History of a Case of Supposed Idiopathic Pernicious Anaemia with Remarks.

The case is that of J.C., male, age 58, married, with no children. His family history is very good, without evidence of tubercular or other hereditary tendency. Healthy from boyhood upwards, with an excellent constitution, he went to sea at the age of eighteen. A sailor for twelve years, he enjoyed first-rate health until at the age of thirty he was laid up in Calcutta with a severe attack of Asiatic Cholera.

For the next twenty-two years he was a tea planter in Darjeeling. During these 22 years, he visited England on holiday twice, at intervals of 8 years. Finally, at the end of 22 years, he came home to this country permanently.

During the first 16 years of his India life, he had three separate attacks of Jeevan, the Jeevan in each case lasting for a week, with convalescence at the end of the second week. About the time of his first holiday to England, he began to complain...
pain in the back & was treated for lumbago. At times slight, & at times so severe that he could not sit on his horse, the pain continued until a year or two after his second holiday when he had an attack of what seems to have been renal gravel. He had intense pain in the usual line, vomited, & passed bloody urine. For a year or so after this he was well, then began to get gradually weaker, suffering much from headache & sleeplessness, feeling unfit for his work, but without symptoms of any definite disease. By the advice of his doctor, he came home to England in May 1891. Benefited at first by the change, his strength soon began to fail again. He had again headache, was sleepless, & had occasional attacks of epistaxis. The debility slowly increased, & he had two syncopal attacks at the end of the year. In January 1892 he was admitted to Sir J. Graham-Stewart Ward in the Edinburgh Royal Infirmary.

So far then, we have a history of a healthy boyhood, hard work, &
See for twelve years; an attack of Asiatic Cholera; three attacks of
Jesse, probably malarial in origin; long continued symptoms of stone in
the Kidney, ceasing permanently after an attack of renal colic; and finally of
the beginning of the last & fatal illness, which is the Subject of this Thesis.

The date of commencement of this it is difficult to fix. The patient was
always subject to attacks of Epistaxis from the time of his boyhood. It suffered
much from headache & nephremia all the time of his residence in India.
After the attack of renal colic however, he appears to have been quite well
for a year or more. We have thereby some ground for concluding that the
general debility & emaciation in which led him to return to this
country were the first symptoms of the disease which ultimately proved
fatal.

Taking this as the starting point, we may say that he had been ill for
two years before his admission to the Royal Infirmary.

Dr. George E. Gibbon, under whose care the patient was in the temporary
absence of Sir Thomas Grainger Stewart, gave an account of the case in the Edinburgh Medical Journal for May, 1892, from which for the sake of continuity I make the following extract:

On admission "the patient complained of weakness, giddiness, headache, and palpitation, with pains in the shoulders. He was 5 ft. 6 in. in height, and the weight was 118 lb. 6 oz. He was well developed and muscular, his complexion somewhat lemon tinted, the lips and gums pale as were also the conjunctivae, there was some oedema about the ankles. The patient had little appetite, but he had also little thirst. There were no feelings of discomfort during digestion, but there was obstinate constipation. The teeth were good, the tongue large, pale, flabby, and indented. There was no change in the size of the liver, or any of the other abdominal viscera. The feeses were wonderfully free. The pulse was of low tension, moderately filled, regular and equal, its rate being 74 per minute. There was a distinct aperistaltic in the fifth interspace,
Two and three-quarters inches to the left of the midsternal line. At the level of the fourth rib the right edge of the heart was two and a quarter inches from the left edge three and a quarter inches from the midsternal line. A continuous venous hum was heard over the veins of the neck, and a systolic murmur of very soft character over most of the precordium, with its maximum intensity in the tricuspid area. The red corpuscles were found to number 8,000,000 per cubic millimetre. Changes in size and shape of the red corpuscles were very obvious, but there was no absolute change in the number of the leucocytes. The spleen and the lymphatic glands were of normal size. In regard to the respiratory system there was a history of frequent attacks of epistaxis. There was no change in the Jendrassik test. The urine was pale and no changes of importance were present in it. The temperature was never at any time abnormal.

