



Thesis

on

A Case of Dermatitis
Herpetiformis Multiformis
with special reference
to its etiology.

Illustrated by Photographs.

By

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I have chosen as the subject of my Thesis a skin disease which has been exciting a great deal of interest within the last few years, and which was so named by Dehring who brought it prominently before the Profession in 1884.

My interest in it was first aroused by seeing a case in D. Jamisson's Clinique, and hearing an instructive account of its history, symptoms and treatment.

Being fortunate to meet with a case while assisting D. Bryan of Little Hampton, I thought it well worthy of study and having looked into the literature on the subject I have ventured to give a short account of the symptomatology, aetiology, prognosis and treatment of the disease as it is generally met with, describing as concisely as possible the phenomena as they were presented to me, and noting anything of special interest in the case.

I may mention that I had sole charge of the case throughout;

and had no assistance whatever
 in its management from any one.
 I had Dr. Bryan's full permission
 to make any use I liked of my
 notes on the case.

After having collected my notes
 on the disease from various sources,
 I received the second volume of
 the Edinburgh Hospital Reports
 which contains an interesting
 article on the subject by Dr. Jamieson.

I find that Dr. Jamieson makes
 special reference to the anatomy
 of the vesicle and to the action
 of arsenic on the bullous element
 of the eruption.

With regard to the former I was
 unable to find any satisfactory
 account of the anatomy of the
 eruption till I read his paper.
 I have therefore made use of it.

As regards the latter it was my
 original intention to enquire fully
 into the action of arsenic on bullous
 eruptions but I was prevented
 by stress of work from completing
 what notes I have on that point.

I trust however that I may have
 succeeded in adding some new fact to the

Literature of this Disease.

The photographs I send of the case, were taken by Mr. King of Littlehampton under very unfavourable circumstances as regards light and immediate surroundings, as the boy was unable to leave the room.

Still I hope they will form a very instructive picture and give a general idea of the character and extent of the eruption as I saw it.

My patient's name is Charles Tate aged 8 years - a scholar at the Board School.

Surroundings :-

His home was a very poor one. He slept in a badly lighted draughty room which had a damp musty odour.

The habits of his friends were not cleanly.

Food :-

Consisted mainly of vegetables, soups &c, with a little milk pudding.

Family History:—

Father and Mother are alive and healthy. His Sister and younger brother are quite healthy. There is no history of any skin eruption in the family, nor is there any specific taint.

Previous Illness:—

He had the usual diseases of childhood but nothing worthy of note occurred in any of them.

He had never suffered previous to this illness, from any skin eruption; but his predisposition to skin disease was shown by the fact that this disease followed directly on an injury to his right elbow.

Present Illness:—

His present illness began six weeks before I was called in.

As the mother was not very observant I have had extreme difficulty in getting an accurate account of the case before it came under my care.

According to her account, the boy fell one day whilst playing with other boys and bruised his Right Elbow.

A few days afterwards, isolated blebs appeared on the injured part, which rapidly ran together.

Similar blebs then appeared on the Left Elbow.

These continued to develop on the arms for some time, then they appeared on the inner surfaces of the thighs, on the buttocks and ultimately on the ankles.

The blebs had no tendency to rupture spontaneously.

They contained at first a clear straw-coloured fluid which in some became milky, in others pustular.

As the old blebs subsided new ones appeared.

He scratched them causing rather painful excoriations of the skin.

During that time red spots also appeared on the skin.

On some of these a papule would form which would develop into a vesicle or bulla.

This process gradually extended on to the trunk and for the first time erythematous patches were noticed of various sizes and shapes some of which had a vesicular margin.

Some of these patches would enlarge and run together.

This then is the course of the disease as I got it from his mother and the process was still going on when I was called in on the 26th of June /93.

She told me that during the evolution of the blebs there was intense itching, with occasionally a marked rise of temperature.

The boy was very squamous, and had occasional attacks of diarrhoea which however were not so troublesome as to require any medical treatment.

At intervals of a few days the boy would have an evening rise of temperature.

Between the attacks of diarrhoea the bowels were rather confined.

The eruption had never appeared on the palms or soles;

and the face had remained almost free.

She had never noticed any sores about his mouth.

During the whole of this time the boy's health had remained good and he had taken to bed a few days before I saw him because of the discomfort of getting about. The mother had done nothing for it - except to regulate the bowels and give him plenty of vegetables and soups.

Being very poor she could not afford to buy sufficient milk for him or other necessary articles of diet.

State on Examination:-

The boy is anæmic and rather emaciated, having a careworn expression. He has lost flesh since the illness began and has had very restless nights.

