Regressive vicariation thanks to Bicom 2000 — **documented with reference to darkfield images**

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Dear colleagues,

We all recognise the effectiveness of bioresonance therapy from our own experiences. For some time now, I have wondered how the effects of this therapy, and in particular of Bicom 2000, can be proved repeatedly and objectively.

Since I have long worked with a darkfield microscope, it seemed obvious to use this diagnostic method. In order to make this clearer, however, I decided to invoke a second naturopathic method and to combine the two.

Naturally, I know that many of you already work with the darkfield method and that for many *Reckeweg's* homotoxin doctrine is a well-known concept. However, many of you will also be completely unfamiliar with these concepts. Thus, for better understanding, I will introduce a few basic principles of these two methods before I proceed to describe my own trials.

DR. H.-H. RECKEWEG'S HOMOTOXIN DOCTRINE (1905-1985)

According to *Reckeweg,* diseases are biologically rational processes in which the objective is to excrete exogenous or endogenous toxins. The organism tries, in this way, to heal or at least to alleviate the damage produced by these homotoxins (i. e. „substances which are poisonous to man").

The toxin defence system of the organism acts in several phases. From this *Reckeweg* drew not only conclusions for assessing the disease process, but also the healing process.

These defence reactions are related to 3 fundamental pathophysiological principles:

- Elimination
- Deposition
- Degeneration and deformation,

Dr. Reckeweg subdivided these basic principles still further. In this way, he developed a differentiated division of the different phases of disease, the so-called table of 6 homotoxic phases (Fig. 1 on page 79).

Table of the 6 homotoxic phases

1. Excretion phase	physiological excretion processes, e. g. urine, faeces, sweat, etc.
3. Reaction phase	pathological excretion processes, e. g. eczema, abscesses, inflammation, etc.
5. Deposition phase	benign depositions lipoma, kidney stones, gall stones, etc.
7. Impregnation phase	pathological toxin storage and damage to cellular functions, e. g. viral diseases etc.
9. Degeneration phase	destruction of intracellular structures, e. g. cirrhosis of the liver, arthroses etc.
11. Neoplasm phase	structural change to the genetic material in the cell nucleus, malignant neoformation

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PHASE CHARACTERISTICS OF HOMOTOXICOSES (according to Reckeweg)

	HUMORAL PHASES			CELLULAR PHASES		
	0 Tendency to self-healing maintained 0 Enzymes remain intact 0 Excretion principle 0 Favourable prognosis dispositional diseases			0 Deterioration depending on constitution 0 Enzyme damage 0 Condensation principle 0 Gloomy prognosis constitutional diseases		
Phase according to Reckeweg	Excretion phase	Reaction phase	Deposition phase	Impregnation phase	Degeneration phase	Neoplasm phase
Main principle	Excretion Adequate physiological excretion functions	Response Pathological excretion processes, Activation of various defence functions (e. g. all inflammations)	Deposition Benign deposits of homotoxins, Demarcation of homotoxins from normally operating cell groups / tissues	Cell poisoning Damage to cellular functions and structures	Degeneration Destruction of intracellular structures and accumulation of degeneration products	Neoformation Structural change to genetic material in the cell nucleus, uncontrolled growth in affected tissue
Possible manifestation	Health Incipient problems with homeostasis	Acute diseases	Latent or subclinical diseases, incipient chronic diseases	Chronic diseases, latent diseases	Chronic-degenerative conditions	Malignant neoformation

(from Dr. v. Ingelheim)

Fig. 1 Phase characteristics of homotoxicoses (according to *Reckeweg)*

The first three phases — the **humoral phases —** relate in particular to excretion. In the case of the fourth to sixth phases — the **cellular phases —** cell damage is triggered.

Reckeweg described the transition from the humoral to the cellular phase as the **biological section,** this being important when assessing a prognosis: to the left of the biological section the organism can still compensate for the problem condition; to the right, compensation proves very difficult and ultimately impossible.

The homotoxicosis phases can change as a disease progresses. This change in overall symptoms is often associated with a change in the actual tissue involved. This is well-known, even in traditional medicine, above all in the case of asthma and eczemas. The stress changes from entodermal to ectodermal, and vice versa. This phenomenon of tissue change is termed vicariation.

In the event of a change to the right and/or down the 6-phase table, we refer to progressive vicariation (deterioration and poorer prognosis), in the case of a shift to the left and/or up we refer to regressive vicariation (improvement, better prognosis).

