer 1934)

Biern (1934) finds the sedimentation test useful in diagnosis of retention of secundines in abortion and in the diagnosis of ectopic pregnancy and other abnormalities which are characterized by hemorrhage or infection. Here the rate will be definitely increased over that which would have been the normal rate for the corresponding month of gestation. In incomplete abortion with the sepsis Lesser and Goldberger (1935) find the blood sedimentation rate only slightly increased from the normal of the corresponding month of pregnancy, but agree with Biern that with the retention of secundines and the onset of sepsis a sharp rise in the velocity of the settling blood will be noted and in unruptured ectopic pregnancy the reading is only slightly higher than the normal for the corresponding week of pregnancy.
With rupture or free hemorrhage into the peritoneal cavity, the sedimentation reading is accelerated to 80-100 mm in one hour, (Westergren technique).

Schattenberg (1932) agrees with the above, and uses the test in differentiating between ectopic pregnancy and acute exacerbation of a chronic inflammatory process. A rapid sedimentation rate points toward infection, while a slow rate may indicate ectopic pregnancy. Nitschmann (1927) in tubal pregnancy finds the rate is not accelerated unless there is an inflammatory reaction, and uses the test in differentiating between intact ectopic pregnancy and salpingitis.

Falta (1927) states that with an impending abortion the sedimentation speed is more marked. The same is true with endometritis. He adds that the first eight or ten days of puerperium the sedimentation rate remains the same as during labor, but returns to normal within three weeks.

The sedimentation rate has been used quite extensively in the field of gynecology. Most writers on the subject (Friedlander 1924, Baer 1925, Nitschmann 1927, Polak 1928, Schattenberg 1932, Grodinsky 1933) believe that the test is of value in determining the most favorable time to perform an operation. In the past, operation has always been deferred until the temperature is
CUTLER BLOOD SEDIMENTATION TEST

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ILLUSTR. 8
normal or nearly so for some time with a stationary or falling leukocyte count. Such observations coupled with the general clinical picture and local findings were the guides in determining when to operate. Polak (1928) states that he previously felt safe in discharging a patient when she had a normal temperature for one week and leukocyte count even in the presence of a pelvic exudate. His statistics show that many of these patients on returning home have been seized with severe pelvic pain accompanied by a rise in temperature, and most of these women were returned to the hospital. He believes this could have been avoided had their sedimentation rates been normal before their dismissal. It is also the consensus of opinion of the above men that a patient showing a sedimentation rate of over 18 mm. in one hour, (Cutler technique) if operated is apt to have a stormy convalescence.

In pelvic infections, according to certain investigators (Schattenberg 1932, Grodinsky 1933, Nitschmann 1927) the sedimentation test is far more reliable than the leukocyte count, and that the acceleration of the sedimentation rate occurs before the rise in temperature and increase in leukocytes, and remains for a time after the others have gone down. It is well known that the leukocyte count fluctuates during a 24-hour period, and
herein lies the danger to the surgeon who might take a blood specimen at the lower level and make the mistake of operating at this time. Had the count been repeated in several hours it would have been found dangerously high. Polak (1928) states that an error of this kind would never happen if the sedimentation rate is taken, as it never yields to erratic results. The sedimentation rate is of value as a prognostic aid which shows a gradual slowing as the patient's condition improves. The regular fluctuations in these tests do not occur as they do in leukocyte counts.

Baer and Reis (1925) state that the rate is directly proportional to the virulence of the infection.

Williams, (1927) however, does not express the same opinion as the other investigators mentioned here. He concluded that a rapid sedimentation time did not gauge to any unusual degree the postoperative morbidity, and that the temperature chart and leukocyte count are the more stable and reliable criterions in determining the diagnosis and prognosis.

Much has been written in the past on the value of the sedimentation rate in differentiating between appendicitis and salpingitis. Many writers (Grodinsky 1933, Smith 1935, Schindler 1935, Lesser 1935, Bouton 1937, Biern 1934) find that catarrhal or suppurative
appendicitis cause no increase in sedimentation rate unless they are well established cases of either abscess or generalized peritonitis. These men have all confirmed the results by operation. The following graphs (Bouton), illustration 9, are an excellent example of the accelerations in sedimentation rate which result from an acute gangrenous appendix:

"E.B., a female nurse of 19 years, suffered an attack of acute appendicitis during February, 1934. Prior to this she had been in good health.

"She underwent an operation on February 14, at which time an acutely gangrenous vermi-form appendix was removed. No other pathology was found.