His tongue is slightly furred and moist. He complains of slight headache and intolerable itching especially in the perineum.

He has no appetite.

His respiratory system is normal. His pulse is fairly strong, full and regular, 110.

There are no cardiac murmurs. The urine is somewhat diminished in quantity, but is otherwise healthy.

His temperature is 99°.

Integumentary System:-

The eruption is seen typically on the back. I have therefore described it first in order to save repetition.

When I saw him for the first time I found macules, erythematous patches, and papules arranged somewhat symmetrically.

The patches varied in size and shape, some being round others having a sinuous outline.

The patches were bright red in colour, and more or less infiltrated, the margins being raised like the wheals of urticaria.

Three days later, I found that fresh macules and erythematous patches had appeared of various sizes - the largest being about the size of the palm of the hand.

Many of the previous papules now bore vesicles.

One patch bore a large bulla on its centre.

The margins of some of the older patches had now become distinctly beaded, of others vesicular.

Two days after this I found that fresh patches had appeared on the upper third of each scapula.

Between the angular scapularum I found two central patches one of which was about $2\frac{1}{2}$ inches broad.

Over the lower dorsal region is another central patch 2 inches by 3 inches. There are also fresh macules and tubercles.

During the evolution of the fresh macules and patches, in the short space of five days marked changes had been going on in the previous ones.

Macules had now developed into papules, papules into vesicles and vesicles into bullae.

The raised margins of the patches had become vesicular and the vesicles running together had in some parts formed large irregularly shaped bullae.

Some of the erythematous patches had extended at the margin while still maintaining their infiltrated and beaded character so that a curious concentric arrangement was produced, larger circles containing smaller ones.

Other patches meeting, had coalesced, and at their junction bullae were produced by the running together of the vesicles which gradually subsided as the free part spread as rapidly as before.

In this manner was the sinuous outline of some patches produced.

One bulla, an inch in diameter, stood alone on an erythematous base with a raised margin. Its contents were quite clear.

Bullae which previously contained clear straw-colored fluid, now contained milky or pustular fluid. Other bullae had entirely disappeared.

In the centre of the larger patches were dry scales due partly to collapse of the bullae and partly to scratching. These scales varied in colour from yellow to bluish-black according as blood had or had not been effused. There was no bad odour perceptible.

As regards the rest of the body I shall briefly describe the extent of the disease noting anything of special interest.

Behind the **EARS** were patches of eczema probably caused by scratching.

On the back of the **NECK** were a few macules and papules but no vesicles.

There were one or two vesicles on the **skin** surrounded by healthy skin. Otherwise the face was free.

On the **ARMS** the process was subsiding. The left was more affected than the right.

On the right elbow were seen a few isolated blabs containing a milky fluid and having areolae.

On the posterior surface of the right arm were a few dry scaly patches, but there was no ulceration where the scabs had fallen off.

The forearm was now almost free. There was a bulla on the back of the hand.

On the outer surface of the left arm was seen a large somewhat infiltrated erythematous patch, covered in the centre by dry scales and a few black scabs produced by scratching, and bearing on its margin numerous vesicles some of which were running together.

A smaller patch on the elbow showed the same appearance. On the flexor surface of the forearm

are two erythematous patches with distinctly bullous margins.

On the back of the hand is a papule. The extensor surfaces of both upper extremities were more affected than the flexor and both palms remained free.

Both shoulders were similarly affected, especially the left.

On the chest and abdomen were seen a few isolated bullae varying in size from a split-pea to a bean; some with clear fluid, others with more or less milky fluid. The former were surrounded by healthy skin, the latter seemed to develop an areole.

Within a week of my first visit I found erythematous patches developing over both infraclavicular regions and over corresponding parts of the abdomen, with margins showing the gradual transition from mere infiltration to bullae.

On the buttocks were two well marked irregular patches

with bullous margins evidently produced by the coalescence of two or more patches.

These showed dry scales and scabs varying in colour from yellow to brown or black.

Although he had caused extensive excoriations there was no ulceration under the scabs.

The scrotum and perineum were in a moist eczematous condition & excoriated.

On the front of the right thigh was a large patch extending round to the ham — 4 inches broad — with a bullous margin which is well brought out in the photograph.

Outside this one is another $2\frac{1}{2}$ in. in diameter.

The smaller ones are seen.

The margins of the larger ones

contained pustular fluids.

There is a patch of Leucoderma on the ham.

On the front of the left thigh was seen a similar patch to that on the right, being about $3\frac{3}{4}$ inches in length and bearing a pustular bullous margin.

