FATTY DEGENERATION OF THE BLOOD
AND
THE SCHARLACH GRANULATION.

BY C.T. HAND NEWTON.
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The occurrence of general fatty degeneration is well known, and has been the subject of extensive investigation. Its chief causes are profound anaemia and certain forms of toxaemia, which in some cases are definitely of bacterial origin, while in others this is assumed to be the case. But the toxic process is not necessarily the result of a bacterial or allied indirect cause, for definite fatty degeneration results from certain inorganic poisons, e.g. Phosphorus, Arsenic, Antimony, Chloroform and Iodoform.

In the profound anaemias the fatty degeneration results from the reduction in the oxygen-carrying power of the blood, while in the cases of toxic origin it results from the injurious action of the toxins upon the cells of the tissue.

The subject of fatty degeneration has been widely studied as affecting many important organs
and tissues of the body, particularly with reference to the heart, liver, kidneys and arteries. The exciting poison is certainly carried to the affected organs by the circulating blood, and this suggested to Shattock and Dudgeon that a similar change might occur in this tissue also.

Conceiving of the blood then as a complex tissue, and subject like any other to parasitic and bacterial infection, or partaking in the morbid changes of many other specific diseases, they imagined it would likewise be subject to degenerations, and more particularly to the chief and most widespread of all of them, viz. fatty.

In considering diseases of the blood there is a double question involved: there are, first, the changes in the blood arising from disease of the blood forming organs; and, secondly, the abnormal changes of the formed blood, arising from lesions proper to the blood itself.

Previous to the investigations of Shattock and Dudgeon, the blood degeneration which had been most studied was the so-called "Glycogenic Reaction of the Blood". This consists in the appearance of mahogany-coloured granules of variable size in the leucocytes and plasma. Czerny claimed that these granules were in reality not glycogen, but of a carbohydrate substance more nearly related to
amyloid. Huppert's later studies on the blood of animals support the belief that the substance thus demonstrated is really glycogen.

Ewald thinks it is probable that many colorless globules visible in leucocytes treated by ordinary staining methods are referable to the former presence of glycogen, which is soluble in water, and of which the reactions are largely destroyed by heat.

Most authorities now consider that theiodophilic degeneration is glycogenic. Gabritschewsky using this method, has found intra- and extra-cellular glycogen in the blood of both healthy and diseased persons. Extra-cellular glycogen, in the form of fine or coarse granules, he found to be the only form usually present in normal blood, and it was increased in diseases in which intra-cellular glycogen was abundantly present. He considered that extra-cellular glycogen is derived from the disintegration of leucocytes but has put forward nothing to prove this origin.

Livierato also found extra-cellular glycogen in normal blood but never glycogen within the leucocytes. Many other workers have investigated the subject and are almost all agreed that the appearance of glycogen granules within the leucocytes is pathological.
Czerny attempted to decide the exact chemical nature of the granules, and decided they are glycogen for the following reasons:

(1) Because of their reaction to Iodine.
(2) The disappearance of their brownish stain on heating.
(3) Their complete digestion by saliva.

Gulland finds that the amount of extracellular glycogen varies greatly in different cases, and the only disease in which it seems to be uniformly increased is diabetes. In all other conditions it follows no ascertained law and is to be disregarded. He agrees with Best that the substance stained is not pure glycogen but probably a rather loose combination of glycogen with some other substance, possibly proteid in character. Gulland believes the change is not a degenerative one but is probably associated in some way with the resistance of the organism to poisons, and especially to those which produce positive chemotaxis.

Whatever the exact nature of this degeneration, it presents marked differences from the granules described by Shatlock and Dudgeon, and these will be discussed later.

**Technique.**

In my own investigations I have rigidly
adhered to the technique adopted by Shatton and Dudgeon, in order that the results may be fairly compared.

It is essential to exclude the use of fat solvents, such as absolute alcohol and ether, as fixing reagents, and essential also that the blood should not be dried. Blood clots were at first dried, but proved less satisfactory than fresh blood films.

The blood film is made in the usual way and at once placed, with the film downwards, in a specially devised chamber of formol vapour.