"The patient's post-operative course was uneventful, and she regained her usual good health within a normal time limit.

"This series furnishes a clearcut illustration of the change in the rate of sedimentation of the red blood corpuscles in an otherwise healthy individual, brought about by an acute inflammatory process in the body, with a temporary slight rise of the curve following surgical intervention, a renewed marked
SEDIMENTATION CHART

Name: E.B.  Date: 2-13-34

Sed. I.: 31  Time: 11:45 a.m.

MINUTES  0  10  20  30  40  50  60  70  80  90

MM.  5  10  15  20  25  30  35  40  45  50  55  60  65  70  75  80  85  90  95  100  105  110  115  120  125  130

RBC 4,440,000  HB 85  CI .96
WBC 13,600  Polys 76  Lymphs 11
Bnds 13  Yg. F.

Acute gangrenous appendicitis

Illust. 9

Myel. Monos.
Eos. Bas.
SEDIMENTATION CHART

Name: E.B.  Date: 2-14-34

Sed. I.  62  Time: 10:15 a.m.

MINUTES 0 10 20 30 40 50 60 70 80 90

MM. 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100 105 110 115 120 125 130

RBC 4,440,000  HB 85  CT .96

WBC  Polys  Lymphs

Bnds  Yg. F.

Two hours prior to operation

Myel  Monos

Eos  Bas

Illust. 9 (cont)
SEDIMENTATION CHART

Name: E.B. Date: 2-15-34

Sed. I: 53 Time: 8:45 a.m.

MINUTES 0 10 20 30 40 50 60 70 80 90

MM. 5

RBC 4,290,000 HB 80 CI .95

WBC 7,200 Polys 63 Lymphs 26

Bnds 9 Yg. F. 2

Day following operation Myel. Monos.

Eos. Bas.

Illustr. 9 (cont)
SEDIMENTATION CHART

Name: E.B.  Date: 2-16-34

Sed. I: 91  Time: 8:45 a.m.

MINUTES 0 10 20 30 40 50 60 70 80 90

MM. 5

RBC 4,120,000  HB 79  CI .96

WBC 12,800  Polys 72  Lymphs 13

Bnds 13  Yg. F. 1

Drop caused by surgical trauma

Myel  Monos 1

Eos  Bas

Illust. 9 (cont)
SEDIMENTATION CHART

Name: E.B.  Date: 2-22-34

Sed. I. 61  Time 9:10 a.m.

MINUTES 0 10 20 30 40 50 60 70 80 90

MM. 5

RBC 4,120,000  HB 85  CI 1.03

WBC 10,200  Polys 62  Lymphs 29

Bnds 7  Yg. F.

Myel. 1  Monos.

Myel. 1  Monos.

Uneventful

Illust. 9 (cont)
SEDIMENTATION CHART

Name: E.B. Date: 2-28-34

Sed. I. 17 Time: 10:30 a.m.

MINUTES 0 10 20 30 40 50 60 70 80 90

MM. 5

RBC 4,070,000 HB 82 CI 1.00

WBC 8,100 Polys 78 Lymphs 17

Bnds. 5 Yg. F.

Good prognosis

Myel. Monos.

Eos. Bas.

Illustr. 9 (cont)
SEDIMENTATION CHART

Name: E.B.  Date: 2-11-34

Sed. I: 9  Time: 10:30 a.m.

MINUTES  0  10  20  30  40  50  60  70  80  90

MM.  5

10
15
20
25
30
35
40
45
50
55
60
65
70
75
80
85
90
95
100
105
110
115
120
125
130

RBC 4,510,000  HB 82  CI .91

WBC

Polys

Lymphs

Bnds

Yg. F.

Patient clinically in good health.

Myel.

Monos.

Eos.

Bas.

Illust. 9 (cont)
SEDIMENTATION CHART

Name: E.B.  Date: 1-29-35

Sed. I. 9  Time: 10:00 a.m.

<table>
<thead>
<tr>
<th>MINUTES</th>
<th>0</th>
<th>10</th>
<th>20</th>
<th>30</th>
<th>40</th>
<th>50</th>
<th>60</th>
<th>70</th>
<th>80</th>
<th>90</th>
</tr>
</thead>
<tbody>
<tr>
<td>MM.</td>
<td>5</td>
<td>10</td>
<td>15</td>
<td>20</td>
<td>25</td>
<td>30</td>
<td>35</td>
<td>40</td>
<td>45</td>
<td>50</td>
</tr>
</tbody>
</table>

RBC 4,610,000  HB 90  CI .97
WBC 8,100  Polys 78  Lymphs 17
Bnds. 5  Yg. F.

