3. Inhibitory and Circulatory Factors Combined.

This is most conveniently illustrated by the congenital mobile colon, but, in preface, it is only fair to insist that the actual position of the colon means nothing whatever. What is really important is its means of support in the upright position.²⁵ The mesentery is *not* (and never was designed as) a supporting structure. As a result of this pathological imposition there are two results:

(a) Sympathetic inhibitory stimuli (with consequent general stasis, flatulence, etc.)—a mild mimicry of the mechanism and symptoms already portrayed in the picture of acute appendicitis (Fig. 2).

(b) Again the most collapsible structures suffer first. The resulting tension on the mesenteric veins leads to venous stasis (evidenced clinically by the tortuous arteries so commonly seen during operations on these subjects), and accentuates the

FIG. 3.—Showing tension on mesenteric vessels and concomitant sympathetic fibres in colopostosis.

existing intestinal inefficiency due to inhibition (Fig. 3). In subjects of visceroptosis these two factors combine to produce the symptoms which are in the experience of us all.

As an example, we are all familiar with the patient of sturdy athletic build, with well-fixed colon, who suffers no symptoms from (or clinical evidence of) constipation, even if this extends over several days. A similar constipation in the subject of visceroptosis leads to fœtor of the breath, discomfort and distension, flatulence, anorexia, etc. Is not this comparison again a mild mimicry of the picture we have already considered when we contrasted the absence of constitutional symptoms associated with chronic obstruction with the profound changes seen in strangulation? Can it be doubted that the venous factor is equally responsible for the symptoms in chronic mesenteric tension (visceroptosis) as it is in acute strangulation?¹⁰

Again, the end-result of venous stasis and the temporary intestinal inefficiency which results, ultimately is fibrosis. Fibrosis not only involves the muscle, but, far more serious, may (and in long-standing cases undoubtedly does) involve Auerbach's plexus, thereby dislocating the whole neuromuscular mechanism of the intestine.⁴

Surely it is our duty to anticipate this eventual and incurable sequel of venous stasis by imitating Nature's normal method of mesenteric fusion, and performing the simple operation of colopexy, in suitable cases. The success of such a procedure, carried out on sound principles, depends on the selection of cases for operation; and, from what has been said, this is often no simple problem.

In conclusion, it is submitted that the term "ileus" should present to us a picture of intestinal inertia of two types: (1) active ileus—a physiological and defensive mechanism due to inhibitory nervous impulses; and (2) paralytic ileus—a pathological and progressive condition due to venous obstruction.

In chronic form the symptoms are a mild mimicry of those we are familiar with in acute abdominal crises; but the principles on which treatment should be based are identical—namely: "active (physiological) ileus" should always be imitated and supplemented; while every effort should be directed towards the prevention of increased venous pressure (from any source) and the possible onset of "paralytic ileus."

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URGENT UTERINE HAEMORRHAGE OF CONSTITUTIONAL ORIGIN.

BY

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LOCAL or pelvic causes account for by far the majority of cases of abnormal uterine haemorrhage, which, according to Beckwith Whitehouse, may be classified into four groups: epimenorrhoea (menstrual periods too frequent), menorrhagia (periods profuse), menostaxis (too prolonged), and metrorrhagia (irregular bleeding). Minor degrees of any of these conditions are occasionally met with associated with constitutional disturbances or disorders of metabolism, such as myxoedema, exophthalmic goitre, acute infectious diseases, and the anaemias. Shaw,¹ in his very detailed work on irregular uterine haemorrhage, has noted that in 17.5 per cent. of his series of 200 cases of excessive uterine haemorrhage no explanation for its etiology could be offered. He states that it is always extremely difficult to attribute menstrual abnormalities to general metabolic and endocrinal disturbance, for in the majority of cases there is no obvious association, and it is undoubtedly true that, in the past, any association that there may be has been grossly exaggerated. He admits that it is possible that in many of the 200 cases investigated some such conditions have been present. Schmitz,² in a survey of 2,184 consecutive hospital records and 2,126 records of out-patients, found that about 36 per cent. of them complained of uterine haemorrhage. No diagnosis as to the cause of the bleeding was made in 22 cases of the in-patient series and in 23 cases of the out-patient series. Linton, Marks, and Smith³ remark that possible factors in the etiology of abnormal flowing without demonstrable pathological conditions are, among others: local or systemic endocrine dysfunction, subclinical constitutional blood disease, and systemic blood disease. According to Munro Kerr,⁴ the common blood diseases, such as anaemia and chlorosis, are associated with amenorrhoea; far more grave disorders, such as scurvy, purpura, etc., are generally accompanied by menorrhagia. Gow is quoted as having shown, in a study of mitral stenosis, that in about 20 per cent. of cases only was menorrhagia present, despite the hitherto general belief that the two conditions were associated. Munro Kerr states that haemophilia is a possible cause of uterine haemorrhage, but Osler⁵ quotes Bulloch and Fildes's claim to have established the fact of immunity in females and their denial of the authenticity of all the published cases. In Grandidier's series of 334 cases of haemophilia uterine bleeding was not instanced. Meigs⁶ has described a few interesting and unusual cases of benign uterine bleeding, excluding the usual causes, such as fibroids, inflammation, polypi, etc. They fell under the following headings: hyper-