The chamber used by them, and also by myself, was a deep oblong glass trough with a ground edge, and was slightly larger in sectional area than an ordinary microscope slide. This was packed nearly to the top with cotton wool thoroughly soaked with formol, (40 per cent), and on the top of the wet wool was laid a grating of perforated zinc near either end of which was soldered a cross strip of zinc wire, the latter being added to prevent the film from touching the grating through which the vapour passed and fixed the film. The chamber was covered with a piece of over-lapping plate glass, and vaseline was painted over the edge.

The film, being prepared, was placed in the formol chamber and allowed to remain there for at
least 15 minutes, and was then placed vertically in a glass of Scharlach for 24-48 hours.

The use of alcoholic solution of Scharlach as a fat stain requires much care for the dye readily precipitates, and this I found personally was very difficult to avoid. The solution used was prepared by saturating 75 per cent absolute alcohol in the cold, and subsequent filtration through paper first wetted with alcohol.

After 24-48 hours had elapsed the slide was removed from the Scharlach solution, washed for a few seconds in 75 per cent alcohol, then in distilled water, and after this stained in a bath of haemalum for three minutes. This was followed by washing first in distilled and then in tap water, the film being finally mounted in Farrant's medium and a coverglass applied. I have found that many of the films prepared in this way do not retain their characteristic features for any length of time.

In their article published in April 1906, Shatlock and Dudgeon give the results of their examination of the blood by this method in 78 cases.

The following is a list of diseases in which the blood was examined for fatty degeneration by them:-
Pernicious Anaemia.
Toxaemia of Pregnancy.
Subphrenic Abscess.
Chlorosis.
Diphtheria.
Appendicitis.
Lymphadenoma.
Acute Meningitis.
Post-Nephric Abscess.
Anaemia of undetermined origin.
Myelaemia.
Influenza.
Exophthalmic Goitre.
Acute Pneumonia.
Chronic Bright’s Disease.
Purpura.
Rheumatic Pericarditis.
Anaemia (Secondary)
Septic Nephritis.
Myxoedema.
Pleurisy with Pericarditis.
Septic Broncho-Pneumonia.
Carcinoma of Pylorus.
Sub-Acute Rheumatism.
Diabetes with Lipaemia.
Hepatic Suppuration.
Ulcerative Endocarditis.
Rickets.
Acute Cerebro-spinal Meningitis.

Diseases in which fat was found in the leucocytes.

Chlorosis.
Toxaemia of Pregnancy.
Chronic Bright’s Disease &
  Chlorotic Anaemia.
Acute Pneumonia.
Purpura.
Carcinoma of Pylorus with
  Chlorotic Anaemia.
Diabetes with Lipaemia.
Myelaemia.
Influenza.
Lymphadenoma.
Pleurisy with Pericarditis.
Acute Cerebro-spinal Meningitis.

In the foregoing list of diseases in which fat was found in the finely granular polymorpho-
nuclear leucocytes, some in which a fatty degen-
eration might have been particularly anticipated, are conspicuous by their absence. Two such would certainly be diphtheria and pernicious anaemia. In both these diseases, however, Shattock and Dud-geon found the leucocytes laden with fine granules, which, though deeply stained, do not exhibit the transparency and proper red colour of ordinary fat. These granules are of a deep brown colour and the phenomenon itself has been called the "Scharlach Granulation".

This Scharlach granulation they observed in the following diseases:

- Subphrenic Abscess
- Exophthalmic Goitre
- Pneumonia with Empyema
- Pernicious Anaemia
- Diphtheria (6 cases)
- Appendicitis
- Pericarditis (Rheumatic)
- Acute Meningitis

The following is an analysis of the cases which I have myself personally investigated.

Case 1. Lymphadenoma.

History: Male, aged 17, was admitted to the Manchester Royal Infirmary in April 1910. There extensive involvement of the glands on the right side of the neck, with some less prominent glands on the left. A few glands in the right axilla were affected but not those of the groins.
The patient was operated on in 1904, and the glands then enlarged were removed but recurrence took place within a year.

A complete blood examination showed:

Red Blood Corpuscles 4,032,000 per C.M.M.
White Corpuscles 10,000 "
Haemoglobin 60 per cent.

