

Nov 1913 (22

THE S I S

on

"Haemorrhagic and Gangrenous Varicella"

with notes of two cases.

by

HUGH COCHRANE STORRIE, M.B., Ch.B.

ProQuest Number: 27555606

All rights reserved

INFORMATION TO ALL USERS

The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



ProQuest 27555606

Published by ProQuest LLC (2019). Copyright of the Dissertation is held by the Author.

All rights reserved.

This work is protected against unauthorized copying under Title 17, United States Code
Microform Edition © ProQuest LLC.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

Introduction.

Until comparatively recent times Varicella was regarded as a trivial disease, but during the last few years several more or less serious conditions have been recognised to complicate even an ordinary attack. Many still believe it to be an exanthem in which no complications occur. Varicella, in view of the fact that fatal results have occurred from such complications as nephritis, septicaemia and pyaemia, severe inflammatory eye conditions and haemorrhagic and gangrenous forms, must be given more consideration specially when the disease occurs in districts where starvation, neglect and unhygienic conditions exist.

Also there are the unsettled questions as to what part tuberculosis plays in the occurrence of the gangrenous form and what part this form plays in setting up or lighting up afresh tubercular disease in badly nourished infants and children.

Having been fortunate enough to have two cases of this condition in one of my wards at the Grove Fever Hospital, London, I have selected this subject for my thesis. There are very few cases on record and the literature on the subject is scanty.

The occurrence of an outbreak of varicella of a somewhat severe type and a close study of these two serious cases have enabled me to collect sufficient clinical material to write this thesis which, I trust, will help to throw further light on the subject.

Reference to the literature leads me to believe that complications of varicella are few in number and somewhat rare in occurrence. Quite a number of cases with multiple gangrenous skin lesions are on record, but there are very few cases in the literature of the true haemorrhagic type and still less in which a single extensive gangrenous lesion was present.

Several cases have been described in which a haemorrhagic stage was present before the gangrenous stage was reached. At present there are only some ten or twelve cases of the true haemorrhagic type on record.

The cases under my care both exhibited one extensive gangrenous lesion;

the second one, in addition, presented a haemorrhagic condition before the gangrenous stage was reached. It would appear as though there is some close association between the two types, so much so, that it is probably not out of place to discuss the two types at the same time and in the same paper under one heading.

I have been able to gain access to a fair portion of the literature and propose to give a resume of it in the following pages and a detailed account of the two cases under my care. The rarity of the condition makes me give a much more detailed account of the cases than is probably usual.

Historical Outline.

We are indebted to Heberden for clearly pointing out that smallpox and chickenpox are separate diseases. Ingrassias, a Sicilian physician, in a work on "Preternatural Swellings", published in 1553, appears to have given the earliest clear description of chickenpox. Some forty years later Vidius described the condition as a variety of smallpox and like Ingrassias called it "Variola Crystallina". Towards the end of the seventeenth century several English writers made reference to a variety of smallpox which they called "Chickenpox". The name, "Varicella", was apparently first used by Vogel in 1764, and somewhat earlier Fuller asserted that smallpox and chickenpox were separate diseases, but it was not until 1767, when Heberden made out clearly, points of difference between the two diseases, that this separation was more or less universally accepted.

Several eminent physicians, including Thomson of Edinburgh and Hebra of Vienna, did not accept the distinction between the two states, and for some time to follow the various opinions and writings tended to confuse rather than to elucidate. The two diseases were universally accepted as being separate after the smallpox epidemic in Germany in 1870-73. The condition which seems to have caused most confusion was that in which a mild attack of smallpox was present.

Other names used were Spuria, Volatica, Crystallina and Variola, Crystallina, Nathae, Ligitime, Glasspox, Swinepox, Hives and Waterpox.

Varicella Gangrenosa was first introduced into the literature by Hutchinson in 1880. Although he had been aware of the condition for some years before that date, he had refrained from making observations until he was sure of his ground. Whitley Stokes in 1807 had apparently observed the condition and used the names "Pemphigus Gangrenosus", "White Blisters" and "Eating Hives", but he did not consider it to have any connection with varicella. Several years later Pagge used the name "Rupia Escharotica" for what appeared to be the same condition. Although Stokes, Pagge and several other writers had observed the condition, none of them had recognised its true significance - this was left to Hutchinson. The names of Barlow, Drewitt, Abercrombie, Haward, Bowley, Payne, Bellamy,

Crocker, Jamieson and Andrew may be associated with these others in the earlier recognition of the condition in literature.

Of late years more attention has been given to the complications, but the records did not seem to increase very appreciably.

Crocker introduced the name "Dermatitis Gangrenosa Infantum". This has, however, a wider range of cases and includes all those cases of gangrene of the skin occurring in infants and children, who are badly nourished, after varicella, vaccinia, measles and other non-specific skin eruptions of a pustular, papular or vesicular nature.

The first work on Haemorrhagic Varicella appears to be by Storer in 1828. Nothing more appeared for some fifty years and then cases were reported by Demme, Hamann, Andrew and others.

Resume of the Literature.

Hutchinson's name will always be associated with the condition Varicella Gangrenosa. In a communication on "Gangrenous Eruptions in connection with Vaccination and Chickenpox", he said that he had been aware of the condition for some ten years. This article appears to be the first on the subject and was written in 1879. A case of Vaccinia Gangrenosa had come under his notice earlier in 1879; almost immediately William Stokes published details of a somewhat similar case.

Hutchinson recognised that there was a distinct pathological similarity between vaccinia and varicella and, looking back on the cases which had come under his notice, he came to the conclusion that a gangrenous state occurred in varicella as well as in vaccinia. These cases of gangrenous vaccinia seemed to have made him come to this conclusion after having been cognisant of it for some time.

The first cases he saw presented deep, abruptly margined, punched-out ulcers extending right through the skin and chiefly situated at the flexures. He depended on the history of the cases and the presence of healing vesicles on other parts of the body for a diagnosis of varicella. He recognised that varicella gangrenosa was a rare condition, that it occurred usually in healthy children and frequently in one of four or five in a family. He attributed this to the exhibition of an idiosyncrasy. These cases usually exhibited much constitutional disturbance, but recovery was usual though death might occur in a few cases.

Not only did he recognise that a varicella eruption might become gangrenous; he had observed bullous and petechial conditions in the skin lesions and frequently serious eye complications, such as Iritis, Irido-Choroiditis and Panophthalmitis. As a result of these complications deep scars and serious damage to the eyes were frequently found.

Hutchinson, however, was not the first one to take note of the condition; he recognised the state, described by Whitley Stokes in 1807 as "Pemphigus Gangrenosus", "White Blisters", "Eating Hives" and "Burnt Holes", to be the same.

Probably a lapse of sixty years had altered the condition somewhat. It seemed to have been fairly common in Ireland at that time.

The casts in Guy's Hospital Museum labelled "Rupia Escharotica" appeared to Hutchinson to be from similar cases. This name was used first by Fagge who had also observed but had not recognised the full significance of the condition.

In the discussion which took place after the communication had been made to the Royal Medical and Chirurgical Society several opinions and cases were given. Crocker spoke of a case he had seen in which the skin lesion was a papule, which became pustular and finally gangrenous. The child, whose parents were consumptive, was unhealthy and died. He noted that a history of varicella was not obtained in all the cases and that gangrene did not always begin in the varicella lesion, but sometimes in apparently healthy skin. Greasy applications were not so good as watery ones in his experience of the treatment.

Barlow had seen fifteen such cases; in all of them the lesion appeared first as a vesicle, not a papule, and without any inflammatory areola. The vesicle collapsed leaving a punched-out or trephine ulcer.

He did not feel inclined to believe in idiosyncrasy, because all of his cases were in unhealthy children and tuberculous conditions or family histories were often obtained. Tubercular disease had been found in six of his cases post-mortem. He, however, did not attach too much importance to these findings because he knew tuberculosis was very common in children.

Parker mentioned a case of Herpes in which the vesicles contained dark blood-stained serum and formed gangrenous ulcers.

Fagge agreed with Crocker and Barlow that varicella was not the only condition in which such gangrenous ulcers of the skin were found.

Habershaw^N thought it was rather a confession of ignorance to suggest the exhibition of an idiosyncrasy in such cases and suggested the possibility of congenital syphilis, previous exanthem or some peculiarity in diet as causes.

Drewitt had one case in which the gangrenous ulcers were on the inner sides of the thighs. He suggested that locality in a general eruption might have something to do with the occurrence of gangrene.

This discussion showed that the condition had been observed by several others so that its recognition and acceptance were not difficult points.

Very soon after Hutchinson's communication, Abercrombie published a case of gangrenous varicella in a child fourteen months old. He had never been healthy, but there was no specific or tubercular history. Three other children in the family had ordinary attacks at the same time. When seen two weeks after the eruption had first appeared there were large definitely shaped ulcers on the head, some of which still had sloughs. Smaller similar lesions were present on the face, buttocks, and over the right clavicle. The ulceration spread and child died four days later. A post-mortem examination showed some pneumonic mischief, right sided pleurisy, tubercular mesenteric glands and an ulcer at the end of the ileum.

Some time afterwards, Haward described a case in a boy one year old; he was badly nourished, only weighed six and a half pounds and looked very ill - the constitutional disturbance was marked.

There were large crusted ulcers on the scalp, black circular gangrenous areas on the trunk and papules and vesicles were present all over - some with crusts. The eruption appeared one week before the child was seen by Haward. The family history was good; the child had always been puny; he had been successfully vaccinated two and a half months before.

He died four days after Haward had first seen him.

Post-mortem the largest ulcers were found on the abdomen; muscles were exposed at the bottom of some of the ulcers. There was some pleurisy with effusion and secondary abscesses in the lungs.

The gangrenous process had come on suddenly, spread rapidly and soon

caused death from pyaemia.

In 1885 Bowley gave some details of three cases in children eighteen, ten and eight months old. In all large sloughy ulcers were present. He suggested that a septic change in the secretion of the vesicle might account for the gangrenous condition setting in.

At the same time Payne described an interesting case in a girl one and a half years old. He only saw her one month after the eruption had appeared; she then had some twenty or thirty ulcers all over the body. The family history was good, but child was marasmic and fevered. One other child in the family had at the same period an ordinary attack which left her with large scars.

The child died. Post-mortem examination revealed acute military tuberculosis. Payne said that this corresponded with what Barlow and Abercrombie had found.

He concluded that varicella, occurring in a tuberculous child, was liable to assume an ulcerative form and that a fatal issue was common. The presence of the tuberculous and varicella poisons was the cause of death. When no such result occurred he presumed that the child was cachectic and thus predisposed to a severe attack.

The presence of tubercle and gangrenous varicella might be a coincidence, he thought, though the presence of tubercle might be a factor in explaining the occurrence of gangrene.

The acute military process in his case was so recent that it might have begun after the varicella had appeared.

The case which came under Jamieson's care in 1886 was in a boy one year and nine months old. He had just recovered from measles and whooping cough when he developed chickenpox. There was a family history of tubercle; the boy had never been healthy and was rachitic. When seen two weeks after vesicles had appeared there were several small circular ulcers chiefly on the back, face, head and right thigh. Ulceration extended and fresh vesicles continued to appear. There was some broncho-pneumonic mischief and the boy became very weak. This was made more evident by an attack of diarrhoea. However, at the end of the fourth

week he began to pick up and in the end made a good recovery. The ulcerative process seemed to have gone on for a certain time and then for some unknown reason ceased. Fretfulness and pallor were very marked signs in this case.

An article which threw considerable light on the subject was written by Crocker in 1887 on "Multiple Gangrene of the Skin in Infants and its Causes". There were, he said, many grades of the condition, from the simple inextensive ulcer, which was frequently accompanied by much skin irritation, to the large extensive gangrenous lesions, some of which had been haemorrhagic in an earlier stage, and these were often the cause of death from exhaustion. Tubercle, congenital syphilis and rickets were predisposing causes and the actual cause microbic. He was convinced that varicella was not the only condition in which such lesions were found and gave details of cases under four headings to substantiate this.