Erythematous patches were also seen with extending infiltrated margins. The largest extended round the outer aspect of the thigh to the ham where it was 4 in. long. This patch was covered with scales of a yellow to a bluish black colour. The skin was indurated whilst the contents of the marginal bullae were partly clear and partly pustular.

The extent to which the legs were affected is well shown in the photographs. Note that the process extends round both legs. When I first saw him the lower third only of the left was affected, but within 6 days it had extended up to the junction of the upper $\frac{1}{3}$ rd with the lower $\frac{2}{3}$ ^{rds} in front and the popliteal space behind.

The right was less affected and the process did not extend much beyond the lower third.

All of these patches were covered with thick adherent scales and dry scales. At some parts there was oozing of serum from scratching

DIAGNOSIS

In arriving at a diagnosis of this case one must consider:-

1. The History and Course of the Disease.

There was no syphilitic history obtainable nor could any cause be assigned by his mother.

Curiously enough it followed upon the receipt of an injury to the right elbow, spreading thence over the greater part of the body but showing a partiality for the Extremities.

It was most marked however over the elbows, Shoulders and Ankles.

The patient had the usual febrile disturbances at the commencement of the illness.

2. Type of the Eruption.

The type was multiform.

Macules, erythematous patches, papules, wheals, vesicles, bullae & pustules were all seen variously combined.

Thus, there were erythematous patches with infiltrated urticarial margins of various sizes and shapes.

The patches tended to spread peripherally.

Papules, Vesicopapules and Vesicopustules were all seen in the various stages of evolution.

The tendency of the eruption was to pass from one variety to another e.g. a macule developed into a papule, a papule into a vesicle, a vesicle into a bulla or pustule and so on.

The grouping of the elements was herpetiform without any definite relation to the course of nerves.

One noted too, that the development was irregular, thus, some bullae arose on healthy skin, others were secondary to a macule or papule.

The pustules were never primary.

3 Subjective Symptoms

Itching was most intense before and during the evolution of the vesicles.

The patient also complained of burning and pricking sensations.

4. Chronicity of the Disease

One part of the eruption would subside or fade away whilst another was commencing.

Excoriations and pigmented patches were present to a slight extent.

5. Sex.

My case was in a boy.

6. Relapses

In the Spring of 1854 he came to me and had a few bottles of medicine but there was nothing special to note about it.

After I left Littlehampton he had a serious relapse which is referred to in the after history of the case.

7. General Health.

All through his illness his strength was well maintained and his general health did not suffer to any appreciable extent.

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What then could this peculiar eruption be?

Is it a case of

- a. Dermatitis Herpetiformis?
- or b. Erythema Multiforme?
- or c. Pemphigus?
- or d. Bullous Syphiloderm?
- or e. Herpes?
- or f. Impetigo contagiosum?
- or g. Bullous urticaria?

The symptoms and course of the disease all correspond with those met with in Dermatitis Herpetiformis.

But as the other diseases enumerated above bear certain resemblances to it, it will be necessary to exclude them before arriving at a definite conclusion as to the nature of the case.

b. Is it Erythema Multiforme?

As the name implies it is also multiform in character and relapsing.

(2) But it has a greater tendency to appear in Spring & Autumn than Dermatitis Herpetiformis has (McCull Anderson)

- (6) As a rule the duration is less but it may be prolonged by successive crops (Anderson).
- (7) There is not the same Herpetie grouping of the lesions as in Dermatitis Herpetiformis, nor do the patches have the same tendency to form vesicles bullae or pustules as was the case with my patient (Harrison)
- (8) Vesicles and bullae are usually secondary in Erythema Multiforme. In my case some were primary.
- (9) Distribution of Erythema Multiforme is chiefly on the back of the hand and forearm, dorsum of the feet and front of the legs (Anderson)
The eruption in my case at first showed a partiality for the extensor surfaces but afterward it was general over the greater part of the body.
- (10) The itching in Eryth. Multiforme is slight and may be absent altogether. In my case it was intense.
- (11) In Erythema Multiforme the Constitutional disturbance is slight.

c. Is it Pemphigus?

- 2) In pemphigus there is no herpetic grouping as in Dermatitis Herpetiformis. Bullae as a rule are isolated. In my case some bullae were surrounded by a circle of vesicles.
- (3) We have not the multiform character of lesion that was seen in my case. Moreover in Pemphigus the bullae are large and the erythematous patches absent.
- (4) Itching and burning sensations are absent in Pemphigus as a rule.
- 5) There was an inflammatory look in my case that is not seen in Pemphigus.
- 6) The general health in Pemphigus is very bad. It may in some cases appear good. The constitutional disturbance is most marked in children.

d. Is it a Bullous Syphilitide?

