BULLOUS CONTAGIOUS IMPETIGO OF THE NEWLY BORN

(PEMPHIGUS NEONATORUM).

By

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The disease known as pemphigus neonatorum or bullous contagious staphylococcic impetigo of the newly born has shown in most countries a tendency to increase in incidence since the war. Almost all maternity hospitals and wards that have been open for any length of time have had at least one epidemic in the last ten years. Brennemann (1928) writes: "Hospitals in which impetigo neonatorum does not occur either have no maternity wards or else do not recognize the disease."

The disease is perhaps recognized more often than it was, and some stress has been laid on it in the Central Midwives Board Reports, 1927 and 1934. Attention has been drawn to it much more in the last ten years in American literature. However, the significance of a few bullous spots on an otherwise normal, healthy baby a few days old is often missed, and before the resulting epidemic has ceased one or more deaths may have occurred. As a disease entity it is really only seen by the personnel of the ward nursery. Paediatricians and dermatologists see relatively few cases. This paper is an attempt to summarize the more recent literature, and to stress the significance of the first lesion appearing in the ward nursery. A practitioner seeing a child with healing lesions discharged one or two days before from hospital will know that the wards of that hospital are dangerous to babies born there in the next few days or weeks. Irwin Rubell (1931) noted that in private practice cases

were frequently encountered coming from various hospitals, and he states that epidemics are more common than one would suppose as so many are hushed up. In this country the midwife in attendance is bound to notify the infected case to the local supervising authority at once, and to disinfect herself and her appliances to the satisfaction of that authority. The disease, however, still seems to be increasing, and the probable reasons are (a) failure to recognize the nature of the disease, (b) inadequate cleanliness and sterility in the maternity department, (c) failure to recognize the significance of the first case, even if diagnosed, and (d) the tendency to hush the whole thing up. More and more cases go to maternity hospitals nowadays, and often the attendance has increased while the building has not. Overcrowding is a very definite predisposing factor.

The two epidemics here described are of interest in that they present most of the usual features of the disease, and also bring out a few points hitherto inadequately stressed.

HISTORY AND BACTERIOLOGY.

Ochene in 1773 seems to have been the first person to have described the condition. Epidemics of pemphigus neonatorum were recorded in England as far back as 1834 by Rigby, in France by Leseque and Trousseau in 1850, and in Leipzig by Moldenhauer in 1874. In this year also Emile Widal noted that the bleb contents were self-inoculable and might be inoculated from person to person. Call gave one of the first accounts in America in 1904. Dohi and Dohi reported a Japanese epidemic in 1912.

In 1916 there were eight epidemics in Chicago alone (Falls).

In 1891 Almquist described an organism obtained from the vesicle which resembled S. aureus - a diplococcus producing a vesicular eruption on intradermal injection very unlike the follicular infection usually caused by this organism in older subjects - and suggested giving it the name "Micrococcus pemphigi neonatorum".

In 1900 Matzenhauer isolated a Staphylococcus aureus from the vesicles of cases of pemphigus neonatorum and impetigo neonatorum and insisted that they were the same disease. His observations were repeatedly confirmed. In the same year Sabouraud obtained a mixed growth of staphylococci and streptococci from a crusted form of the lesion and blamed the latter organism, and in the same year Bloch found staphylococci in the skin-lesion but streptococci on blood-culture of a fatal case.

In 1905 Whitfield described a case in which the date of onset was the 17th. day after birth, two days later the mother developing a lesion on the hand. Cultures in ascitic fluid gave a pure growth of streptococci from the child, and streptococci and Staph. aureus from the mother. Since then a large number of workers (Call (1904), Maguire (1903), Cole and Ruh (1914), Falls (1917), Belding (1926), Schultheiss (1923) and many others) found staphylococci, and the Staph. aureus became generally accepted as the usual causative organism.