In the first series he had five cases in which there were no antecedent histories of varicella. All of these cases had skin eruptions which after a time developed gangrenous ulcers.

In the second series were six cases which all showed varying degrees of ulceration accompanying or following varicella. The vesicles either became gangrenous in a few days or gangrene occurred some two or three weeks after the eruption had come out, and when it appeared to be healing.

Two of these cases ended fatally; one of them presented much the same characteristic signs as Howard's case did.

As a third class he mentioned two cases of gangrenous ulceration following vaccination. The ulceration in neither case was extensive.

The fourth series he gave as an appendix and included cases in which gangrene of the skin occurred locally. He had four such cases. The lesions usually occurred round the genitals and commenced as a vesicle which soon became pustular and finally gangrenous. The ulcers were punched-out and deep. This series, he thought, was closely related to Noma.

After reviewing his cases and those in literature he came to the conclusion that if ulceration occurred when the eruption was still present, the head

and upper part of the body were generally affected and that the buttocks and thighs were attacked in those cases occurring after vaccinia, apart from varicella or some weeks after the varicella eruption had come out, and when fresh papules and vesicles appeared.

Severe and extensive ulceration usually caused marked constitutional signs. Tuberculous-pyæmic and pulmonary conditions were terminal complications often.

He noted that most cases occurred in girls and often under one year of age.

Although most such cases occurred after varicella and vaccinia, one might see it occur in any pustular or non-specific condition in young children. Febrile states were an almost constant occurrence when ulceration was present. No matter what the original skin lesion had been, the gangrenous lesion usually commenced as a vesicle which soon was pustular and crusted. Under the crust ulceration went on.

This article was undoubtedly of considerable importance and a valuable addition to the literature on the subject.

The case reported by Bellamy in the same year is of considerable interest. The patient, a girl four years old, had been running about for three days with the eruption developed. Then she began to complain of pains in her legs; small circular black-looking areas appeared on the inner sides of the knees. These spread rapidly and others appeared. Both legs presented a bluish-black lividity. There were soon patches on the thighs, discolouration of some of the toes and blebs on each foot. Patches appeared later on the arms, back, face and left ear. General signs were very marked; pallor, sallow complexion, dull heavy expression, tongue dry and brownish, weak rapid pulse, fever and excessive thirst. Both legs were cold and there was no pulse in the right femoral artery. Depression and drowsiness continued and next day petechiae appeared. All the patches increased in size and some bleeding occurred from the nose. She was very soon comatose and died.

Two other children had ordinary attacks at the same time. The family

history was good and the girl appeared to be healthy.

Post-mortem, haemorrhages were found in the lungs, heart and spleen. There were many petechiae and several gangrenous patches on the legs. There were also haemorrhagic infiltrations into the subcutaneous tissues, and muscles. There seems to be no doubt that the case was one of haemorrhagic varicella going on to gangrene in some parts.

Buchler described a case of gangrenous varicella in a girl four years old. She was one of four who developed varicella; the other three had ordinary attacks. The family history was good, but the whole family had had attacks of malaria immediately before.

There were marked prodromal signs; some eighteen or twenty vesicles chiefly on the back, buttocks and chest became filled with greenish-yellow pus and surrounded by an inflammatory areola. These were soon gangrenous and on the fourth day deep, well-defined, punched-out ulcers, penetrating the whole thickness of the skin and subcutaneous tissues, were visible. All were foul smelling and tender to palpate.

The largest ulcer was over the left mastoid region and exposed the cartilage of the auricle.

Constitutional symptoms were severe; an attack of diarrhoea supervened, she became comatose and died on the ninth day of illness.

Buchler believed that the cause of gangrene here was the insanitary surroundings in which patient remained and did not credit a constitutional condition as a cause.

Woodward preferred the name Varicella Gangrenosa to any of the others which had been used. He had two cases in girls one and three years old. Both were healthy and had good family histories; in each case the ulcers were present chiefly on the buttocks and thighs. Healing under treatment with ichthyol ointment occurred rapidly.

He observed clinically that the ulcerative process seldom went any further after the sloughs had separated and that although the lesions might occur

on any part, the buttocks and thighs were the favourite situations.

Andrew, in 1890, described two very interesting cases.

The first was in a boy, eight and a half years old. For some seventy-two hours he had blood in the vomitus and stools, accompanied by much restlessness and sleeplessness; there were no abdominal signs. At the beginning of the third day he was collapsed, had an irregular weak pulse, sighing respirations and cold extremities. Soon, however, vomiting ceased and a haemorrhagic rash appeared on the elbows and ankles. Towards the end of the day he had recovered slightly and a general rash, resembling an aborted varicella rash, appeared, accompanied by many haemorrhagic spots on the chest and lower extremities. There was only one definite vesicle in the rash. With the development of this rash the boy's condition rapidly improved. Convalescence was rapid and uneventful and he was sent away on the twelfth day well.

There was no albumen; vaccination had been successful three months previously. This case occurred in a school epidemic.

There must have been considerable interest in the case and much speculation as to the diagnosis before the vesicular rash appeared. Much importance has been attached to this case as it is one of the few true haemorrhagic cases on record.

The second case was of the gangrenous variety and occurred in a strumous boy, nine years old. Severe constitutional disturbance - dusky colour, furred tongue, offensive breath, constipation, fever and frequent pulse - was present with the rash at first. The rash was dark in colour, but not haemorrhagic. The vesicles were numerous, large in size, soon became pustular and ulcerated, discharging offensive pus. This state lasted for several days during which period he looked septicaemic.

Recovery ultimately took place.

Andrew classified such cases thus:-

- (a) Those with intravesicular haemorrhages. He had details of such a case from his friend Cronk.
- (b) Those in which the eruption of vesicles may be preceded for two or three days by a bluish erythema with small petechiae. He

quoted cases of such a nature from Baader and Berard and De Lavit.

(c) Those in which a haemorrhagic nephritis occurred.

Such a case was described by Hoffman.

The case reported by Staniforth occurred in a boy seventeen months old. There was a prodromal scarletiform rash: he had had Measles four months before. On the sixth day of illness all the vesicles had dry black crusts surrounded by an erysipeloid areola; by the tenth day all were deep, circular, punched-out ulcers, many of which had ashen-gray sloughs at the base. The face and head were free. The deep fascia was exposed in some of the ulcers. Restlessness, feeble rapid pulse and fever were prominent constitutional signs. The ulcerative process did not cease until some twenty-five days had passed. The family history was good and child was healthy; he had been vaccinated. A satisfactory recovery was made.

Amongst the earlier haemorrhagic cases is that reported by Hamann. The boy, six months old, was one of four who took chickenpox. On the third day numerous vesicles, specially on the face and head, contained blood. The appearance of child was unsightly and alarming; there was much cerebral depression which at one period bordered on coma. Restlessness and fretfulness were prominent symptoms. There were no visible lesions on the mucous membranes or bleeding from the bowel, stomach or kidneys.

Recovery was complete in ten days.

The case described by Schwartz is of considerable interest. The patient, a boy of two and a half years, was one of five in a family who took varicella. On the third day the eruption was copious and tended to become confluent in places. Several vesicles were dark in colour and round several was a purpuric areola. On the fifth day some vesicles on the back and thighs became confluent. Vesicles still continued to appear and next day the back and nates appeared as one huge blister filled with purpuric serum. Vesicles appeared in the mouth. Severe constitutional disturbance was present all the time. There was considerable faucial oedema and diarrhoea set in.

On the seventh day the large vesicle on the back burst, leaving a large raw surface from which exuded purpuric serum. The faucial condition became worse and he began to go downhill. Where the skin was not purpuric it was cyanotic. He gradually became comatose and died next day.

Schwartz apparently did not recognize the condition at first and changed his diagnosis afterwards. He put the cause of death thus: "Haemorrhages into the subdural and arachnoidal spaces of the brain and oedema of the glottes resulting from purpura haemorrhagica which had complicated the varicella." He, however, did not say whether he had a post-mortem examination or how he arrived at this diagnosis.

The case was in all probability one of haemorrhagic varicella.

Stern makes out that varicella is a much more serious condition than is generally supposed. He had seen a case in which there were an inflammatory lesion at one elbow-joint, metastatic parotitis, otitis, multiple abscesses and infiltration of the right lung. Post-mortem pus was found in the mediastinum, pericardium, bronchi, spleen and kidneys. The organism present was the *Staphylococcus Aureus*.

The first case in literature in which the scrotum was affected was reported by Spivak in 1895. Patient was a healthy boy, two years old; three other children in the family had ordinary attacks at the same time. From the first the attack was acute; on the third day he was delirious and there was redness of the scrotum. This state continued for three days during which time the redness spread on to the left groin. Then a black pin-head sized spot appeared on the left side of the scrotum, soon followed by several other similar spots which all coalesced to form a distinct discoloured area. In a very short time deep ulceration occurred. Spivak saw the boy on the eighth day of illness and then the penis, scrotum and left groin were much swollen and tender, a dirty crust and detritus were present on the left side of the scrotum and there was a very offensive odour. When the crust was removed it was found that ulceration had gone so deep as to expose the tunica albuginea of the left testicle. Pus exuded from a small opening in the left groin. The bubo was painful but the

ulcer was not.

The boy's general condition had apparently not suffered much.

Bichloride wash and iodoform gauze were used to the parts.

The bulbo was incised freely under chloroform and the cavity packed with gauze. Two grains of Quinine were given night and morning for a few days only. After this treatment healing went on satisfactorily and was complete at the end of five weeks.

Cantrell discussing the case said he had seen gangrenous lesions follow Miliria Rubra, Eichen Planus, Purpura &c. The lesion went through the following stages, erythematous spot - papule - vesicle - vesico-pustule - pustule with crust and ulcer with ashen-gray slough under the black crust.

Spivak called attention to three points in his case (a) a single lesion (b) large in size (c) only case in literature where scrotum was affected.

A boy, twenty-two months old, came under Griffiths' care with Pneumonia of five days' duration. He was very ill and had meningeal signs; he recovered however. During his convalescence he developed a diphtheritic tonsillitis and four days later measles developed. Three days after the measles rash had come out vesicles appeared; some of these became bullous. Some five days later ulceration had occurred. The ulcerative process was almost confined to the trunk and arms at first. Vesicles continued to appear and the ulceration extended and soon appeared all over the body. Numerous ulcers were present. He rapidly got worse and died on the twelfth day of illness with signs of a broncho-pneumonia. Nothing out of the usual was found post-mortem.

In this case the gangrenous process did not occur in the usual way. The vesicles soon became bullous; the bullae burst, dried up and were apparently healing when ulceration commenced in the erythematous areas which were left after the bullae had burst.

All of the ulcers were very superficial - none of them were through the skin- but most of them were very extensive specially on the scalp and back.

The child had always been healthy; the cause was most probably general and local.

Lockwood's case occurred in a well-nourished healthy girl two years old. When the vesicles appeared she was languid, fretful and vomited. Next day a dusky redness appeared on the trunk. Around many vesicles in this area rapid ulceration occurred and several vesicles all over the body had become haemorrhagic. Bleeding occurred from the nose and mouth and continued during the whole illness. The skin over the right groin became puffed up with a large effusion of blood which formed a large bleb. There was extravasation of blood over the pubes and genitals.

In many of the lesions there was no relation between the haemorrhagic and the gangrenous tendency, and skin which was free of vesicles was the seat of haemorrhage and gangrene also. On the back and front of the trunk were large patches where the epidermis was only affected exposing dry purplish-red surfaces. The extremities were free of deep ulcers - most were seen on the head and trunk. There was very offensive foetor. The eyes soon became affected by swelling and ulceration of the eyelids. The largest ulcers were outside the dusky red area - one on the inner aspect of the left elbow being the most extensive.

Restlessness was a prominent symptom. Child died on the eighth day with signs of a broncho-pneumonia. There was no examination.

The only case on record of gangrenous varicella accompanied by acute nephritis was reported by Silver in 1897. The boy had had a severe attack of gastro-enteritis during a very hot summer and autumn, but made a good recovery in the country.