Differential Count of Leucocytes:

Polymorphonuclear Leucocytes 77 per cent.
Lymphocytes 15 "
Large Mononuclears 7 "
Mast cells 1 "

Scharlach Reaction.

In a certain number of the polymorphonuclear leucocytes, and in the plasma, there are to be seen some transparent red granules. These vary somewhat in size, but are not present in the mononuclear leucocytes or red corpuscles.

Case 2. Recurrent Appendicitis.

A woman, aged 21, was admitted to the Manchester Royal Infirmary in April 1910. On admission there were well marked signs and symptoms of appendicular abscess. Appendicectomy was performed and the abscess cavity drained. Suppuration persisted for the 4 weeks she remained in hospital, associated with an average daily temperature of 102 F., and a pulse of 118. She had frequent rigors, and when she left the hospital there was no improvement in her condition.
The blood examination was made on the day following the operation.

Red Blood corpuscles  4,100,000 Per C.M.M.
Leucocytes           13,200   "   "
Haemoglobin          70        " ' "

Differential Count of Leucocytes.
Polymorphonuclear Leucocytes  85 Per cent.
Lymphocytes            12        " ' 
Large Mononuclears     2         " ' 
Eosinophils            1         " ' 

Scharlach reaction.

Many of the polymorphonuclear leucocytes show fine red granules which are neither present in the plasma nor in the lymphocytes and large mononuclears.

Case 3.
Acute Appendicitis.

A woman, aged 50, was admitted to the Manchester Royal Infirmary in April 1910. For 14 days she had had abdominal pain and vomiting. On admission a hard mass, the size of an orange, was palpable in the right iliac fossa. The abdomen was opened and a drainage tube was inserted after evacuation of a considerable quantity of pus. The appendix was not removed.

Two days later the patient developed lobar pneumonia, and death occurred seven days after admission.

The blood was examined the day after the first signs of pneumonia were noticed.
Red Corpuscles 4,720,000 Per C.M.M.
Leucocytes 24,000 " "
Haemoglobin 90 Per cent.

Differential Count.
Polymorphonuclear Leucocytes 88 Per cent
Lymphocytes 9 " "
Large Mononuclears 3 " "

Scharlach reaction.
Some of the polymorphonuclear leucocytes contain clear red granules which are also present in the plasma.

Case 4.
Tetanus.
A man, aged 47, was admitted to the Manchester Royal Infirmary on April 6th, 1910, with the following history:

On March 10th, while cutting a hedge, he wounded the back of his right hand.

On April 1st he noticed some stiffness of his jaw, and later stiffness of his chest and abdomen. On April 5th he complained of pains in the legs.

On admission patient could only open his mouth with great difficulty for \( \frac{3}{4} \) inch, the masseter muscles being in a condition of clonic spasm. Slight risus sardonicus. On palpation the muscles of the abdomen were rigid and boardlike, while there was also some rigidity of the hamstring muscles.

Patient was treated with anti-tetanic serum and made an apparently complete recovery.
Scharlach reaction.

No granules, red or chocolate, are present in any of the polymorphonuclear leucocytes, mononuclear leucocytes or plasma.

Films were prepared on three separate occasions.

Case 5.

Tetanus.

A man, aged 35, was admitted to the Manchester Royal Infirmary on April 9th, 1910.

The symptoms were definite but the case was not so serious as Case 4. Patient was treated with anti-tetanic serum and recovered.

Scharlach reaction.

No red or chocolate granules were found in any of the films prepared.

Case 6.

Parenchymatous Goitre.

A girl, aged 18, was admitted to the Royal Infirmary, Manchester, in May 1910. The goitre first appeared in 1906 and steadily increased in size till the date of her admission to hospital.

A hemi-thyroidectomy was performed, and the wound healed by first intention.

Blood examination. (Before operation)

Red Corpuscles 4,389,000 Per C.M.M.
Leucocytes 9,800 " "
Haemoglobin 80 Per cent.
Differential Count.

Polymorphonuclear Leucocytes 70 Per Cent per C.M.M.
Lymphocytes 21 " "
Large Mononuclears 6 " "
Transitional Leucocytes 3 " "

Scharlach reaction.