Engman (1901), Falls (1917) and others have reproduced the lesion by direct inoculation into the skin, and recovered in pure culture the original organism. The latter worker claims to have fulfilled all of Koch's postulates. Whitfield (1903) noted the association of

acute pemphigus neonatorum with impetigo contagiosa of adults, and thought it strange that one should be a staphylococcal if the other were a streptococcal lesion. On insufficient evidence he took the streptococcus to be the causative organism. In the same year Adamson reviewed the literature, and thought that investigations by special cultural methods would reveal the primary cause as streptococcal.

At present almost all workers consider the lesion a staphylococcal one. Early workers thought it likely that the organism was a specific strain, for it presented unusual features. Clegg and Wherry (1906) examined an outbreak at Manila, and agreed with Almquist that the coccus was very similar to Staphylococcus aureus on culture, but differed in that litmus milk was coagulated in a week and no indol was produced in broth. They found that the diplococcal appearance seen in the direct smear from fluid from the bullae could be reproduced in milk and serum broth cultures. Auto-inoculation produced typical lesions within 30 hours. Most authors, however, think it likely that the organism is a non-specific Staphylococcus aureus. Falls (1927) draws attention to the frequency with which the disease is associated with other staphylococcal lesions - pustular acne, boils, etc. - and quotes a case where an instructor demonstrated engorged breasts in a newly born baby and expressed some "hexenmilch". A few days later a suppurative mastitis developed, which was opened and Staphylococcus aureus isolated. Twenty-four hours later typical small pemphigoid lesions developed on the adjacent chest-wall. He states in conclusion, "One may think that the Staphylococcus aureus, particularly

one which has been producing a skin-lesion, may, on contact with newborn skin under certain conditions, give rise to typical pemphigus", and "it is probable that Staphylococcus aureus from lesions other than pemphigus may produce typical pemphigus lesions and be the starting-point in an epidemic of this disease".

Krigbaum (1926) found a Staphylococcus in all his cases. He was unable to infect himself, other parts of the same patient or another child by rubbing a culture into the skin. Carter and Osborn (1936) found 17 different strains of Staphylococcus aureus and two of Staphylococcus citreus in their series of cases. Benians and Jones (1929) found nothing to suggest that the Staphylococcus aureus was of abnormal specific type, and nothing to suggest a filterable virus as a cause.

There is still much bacteriological work to be done. August von Reuss (1935) writes: "That we are not dealing here with a specific type of staphylococcic pyodermia, but rather with a manner of skin-reaction especially peculiar to earliest infancy is shown by the results of inoculation experiments; older children and adults after being inoculated with contents of pemphigus vesicles do not develop pemphigus but impetigo contagiosa instead. What we called pemphigus neonatorum is in no way a clinically uniform disease, but a disease group, the representatives of which exhibit the greatest differences in regard to the size and extension of the efflorescences, the contagiousness and the disease course." He states, however, that the epidemic form seen in England and the United States seemed relatively rare in Germany.

It is interesting at this point to note the confusion still present in the bacteriology and aetiology of the subject in the most recent text books on diseases of infancy. Porter and Carter (1938) still regard the condition as streptococcal, staphylococcal lesions being responsible for purulent folliculitis and allied lesions. Stone (1938) describes the condition much as we do in this paper but still retains pemphigus neonatorum as a distinct and separate entity although he notes that the border line between pemphigus neonatorum and bullous impetigo neonatorum is "very shadowy" and that some authors do not recognize any difference in the two conditions. He gives a mortality of 50% for the former condition and says that the latter is rarely fatal. He further subdivides exfoliative dermatitis, due usually to Staphylococcus aureus, noting that it is a dangerous and frequently fatal condition. Tow (1937) also recognizes pemphigus neonatorum as a separate condition and states that impetigo may be due to staphylococci, streptococci or (rarely) pneumococci. Pemphigus, he states, may be mild or severe. He devotes a separate section to exfoliative dermatitis but notes that many observers do not recognize it as a separate entity.

