Contribution on

(Idiopathic)

Symmetrical Gangrene

By

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1889
Since Raymond published his thesis
calling attention to this condition in
1862, many observers have related
cases which they have met with; but
the clinical, and especially the
pathological observations yet recorded,
do not afford sufficient evidence upon
which to base a satisfying and
definite statement as to the pathology
of this interesting disease.

It is with the hope of assisting in
building up a satisfying knowledge
of this disease, that I contribute accounts
of two fatal cases I have met with, and
observations of some of the cases recently
reported by other observers, together with
remarks upon the series of cases, and
some valuable pathological evidence
obtained from the first case.
Case I

Mary. E. H. — age 1 year 7 months

Previous History — The child was perfectly healthy when born, no eruption or rashes and was well formed.

Labour was easy and the mother was in good health, but the husband was out of work while the mother was pregnant, and she had to work hard and did not get good food, and the child also appears to have suffered from want of good food. The neighbourhood in which the parents live is poor and unhealthy.

The mother has been married six years, and has had two other children, boys, now alive who were and are healthy.

She has never miscarried and the child was born at full term.

The mother is healthy and there is no history of Syphilis. The father is strong and healthy, and has no history of Syphilis. The parents' relatives are all healthy.

The child was weaned until three months ago, and took the breast well, but would take nothing else.

The mother
The mother was well and the milk apparently good.

Three months ago the child began to eat, but for the last six weeks she
will only take milk, of which she uses
a pint per diem.

Until three months of age the baby
survived, and was then vaccinated.
The arm inflamed and swelled, and
for twelve weeks was very bad, and
the child was attended by a dispensary
doctor.

During this time she began to have fits,
The first fit taking place the next day
after being vaccinated. During eight
weeks the head fits night and day-
beginning with a scream and lasting
about a quarter of an hour each.

During this time the size of the head
increased, and the diagnosis of hydro-
cephalus was made.

The fits gradually diminished and
stopped in three months, and the child
began again to thrive.

A month later the patient was febrile
in the sun one day, and the skin of
the face became burnt and blistered,
and the
and the face became inflamed and raw for a fortnight. This was diagnosed as syringes.
From this time the child became weaker and lost flesh, but had no fits.
Six weeks ago - June 16th - patient began with fits severely. The same as previously. These have continued until a week ago when they stopped suddenly, and since then there have been none.
Two weeks ago the patient became edematous all over - feet, legs, body, arms, hands, face. (There had been no antecedent rash or scarlet fever in the neighborhood.)
In three days the edema passed off, but as it passed away purple spots appeared on the face and body, and the nose became purple.
The hands and feet remained discolored, and the fingers and toes gradually became gangrenous, but the discoloration and congestion of the nose passed among.
There has been some diarrhea for the past few weeks.
The physician who attended the case at this time furnishes the following account -
On June 29th I was asked to see the child who had convulsions. The child was sleepless at night, moaning and pressing its head into the pillows - there was perspiration on the head and forehead. The pupils were somewhat dilated. Temperature and pulse normal. Next day there were no more convulsions and the child was decidedly better, and was not seen again until about a fortnight later (July 10th) when it was reported that the child had suddenly got worse.

I found the body oedematous, and small purpuric spots on the cheeks and nose, each spot being about the size of a sixpence. The fingers and toes were purple. The urine was very offensive and the stools were loose, clay-coloured motions. It was impossible to collect any urine for analysis. The affected parts were carefully wrapped in cotton wool. In two days the oedema was entirely gone, and two days afterwards the purpuric spots on the face and nose disappeared; but the fingers and toes had become
August 1st. Present Condition.
The child is of fair complexion with white waxy skin. It is a very sickly looking baby, with prominent forehead and large head, and the anterior fontanelle is still quite closed.

Right hand. The thumb and all four fingers are gangrenous. There is a deep ulcerating groove across the back of the hand, and the forefinger is the worst, being only attached by a bone. The fingers are black and dead at the joints. The thumb is only destroyed for the distal phalanges.

The extent of the gangrene when first seen is shown diagrammatically in the accompanying drawings —
**Left hand** - The thumb is a little elevated at the tip, but not black; the terminal phalanges of the fingers are black and pointed; there is no line of demarcation, as is faintly indicated in the right hand. The blackness shading through purple into the ordinary skin.

The back of the hand is normal, and its whole condition is much better than the right hand.

The hands are apparently not very painful. The child attempts to rub the hands together, and can move the fingers of the left hand. The only whines when they are exposed to a dressed right foot.
Right foot - This is worse than the left foot. The whole toes are black and there is a line of ulceration across the back of the foot, and a purple patch across the plantar surface above the toes.

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Left foot - The tips of each toe, for a patch the size of a three-penny piece, are black and dry and gangrenous. There are rings of ulceration above each patch, and in the plantar groove of the great toe there is more extensive ulceration. Above the black patches the toes are purplish.

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Diagram of the right and left foot with descriptions of the condition.
The body is essentially thin, though somewhat stunted. There are no marks or blemishes on the body, arms, or legs. The nose has a small reddish patch near the lips.

The teeth are not well developed; there are six in each jaw. The right incisors are fairly well developed, and are not pegged, but have some unhealthy inflammation of the gums around their crowns.

The tongue is not subject to blanch. There is no stomatitis, but the lips are cracked and crusty with a little yellowish discharge. The child lies quite quiet and good when undisturbed.

The left upper extremity appears somewhat smaller than the right, and the child can move the fingers and toes. Apparently there is no pain in the hands or feet, even when touched or moved.

Urine - The urine is smoky and blood-tinged. It is cloudy containing a fine flocculent material which does not settle but remains suspended for days. The color is a deep pinkish smoky tint. Under the microscope red
red blood corpuscles are present in large quantity. There are a few large granular cells, like white corpuscles. There are a few hyaline casts, some yellow granular casts, and occasionally patches of granular material. Even when examined a few hours after passing the urine swarms with active bacteria.

On boiling, the flocculent material becomes a little more marked, and slowly deposits as a nearly fine white deposit. Apparently there is not more albumen present than the blood will account for.

Heart: Position and sounds normal; no murmur. A good well marked pulse can be felt at the wrist.

Pulse 120 per minute. Temperature 99.2.

The hands and feet were bathed with carbolic lotion (one to forty) and wrapped in lint + carbolic oil. Diet milk with a little red beef juice occasionally.

August 3. The child is no better. The left hand and the feet are slightly worse, the right hand is considerably worse.
worse; the groove at the root of the fingers has extended and deepened, and all the fingers, except the little one, are now in the condition which was previously confined to the index finger. The index finger tendon was stuffed out of the groove as a whitish slough. There is little discharge or odor. The child seems weaker in himself, but cries little. She takes milk and the beef-juice well and moves the fingers and toes. Raw white egg was ordered in addition, and indigo was powdered in the manner of the powders.

August 4. The child looks fairly well, sleeps well, and takes food very well. There is an offensive odor from the child independently of the ulcerating portions. Bowels constipated, motions dark. Very little urine is passed. There is slight stoolsea on the right side. The right hand is much the same as yesterday, and she uses it often. The left hand is no worse. The child does not move this hand much; it is kept strongly abducted. She looks quite

...
The Right foot shows that the gangrene has spread further on the dorsal surface, and the discolored patches on the sole have become black.

Left foot: No further blackness, but the general purplish discoloration of the toes has become darker.

August 8. Child much the same, but somewhat weaker. Swelling is now present. The index and second finger of the right hand are loose, the metacarpal phalangeal joint being open, and the bone of proximal phalanx protruding at the back. The little finger of the left hand has fallen off, and otherwise there is more sign of reaction, and a better line of demarcation on the left hand and on both feet than formerly.

Temperature 99.2°.

August 9. The child was photographed to-day. (Copies of these photographs accompany these notes.)
August 10 - The second and third fingers of the right hand fell off today at the joint of the proximal with the second phalanx.

August 11 - The little finger of the right hand came off at the same joint. The index finger now only hangs by a bit of dead skin on the right hand, and where the other three fingers were, the dead bone of the proximal phalanx protrudes. The granulation surface left however looks much healthier, indeed there is now healthy demarcation in both hands. The child constantly moves the right hand and gets the dressings off. The big toe of the right foot is still in a bad state and the groove at its base is ulcerating. The baby seems fairly well in itself, takes very well of milk, while of egg and Beacon's food. The offensive odour is less and the urine increased in amount. Bowels regular. Pulse good.