J. Gibson then says "that this case was one of Pernicious Anaemia then could be no reasonable doubt".
the only characteristic symptoms of the disease which were absent in our patient were retinal hemorrhages & dark urine. In the first few days after admission the patient was treated by means of arsenic in various forms, but this had to be discontinued as the drug produced great gastric & intestinal irritation, showing itself by vomiting & diarrhoea.

He was becoming weaker, & the edema increasing, but his blood remained until 16th Feb. in the same condition — that is contained 800,000 red corpuscles. On Feb. 18th he was put on brom. chlorid & brom. perchlorid. On March 2nd the red corpuscles were 920,000 in number, the patient had gained 3 lbs. in weight. On March 3rd, 68.9 liters of blood were transfused into the patient's right arm. On March 4th the red corpuscles numbered 1,120,000.

On March 9th they had fallen to 102,000, & on March 12th to 920,000.

Dr. Green says, "His energy during this time was steadily failing. The edema was gaining on him, the condition of his heart was becoming
worse & worse.

The patient objected to traces, just as a second time. On March 18th. following Hunter's suggestion, the use of naphthite was begun - two grains three times daily. On March 22nd., the reds were 1,000,000. On April 2nd. they were only 800,000; the weight was 10st. 15lbs.

During the treatment by means of naphthite a good deal of intestinal disturbance had from time to time troubled the patient. On April 9th. a severe attack of diarrhea set in.

"Whether the drug was the cause or not was uncertain, as at that time there was an epidemic of diarrhea in the ward, but the attack was severe and lengthy, as in spite of all kinds of medicines the symptoms did not abate until a fortnight had passed."

On the 16th. April, about a month after the naphthite treatment was begun, the number of red corpuscles was 1,600,000, and the colour of the skin & mucous membrane was greatly better than at any time since the patient's admission.
The sedatives had quite disappeared, but his weight, as might be expected, was only 108 lb. On the 19th April the administration of the naphthol was resumed, but the patient took only two pills daily. On the 25th April the red corpuscles numbered 1,760,000.

On the 2nd May they numbered 1,850,000. "The patient had improved in every way, his strength had greatly returned, his appetite was much better, and his appearance had undergone much improvement."

On May 26th the red corpuscles numbered 2,080,000, weight 128 lb., the strength much increased, so that the patient was able to take a short walk without fatigue. He was now permitted to return to his home in the country, with strict injunctions to continue the use of naphthol and iron.

June 9th. On this day I saw the patient for the first time. He complained of great tenderness of the tongue while eating, and some hoarseness and cough, but his appetite was good, and he felt that he
was improving. The tongue was very pale, flabby, and fissured. There was slight bedema of the feet, ankles, and there were a few crepitations in both lung bases posteriorly. The lesion of the skin was not marked, but there were a few purpuric-looking spots on the arms, chest, and the tender side of the tongue. There was no headache, no palpitation, not much breathlessness, the pulse 72, moderately filled, regular, tension low. Heart action feeble.

June 18th. The reds were counted with Gomer's haemacytometer to be 1,840,000, the patient still improving in strength and energy.

July 3rd. The weight had risen to 128 st. 1 lb., the number of reds to 2,520,000, and the patient was able to walk four miles without fatigue.

August 5th. Red corpuscles 3,830,000.
Weight 128st. 5 lbs. Homeseness cough much better. Complained of chiefly of pains in the legs, had a sensation in the hands as if they were swollen.

Sept. 5th. Red corpuscles 3,620,000.
Weight 128 st. 3½ lbs., bowels regular but with a tendency to looseness.
Complained of a peculiar sinking sensation during defecation, had had occasional attacks of epistaxis.

Diet.
Oct. 25 - Red capsules 3.970.000
Nov. 3 - Red capsules 3.998.000
Dec. 3 - Red capsules 4.080.000

The capsules not by any means however normal in shape or size, the weight remained about 12 st. 11 lb.