That covers *Reckeweg.*

These basic concepts of homotoxin teaching are a prerequisite to understanding what I am going to talk about.

Now, before I can explain the link between the *Reckeweg* phases and blood images in a darkfield in more detail, I must also go into a little more detail about a few basic terms used in darkfield diagnostics.

PROF. ENDERLEIN'S DARKFIELD BLOOD TEST

Darkfield tests on live blood provide information about the abundance and upward development of the tiny protein bodies present in all humans, endobionts. These are passed on with the egg cell and live in symbiosis with us. Every mammal has, in each individual cell, the symbionts Mucor racemosus and its companion Aspergillus niger. These two have the task of serving the organism in enzyme and energy processes and in blood clotting. The endobionts are present here in their apathogenic lower valency form. Under certain conditions, however, they can develop into pathogenic forms. According to *Prof Enderlein,* microorganisms and higher, more complicated structures such as bacteria, viruses and moulds are produced by the upward development of these protein colloids.

The higher valency pathological endobionts are parasitic and therefore develop a metabolism of their own. This places an even greater strain on the human host organism.

A prerequisite for the upward development of endobionts is a change in milieu towards overacidification and the presence of excess protein. As a result of stress, environmental problems and increased consumption of protein, this frequently occurs in the modern day and age. Any chronic disease, according to Enderlein, is accompanied by an upward development of endobionts. The more acidic and toxic the milieu, the more highly developed forms occur. And the more highly developed these forms become, the more harmful they are and the more diseased is the organism!

Reckeweg describes in his `Cyclogeny' the various possibilities for upward development of endobionts along three different cyclodes (development paths) which provoke corresponding different states of disease.

In contrast to a traditional blood test, in a darkfield test the milieu and the stresses acting on the blood are assessed by examining endobiontic change in the cellular elements. Therefore, the test is performed not on stained dry blood, but with live blood.

The upward development of endobionts is regarded as a cause of disease and is also important with regard to the prognosis for the disease. Accordingly, the various stages of development can also be related to the Reckeweg phases.

You can see from Fig. 2 on page 81 which darkfield images correspond to which homotoxicosis phase.

SEQUENCE OF TESTS WITH Bicom 2000

And now to my series of tests. In order to assess the effectiveness of Bicom 2000, I followed the procedure set out below:

- Before therapy, blood was taken from the patient, from the fingertips. The patients were fasting when the blood samples were taken. These drops of blood were observed under the darkfield microscope and the image was allocated to the relevant homotoxicosis phase.
- Then basic therapy and a course of therapy based on Cross-linked Test Technique was administered with the Bicom 2000.
- 10 minutes after completion of the therapy, blood was again taken, observed under the microscope and again allocated to the correspon-

PHASES of HOMOTOXICOSES (according to Reckeweg) — in relation to DARKFIELD DIAGNOSTICS

	HUMORAL PHASES			CELLULAR PHASES		
Phase acc. to Reckeweg	**Excretion phase**	**Reaction phase**	**Deposition phase**	**Impregnation phase**	**Degeneration phase**	**Neoplasm phase**
Main principle	**Excretion**	**Response**	**Deposition**	**Cell poisoning**	**Degeneration**	**Neoformation**
Characteristics in darkfield image	0 Protite veil 0 Many plasmatic low valencies 0 „postprandial" image	0 Many protites 0 Low to medium valencies 0 Reactive leucocytes 0 Diecothecites	0 Symplasts 0 Roll formation 0 Reactive and rigid leucocytes 0 Endobiosis of leucocytes	0 Intracellular endobiosis prevails A 0 Rigid leucocytes 0 Honeycomb production 0 Filit production	0 Poicilocytoses 0 Autolytic leucocytes 0 Increase in honeycomb and filit network production	0 Many high vale ncies 0 Sclerotic plasma forms 0 Increase in all previous endobiosis indicators
Possible manifestation	**Health** **Incipient problems with homeostasis**	**Acute diseases** **Defence mode!**	**Latent or subclinical diseases, incipient chronic diseases**	**Chronic diseases, latent diseases**	**Chronic-degenerative conditions**	**Malignant neoformation**

(from Dr. T. Rau)

Fig. 2 Phases of homotoxicoses (according to *Reckeweg)* — in relation to darkfield diagnostics

ding *Reckeweg* phase. Thus it was ensured that, apart from Bicom 2000 therapy, no other effect such as medicaments or diet could have contributed to the change in the blood.