August 12 - The child had a restless night and appears
and appears weaker this morning and seems to feel pain in the right foot and to have lost flesh.

August 14. Child seems better. The big toe of the right foot was loose, and removed. The right hand is forming a healing line and healthy granulations. The loose splintered distal phalanx of the thumb was removed, and the loose dead bone of the proximal phalanges of index and little fingers. Two fingers of the left hand have now come off in the dressing. The child does not smell so offensively as previously. Urine is still bloody, but less so, and the secretion is increased in amount.

August 16. There is a good healing line round all the splintered portions, especially in the right hand, but the baby is perceptibly weaker.

August 18. The baby commenced having convulsions yesterday afternoon, and died quietly this morning. Temperature yesterday was 102° F.
Unfortunately the post-mortem examination could not be as thoroughly carried out as I desired.

However permission was obtained to open the head, and as the gangrenous extremities were fastened up in bandages I took the opportunity of undoing them and removing portions of the hands and feet, which I send with these notes.

The body was poorly nourished. On opening the head, the brain was found to be soft, and about eight ounces of fluid escaped from the ventricles. Excepting the distension and flattening of the brain against the skull, nothing abnormal could be observed. I cut away the pons and medulla, and as much of the cord as I could reach. These parts were hardened and sections of each accompany these notes.

Specimen I. Pars and part of cerebellum

"   II Pars and Medulla

   III Medulla

IV Medulla (lower level)
Specimen V  Cord

These specimens furnish only negative evidence, and excepting a little increase of leucocytes in the perivascular spaces there is nothing remarkable.

It is otherwise with the remaining specimens, which furnish evidence of the same time interesting and important. The changes found in the peripheral nerves and arteries were so well marked and important that I have had drawings made of them which show with clearness the conditions found (which see).

Specimen VI. Median Nerve - Stained

This specimen shows degeneration of the axis cylinders, and leucocytes in the nerve bundles, and some dilatation of the lymphatic spaces - a parenchymatous neuritis.

Specimen VII. Median Nerve Transverse and longitudinal appearances (both shown in the drawings) - Stained Gramic Acid.

In the transverse view we find the medullary sheaths swollen and of all shapes.
shakes, and in many cases there are no axis cylinders.

In the longitudinal views we find moniliform arrangement of the axis cylinders in some cases and irregular contortions in others, while here and there are axis cylinders, and here and there a normal nerve with nodes of Ranvier.

Specimen VIII. Section of a portion of the right wrist, showing a nerve and an artery, stained haematoxylin.

On examining the artery we find that the middle coat is thickened. This thickening is not due to a pathological change, but to a contraction of the normal coat of the artery. That this is the case is proved by the arrangement of the internal coat, which is found to be thrown into deep folds.

Specimen IX. Section of the left wrist—stained haematoxylin—and showing arteries and nerves in similar conditions to those already observed and which were made from the right wrist.
These sections of peripheral arteries and nerves—and the excellent drawings of them—are of extreme interest.

The neuritis has been demonstrated previously by St. Affleck, Pithes and Vailland, and others, but the combination of the arteries, although stated as being present, has, I think, never before been conclusively demonstrated. I think that these two conditions taken together will be of immense service in clearing up the difficulty which still surrounds the pathology of this disease.
Case II

The second case differs from any of the cases referred to by Raymond, but as the symptoms were spontaneous and symmetrical, I think it is worth recording. Unfortunately the account is fragmentary, and not as full as the case deserves, although it is as full as was possible under the circumstances as the case did not come under any notice until the day on which the child died, having been previously taken from one doctor to another, and it had been under five different doctors besides having undergone some treatment prescribed by a chemist. I saw, and examined the child six hours before death, but the parents refused to allow anything to be done and therefore an autopsy was impossible. The account has been collected from various sources, mostly from the mother, from whom I managed to obtain some particulars by a system of cross-questioning.

Agnes Q., aged 1 year

A satisfactory family history could not be obtained. There appeared, as far as I was able to obtain answers to my questions, to be no history of epilepsy, but one of the patient...
The patient's sister had died of consumption at six years of age.

The child first began to be ill during the first week in October 1885. The first symptom was 'inflammation of the eyes'.

She was taken to a doctor who ordered an ointment, which, the mother says, was 'too strong', as after using it the inflammation began to spread over the face, involving the cheeks. Another doctor was called in (who had attended the child which died of consumption a year previously) and he ordered poultices to be applied over the inflamed area. The eyes were entirely closed with the inflammatory swelling, but this somewhat subsided and on December 2nd the child opened her eyes slightly for the first time.

On December 3rd the child was admitted into hospital and its eyes examined, when the oculist described it as a case of 'Catarrhal Conjunctivitis with slight Corneitis of the left eye, and rather severe Corneitis of the right eye'. This conjunctival inflammation was thought to have been increased by the applications obtained from the chemist. Strict darkness was enjoined and during its stay...
to stay in hospital the skin got much better - almost quite right! The eyes also got much better but never right. There was a constant discharge from the nose. The urine could not be collected, but the diuresis was not stained; but the odor of the urine was very offensive, as also were all discharges from the patient, and there was an offensive odor from the patient herself.

The patient became very cross and irritable, and on December 24th there was a bluish exudation over the nose and cheeks. On the night of this date the patient cried all night.

December 28. 8 A.M. The child appeared all right. At 8:15 A.M. the upper lip was observed to be swelling, and bleeding slightly. Slight bleeding then commenced from the eyes and nose, and the upper lip became much more swollen. The child was crying pitiously all the time. The lip then became darker in color, and this discoloration spread, and could be actually seen growing darker, and in
...and in two hours the color had turned to black. The bleeding from the eyes and more continued and at 12 o'clock from the child was black over the greater part of the face.

At this time the mother, who had been sent for, arrived at the hospital, and took the child immediately out of bed and home with her.

It was on the following day that I heard of, and found the address of, the child. I called and examined it. The mother says that on bringing the child home she noticed a discolored spot on the inner right ankle, which was cold and hard. Next day (being the day in which I called and in which the child died) the other foot was found to be discolored and cold, and when I called at 4.15 P.M. on December 29th the condition was as follows—

The child lay in its mother's arms, quiet but making a low whining noise. It had vomited everything which had been given it. Since coming home again the face presented a curious appearance. The skin over the affected...
parts was black, shining and brittle — exactly in appearance like coal and looking as if it had been cleansed by fire. The blackened portion extended upwards, just involving the eyebrows, the eyelids (which were swollen and completely closed) the cheeks — as far back as and involving the ears, the nose, upper lip, and down to the level of the angle of the mouth. The lower lip and chin were not discolored, nor was the forehead involved. The blackened part appeared swollen and hard and was cracked in places shining discolored and gangrenous tissue underneath. The neck, trunk, arms and hands were natural. The legs however were involved. The right leg, which was the worse, was swollen, tense, hard and suggested a fluid of solid edema all over. The foot was deep purple in color and this color extended up to the knee — above which, up to the groin the leg was a dead white color. The entire limb was quite cold and no pulse could be felt.

The left leg was also discolored but
not quite so deeply as the right; the white appearance and the coldness of the thigh being not quite so marked.

The child recognized its mother's voice, and showed this by putting its hands into her dress as if to get to the breasts; but it made no sound except the low whining noise before mentioned.

The color of the lips deepened and they both turned black up to the knees, and the child died about 9 p.m. the same evening.

It is false reported that the mother refused to allow any examination and was indeed sullen over the entire case.
Abstract of some of the cases recently reported by other observers:

**Case III**

Reported by Dr. Upland to the British Medical Association, Glasgow, August 1868.

M. R., aged 16. Admitted to Edinburgh Infirmary on January 20th, 1867, with severe pain and swelling of both feet, especially the left, which had existed since January 3rd. It began after exposure and walking in melted snow for two days previously, when there was extreme chilliness of the feet which soon passed off.

Two days afterwards, pain in both feet, which returned each day after meals and lasted several hours. Pain increased and by Jan 15th she was confined to bed. On Jan 23rd there was intense pain in both feet which were swollen and blue; the toes being extremely edematous, as if they would burst. The color of the feet varied from nearly black to pale, and the pain showed exacerbations. Pain continued without relief until admission.