Text books of dermatology also vary considerably, but many now agree that pemphigus neonatorum and bullous impetigo neonatorum are the same condition and that the staphylococcus is the usual causative organism. The vast bulk of literature backed by careful bacteriological study is in favour of a (probably non-specific) strain of staphylococcus aureus being the usual causative organism.

The last word has not been said on the subject, but it is interesting to note what a wealth of speculative and immaginative pathology and false reasoning from incorrect premises led to the view that a streptococcus was the sole cause. Many authors thought it unlikely that a streptococcus should cause impetigo contagiosa in adults and a staphylococcus in infancy and assumed that the streptococcus "must be the cause". Relatively few aspirated intact bullae of a large number of cases from different epidemics and studied the growth of bacteria. Those who did discovered staphylococcus aureus in almost every case. A complicating factor appears to be that impetigo in adults is by no means always due to a streptococcus. Paul Tachau (1938) noted that while in Germany the cases he saw were usually streptococcal; now in the U.S.A. staphylococcal impetigo predominated in his practice. J.E.M. Wigley (1938) has noticed that in a Children's Hospital in London staphylococcal impetigo predominates.

Our knowledge of the staphylococcus is still very unsatisfactory and the factors governing pathogenicity incompletely worked out. Even if it is granted that the staphylococcus aureus is almost always isolated from the bullae of contagious impetigo neonatorum and that there is much evidence that it is the causative organism, it is still completely uncertain why it causes the disease in some cases and not in others. Infants in an environment abounding in all strains of staphylococcus may and probably will remain unaffected, yet one handling by a nurse who has been handling an infected child may cause the

typical lesion.

The skin of the newly born infant is very sensitive to irritation and infection and it appears particularly so to staphylococcal infections. It is of interest at this point to note that extracts from certain strains of staphylococci contain a substance which increases the permeability of the skin in experimental animals to invasion by staphylococci and other organisms (Duran-Reynals, 1933. 1935). It is a possibility that some such "spreading factor" may play a part in this condition. Further bacteriological studies are needed and the little work on virus infection of skin - so far negative in bullous impetigo neonatorum - verified and carried further.

EPIDEMIOLOGY.

Almost everything has been blamed as the starting-point of an epidemic: septic lesions on hands or face of nurses, mothers or visitors, breast abscesses, infected milk (Mellon et al., 1925), infected clothes, hands of the doctor infected by cases elsewhere or from cases of sepsis, nose and throat infections in attendants, etc. Midwives may start an epidemic by bringing in infection from district cases (Zechmeister, 1886). Labhardt and Wallart (1908) and others describe congenital cases starting epidemics. Cole and Ruh (1914) draw attention to infected vaginal tears as a source. Any staphylococcal condition in an infant may be followed by bullous lesions, as in the case quoted by Falls above, and the case in the first epidemic described in this paper.

It seems, then, that an epidemic begins (a) by infection by carrier from cases elsewhere, (b) sporadically

from other non-specific staphylococcal lesions. Once the epidemic is started new cases result usually from infection carried on the hands of the nurse or doctor from case to case, from contaminated laundry or from actual contact. Carter and Osborn consider the site of the lesion to be often an indication as to the mode of infection. If on hands or exposed parts of the babies the nurses' hands are probably responsible; if on the body the laundry is to blame. The mother herself may act as a carrier. Bowman (1936) considers visitors may carry the infection to the mother, who conveys it to the baby, neither of the first two suffering from any lesion. Rubell (1931) considers that the handling of the laundry is the most important single factor in controlling an epidemic. An over-hot, moist skin is more liable to become infected than a cool dry one, but there is no marked seasonal incidence. Neff (1929) found that the spreading depends upon a lack of aseptic precautions rather than the season. Almost any child may develop the lesions, but at Manila it was found that all cases of pemphigus were among the white infants; the coloured seemed to have a greater immunity. This seems open to doubt and lacks confirmation. A child may, if allowed inoculate himself in fresh places from the primary vesicle or vesicles. Scrubbing or otherwise maltreating the tender skin of the infant undoubtedly predisposes to infection, and Kringbaum (1926) has shown the bad effect of misusing liquor cresolis co. in the wards of a hospital.