There is no history or evidence of Syphilis in the boy. The bulla in Syphilis is purulent and when it dries up a thick

crust- is formed which when removed reveals an ulcer.

No such ulcer seen in T. &.
Further the pustules in Syphilis are not grouped with any regularity.

e. Is it Herpes?

Herpes agrees with Dermatitis Herpetiformis in the grouping and in the angular outline of some of the blebs but Herpes follows the course of a cutaneous nerve being usually unilateral and is less extensive and altogether it runs a definite course.

f. Is it Impetigo Contagiosa?

Impetigo is always due to the inoculation of contagious pus whatever its source may be. The pustules are isolated.

Though my patient slept with his brother the disease was not communicated to him.

This fact excludes Scabies

g. Lastly was it a bullous form of Urticaria? In it there is no symmetry, no grouping & no circinate arrangement as in Dermatitis Herpetiformis.

Etiology.

The cause of this affection is very obscure.

Bazin and others believe it is a rheumatic affection, but this view is not generally held as there is not sufficient evidence to support it.

Most authorities hold that in the case of Erythema Nodosum there is a rheumatic element present. I could get no history of rheumatism in my case.

Now can it be said to be due to any digestive disturbance. It is true that in urticaria which is nearly allied to it (i.e. Derm. Hæm. psor.) we have an eruption which in some cases bears a direct relation to the ingestion of certain articles of diet.

But the result is due rather to a reflex nervous irritation of the skin, through the nerves of taste or the splanchnic nerves which supply the digestive tract.

In my case though no such cause could be assumed, the food was of such poor quality that the boy was undoubtedly rendered less liable to resist any illness.

Exposure to Cold is believed by some to be an exciting cause of Dermatitis Herpetiformis.

Elliot in the Brit. Journal of Dermatology 1891 ascribes two cases of Dermatitis Herpetiformis to shock and most people believe that nervous exhaustion predisposes to an attack.

Duhring has also recorded a case due to shock.

In women pregnancy is a frequent cause of Dermatitis Herpetiformis. It occurs usually in the later months.

Many have it in some pregnancies only, others have it in all.

It may not appear till after parturition, and more especially if there has been any undue hæmorrhage.

D. Jamieson looks for a probable cause in the lowering of the blood pressure which is met with chiefly in anæmic subjects; and which would in those of a neurotic disposition give rise to lesions similar to those met with in Dermatitis Herpetiformis.

In support of his view he alludes to the Iodide rashes which bear a somewhat close resemblance to the multiform eruption of D. Herpetiformis.

He with other observers has noticed that Iodides aggravate the eruption of D. Herpetiformis.

In my case the only cause that could be discovered was a direct injury to his right elbow.

Nowhere have I seen mention of any case following an injury.

But here we have the history of a fall whereby his right elbow sustained an injury at the seat of which this eruption first manifested itself.

Pemphigus as is well known often succeeds an injury.

But in my case though the eruption was said to be bullous at first, I am inclined to think that the boy's mother was not sufficiently observant to notice the macules and papules that preceded or accompanied the vesicles.

From the first there was in-
-tense itching and the course
of the disease was that of Dermat.
Herpetiformis and not of Pemphigus.

I see no reason for believing
that Pemphigus can assume
the characters of or be converted
into Dermatitis Herpetiformis.

Still the fact that this case
followed an injury is interesting.

Moreover there does not seem
to have been any nervous
shock accompanying the injury
(as it did not prevent his
resuming play with his com-
-panions).

Two great theories as to
the causation of this disease
are :-

- I That it is an Angioneurosis.
- II That it is caused by the
non elimination of Leucocytes
from the system.

T. What is meant by an Angioneurosis?

Angioneurosis (angion blood, neuron nerve,
& osis disease) is a neurosis (generic name for
disease of the nerves) in connection with the blood vessels
of a part.

The Theory of Angioneurosis (according to Kaposi (New Syd. Soc. Publications) is as follows:—

1. There is first of all contraction of the fine bloodvessels in the capillary layer of the skin.

This he attributes to stimulation of the vasoconstrictor nerves.

Umana however says (New Syd. Soc. Publication) this stage can't be looked upon with certainty (as Angioneurotic) but the appearance only suggests it.

2. This stage is followed by dilatation of the vessels and temporary paralysis causing slowing and stasis of the blood-current.

This is evidenced by the hyperaemia visible in the skin.

He attributes this to stimulation of the vaso-dilators or paralysis of the vasoconstrictors.

The simplest form is seen in the macule. In other cases we have effusion of serum into the cutis causing the erythematous patch to be slightly raised above the skin as in Erythema nodosum.

or Erythema papulatum.