On admission she suffered extremely and sat up in bed until the feet changing very, as this gave relief. Both feet showed extreme nervous engorgement and swelling.
particularly the dorsum and toes of the left foot; where there was a large dark bluish
suggested gangrene. The congestion extended as far as the ankles and up the inner aspects
of both legs, but it occasionally alternated with the appearance of pale lividity.
Any attempt to touch the feet caused screaming, and she showed extreme nervous
excitement and irritability. There was pulsation in arteries at both ankles.
Coldness and numbness of feet. Cerebral and mental functions normal. Pulse
120. Temperature 101.

The gangrenous condition of both feet became more marked. The whole of the anterior
half of the left foot became shrivelled, mummified, and black as coal, and all
the tissues with had perished. In the
right foot the gangrenous area was almost
as extensive, but more superficial, chiefly
affecting the outer skin, involving especially
the four outer toes, the first toe being quite
healthy.

The gangrenous mass of the left foot began
to separate - a distinct time of demarcation
forming in the early part of February.
The right foot improved and there was
only
only ulceration of the skin of the dorsum, and the points of the four toes were destroyed.
Pain was less vascular engagement of the surrounding parts better.
Blood cupules 4:300,000, no anaemia. Urie: no albumen, but phosphates.
March 1. Left foot amputated and with slight sloughing of the edge of the heel flap healed well.
Patient was developed new complication coughing up blood but no tubercle bacilli or lung tissue.
Right foot points of the four toes separated, near the base of the fifth toe was lost but only the distal phalanges of the second toe the great toe remaining healthy.
On November 3rd a creeping ulceration in right great toe and slough of adjoining toe.
November 4. Great pain and these toes turned almost black. The slough of the left foot was not affected. In two days the pain was better and the dark appearance of the toes less marked and patient was soon well.
December 2. Lesion feet became purple almost black but by the 7th this had passed.
passed away
April 24th 1868  Keeping well, but subsidence during the winter tended to bring on the pain and discoloration. Catacreas has returned which had been suspended during the entire illness.

The amputated foot was examined, when the blood vessels leading to the diseased parts were quite healthy. The internal plantar nerves was dissected out, and feelings made as far from the diseased part as possible. The nerve was found to have suffered extensively from neuriitis, and was undergoing degenerative changes, many of the bundles being entirely destroyed and replaced by fatty matter. These appearances were so striking and unmistakable, as to preclude the theory that they were secondary to the state of the gangrenous tissues.

Case IV  Reported by Dr. Aplleck on the same occasion.

J. P. aged 16; admitted the Edinburgh Infirmary April 20th 1868. Severe pains for a week in hands, especially the right.
Liable to such attacks for the past five months occasionally affecting both hands and feet; and I varying severity. Right days before admission, after a long walk, she felt much pain in her feet and hands. This passed away from the feet, but the right hand became worse, and the tips of the fingers blanched. Pain was severe and continued for four days, being more severe at night, and worse in the middle and ring fingers.

On admission she appeared healthy, but suffered severely. In the right hand the fingers were blanched and corpse-like, especially the second and third phalanges, and the nails were absolutely colorless, and the points of the fingers of varying hardness. There was great pain, but the fingers and a large part of the dorsal aspect of the hand were anaesthetic to the touch.

The fingers of the left hand were also pale and painful but to a much less extent. Also pain in the toes of both feet, but the appearance was normal.

The various systems of the body were normal with one exception - the heart and vessels generally were normal, but in the right arm
arm. There could be felt no brachial, radial, or ulnar pulse. The right subclavian and axillary arteries could be felt, but they were smaller than the left, also the right arm was half an inch smaller in circumference than the left—when the arteries were normal.

April 9. On being exposed the whole of the right hand was of a uniform pale cuticle appearance and very painful, but in a minute or two the colour changed to deep purple a livid, and became less painful.

April 10. Same symptoms.

April 11. A dark spot on the tip of the middle finger and on the 12th a similar spot appeared on the tip of the ring finger. The pain varies but the patient is always irritable. Considerable areas on the dorsal and palmar aspects of both hands are more or less anaesthetic, and these areas are constantly changing.

April 19th. Condition not much changed. Pain less. Two gangrenous patches of skin on the ends of the right middle, and ring fingers are separating. Still pallor and engorgement of the hand when exposed.

May 2.
May 4. The gangrenous tip of the middle finger came away and on May 12th that of the ring finger. Healthy granulating surfaces left. Patient improved steadily.

May 31. Dismissed, feeling quite well although frequently examined, beyond the pulsation of the carotid, no brachial, when a radial or other pulse could be observed.

July 25. Again seen. Right hand felt cold and the fingers generally, especially the three first, were of a deep purplish tinge contrasting strikingly with those of the other hand, but painless. Creatures of the finger tips are healthy. She could use the hand and arm well, but the measurements showed the same diminution as before, and the absence of all pulses was again confirmed.

Case V. Reported by A. Turnbull (Glasgow Medical Journal December 1888)

Alice M. Born January 20, 1881. Family well grown; organs of body healthy. Action of heart marked irregular, varying from 15 to 16 beats per ten seconds; pulmonary second sound accentuated. Family history good. When two years old began to suffer pain in
feet and legs. In the spring of 1883, after exposure, the legs and feet became red, and
a warm bath was given by the patient's mother.
Soon after the bath, a large black patch
appeared on each arm - on the outer aspect
above the elbows. These patches had disappeared
before bed time.
In March 1883 a black patch appeared
which was said to cover the whole of one arm,
the rest of the body remaining perfectly free.
From this time onwards till July 1883 she
had several attacks, in one or more of which
the ears were affected. During this month
the head for the first time, an attack of
haematuria. Subsequently proved to be
paroxysmal. From then till now, she has
had numerous attacks of local asphyxia,
and repeated attacks of paroxysmal
haematuria. These two affections never
occurred simultaneously, but always
alternately.
Several slight attacks which soon passed
were reported and in October 1886 a severe
attack occurred, two patches remaining
and becoming gangrenous. The feet as far
as the ankles, and the hands were of a
deep purple tint. The colom was equally
diffused.
diffused and did not disappear as pressure. The discoloration passed off in about thirty minutes—passing off in a peripheral direction the fingers & toes being more or less livid when the hands and feet were of normal colour.

On the left foot two patches of gangrene occurred, in the little and adjacent toe. The necrosis was superficial, and well defined scars remained. The gangrene being asymmetrical. Another attack not followed by gangrene occurred June 22nd, 1887.

In the spring of 1888 she was stripped for a bath, and had an attack varying from the preceding ones. Two large patches appeared in the left forearm—extensor surface, just above the hand, and one below the knee, inner aspect of the leg. The mother could not say that the two patches were confined to the left side of the body. The patches were described as looking like large scalds, and within these apparently scalded areas were numerous white spots, projecting above the general surface.

The child has had at least—linearly attacks, gangrene only occurring in one and they seem always to follow cold—either cold winds
winds on being stripped - the child's father

can predict an attack, as the eyelids become
bloodless, and the eyeballs and lips
assume a yellowish tint.

Case VI. Reported by Dr. Walmsley of Rochdale and
referred by Dr. Laidlaw, M.D. cit.
Scarlet fever occurred in a family and one
of the children was sent to hospital. Another
of the children was found to have suffered a
slight attack, and desquamation was taking
place. He had been playing out of doors
on the fourteenth day of his illness. A purple
patch appeared on the calves of his legs next
morning. He was sent to the hospital.
Temperature remained normal, and his general
condition appeared excellent. Other purple
patches appeared and extended to the
dye crease.
Death occurred suddenly thirty-six hours
after admission to the hospital. No
post-mortem was made.

Case VII. Shown to the Medical Society of
London by Dr. Reesor, March 18, 1849

The patient
The patient—a young woman—had been subject to repeated attacks during the past six years. The left arm turned quite black for a time. There was generally some loss of sensibility for a time, and now the symptoms came almost daily. A patch on the hands Achilles also turned black.

