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 Virchow who brought it prominently before the profession in 1864.

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

Being fortunate to meet with a case while assisting Dr. Bryan of Littlehampton, 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 vesicles 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 regard 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 the Disease.

The photographs I took of the case, were taken by Mr. Reig of Littlehampton under very unfavourable circumstances as regards light and minute surrounding, 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 [illegible] aged 8 years — a scholar at the Board School.

Surroundings: —
His home was a very poor one. His 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, soup, tea &c., with a little milk pudding.
Family History:
Father and mother are alive and healthy. His sister and younger brother are quite well. There is no history of any skin eruption in the family, nor is there any specific taint.

Previous Illnesses:
He had the usual diseases of childhood, but nothing worthy of note occurred to any of them. He had never suffered previous to this illness from any skin eruption, but his predisposition to skin diseases 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 surface 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 other pustules. 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 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.

Thus then is the course of the disease as I see it from his brother and the process was still going on when I was called in on the 26th of June 1923.

The tells me that during the evolution of the blebs there was intense itching, with occasionally a marked rise of temperature. The boy was very irritable and had occasional attacks of diarrhoea which however were not of 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 sore 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 soupes. Being very poor she could not afford to buy sufficient milk for him or the necessary articles of diet.
State on Examination:
The boy is anaemic and rather emaciated, having a carious 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, if others vesicular.

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

Between the angularis scapularum I found two central patches, one of which was about 2½ inches broad.

Over the lower dorsal region is another central patch 2 inches by 3 inches. There are also fresh macules and papules.
During the evolution of the first 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, papule 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 infiltrate.

And beard character so that a curious concentric arrangement was produced, larger circles containing smaller ones.

The 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 circular 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 purulent 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 color, 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 chin 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 violaceous blisters containing a sticky fluid and having cariolas.

On the posterior surface of the right arm were a few dry, scaly patches, but there was no ulceration where the scales 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 spots 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-pla 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 areola.

Within a week of my first visit I found erythematous patches developing over both infracostal 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 triangular patches
with bullous margins evidently
produced by the coalescence of
two or more patches.
These showed dry scales and
scales varying in colour from
yellow to brown or black.
Although he had caused ex-
tensive excoriations there was
no ulceration under the scales.
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
was well brought out in the photograph.
Outside this one is another
2½ inch in diameter.
The smaller ones are seen.
The margin of the larger one
contained serous fluids.
There is a patch of leukoderma
on the ham.
On the front of the left thigh
was seen a similar patch to
that on the right, being about 3½ inches
in length and bearing a frustular bullous
margin.
Erythematous patches were also seen with extending infiltrated margins. The largest extended round the outer aspect of the thigh to the knee where it was 4½ 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 punctate.

The extent to which the legs were affected is well shown in the photographs. Note that the process extended round both legs. When I first saw him the lower third only of the left leg was affected, but within 6 days it had extended up to the junction of the upper ⅓ with the lower ⅔ 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 point there was oozy exudate 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, and pustules were all seen variously combined.

Thus, there were erythematous patches with infiltrated urticarial margins of various sizes and shapes.
The pustules tended to spread peripherally.
Papules, vesiculous papules and vesiculous pustules 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 haphazard 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 prickling sensations.

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. 

My case was in a boy.

6. Relapse

In the spring of 1854 he came to me and had a few bottles of medicines 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.


All throughout his illness his strength was well maintained and his General Health did not suffer to any appreciable extent.
What then could this peculiar eruption be
Do it a case of
a. Dermatitis Herpetiformis?
or b. Erythema Multiforme?
or c. Pemphigus?
or d. Bullous Syphiliticum?
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.

7. Is it Erythema Multiforme?
As the name implies, it is also Multiform in Character and Relations.
(2) But it has a great tendency to appear in Spring & Autumn that Dermatitis Herpetiformis has (McCall Anderson)
(6) As a rule the duration is less but it may be prolonged by successive eruptions. (Anderson)

(7) There is not the same herpetic grouping of the lesions as in Dermatitis Herpetiformis, nor do the patches have the same tendancy to form vesicles, bullae or pustules, as was the case with my Patient (Anderson).

(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 external surfaces but afterward it was general over the greater part of the body.