In some of the Polymorphonuclear leucocytes can be detected brown granules, varying in number in different leucocytes. None are present in the mononuclear leucocytes or plasma.

Case 7.

Gastric Ulcer.

A woman, aged 35, was admitted to the Manchester Infirmary in May 1910. Diagnosis was confirmed at the operation, and a gastro-enterostomy was performed.

Blood examination.

Red Corpuscles 4,035,000 per C.M.M.
Leucocytes 7,400 " "
Haemoglobin 75 per cent.

Scharlach reaction.

No red or brown granules can be found in any of the leucocytes.

Case 8.

Sub-Acute Appendicitis.

The patient, a woman, was admitted to the Manchester Royal Infirmary in May 1910. There was a history of abdominal pain, nausea and vomiting extending over 5 days. On admission her temperature was 99 and her pulse 90. There was marked tenderness at McBurney's point, but no other definite
signs. Operation was performed a week later when an inflamed and thickened appendix was removed. Patient made a rapid and uncomplicated recovery.

**Blood examination (Before the operation).**

Red Corpuscles 4,830,000 Per C.M.M.
Leucocytes 13,300 " "
Haemoglobin 90 Per cent.

**Differential count.**

Polymorphonuclear Leucocytes 68 Per cent
Lymphocytes 26 " "
Large Mononuclears 2 " "
Eosinophils 1 " "
Transitional Leucocytes 3 " "

**Scharlach reaction.**

A few of the polymorphonuclear leucocytes show Scharlach granules which are not present in the other leucocytes or the plasma.

**Case 9. Chlorosis.**

A woman, aged 22, was admitted to the Royal Infirmary, Manchester, in May 1910, complaining of headaches and shortness of breath. A typical case of chlorosis both in general symptoms and in the condition of the blood.

**Blood examination:**

Red Corpuscles 3,320,000 Per C.M.M.
Leucocytes 7,000 " "
Haemoglobin 40 Per cent.
Differential count.

Polymorphonuclear leucocytes 67 Per cent.
Large Mononuclears 3 " "
Lymphocytes 30 " "

Scharlach reaction.

Very many of the polymorphonuclear leucocytes contain red droplets which vary greatly in size and number of different leucocytes. These granules are also abundantly present in the plasma.

Case 10.

Pernicious Anaemia.

The patient, a male aged 39, was admitted to the Manchester Royal Infirmary in May 1910. The case was a severe one with a history of symptoms extending over two years.

The lemon-yellow colour of skin was well marked, and the patient suffered from severe palpitation and frequent attacks of diarrhoea and vomiting.

Red Corpuscles 1,192,000 Per C.M.M.
Leucocytes 4,200 " "
Haemoglobin 55 Per cent.

A film stained with Leishman showed considerable irregularity in size and shape of the red corpuscles and also well marked polychromatophilia. Some normoblasts and one or two megaloblasts were detected.

Scharlach reaction.

Many of the polymorphonuclear leucocytes show numerous transparent red granules, and in addition
In one or two polymorphonuclears the granules are darker and non-refractile, but not typical Scharlach granules.

Case 11. Pernicious Anaemia.

The patient, a man of 42, was admitted to the Manchester Royal Infirmary in May 1910.

The symptoms were well marked and the disease had lasted for several years.

Blood examination.

Red Corpuscles 2,100,000 Per C.M.M.
Leucocytes 3800 "
Haemoglobin 50 Per cent.

A blood film showed marked irregularity of the red corpuscles, polychromasia, and the presence of many nucleated red cells.

Scharlach reaction.

Many of the polymorphonuclear leucocytes contain red granules similar in character to those previously described. Some red translucent droplets are present also in the plasma.

Case 12. Oxalic Acid Poisoning.

The patient, a woman aged 62, was admitted to the Manchester Royal Infirmary with the history of having swallowed two teaspoonfuls of oxalic acid. On admission she was very cold and collapsed. Her pulse was weak and feeble, and she frequently
vomited a dark reddish-brown material.

She complained of severe pain in the throat and stomach, and was fed per rectum for several days. Death occurred on the 6th day.