It was shown that, as a result of therapy using only the BiCom 2000, the organism had moved to a different starting position. Changes occurred in the blood image which may be characterised as regressive vicariation.

It is striking that this phenomenon is currently expressed much better with stresses in the humor- al phases than with stresses in the cellular phases. And yet even in patients with malignant neoplasia, there was a clear improvement in the direction of the biological section (see Figs. 3 to 16).

Changes noted after treatment with the Bicom 2000

- Massive activation of leucocytes and a clear increase in the number of leucocytes.
- Intracellular endobiosis clearly decreased.
- The erythrocyte shapes changed in the direction of isocytosis.
- Diecothecites were produced (positive auxiliary cells)
- Protite activity increased

Almost all patients were allocated homotoxicosis phases after therapy which lay at least one step further left in the table of hornotoxicosis phases.

The detailed results of the trial are given in the following tables:

Change in darkfield after therapy with Bicom 2000
(number of test subjects: 26)

Increase in leucocyte activity:	84.6 %
Decrease in roll formation:	65.4%
Decrease in anisoc osis:	5 0 %
Increase in number of protites and production of diecothecites:	5 0 %
Decrease in intracellular stresses on erythrocytes:	46.1
Clear shift of pH to higher values (more basic)	65,4%

Regressive vicariation after therapy with Bicom 2000
(number of test subjects: 27)

Deposition phase --> Reaction phase	46.1
Impregnation phase Deposition phase	11.5%
Impregnation phase --> Reaction phase	15.4%
Degeneration phase —> Impregnation phase	7.6%
Degeneration phase —> Deposition phase	3.8%
Homotoxicosis phase	15.4%

This means that Bicom 2000 therapy brings about regressive vicariation in the blood!!!

Now to the following darkfield images

Various development phases are visible in any blood smear under the darkfield microscope. Each of the following figures correspond to one prevailing image.

Patient (male): born 1936, **status post colonic carcinoma**

Before therapy:

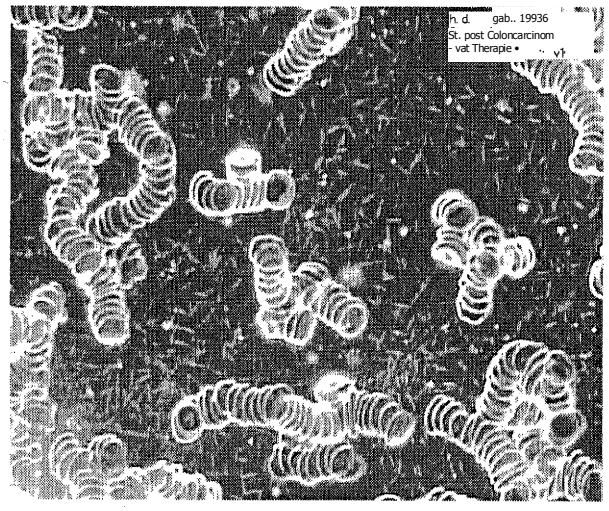

Fig. 3

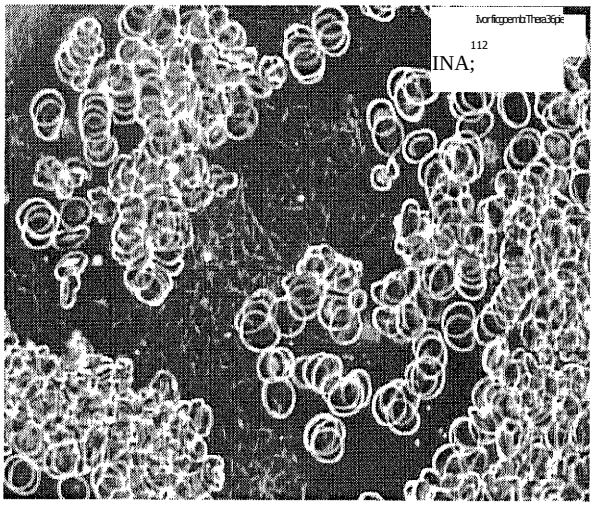

Fig. 4

Heavy roll formation in erythrocytes, close flit network, sporoid symprotites.