No change had been made in the treatment since the patient came under my care. He had taken the combination of naphthal and iron almost uninterruptedly. He had had dyspepsia perineum part of it peptonized with a moderate allowance of stimulant. He was now so well that he was able to travel to London on business and return without any bad effects ensuing. He had still a slight cough, but there were no menorrhagia, no breathlessness and no palpitation.

1893.
Jan. 3 - Capsules 10 - 4,810,000
Weight 12 st. 11 lb.
Feb. 3 - Capsules 3,150,000
Weight 11 st. 12 lb.
March 3rd: Corporation 3, 290.000.
Weight 11st. 8½ lbs. Physical signs in the lump now normal, the cough very much better. The dose of Naphthol increased to give pains three daily & causing no diarrhoea.

April 8th: Corporation 3, 310.000.
Weight 12st. began digen Arsenic in addition.

May 5th: Corporation 3, 760.000.
Running 1½-backs of hands & face noticed. Arsenic well borne & continued in increasing doses up to 15 mins.

June 5th: Corporation 4, 650.000, weight 11st. 11½ lbs. No maximum. the pulse 90, the patient well, but the corporation not normal.

July 30th: Corporation not counted.
Aug. 30th: Corporation not counted.
Sep. 3rd: Corporation not counted & Arsenic stopped.

Oct. 30th: Corporation 3, 120.000, weight 11st. 10 lbs. Arsenic begun again.

Nov. 3rd: Corporation 3, 840.000, weight 11st. 10 lbs.

Dec. 5th: Corporation not counted.
Patient well & able to go to Aberdeen.
Jan. 3rd. Condition 4. 5-80. 000. But not normal in shape. Patient able to take in good deal of exercise. His cough however never alto get away.

In the end of January he had a mild attack of influenza. The acute symptoms lasting only a very short time. There were no complication. But the attack left the patient manifestly much weakened. The pulse never fell to its normal rate, but remained very constant. After 84 per minute.

The soft systolic murmur previously mentioned was again audible. He still complained of the feeling sensation during defaecation. The motions were slightly loose. He was put on digitalis, iron and arsenic.

I was in bed till the middle of March. During April a great part of May. he was able to move about out of doors, but never seemed to recover strength.

May 28th. The patient came to me complaining of utter exhaustion, throbbing in the ears, and complete loss of appetite. The red corpuscles numbered 1.850.000. He was practically in the same condition as described.
by Dr. Gibson, except in the case of the peculiar: the pulse was now 84, there was marked pigmentation of the skin, the bowels were loose, there was no delirium. He had been taking naphthalin constantly, except during the influenza attack. He was again put on arsenic, gradually increased up to 15 m. doses three times daily. For two or three nights in succession at this time he had attacks of intense agonising headache. It was relieved by antipyrine after the third night did not return.

June 4th. At the suggestion of Dr. Gibson patient was tried with 6 c. bone marrow, ½ gr. three times daily. He found it very nauseating and never felt anything but sick after taking it. This sickness so started, continued more or less constantly until the day of his death.

June 6th. Salol, in 10 m. doses was substituted for the naphthalin so that he now had Salol, arsenic and marrow. The urine was dark during administration of Salol, but never at any other time.
June 18th. Red Corpuscles 1,700,000.

No improvement following the use of these drugs. I advised the daily injection into the rectum of 60 c.c. of defibrinated sheep's blood, fresh and warm. The result could not be obtained every day. The injection always remained, the bowels moved loosely once daily.

June 25th. Red Corpuscles 2,270,000.

July 2nd. Red Corpuscles 2,310,000.

July 23rd. Red Corpuscles 3,270,000.

Unfortunately he began to suffer from piles & the rectal treatment had now to be stopped.

July 28th. Dr. Gibson saw the case with me & advised the continuance of arsenic, the marrow to be taken if possible. The patient was allowed up & was out on one or two occasions in an open phaeton.

July 30th. Red Corpuscles 2,590,000.