A further stage is seen where the serum forces its way under the epidermis raising it up in the form of a vesicle or bulla according to the amount of serum effused as in Dermat. Herpetiformis and bullous forms of Erythema.

Uwua says

"In all cases the chief characteristic of an Angioneurosis consists in the fact, that the enlargement of the capillaries is not normally compensated for by an adequate fluctuation in the calibre of the veins, and by an increased rate of flow, but that on the contrary, a stoppage takes place."

How then is this result brought about?

We must assume that some stimulus has been applied to the nerve fibres in their course from the brain centres to their terminations in the skin.

1. The blood might contain some abnormal constituent

to the vasomotor centres in the medulla, and so cause direct stimulation of the vasomotor tracts.
 or 2. It is possible that these centres might be indirectly affected by stimulation of the peripheral endings of these nerves.
 or 3. The higher brain centres which control the bloodvessels may be stimulated directly or indirectly.

Thus Herpes Zoster has been observed to follow emotional disturbances.
 or 4. Stimulation to the periphery may cause it as seen in wheals caused by stinging nettles &c.
 (Hamilton - Text Book of Pathology).

With regard to the stimulus in angioneuroses Ueber suggests that there is a special irritability of the musculature of the veins which causes them to respond in a prejudicial manner to the increased blood pressure. To the arteries he would attribute the same properties of responding to variations in the blood pressure, but the pressure being greater in them, and their being in a state of paresis,

They do not contract spasmodically as would the veins. Thus he would infer that the blood pressure in these areas was sufficient stimulus to account for the Angioneurosis.

Localisation

in the skin is accounted for by Auspitz by a. Nerve Disturbances.
b. Local Injury.

He says that the altered tone of the vessels does not depend upon the local inflammatory processes but upon the injuries affecting the vascular nerve centres.

This however would not account for the appearance in Dermatitis Herpetiformis.

These he would account for merely by stimulation or paresis of the Vasodilator or Vasoconstrictor fibres.

Something else is required and we may not unreasonably suppose that there is some local irritant at the points of origin of the various macules and papules & patches whatever that irritant may be and that the Angioneurosis is the result of a reflex action.

In my case the lesion began on one elbow, then it appeared on the other. Was then a stimulus conveyed along the afferent fibres to the cells in the spinal cord belonging to the part-injured and being sufficiently strong to overcome the resistance of the efferent fibres which form communications with the cells which belong to the corresponding part of the other side of the body thus cause reflexly the lesion on the other elbow?

I myself being subject to eczema have frequently noticed that if I scratch one limb I automatically begin to scratch the corresponding area on the other limb; and I have in this way lighted up a fresh patch of eczema where it did not previously exist.

In neurotic subjects the resistance to impulses would be diminished.

This reflex theory might in some cases at least account for the symmetry observed.

The fact that Pregnancy and certain uterine diseases are known to cause Dermatitis Herpetiformis would support this reflex theory.

I incline to the belief that in Dermatitis Herpetiformis the symmetry observed points to a central origin.

Stephen M. Kenzie (Brit. Jour. of Dermatology) thinks that nerve lesions determine the locality rather than the form of eruption. The latter would depend on the idiosyncrasy of the patient. Thus we might have different eruptions resulting from one nerve lesion, or one form of eruption resulting from multiple nerve lesions.

What properties then have Angioneuroses in common?

1. There must be some stimulus to act upon the nerve centres of the vessels.
2. Their evolution and development is usually a rapid one while they run a typical course.

3. The distribution of the Angioneurosis is Symmetrical
4. There is practically one type of eruption Common to all, to which most of the Varieties can be referred.

As regards bullous eruptions it has not been shown whether the action of the vaso motor nerves is Primary or Secondary.

What facts then would justify our considering Dermatitis Herpetiformis an Angioneurosis?

1. It occurs chiefly in those of a neurotic disposition
2. The eruption is usually Symmetrical.
3. There is an urticarial element in it and as is well known urticaria is often produced by abnormal nerve influences.
4. The paræsthesiæ accompanying it viz itching, pricking &c would favour the view.
5. The result of treatment by nerve tonics is satisfactory.

These reasons alone would not justify us in referring all the Symptoms to nerve influences.

II What is meant by the Leucomaine Theory?

"Leucomaines" is the term used by Gautier to denote those basic alkaloids which are formed by the decomposition of albuminous bodies in the living tissues and are therefore to be regarded as products of their normal metabolism (Hamilton).

It is supposed by upholders of this theory that Dermatitis Herpetiformis is caused by their non-elimination by the skin.

Gautier in 1881 succeeded in extracting from the muscular juices of large animals five new alkaloids.

He found that they exercised a powerful influence over the nerve centers.