Marked constitutional symptoms were present from the very first. Restlessness was very marked; a convulsion lasting twenty minutes occurred. The eruption was profuse and the eyes and mouth very much congested.

Gangrenous lesions were present on the fifth day on the right side of the neck. The right parotid became much swollen and congested. The child continued to get weaker. Blood and serum flowed freely from the mouth, the gums bled easily and there was blood and mucus in the stools. The output of urine was scanty; there was abundant albumin and granular casts, while the urine was smoky.

The condition gradually got worse; he vomited blood and mucus. The whole condition in the end seemed to be uraemic. On the eighth day of illness he had a severe convulsion and died ten minutes later. No examination was allowed.

The whole history seems to indicate that there was a large uraemic element in the case all along. How much the haemorrhagic condition was due to the uraemia is difficult to say.

In 1898 a well-nourished, healthy, girl of seven months came under Turner's care. She was one of a family of five who developed varicella. From the first the rash was profuse and convulsions occurred. On the third day erythematous patches appeared on the abdomen and one on the inner aspect of the left thigh. During the next two days several pocks had become deeper and broader and punched-out; inflammatory areolae were present. Boracic ointment was applied, but the lesions continued to get larger and yellowish sloughs began to form.

Later on the abdomen became distended and the skin shiny.

On the eighth day the skin, which had been getting gradually duskier, sloughed 'en masse' across the abdomen, leaving a gangrenous area four and a half by three and a half inches, and another on the left side two by one and a half inches. Each had deep, well-defined edges and a yellowish slough at the base. There was much redness and swelling all round. The child rapidly got worse and died next day. No post-mortem.

The child was healthy, but lived in a poor district under unhealthy conditions. Turner thought the causal agent in his case was a compromise of Hutchinson's and Goodhart's views on that point.

The apparent frequent association of gangrenous varicella with tuberculosis was discussed in an article by Edwards in 1903. He gave some details of a case in a boy twelve years old; there was much fever at first and practically all the vesicles became gangrenous. Although all were surrounded by inflammatory areolae the degree of gangrene could not have been severe as only a few ulcers formed. Recovery was delayed.

Streptococcus and Staphylococcus Albus were found.

He had seen two other cases both ending fatally.

At that time most evidence favoured a very close association between the two states. His own case was in a scrofulous boy. He quoted the case described by Ashly and Wright where the child died two months later from tuberculosis.

He thought that the appearance of the rash gave no indication at all as to whether gangrene was going to occur - and that the cause was an overwhelming invasion of pyogenic organisms, while in others it was due to a tendency to develop gangrene in any illness (Rotch) shown in some individuals.

Although there was a fair amount of clinical evidence in favour of some definite connection between the two conditions he concluded that there was not enough to make any definite statement about the Association even although the evidence appeared to be very much in favour of some association.

Kieffer thought that gangrenous varicella was not a special type of the disease but an intensification of the morbid process due to local and general extraneous infection. Gangrenous varicella was to varicella what rupial syphilides were to the syphilitic exanthem, noma to the acute diseases of children and superficial cutaneous gangrene to typhoid and other continued fevers.

He had a case in a girl, sixteen months old who was healthy and well nourished. On the second day of illness the child was acutely ill and had respiratory troubles. Some twelve vesicles were large in size, became acutely inflamed and surrounded by a brawny dusky areola. The largest one was close to the ziphoid and had an areola three inches in diameter round it.

On the third day all the vesicles had sphacelated crusts; some of these came off leaving deep ulcers in the floors of which were dense grey membranes. The smaller ones retained the crusts and from each came heavy greenish pus.

During the next four days ulceration continued so rapidly in the largest ulcer that he was afraid the ulcer was going to perforate into the abdominal cavity. Seven days after gangrene had set in the crusts came off the

smaller ulcers which dried up while the larger ones all cleared up and healing commenced.

She ran a temperature all the time between 100° in the morning and 104.5° in the evening. Healing was very slow; it was four weeks before the largest ulcer healed.

His treatment consisted of hot carbolised packs until the sloughs had cleared up and then an antiseptic dressing. Bromine was used once with good effect. The clinical points he observed were that ulceration continued as long as the varicella advanced and as soon as it declined, the ulcers cleared up and spread no further; and also the colour and appearance of the slough in the largest ulcer resembled membrane on a surface wound which had become infected with the *Bacillus Diphtheriae*. A child convalescent from Diphtheria had been in the same room as his patient but several cultures from the ulcers revealed the presence of *Staphylococcus Aureus* and *Streptococcus* only.

Kieffer discussing the subject said that there might be numerous, very few or only one lesion as in Spivak's case; and that gangrene might occur in skin quite free of varicella lesions as in Lockwood's case.

He concluded that gangrene usually occurred in the presence of marked physical deterioration, that tubercle, syphilis and rickets were the commoner predisposing causes. Under such conditions gangrenous varicella developed either as a local or general infection. Most cases were due to local infection of the ²pack by virulent pyogenic organisms. This secondary infection in most was due to *Staphylococci* and *Streptococci* and occasionally the *Bacillus Diphtheriae*.

Gangrene also occurred in healthy children.

The part played by tubercle was not definite - it probably caused diminished resistance and thus allowed the infecting organisms more scope.

Septicaemia, pyaemia and secondary joint affections might occur.

Kieffer collected forty three cases in which occurred twenty nine deaths but he thought tubercular disease was the real cause in several of these cases.

Blackham had a case in a girl five months old; there were marked symptoms from the beginning of the illness. Four other children had ordinary attacks at the same time. The eruption was profuse; several vesicles were large in size. During the next three days these large vesicles became pustular and a dusky red areola appeared round each. Brownish crusts formed under which circular, sharply edged ulcers, with sloughy bases, were found.

On the seventh day an axillary abcess formed; this was a streptococcal lymphatic infection from a large pustule on the anterior axillary line.

The ulcers and abcess took a long time to heal.

The child was very healthy and in the end made a good recovery.

During the same epidemic he saw two other patients who had very marked constitutional disturbance and profuse rashes with excessive prurigo but there were no ulcerative lesions at all.

One of the few haemorrhagic cases in literature is that recorded by Rundle. Patient was a boy, two years old, who had always been in good health. When seen on the third day of illness there were about a hundred vesicles visible. On the second day the parents saw some bleeding into a few of the vesicles. On the third day the following haemorrhagic lesions were present.

- (a) Petechiae and ecchymoses scattered somewhat profusely over the trunk, shoulders, thighs and legs - most marked over the sacrum, crests of the ilium and thighs.
- (b) Vesicles and pustules all of a purplish hue and exuding a sanious fluid on incision.
- (c) The bases of ruptured vesicles and pustules presented a haemorrhagic infiltration which could not be obliterated by fluids.

The conjunctival and visible mucous membranes were clear at that period and the general condition surprisingly good - he had no symptoms at all.

Cutaneous haemorrhages continued to appear and on the fourth day an area of subcutaneous extravasation of blood, two inches in diameter, occurred close to the left hip; a few hours later a similar lesion was found over the right

shin and towards the end of the same day a large extravasation was found under the ocular conjunctiva of the right eye. There soon occurred black haemorrhagic staining of the lips and gums. During that night restlessness, delirium, and cardiac depression were present and he gradually sank and died on the morning of the fifth day. No examination was permitted.

The boy had been vaccinated successfully in three places eighteen months before this illness; haemorrhagic smallpox was easily excluded from the diagnosis. There was no history of haemophilia and the condition was not purpuric.

Commenting on this type of case, Rundle said one may not uncommonly get papules with a violet tint and a few intravesicular haemorrhages but cases like the above were fortunately very rarely seen.

Berouilli and Baader had cases with petechial prodromal rashes. Galliard had seen epistaxis, while Comby had a case in a girl eight years old in which there was a purpura followed by haemorrhagic vesicles, ecchymoses in right iliac fossa, pemphigoid bullae and marked prostration ending in death on the eighth day.

He also quoted the cases reported by Andrew and Rolleston.

Another haemorrhagic case came under Porter's care a few months later. A girl, thirteen years old, had been troubled with an irritable papular eczema of face and backs of the hands, specially on the forehead, nose and cheeks, for a few days before varicella developed. She had acute anterior poliomyelitis when four years old, but had otherwise been healthy. Vaccination had been unsuccessful three times in infancy, but it had taken in two places five years ago.

There were prodromal symptoms for three days and then a very profuse general eruption came out. Three areas of confluent vesicles soon appeared on the face and neck. Vesicles continued to appear and on the fifth day the face was swollen and haemorrhage had taken place into the three confluent areas. She presented an alarming appearance, but felt well and had no symptoms. Things quietened down after this and convalescence was uneventful. Crusts formed on all

the vesicles and the three on the large confluent vesicles did not fall off for four weeks.

A mask of carbolised vaseline was used on the face and the body sponged down with one in sixty carbolic lotion frequently.

One vesicle at the left nipple was bullous in nature. On this scar and on one of the confluent vesicle scars keloids developed later.

Watson's case was in a boy, five years old, who was healthy but very nervous and who had had diphtheria a year before. An ordinary vesicular eruption appeared at first. On the third day he complained of pain in the right groin. Next day an irregularly shaped erythematous patch was visible in the groin. This patch spread rapidly and in thirty-six hours reached to the umbilicus, scrotum and the left groin. The scrotum was swollen three times its normal size. A bleb formed in each groin.

Watson thought it was a good picture of erysipelas.

On the tenth day a gangrenous condition appeared on the scrotum. A patch of gangrene also appeared at the bleb in the left groin. These areas were excised; healing ultimately took place and was complete in fifty days. Hydrogen peroxide and boric acid solution were used.. Only about one-third of the scrotum remained.

Constitutional disturbance was severe and child required free stimulation.

Goodall quoted a case by Kreuzeder in which cellulitis of the arm followed by complete sloughing of the arm, occurred in a child nine months old, with a fatal issue.

Alexander had a boy two and a half years old who developed varicella when he was convalescing from whooping-cough and broncho-pneumonia. He also suffered from active tubercular peritonitis.

Round each vesicle there was an intense areola; on the abdomen each vesicle was haemorrhagic while above the knees and on the arms were several cutaneous ecchymoses. There was some intestinal haemorrhage, but he thought it was due to the local intestinal condition.

Several vesicles sloughed so that a gangrenous condition existed as well. He made a good recovery from the complicated varicella attack but was of course still left with the tubercular lesion.

The case of haemorrhagic varicella reported by Elliot has many interesting features. Three boys in one family developed varicella at the same time. On the fourth day one of them, four years of age, had pain in his stomach, did not sleep well at night and next morning vomited blood - mostly clotted.

When Elliot saw him then the vesicles were healing; the eruption had not been profuse but petechiae and ecchymoses had by this time appeared all over, the former chiefly on the face, chest and abdomen, the latter generally distributed over the scalp and lower limbs. The petechiae were pin head in size, purple and in large numbers. The largest ecchymoses were on the legs and thighs, fifteen to twenty in number. In the mouth five circular haemorrhagic areas, of a deep purple hue, were visible on the under surface of the tongue near the edge. There were partially healed vesicles on the cheeks and dorsum of the tongue. The tonsils were large, dusky red in colour and on each were four to six discrete haemorrhagic spots similar to those on the tongue. The heart and lungs were normal but the abdomen was slightly distended and tender in the epigastrium. Glands all over the body were palpable. The boy felt well; early in the day he passed clotted and red blood by the bowel. The urine contained albumin, and blood microscopically.

Next day a few more fresh areas had appeared on the inside of the cheeks and on the hard palate, about half the size of those on the tongue.

On the seventh day the vesicles on the tongue had almost healed. Two fresh haemorrhagic spots appeared on the lower lip. Those on the tonsils had become confluent; here and there were breaks on the surface which exposed the mucous membrane with blood adherent to it. The urine was clearer, but there were still clots in the stools.