After therapy:

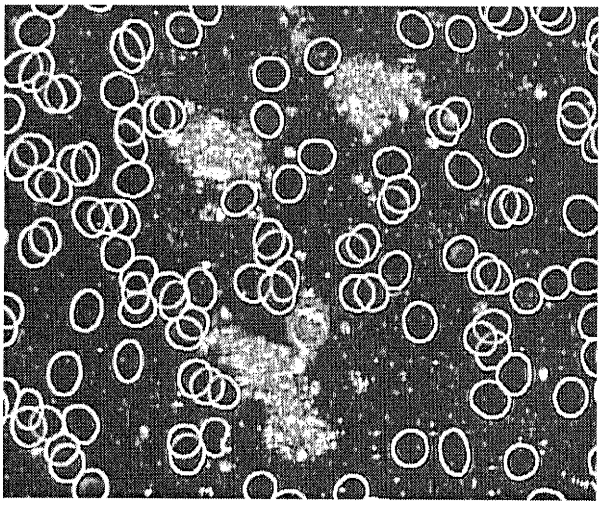

Fig. 5

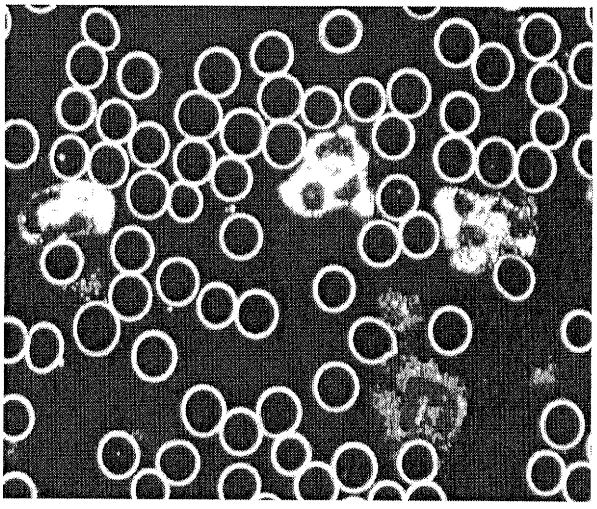

Fig. 6

Much looser erythrocytes, more active leucocytes, looser accumulation of protites.

Patient (male): born 1966, **allergic bronchial asthma**

Before therapy:

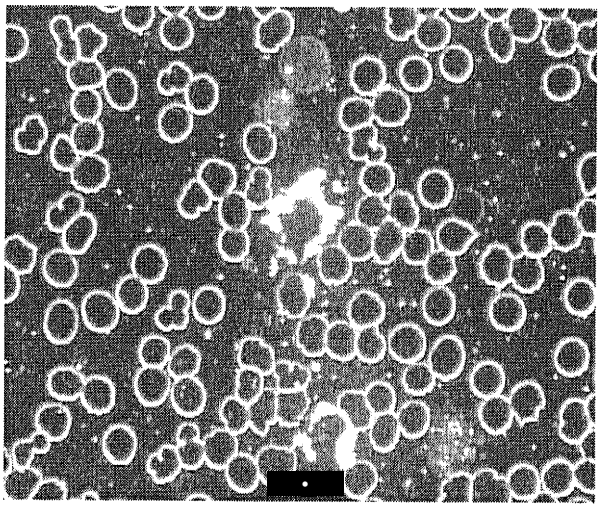

Fig. 7

Roll formation present, autolytic leucocytes, erythrocytes apple- and lemon-shaped.

After therapy:

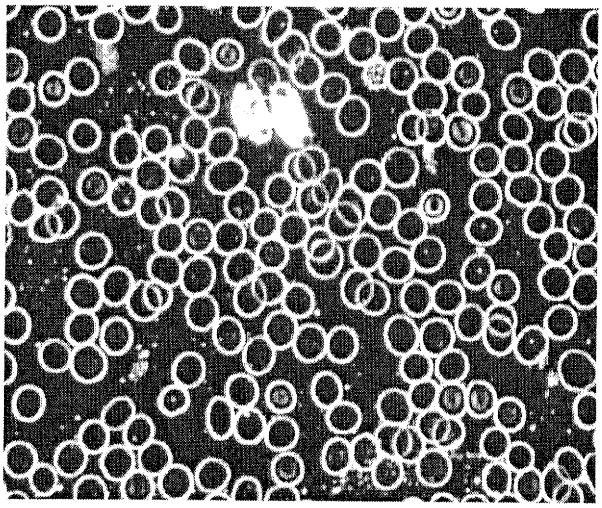

Fig. 8

Reduced roll formation, active leucocytes, increased number of protites.