Normal check up
Myel.  Monos.
Eos.  Bas.

Illust. 9 (cont)
drop reflecting the trauma of the operation, and gradual definite return to this individual's normal.

"The fact that the curves showed no marked initial drop gave added assurance, in conjunction with temperature readings and white cell counts, that there was at no time serious pus retention or excessive tissue destruction."

Biem (1934) felt that the test was an aid in differentiating between salpingitis and appendicitis, as salpingitis gives an increased rate.

Smith, Harper and Watson (1935) found that during the first 24 to 48 hours after the onset of symptoms the sedimentation time is apt to be shorter in salpingitis (Linzenmeier technique). The reason for the shorter time in salpingitis is attributed to the fact that salpingitis is a slower disease process, which tends to be chronic and that the appendix does not have the ability to distend as rapidly as the fallopian tubes. Therefore, the earlier symptoms in appendicitis.

Lesser and Goldberger (1935), after studying 1,000 cases with an average of 1 to 12 readings per case and using the Westergren technique, observed that if the sedimentation rate is less than 20 mm. per hour this
could be considered an absolute assurance that no acute pelvic infection was present. All cases which were clinically typical were found to have readings from 40 to 50 mm. in one hour to a high of 140 to 150 mm. in one hour, depending upon the severity and extent of the acute pelvic pathology. They also took readings every week or two to determine the progress of the disease under treatment, and the optimum time for operation. It was consistently observed that long after temperature, leukocyte count, pain and tenderness had subsided, the sedimentation test became a much more accurate prognosticator of the best time to operate. No cases were brought to surgery unless the sedimentation rate was below 25 mm. in one hour, and as a result in several hundred operations no acute processes were encountered, and this was also supported by a low post-operative mortality.

Grodinsky (1933), Lesser and Goldberger (1935), and Biern (1934) found that the accelerated sedimentation rate in pyonephrosis, pyelitis, pyelonephritis, prostatitis and perinephritic abscess, and seminal vesiculities, do occasionally resemble acute appendicitis, and in these cases the sedimentation rate was high. Murphy (1937) has found that the erythrocyte sedimentation test is an important aid in determining the degree of activity
of the kidney lesion. Although the reaction is not infallible as a criterion, it was found that if the rate is normal that the chance of the complete healing of the kidney is good. But on the other hand if the rate is rapid it may indicate progressive renal damage.

Lesser and Goldberger (1935) found that acute surgical gallbladder, varying from acute suppurative to rupture, gives readings from a moderate 25 to 40 mm. in one hour to a high of 60 to 80 mm. in one hour (Westergren technique).

Ruptured peptic ulcer accelerates the rate, depending on the duration of the rupture. Biern (1934) finds the rate is retarded in peptic ulcer unless peritonitis is present.

Tuberculosis peritonitis (Lesser 1935) gives a rate from 75 to 90 mm. in one hour, differentiating it from appendicitis. They added that the group of acute chest conditions with referred abdominal manifestations also enters into the differential diagnosis of the acute surgical abdomen. The several cases with apparent acute surgical conditions of the abdomen and high sedimentations have subsequently proved to have acute pulmonary pathology such as pneumonia and acute tuberculosis. It was believed that rheumatic fever might be differentiated from appendicitis as every case of the fever,
if in its earliest incipient stage, shows a sedimentation velocity of a high 60 to 80 mm. in one hour to a severe 70 to 140 mm. in one hour.

Illustration No. 10 clearly demonstrates the nonspecificity of the test, which may be of value in giving the approximate readings for the diseases listed.

Lintz (1935) made a determination of the sedimentation rates of 30 chronic sinuses, 13 cases of chronic tonsillitis, 5 with both tonsillitis and sinusitis, and 27 with dental periapical infections. Of these cases 92 per cent had normal sedimentation rates.