A tracheotomy had to be done that morning to prevent him choking by a piece of gum which he had inadvertently swallowed. He was restless during that night, but the tube did not appear to upset him much. Next day he felt well. The urine was clearer; a pin head sized haemorrhagic spot was now visible on the conjunctiva of the left lower eyelid and a similar one immediately below the iris.

The ecchymoses on the scalp and legs had begun to fade while the spots on the tongue and cheeks were practically healed. On the following day, the ninth of his illness, the stools and urine were clear. The petechiae were fading and dusky in colour, while the tonsils were less swollen and cleaner. The boy seemed bright and well, and at night appeared to fall into a natural sleep, but in half an hour he woke up, had a slight convulsive seizure in which he put his hands up to his head and passed into a general spasm and was dead in two or three minutes. An examination was unfortunately not allowed.

The blood in the urine in this case was from the bladder; a smear film was like an ordinary blood film.

Elliot observed no change in the coagulability of the blood when he did the tracheotomy, and was of the opinion that the issue did not depend on the amount of blood lost but on the site of the haemorrhage. He referred to the cases reported by Rundle, Porter, Andrew, Rolleston, Hamann, Comby and Alexander.

He thought varicella was not such a benign disease as most were inclined to think, because deaths occurred from gangrene, erysipelas, pyaemia, nephritis, bronchitis, broncho-pneumonia and parotitis.

These three cases reported by Rundle, Porter and Elliot give a good clinical picture of the haemorrhagic form of varicella and would have been more or less complete had there been some gastro-intestinal bleeding.

A case of Dermatitis Gangrenosa Infantum was observed by Adamson in a child one year old. Gangrenous ulceration occurred about four weeks after an attack of varicella. The ulcers were all over, but chiefly on the lower back where twelve large ulcers were present. This condition had been present for two weeks when he first saw the child; the child very soon died. There was nothing of unusual interest discovered at the examination.

The chief point of interest in the case was that the *Bacillus Pyocyaneus* was present in cultures from the lesions as well as *Streptococcus* and *Staphylococcus*. Ehlers, Hitschmann and Kreilich has found the same organism.

Palpebral gangrene in varicella must be very rare as only four cases are in literature at present. Rolleston reported one in a healthy boy sixteen months old. Scarlet fever and varicella were present at the same time. One

vesicle over the external canthus of the right eye had been scratched. The lids became oedematous. The following day the oedema was less but a patch of gangrene appeared and spread along the lower eyelid. The whole surface of the lower lid was involved; the slough separated exposing muscle; there was no foetor. The wound healed satisfactorily.

Restlessness and fretfulness were present. The stools were loose and green. Staphylococcus and Streptococcus were found in culture.

Rolleston quoted three cases by Romer, Isola and Homer.

In this case there was only one gangrenous lesion. No evidence of tubercle was found and the history was good.

Knowles made an exhaustive search in the literature on this subject and classified the cases thus:-

(a) Varicella with haemorrhagic lesions.

Of this type were the cases noted by Hamann, Andrew and Cronk. In these cases haemorrhage took place into the vesicles.

(b) Varicella with haemorrhagic and gangrenous lesions.

Gangrene follows haemorrhagic lesions in this class of cases. Crocker had one of this type and Knowles quoted two cases of Demme's, one in a boy three years old. On the second day of illness he vomited blood-streaked masses and the vesicles became a dirty grey colour. Next morning the colour was violet. Haemorrhage also occurred from the bowel. Several vesicles became confluent and bluish-black in colour. Crateriform ulcers formed. The child died; nothing unusual was found post-mortem.

The second case was in a boy, two and a half years old, who had just recovered from pertussis and who had pulmonary trouble.

Vesicles behind the ears became violet coloured, those on the abdomen and chest had a violet areola. Numerous small gangrenous lesions occurred on the face, chest and abdomen. The child recovered.

(c) Varicella of the usual type but associated with petechial rashes.

These rashes usually precede the vesicular eruption. Storer, Baader and Berard and De Lavit described such cases.

In Storer's case there were vibices on the forehead and left temple of a brownish venous colour, petechiae on the glutei and bright red areola round some of the purpuric spots. Vesicles appeared with the above. Death occurred suddenly; there was nothing post-mortem.

Bluish erythema with small petechiae scattered throughout it preceded the varicella eruption by three or four days in Baader's case while in Berard and De Lavit's there was malaise and restlessness for a day followed by a general petechial rash, most marked in the

neck, chest, hypogastrium and genitals. Two days later there was epistaxis and some days later the varicella rash came out. Patient was a boy over six years old.

(d) Varicella with haemorrhages from the stomach, bowel and kidneys.

In this class are found those described by Andrew, Hoffman and Demme. Hoffman had a case with haemorrhagic nephritis occurring on the seventh day of a severe attack.

Demme had one case with haematemesis and a second in a child one and a half years old. This child had haematemesis and malaena followed eighteen hours later by a varicella rash.

(e) Gangrenous non-haemorrhagic varicella.

In this class are the cases described by Hutchinson, Stokes, Fagge, Buchler, Payne, Woodward, Barlow, &c.&c.

Gangrene occurs in these cases without the exhibition of any haemorrhagic state at all.

He had a case, in a healthy boy two and a half years old, in which the clear vesicles became cloudy and were haemorrhagic ten to twelve hours after coming out. Vesicles appeared in the mouth and fauces and probably also in the larynx as he became croupy before death occurred. Gangrenous lesions developed all over the body.

Knowles concluded that there was a true variety of haemorrhagic varicella and that it might go on to the gangrenous type : that the virulence of infection and the resistance of the patient were the important factors in determining the issue: that a true form of gangrenous varicella existed in which the vesicles became purulent and then gangrene, usually of an ecthymatous or impetiginous type, occurred.

He thought the term *Dermatitis Gangrenosa Infantum* ought to be used whether the gangrene followed varicella or not.

Etiology.

Tuberculosis, congenital syphilis and rickets are the more common predisposing causes. These probably act by lowering the child's vitality. The majority of children who develop gangrenous lesions are badly nourished, live in the poorer districts amidst dirt and grime and are always in unhealthy and unhygienic surroundings and conditions. There is no doubt that these ill-cared-for and neglected children are predisposed to the severer form by such. Buchler ascribes such conditions to be the cause in his case.

Gangrenous lesions also occur in healthy, well-cared-for children as well. We must put this down to the exhibition of an idiosyncrasy on the part of the patient or to the fact that certain people show a distinct tendency to develop the severest form of each and any disease they may chance to develop.

It is not always in the badly nourished scarlet fever patient that one gets the septic throat with sloughing of the tonsils, uvula or soft palate; the healthy person may develop gangrene of the lung in pneumonia or gangrene of the appendix.

Gangrenous varicella is not a special disease; it should be considered as the severest form of an otherwise usually benign exanthem. Previous illness leaving a child debilitated from prolonged fever or severe toxæmia must necessarily predispose to a severe exacerbation. Several cases are in literature where pneumonia, gastro-intestinal disturbance, diphtheria, and whooping cough seemed to predispose. It is well recognised that scarlet fever patients tend to develop severer attacks as a rule.

Local causes are present much more frequently than general ones. The fact that in the large proportion of cases, only a limited number of gangrenous lesions occur, shows this. There seems to be no doubt that a child, recovering from one of the eruptive fevers or some skin eruption, always tends to develop a severe attack. This is well seen in scarlet fever patients and in the case reported by Porter. The skin in all such cases is probably in a toxic state and unable to offer its usual resistance to outside infection.

Dirty clothes in contact with the eruption must lead to infection of the parts in contact, specially if there be much rubbing.

The most common local cause is probably scratching with dirty finger nails. I have no doubt that this was the cause in my first case, while in the second case under my care I had good reason to believe that the gangrenous lesion commenced in a part which had a short time previously been the seat of an acute inflammatory process. Gangrene seems liable to occur thus, in a part which has been infected with pyogenic organisms immediately before.

Locality in a general rash seems to have some predisposing influence in certain cases. Thus gangrene although it may occur in any part in varicella, usually occurs on the buttocks, thighs and lower back and less frequently on the scalp.

P A T H O L O G Y.

The cause of varicella remains unknown. General opinion favours an infection by some micro-organism but this has not been definitely discovered yet although protozoa and unicellular organisms have been described by Pfeiffer and De Korte.

There is a general concensus of opinion that the haemorrhagic and gangrenous forms are due to secondary infection. There are too few cases on literature of the haemorrhagic type to say anything definite about the pathology of that variety. The infection is most probably a general one.

Various organisms have been found in the lesions present in the gangrenous cases. Streptococcus Pyogenes and Staphlococcus Pyogenes Aureus have been found in many cases either in pure culture or mixed. (Adamson, Rolleston, Kieffer, Edwards, Baudouin and Wickham) Adamson found Bacillus Pyocyanus present in one case mixed with Streptococcus and Staphlococcus. Ehlers discovered the same bacillus in two of his cases as did Hitschmann and Kreibick also. (Crocker).

Krjukoff found the Bacillus Diphtheriae present in the blood stained pus from the ulcerating skin lesions. The same bacillus has been found by Labbe, Demarque and Dettinger. (Knowles).

Until the Pathology of uncomplicated varicella is known however we are likely to remain in the dark as to the true nature of these two complications.

Much has been written and said about the frequent association of tuberculosis and gangrenous chickenpox but no one has so far found any tuberculous condition in any of the lesions. Histological and bacteriological examinations in several cases have been negative. Barlow first drew attention to the association of the two diseases. He however did not lay too much stress on the finding of tuberculous lesions in his cases. If tuberculosis was the cause of gangrene occurring in chickenpox then we should see numerous cases of

gangrenous varicella in every epidemic but we do not.

Post-mortem examinations have not thrown much light so far on the subject. It would be interesting, specially in the haemorrhagic and tuberculous cases, to know exactly what state the suprarenal glands were in. No attention has so far been paid to this.

CLINICAL SIGNS AND SYMPTOMS.

Several writers recognise three forms of Varicella Gangrenosa; this division is probably a good one for purposes of description and seems to answer the clinical picture fairly well.

The mildest form is that in which superficial ecythematosous sores are present. This form has been found frequently by several writers and has appeared in epidemic form in children grouped together in schools and asylums, while others regard this form as being not uncommon in dispensary work. There seems to be no doubt that this form is common but that little attention is paid to it because in most cases the sores are not numerous and heal up readily. Quite a number of simple varicella cases show a degree of ulceration when the crusts fall off. Those cases which came under notice are probably in unhealthy, badly nourished tubercular children, living in insanitary places and with dirty clothes etc. The cause is in all probability due to scratching; when this is kept up and dirty clothes are allowed to rub on the sores one can quite well understand why so many of the ulcers are so persistent and heal up very slowly.

Such cases seldom show any constitutional symptoms.

The second form includes those cases in which there are deep ulcers with sharp angry-looking edges and black gangrenous crusts. To this class Hutchinson gave the name Varicella Gangrenosa.

In these cases the vesicle instead of drying up becomes the seat of an acute inflammatory process. The vesicle may become bullous - more frequently however a brownish or black crust forms; this may be umbilicated. Usually there is a pustular border round this central depressed crust and an inflammatory dusky red areola of varying diameter. At first the crust is adherent and under it more or less active ulceration goes on resulting in the formation of an ulcer of varying size and depth with a dirty black, ashen grey or yellowish

central slough. The ulcerative process may spread and, if the ulcers are close together, two or more may coalesce to form large sharply-edged deep ulcers frequently circinate and irregular in outline. After a varying interval the central slough separates and the ulceration as a rule ceases and healing occurs rapidly.

Great variation exists in this class of case. The ulcers may be on any part of the body - more frequently on the scalp, lower part of the trunk and thighs; there may be many or comparatively few; the process may be very acute or several days may elapse before definite ulcers are formed; the size of the ulcers varies greatly as does the depth. In almost all cases however the whole thickness of the skin is involved; it is only in the severe cases that one gets muscle exposed.

When ulceration occurs rapidly one is more apt to get large deep angry-looking ulcers extending to the deeper subcutaneous tissues and muscles. There is a tendency for numerous ulcers to form when the eruption of vesicles is profuse.