thyroid, hypothyroid, pituitary disorder, thrombopenic purpura haemorrhagica, atypical purpura haemorrhagica, endocrine, and cases with no definite pathological findings. Possibly, in many clinical cases of mild or moderate uterine haemorrhage a general cause is overlooked. When bleeding is severe it almost always has a local cause.⁷

Profuse uterine haemorrhage, severe enough to endanger life and of constitutional origin, is undoubtedly uncommon, and in a review of approximately 19,000 in-patient records (from 1920 to 1930) in the Royal Samaritan Hospital for Women, Glasgow, I have encountered only seven cases of such a category. The main features of these cases are detailed below.

CASE I.

This girl, aged 15 years, was admitted urgently to hospital on February 22nd, 1921, suffering from profuse haemorrhage from the vagina. It was of sixteen days' duration, and hitherto she had not menstruated. Bleeding was continual, and had necessitated her remaining in bed. On examination she was found to be blanched, the lips, gums, and conjunctivae being almost colourless; she was poorly nourished, and haemic murmurs were present. No pelvic abnormality was detected on rectal examination.

Four years previously purple spots appeared one day "under the arms and breasts." On the following day epistaxis commenced, and was accompanied by sickness and vomiting. On two further occasions, at two-monthly intervals, nasal bleeding recurred. It was noticed by her mother that, prior to each haemorrhage, the patient became excited, and there was apparently involuntary blinking of the eyes. Each attack of epistaxis lasted about twenty-four hours and ceased spontaneously. Since 1917, too, successive crops of purple spots appeared and disappeared, and on one occasion there was some diffuse purple discoloration of the legs; there was no history of trauma, and there was no history of "bleeders" in the family.

Blood examination: The red cells numbered 1,600,000 per c.mm., the white cells 30,000. Polychromatophilia, poikilocytosis, and nucleated red cells were seen.

On March 8th she was transferred to Glasgow Victoria Infirmary for medical treatment. On admission she was blanched, breathing was shallow, the pulse small and weak, and she complained of thirst. She was given subcutaneous and intravenous salines. No abnormal constituents were present in the urine. On the following day she was examined under light general anaesthesia, and the uterus and appendages were found to be apparently normal. The cervix was dilated and the uterus curetted. She was given pituitrin (1/2 c.cm.) four-hourly, and 3 ounces of saline containing 30 grains of calcium lactate, four-hourly, per rectum; this was continued for seventy-two hours. For three or four days after admission she rallied, but then became delirious and developed gangrene of the right foot and of the toes of both feet. For ten days she was given, by the mouth, a mixture containing the chloride, lactate, and iodide of calcium. However, her condition became worse, and she died on March 20th—that is, six weeks after her first uterine haemorrhage commenced. Post-mortem examination revealed no features of note.

Unfortunately a detailed blood examination, including platelet count and bleeding time, was not carried out in this case, but from the history a presumptive diagnosis of thrombopenic purpura haemorrhagica may be made. Price⁸ states that in purpura haemorrhagica, menorrhagia in the adult is frequent and is a dangerous symptom. Meigs's⁶ case of this condition was that of a girl, aged 14 years, who had had four transfusions to stop nasal bleeding. Later, she had continuous uterine haemorrhage for three months, at the end of which her haemoglobin was 35 per cent. Transfusion and splenectomy were performed. Two days later the platelets and bleeding time were normal, and uterine bleeding stopped almost at once. Her periods subsequently were normal and regular. Linton, Marks, and Smith⁹ report the results of treating by radium a series of 250 cases of uterine bleeding not due to malignant disease. One case (a young girl) in this series was due to idiopathic thrombopenic purpura, and was treated by three local radium applications and intensive ultra-violet ray treatment over the spleen; she is now apparently well, nine years after her first admission.