These, he showed, were produced naturally in the tissue metabolism along with urea and Carbonic Acid.

If this be so how do we escape any deleterious effects they produce?

1. It has been shown that they are eliminated by the excretory organs viz bowel, kidney, skin &c.

If therefore the elimination be impeded in any way they accumulate in the blood and various tissues and so produce many of the manifestations of the various diseases that are attributed to them.

Thus if the skin failed to perform its functions normally we would have certain lesions produced which we could assign to a more or less definite cause and to which perhaps we could give a descriptive name.

2. It has also been shown that they can be rapidly destroyed by Oxygen.

If then the blood be deficient in Haemoglobin as in Anaemia or in cases where deficient aeration occurs in the respiratory organs, they accumulate in the blood and thus exercise a deleterious influence on the nervous centres.

In the case of Dermatitis Herpetiformis the poisonous matter would probably affect the nerve centres of the skin chiefly.

Brook in a paper which he read before the Clinical Soc^y of Manchester says that "Blood diseases as a cause of skin diseases arise from the action of poisons — autoinoculations as in gout, rheumatism &c or from drugs or foods."

Referring to the subject of Leucoderms, Dr. Jamieson in his 'Handbook' mentions an interesting case where the eruption bore a direct relation to the ingestion of meat.

Such then are the two theories, as to the causation of Dermatitis Herpetiformis.

Stephen M^c Kenzie regards Dermatitis Herpetiformis as

1. a functional neurosis, especially where the urticarial element predominates
- or, 2 a peripheral neuritis,

in Support of which view
he asserts :-

a) That it may arise from
Cold just as Multiple Neuritis
does.

b) That it usually occurs about
the period of life when
Multiple neuritis is most met
with

In some cases of
Pimphegus Déjerine and Kelown
have described a paramechymatous
neuritis of the nerves endings
beneath the bulla.

It is evident from the number
of causes given that authorities
are not yet agreed as to the
real causation of Dermatitis Herp-
etiformis. It seems to be
held that in the majority of
cases at least there is a nervous
element running through
it, but how and where the
exciting stimulus is applied
to the nerve structures, is
a matter of dispute.

As the disease becomes
more generally recognized

and more attention is directed to this subject, we may in time arrive at some feasible explanation of the phenomena observed.

Histology.

As I pointed out whilst discussing the phenomena peculiar to angioneuroses, there is a dilatation of the superficial bloodvessels with a slowing of the blood current, then a certain amount of exudation takes place which if abundant would give rise to vesicles or bullae by raising up the epidermis.

Vidal excised a wheal (urticaria) during life and found the superficial and deep vessels dilated and full of blood. The walls remaining healthy.

Both blood ^{vessels} and lymph-vessels were surrounded by leucocytes which were seen throughout the whole thickness of the cutis, forming at certain parts small clusters.

A few were to be seen between the deepest cells of the epidermis, the deepest layer of which was normal.

Another piece was excised from a wheal on which a vesicle had developed.

The fluid contained serum albumin, but the deeper layer of cells was more invaded by leucocytes than in the other case.

The vesicles in Heber's Zoster are formed in the same way as in eczema, the process proceeding from the papillary layer in which the vessels are dilated, the papillae are enlarged and topped with the corium & sometimes even the subcutaneous layer infiltrated with leucocytes.

(Crocker - Skin Diseases. Milton - Pathology).

The effused fluid forces its way between the rete cells elongating and compressing them as well as sweat ducts & hair follicles whereby a meshwork is produced which contains in its interspaces connective tissue cells which have come through the rete.

The seat of the vesicle in eczema is in the upper part of the rete or just beneath the

horny layer while the vesicles in Dermatitis Herpetiformis are situated between the mucous and horny layers of the cuticle (Jamison).

The anatomy of the vesicle in Dermatitis Herpetiformis has been carefully studied by Dr. Jamison (Edin. Hosp. Reports Vol II.) He found that the hairs passing through the vesicle were quite healthy as were also the sebaceous glands and follicles accompanying them. Near the vesicle the corium was somewhat oedematous, the bloodvessels being dilated and surrounded by leucocytes.

In its upper part the duct of the coil gland was surrounded by cells, the lining cells being broken up; otherwise the duct and coil itself were healthy.

The vesicles were seated beneath the stratum granulosum, the demarcation between the corium and rete being indistinct as cell processes extended from the rete into the corium.

He found that "the vesicles

were chambered, the separation being made by columns of imperfect rete cells. The Loculi were rounded and partitioned irregularly by fine structureless trabeculae, the contents being blood and lymphoid cells "

Pigmentation is due to the escape of red corpuscles by diapedesis and the staining of the tissues by the products of their pigment. Where blood is effused we find staining of the tissues (around for the same reason. This staining may persist for years. (Coats - Pathology).