Patient (male): born 1923, hypernephroid carcinoma

Before therapy:

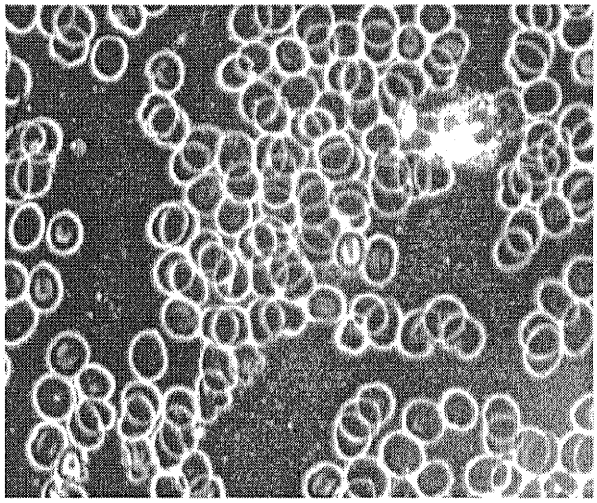

Fig. 9

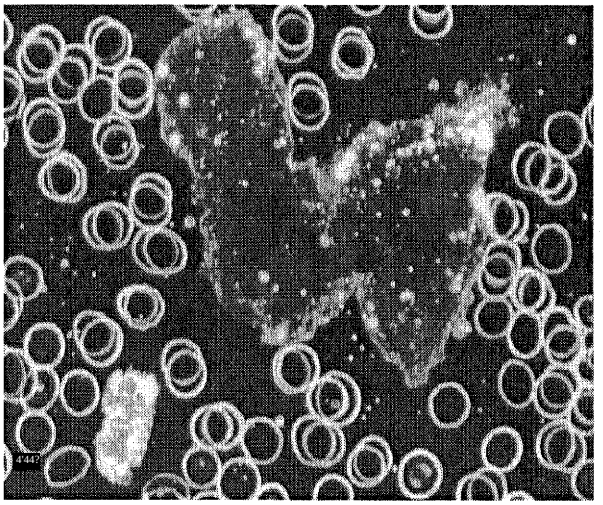

Fig. 10

Erythrocytes clumped together, giant symplast, slight roll formation, scarcely any protites.

After therapy:

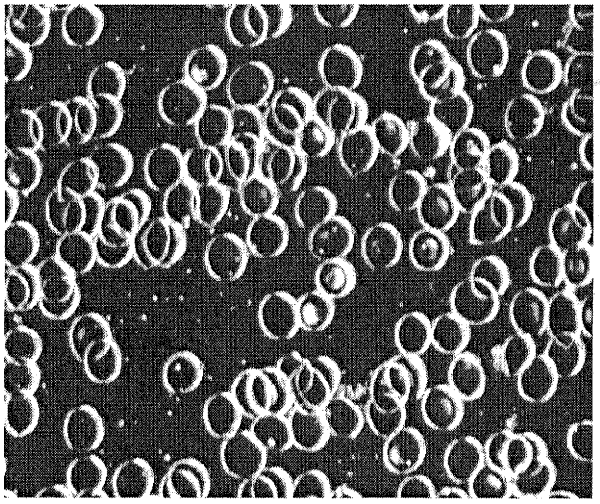

Fig. 11

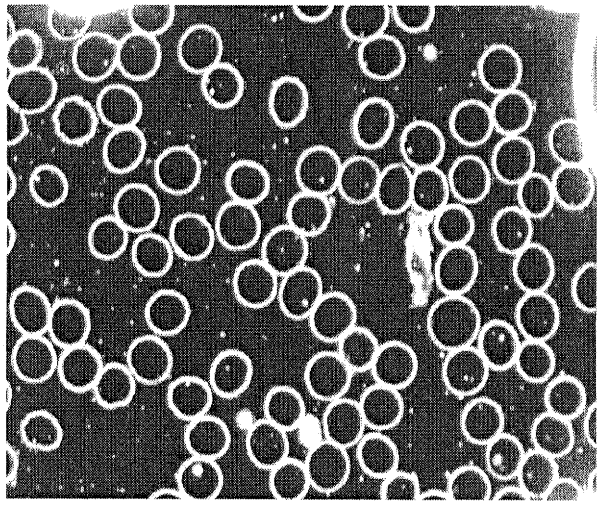

Fig. 12

Less clumping together of erythrocytes, clear increase in number of protites, erythrocytes much more rounded.