CASE II.

A girl, aged 14 years, was admitted to hospital on May 25th, 1922, as an urgent case of profuse uterine haemorrhage. Bleeding had commenced ten weeks previously, and had continued without interruption. On examination she was blanched, the pulse rapid, and the heart sounds soft but pure. The only feature of her past health was that "she had always been anaemic."

She was given elixir calcium chloride, 1 drachm thrice daily, and the foot of the bed was elevated. Two days later the pulse was still rapid and very poor in quality, and rectal salines were administered. Her condition remained serious for several days, but, when improvement showed, she was examined per rectum under anaesthesia, and no gynaecological abnormality was detected.

Blood examination (ten days after admission): The red cells numbered 4,775,000 per c.mm., white cells 11,875; haemoglobin 45 per cent.; colour index 0.47. The erythrocytes were pale in colour, and a few nucleated red corpuscles were seen. There was some poikilocytosis. No myelocytes were present, polymorphonuclear leucocytes predominating.

The diagnosis in this case was that of chlorosis. The usual medical treatment was employed, and she was dismissed well on June 23rd, 1922.

Almost eight years later (May 9th, 1930) I saw this patient, and was informed that periods had been normal and regular for several years. She was of small stature and generally underdeveloped, her appearance suggesting that she was still very anaemic. I examined her blood, with the following results: red cells 4,500,000 per c.mm., white cells 7,800; haemoglobin 50 per cent.; colour index 0.5. The erythrocytes were pale in colour, and "ring-staining" was marked. There was some poikilocytosis. No nucleated red cells or anisocytosis were present. No myelocytes were seen, polymorphonuclear leucocytes being in normal proportion. The blood picture, it is interesting to note, was still of the chlorotic type.

CASE III.

A patient, aged 45 years, was admitted to hospital as an urgent case on April 29th, 1924, complaining of very profuse menstruation. For twelve months prior to admission menstrual periods had been very irregular; her three most recent ones had extended for five to seven days, bleeding being very profuse and large clots being passed. On examination she was found to be very pale and the pulse thready; systolic murmurs were present at the apical and aortic areas of the heart.

Puberty was at 12 years of age, and menstruation had been regular and normal until the age of 44. With the exception of a slight attack of pleurisy, her past health had been good.

Blood examination: The red cells numbered 3,300,000 per c.mm., white cells 10,400; haemoglobin 80 per cent.; colour index 1.2.

Two days after admission she was examined under general anaesthesia; the uterus was dilated and curetted and the curettings examined microscopically. A small mucous polyp was found, but neither in it nor in the endometrium was there any evidence of malignancy. On the day following operation she developed phlebitis of the left leg. Three days later her condition became critical, and despite liberal stimulation and treatment with brandy, digitalin, strychnine, and salines, she died on the eighth day after admission to hospital.

Unfortunately a more detailed blood examination was not made in this case, and a suggested diagnosis of pernicious anaemia could not be made with certainty.

CASE IV.

This patient, a woman aged 43, was admitted to hospital on April 21st, 1926, as an urgent case of uterine haemorrhage. Bleeding per vaginam had been constantly present for the preceding three weeks, and for four days prior to admission had been extremely profuse. An ergot mixture from her physician had made no appreciable difference. She was found to be exsanguinated and in a critical condition. Her temperature was 96.4°, pulse rate 100, and respirations 20 per minute. Examination of the pelvis revealed no local or pelvic abnormality.

The patient had suffered for several years from "internal bleeding piles." There was also a history of myxoedema three years previously, and for this she had had thyroid treatment, which had latterly been discontinued. She had been married sixteen years, had five children and no miscarriages, and labours and puerperia had been uneventful. The menopause was eighteen months prior to her urgent admission to hospital, and in this interval there was no uterine bleeding whatever.

Blood examination: The red cells numbered 980,000 per c.mm. A blood film showed a polymorphonuclear leucocytosis; the red cells were of normal shape, and no poikilocytosis or nucleated red cells were present.