Aug. 11th & 18th. Patient complained of bed again. Suffering very acute from innumerable external piles, with retention of urine necessitating the use of the catheter. There was slight constipation. Passage of the catheter was followed on one occasion.
by acute tertian fever.
In spite of fever, the urine became ammoniacal and the
microscope showed pus corpuscles in abundance. All previous drugs were
stopped except Soda and Bismarck Acid
was given in 15 gr. doses every evening.
Aug. 18th-27th. Patient quite suffer-
mg but very weak.
Aug. 27th. Pulse again very painful,
causing retention of urine. Other
measures failing to give relief, the
catheter was again necessary.
Aug. 28th. Catheter used for the last

Aug. 29th. Patient had a fever
regain at noon with high temperature
(104° F.) followed by a drenching sweat.
The temperature was normal in the
evening, but rose again about bed-
time. He had a good night after
a dose of Aspirin.
Aug. 30th. He was feverish again,
with more or less mental slumber,
thirst, dry tongue, & great headaches.
He was put on Quinine - 5 gr. every
four hours. Dr. Irvine & Wibworth
saw him in consultation with
me, & daily after this - Quinine
continued.
Aug. 31st. Still feverish at some part of the day. Diarrhea continued in 1887. Aches. \textit{Boric Acid} at night. Burgundy Pyrexia continued in varying degree till 12th September, when it came to an end. The urine was then better, \textit{&} the Piles had ceased troubling, but the appetite was poor.
Sept. 16th. Patient vomited. This was the first time since he had been in the Infirmary, but it now continued almost daily until the end. The bowels also were now troubled more than Bismuth Subnitrate in 30 gr. doses kept the diarrhoea in check for a time. Patient complained of the peculiar sinking sensation with every motion.
Sept. 21st. He was now always in bed. He complained of throbbing in the ears, great exhaustion & sinking. He lay with his eyes closed, was disinclined to talk, was deep & insensible, the expression was mournful, the skin of the face pigmented, \& looked as if tanned by the Sun, but the tanning extended right up to the roots of the hair.
The neck was more deeply tanned than the face. The sclerotics were white. The conjunctivae of the eyes were pale—he was markedly anaemic. The muscles were soft and flabby. He was somewhat emaciated, the lower limbs shrunken to about half their size, the fat seemed to be gone. The upper limbs were not so much wasted as the lower. In the face pallor was much more striking than emaciation. The abdomen was sunken. On the left side of it there was a large patch of blanched skin, extending upwards as far as the ribs, and downwards as far as Poupart's ligament, reaching to the linea alba below the umbilicus. To about 1 1/2 inches from it, about the umbilicus laterally, it was continuous with a piebald patch, which extended backward to the spine. In colour it was dead white, not the natural colour of the hair, nor it was white. The margins were quite sharply defined, the skin quite smooth, soft & pliable. The piebald appearance in the umbilical region is caused by small islets.
8) smooth coloured skin on a ground
10) deciduous skin: the white
was continuous with the white of
the patch in front. Restraining con.
trust to the whiteness of these patches
there was pigmentation of various shades
over all the rest of the body. The
right half of the abdomen & both of
the groin were of a dark mouse colour.
So were the nipples, the back of the
spine, & the perineum. From the
rest of the trunk & the limbs of the
upper & lower, the pigmentation was
merely decided but no others. In
there was a light chocolate coloured
patch on the left hip, between the
anterior superior spine of the ilium
& the great trochanter.
Below Poupart's ligament. on the left
side. Over the left lower ribs, were
numerous small white patches about
twenty, each about the size of a
fingernail. There were a few
similar white marks on the left
jerk of. There were no white patches
on the right side. (Before death
occurred there were two or three very
lightly developed in the right side
of the chest.) The white patches
were gradually increasing in size. The large patch on the abdomen began in distinct. No pigmentation was observed in the abdomen. There was no discoloration of the lips or of the buccal mucous membrane. The red corpuscles measured 690,000. They did not form rouleaux. They varied in shape, some were kidney shaped, some were rounder, some irregularly biscuit shaped, some presented a budding appearance.