Scharlach reaction.

Scattered throughout the plasma and also in the polymorphonuclear leucocytes are very great numbers of red granules varying considerably in size and intensity of redness. No brown granules are to be seen.

(Several films were prepared at different times and all showed the same condition.)

Case 13. Carcinoma of Vagina.

Patient, a woman aged 46, was admitted to the Manchester Royal Infirmary in June 1910.

The condition was inoperable. Her temperature rose occasionally to 99.5 but never higher. There was slight anaemia of the chlorotic type present.

Scharlach reaction.

Some of the polymorphnuclear leucocytes show small red droplets or granules which are also present in the plasma.


Patient, a man aged 40, was admitted to the Manchester Royal Infirmary in June 1910.
He had had dysentery in India two years previously and had not been well since. The liver was enlarged and tender. An abscess was evacuated and the pus found to contain amoebae dysenterica which were also detected in the stools.

Blood examination.

Red Corpuscles 4,300,000 Per C.M.M.
Leucocytes 11,000 " "
Haemoglobin 67 Per cent.

Differential count.

Polymorphonuclear leucocytes 78 Per cent
Lymphocytes 14 " "
Large Mononuclears 6 " "
Eosinophils 2 " "

Scharlach reaction.

Almost all the polymorphonuclear leucocytes contain red granules which are also abundantly present in the plasma.

Case 15.

Acute Yellow Atrophy.

Patient, a woman aged 27, was admitted to the Manchester Royal Infirmary in May 1910. Severe jaundice, diminution in liver dulness, vomiting and haematemesis, coma and death were the leading symptoms. Leucine and tyrosine were detected in the urine and the diagnosis was confirmed by post-mortem examination.

Scharlach reaction.

Some of the polymorphonuclear leucocytes contain fine red granules, which are of small size.
A few are to be seen in the plasma, but not in the mononuclear leucocytes.

Case 16. Sciatica.

Patient, a man aged 33, was admitted to the Manchester Royal Infirmary in June 1910.

The case was of moderate severity but presented no unusual features.

Scharlach reaction.

No abnormal granules are present in the leucocytes or plasma.

Case 17. Sub-Acute Osteomyelitis.

Patient, a boy aged 13, was admitted to the Chelsea Hospital for Children in March 1911. There was swelling and tenderness over the lower end of the right tibia. There was a moderate degree of constitutional disturbance.

The bone was opened and some thin sero-purulent fluid escaped, which, on examination, yielded a pure culture of staphylococcus aureus.

Blood examination:-

Red Corpuscles 4,920,000 per C.M.M.
Leucocytes 14,600 " "
Haemoglobin 95 Per cent.

Scharlach reaction.

A few of the polymorphonuclear leucocytes show coarse chocolate granules which are not present in the plasma or mononuclear leucocytes.
Case 18. Acute Osteomyelitis.

Patient, a boy aged 11, was admitted to the Chelsea Hospital in March 1911.

The lower end of the right femur, close to the epiphysial junction was the part affected.

There was severe constitutional disturbance, the temperature being 102.5 at the time of operation.

The bone was trephined, pus liberated, and the wound drained.

Blood examination.

Red Corpuscles 5,100,000 Per C.M.M.  
Leucocytes 17,200 " "  
Haemoglobin 90 Per cent.

Scharlach reaction.

Many of the polymorphonuclear leucocytes contain dark brown granules, varying in number in different leucocytes, in some only a few being present while in others they are so numerous that the leucocytes resemble a mulberry in appearance.

No Scharlach granules are present in the plasma or mononuclear leucocytes, and no red granules are present at all.

Case 19. Severe Burn.

Patient, a girl aged 2½, was admitted to the Chelsea Hospital in March 1911. There was a severe burn involving the right side of the neck and the upper part of the chest. Sepsis followed with
marked constitutional disturbance, the pulse being very rapid and the temperature sometimes reaching 0 104.5 F.

Leucocyte count 16,300 Per C.M.M.

Scharlach reaction.

In one or two of the polymorphonuclear leucocytes can be detected granules of a brown colour.

Case 20.