Patient (female): born 1958, diagnosis: **hepatopathy, art. hypertonia**

Before therapy:

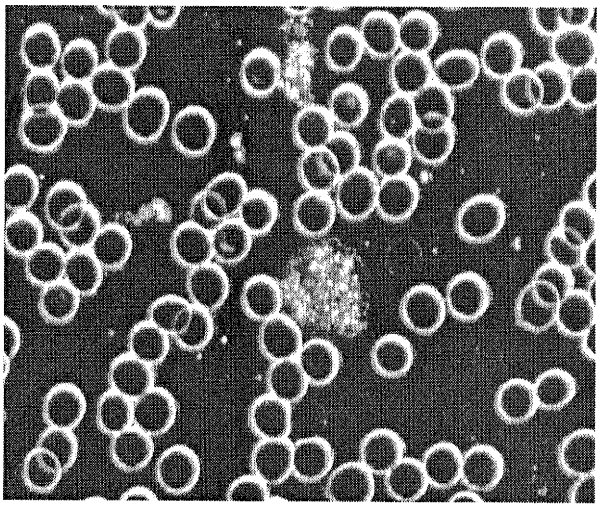

Fig. **13**

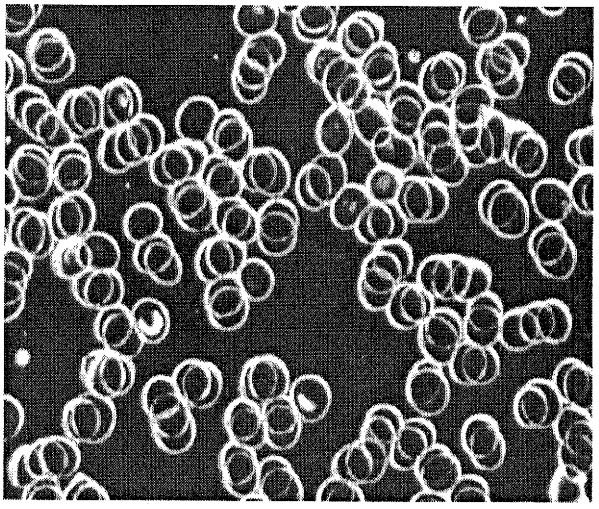

Fig. 14

Relatively loose roll formation, leucocytes rigid to autolytic, scarcely any protites.

After therapy:

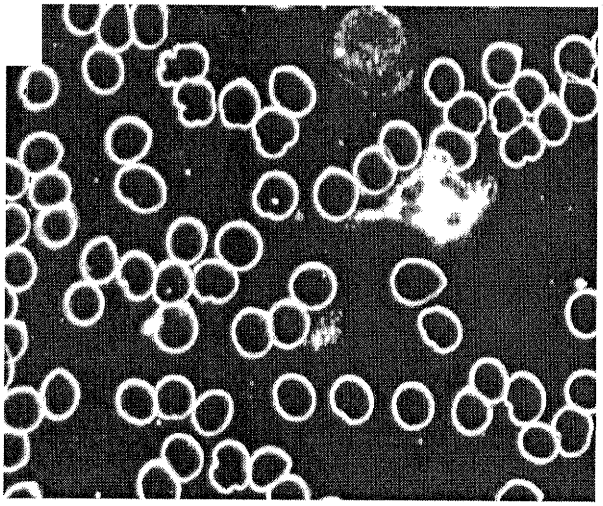

Fig. 15

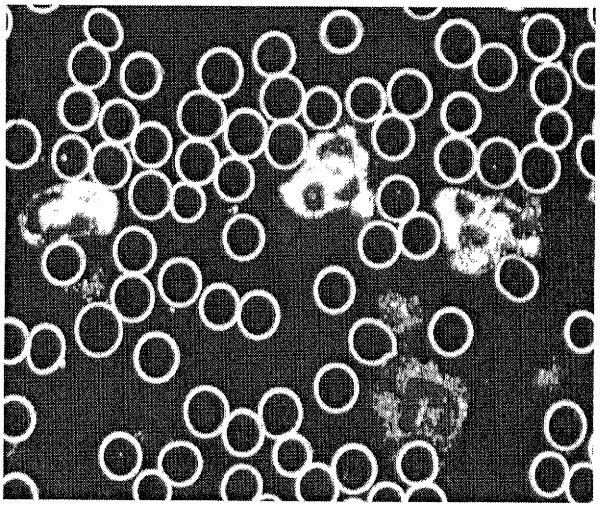

Fig. 16

Clear increase in leucocyte activity, no more roll formation!