The patient was treated with the usual measures for the arrest of bleeding per vaginam, and rectal salines were administered. Her condition, however, became worse, the pulse became weaker, and she died four days after admission to hospital. A post-mortem examination was refused.

The features of this case were: (1) eighteen months of complete amenorrhoea and apparently normal health after the menopause; (2) one attack only of severe uterine haemorrhage (this proved to be fatal); (3) a history of myxoedema; and (4) an extremely low red blood cell

count and a normal film. (Unfortunately, for some reason not apparent, the haemoglobin percentage was either not estimated or not noted.)

CASE V.

This patient, who was 27 years of age, was admitted to hospital on May 14th, 1928, with a complaint of very profuse haemorrhage per vaginam. Three weeks previously a menstrual period commenced, and for the first twenty-four hours was accompanied by epistaxis. Severe uterine bleeding continued throughout three weeks. On examination she appeared almost exsanguinated. The urine contained much albumin.

She had had scarlet fever during childhood. When 20 years of age she was a patient in Glasgow Royal Infirmary, and was under treatment for nephritis. For three years prior to the attack of menostaxis, oedema of the face and legs was absent, but albuminuria was constant. Since puberty menstruation had been scanty but regular.

Her condition improved with general measures and rest in bed. No abnormality was detected in the pelvis. On May 25th, 1928, she was transferred to Glasgow Royal Infirmary for medical treatment.

Blood examinations: On May 25th there were 830,000 red blood cells per c.mm., the white blood cells numbered 2,600; haemoglobin 18 per cent.; colour index 1.0. On May 29th the red cells numbered 1,300,000 per c.mm.; haemoglobin 17 per cent. A film showed an ordinary secondary anaemia. On June 7th there were 1,270,000 red cells per c.mm.; haemoglobin 18 per cent. On June 18th the red cells numbered 1,710,000 per c.mm.; haemoglobin 26 per cent. A film showed ring-staining of red cells and poikilocytosis. No nucleated red cells were present.

On account of a persistent cough and an evening rise of temperature the suspicion of tuberculosis was raised, but a skiagram of the chest and a von Pirquet test proved negative. A detailed examination by the urologist was made, but beyond a well-marked cystitis, seen per cystoscope, no gross abnormality was detected in the urinary tract. On June 11th and 13th the patient had severe attacks of epistaxis. On the latter date a blood culture was made, but this was negative after ten days' incubation. Her general condition improved, and there was no further uterine bleeding. She was dismissed, much improved, on August 15th, the diagnosis being chronic nephritis. There was, further, no recurrence of uterine haemorrhage, but her general health deteriorated, and she died at home on November 15th, 1928.

In this case of a young woman, aged 27, no explanation of the single, very profuse uterine haemorrhage is apparent beyond the condition of chronic nephritis. Osler³ states that haemorrhages, particularly epistaxis, are not infrequent in chronic interstitial nephritis. Berkeley⁹ writes that haemorrhage from the uterus may occur in chronic renal disease, in which not only is the composition of the blood altered, but the blood pressure is raised and arterial degeneration is present. General blood-vascular diseases may affect the uterine vessels in the same manner as those of other organs. In elderly women they may be atheromatous, and Eden and Lockyer have, in one case, found definite sclerotic changes in the arteries in a uterus removed for uncontrollable bleeding. Certain diseases, such as chronic nephritis and general arterio-sclerosis, sometimes occur in connexion with chronic metritis.¹⁰

CASE VI.

This patient, aged 30 years, admitted on October 9th, 1929, complaining of profuse bleeding per vaginam of ten days' duration. Hitherto her menstrual periods had been regular but excessive, the flow lasting for about eight days. On examination she was found to be blanched, the mucous membranes being almost colourless. The uterus was slightly enlarged, but no other abnormality was detected. Her past health had been very good. She had been married for eight years and had no family.

Rest in bed and haematinics were prescribed; the haemorrhage ceased, and she was dismissed, much improved, on October 31st. On December 9th she was readmitted as an urgent case of uterine haemorrhage, bleeding having been present for fourteen days.

On examination she was found to be exsanguinated. The pulse was rapid and thready, and a systolic cardiac murmur was present. Despite measures for the arrest of haemorrhage, bleeding continued. An attempt to make a detailed blood examination failed, as sufficient blood—almost colourless as it was—could not be obtained by the usual method. Thirty-six hours after admission a blood transfusion was performed, but during the course of it the patient died.