PATHOLOGY.

The lesion is usually considered to be a superficial one, the effusion lying between the stratum granulosum and stratum corneum as in the other forms of bullous impetigo. There has not been much work done on the histology of the disease in childhood and, in fact, accounts of the histology of the normal skin over different areas of the new born are conspicuous in the literature by their absence. Carter and Osborn thought the condition essentially a "sub-epithelial pyo-dermatitis", the line of cleavage lying between the epidermis and dermis, and disagreed with Maguire (1903), who said that the exfoliation was due to horizontal cleavage of the epidermis between the stratified and the malpighian layers, occurring along the lower edge of the stratum lucidum. They found that sections of skin taken from stillborn or early neo-natal infants showed a very primitive condition, it being impossible to note differentiation of stratified and malpighien layers. The stratum lucidum was not apparent, the epidermis only two or three cells thick and the papillae primitive or absent. On their post-mortem and other evidence they placed pemphigus neonatorum with the other "pyodermatites" and concluded that the only difference between exfoliative dermatitis and subepithelial dermatitis was one of degree. They state "In folliculitis the infection is confined to the hair follicles and sebaceous and sweat glands, and in "pemphigus neonatorum" the organisms are situated below the epidermis where there is ample scope for rapid spread under the growing squamous epithelium."

We have quoted their work rather fully as it is one of a very few recent papers on the histo-pathology of the pyogenic skin infections of childhood. Their work came in for criticism, Muende (1936) remarking that the condition, if coccal in origin, should not be called dermatitis, and pointing out that post-mortem an artificial line of cleavage almost invariably results at the epidermo-dermal junction. If preparations were examined during life a flaccid bulla was found due to fluid collecting among the upper layers of the stratum Malpighii.

Dore and Franklin (1934) state that though bullae are usually superficial, starting between the mucous and horny layers, they may also be subepithelial and elevate the entire dermis. It seems to us that there is no good reason why this should not occur in bullous impetigo. Most lesions appear superficial and heal within a few days, but in the more severe spreading type with extensive flaking of skin there is what does appear to be a subepithelial dermatitis. The two conditions are definitely one, for we have seen the former develop into the latter. The content of the intact bullae is sero-purulent fluid, the cells being largely polymorphonucleus with some mononucleus and epithelial cells. Red cells may or may not be present.

CLINICAL PICTURE.

An infant a few days old - nearly always in the first 20 days of life - develops a small reddened area rapidly becoming bullous with clear fluid content, which turns slightly turbid in 12 - 48 hours. Falls considers

that a lesion that takes longer to develop into a bulla than a few hours is not pemphigus neonatorum. In most cases he has seen the initial red macule in a few hours become a clear bleb, which in turn becomes cloudy, bursts and leaves a red weeping surface. The skin around may be perfectly normal or slightly reddened. At the end of 48 hours or so the vesicle shrinks or ruptures, leaving a moist red area with shreds of vesicle wall adherent. The size of the individual lesion is from 1 mm. to several centimetres in diameter, but is usually from 0.5-2 cm. The lesions enlarge peripherally and may coalesce, but also come out in fresh crops on different parts of the body. After 3 - 12 days the lesions stop spreading and new crops cease to appear. In a typical case there is no crusting and no frank pus-formation unless there is superadded infection from other organisms - frequently streptococci. The whole disease process is usually over in 10 - 24 days, but may take a longer course if the lesions are very extensive, or secondarily infected. The skin is not scarred, and the infant's final appearance is exactly as before the infection. The lesions may appear anywhere on limbs, head and trunk, but the palms of the hands and soles of the feet are immune. Flexures and warm, moist areas generally fare worst, and it is here the lesions tend to spread and become contaminated.