Varieties.

The varieties of Dermat. Herpetiformis
 (as enumerated by Dühring

are:—

Dermatitis Herpetiformis	Erythematosa.
"	" Papulosa.
"	" Vesiculosa.
"	" Bullosa.
"	" Pustulosa.
"	" Multiformis.

D. H. Papulosa is rarest.

The names are sufficiently
 descriptive not to require more
 than mention.

In Dermat. Herpet. Multiformis
 some or all of the preceding
 varieties are represented.

Prognosis.

Under proper treatment a case
 should get well in a few months
 but it recurs, the succeeding
 attacks getting weaker till they
 cease altogether.

In my case I learnt that
 the second attack was not severer
 than the first.

Other cases last for years
 with intermissions.

Duhring records a case that lasted over 12 years.

The prognosis though good should be guarded as some fatal cases have been recorded.

Thus Stephen McKenzie records a case which lasted without intermission for $5\frac{1}{2}$ years then proved fatal.

Brocq has also recorded a fatal case.

In one case (mentioned by Pringle in a paper read before the Harvian Society - Nov 3/92) perforation of the ileum was discovered with ulceration of ileum and caecum, which would suggest an internal manifestation of the disease.

Treatment.

Internal.

Malcolm Morris in his Presidential address to the Harvian Society says:— The knowledge of the intimate pathological connection between the nervous system and the skin gives the key to the successful

treatment of many cases of Dermat. Herpetiformis, Eczema &c which defy all local measures."

Amongst the nerve tonics that are usually recommended are Phosphorus, Quinine, Nux Vomica and above all Arsenic.

Tinct Belladonnae has been tried and seems to act well when Arsenic fails

one may give 15 to 30 grains three times a day.

Ichthyol has been used with varying success.

Thiol has been used with doubtful success.

Arsenic ought to be given in full and increasing doses.

Jonathan Hutchinson (in the B. M. J. June 1891) says Dermatitis Herpetiformis yields at once to it. He finds that in Pemphigus no fresh bullae appear as a rule after Arsenic is given

I observed that after my patient had been taking Arsenic for 4 or 5 days there were no fresh bullae developed.

Hutchinson says Arsenic has an unfavourable influence on elderly people, especially when the mucous membrane of the mouth

is implicated or where symptoms of nerve degeneration are present.

I found that as I increased the dose in Tats he became less able to tolerate the drug.

I therefore had to control the diarrhoea it occasioned by suitable measures.

I am interested to find that Dr. Jamieson (Edin. Hosp. Rep. Vol I) had to combine Arsenic with astringents in one of his Cases. Page 173

For the insomnia and restlessness we may have to employ Sedatives such as Bromide, Chloral, Opium, Camphor Indica &c. The Tr. Camph.

Indica may be given in doses of 10 to 15 grains three times a day.

Antipyrim is useful in doses of 20 to 30 grs when the tubercular element predominates.

As already mentioned, Iodide of Potash aggravates the eruption.

Alcohol should be restricted. To improve the blood, some of the preparations of Iron may be given.

If Gout is present we may give
Colchicum.

In such a case Sulphide
of Potass baths, $\mathfrak{z}\text{ij}$ to the bath,
would do good.

External.

Various soothing remedies
afford temporary relief viz
dusting powders, lotions & ointments
Amongst the lotions, one may
try Calamine Lotion, Ichthol
Lotion $\text{℥}\text{ii}$ $\mathfrak{4}$ (Jamieson) Boric Starch
Dressing, Carbolic Acid Lotion
especially where itching is intense,
Lead Lotion etc.

Stephens McKenzie has found
Glycerine of Lead very useful.

Soothing ointments are also
serviceable such as Subnitrate
of Bismuth $\mathfrak{z}\text{i}$ to $\mathfrak{z}\text{ij}$,
Ichthol 20% with or without
the addition of Calamine or Oxide
of Zinc. Ichthol Varnish
40% might be used in
cases where the vesicles are
unruptured.

During finds the vesicular form most amenable to treatment.

He recommends Tar preparations, Thymol, Ichthol, Carbolic Acid, Hydronaphthol, Resorcin, Liquid Extract of *Grindelia Robusta*.

In the erythematous varieties he would use *Liq. Carbonis Ditesis* of the strength $3i$ to $3iv$

In the vesicular and pustular varieties he has derived most success from a Sulphur Ointment $3ii$ to $3i$.

The rubbing must be long and thorough and sufficiently forcible to break down the pustules etc.