In those cases occurring some, two to four weeks, after the vesicles have first appeared the lesion starts usually as an eruption of papulopustules which go through the same process. In these the ulcerative process usually tends to be subacute.

Sometimes gangrenous patches of skin are present as well as the gangrenous ulcers.

Both of these forms occur more in girls and usually in children under three years of age.

Constitutional symptoms vary considerably in these cases and it cannot be definitely said that the number, size, depth and situation of the ulcers have any direct relation to the amount of systemic disturbance.

There may be no constitutional upset at all; on the other hand there may be much fever, wasting, vomiting, diarrhoea, exhaustion and convulsions.

Cardiac and pulmonary complications are not uncommon and almost one half of the cases show signs of general sepsis; pyaemia occurs in a few as a terminal event. It may be said however that generally the constitutional disturbance varies as the degree of severity and number of ulcerative lesions.

In the third form of the condition diffuse gangrenous lesions are found on one or both lower limbs and occasionally on the trunk. There are few cases of this nature seen fortunately because it is the most fatal form. The cases reported by Bellamy, Turner, Watson and Spivak and those under my own care are examples of this type.

In this class one usually finds a single, more or less extensive lesion with gangrene of the skin, subcutaneous tissues and deep fascia so that the deep lying tissues - muscle, periosteum and glands - are usually exposed. Constitutional symptoms are as a rule severe and the cases usually end in a septicaemic or pyaemic condition about the seventh day. Secondary infection of one vesicle may lead to extensive necrosis and pyaemia (Munro). Clinical evidence goes to show that these cases tend to occur in children over three years of age and in previously healthy children. The gangrenous process may set in at any period of the illness; but usually during the first days and is invariably acute.

I have verbal details of two cases of this type in both of which the large single gangrenous lesion was on the thigh and spread so deeply and rapidly as to expose the hip-joint in each case. Both cases ended fatally.

In the haemorrhagic cases different forms of haemorrhagias are seen.

A prodromal bluish erythema with numerous petechiae may occur or the petechiae may appear alone.

The commonest lesions however are the intravesicular haemorrhages and bleeding from the stomach and bowels. Haemorrhage may occur from the kidneys as in Hoffmann's case, from the bladder as in Elliot's case or from the nose, mouth, gums and lips as in Rundle's case. It thus appears that

haemorrhage may occur from any of the mucous membranes and it is possible that there may have been a cerebral haemorrhage causing the sudden death of Elliot's patient. There were meningeal haemorrhages in Schwartz's case but he attributed that to a purpuric condition. Epistaxis has been observed in a few cases.

Purpuric spots may appear also and conjunctival haemorrhages have been noted.

Less frequently there occur areas of ecchymoses over which blebs, filled with purpuric serum, occasionally developed.

A haemorrhagic stage sometimes develops before gangrene sets in; this is seen frequently in the second grade of the gangrenous type and in the third grade the skin may be haemorrhagic before gangrene is noticed. This is what occurred in my second case and also in Bellamy's case.

DESCRIPTION OF CASES.

Case 1. On 3rd. December, 1912, a healthy, well nourished boy, 6 years old, was admitted to hospital suffering from scarlet fever. The attack had commenced on the 25th. November with headache, followed by vomiting and sore throat on the 30th. and a general rash two days later.

On admission a general punctate erythematous rash, furred tongue, faucial injection and enlarged, tender cervical glands were present. The attack was comparatively mild in degree - the only point of interest in the course of the illness was that the tonsils became enlarged and covered with deposits. On admission, and when the tonsillitis set in, the temperature reached 99.8° . These were the highest readings during the illness.

The tonsillitis and adenitis subsided quickly. He was allowed up in clothes on the 16th. December.

On 31st. December he developed profuse nasal discharge. Two separate cultures showed the presence of numerous rod-shaped bacilli, only a very few of which showed staining with toluidine blue. The condition was not considered to be diphtheritic.

Patient had been exposed to chickenpox in the ward on 17th. December and so had to be kept in quarantine. The exposure was from a girl who was admitted on 9th. December with scarlet fever and who developed chickenpox on 17th. December. She was supposed to have had chickenpox. Patient developed chickenpox on 2nd. January 1913. Fresh vesicles appeared during the next three days; the eruption was not however copious.

There was nothing in the boy's condition to attract special notice until the morning of the 5th. January when his temperature was 101° . He felt well; no apparent cause was discovered at that time. A few pustules were now present. He had been using his fingers to allay irritation in his nostrils and had been scratching his skin in several places.

Towards evening the temperature rose and at eight and twelve o'clock was 103° . The pulse was 144 - somewhat frequent but of good quality. The only

constitutional symptom was slight headache.

During the night he was somewhat restless.

After being sponged down early in the morning a patch of redness was noticed over and below the middle of the left clavicle, about the size of a crown-piece. There were two pustules in this area about one and a half inches apart.

He was now much more acutely ill. He had headache but no sickness or vomiting. The breath was foul, the tongue was thickly furred - previously it had been clean - and there was considerable faucial injection and swelling of the tonsils. He was very fretful; he lay in bed slightly on his left side with the head inclined to the left side - an attitude not unlike that adopted by the person who has a fractured clavicle. Any movement of the head, arms or trunk which disturbed the region, where the redness was, made him cry out.

The patch of redness spread very rapidly and in the short space of four hours, it had spread right over the left shoulder - downwards on to the chest wall to the level of the fifth rib and across the chest and front of the lower part of the neck on to the anterior aspect of the right shoulder. The reddened area was swollen, the skin was not glazed at all and the spreading edges were fairly well marked. The skin was very tender indeed, the slightest touch causing him to cry out. There was very acute hyperaemia. The cutaneous tenderness made it difficult to palpate but judging from the amount of swelling there appeared to be some involvement of the subcutaneous tissues. The swelling immediately above the manubrium ~~sterni~~ was soft and puffy. Immediately over the middle of the clavicle there was one pustule and an inch and a half below and nearer the middle line was a second one. Before this condition occurred there had been no redness or swelling round these pustules to indicate that any acute inflammatory process was going on and when the red patch appeared each pustule only showed a small collection of pus with very slight umbilication.

Fomentations wrung out of one in forty carbolic were applied every three hours. One grain of calomel and thirty of jalap powder were given. Fluids

only were given by the mouth.

The temperature gradually rose and in the evening was 104.8° . Tepid sponging reduced it to 101.2° . He was very restless during the night. Next day - i.e. on the 7th. - the redness had spread still further but very much less rapidly. The parts were if anything more swollen. The skin remained very hyperaemic and tender. No fluctuation was made out.

By this time the constitutional disturbance was becoming more evident. He presented the appearance of a person becoming bowled over by a severe toxæmia. The temperature continued to run about 104° . He was very thirsty and took considerable quantities of fluid nourishment. As the bowels had not moved a soap and water enema, and some castor oil by mouth, were administered. A copious movement of the bowels seemed to make him much easier.

The carbolic fomentations were continued as before. Towards evening the temperature fell and at midnight was 100° . The pulse was 140-144. He had another very restless night; slight headache was still present. Any movement of his body made him cry out.

On the 8th., the temperature again rose to 102.5° in the forenoon and the pulse had become more frequent. 160. The redness had not spread any further however. Over the right shoulder and on the chest wall the redness was less marked but over the left shoulder the state of affairs suggested that there was still going on a very acute inflammatory process. There was more swelling in that area and the tenderness and hyperaemia excessive. No fluctuation could be obtained at any point. Boracic fomentations were now applied every three hours.

There was no tendency for the redness to extend onto the face or scalp although the face was frequently flushed.

The child was by this time so ill that it was deemed necessary to make exploratory incisions.

The condition had come on and spread so quickly and the boy had become so ill that it appeared as though nothing would make any impression on the condition.

Under light chloroform anaesthesia the two pustules were lightly scraped with a small blunt spoon. The lower one was quite superficial but the upper one had evidently perforated the whole thickness of the skin as, after scraping it, a distinct circular aperture about $\frac{1}{8}$ of an inch in diameter was seen. A probe was passed through this and could be moved about for a considerable distance in all directions. The aperture was punched out in appearance and from it exuded a small quantity of thin, watery purulent fluid.

With the probe in position a large free incision was made from above downwards and smaller ones on each side of it. A small amount of thin watery purulent fluid came away and it appeared from several small pockets, not from any large cavity.

The incisions exposed the cellular tissues which were found to be in a complete state of sloughing - a large yellowish slough was what appeared at the bottom of the wound. No muscle was visible. The incisions did not by any means reach to the edges of the affected area but were made large enough for adequate drainage. The skin all round was considerably undermined and in parts looked as if it would ultimately slough.

A large one in forty carbolic fomentation was applied and allowed to remain on for eight hours, at the end of which time it was found that a small area of skin - about one square inch in size - had become quite black. This area was free at three edges where three incisions had been made. A boracic fomentation was applied at this time. When this was changed six hours later no further discolouration had taken place, nor had any at the end of another six hours.

There was really no very apparent change in the wound itself except that the edges had retracted considerably exposing the slough more. It was now dressed every three hours with lint wrung out of hot normal saline solution and then dipped in a solution of ten per cent ichthyol in glycerine.

The redness at this period began to spread over the top of the left shoulder on to the back and deltoid regions and also into the axilla. The parts

which had been early involved still remained reddened, swollen and tender.

On the 10th. both pulse and temperature were lower in the forenoon but at night were elevated again. The wound now looked like a huge gash so much had the free edges receded. The yellowish slough was somewhat blackened in parts but no more discolouration of the skin had occurred. The wound was four inches long and at its upper and lower ends two and a quarter and three and a half inches broad. The same fomentations were being used and normal saline solution and hydrogen peroxide were used to clean up the wound.

Generally the boy's condition had somewhat improved but he still looked very toxic although he had practically no constitutional symptoms.

Next day both pulse and temperature were within reasonable limits. The wound was beginning to look much healthier even although the skin edges remained red and much undermined. The slough was separating rapidly exposing muscle in places. There was no excess of discharge which was quite sweet - it had never been offensive. The undermined skin on the outside was becoming adherent. The redness, swelling and tenderness in the surrounding tissues were rapidly disappearing.

Patient's general condition had become much more satisfactory.

On the 12th., four days after the incisions had been made, a great change was noticed in the wound. The redness of the skin was limited to a varying margin of one to two inches all round except at the uppermost margin where it tended to spread and had involved a pustule which had formed a small ulcer now. There was still tenderness and slight swelling. The slough had practically all separated leaving a healthy granulating surface and exposed muscles.

The boy had wasted considerably during this acute process but with a healthy appetite and normal pulse and temperature a distinct change for the better was visible. 14th. During the last two nights he had slept very well. The dressing had only been changed four times in the last twenty-four hours. There was no excess of discharge and the surfaces were very clean. Fibres of

the Platysma Myoides, Pectoralis Major and other smaller muscles were visible. The early granulation tissue appeared healthy while the previously undermined skin had become adherent all round except in one or two small places.

The redness and swelling of the skin had practically all disappeared. There was still some tenderness but movement was free and easy. The edges had receded so much as to leave the outline of the outer three-fourths of the clavicle quite distinct.

Hydrogen peroxide and saline solution were being used still to clean the wound but the lint dressing was only being wrung out of saline.

On the 22nd., fourteen days after the operation, the boy's condition was very satisfactory. He was able to sit up and use the arm quite freely. Pulse and temperature were normal. He had no signs or symptoms, had regained his colour and much of his weight and had entirely lost the toxic appearance he had for several days.

The wound was by this time a healthy granulating one and in shape had become quite like a maple leaf. The granulations were sound, there was no excess of discharge and the edges were growing and adherent all round except at one small part between the two ~~sternal~~ ends of the ~~sterno~~-mastoid muscle where a small pocket existed. In size the wound was distinctly smaller but retraction had been so marked at the lower and innermost end of the wound as to expose completely the whole outline of the clavicle.

The loss of the subcutaneous tissues had made no appreciable difference in the outline of the parts. Dressing was done night and morning at this time. Red lotion was used now as a dressing.