Case VIII Shown to Sheffield Medical Clinical Society by S. Porter—March 28, 1849.
Patient suffering from attacks of local asphyxia in both hands and feet, independently of exposure; sometimes three or four in a day, alternating with paroxysmal attacks of rigors and pain, and vomiting usually followed by slight jaundice. No icterus, though albumen, as well as bile, had been present in the urine after an attack, but no reaction with the guaiacum test. In the intervals, the hands and feet recovered their natural color and warmth. No pain in hands and feet during the attack. The patient was a married woman, aged 47 years. She had
She had had the attacks in the fingers about four months, had not suffered previously from cold fingers or chilblains, and had not been especially exposed to damp or cold. Nine children living. Carotid still regular; family history good; no chills or aches. Abdominal tenderness over hypochondrium and Xiph." hypochondrium; field of vision contracted.

The three following cases are from notes furnished by Dr. B. Heath of London.

Case IX Family S.B. Aged 31. Slender.

Family history. Mother died of right hemiplegia at the age of 64. Foster of bad leg; aged 40.

Menstruation commenced at 13 and has been regular till lately. In two or three years they have lasted ten days but are and excessive in quantity—whch varies at alternate months and is dark-colored. Rough hernial at apex—lonest between sternum and apex. None at base.

Had nausea during right infra—

clavicle region.

When about 16 (1870) one day while dressing
dressing, noticed that the left little finger went white and 'died away.'
From that time on and off, one or more fingers have been liable to go dead from cold, or from sudden fright or start. She gradually got worse. The fingers turn very blue, or even quite black before their recovery, and this dark colour rarely occurs without the previous deadness. No pain during the local synechoe, but excessively painful when the circulation is being re-established and aching and tingling right up the arm. Sensibility returned, but not abolished and movement is difficult.

During recovery the appearance is remarkable. In some parts the dead shiny white of synechoe is seen, while the chief spot is black or blue-black in colour. In the midst of these dusky areas—in some cases in the back of the hand and on one finger, a patch of purple colour appears and gradually spreads until the hand finally looks 'like raw beef' as the patient describes it.

Eight years ago (1880) the patient the right index finger got 'poisoned' and a whirlwind followed. No bone involved and the nail
The nail did not come off, but it is now covered over the end of the finger and does not fit closely.

In several years she has had what she describes as 'corns' in the tips of all the fingers, which she can peel off. Usually there is left a round but slightly punctured superficial scar. Sometimes a breach of surface is left which takes some days to heal. Evidently these are dry gangrene of superficial pieces of skin. Similar phenomena occur on the dorsal aspect of the fingers. They occur on both hands, but not on the feet. The feet never get ulcerated or sore. They 'go dead,' not blue. She is not subject to clubbing. The rains go blue-black, but no destruction of tissue has occurred.

The other two cases though not typical present interesting features which entitle them to be recorded.

**Case X** Miss E. L. age about 40 (b. 1882). Suffered from 'dying away' of the fingers for many years. She says she began ten years ago with a 'small ulcer' in mouth getting to its worst in two weeks, very bad.
very hot and painful, then gradually getting well. Teeth being drawn had no effect on these.

Two years ago I felt a pricking stinging sensation in several fingers, between the nail and second joint. These broke into deep ulcers, like broken chilblains. Cold and heat irritate the fingers. As they heal they desquamate in large flakes. The inflammation comes under the nails and causes them to be mis-shapen and also found the end of the nail. Later the left foot became similarly affected. Both ankles and hands are worse at the menstrual periods, beginning about a week before, then getting a climate and improving again a little. The fingers smart very much and when warm in bed the feet are almost unbearable. She never had chilblains previously. The ulceration was worse at the dorsum of the fingers over the two terminal phalanges. Here they looked like very bad chilblains. The whole fingers and sometimes the hand also were swollen. The neighbourhood of the ulcers was bluish, and the ulcers showed a raw surface covered with thin pus.
pus. The tips of the fingers under the nails were similarly affected. Both hands and feet were very painful, worse at night when they used to swell and get very red and list. No treatment did more than palliate the condition of the fingers, but the general health improved.

Case XI  A. F. aged 54. has for ten years had attacks of deadness of the fingers-palpable white, numb & devoid of sensation.
This seems from even a slight degree of cold. The cold sometimes causes ecchymosis pain without the actual 'dying away' of the fingers. As they recover there is burning and tingling.
For several years has had attacks of inflammation round nail - destroying the nail in one instance. During the winter had a number of superficial ulcerations of the skin over the fingers. They did not discharge, but became red and the skin thickened and then desquamated leaving either as tissue or thin, smooth, parchment-like scars. On the left hand the inflammation has only attacked the neighbourhood of the nails - not the fungus.
numbness. There is no affection of the finger tips. She gets 'feathering' about the toes but they do not 'bleed'. This patient has no pronounced blueness of the hands or feet, only the fingers. She suffers from hemorrhoids which bleed a little.

Cases V and VI seem scarcely to merit the diagnosis of Raynaud's disease. However, they show the difficulty in diagnosing true Raynaud's disease from slighter conditions which resemble it; as Case II shows the difficulty of excluding grave forms from such classification. Raymond Ewens says (Raynaud’s Essay Med. & Phys. Soc. vol CXXI, p. 112) — “if the gangrene commences by a diffuse lived colon, it is almost always confounded at the commencement with chilblains...The itching and painful sensation are naturally referred to a very common affection”. “It is probable that these (chilblains) have some relation with gangrene of the skin”. I will only say - simultaneously
Case XII. Dr. Southey (Pathological Society of London, December 5, 1862).

The body of a child aged 2½ years who died with symmetrical gangrene. She was of healthy parentage, and had been previously quite healthy, except that two or three months ago she had a febrile attack with some purpuric spots. On Friday, December 19th, she had a pain in the head; on the following morning, she seemed quite well again, but in the afternoon complained that she had a numbness in her leg, but when her father pressed it, she said that it pained her, and he then noticed some lividity over her calf.

Soon after, she was sick, febrile, and complained of headache; at six p.m., she was worse and the lividity of the calves had extended both upwards and downwards; in the night the backs of the arms were likewise affected and at about 6 p.m. petechiae formed.
formed upon the buttocks.
She was admitted into St. Bartholomew's Hospital at noon on the third of December, looking mortified; the pulse rapid and febrile. The tibial arteries could not be felt; the lungs and heart were normal; at 6 p.m. she had taken some nourishment; she was ordered a warm bath and some nitro-glycerine; the cheeks were beginning to show a slightly livid redness.
At 9 p.m. convulsions came on, and continued until she died at midnight, thirty-two hours after the first symptoms. St. Moore had cut up the left femoral and tibial arteries, but no umbilus or other obstruction could be found. The theory advanced had been that there was spasm of the arteries, and then migration of blood elements into the skin, for the gangrene was quite superficial.
St. Norman Moore had examined the blood microscopically and said that it was whitish comparatively, containing many fat globules, something like the so-called "raspberry-juice blood" found in some cases of diabetes. Only an ounce of urine could be obtained and it contained some albumen.
Case XIII. Mr. A.S. Murray (British Medical Journal. January 9th 1886) relates a case of a boy aged 3 years who had been fairly healthy all his life. Mr. Murray had operated for club-foot some months before the attack. The last division of tendons took place on December 29th 1884, after which the leg was put in irons. He remained in good health until March 4th 1885, when, in the afternoon, he began to scratch the left knee and thigh. Shortly afterwards, he began to cry, and said his leg was sore. His mother then took off the instrument and boot, and noticed a slight discoloration on the upper and inner part of the knee, not as large as a sixpence. It quickly extended down as far as to where the padding of the splint had reached. About 4 p.m. an angry red line was noticed round the discoloration. Next morning the patch was much larger and darker. On the morning of March 5th, the discoloration extended from about three inches above the knee to about two inches below it on the inner and under surface of the joint. No other part was affected, the child was suffering great pain in the diseased part, and also...
and also complained of pain in the extremities. The pulse was extremely rapid and irregular. The breathing was very quick. The temperature was below the normal. The surface of the body was peculiarly cold.

In the course of an hour, both feet had become black and much swollen. In about an hour more, the hands and arms had assumed a dusky hue, and were much swollen. On the morning of March 6th, another discolored patch appeared on the back of the left thigh; and towards the afternoon another came on the left cheek.