This case of a young married woman is one from which a lesson is to be drawn—namely, the necessity for detailed general and blood examination in the absence of a local

cause of profuse uterine haemorrhage. She was dismissed from hospital, apparently much improved after her first residence, only to return and die of a recurrent haemorrhage two months later. The actual cause of bleeding in this case was unknown, except that it was not due to any appreciable pelvic abnormality. One patient in the series of Linton, Marks, and Smith (loc. cit.) was moribund on admission, from excessive flowing of ten days' duration, and she died on the following day; no pathological basis for it is mentioned.

CASE VII.

This patient, a woman aged 49 years, was admitted to hospital on December 3rd, 1929, as an urgent case of uterine haemorrhage. The history of the illness was that in September, 1929, her menstrual period lasted for ten days; two months later bleeding recommenced and persisted for four weeks—that is, until admission. At no time had there been any pain. On examination she was found to be exsanguinated and the pulse feeble and rapid.

Prior to September, 1929, menstruation extended for four or five days, and recurred at irregular intervals of from four to seven weeks. She had been married for seventeen years and had four children. After her first confinement (twins), in 1913, she had very severe post-partum haemorrhage, and was confined to bed for six weeks. The second puerperium (in 1914) was also associated with post-partum haemorrhage, but not as severe as in the previous one. In the following year she had an ante-natal haemorrhage, and developed pneumonia and enteric fever during the puerperium. For many years she had a tendency to bleed unusually readily.

On the day of admission 1 c.cm. of haemoplastin was given; rectal salines with glucose and sodium bicarbonate were administered four-hourly. A blood transfusion (500 c.cm.) was performed. On December 7th a curette was introduced into the uterus, but no evidence of malignant disease or of other gross cause for haemorrhage could be found.

A blood count taken on December 6th showed: red blood cells 1,100,000 per c.mm., white cells 10,000; haemoglobin 28 per cent.; colour index 1.3; polymorphonuclear leucocytes (including transitional neutrophils) 86.5 per cent., eosinophils 1 per cent., mononuclear lymphocytes (including basophil myelocytes) 2.5 per cent., small lymphocytes 10 per cent.

She was transferred to a medical ward at the Victoria Infirmary on December 7th. On admission there she was described as being *in extremis*, and another blood transfusion (500 c.cm.) was given. She consumed twelve pints of water and two pounds of glucose in forty-eight hours.

A blood examination was repeated on December 10th, and showed: red cells 1,180,000 per c.mm., white cells 16,700; haemoglobin 32 per cent.; colour index 1.45. A film showed poikilocytosis, polychromatophilia, and polymorphonuclear leucocytosis. Another blood transfusion (500 c.cm.) was given on the following day, and a blood examination on December 12th showed: red cells 1,220,000 per c.mm., white cells 12,600; haemoglobin 32 per cent.; colour index 1.3. A film showed similar features as in the one made two days previously.

A swelling appeared in the right submaxillary region and increased in size, the cheeks became oedematous, and the tongue greatly swollen. Mechanical respiratory distress became severe. Slight haemorrhage per vaginam continued throughout. She died on December 15th, 1929.

Post-mortem examination revealed no macroscopic features of note. The liver and spleen were normal. A film from the bone marrow, however, showed the characteristic features associated with anaemia (pernicious) of the aplastic type.

There can be little doubt that the fatal uterine haemorrhage in this case was due to the constitutional disease, aplastic pernicious anaemia. A feature of interest was the history of post-partum and ante-partum haemorrhages at her confinements, some fifteen years previously.

Severe uterine haemorrhage in pernicious anaemia is rare. Linton, Marks, and Smith (loc. cit.) record a case of a patient who was treated with radium on account of fibroids, the presumed cause of her menorrhagia, but two years and eleven months later she died of "pernicious anaemia" (State Records).

Conclusions.

I have described seven cases of urgent uterine haemorrhage in which no local or pelvic cause was discoverable, and which were presumably of general or constitutional origin.

When bleeding is severe and the true diagnosis at an early stage is missed, the prognosis is grave; four of the above patients were dead within a month of admission to hospital, one within two months, and one (Case V) within six months.

Other factors than dysfunction or disease of uterus and ovaries are responsible for some cases of uterine bleeding. Cases occur in which it is to be attributed to a pathological condition of the blood.