On the 29th., a blotchy erythematous rash appeared all over his body. The cause of this was indefinite but in case it might have been due to absorption of the red lotion solution, this was stopped and normal saline solution used again. His general condition remained good.

The rash disappeared in a few hours.

He was allowed up with the arm slung across the chest and fixed there.

The wound was healing well; there was no excessive growth of granulation tissue and the discharge sweet and not excessive. On the 3rd. February he developed an acute attack of rheumatism which yielded in a few days to salicylate treatment. Both tonsils were much inflamed and had large deposits. Four separate cultures showed a mixed streptococcal and staphylococcal infection. The throat did not clear up until the 12th. The wound was in no way affected. Red lotion was again used.

20th March. The wound had now healed up completely.

The boy's general condition had remained good. He had unimpaired movement of the arm. The whole result was very satisfactory. Culture from the wound after incision gave rise to a pure growth of *Staphylococcus Pyogenes Aureus*.

The photograph shows the parts on the 10th., two days after the incisions had been made.

There seems to be no reason to doubt that this case was one of the severest types of varicella gangrenosa. The uppermost vesicle had evidently become secondarily infected with a virulent type of *Staphylococcus Pyogenes Aureus*. This infection had been so acute as to cause a gangrenous perforation of the skin at the base of the vesicle. The virulent pyogenic organisms once free in the loose cellular tissue continued their destructive process and set up an inflammatory process so acute as to cause complete necrosis of the affected area.

At first one was inclined to ask if the condition were diphtheritic particularly as he had been using his fingers to his nose and then scratching himself but no bacilli were found in cultures. The infection appears to have been a pure *Staphylococcus Pyogenes Aureus* one.

Free incisions so as to allow of free drainage and the use of mild antiseptics frequently are points to be observed in the treatment of this case.

The boy was healthy and the family history was good.

Case 2. A well nourished, healthy boy, four years old, was admitted to hospital on the 24th. November suffering from scarlet fever. The attack had commenced on the 20th., with discharge from the nose followed by headache and vomiting on the 23rd. and a rash on the neck and chest on the 24th. On admission there was a general punctate erythematous rash present, with faucial injection, peeling tongue and enlargement of the cervical glands. The temperature was 101.6° on the night of admission.

The attack was comparatively mild in degree; there were no complications and there was very slight desquamation.

He was allowed up in clothes on the 14th. December.

He was in the same ward as the patient described in the first case and so was in quarantine for chickenpox.

On the 13th. January 1913, he developed chickenpox. The attack appeared to be comparatively mild; there was not an excessive eruption of vesicles. Four days later the temperature was 99° , a punctate erythematous rash suddenly appeared all over, there was stomatitis and considerable injection of the fauces. The boy was quite off colour. There were a few pustules.

Next day - the 18th. - the rash had completely disappeared, the tongue was red and well papillated and the fauces still markedly injected. His general condition however was better.

At four o'clock next morning, he was very much flushed, looked ill and had a temperature of 101.4° . No rash was visible. Three hours later a patch of redness, about the size of the palm of the adult hand, was discovered on the posterior aspect of the right thigh about its middle. This had apparently developed quickly and in about five hours' time was almost three times as large. A small septic focus appeared on the left thumb also. The boy had become now acutely ill; he had headache and was very pale. The temperature continued to rise, the pulse was frequent, 156, and thready, he was very thirsty and lay quite flat on his back in bed.

The patch of redness on the thigh was markedly swollen and indurated.

It had rather an indefinite spreading edge and was tender to touch. When first noticed this patch was free of pocks but right in its centre there was a small pin head sized papule. The patch in its rapid spread soon involved on the inner aspect of the thigh a vesicle which was slightly crusted and quite black in colour. Before the redness appeared there had been no signs of any inflammatory process around any of the vesicles or pustules.

The difference in the size of the two thighs was very apparent. Fomentations of normal saline solution and ten per cent ichthyol in glycerine were applied every three hours.

During the afternoon, the punctate erythematous rash reappeared. Towards the evening the boy's condition became much worse. The redness had spread up - down and round the thigh so as to extend from the hip to the knee and there was much more spread on the inner side of the thigh than on the outer. The thigh had become even more swollen and appeared to be very tense but palpation revealed the fact that there was considerable softness and at places some pitting on pressure. In the centre of the patch, which had appeared first, there was now present an area of skin, about the size of a penny, which was bluish-black in colour. The parts remained very tender and were acutely hyperaemic. He was now more inclined to lie on his right side with both the hip and knee flexed. Extension caused him to cry out.

The constitutional disturbance became much more marked. The pulse was 160 - very thready; the temperature 104.2° ; he had vomited and had become restless and fretful.

20th. He was very restless during the night, cried out for drinks frequently and had practically no sleep. In the morning he looked very ill, the pulse was of worse quality and the temperature 102° .

The redness had spread still further but very much less quickly. The scrotum and the left thigh at the gluteal fold had become involved and the parts all over were more swollen. The bluish-black area noticed last evening now measured three and a half inches along the thigh and six and a quarter round

the thigh so that it had spread rapidly and more so on the inner side of the thigh. In this discoloured area there were three small bullae from which, on puncture, watery sanious fluid came. This was evidently a haemorrhagic condition. Much of the swelling was now due to oedema on the posterior aspect of the thigh chiefly in its upper parts.

Under light chloroform anaesthesia in the afternoon five exploratory incisions were made on the posterior and inner aspects of the thigh. There was a free flow of watery sanious fluid. The subcutaneous tissues were dark yellow in colour but there was no evidence of pus or of sloughing. Large fomentations of normal saline solution were used at this time and changed every three hours. At each dressing a moderately free flow of blood stained watery fluid occurred. After two or three dressings the subcutaneous tissues became rather sloughy in appearance.

Towards evening the discoloured area had spread upwards and outwards from the inner aspect of the thigh towards the groin in a narrow strip about an inch and a half broad. This strip was quite black in colour. Sickness and vomiting had become more persistent. Restlessness was marked; thirst was excessive and was rendered more troublesome because almost everything he took was soon vomited.

The pulse was very poor indeed so three minim doses of liquor strychninae were given every three hours by mouth.

On the 21st. he was still very restless and had not slept at all during the night. Vomiting persisted even plain water was immediately returned. About eight o'clock he had a rigor lasting almost five minutes.

The discoloured area on the thigh had by this time become quite gangrenous and had spread in front and on the back of the thigh. Patient's colour was bad and the skin generally cyanosed. The pulse was so weak and irregular as to be uncountable at the wrists. The left side of the face was twitching frequently; he remained restless and fretful. He vomited persistently; towards evening his condition was such as to make me think he was going to die soon.

During the afternoon he was given the following mixture:-

Rx.	Ac. Hydrocyan Dil. m.	32	<u>32</u>
	Sod. Bicarb.	3	<u>3</u>
	Mag. Carb.	3	<u>3</u>
	Tr. Digitalis.	3	<u>3</u>
	Aqua ad	3	<u>3</u>

Sig. 3 $\overline{\text{ii}}$ every two hours.

This mixture seemed to allay the vomiting as it ceased about nine o'clock at night. There was at that hour no further change in the condition of the wound except that the redness and swelling were slightly less marked. The boy seemed to pick up a little as his colour improved and the pulse became a little steadier. The temperature then was 100.4° . The scrotum had become more swollen however and that part of it adjacent to the thigh had become discoloured. There was some foetor now and the superficial layers of the cuticle on the gangrenous area were commencing to peel in large pieces.

During the night he only vomited once slightly. The thirst remained excessive while the tongue kept moist. He slept in all about three hours. When the wound was dressed in the morning it was seen that the gangrene had spread further. Almost one half of the scrotum was bluish-black in colour and on this part were two small bullae. The original patch of gangrene had by this time become a dull greyish colour but kept quite moist. The narrow strip in front of the thigh however remained black and was inclined to be dry.

Except at the scrotum and at its uppermost margin the gangrenous area now seemed to have definite separation edges beyond which the sound tissues remained reddened but much less swollen.

Much difficulty was experienced at this period in dressing the parts, because he kept the knee and hip flexed and the area was so big. The parts around the scrotum were in addition very tender to touch. All this time there was present the punctate erythematous rash.

Towards evening the patient had improved somewhat in his general condition. No more vomiting had occurred, he was much less fretful and restless and was retaining a fair amount of fluids by the mouth. There was some

twitching of the left side of the face still. The pulse was of better quality, 152, and the temperature 99°. The swelling of the scrotum was spreading up on to the lower abdomen over the pubes while both groins became red, swollen and very tender to touch. The strychnine by mouth was stopped; the other medicine however was continued. The solution of sodium citrate and sodium chloride, as advocated by Wright, was now being used for the fomentations which were as yet being renewed every three hours.

His colour had improved but was still cyanotic.

23rd. Patient had a much better night and slept soundly at intervals. Dressing the parts made him restless for a little. There was only slight vomiting at five o'clock; he remained thirsty but was more reasonable in his demands for drinks. A slight general improvement was noticed. The thigh and scrotum were looking somewhat cleaner; there had been no further spread of the gangrenous process. What now was seen was a large dull, moist, whitish-grey, shrivelling area of necrosed skin extending from below pouparts ligament in front round the inner side of the thigh and then right back to the buttock where it was more evident on the inner side. The scrotum was less swollen and the discolouration less marked but there was some superficial peeling of the cuticle. A definite separation edge was present all round by this time from which the gangrenous area was receding leaving some undermining at places. In the perineal region there was only a very narrow strip of reddened skin between the gangrenous parts of the thigh and scrotum; this strip was completely undermined. The skin beyond the separation edge remained reddened and swollen but seemed to be subsiding. The parts in the right groin were less swollen but still very tender to touch and on movement. The skin on the outer aspect of the thigh had almost resumed its natural condition while that over the knee was reddened but not swollen.

The position of the leg remained in a state of flexion with abduction now. The dressing was done as before.

All over the body traces of the punctate rash were visible and a slight degree of cyanosis still existed. The temperature was 98° - 99° and the

pulse 140-144. Twitchings were still visible. During the day he slept several hours. The gastric mixture was stopped and the strychnine given again. There had been no more vomiting but the stools had become greenish in colour and there was a trace of albumin.

24th. Patient had a good night's rest and slept for several hours. At four o'clock he complained of pain in the left axilla and three hours later cried out with pain when the left elbow was moved. He vomited once after taking a large drink of milk. The pulse in the morning was more frequent and the temperature had risen. His face was more flushed. The condition of the thigh was more satisfactory. The large slough, which was practically the whole posterior aspect of the thigh, had separated still further. More than half of the scrotum was sloughing as well. There was less redness and swelling of the rest of the thigh.

The separation edge was rather angry-looking; the foetor had by this time become offensive so iodoform powder was dusted over the parts. It was impossible to tell at this period how much of the subcutaneous tissue was going to slough; it looked very doubtful all over. There was nothing apparent to account for the pain in the left axilla but the left elbow was slightly but distinctly swollen, all round. No crepitations were elicited, but there was a faintly discoloured small area immediately behind and on the same level as the external condyle of the humerus. There was if anything more swelling over this part and it was distinctly tender to touch. Movements made him cry out with pain; supination seemed to cause him most pain. There thus appeared to be some acute condition either in or around this joint but what it exactly was, was difficult to tell. Boracic fomentations were applied every three hours. The temperature at midday was 101.6° . This joint affection made his condition less satisfactory. Several vesicles were at this time covered with black crusts but there was no inflammation round any of them.

25th. During the next night he slept some six hours; he took fluids well and only vomited once. Both pulse and temperature were elevated. At six

o'clock in the morning he complained of pain in the right elbow. The thigh now looked very ugly. The sloughing area had receded more and fallen below the skin level making it crateriform in shape. The iodoform powder was all swabbed off as there seemed to be too great a risk of absorption from the large raw surface. Fomentations were applied every four hours now. Although ugly to look at, the parts were really in a fairly healthy state at this period.