Tuberculous Peritonitis.

The patient, a boy aged 8, was admitted to the Chelsea Hospital in March 1911.

The abdomen was moderately distended and contained some free fluid. Patient showed marked wasting, and had occasional vomiting but no pain. Scharlach reaction.

No brown or red granules can be found in the leucocytes.

Case 21.

Tuberculous Peritonitis.

The patient, a boy aged 5, was admitted to the Chelsea Hospital in March 1911.

The abdomen was prominent and palpation yielded a dough-like resistance most marked on the right side. There was no free fluid present and there was only a moderate degree of wasting. No signs of tuberculosis elsewhere. Scharlach reaction.

None of the polymorphonuclear leucocytes contain any abnormal granules.
Case 22. Acute Lobar Pneumonia.

The patient, a boy aged 5, was admitted to the Chelsea Hospital in March 1911. The lower lobes of both lungs were consolidated and death occurred on the 4th day after admission.

Blood examination.

Red Corpuscles 4,670,000 Per C.M.M.
Leucocytes 22,700 " "
Haemoglobin 85 Per cent.

Scharlach reaction.

No granules, red or brown, can be detected in any of the leucocytes.

(Several slides were prepared at different times but none showed any abnormal granulation.)

Case 23. Diphtheria.

The patient, a boy aged 5, was seen at the Out-Patient Department of the Chelsea Hospital in April 1911. There was a marked membrane involving the tonsil and the neighbouring part of the palate. A swab was taken and a positive bacterial result obtained.

Scharlach reaction.

In some of the polymorphonuclear leucocytes can be seen very fine brown granules somewhat lighter in tint than those previously met with.

Case 24. Phthisis.

The patient, a boy aged 6, was admitted to the
Chelsea Hospital in April 1911.

There was marked dulness on percussion at the apices of both lungs and also over the root of the right lung. The breathing in these situations was bronchial with crepitations. The illness had already extended over some months. There was severe night sweating and Von Pirquet's reaction was positive.

Scharlach reaction.

Most of the polymorphonuclear leucocytes appear normal but on careful searching one or two can be found containing Scharlach granules.

Case 25. Acute Nephritis.

The patient, a boy aged 6, was admitted to the Chelsea Hospital in March 1911.

On admission there was a moderate degree of oedema with blood and albumin in the urine. When the blood was examined the boy had been 17 days in the Ward and his condition was very much improved, there being no blood in the urine and only a trace of albumin.

Blood examination.

Red Corpuscles 3,980,000 Per C.M.M.
Leucocytes 6,800 " "
Haemoglobin 65 Per cent.

Scharlach reaction.

The majority of the polymorphonuclear leucocytes appear perfectly normal, but one or two show
dark brown granules. No red granules are present.

Case 26.  
Epithelioma of Floor of Mouth.

The patient, a man aged 45, was admitted to the Cancer Hospital, London, in March 1911.

The growth started 7 months ago and the extensive involvement of the glands of the neck rendered the case inoperable.

Scharlach reaction.

A few of the polymorphonuclear leucocytes show well marked Scharlach granulation. No red granules are present.

Case 27.  
Rodent Ulcer.

The patient, a woman aged 48, was admitted to the Cancer Hospital in March 1911.

There was extensive deep ulceration of the face exposing the termination of the common carotid. Patient was being treated with X Rays with no improvement.

Scharlach reaction.

No abnormal granules are present in the leucocytes.

Case 28.  
Pneumonia.

The patient, a boy aged 6, was admitted to the Chelsea Hospital in April 1911.

Physical signs showed consolidation at the base of both lungs, apparently of the lobar type.
The blood was examined four days after admission. The pulse was then 154 and the temperature 103.5.

Leucocyte count 18,800 Per C.M.M.

Scharlach reaction.

In neither the leucocytes nor the plasma can be seen any red or brown granules.

Case 29.

Empyema.

The patient, a child aged 9, was admitted to the Chelsea Hospital in April 1911.

There was marked dulness at the base of the left lung, and the breath sounds at this situation were bronchial in type and distant in character. An exploring needle withdrew some pus which on examination proved to be pneumococcal.