By this time, the feet and hands were quite gangrenous; all pain left the child about 10 o'clock, and he died at 10:30 p.m. The urine appeared normal. The disease spread so rapidly in the arms, that the proper care could actually be watched.
These two cases (XII and XIII) are similar in character to Case II. They are remarkable for the suddenness of the onset of the symptoms, and their severity, and for the rapidity with which the gangrene spreads. As in Case II death occurred within 48 hours, in Case XII within 36 hours, and in Case XIII within 26 hours, from the commencement of the symptoms. They are also remarkable for the extent of the gangrene, involving as much as the back, deep up to the spine, and the greater part of the face and the ears. In Case XII embolism or thrombosis cannot be ascribed as the cause. In Case XII, the arteries were examined and no obstruction found. They must be ascribed to spasm of the arteries along with some hypoxic change, and constitute a severe form of the disease described by Raymond, and of which one observer states that "life is not endangered in this disease." These five fatal cases (Cases I, II, VI, XII, XIII), with others (British Med. Journ. Vol. II. 1882, p. 1167) considerably modify this statement.

Case XIV. Reported by J. Callahan Powell

British Medical Journal January 30th, 1886

G. P., aged 48, admitted July 14th, 1887. He had been a rough rider in horse artillery for twenty-two years, and subsequently drill instructor to police for six years. During all this time his health has been excellent. History good - no syphilis, gout in meanwhile family history good. On May 12th he felt himself ill with a sore throat. This was followed in a week's time by sudden loss of voice, for which he was treated by a practitioner, who said he suffered from diphtheria. During his convalescence, three weeks later, when dressing in the morning, he discovered his nose had become blue and swollen during the night - "in fact resembling an overgrown mulberry". This was followed in a day or two afterward by a similar appearance in the knuckles of the fingers of his right hand and in his ears. In the following week, the ring and little fingers of the left hand were attacked in the same manner, and gave him considerable pain. The discoloration of the fingers steadily increased until, at the end of three weeks, they were quite black along their entire length. His blackness then

[Missing text]
needed to the middle joints, where blebs formed between the sound flesh and the swelled joint. Subsequently, large patches formed in the second and third toes of the right foot, and along its outer margin. This numbed and impaired became swollen and painful, the latter being livid, and an ulcer forming near the tip. This was stopped, so that he could not breathe through it, and was only cleared by squeezing out some tough brown stuff which came away with much difficulty.

Careful examination on the day after admission showed that, in general, he was a well built man, above the average size, and fairly healthy looking, although he stated that he had lost much flesh and become much weaker during his present illness. He had no areas sensitive nor had he any signs of general anaemia. His nose was cold and livid. The edges of the redness of both ears were dull; a small dry slough separating from the left. The unguis, and part of the middle phalanges of the ring and little fingers of the left hand, and all the fingers of the right, were black, dried up, and completely unrecognizable. The remaining fingers of the left hand and both
both thumbs were cold, glossy, devoid of hairs and of a deadly red colour at the tips. The ungual phalanges of the second and third toes of the right foot were also splintered. The splintered portions of the fingers were separated from the small fleshly bullae containing turbid serum, but there was no distinct line of demarcation. Careful physical examination of the chest did not reveal anything abnormal. No cold symptoms were detected in the radial venous or pulmonic. The voice had been regained. The temperature was normal. The urine contained a little albumen, but no casts nor sugar. The blood, examined under the microscope, showed an increase of the white corpuscles. The digestive functions were well performed. He complained of nothing but slight pain and tingling in the fingers occasionally. Under liberal diet he improved rapidly and in September third went into the country. On his return a month later a well defined line of demarcation appeared on the gouty fingers. The sloughs separated from the ears and took an interesting interlacement of granulating surfaces underneath, which
which rapidly healed. The stumps of the gangrenous fingers were slow in healing, and his thumbs and ears were still cold and slightly discoloured, but he was sufficiently well to resume his duty as instructor of police. There was no possibility of ergotism. The absence of any general anaemia or any disease of feebleness of the circulation, the presence of swelling prior to the shrinking of the gangrenous parts, and the occurrence of the disease in summer, make the case more remarkable. August 19th. Patient looks and feels quite well.

Case XV. Reported by Dr. F. Weaver
(Lancet, November 3rd, 1888).

D. H., aged 45; Widows for ten years.
No history of rheumatism, appendicitis, chlorosis, or goitre, and no exposure to malaria. Always good food - no excess of alcohol. Family history good.

No miscarriages. No history of nervous or vascular disease in family.

Patient had been feeling poorly for three or four weeks; appetite failed and fingers at times felt stiff. On January 1st she went...
She went out to shake carpets and found that her hands became stiff, could move fingers, but could not hold anything. Next day in the morning in washing the children in warm water, fingers became stiff and painful for the first time, and she could not do anything. Whole length of the fingers and the ends of the thumbs were painful, and were blue-looking and cold. She could not sleep because of the pain; attempting to warm the fingers made the pain "dreadful". Pain generally commenced in the right hand but in a few minutes would follow in the left hand, while the pain lasted she was very thirsty. This state lasted on and off for three weeks, there being also two or three attacks of pain during the day but the worst at night. The hands were rubbed during the first week, wrapped in cotton wool during the second, and linseed meal poultices applied during the third. Patient said that the poultices gave her most relief. About a week after the first application of the poultices, the fingers in their whole length, from the neck, palms, and fingers joint downwards swelled up like bladders. They remained swollen for twelve hours, and then the swelling
Swelling went down as suddenly as it had come on, and the skin then appeared to dry up. The skin on the palmar surface of the fingers and thumbs, from the metacarpal-phalangeal joints down to the last inter-phalangeal joints, then began to peel, and the ends of the fingers below the peeling began to turn black, but did not peel. The skin on the dorsal surface of the fingers and thumbs was rough, but did not peel. There was pain in the thumbs at the same time as in the fingers, but not so great, nor did they turn black. About this time, as the hands got a little easier, a "jumping pain" commenced on the palmar surface of the patient's heels, and when she put her feet near the fire she big toes of both feet swelled and "bunched" (just as the fingers had done) at their ends. The pain in the feet lasted about a week.

March 5, 1884. Patient is thin and blue-clothing, as if from cold. Right-hand. There is a dry, black, slough, on the palmar surface of the first finger, extending above the last inter-phalangeal joint; on the dorsal surface of the same finger a brown discoloration extends a little above the nail. The skin is peeling.
is feeling up about a quarter from inch further on to the finger than the thumb extends. There is a small ulnar on the end of the ring finger, and a larger one on the tip of the little finger. There is a distinct depression on the end of the middle finger, but no slough. The patient cannot pick up, or feel, a pin. She feels pressure on the ends of the fingers, as they are rather sore. Sensibility is  

shunned | middle finger and palm of hand unaffected.  

Left hand _There are sloughs _on all four fingers_; none of these extend round to the dorsal aspect of the fingers. 

Nails normal on thumbs and fingers of both hands, except on the first and little finger of the right hand; the patient states they are not growing on these. 

**Pulses at wrists:**  

In the right forearm

There is a distinct radial pulse, and a faint ulnar pulse; in the left forearm the radial pulse is with difficulty felt, but there is a distinct ulnar pulse. The feet are cold; no sloughs. Feet are always cold. The posterior tibial arteries are easily felt at both ankles; the dorsal arteries of the foot is distinctly felt in the right foot, but not in the left. 

**Renal:** Explain - the patient has always...
has always passed a large quantity of urine; had to get up once a night for the last few years. Never noticed any change peculiar in urine during illness. Urine (March 14th, 1877) Sp. gr. 1.018, acid reaction; no albumen or sugar; slight cloud of mucus; nothing microscopically.

Last menstruation was first week of the new year, during present illness - menstruated twice together within a short time, and lost a great quantity the second time - no menstruation since. Memory good, hearing and sight good. Knee joints normal and well marked, no ankle clonus. No affection of joints, and walks well. Heart normal; no murmurs. Pulse 76 per minute. No sign of arterial degeneration, except the arterial condition in the forearm. No emptiness or discolouration (except fingers).

Lungs normal; bowels regular, no diarrhoea or vomiting.

March 14: Fingers of both hands from knuckles to tips of a peculiar straw-blue colour and very cold. Right arm - distinct thump in weak radial pulsation, and a faint ulnar pulse; left arm, radial artery seemingly made out, but distinct ulnar pulse. In the feet - posterior tibials on both sides were...
were distinctly felt; dorsal arteries not felt in either foot.