Westergren (1928) found that there was little or no effect on the sedimentation rate in influenza. Gallagher (1934) found little acceleration of the sedimentation rate in common colds. Gold (1936) studied whooping cough and noted that the rate is retarded and remains normal or subnormal in the ordinary uncomplicated case without fever. He believes the sedimentation rate completes a diagnostic triad of symptoms which are; suspicious cough, lymphocytosis in association with leukocytosis, and a retarded (subnormal or normal) sedimentation rate. In general, apparently healthy children were found to have a slightly increased sedimentation rate. This he explained was due to some unrecognized minor pathology as tonsils,
<table>
<thead>
<tr>
<th>No. cases</th>
<th>Diagnosis</th>
<th>Norm. Av. 15 min reading</th>
<th>Av. 45 min reading</th>
<th>Average reading rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>26</td>
<td>Bronchopneumonia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Lobar pneumonia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Bronchitis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Acute upper resp. inf.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Pleurisy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Acute rheum. arth.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Rheum. heart disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Subacute bacterial endocard.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Atrophic arthritis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Non-acute appendicitis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Appendicitis &amp; peritonitis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Otitis media &amp; mastoiditis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Sinusitis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Tonsillitis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Abscesses</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Acute genito urinary inf.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Local infs.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Illust. 10</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. cases</td>
<td>Diagnosis</td>
<td>Norm. Av. 15 min reading</td>
<td>Av. 45 min reading</td>
<td>Average reading</td>
</tr>
<tr>
<td>----------</td>
<td>--------------------------------</td>
<td>--------------------------</td>
<td>--------------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>3</td>
<td>Burns of extremity</td>
<td>1</td>
<td>12</td>
<td>40</td>
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<tr>
<td>5</td>
<td>Ulcers of extremity</td>
<td>1</td>
<td>16</td>
<td>47</td>
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<tr>
<td>13</td>
<td>Peptic &amp; duodenal ulcers</td>
<td>1</td>
<td>4</td>
<td>16</td>
</tr>
<tr>
<td>8</td>
<td>Stomach carcinoma</td>
<td>1</td>
<td>12</td>
<td>45</td>
</tr>
<tr>
<td>12</td>
<td>Other carcinoma</td>
<td>2</td>
<td>13</td>
<td>41</td>
</tr>
<tr>
<td>4</td>
<td>Benign tumors</td>
<td>1</td>
<td>22</td>
<td>49</td>
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<td>10</td>
<td>Gen. Arteriosclerosis</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Cerebral hemorrhage or thrombosis</td>
<td>1</td>
<td>4</td>
<td>25</td>
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<tr>
<td>24</td>
<td>Hypertension &amp; cardiovascular renal disease</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>9</td>
<td>Syphilis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pernicious anemia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>a. RBC above 2.6 million</td>
<td>5</td>
<td>19</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>b. RBC below 2.6 million</td>
<td>11</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Diabetes mellitus</td>
<td>1</td>
<td>7</td>
<td>31</td>
</tr>
<tr>
<td>3</td>
<td>Enteritis &amp; Colitis</td>
<td>0</td>
<td>16</td>
<td>77</td>
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Illustr. 10 (Cont)
<table>
<thead>
<tr>
<th>No. cases</th>
<th>Diagnosis</th>
<th>Norm. read.</th>
<th>Av. 15 min reading</th>
<th>Av. 45 min reading</th>
<th>Average reading rate</th>
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<tr>
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<td>Amebic dysentary</td>
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<td>24</td>
<td>60</td>
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<td>Meningitis</td>
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<td>5</td>
<td>27</td>
<td>22</td>
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<td>Tabes dorsalis</td>
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<td>3</td>
<td>9</td>
<td>4</td>
</tr>
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<td>12</td>
<td>Post-operative</td>
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<td>26</td>
<td>80</td>
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<td>Fractures</td>
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<td>20</td>
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<td>Hypertrophy of prostate</td>
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<td></td>
<td></td>
<td>15</td>
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<tr>
<td>6</td>
<td>Non-acute gonorrhea</td>
<td>3</td>
<td>3</td>
<td>13</td>
<td>5</td>
</tr>
<tr>
<td>10</td>
<td>Chronic cholecystitis</td>
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<td>8</td>
</tr>
<tr>
<td>7</td>
<td>Kidney dis. non-nephritic</td>
<td>2</td>
<td>11</td>
<td>39</td>
<td>15</td>
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<tr>
<td>15</td>
<td>Tuberculosis</td>
<td></td>
<td></td>
<td></td>
<td>22</td>
</tr>
<tr>
<td>6</td>
<td>Misc. lung cases</td>
<td>2</td>
<td>5</td>
<td>27</td>
<td>11</td>
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<tr>
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<td>Non-infect. heart dis.</td>
<td>6</td>
<td>4</td>
<td>29</td>
<td>12</td>
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<tr>
<td>11</td>
<td>Thyroid</td>
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<td>Misc. mental &amp; nervous</td>
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<td>5</td>
<td>20</td>
<td>8</td>
</tr>
<tr>
<td>6</td>
<td>Misc. eye diseases</td>
<td>4</td>
<td>3</td>
<td>17</td>
<td>7</td>
</tr>
</tbody>
</table>

Illust. 10 (Cont)

The above results were obtained by the Westergren technique, and are quoted in part. (Haskins, 1930-31).
adenoids, dyspepsia, or flatulence.