The left elbow was less painful to touch and on movement but was otherwise as it appeared yesterday. The right elbow however had become swollen and painful; there was a faint discolouration over the head of the radius which was very tender to touch and on movement. Fomentations were applied here also every three hours.

A small septic focus also appeared on the back of his neck.

During the course of the day he vomited several times and in the afternoon his temperature was 104.4° . Later on he became more fretful and mildly delirious. The general condition was thus much less satisfactory. The strychnine was stopped and the gastric mixture begun again.

26th. Although he slept some five hours he had not such a good night as he was restless. He vomited twice but took fair quantities of fluids. The pulse and temperature remained high; his colour got worse and he was delirious at intervals.

Early in the forenoon a rapid change occurred. Vomiting became persistent; his colour got worse and his face became quite pinched. The tongue, previously moist, got very dry and the breath became 'fruity'. On several occasions the vomit consisted of mouthfuls of chocolate coloured mucus - at other times it was greenish in colour. He became markedly delirious.

The slough on the thigh had separated considerably and on the inner aspect of the thigh almost two inches of the adductor longus muscle were exposed; the subcutaneous tissues were evidently going to slough now. The foetor was less offensive; the skin was more undermined in the perineal region. The sound skin on the thigh was by this time only represented by a strip on the outer side

about four inches broad. This was due to retraction chiefly. The sloughing area had become much smaller in size; there was an extensive red raw surface.

The elbow joints remained in much the same condition. The conditions, whatever they were, had not advanced and there was not sufficient evidence to warrant incisions being made.

Towards the evening he went downhill rapidly and it soon was evident that he was going to die. He wandered considerably, the temperature reached 105.8° - it was reduced to 102° with sponging the chest - , the pulse became almost imperceptible at the wrists - 160. Considerable tenderness seemed to be present over the left patella and the knee was painful on movement but no swelling was visible nor was there any discolouration.

The heart's sounds became very weak and distant. Vomiting and delirium continued. His face got very pinched and his colour dusky. He gradually got quieter and early next morning became quite comatose. He died quietly at five o'clock on the 27th. in the morning. Before he died the breathing was irregular and he was quite pulseless for several hours.

There were no further indications of any joint troubles.

The albumen had not increased in amount.

Unfortunately a post-mortem examination was not allowed.

The history of this child is rather interesting. When he was born he was well nourished, healthy and weighed $9\frac{3}{4}$ lbs. He thrived very well for six months and then suffered acutely from wasting disease. During this time he got very thin and was not expected to live. This wasting continued for several weeks after which he picked up and made a good recovery. Since then he had been a healthy child.

About three months before he took scarlet fever his doctor treated him for a boil on the back of the right thigh. This boil seems to have been situated at the place where this gangrenous condition commenced.

The parents were unhealthy looking but moderately respectable. There was no history of tubercle, syphilis or other constitutional disease.

The boy himself was healthy, there were no evidences of tubercle, syphilis or rickets.

Culture revealed the presence of numerous *Staphylococcus Pyogenes Aureus* and a very few *Streptococci*.

With regard to the nature of this case. The condition commenced in a part which had evidently been the seat of some inflammatory condition three months before. The pin-head sized papule, visible in the centre of the patch at first, appeared to me to be either an aborted vesicle or an early vesicle which had become very early secondarily infected and had really not reached the vesicular stage. This is one possible cause of the complication. The condition was early regarded and treated as an infective process. The occurrence of the bluish black discolouration made one realise the degree of virulence of this infection. This appeared to be a haemorrhagic stage in the process. Early incision showed the discolouration to be haemorrhagic. It was doubtful at that time as to what was going to occur in the cellular tissues. The discoloured area soon became gangrenous. The process spread considerably and soon involved a large area. The size of the affected area, once separation began to take place, appeared very much larger on account of the retraction of the healthy parts. In the end the subcutaneous tissues tended to slough completely.

After a partial recovery, he became much worse. This was coincident with the occurrence of joint troubles.

The terminal stage appeared to be pyaemic.

The photograph was taken immediately before the exploratory incisions were made and shows the state of affairs fairly well.

The infection in this case seems to have been more virulent than in the first one. In the first case a localised gangrene occurred; in the second the infection went a degree further and caused a pyaemic condition in the end.

DIAGNOSIS.

There ought not to be much difficulty in the diagnosis of such cases. A guess can only be made in those haemorrhagic cases in which there is some ~~cutaneous manifestation or~~ haemorrhage from the intestinal tract or internal organs. The nearest one can get is probably a haemorrhagic type of one of the exanthemata, until vesicles develop.

The gangrenous type may offer some difficulty when the lesions only become ulcerative say two, three or four weeks after the vesicular eruption has come out and when the vesicles have apparently almost healed up. The history has usually to be depended on in these cases.

The presence of other cases in the house or the locality must help one also and there are frequently the remains of the crusts and scars to be seen. Pemphigus may cause a mistake to be made. Probably the diagnosis may be confused with Impetigo Contagiosa. Woodward got a leading dermatologist to see one of his cases and he made this mistake which he corrected only after reading up the current literature and the history of the case.

When these gangrenous ulcers are confined to the buttocks of a badly nourished infant the condition may be mistaken for a congenital syphilide.

P R O G N O S I S .

A guarded prognosis should be given in each case.

The haemorrhagic cases are very deceptive and one really cannot say what is going to happen. There never seems to be an excessive loss of blood but the prognosis seems to depend more on the situation than on the amount of blood lost. Some petechiae or a few intravesicular haemorrhages ought not to cause alarm but when petechiae, ecchymoses, effusions, bleeding from the stomach, mouth, bowel, kidneys or bladder are present and progressive there is good reason to fear a fatal issue.

Where superficial ulceration exists only the prognosis is usually good otherwise it varies according to the age, the number and degree of the lesions, the amount of constitutional disturbance and the presence of tubercular, pyaemic and inflammatory lung lesions.

It has been noted in several instances where extensive ulceration and severe constitutional disturbance were present, so much so as to make the cases appear almost hopeless, for sudden changes to occur for the better and rapid recovery to ensue. There is thus always some hope no matter how bad things may appear.

T R E A T M E N T.

General.

As most cases of gangrenous varicella occur in children who have been more or less neglected and improperly fed and as the more frequent lesion is one of more or less extensive gangrenous ulceration of the skin, superficial and deep subcutaneous tissues, dietetic, antiseptic and hygienic conditions of the simplest but most efficient nature are called for.

Provided the patient is not too acutely ill or in a state of collapse, open air treatment should be adopted. All such cases should be treated in hospital where suitable balcony accommodation is available. In my experience nothing seems to make a child sleep, rest and feed so well as a sojourn of several hours each day in the open air or entirely in the open air night and day. The most striking cases of this are those scarlet fever cases with foul septic throats and profuse discharges, and the measles cases with pulmonary trouble. The possibility of tubercular disease being present is another very good reason for adopting open air treatment.

I do not mean to infer that the fresh air has any direct action on the ulcerative process. The general condition is treated by this method and it is constitutional errors which seem in many to be the cause.

With regard to food each case must really be taken by itself. Where there is fever and constitutional symptoms a simple nourishing fluid diet is essential. Gastro-intestinal troubles are so liable to crop up in these badly nourished children and infants as to make cleanliness of the utensils and regular feeding very essential.

If no fever exists the feeding ought to be pushed so as to make up for past neglect and starvation.

A point must be noticed however in the feeding up process. As most of these are ill nourished they have most likely been accustomed to unwholesome food since birth. I have noticed several scarlet fever patients who only

wished to have "beer, dripping and bread" to eat. So that some caution must be exercised in giving food - much too rich for them and which probably they are quite unaccustomed to. Barley, albumen and lime waters are of much use in those cases either alone or to dilute milk.

Stimulants are frequently needed. Brandy is very suitable and useful. It may be given alone or beaten up with an egg.

Port Wine frequently answers satisfactorily as, besides acting as a stimulant, it tends to check diarrhoea.

Sherry or Port Wine Whey and Raw Beef Juice are also useful.

Quinine in one or two grain doses in milk every four hours and five grain doses every three hours of Sulphocarbolate of Soda were used by Crocker.

Sodium Salicylate has also been used.

Iron, Arsenic and the compound syrups are the best tonics.

Malt and Oil, Cod Liver Oil and Virol are all of use in suitable cases.

Opium probably acts best if any sedative be required for pain, restlessness, sleeplessness or delirium. Morphia with Atropine is best. I found small frequent doses of Hydrocyanic Acid of great use in allaying persistent vomiting.

Free cardiac stimulation is probably best obtained from hypodermic injections of strychnine in large doses.

LOCAL TREATMENT.

In those cases, where multiple gangrenous lesions exist, boracic acid baths or wet dressings frequently used seem to be of great service until the sloughing stage has finished after which a simple dressing acts suitably. Iodoform or iodol vaseline, Iodide of starch paste may also be used as dressings.

The most efficient of all however seems to be ichthyol either as an ointment or a watery or glycerinated solution.

I found a ten per cent ichthyol solution in glycerine of great use. As the local tissue vitality is probably particularly low antiseptics may do harm so that a normal saline solution may act very satisfactorily. Soothing lotions are necessary if there be much irritation of the skin or ulcers. Pasteur used a warm solution of chlorinated lime on lint to relieve. Other applications which have been used include, Carbolic Oil, Nitrate of Mercury and Boracic Acid ointments; Perchloride of Mercury 1-2000 to 1-5000; Boric acid ointment with Resercin (2-3 gr. to the ounce); Aristol, Boric acid powder with acetanilid (10-20 gr. to the ounce).

The most efficient treatment seems to be obtained from the use of Boracic Acid, or ten per cent Ichthyol in glycerine, foment until the sloughs have separated and then a simple ointment dressing or foment of normal saline solution.

Hydrogen peroxide is extremely useful for cleaning up the wounds and red lotion dressings seem to assist the healing process very considerably. Crocker also used subcutaneous injections of 1-40 carbolic solution round the base of the ulcer or gangrenous patch.

Above all good nursing must be secured. Much can be done to make these patients much more comfortable by a good nurse. A little coaxing and kindness do much often to cheer up.

If there be any tendency to scratch, the nails must be cut short and kept clean, and if necessary gloves and cardboard splints used. There is no

doubt that many lesions are made worse and fresh ones caused by scratching with dirty fingers. When restlessness, sleeplessness or mild talkative delirium are present a good plan is to tempt patients to go to sleep with hot drinks, cold applications to the head or chest and if need be cold tepid sponging of some part of the body - the chest, neck and face for preference.

A temperature above 103° ought to be brought down by sponging; these patients do not seem to stand high temperatures well at all.

The nursing difficulties are increased considerably in these cases with lesions round about the buttocks. If there be any intestinal derangement it means that dressings are much soiled and consequently further infection of the ulcers. Great care and frequent attention are thus necessary. Other conditions occurring in the course of the disease such as joint pains, pulmonary lesions, etc., must be treated, as they occur, on general principles.

Serum and vaccine treatment has not been used by anyone so far; it remains to be tried. How much success will attend the attempts it cannot be said but in certain cases it ought to be tried.

GENERAL REMARKS.

On reviewing the literature it is found that the views first recorded in literature by Hutchinson have not been materially altered although much has been added and many new points brought out since 1880. It is somewhat difficult with the subject in its present incomplete state to draw definite conclusions but some opinions may be expressed. The experience I had of varicella occurring in convalescent scarlet fever patients makes me confirm the opinions of previous observers that the attack of varicella tends to be severe. Fourteen, including the two cases described in this paper, out of seventeen patients, who took varicella, developed severe attacks.

It was very unusual to have two gangrenous cases occurring in the same ward within a few days of one another. This raises the question as to whether these gangrenous forms are infective and whether they should be isolated. Andrew's two cases occurred in a school epidemic in which twenty four boys developed varicella. Epidemics of the milder type of gangrenous varicella have occurred in schools, asylums and in dispensaries. The question of infectivity and isolation is well worth consideration under such circumstances, and specially if the type of the cases belongs to the same class.