July 11. Side of fingers - All day, except that in the end of the first finger of right hand, tone was disappeared, leaving flat white scars; nails growing on all fingers but first of the right hand; fingers still become blue when cold. No pain in the toes now.

September 19th. No optic neuritis; fundi are normal.

November 4th. Feeling better. Hands warmer. The thorn from the first finger of the right hand came off in August without loss of bone, but of the whole pulp of the finger. The fingers are now all normal, except the first and little fingers of the right hand, which are very pinched and have badly formed nails. No return of numbness.

One day or going out without evening a line across the back of the hand, he had an attack of pain and loss of use of one hand, but the attacks passed.

December 16th. No tenderness along median and ulnar nerves of both arms - if anything less sensitive. Lethargy pains in feet.

December 22. Pulse 100, and cannot be alleviated.
Obliterated by pressure. Veins of arms
surround April 5th, 1888. Began to menstruate
early in January of this year - ceasing
the first two months, slight the last two
months. Cannot tolerate anything
cold, and since menstrual discharge in
January her feet have been tender
and cold.
July 26th. Patient in good health, and
the condition of the arteries improving.

A peculiarly noticed in this case was
the varying state of the arterial pulsations
in the arms... which... Weaver gives
in a tabular form, and which shows that
the condition varied considerably;
since, as Weaver remarks, this
condition must be due to spasm of the
arteries.
### State of Pulsation in Arteries in Forearm

<table>
<thead>
<tr>
<th>Date</th>
<th>Right Arm</th>
<th>Left Arm</th>
</tr>
</thead>
<tbody>
<tr>
<td>March 9, 1887</td>
<td>Radial: Distinct</td>
<td>Radial: Very faint</td>
</tr>
<tr>
<td></td>
<td>Ulnar: Faint</td>
<td>Ulnar: Distinct</td>
</tr>
<tr>
<td>July 11</td>
<td>Radial: Hardly felt</td>
<td>Radial: Faint pulsation in both</td>
</tr>
<tr>
<td>Nov 1</td>
<td>Radial: Doubtful if felt</td>
<td>Ulnar: Doubtful if felt</td>
</tr>
<tr>
<td>Dec 1</td>
<td>Radial: Cannot be felt</td>
<td>Ulnar: Distinct</td>
</tr>
<tr>
<td>Feb 22</td>
<td>Radial: Cannot be felt</td>
<td>Ulnar: Faint</td>
</tr>
<tr>
<td>April 2, 1888</td>
<td>Radial: Very faint</td>
<td>Ulnar: Faint</td>
</tr>
</tbody>
</table>

Other cases of this disease have been reported, but cannot be included here, as they would extend this already lengthy series beyond all reasonable or manageable proportions. I have thought it necessary to quote at some length the above series of cases, in order that we may have before us sufficient...
Sufficient material from which to form opinions as to how the clinical facts observed correspond with the observations and theories which have been made in connection with this disease.

The symptoms of the disease as originally described are the occurrence of discoloured or black patches on the body, arranged symmetrically. These patches are generally preceded by ischaemia, and occur especially at the ends of the fingers or toes; also at the ears and tip of the nose etc. These patches may be transient and may recover without gangrene occurring.

In Case V there were over twenty attacks, in only one of which did any gangrene take place. The discoloration may not pass off, but may be followed by gangrene — usually superficial — involving the skin and tips of the toes or fingers; but in the first of these cases it was more extensive — as also in Case III. In Cases XII and XIII it was far more extensive, while in Case II both legs became gangrenous as well as the greater part of the face.
Pain is usually a marked symptom; in cases III and IV it was especially marked, as also in Case VIII of Raymond's Series. But in Case I, it seemed to be very slight; and in Case VIII it is stated that there was no pain.

Let us see how the facts stated in the above series of cases, agree with the theories of Raymond.

Predispousing Causes. It affects especially the female sex. In Raymond's first series of cases he cited twenty cases in females, and five in males. In his next series (contained in his New Researches, New Syden. Soc. Vol. CXXI), there were two females and four males. In the fifteen cases reported above there were twelve females and three males.

Age. Raymond's cases occurred in the great majority, between 18 and 30 years - the average being 25 years, and the youngest -
Youngest case he had seen was at 3 years, but the first case here occurred in a child aged 1 year 4 months — the youngest I have found on record. The second case, although occurring at 12 months, being atypical.

Nothing of importance can be deduced from the temperament, constitution or previous illnesses, in the above cases — as also was the case in Raymond's cases.

In no cases had any heredity or factor evident played any part in its production, although the winter and spring seem in the majority of the cases to have been most favourable to its development.

Inciting causes — usually exposure generally to cold — but often an inappreciable degree, as in Case V; but in Case I it rather seemed to be after exposure to the sun than cold.

Raymond mentions that the condition may be caused by infection, and this is confirmed by Case X, which I hold confirms the theory of nervous origin of the disease.
As to Antérulic - there was no evidence
of this, in any of the cases, while in Case I
the arteries were found to be healthy
(see microscopic specimens). Also in
Case III the arteries were examined and
found to be healthy.

It is interesting to notice that it is distinctly
stated in Case I, that the discoloration
did not disappear in pressure - as it is
described by Raymond as invariably
doing.

In Case IV there is a deficient condition
of the circulation of the limb - similar to
the condition observed in Cases XVIII and
XXII of Raymond. This condition
however did certainly not cause but
may have contributed towards the
attacks

Although Raymond in his second series
of cases describes a case in which a
state of contraction of the retinal arteries
was observed (New Researches - Case I,
pp 155-160) and assumes that this is
a visible proof that the arteries are
in a state of spasm generally - it has
not previously been demonstrated absolutely
that the
that the arterioles in the affected limbs are contracted. That such is the case, is absolutely demonstrated by referring to microscopical specimen VIII from Case I. The thickening of the middle coat being physiological and not pathological, and due to a contraction, as shown by the increased folding of the internal coat. (See also accompanying drawing of artery.)

Interischemia - there are no symptoms at all pointing to this condition.

Paresthesia - can in all the above cases be absolutely excluded.

As to the condition of the blood - more evidence is required. In Case III the condition of the blood was good, the red cells numbering 4,300,000 per cubic millimetre, and also in cases reported by Sir Barlow (appendix to Raymond's Cases, 2nd ed.) the state of the blood will not account for the condition; nor does the examination of the blood in Cases XII and XIV reveal any important change.

Nervous influences - that panenceph may be caused by nervous influences
is a well demonstrated fact; as gauzene occurs after injuries of nerves. Irritation of nerves may produce gauzene. If the parts supplied by these nerves we get stimulation of the sensit-motor function, and spasm of the vessels, the spasm of the artieoles may give way and recovery occur, or it may persist and cause gauzene. Stimulation of the nerve produces spasm of the artieoles as proved by Raynand's experiments (New Researches, Op. cit.) that peperinal neuritis can produce gauzene, seems to have been proved by the experiments of Pitres and Vailland (Beyelle Medecin de Paris 1887). They produced neuritis by means of hypodermic injections of sulphuric ether. Cutaneous anaesthesia, disorders of motility and even serious trophic lesions, were observed to follow such injections, and the experimental researches of Amogne and Salvat proved, that the mechanism of these lesions was a neuritis.

When an injection of half a cubic centimetre of sulphuric ether was made deeply into the cellular tissue of the muscles
The muscles on the back of the thigh of a guinea-pig, paralysis of sensation and motion resulted in the parts of the limb below the level of the injection. Generally the anaesthesia occupied the two anterior toes, and the outer aspect of the leg. After a few days these phenomena may be accompanied by oedematos swelling of the foot, ulceration of the toes, and decrease, falling off of the nails, etc. The anaesthesia and the paralysis are manifested immediately after the injection, and in a short time they reach their fullest extent, and when developed, may persist for several weeks or months.

Pitres and Vailland found on histological examination, the nerve above the level of the lesion normal—below there was Wallerian degeneration. Also in Case III, the neuritis seems to have been demonstrated as the cause as it is stated by Dr. Affleck, that the appearances in the nerve were so striking and unmistakable, as to preclude the theory that they were secondary to the state of the foot. Also severe neuritis is found in Case I (see Specimens VI and VII).
Cases which have an important bearing on the relation of peripheral neuritis to this disease are reported by different observers.