A rib was resected to ensure free drainage of the empyema.

Scharlach reaction.

No abnormal granules can be detected.

Case 30.

Empyema.

The patient, a child aged 11, was admitted to the Chelsea Hospital in April 1911.

There was nothing unusual in the symptoms and physical signs and the diagnosis was confirmed by exploration.

Scharlach reaction.

No abnormal granules can be detected in the blood.

(Several other slides were prepared from this
case but the result was always negative.
Shattock and Dudgeon as the result of their investigations came to the following conclusions:—

**Fatty Degeneration of the Blood.**

In the toxic cases the fatty change present in the polymorphonuclear leucocytes results from the direct action of the toxic substances present in the blood itself, i.e. the change is a true fatty degeneration, and not merely a storage of fat by a healthy cell. The fineness of the fatty points, their varying number in different cells in the same film, and the fact that they occur in abundance in cases in which the condition of the patient excludes the possibility of so large an amount of fat being merely ingested from the blood by the circulating leucocytes, all point to this conclusion.

The fact that such an acute fatty degeneration actually does occur in the renal epithelium and cardiac muscle, proves the ability of the toxins to effect such damage also on the circulating leucocytes.

The degeneration of the leucocytes occurs in the blood, for if it had occurred whilst the cells were still in the marrow they would not be likely to enter the circulation.

The free droplets of fat in the plasma are probably derived from the disintegration of leucocytes which have suffered very severe degeneration.
This is not the sole source of the free fat in the blood, for in some cases its amount is too great for such an explanation.

Most of the free fat must be regarded as derived from the intestine, or as transferred from the common connective tissue to the blood plasma for the use of the more important organs. The fat in some of the leucocytes in the circumstances is doubtless ingested, but the fact that in some of the cases where the fat in the leucocytes was pronounced, no free fat was present in the plasma, shows that this cannot be advanced as the sole explanation.

The presence of fat in the polymorphonuclear leucocytes does not indicate a grave prognosis.

The fatty changes are limited to the polymorphonuclear leucocytes, i.e. to the cells which take the most active part in phagocytosis: and do not occur in the lymphocytes which are almost devoid of phagocytic power.

In Chlorosis, the fatty changes occurring in the polymorphonuclear leucocytes are attributable to the deficient oxygen-carrying power of the blood; and as the reduction in this function affects the tissues supplied by the blood, so may it affect the nutrition of the leucocytes in the blood itself.
Scharlach granulation.

That the granulation is not a mere precipitate of Scharlach within the cytoplasm of the cells that have been imperfectly fixed, is shown by the fact that the diphtheria blood films, (in which, amongst others, it occurred) were fixed in formol vapour for over twenty-four hours.

The granules of the normal eosinophil leucocytes exhibit a somewhat similar coloration though of a less intense degree, but while they are equally well coloured in both pathological and normal blood films after the films have been treated with absolute alcohol, the Scharlach granules, on the contrary, are not demonstrated by means of Scharlach if the films are first treated with absolute alcohol.

The Scharlach granulation is not due to the coloration of the proper granules of the cell, for in some of the leucocytes comparatively few of the brown granules occur, and in these cells the proper granules are recognisable though unstained.

The granulation is not unlike the glycogenic, as displayed in the leucocytes after treatment of blood films with iodine solution. The Scharlach granulation, however, is not to be observed in control films if treated with absolute alcohol before they are transferred to the dye.
The Glycogen reaction, on the contrary, is as readily obtained after this treatment as without it.

The absence of the Scharlach granules when the blood films have first been treated with absolute alcohol excludes the possibility of their being blood pigment which has been ingested by the leucocytes.

The Scharlach granulation is not due to a substance allied to lecithin or myelin for neither of these is coloured with the Scharlach solution.

The granulation is indicative of a degeneration allied to that known as fatty.
The following is a list of the diseases in which the blood was examined for fatty degeneration by myself.