No writer has so far drawn attention to any point or points which might lead one to be prepared for a haemorrhagic or gangrenous form developing. Some cases have exhibited marked prodromal symptoms, severe constitutional disturbance and a copious rash. These however may only mean a well marked simple attack and moreover most cases of the gangrenous type only present at first ordinary symptoms and an average rash.

With the haemorrhagic form it is however somewhat different because there are frequently prodromal signs of a haemorrhagic character. Probably the best that can be done at present is to prepare oneself for gangrenous lesions developing in a child who appears to be predisposed from any cause. This however does not help in those cases occurring in apparently healthy and

well nourished children, and specially when the onset is somewhat sudden.

The appearance of the rash may help to a certain extent. When pustules and discoloured crusts form early and a surrounding inflammatory areola presents, one is justified in suspecting that gangrenous lesions are about to form.

A large number of gangrenous and haemorrhagic cases occur during the winter months, December, January and February.

The most frequent constitutional signs seem to be a varying degree of fever, fretfulness and restlessness while general cyanosis is a more or less constant sign in the severer gangrenous cases.

The appearance of a scarletiniiform prodromal rash does not seem to have any diagnostic significance as it only appeared in the case reported by Staniforth. During the epidemic in my wards I saw four cases with well marked scarletiniiform rashes appearing during the earlier days of the varicella rash. My second gangrenous case showed such a rash during the whole illness so that there may be something in its appearance and persistence.

It has been noted by several observers that the ulcerative process goes on for a certain period and then for some reason, so far unexplained, ceases, the sloughing base separates and healing occurs rapidly. This period may be a critical interval in the disease.

Reference has been made by several writers to the appearance of the slough in the gangrenous cases. Thus Kieffer thought the slough was like the membrane found on a surface wound infected by the Klebs-Löffler Bacillus. The slough in my first case was very much like such a membrane but no bacilli were found on several cultures. The organism involved in the secondary infection, may belong to the same group as the Klebs-Löffler Bacillus but it so far has not been successfully cultured.

Goodhart and Kieffer seem to have put Varicella Gangrenosa in its most appropriate place in relation to Varicella when they said that it bears to Varicella the same relation as does Cancrum Oris to Measles, Rupia to the

syphilitic exanthem and Vaccinia Gangrenosa to Vaccinia.

The risk of gangrene occurring is present in all the exanthemata and there seems to be no apparent reason to separate varicella from the others. The same may be said with regard to Haemorrhagic Varicella. This form is as rare in Varicella as it is in the other exanthemata.

With regard to the association of tuberculosis and gangrenous varicella, there now seems to be sufficient clinical evidence to show that, while the presence of tubercle may be the cause of some cases of varicella becoming gangrenous, it is probably only a causal agent in that the tubercular toxin, acting on the tissues, causes them to be of particularly low vitality and thus renders them susceptible to any extraneous infection.

The earlier writers, with the exception of Hutchinson and Stokes, were inclined to believe that tubercle was the actual cause and certainly post-mortem and clinical evidences gave them support in their belief but there are now too many cases on record of gangrene occurring in apparently healthy children, in whom no evidences of tubercle were found, to support that earlier view entirely.

If tubercle were the cause of gangrene occurring in varicella then we ought to see many more cases of gangrenous varicella - in fact epidemics of it - because tuberculosis is so frequent in children at the 'exanthem' age. Barlow recognised this and therefore did not lay too much importance on the point when he found tubercular lesions post mortem so frequently in cases of gangrenous varicella.

One is probably justified in acquiescing with Payne's view that varicella, occurring in a child suffering from tubercular disease, is liable to become ulcerative.

Eustace Smith frequently found children in a very weak condition after an attack of varicella; a more or less quiescent tubercular lesion under such circumstances may become active and even cause a rapidly fatal issue. This probably occurred in Ashby and Wright's and Payne's cases amongst others.

Sequeira and Kieffer both agree that many of the deaths occurring during an attack of varicella or soon after it are really due to tubercle. In such cases this result may be brought about by the secondary infection of a pre-existing tubercular lesion in the same way as a secondary infection of a psoas abscess ultimately causes a fatal issue.

As the matter stands at present there seems to be some fairly close association between tuberculosis and the mildest form of Varicella Gangrenosa and to a lesser degree with the second form. Clinical evidence is however against any association between tuberculosis and the severest form of the gangrenous type while in the haemorrhagic types there has been no evidence of any connection with tuberculosis at all.

When this subject has been fully worked out it will probably be found that varicella bears a somewhat similar relation, but in a less marked degree, to tuberculosis as does measles.

Andrew's classification was quite a satisfactory one but I think Knowle's five classes might be reduced to three viz:-

(a) Gangrenous cases.

In this class we may recognise three types of cases as described.

(b) Haemorrhagic cases.

In this class may be included all cases which show only a haemorrhagic lesion.

(c) Haemorrhagic and gangrenous cases.

In this class we may include all the cases in which there is a haemorrhagic stage either before or accompanying the gangrenous lesions.

Through time it may be found necessary to rearrange the three types of the gangrenous cases because some are inclined to believe that the severest form has a pathology and etiology of its own. Undoubtedly there are grounds for such a belief.

B I B L I O G R A P H Y.

- Abercrombie. Trans. Path. Soc. London. XXXI. p. 333. 1880.
- Adamson. Brit. Jour. Child. Dis. p. 78. 1905.
- Adamson. Skin Affections in Childhood. p. 113. 1907.
- Alexander. B. M. J. I. p. 276. 1909.
- Allbutt and Rolleston. System of Medicine.
Vol. II. pt. I. p. 475 and Vol. IX. p. 66. 1906.
- Andrew. Trans. Clin. Soc. London. XXIII. p. 79. 1890.
- Ashby and Wright. Diseases of Children. p. 310. 1896.
- Baader. Quoted by Andrew and Knowles.
- Baudouin and Wickham. Quoted by Knowles.
- Barlow. Lancet. II. p. 751. 1881.
- Bateman. Cutaneous Diseases and on Fever. p. 207. 1814.
- Beardsley. Jour. Am. Med. Ass. LIV. p. 1784. 1910.
- Bellamy. Clin. Soc. Trans. London. XX. p. 195. 1887.
- Bérard and De Lavit. Quoted by Andrew, Knowles.
- Berouilli. Quoted by Rundle.
- Blackham. B. M. J. II. p. 1205. 1905.
- Bowley. Lancet. I. p. 987. 1885.
- Büchler. Amer. Jour. Med. Sciences. XCVIII. N.S. p. 265. 1889.
- Cantrell. New York Med. Jour. LXI. p. 86. 1895.
- Chapin and Pisch. Diseases of Infants and Children. p. 271. 1912.
- Churchill. Diseases of Children. p. 784. 1870.
- Clemmey. B.M.J. I. p. 474. 1895.
- Comby. Quoted by Rundle.
- Crocker. (Lancet I. p. 987. 1885.
(Trans. Med. Chir. London. LXX. p. 397. 1887.
(Diseases of the Skin. p. 330. 1893.
- Cronk. Quoted by Andrew.

- Denme. Quoted by Andrew, Knowles.
- Demarqué. Quoted by Knowles.
- Dieulafoy. Textbook of Medicine. Vol.II. p.1575. 1910.
- Drewitt. Lancet. II. p.751. 1881.
- Edwards. Arch. Ped. XX. p.570. 1903.
- Ehlers. Quoted by Adamson.
- Elliot. Med. Record. XXXIX. p.562. 1891.
- Elliot. Cleveland Med. Jour. IX. p.37. 1909.
- Evans. Diseases of the Skin. p.144. 1912.
- Fagge. Lancet. II. p.751. 1881.
- Fagge. Principles and Practice of Medicine. Vol.I. p.243. 1888.
- Fagge and Pye-Smith, Ibid. p.205. 1901.
- Freeth. B. M. J. I. p.679. 1906.
- Galliard. Quoted by Rundle.
- Gaucher. Diseases of the Skin. p.25. 1910.
- Goodall. Medical Annual. p.599. 1907.
- Goodall and Washbourne. Infectious Diseases. p.213. 1908.
- Goodhart. Diseases of Children. p.227. 1891.
- Goodhart and Still. Ibid. p.300. 1910.
- Griffith. Univ. Med. Mag. (Aug.) p.837. 1896.
- Habershon. Lancet. II. p.751. 1881.
- Hamann. Med. News Phila. LXI. p.362. 1892.
- Hatfield. Arch. Ped. XVIII. p.236. 1901.
- Haward. B. M. J. I. p.904. 1883.
- Henoch. Lectures on Children's Diseases. Vol.II. p.263. 1889.
- Hitschmann. Quoted by Adamson.
- Hoffmann. Quoted by Andrew, Knowles.
- Holt. Diseases of Infancy and Childhood. pp.936, 997. 1909.

- Horner. Quoted by Rolleston (J.D.).
- Hutchinson. Lancet. II. p.751. 1881, and
Med.Chir. Trans. London. LXV. p.1. 1882.
- Isola. Quoted by Rolleston.
- Jamieson. Med. Chir. Trans. Edin. VI. W.S. p.19. 1886-87.
- Ker. Infectious Diseases. p.195. 1909.
- Kieffer. New York Med. Jour. LXXXII. p.1. 1905.
- Knowles. Ibid. XCIII. p.876. 1911.
- Kreilich. Quoted by Adamson.
- Kreuzeder. Quoted by Goodall.
- Krjukoff. Quoted by Edwards, Kieffer and Rolleston.
- Labbe. Quoted by Knowles.
- Lipes. Quoted by Knowles.
- Lockwood. Arch. Ped. XIV. p.680. 1897.
- Matthews. B. M. J. I. p.269. 1901.
- Morris. Diseases of the Skin. p.553. 1911.
- Munro. Manuel of Medicine. p.51. 1911.
- Neal. Lancet. II. p.1163. 1891.
- Neech. Lancet. I. p.515. 1906.
- Oettinger. Quoted by Knowles.
- Osler. Principles and Practice of Medicine. p.128. 1910.
- Osler and McCrae. System of Medicine. Vol.II. 1907.
- Parker. Lancet. II. p.751. 1881.
- Payne. Lancet. I. p.987. 1885 and
Trans. Path. Soc. London. XXXVI. p.471. 1885.
- Pfaundler & Schlossmann. Vol. II. p.330. 1908.
- Phillips. B. M. J. II. p.1355. 1895.

- Plant. Quoted by Edwards.
- Porter. Lancet. I. p.1359. 1907.
- Roberts. Theory and Practice of Medicine. p.198. 1909.
- Rolleston, J.D., Med. Chron. XIX. p.215. 1909.
- Römer. Quoted by Rolleston.
- Rotch. Pediatrics. p.603. 1906.
- Rundle. Lancet. I. p.1692. 1906.
- Savill. System of Clinical Medicine. p.473. 1912.
- Schwartz. New York Med. Jour. LXI. p.86. 1895.
- Sequeira. Diseases of the Skin. p.154. 1911.
- Sheffield. Diseases of Children. p.321. 1911.
- Silver. Arch. Ped. XIV. p.684. 1897.
- Eustace Smith. Ibid. p.50. 1889.
- Spivak. New York Med. Jour. LXI. p.505. 1895.
- Staniforth. B. M. J. I. p.18. 1890.
- Stelwagon. Diseases of the Skin. p.398. 1905.
- Stern. Pediatrics. II. p.559. 1896.
- Stokes, Whitley, Quoted by Hutchinson.
- Stokes, Wm., Dublin Jour. Med. Science. 1880.
- Storer. Quoted by Knowles.
- Taylor. System of Medicine. p.79. 1911.
- Turner. B. M. J. II. p.716. 1898.
- Watson. New York Post-Grad. XXIII. p.442. 1908.
- Wheeler. System of Medicine. p.42. 1894.
- Whitla. Practice of Medicine. Vol.II. p.1817. 1908.
- Wright. Lancet. II. p.496. 1907.
- Woodward. Pacific Med. Chir. Jour. (San Fran.) XXXII. p.169. 1889.

Case 1

Varicella Gangrenosa.

Case II

Haemorrhagic and Gangrenous Varicella