D. Jamison has also found Sulphur of great service especially in Children.

The same authority recommends the addition of starch to the bath of Potassa Sulphurata, after which he would apply Calamine Lotion.

When I first saw my patient I gave him the usual nerve tonics such as Quini, Phosphorus, Iron, Cod Liver Oil, Nux Vomica etc

Externally I applied Calamine's Lotion.

The Tonics improved his general health but did not restrain the evolution of the bullae, nor did the Calamine Lotion have the desired effect. I therefore substituted Lead Lotion with certain precautions as to its use. This succeeded better than the Calamine Lotion which caused great smarting owing perhaps to the excoriated state of the skin.

I then tried Carbolic Acid which gave marked relief. At the same time I gave Arsenic and Bicarbonate of Potass - beginning with 2 minims of Fowler's Solution and gradually increasing it up to 5 minims. I was unable to push it farther on account of the gastric disturbance it produced and I found that the limit was about 3 minims three times a day.

After 4 or 5 days of this treatment no fresh bullae arose and the boy made a speedy recovery and was able to go for short walks in less than 6 weeks from my first visit.

Such marked improvement following the administration of arsenic naturally led me to ask why bullous eruptions were thus affected by Arsenic.

But as I explained I have been unable to enquire fully into this interesting subject.

Lauder Brunton in his Book on Therapeutics says "Arsenic absorbed into the blood appears to modify tissue change. A solution of Arsenious Acid added to blood retards Coagulation.

Minute doses accelerate the pulse
Larger ones diminish the pulse and
blood pressure.

Ruizer and Murrell noticed that in frogs poisoned by Arsenic the cuticle could be stripped off the whole body a few hours after the drug had been taken.

This was found by Munn to depend upon softening of the protoplasm of the columnar layer of cells in the epidermis.

Other epithelial structures are affected as well and Coriul has found fatty degeneration.

of the epithelium lining the
alveoli of the lungs in animals
poisoned by Arsenic (Ruijs- Therapeutics)

Mitchell Bruce says that
Arsenic seems to increase in a
salutary manner the metabolism
or vital activity of all the organs.

As to the effect on the nerves,
Lauder Brunton says that "in frogs
it produces apparent paralysis
due to really diminished
Sensibility of the Posterior Cornua of
Grey Matter i.e. of the pain conveying fibres"

Mitchell Bruce says it diminishes
the sensibility and reflex irritability
of the nerve centres (Mat. Med. & Therapeutics)

After-History.

I had left Little Hampton before
my patient had a serious relapse
but when he was convalescent
I had an opportunity of seeing
him again. He was feverish at
the commencement of the illness but
had no diarrhoea.

The itching was intense, and he
tells me that since the first attack

He has never been free from pruritus.

This is partly explained by the presence of eczema in the Perineum. He had no sores in the mouth or pharynx. The same localities were affected as before and this attack ran a similar course to the previous one - being however more severe.

This I cannot account for. I know however that it was treated as a case of Pemphigus by my successor.

I found a few patches of leucoderma where the process had been severe, especially on the buttocks, shoulders, and legs. The right leg has a large patch of leucoderma with a faintly pigmented margin.

One distinctly pigmented patch is seen on the chest. Others are of a dusky red colour merging into a purplish hue. Other patches are seen on the abdomen in the middle line.

His back is now almost clear of any eruption, but both scapular regions are covered by large dark red patches. Other smaller patches dusky red in colour are seen, but no bullae no scaling are visible.

In this illness the eruption first appeared in the Right Popliteal space and spread thence upwards on to the front of the thigh and abdomen. It then extended on to the chest and back and arms.

There was no determining cause. He complains of nothing but itching.

Tongue is clean. Pulse is good. He is practically quite well again and running about.

Amongst the works which I have consulted are McAll Anderson's "Diseases of the Skin"; Mitchell Dozier's "Materia Medica & Therapeutics"; Land & Bonbrun's "Therapeutics"; Coats' Manual of Pathology; Radcliffe Crocker's "Diseases of the Skin"; Hamilton's "Text Book of Pathology"; Jamieson's "Diseases of the Skin"; Living's "Handbook of Diseases of the Skin"; Rejzer's "Handbook of Therapeutics" &c. Amongst the publications I may mention Selected Monographs on Dermatology - New York Loan Society Publication - in which I consulted papers by Kaposi, Anna, Duhring &c.; Reference Handbook of the Medical Sciences; Edin. Hospital Reports Vol I & II; British Journal of Dermatology in which I consulted papers by Stephen Mackenzie, Jonathan Hutchinson, Duhring &c.; Journal of Cutaneous & Genitourinary Diseases; Brit Med Journal and Lancet and others.