Moutstein (cited by Loebner, "Handbuch der speziellen pathologischen Anatomie und lokale Differentialdiagnostik. Vienna. 1866. p 35") had a patient, a man aged 51, for whom amputation of the right leg in the upper third was performed, on account of gangrene of the foot, which had commenced two months previously. A week after the operation he died with a high temperature. This urine was normal. The gangrene had led to the separation of the first, second, and fifth toes, whilst the third and fourth were isolated; but the skin over all the toes was involved in the gangrenous process as well as that covering the heel, the inner side of the foot, and the dorsum. The vessels generally in the lower extremity were free from abnormal contents, only in the capillary vessels adjacent to the gangrenous focus were microscopically liquefied albumin present. The peripheral arterial artery showed many calcaneal plates, but no Kromboli adherent to them.

The posterior
The patient's fibula nerve was greatly thickened in its lower part; microscopic investigation showed great wasting of the myelin with collapse of Schwann's sheaths, and chronic inflammatory proliferation of the interstitial connective tissue, especially in the area close to the gangrenous area. The nerves in the peripheral limbs showed similar changes to those in the gangrenous limbs.

The nerve roots of the lumbar region were only affected with neuritis on the right side. Brain and cord were markedly anaemic, and the examination of the viscera gave negative results.

This case cannot be definitely classed as Raymond's disease, but the double-sided affection of the nerves, more extensive on the gangrenous side, is very suggestive. Pitres and Vailland (Archives de Physiologie normale et pathologique, 1885, p. 106) named these cases. The first was a young woman aged 24, of feeble intelligence from childhood, but from 18, began to suffer from tremors and stiffness of limbs, until at length walking became impossible. The lower limbs passed into a state of severe contracture, and the patient was bedridden.
bedridden and delirious. After a time
the feet were noticed to be cold, blue, and
insensitiveness; they gradually became
gangrenous; the left foot underwent
spontaneous amputation, and the
right was at last separated. Numerous
eschars appeared in various parts of the
body; many of these suppurated, and
the patient died from exhaustion.

In post mortem examination, the tibial
arteries were seen each to terminate in
a cutaneous cul-de-sac, which was
surrounded by foamy granulations.

In no part of the arteries of the lower limbs
were adherent lymph found, only here and
there epithelial on the arteries and its branches
and the veins of the limbs generally were
healthy, and examination of the viscera
gave negative results. In the nervous
system there was found chronic hydro-
cephalus of the lateral ventricles, and some
 undue adhesion of the pia mater to the
center of the hemisphere, and the thalamus
was greatly thickened. There was a slight
diffuse sclerosis of the dura mater and
part of the cord, affecting the whole of the
anterior lateral columns, and the whole of
the posterior columns except their anterior
nervi.
fifth. The spinal ganglia and nerve roots, so far as they were examined, were normal. The principal nerve trunks were carefully examined throughout the body. Those of the upper limbs were normal, and the nerves of the thighs were also normal. The anterior and posterior tibial of both sides, presented changes of varying extent, but which were fairly symmetrical. The changes consisted in extensive stripping of nerve fibres with empty sheaths, presenting numerous myelinated and, at long intervals, varicose dilatations, which contained masses of granular protoplasm and drops of myelin. Between the fibres in many places were found abundance of macrophages infiltrated with small granules, and having the aspect of Shultz's encysted cells.

Their second case is that of an old woman, aged 56, a ray gatherer who had been subjected to great hardships, and for five months, along with a sensation of considerable fatigue, had found that in walking she no longer felt the soil on which she trod. Two months before her admission to hospital, blisters formed on the soles of her feet. These she pinched, and they gave little trouble.
About the same time she began to suffer from obstinate diarrhoea. Three days before admission the feet became swollen, painful, and covered with reddish patches on the dorsal surface. Fresh bullae formed on the feet. They were perfectly cold, and anaesthesia on the left side extended up to the ankle, on the right side to the middle of the tarsus. The line of separation formed at this level on both sides, but the patient died from sepsis and diarrhoea before actual separation had taken place. Post-mortem examination showed necrosis of the plantar and tibial nerves, but the vessels of the limbs were normal, and the brain, spinal cord, and viscera were also normal. Piché and Viallard meet the objection that in the above case the necrosis might have been consecutive to the gangrenous process by recording the results of an examination of peripheral nerves in a case of gangrene of embolic origin. The nerves in the gangrenous extremities were found to be normal throughout. They are inclined to regard the peripheral necrosis in their case as due to the pressure, and they hold that most.
A. Raymond's cases of gangrene were of like origin.

Dr. Wigglesworth (Pathol. Trans. 1861, p. 61) records a case of very extensive peripheral neuritis in a woman aged 36, who was the subject of epileptic dementia, and of chronic Bright's disease, and who suffered repeated attacks of spontaneous gangrene of fingers and toes.

Boehncieg (über Hypemische Gangrén, and Beitrage z. Erforschung d. Venenblutverwesungen, Vienna 1876) admits the soundness of the conclusions of Dries and Vaillard, but he resisting the universality of their propositions. He reports a case of a man aged 51, who developed gangrene of the left hand independently of vascular causes. The post-mortem examination showed chronic hydrocephalus and Syringomyelia. Only a slight degree of atrophy was found in the peripheral nerves, and was held to be secondary to the cord lesion. Boehncieg maintains that the gangrene was caused by the central lesion, but in view of the existence of the nerve changes, slight and non-inflamatory, though they were, this conclusion seems hardly satisfactory.
But we find cases of severe neuritis in which we get no gangrene, a where the gangrene is not proportional to the neuritis present.

Hughes (St Louis Western Medical Reporter 1896) relates two cases of severe plantar neuritis. The pain in both cases was intense, being especially severe at a patch under the calcaneo-cuboid joint, and the plantar aspects of the four inner toes. In the first case there was pallor of the foot and toes, but no ptyalism. In the second case there was flushing of the foot.

Cases of severe peripheral neuritis in which gangrene did not follow, whereas the gangrene was not proportional to the neuritis, are related by Mr. Loppy and Ch. Archard (in the second number of Archives de Médecine Experimentale). The first case is one in which other conditions were associated with this affection. It is the report of a case in which well-marked symptoms — as severe and persistent pain, followed by muscular paresis, and washing of all the extremities—occurred about nine months before the

patient's
patient's death from an attack of cerebral hemorrhage and pneumonia. Degeneration of nerve fibres was found in the main nerve twigs of the limbs in varying degree, but in all more marked in the peripheral nerves. In addition there was found obliterating arthritis in the nerves - e.g. in the facial - and it was to the resulting loss of function that the neuritis was attributed. Indeed, a parallel is drawn between the changes thereby produced in a nerve twigs and those of cerebral softening from arterial thrombosis. It is remarked that, had the patient not succumbed to pneumonia, senile gangrene would have developed, and might have been referred erroneously to the neuritis; whereas both conditions would have owed the same origin - obliterating arthritis.

The next case is a case of tabes, complicated with cutaneous gangrene, in the left great toe, but with neuritis changes far more marked in the nerves supplying some of the other toes. Hence the writer does not attribute the gangrene to the neuritis, nor could
nor could they assign pressure as its cause; instead, refer the gangrene as well as the neuritis to the disease of the cord.

Another factor in the production of peripheral neuritis in this case was the presence of tuberculous, from the effects of which the patient died.

In the same journal M. Dujour and Sollier relate an interesting case dealing with the subject of peripheral tabes; to which M. Dujour had previously drawn attention. The case was one of a man aged fifty-four, who for fifteen years had suffered from incoordination of the lower limbs, marked lightening pain, and disturbance of sensation. The plantar reflex was, however, present. This patient also died of phthisis. The spinal cord and nerve roots were found to be healthy, but there was very marked peripheral neuritis, especially in the cutaneous nerves of the lower limbs, less marked in the muscular nerves, and slight in the cutaneous nerves of the hands. The sciatic nerves were quite normal.

These cases seem to oppose the theory that the neuritis causes the gangrene as in the first
In the first case the neuritis was due to obliteration of arteries in the sciatic nerves, and the neuritis was from want of nutrition to the nerves; a similar condition was present in arteries in other parts. In the second case the neuritis was much worse in the toes which did not gangrene, than in the toe which did gangrene, proving that the gangrene was certainly and proportionally the neuritis present; although the neuritis probably was an important factor in its production.