As to size, some corpuscles were very much larger, some very much smaller than the normal corpuscles. In addition there were a few small rounded globules corresponding in appearance to Heister's description.

1) Sehrhorst's corpuscles—that is, they were (1) about one third the size of a normal red corpuscle.
2) they were perfectly globular.
3) they were deeply pigmented.

Haemoglobin was 12.9% in quantity. The percentage of corpuscles was 14. The value of each corpuscle was 12. Haemoglobin therefore was 14. not in excess. White corpuscles were very difficult to
find. There was a good deal of
thirst, no appetite, a tendency to
vomiting, bowels relaxed. The liver
and spleen were normal. There had
at any time been epigastric pain,
nor tenderness. The respiratory
system was normal, the pulse
regular, 96. moderate, large.
1) Blood tension. The heart not
appreciably altered in size
from before. Its action was weak
the heemic murmurs were loud.
Temperature 98.4, urine normal.
There was no glandular enlargement
in any part of the body.

Sept. 22th. Had a fairly good night,
slightly restless, vomited once,
pulse 96, temperature normal.
Piles continued easy. No headache,
ache, nor pain anywhere. Very slight
epistaxis. No sickness during
day. Bowels moved three.
Diet raw beef juice & milk.

Sept. 23th. Had a good night.
Pustula very marked in the face.
Temperature slightly subnormal.
Bowels three times. Vomiting once.
Pulse 96, very soft, settled some. Very weak & prostrated. Morphine supporting at bed time.


2.m. every three hours. Injected 13 m. of Peritonate into the back.
Patient vomited. Bowels three times.


Oct. 1st. Siptane.
every four hours. Lead up in pill, with decoction of Sograis every three hours. Standard Landau enemas.

Blood examined. Red corpuscles 490,000. Average of corpuscles in two squares was 9.4. Haemo-

globin 10%. Valency corpuscles in Haemoglobin 1/5. Haemoglobin therefore not in excess.

A few red corpuscles seen again. White corpuscles not so distorted. Whites not to be seen.


with Landauenem.


very marked. Set burnt brandy 1/2 a pint every three hours containing Euphr. Symp. 6t. with Pulse 88. 6t.

Flannel bindle was also put round abdomen.

Clinical Research Committee report on urine as follows - reaction acid. Specific gravity 1.008. No albumen. No sugar. No blood. Colour a deep yellow, but not the deep colour of the urine of Pernicious Anaemia. No characteristic Urobilin Spectrum.

Oct. 5th. Bowels moved about once in four hours. No vomiting. Pulse 102.

Oct. 6th. Very weak, stupor and unable to answer intelligently. Had a syncopal attack. Dying slowly, but easily aroused to take stimulants. Pulse smaller, 102. Temperature 98.4.

Oct. 7th. Bowels moved four or five times. Pulse very weak. Mental stupor. Unable to ask for what he wanted.

hours. Oedema of hands & feet.
Gastric & vomiting. Breathing laboured more
or less all day. Extremities cold.

Oct. 9: Death at 7.30 A.M.

Interesting as this case was in the
earlier stages from the profound
degree of anaemia present, & the
marked alterations in the composition
of the blood without evidence of any
definite cause, from the actual
diagnosis of Pernicious Anaemia by
a competent observer, & the re-
markable rally which took place under
medical treatment based upon
Hunter's pathological researches, in
cases of that disease, it became
doubly so in the later stages when
the gradual development of
pigmentary changes in the skin
suggested the probable presence both
of Addisoni's Disease & of Bitiligo.

For suggestive as the general
pigmentation was of Addisoni Disease,
the white patch on the abdomen
was no less suggestive of Bitiligo.

Two questions as regards diagnosis,
presented themselves. I. Was Addison's Disease really present, or was it only simulated? The pigmentation skin changes due to bilirubin bear a close resemblance to the pigmentation skin changes in Addison's Disease, & the symptoms of idiopathic Per-
necious Anæmia bear a close resemblance to the constitutional symptoms of Addison's Disease.