There has not been much written on catarrhal jaundice, but Rosenthal (1938) supports the work of Gram (1921) by stating that he found a normal sedimentation rate in catarrhal jaundice. At this point I will briefly give a case history of a patient on whom I had the opportunity to do four tests. These tests had an unusually high sedimentation rate, which clearly indicated that there must have been an extreme amount of tissue destruction. This conclusion was later confirmed in the autopsy report of Dr. S. Miles Bouton:

A physical examination of the patient, S. W., in July, 1936, revealed obesity, a blood pressure of 220/108, rectocele, arthropathy of the fingers, with little else of note. The blood Wassermann was negative in 1934. On January 22, 1937, the patient appeared jaundiced and was put to bed, with a temperature of 99.2. Urine was dark, stool normal, the liver enlarged, the abdomen distended. The urine showed a low specific gravity, and the white blood count was 20,200 with 3 per cent lymphocytes. On February 3 her white blood count was 8,600, with a normal differential count. In May the patient was reported as up and dressed. In June her weight was recorded as 132 pounds. In August, 1937, she was put to bed again, when edema of the feet
and eyelids were noted. Blood pressure was 110/60, rate 84. There was marked jaundice, some weight loss, and a mild temperature, usually about 100 plus to 102 on one occasion. Urinalysis early in August showed one pus albumen, with nothing else of note. In September general edema was present, the abdomen was markedly cis-tended, and 2500 cc. of fluid (clear, amber) were removed by paracentesis. The patient appeared more comfortable, bowels and kidneys were functioning, but she was drowsy most of the time and slept much. The ascites increased, and a second tap relieved her of 3500 cc. of bile-stained fluid, in October, 1937. On October 9 and 10 the patient received morphine sulphate, because of unrelieved discomfort. She died October 11, 1937, at 1 A.M.

CLINICAL DIAGNOSIS: Dementia precox; Carcinoma of the liver (?)

ANATOMIC FINDINGS:

1. Obesity.
2. Surgical scar hernia of right abdominal wall.
3. Chronic cholecystitis and choledolithiasis, with periportal fibrosis and adhesions, chronic hepatitis and beginning cirrhosis.
4. Ascites.
5. Edema of external genitalia and perineum.
6. Pulmonary compression atelectasis, with pleural transudation. Myocardial degen-
eration with hypertrophy and dilatation of heart.

7. Moderate cerebral edema.

CAUSE OF DEATH:

Chronic cholecystitis, with periportal fibrosis, hepatitis and cirrhosis, and ascitis.

Myocardial degeneration.

(See illustration 11.)

ANEMIA

Since Fahreus (1929) first demonstrated that by diluting red cells with plasma the velocity of sedimentation was accelerated, much of the research has merely confirmed his work. After a careful review of the literature I find that very little has been added to his work concerning anemia.

Gram (1921) was perhaps the first man to follow the work of Fahreus. After analyzing the fibrin count of 542 plasmas, it was his belief that the velocity of sedimentation was due to the amount of fibrin in the plasma, and found a decreased fibrin count in diseases having a retarded sedimentation rate as in polycythemia. He also did concentration and dilution experiments, and pointed out the influence of the number of red cells on the sedimentation rate. He described a method of correcting the sedimentation rate by fixing a certain heme-
SEDIMENTATION CHART

Name: S. W.
Date: 8-20-37

Sed. I: 73
Time: 9:50 a.m.

MINUTES 0 10 20 30 40 50 60 70 80 90

RBC 2,850,000 HB 70 Cl
WBC 9,300 Polys 61 Lymphs 19
Bnads 17 Yg. F.
Myel. Monos. 3
Eos. Bas.

Illust. 11
SEDIMENTATION CHART

Name: S.W.  Date: 8-24-37

Sed. 1  140  Time: 2:57 p.m.

MINUTES 0  10  20  30  40  50  60  70  80  90

MM. 5  10  15  20  25  30  35  40  45  50  55  60  65  70  75  80  85  90  100

RBC 2,170,000  HB 50  CI
WBC 6,250  Polys 41  Lymphs 9

Fragility 27%

Bnds 49  Yg. F.
Myc.  Monos 1
Eos.  Bas.
SEDIMENTATION CHART
Name: S.W.  Date: 8-25-37

Sed. I. 133  Time: 9:55 a.m.