1. Lymphadenoma.
2. Recurrent Appendicitis.
3. Acute Appendicitis.
4. Tetanus.
5. Tetanus.
6. Parenchymatous Goitre.
7. Gastric Ulcer.
8. Sub-Acute Appendicitis.
11. Oxalic Acid Poisoning.
12. Carcinoma of Vagina.
13. Amoebic Dysentery.
15. Acute Yellow Atrophy.
17. Sub-Acute Osteomyelitis.
18. Acute Osteomyelitis.
19. Severe Burn.
20. Tuberculous Peritonitis.
22. Lobar Pneumonia.
23. Diphtheria.
24. Phthisis.
25. Parenchymatous Nephritis.
26. Epithelioma of Floor of Mouth.
27. Rodent Ulcer.
28. Pneumonia.
29. Empyema.
30. Empyema.

In the following diseases fat was found in the leucocytes.

Lymphadenoma.
Recurrent Appendicitis.
Acute Appendicitis.
Chlorosis.
Pernicious Anaemia (2 cases)
Oxalic Acid Poisoning.
Carcinoma of Vagina.
Amoebic Dysentery.
Acute Yellow Atrophy.
In the following diseases the blood showed Scharlach granulation:

- Parenchymatous Goitre.
- Sub-Acute Appendicitis.
- Acute Osteomyelitis.
- Sub-Acute Osteomyelitis.
- Severe Burn.
- Diphtheria.
- Phthisis.
- Parenchymatous Nephritis.
- Epithelioma of Floor of Mouth.

When the cases examined by Shattock and Dudgeon, and also those of my own series, are analysed it will be found that most of them may be considered as of toxic origin. In some cases this toxaemia is acute, e.g. Appendicitis and acute yellow atrophy, while in others it is of a more chronic nature, e.g. lymphadenoma.

The intensity of the toxaemia does not appear to be the only factor determining the variety of the resulting granules, for while the blood of some acute cases shows Scharlach granules, in other cases, equally acute, fatty degeneration of the blood is present.

Nor does there appear to be any constant relation between the appearance of fatty or Scharlach granules and a leucocytosis, for in some cases the leucocytes are about normal in number, and in others they are greatly increased.

Certain of the cases were associated with severe constitutional disturbance, including...
considerable elevation of the temperature, but the fatty change was not confined to such cases but was, indeed, found to be best marked in certain cases in which the temperature was very little affected, e.g. Chlorosis and pernicious anaemia.

Similarly the Scharlach granulation was found very highly developed in two cases of osteomyelitis in which there was pyrexia, but was also found to occur where there was little or no elevation of the temperature in goitre, phthisis and nephritis.

The appearance of both forms of granulation then appears to bear no constant relation to either pyrexia or leucocytosis.

Shattock and Dudgeon found fatty degeneration present in acute pneumonia, and Scharlach granulation in empyema. I investigated two cases of pneumonia at different stages of the disease, and two of empyema, but in none of the many slides prepared from each case could I discover any abnormality in the blood.

Two of my cases of appendicitis showed fatty degeneration of the leucocytes and one Scharlach granulation, while Shattock and Dudgeon found brown granules in the leucocytes of the only case of appendicitis they investigated.
The Plate.

The illustrations in the plate are taken from my own slides, and were drawn by Mr. Richard Muir.

Case I shows marked fatty degeneration of the blood: Case 18 Scharlach granulation, while Case 21 shows the appearance of normal blood after staining for 48 hours with Scharlach solution.

Many of the slides prepared failed to retain their original appearance, becoming considerably altered after some time had elapsed.
Literature Consulted.

S.G. Shattock & L.S. Dudgeon,
"Fatty Degeneration of the Blood."
Transactions of the Pathological Society of London, 1907.

R.G.M. Buchanan,
"The Blood in Health and Disease" 1909.

J. Ewing,
"Clinical Pathology of the Blood."

William Osler,
"The Principles and Practice of Medicine"

J.M. Beattie & W.E. Carnegie Dickson,
"Textbook of Special & General Pathology."

Richard C. Cabot,
"Chapters 14 & 15 System of Medicine"
(Osler & McRae) Vol. 4, 1908.

J.H. Drysdale,
"Clinical Examination of the Blood & its Significance."
(Allbutt & Rolleston) Vol. 1, 1905.

G. Lovell Gulland,
"The Glycogen Reaction in Blood".
British Medical Journal,