The mechanism of haemorrhage in these cases is not clear, but it may depend on disordered calcium metabolism—for example, increased calcium output or low blood calcium.

In cases of severe uterine haemorrhage without obvious pelvic or local cause, a systemic investigation and a detailed blood examination (preferably by a haematologist) should be made at the earliest opportunity.

I am indebted to the surgeons of the Royal Samaritan Hospital for Women, Glasgow (Drs. Shannon, West, Gardner, and McIntyre), and to the medical superintendents of the Glasgow Royal and Victoria Infirmarys (Drs. Grant and McCall Smith respectively), for access to records and notes in some of these cases.

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PRODUCTION OF PERNICIOUS ANAEMIA (BARTONELLA ANAEMIA) IN RATS BY DEFICIENT FEEDING.*

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THE dietetic survey of various classes of women in Bombay suggested that the disease occurring in pregnant women known as "pernicious anaemia of pregnancy" was closely connected with a relative deficiency of vitamins A and C in their diet. In this connexion a series of experiments was carried out at Coonoor in which diets relatively deficient in these vitamins led to the production of a severe anaemia in intact albino rats, especially in the female animals; it was further shown to be a true "Bartonella anaemia." The term "Bartonella anaemia" is used to describe the well-known anaemia of rats which occurs after splenectomy in infected stock, and is associated with the appearance of Bartonella "organisms" in the red cells; it is used without prejudice as to the exact nature of these small bodies.

DIETETIC EXPERIMENTS.

The diets used had one common fault—a relative deficiency in vitamins A and C.

Series I.

These experiments have been reported before by McCarrison,¹ in his work on the production of urinary calculus by faulty feeding. In them two types of diet were used, the one based largely on oatmeal, and the other on atta (whole wheat flour). These diets contained no vitamin C and only a little vitamin A, the latter being obtained from the small quantity present in the grains used. Vitamin B was judged to be present in adequate amounts.

Cases of a severe, and frequently fatal, anaemia occurred among the rats fed on these diets; among those fed on the oatmeal diets there was a death rate from this disease of 13.8 per cent.; among those fed on the atta diet there was a death rate of 12.4 per cent. Milk added to the oatmeal diet appeared to afford the rats protection against this anaemia. The disease was much more frequent in

females; out of a total of seven pregnant animals in one experiment five died of this anaemia.

The diagnosis of Bartonella anaemia was made on an examination of the blood slides. The characteristic appearances in the blood will be described in the account of the next series of experiments; in these the blood examination was much more complete.

Series II.

Six diets, two natural and four synthetic, were used in this series of experiments. The two natural ones were based on diets in common use among the hospital class in Bombay; one resembling that used by Mohammedan women and the other based on a diet used by Hindu women. Of the four synthetic diets the first was a basic one without vitamins A and C except such traces of vitamin A as might be found in the olive oil used (very

FIG. 1.—Blood from splenectomized anaemic rat showing "Bartonella organisms" in the red cells. (×1,000.)

little if any present, as the animals went downhill rapidly); the others were the same as the first with the addition of orange juice to the second, cod-liver oil to the third, and both orange juice and cod-liver oil to the fourth.

Cases of Bartonella anaemia occurred spontaneously among the rats fed on the Mohammedan diet and among those fed on the basal synthetic diet. Pregnant animals living on the Hindu diet were, however, susceptible to inoculations of Bartonella-infected blood; in this respect they were unique, as we failed to produce an anaemia after inoculation either in well-fed rats, whether pregnant or not, or in pregnant rats when they were fed on the Hindu diet to which vitamins A and C were added in the form of cod-liver oil and tomato juice.

The amount of vitamin B and salts and also of fat varied in the different diets. The Mohammedan diet, on which the animals developed the anaemia most rapidly, was markedly deficient in vitamin B (poor growth from the beginning of the experiment) and salts, and at the same time had a high content of vitamin-A-free fat. The Hindu diet, on the other hand, had more vitamin B and a very low fat content. The synthetic diets had what has been shown to be an adequate amount of vitamin B and salts. The animals on complete synthetic diet grew well. The common factor in the diets which led to the production of the anaemia was, therefore, a relative deficiency of vitamins A and C, or very possibly some other dietetic fault associated with this deficiency.

* Summary of a paper appearing in the *Indian Journal of Medical Research*.