MINUTES  0 10 20 30 40 50 60 70 80 90

MM.  5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100 105 110 115 120 125 130

RBC  2,930,000  HB  80  Cl
WBC  7,150  Polys  38  Lymphs  22
Bnds.  40  Yg. F.
Meyl.  Monos.
Fragility 27% Na Cl
Eos.  Bas.  2

Illust. 11 (cont)
SEDIMENTATION CHART

Name: S.W.  
Date: 8-31-37

Sed.  157.5  
Time: 2:00 p.m.

MINUTES 0 10 20 30 40 50 60 70 80 90

RBC 2,810,000  
HB 60  
Cl  

WBC 11,450  
Polys 55  
Lymphs 8  

Bnds 37  
Yg. F.  
Myel  
Monos  

Eos  
Bas  

[Graph showing sedimentation curve with measurements and cell counts]
The hemoglobin values given in these graphs were determined by the Tallqvist method and are therefore not dependable.

The relationship which exists between the red cell count and the acceleration in sedimentation rate is negligible, as is demonstrated by the preceding charts.
globin percentage as normal, using 100 per cent as the standard of normality.

Rubin and Smith (1927) found that a lower hemoglobin frequently accelerates the sedimentation rate, and believed this could be correlated to the red cell count, and a low red cell count gave a corresponding acceleration in sedimentation rate.

Hubbard and Geiger (1928) allowed blood to stand in settling cylinders, and as soon as the cell had settled sufficiently they pipetted off some of the clear serum and a sedimentation test was done on the remainder. To some of the cylinders they added serum. Increased concentrations were found to retard the rate and vice versa. They also mention that in many diseases anemia is present, and if the effect upon the infectious process is to be studied adequately some attempt must be made to differentiate the effect of this secondary factor from the primary factor, anemia.

Morris (1924) found no relationship between the rate of sedimentation and red count, white count, or hemoglobin count.

These investigators (Morris 1924, Hubbard 1928, Rubin 1927) found the velocity of settling to be retarded in polycythemia and rapid in anemia.

Foster (1921) in attempting to explain the various
factors causing accelerated sedimentation rates stated that a stimulation of fibrin production, or at least a rise in fibrinogen values, was caused by the following factors: Food, hemorrhage, any type of inflammatory or tissue injury, and even certain general intoxications without obvious cell injury, and also added that polycythemia inhibits the production of fibrinogen.

The various factors, such as temperature, anticoagulants, deviation of the tube from vertical, etc., have been discussed under the Phenomenon of the sedimentation rate.

Walton (1933) applies the original formula of Blacklock (1921) which is as follows: \( \frac{X}{Y} - 1 \). \( X \) represents the original percentage and \( Y \) the desired percentage. He applied this to blood by substituting the number of millions of red cells for \( X \), and 5 million for \( Y \). Schindler (1935) also uses this method. Thus, if a patient has an erythrocyte count of 4,000,000 the calculation would be \( \frac{4.0}{5.0} - 1 \) equals 0.2 ccs., and this 0.2 cc of plasma per 1.0 cc of blood would be removed after centrifuging the cells. If the count should be above 5 million, the calculated amount of plasma would be added to each cc of blood.

Many (Newham 1926, Hubbard 1928, Walton 1933) have done dilution experiments in vitro, and demonstrated
that the sedimentation rate accelerates proportionately to the count of cells added or subtracted from the plasma.

Others (Rourke and Ernstene 1930, Rourke and Plass 1929) have worked out correction charts using hematocrit readings for standards of comparison. Wintrobe and Lansburg (1935) used normal blood with dry potassium oxalate as an anti-coagulant in their experiment. Dunn and Sharpe (1936) constructed nomographic alignment charts to correct the volume of packed erythrocytes for the amount of shrinkage due to the anti-coagulant, and to calculate the mean corpuscular volume of the cells, the mean corpuscular hemoglobin count, and the mean corpuscular hemoglobin concentration.

It has in the past been the general consensus of opinion that decreased number of red cells caused an accelerated sedimentation rate. It will, however, be found that the majority of investigators have done their experiments in vitro and very little work has been done in vivo.

Gregg (1937) has recently completed a series of experiments using the blood of rabbits made anemic by repeated bleeding. He uses the dilution method of Walton (1933), setting up a series of four sedimentation tubes containing blood in descending concentra-
tions from 5,000,000 to 2,000,000 cells per cmm. The hemoglobin was also determined by the Haden-Hauser hemoglobinometer. Whole blood was sedimented in comparison with the dilution. His results show that the whole blood did not settle as rapidly as the diluted blood.