The coincidence therefore of Bilirubin + Pernicious Anæmia would produce a combination of symptoms closely simulating all the clinical symptoms of Addison's Disease.

II. If it be shown that Addison's Disease was present, may we still adhere to the original diagnosis made by Dr. Gibson?

Can we maintain that the patient suffered at one the same time from two distinct maladies, Idiopathic Pernicious Anæmia + Addison's Disease? Or must we come to the conclusion that Addison's Disease was present alone, the Anæmia the Perinicium being only symptomatic?
To discuss in detail each of the three diseases apparently present will be the easiest way to find an answer to these two questions.

A. Vitiligo - There was a white patch on the skin of the abdomen. The skin of this patch was smooth and soft and the colour was whiter than that of normal skin. The hairs on the patch were white and there was a sharply defined, deeply pigmented margin. The patch was non-congenital and was gradually enlarging. One anomalous feature there certainly was. If vitiligo was present, the asymmetry would be always spreading and symmetrical (Balmano Squire) but there was no corresponding patch in this case on the other side of the body. Absence of asymmetry therefore suggests the idea that vitiligo was not present. If this be so, what was the nature of the white patch? While patches are sometimes seen in cases of Addisoni Disease,
Other appearances were suggestive of the presence of that malady. The facts that in the case before us the patch was steadily enlarging, that there was no antecedent deep injury to the skin, are quite sufficient to differentiate it from the white patches described by Greenhow as occurring in Addison's Disease in the position of creatives. But I find on referring to my notes of Sir Thomas Ambrose Stewart, that Addison's Disease is "often associated with patches of a dull white colour interspersed through a adjoining the brown part."

This is an accurate description of the appearance of the skin over the lower ribs & the lumbar region, but not of the appearance of the abdomen. The patches there were of a dead white colour, unnaturally white. Certainly the extreme degree of anaemia present might account for the blanched appearance, but why were not the smaller white patches equally blanched?
In spite of the want of symmetrical
symptoms I am inclined to the
conclusion that vitiligo was
actually present.
Assuming the presence of vitiligo,
still adhering to Dr. Zieve's
original diagnosis of Pernicin
Anæmia, substantiate Addison's
Disease remains so far a prob.
ability in diagnosis.

B. Addison's Disease. In his
original memoir, Addison says:
"The leading and characteristic feature
are anaemia, general languor,
debility, remarkable paleness
of the hands, action, irritability
of the stomach, a peculiar change
of color in the skin occurring in
connexion with a diseased con-
dition of the suprarenal capsules.
Anaemia, general languor,
debility, remarkable paleness
of heart's action, irritability
of the stomach were all marked
symptoms in the course of J. C.'s
illness. But these symptoms
were also in Pernicin Anæmia.
And changes in the blood highly
suggestive, yet in themselves
actually distinctive, of pernicious anaemia were demonstrable from a very early period in the history of the case. The diagnosis of pernicious anaemia had actually been made. The characteristic of the one leading feature of Addison's disease which does not occur in pernicious anaemia, viz. the peculiar color of column in the skin, become there prominent. Amount importance in diagnosis. In the absence of characteristic bronzing, in fact, no diagnosis of Addison's disease could be made. Marked pigmentation of skin was certainly present. Pigmentation is associated with other conditions besides disease of the Suprarenal Capsule. But there was no evidence of long continued hepatic disease, of syphilis, of tropical or malarial fever, or of very chronic phthisis. All diseases sometimes are accompanied by pigmentation. Pityriasis versicolor certainly was not present. Bagshwan's discoloration was out of the
Question. There is some doubt about the presence or absence of vitiligo; but, assuming its presence, pigmentation was far too general to be referable to the single white patch on the abdomen. Was the pigmentation then characteristic Addisonian bronzing? What were its features?

I. It was not uniform in colour, nor in distribution.

II. The bronzed skin was smooth, soft, and supple.

III. It began earliest and became most marked, first on some of the most exposed exposed parts, viz. the face, hands, neck, and finally on parts naturally more pigmented, viz. the abdomen, gums and genital.