The presence of this neuritis caused then, I think, the need to offer a satisfactory explanation for the occurrence of the gangrene. It may, by interference with the nutritive condition of the part, predispose to gangrene; but that it is the essential and only cause, is I think disproved by these cases. The evidence yet is conflicting, as it has been demonstrated that peripheral neuritis will not itself cause gangrene; at least it has been proved that in many cases it does not do so; while in other cases where gangrene occurs, peripheral neuritis is the only demonstrated lesion.

This agrees
This agrees with the statement of Dr. Borden (Appendix to Raymond's New Researches Op. Cit.) that peripheral neuritis alone will certainly not produce jaundice.

The evidence certainly points to the lesion being in the nervous supply of the parts. And this essential change is still central is, I think, proved by the specimen of the pars, medulla and upper part of the end which accompany these notes; although, a state of irritation (perhaps depending on some change to be hereafter demonstrated) of the end, as suggested by Raymond, would produce the condition.

Specimens V and VI demonstrate the presence of a severe peripheral neuritis, as was also proved in Case III. This would account for the interference with the circulation of the parts beyond, but not for the state of contraction of the arteriolar generally - if this is present.

There is some evidence to prove that this spasm is general - as it was observed in the eye in Raymond's Case I new series - and also in a general blanching which occurred.
which occurred in Case V - the eyeballs and lips especially showing it, previous to the signs of local asphyxia in the extremities manifesting themselves. The spasm may sometimes be general, but the parts where radiation is greatest: viz. fingers, toes, ears, and nose are the parts where the condition produces the most severe results.

In this spasm the veins as well as the arteries take part, producing the stage of local syncope (Raymond, p. 279). The succeeding stage of local asphyxia is caused by the relaxation especially of the vessels, and the blood stagnates in the veins below.

Haemoglobinuria is frequently associated with the disease -

Dr. Barron (op. cit.) cites several cases: One by Mr. Hutchinson (Med. Lines, 1871, Vol. II, p. 678) where the urine became dark after exposure to cold. Dr. Wilks (Med. Lines, 1879, Vol. II, p. 207) had a case of a boy aged 16, with symmetrical gangrene where the urine was at different times dark.
darker in colour and gave the guaiacum test. Granular casts and debris were present, but on several occasions no blood corpuscles could be found. At a later period however some blood corpuscles were present.

Dr. Farley (St. Bartholom. Reports XVI, 1852, p. 157) gives the history of a patient having passed black urine in some of his attacks, but during her stay in hospital with symmetrical jaundice, though a trace of albumen was present, there was no record of haemoglobinuria. In a second case by Dr. Farley (Ibid. cit.) there was for several days a true intermitting haematuria caused by external cold to the surface of the body. Sometimes the blood was apparent by its colour in the sediment, but at other times its presence was only detectible by the guaiacum test. This case was probably of haemoglobinuria.

Dr. Barlow records a case (Transact. Phys. Soc. XVI, 1853, p. 149) where the onset was marked by epigastric pain and haemoglobinuria. The dark urine only...
only appeared once after a fever attack. It gave the quinine test and under
the microscope pigment and crystals but no blood corpuscles.
Dr. Drexler had one case (Penal and
Urinary Medicine Part III 1885 p. 1164)
which is important as the history shows
that the typical attacks of intermittent
haemoglobinuria were on one occasion
replaced by a typical attack of the
parasymmtalic local asphyxia affecting
the hand, and unattended with the
usual urinary affection.
Dr. Reid (Med. Jour. Nov. 19, 1873)
in his own case showed that he suffered
from aseptic and also distinct attacks of
haemoglobinuria related to cold, exposure
and worry. The attacks were associated
with marked proneness to numbness,
tingling, and blueness of the extremities,
the blue patches at times being suggestive
of imminent gangrene.
Dr. John Alexander (Archives of
Pediatrics Oct. 1886) had one case where
in certain attacks of local asphyxia—
chiefly affecting the hands, the child
passed urine @ $p. 1023 and wrist
hematuria. It gave the guaiacum test and microscopically no haematoxylin and some amorphous material, but no blood corpuscles.

Dr. Cavenby (quoted by Dr. Barker - Sp. cit.) also reports a boy aged 12, suffering from paroxysmal haemoglobinuria for five years and the ears were said to be very expanded when the boy was coldly and asked as he got warm. Subsequently gangrene of both ears set in and relapsed several times in successive winters.

Dr. Abercrombie (Sp. cit.) holds that this is due to the same cause as the Raymond's disease, and the presence of bile sometimes noticed (as in Case VIII) to form of the haematic vessels.

Hematuria sometimes occurs. In Dr. Brindley's second case (quoted above) it was present for some days. It was certainly present in Case I. The blood corpuscles being present in large numbers. Also it was probably present in Case II. It was found also in Case V, and occurred here in paroxysmal form as attacks always.
always alternating with the attacks of local asphyxia; as the opthalmic changes
alternated with the local asphyxia in Raymond's Case I New Series.

A haemorrhagic tendency is noticed in many cases; as in these cases of haematuria,
and also in Case II bleeding occurred from the rectum and nose. In Case IV there was
haemorrhagia at first; in Case III the patient coughed up blood although no disease
was found in the lungs. J. Warren (edited in St. Bartholomew's Hospital Reports 1870,
Vol. XXX p. 23) had a case where haemorrhage from the nose took place "permanently for over
a period of two weeks" before the collection of the fingers began.

The interference with vision which is
sometimes noticed (as in Case III) probably
depends on spasm of the opthalmic vessels, as demonstrated by Raymond (Case I
New Series).
Spasm of the arterioles alone could
probably not account for the condition.
Mr. Durean (cited by Dr. Appleyard) gives
his opinion that it could not do so.
It is doubtful whether the stagnation of
the blood in the veins could cause the
gangrene in these cases; it might cause
edema, but of itself, it could probably not
cause the gangrene.

Raynaud says (Sp. cit. 6/62) "I would
say that in the present state of our
knowledge, local asphyxia of the
arthritis ought to be considered as a
neurosis characterized by monocronous
eruption of the skin, méthode erythémateuse
of the gray part of the spinal cord, which
caused the vaasomotor insufficiency".
While allowing that such a condition
might cause the symptoms met with in
this disease, we must maintain that,
so far as the evidence goes at present,
peripheral neuritis appears to play a
more important part in their production.

That peripheral neuritis can cause gangrene
has been shown by Piché and Vaillant —
probably owing to the spasm produced in the vessels, and the interference with the haptic condition of the parts. But that peripheral vasculitis does not necessarily cause gangrene even when severe, is proved by the cases of Mr. Bynn and Aehard, and by many cases of peripheral vasculitis, recorded by others, where no gangrene occurs.

All we can yet state is that in this disease peripheral vasculitis has been demonstrated, and also spasm of the artërioles. It is extremely probable that the cause of the gangrene is to be found in the vasculitis, but evidence has not yet been collected which will point out to us the cause of the vasculitis, nor the exact relation of the vasculitis to the gangrene, and why gangrene should occur in some cases of vasculitis, and not in others. These points can only be cleared up by further evidence and by further opportunities for observation and research as to whether it is the activeness of the vasculitis, or some special form of vasculitis, which causes the gangrene in cases of this disease.
"Symmetrical Gangrene"

Hansarea Seelicia Medain Nerve
Stained with Acetic Acid  X 1/10 oil immersion lens

Lee柚 and Specimen VII

Cust Hayward, 1889.
"Symmetrical Taenurea"
Transverse section median view
Stained orange, X 1/12 oil immersion lens
See text and specimen VI
C. H. Garwood 1889
"Symmetrical Gangrene"
Longitudinal Section of Median Nerve
(See Text and Specimen VII)
Stained with Acidum - x 1/12 oil immersion lens
E. W. Baynard 1889
"Symmetrical Carcinoma"
Transverse section peripheral cutting
Stained Haematoxylin
See Card and specimen VIII

C. W. Baynard, 1879
"Symmetrical Gangrene  
(Eds: Baynard, 1889)  
Case I - Aug 9/68"
Symmetrical Lamproence
(Caudoyward - 1889)
Case J. Aug 9/88
Symmetrical Symphonie
(Asymmetrical 1889)
"Symmetrical Gangrene"

(Bertho.)

Case I. Aug 9/89