(10) The itching in Erythema Multiforme is slight and may be absent altogether. In my case it was intense.

(11) In Erythema Multiforme the constitutional disturbances are slight.
c. Is it Pemphigus?

2) In pemphigus there is no herpetic pustule as in Dermatitis Herpetiformis.

Bulles as a rule are isolated. In my case some bulles were surrounded by a circle of vesicles.

3) No have not—the multiform character of lesion that was seen in my case. Moreover in Pemphigus the bulles are larger and the erythematous patches absent.

4) Itching and burning sensation 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 Tullous Syphilis?

There is no history or evidence of Syphilis in the boy.

The bullae in Syphilis is purulent and when it dries up a thick
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 no sufficient evidence to support it.

Most authorities hold that in the case of erythema nodosum there is a rheumatic element present. I could find no history of rheumatism in my case.

Nor can it be said to be due to any digestive disturbances. It is true that in urticaria which is nearly allied to it (as Dermat. Hospit.) 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 I eat of such poor quality that the boy was undoubtedly rendered less liable to succumb to any illness.
Exposure to cold is believed by some to be an exciting cause of Dermatitis Herpetiformis.

Elliot in the Brit. J. Dermatol. 1891 describes two cases of Dermatitis Herpetiformis in the skin associated with nervous excitement or predisposing to an attack. He also records 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 trouble with haemorrhages.

Dr. Jamieson looks for a probable cause in the lowering of the blood pressure which is met with chiefly in anaemic 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 pastes 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 ni-
tense itching and the course
of the disease was that—of Dermat.
Herpetiformis and not of Pemphigus.
I see no reason for drawing
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 this
Recurrent play with his com-
panions.

Two great theories as to the
Causes of this disease are:

1. That it is an Angioneurosis

2. That it is caused by the
Non-elimination of Endocontus
from the System.

1. What is meant by an Angioneurosis?

Angioneurosis (Angioneurotic, Angio\textsuperscript{\textregistered}Con-,
\textsuperscript{\textregistered}Con, 4th ed.) is a neurosis (generic name for
disease of the nerves) in connection with the Blood Vessels
\textsuperscript{\textregistered}a fact.
The Theory of Angioneurosis (according to Kaposi [newly pub'd. I. p. 7. Publication] is as follows:—

1. There is first of all contraction of the fine blood-vessels in the capillary layer of the skin. This he attributes to stimulation of the Vaso-constrictor nerves.

Unna Lowers (newly pub'd. I. p. 7. Pub.) states that this stage cannot 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 paralytic of the Vaso-constrictors.

The simplest form is seen in the macula. In the cases we have observed, a tumour rises 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 Desert Hypediciform and bullous forms of erythema.

Wenck says: "In all cases the chief characteristic of an angioneurosis consists in the fact that the engorgement 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 carry some abnormal constituent
to the vasomotor centres in the medulla, and to 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 blood vessels 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 which caused by Strangeways, M.D.

(Hamilton Text Book of Pathology).

With regard to the stimulus in Arteriomeob, Vater 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 paroxysm,
They do not contract deeper medially as would the veins.
Thus he would infer that the blood pressure in these areas was sufficient stimulus to account for the angioneurosis.

Localization in the skin to accounts for by A. Neurone Dystomnes & 2. Local Injury.
He says that the altered tone of the vessels also depends upon the local inflammatory processes but upon the injuries affecting the vascular neural center. This however would not account for the appearance. In Dermatitis herpetiformis these he would account for merely by stimulation or paralysis of the vasodilator or vaso-constrictor fibers.
Something else is required and we may not unreasonably suppose that there is some local irritant at the point of origin of the various macules and papules of patches whatever that irritant may be and that the angioneurosis is the result of a reflex action.

(GMSN 36. Soc. Puericult.)
In my case the lesion began on one elbow, then it appeared on the other. Was there 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 afferent 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 the reflex theory. I incline to the belief that in dermatitis herpetiformis the symmetry observed points to a central origin.

Stephen W. Kenzie (Brit. Jour. 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 angioneurosis in common?

1. There must be some stimuli to act upon the nerve endings 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 vasomotor nerves to 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 paraesthesias accompanying it—itching, pricking, and tend towards 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.
What is meant by the Liebmann theory?