Other investigators (Bannick, Gregg and Guernsey, 1937) have found that in a number of anemic patients who had diseases which are generally characterized by accelerated sedimentation rates that the uncorrected rates were high, but that frequently the corrected rates gave normal readings. It was also found that in longer tubes, such as the Westergren tube, that the packing of the red cells did not occur as soon as it occurs in the shorter and stouter tubes. They concluded that if single readings were to be taken at the end of one hour a Westergren tube should be used, as shorter tubes require more frequent determinations.

It is also admitted that anemia causes an acceleration in sedimentation rate, but that it is not always necessary to make a correction, as the increase in velocity caused by the anemia can be allowed. One may correct for the anemia, but it should always be kept in mind the possibility of over-correction.

Bouton (1938) substantiates the work of Bannick,
Gregg and Guernsey (1937) by stating that in not infrequent cases a low cell count in the blood has little affect on the sedimentation curve, and that any correction of the sedimenting rate and especially the use of conversion charts leads to pseudo-accuracy and may nullify the results, but if the sedimentation curve is graphically recorded and used in conjunction with other clinical and laboratory data, it is very valuable.

He further states that pernicious anemia and various leukemias have a definite and constant effect on the sedimentation rate, which is quite different from the velocity caused by an uncomplicated secondary anemia.

He adds that if one takes into consideration the initial velocity of settling and the fluctuations in rapidity during the period of reading, the curve can be directly interpreted from the results of the one hour reading and the final reading after the settling has been completed. However, corrections for anemia do not take into account the character of the curve.

DIABETES MELLITUS AND HYPERTHYROIDISM

Diabetes mellitus is a disease which occurs most often between the ages of 50 and 70. However, many
cases are found in younger persons. The sedimentation tests in younger patients (Kramer 1935) show a horizontal or only a slight increase from normal. It was found that the blood sugar had no influence on the sedimentation rate, but that out of 346 patients 67.8 per cent had accelerated rates, which is probably due to some underlying pathology.

Miller (1935) found that 52 per cent of 29 cases had increased rates, and stated that the daily wear and tear on the body tissue causes only slight variation in the sedimentation rate.

Buchanan (1935) studied 22 cases and concluded that the sedimentation rate is not indicative of pathology or tissue destruction. Inasmuch as he has obtained high readings which have remained accelerated for some length of time even in the absence of obvious disease processes, he maintains that the test leads to false conclusions. However, he neglects to take into consideration that women have a much higher sedimentation rate than men, and of 122 cases 6 were men and the highest reading was 24 mm. in one hour and the lowest 6 mm. in one hour, and for the women the high reading was 27 mm. in one hour and the lowest 6 mm. in one hour, and that 12 of these cases had complications and the remainder had comparatively low rates ranging from 12 to 16 mm.
in one hour.

Cutler (1932) and Biern (1934) agree that metabolic
diseases such as uncomplicated diabetes influence the
sedimentation rate very little.

Mora (1926) did sedimentation tests on 30 cases
of thyrotoxicosis prior to and following operation.
Prior to operation all patients showed a marked accel­
eration in sedimentation rate. The operation did not
uniformly retard the velocity of the sedimentation re­
action. He gave 11 of his patients iodine prior to
surgery, and noted the affect on the sedimentation rate.
Six tests showed a retardation in velocity which was
proportionate to the drop in the basal metabolic rate.
The remaining 5 showed a marked acceleration. He also
found that after maximal removal of the gland that three
showed little change in sedimentation rate, 13 were ac­
celerated and 14 retarded. He concluded that there was
little relationship between the sedimentation rate and
the basal metabolic rate.

Landau (1933) agrees with the other investigators
by stating that diabetes is always accompanied by normal
microsedimentation in uncomplicated cases.

Cutler confirms the above results.
ALLERGY AND SKIN

Schulhof (1933) reports 610 non-selected cases of allergy and finds that the retarded sedimentation rate is prevalent in all allergic individuals unless there is some underlying infection which may accelerate the rate. He adds that there is a frequency of slow sedimentation rates among non-allergic relatives of allergic patients, and that this has not been thoroughly investigated.

Gelford, (1933) however, found the rate remains normal before and during the season in which the allergic individual is sensitive.