IV. The nipples and areola were deeply pigmented (Greenhill says that deep discoloration of the nipples is one of the most distinctive external signs of Addison's Disease).

V. The dark patches were not sharply defined, but faded insensibly into less pigmented...
Skin. This is surely the typical bronzing of Addison's disease. Bronzing so well marked, occurring in a patient suffering from anaemia, general languor, delirium, feeble heart action, insatiable appetite of stomach, points unmistakable to disease of the suprarenal capsule.

Whatever the disease the patient may have had, he undoubtedly suffered from Addison's disease.

In connection with the bronzing in this case, it is interesting to note that, at the time of Dr. Gobin's report, the skin was of a lemon yellow colour. This may or may not have been the lemon yellow colour typical of idiopathic permanent anaemia, but in those born to treatment for Addison's disease we have mention of no less than nine cases of true Addison's disease in which the dominant colour was yellow. On page 39. he mentions a case of Sir William Gull's, of four months' duration, "the face of a yellow or sallowish cast" - a case of
Dr. Muckle’s case, week’s duration, “slightly hazy and slight jaundice, with pearly conjunctivitis.”

A case by Professor Heschel, of a few weeks’ duration, “skin yellowish with a green tinge.” And in appendicitis.

We have case XV, “resembled in colour that of a person recovering from jaundice.” Case XII, “Skin of whole body of a dirty yellow brown.” Case XVIII, “Sallow—dull face at first.” Case XXIV, “clear yellowish brown.” Case XXV, “yellowish brown discoloration of face and hands.” Case XXVIII, “looks jaundiced at first.”

We have seen that anaemia is one of the essential features of Addison’s disease. But though an essential feature the blood does not present in typical cases characters different from those met with in any ordinary case of symptomatic anaemia.

Glehn has says—“The composition of the blood does not undergo any important change in uncomplicated cases of Addison’s disease.”
One case he found that the white corpuscles were slightly in excess, of the but otherwise the blood was normal, with rouleaux formation. In another case the reds were diminished. There was no rouleaux formation so there were a large number of globulin highly refractive nuclei of a red column. At the end of three weeks however the patient was stronger & the blood normal.

Dr. Byron Bramwell says in a private communication: "There is usually a certain amount of diminution of red blood corpuscles, with perhaps a slight relative excess of the white. The percentage of haemoglobin is good."

White Anaemia is present therefore in Addison's Disease, it is not in any very marked degree. The fact that our patient suffered from Addison's Disease would not therefore account for the profound degree of anaemia present. But these changes in the blood were such as are seen in cases of Schioedte's Perinia Anaemia.
Hunter says in the Practitioner, in August 1888—"From the anaemia, symptomatic of wasting disease, whether malignant or not, hemorhagia, anaemia is distinguished by the greater degree of oligocythaemia it presents. If there be no hemorhagia in question, a more or less rapid diminution to, say 20,000 per cubic millimetre, 1 point to anaemia of haemolytic origin—that is, practically, to hereditarian anaemia. That whether malignant disease exist or not. The only other condition, in which, up to 1890, hemorhagia, an oligocythaemia of this intensity is possible, are perhaps malaria and haemoctysis."

In our case, hemorhagia from Cristiania was exceedingly slight. Here was no haemoctysis nor malaria, and yet the red corpuscles on several occasions numbered less than 1,000,000.

Two of the ordinary symptoms of hemorhagia, anaemia were absent, viz., retinal hemorhagia and dark urine. Another symptom,
Bzo. irregular pyrexia was present, but was probably due to bladder complications, while the lemon yellow colour of skin was possibly alg am antecedent of Addison's disease.

Taking into consideration how, even the extreme degree of blem.
erythraemia present, the altered
shape of the red corpuscles, the presence of the so-called Anisocytosis
corpuscles, we are forced to come
to the conclusion that the patient
was suffering from an acute
pothic anaemia, Anaemia.

With regard to the treat.
ment, I would only call attention
to the fact of the extreme value of
Arsenic and rectal blood
injections.

Robert W. McEvedy
Monroe