"Liebmann" 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 1887 succeeded in extracting from the musculature of large animals five new alkaloids. He found that they exercised a powerful influence over the nerve centres. These, he thought, were produced naturally in the tissue's 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, etc. 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 toxic substances 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 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 then exercise a deleterious influence on the nerves.
In the case of Dermatitis Herpetiformis, the poisonous matter would probably affect the nerve centres of the skin chiefly.

Brock in a paper which he read before the Clinical Sect 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 food."

Referring to the subject of Luesmatics, Dr. Jaman in his textbook mentions an interesting case where the eruption bore a direct relation to the migration of meat.

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

Stephen M. Kinzie regards Dermatitis Herpetiformis as 1. a functional neurosis, especially where the utricular element predominates, or 2. a peripheral neurosis.
to support which view he asserts:

1) That it may arise from cold moist as multiple neuritis does.

2) That it usually occurs about the period of life when multiple neuritis is most met with.

In some cases of Pemphigus Foliaceus and Keloid have described a paraneuritis of the nerve endings beneath the bulbus.

It is evident from the number of causes given that authorities are not yet agreed as to the real Nature of the disease. 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 structure is a matter of dispute. As the disease becomes more generally recognized...
And most 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 observed a wheel (verticis) during life and found the superficial and deep vessels dilated and full of blood. The walls remaining healthy. Both blood and lymphatic vessels were surrounded by leukocytes which were seen throughout the whole thickness of the cutis forming at certain points 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 whorl in 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 herpes 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 to form with the corium a Sometimes even the substantaneous layer infiltrated with leucocytes.

(Crooke. Ch. Dis. Inl. Pathology). The effused fluid forces its way between the Hall cells elongating and compressing them as well as tending to form fistulas. Whereby a mesothrich meshwork is produced which contains in its interstices connective tissue cells which have come through the skin.

The seat of the vesicle in eczema is in the upper part of the skin or puck beneath the
 Horny layer while the vessels in 
 Dermatitis Herpetiformis are situated 
 between the mucous and horny 
 layers of the cuticle. (Jaminon).

The anatomy of the Vesicle in 
 Dermatitis Herpetiformis has 
 been carefully studied by Dr. 

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 edematous, 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; therefore the duct 
 and coil itself were healthy.

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

He found that: "The vesicles
were chambered, the separation being made by columns of imperfect plate cells. The loculi were rounded and partitioned irregularly by fine structureless trabeculae, the contents being blood and lymphoid cells.

Pigmentation—due to the escape of red corpuscles. By diaphragma and the staining of the tissues by the products of their degeneration. Where blood is effused in the fluid, staining of the tissues around for the latter reason. This staining may persist for years (Catts—Pathology).
Varieties.
The varieties of Dermat. Herpetiform.
(As enumerated by Duhring)

- Dermatitis Herpetiformis Erythematosa
- Papulosa
- Verrucosa
- Bulla
- Pustulosa
- Multiformis

D. H. Papulosa is variant.

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 until they cease altogether.

In my case I learnt that the second attack was not sever than the first.
Other cases last for years with intermittent.
During record 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 5½ years
then proved fatal.
Brock has also records a fatal case.
In one case (mentioned by Price in a paper read before the Hawaiian Society—nov 3/52) perforation of the ileum was discovered with ulceration of ileum and cecum, which would support an internal manifestation of the disease.

Treatment.

Internal.
Malcolm Morris in his presidential address to the Hawaiian 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 etc. which defy
all local measures."

Amongst the nervous tonics
that are usually recommended are
phosphorus, rhodium, new tonics, and
among all arsenic.

First belladonnae has been tried and
seems to act well. When arsenic fails
one may give 15 to 30 gr. thrice a day
with success. Hahnemann has been used with varying success.

Arsenic ought to be given
in full and increasing doses.

Jonathan Hutchinson, in the B. M. J. June
1871, 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
To complicated or where symptoms of nerve degeneration are present.

I found that as I increased the dose in rats he became less able to tolerate the drug. I therefore had to control the discovery of an occasion by suitable measures. I am interested to find that Dr. Jamieson (Edin. J. Physiol. 1971, Vol. I) had to combine strychnine with antispasmodics in one of his cases.