Uffe (1932) found that out of 150 cases 14 per cent had high fever and all of these showed retarded sedimentation rates; 108 patients had asthma; and the majority showed normal rates. He is in accord with Schulhof that an allergic individual may show a decreased rate in the presence of some complicating infection of short duration, such as acute gangrenous appendicitis and that all allergic patients with accelerated sedimentation rates should be carefully studied for accompanying conditions.

Westcott and Spain (1932) concluded that in uncomplicated non-infectious asthma, hay fever or aller-
gic non-seasonal coryza all have sedimentation rates within the normal range.

There has not been a great deal written on the effect of the various dermatological conditions on the sedimentation rate. Tulipan and Director (1933) studied 115 patients by the Westergren method, and found the sedimentation rate usually normal in lupus erythematosus and erythema multiforme, including the bullous variety, and the generalized group of vesicular dermatoses, including dermatophytids. The sedimentation rate was found to be accelerated in tubercular lesions of the skin, erythema nodosum, and markedly increased in eruptions due to phenolphthalein, dermatitis herpetiformis. The sedimentation rate varied as to the extent of the lesion in epitheliomias and many border-line readings in mycosis, fungoids and erythroderma.

NEUROPSYCHIATRY

Up until 1928 the sedimentation reaction had been studied very little in the field of neuropsychiatry. In that year Goldwyn (1928) investigated over 200 cases in the Worcester State Hospital at Worcester, Massachusetts, and came to the following conclusions: The velocity of settling blood was directly related.
to the amount of mental deterioration, the amount of organic destruction, and the amount of toxicity present. The sedimentation rates were found to be normal in cases of manic depressive psychosis, psychopathic personalities, psychoneurosis, and paranoia. The rates were increased in senile psychosis, psychoses with cerebral arteriosclerosis, paresis, neurosyphilis, psychosis with mental deficiency, psychoses with somatic disease, acute types of alcoholic psychoses, in many cases of epileptic psychoses and involutional melancholia. Normal readings were found in the simple and paranoid types of dementia praecox, and slightly accelerated readings were found in hebephrenic and catatonic types. Unless there were complications, no cases of schizophrenia gave a noticeable acceleration.

Freeman (1933) substantiates the work of Goldwyn in a study of 50 normal and 47 schizophrenia male subjects, whom he selected because of the absence of any discernible infective processes, and found no expressive variations in the sedimentation rate in the various subclasses of the disorder. The results were all found to be within normal range.

Gregory's (1936) observations also coincide with those of Goldwyn, and he goes further to state that the high rates seen in senile psychosis and arterio-
sclerotic psychoses can, in most cases, be explained by the diseases common to persons of advanced age, such as cardio-renal respiratory diseases.

Hoverson (1934) made tests on the blood of male paretics. He believed the daily variations in red cell sedimentation rate might be explained by changes which are produced in normal or abnormal individuals by meteorological conditions, rather than by an encroachment of disease. He found wide daily variations in the sedimentation rate, as much as 100 per cent. The rate was found to be normal in paretics. It was concluded that a correlation existed between daily variations of sedimentation rate and the meteorological changes.

Westergren (1928) expresses harmony of opinion in that most organic nerve diseases show only slightly accelerated or normal rates. Border-line rates were found in tabes and paresis. Increased rates were found in multiple sclerosis during the exacerbations of the disease process; and in epilepsy and neurosis the sedimentation rates were normal. Some investigators (Stainsby 1934, Westergren 1928, Roche, 1932) used the test in determining whether a patient's complaints are on a functional basis or due to some organic disease. However, the test has only a limited value in solving this problem. If a patient shows a normal
sedimentation rate one should not be too hasty in ruling out all organic diseases, as this reaction may be due to some pathological process which does not affect the rate. On the other hand, however, if the sedimentation rate is markedly accelerated, a search must be made for the underlying cause. The usual causes of error, such as pregnancy and minor upper-respiratory infections, should be taken into consideration.

Westergren (1928) finds that polyneurotics show a markedly accelerated rate, sciatica a slight acceleration, and myalgias and neuralgias show no acceleration.

It has been the writer's experience that all types of psychoses usually show no acceleration of sedimentation rate unless, however, the patient has some complicating infection or serious mental deterioration. In syphilis of the nervous system the results were found to be variable, depending upon the activity of the disease. Although there are no graphs to illustrate this, the material is easily obtainable, as all sedimentation tests which are done at the Hastings State Hospital are kept as permanent records.
CONCLUSION

This paper does not maintain that the sedimentation test can diagnose any one disease condition; it cannot do this, as it is non-specific.

The sedimentation reaction of blood merely indicates the degree of activity and severity of tissue destruction, the products of which are absorbed in the blood.
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