For the insomnia and restless ness we may have to employ sedatives such as bromide, chloral, quin cin, Cannabis indica, etc. Tincture of india may be given in doses of 10 to 15 min three times a day.

Antipyrin is useful in doses of 20 to 30 grs. when the intestinal element predominates.

As already mentioned, iodides of potas aggravate 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 3½ 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, Iodine Lotion, 1¼ ½ (Jamaica) Boric Acid Dressing, Carbolic Acid Lotion especially where itching is severe, Lead Lotion etc.

Mepha McKenzie has found Algernine of Lead very useful.

Soothing ointments are also derivable such as Subnitrate of Bismuth 3½ to 3½ Iodine 20% with or without the addition of Calamine or Detergent of Zinc. Iodine 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, ethylcarbolic acid,
hydrasaphthol, resorcin, lejuid extract of St. John's Root.
In the erythematous varieties, he would use Lq. Carbo-di-muri
di 31 to 31
In the vesicular and serpular variety, he has derived most
success from a sulphur ointment 31/ to 31/.
The rubbing must be long and thorough and sufficiently
forcible to break down the
pustules, etc.
Dr. Jamieson 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
lotions.

When I first saw my patient,
I gave him the usual nerve
tonics such as Quin, Phosphorus,
Iron, Cod Liver Oil, Nux Vomica, etc.
Externally I applied Calamine Lotion.

The sores 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 excretales state of the skin.

I then tried Carabolic Acid which gave marked relief. At the same time I gave Arsenic and Bichromate of Potassa beginning with 2 minims of Fowler's solution and gradually increasing it up to 5 minims. I was unable to push it further 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.

Landz Prenton in his book on Therapeutics says that arsenic absorbed into the blood appears to modify tissue changes. A solution of arsenious acid added to blood retards coagulation. Minute doses accelerate the pulse; large ones diminish the pulse and blood pressure.

Rüger and Murrell noticed that 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 Dunn to depend upon softening of the protoplasm of the cuticular layer of cells in the epidermis. Other epithelial structures were affected as well. And Coriul has found fatty degeneration...
Of the epithelium lining the alveoli of the lungs in animals.

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

As to the effect on the nerves, Lewis Brounston says that "in frogs it produces apparent paralysis due to really diminished sensibility of the posterior cornua of Gryp. Matta, 1 2 of the pain conveying fibers." Mitchell Pomer says it diminishes the sensibility and to some inactivity of the nerve centers.

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 told me that twice the first attack
He has never been free from Pustules. This is partly explained by the presence of Eczema on the Fingers. He had no sore in the Mouth or Pharynx. The same locality has affected as before and this attack ran a similar course to the previous one—being however more severe. This I cannot account for. I knew however that it was treated as a Case of Pustulosis by my Successor. I found a few patches of Leueoderma where the process had been severe, especially on the buttocks, shoulders, and legs. The right leg has a large patch of Leueoderma with a faintly pigmented margin. One distinct pigmented patch is seen on the chest. There are of a dusky red colour merging into a pimplish hue. The 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. Dusty red in colour are seen, but no Buboes or Scaling are visible.
In this illness the eruption first appeared on the right side of the face 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 complained of nothing but itching. Tongue is clean. Pulse is good.

As to practically, quite well again and running about.

Amont the works which I have consulted are: McAllister's "Diseases of the Skin"; Mitchell's "Materia Medica and Therapeutics"; Leveson's "Therapeutics"; Calkins' "Manual of Pathology"; Ratcliffe's "Cranke's Diseases of the Skin"; Hamilton's "Handbook of Pathology"; Jamsion's "Diseases of the Skin"; Leavitt's "A Handbook of Diseases of the Skin"; Major. "Handbook of Therapeutics".

Amongst the publications I may mention: Selected Monographs on Dermatology - New York Medical Society. Publication—n which I contributed papers by Verner, Dunn, Dutting, etc.; Reference. Handbook of the Medical Sciences; "New Hospital Reports" Vol. 7 & 8; British Journal of Dermatology in which I contributed papers by Stephen MacKenzie, Jonathan Ashlie, Dutting, etc.; Journal of Cutaneous & Venereal Diseases; Port Med. Journal and others.