After the first few days lesions in all stages of development may be present at the same time. A small individual bulla usually heals in about 5 days. Mucosae are not affected. There is more danger to the child in the first few days of life as the cord attachment may

become infected, and such cases are said often to develop a fatal septicaemia. Usually there is little or no systemic upset. The exceptions are:

1. The first case in a series often has a raised temperature, goes off its feeds and is generally unwell for several days.
2. Umbilical cord infections.
3. Very extensive and/or secondarily infected lesions.

The exfoliative type of lesion is most ugly. The infant looks as if it had been scalded all over. The skin is shed in large flakes, and the completely flayed child often dies.

As the lesions vary to some extent certain workers have classified them under different group headings. Thus McCandlish (1925) recognized three main groups: (a) Vesicles coalescing and rupturing, leaving large areas of exfoliation; (b) large discrete bullae (rare); and (c) small vesicles, which may coalesce but do not lead to the extensive exfoliation considered by many to be identical with Ritter von Rittersheim's disease. In addition to these three groups Dohi and Dohi (1912) and Poole and Whittle (1935) have noticed cases where the bullae have become covered with thin crusts. The latter authors suggest as the three main types - (a) the most common vesicular and bullous lesions without extensive exfoliation or coalescence, (b) the exfoliative type, (c) the crusted lesions mentioned above. In many epidemics the lesions tend to become less severe in later cases, and the first case of severe exfoliative type is followed by other cases showing the milder bullous lesions.

The latent period between infection and vesicle-formation appears to be anything from 1 - 6 days, usually 2 - 4, but Bowman considers 12 - 24 hours to be nearer the truth. In view of the infection by carriers the incubation period is hard to estimate.

It is of interest to note that while the disease is found usually in the newly born, older children can also become infected. This was so in the second epidemic described in this paper, and from France between the years 1925 - 1928 there came reports of epidemics of the same character in older children. Weiland (1922) described an epidemic where both mother and child developed the disease. The adults suffered little discomfort, and the lesions were much milder than those of their children. It seems that the older the subject, the shorter the course of the disease and the less effect on the patient.

PROGNOSIS

The mortality figures of different epidemics vary widely. This is not surprising, for the higher the number of immature babies, puny babies and infants in whom the cord has not separated the higher will be the number of deaths. The lesions in some epidemics are also much more severe than in others, and it has been noted that epidemics starting with a severe fatal case have a higher mortality than others Garot (1927), for instance, records an apidemic in which the first case was fatal, and 3 out of 9 patients died. Falls records a mortality of only 4 out of 100. Nursing care, and adequate asepsis, seem to keep the figures down. In fatal cases death may result from (a) septicaemia,

usually the result of a cord infection from a spreading area; (b) the severe exfoliative type of disease; (c) intercurrent infection; or (d) inanition. Groups (a) and (b) commonly co-exist, and death nearly always takes place within the first 6 days. In groups (c) and (d) death occurs later. Concurrent infections may rarely occur and complicate the picture, such as the staphylococcal conjunctivitis reported by Benians and Jones (1929) and Schultheiss (1923). The staphylococcal pneumonias at the end of the second epidemic reported in this paper are therefore of interest.

TREATMENT.

Prophylaxis. - Overcrowded, badly run wards and nurseries, with imperfect asepsis and lax rules regarding visitors are the most frequently affected by pemphigus. Glass partitions for every cot have been advocated, but the cost is usually prohibitive, and unless every cubicle has its own nurse and washing facilities epidemics would spread just the same. Visitors should view the infants from a reasonable distance and not handle them. Nurses should be told to report septic lesions as soon as they occur - whether on themselves, mother or child, and should be sent off duty if they have themselves such a lesion on the hands. A nurse with chronic nose and throat infection should be transferred to a department where there are no infants. Children should not be vigorously rubbed with towels, for a skin red and irritated is more subject to infection. Should any sepsis occur in child or mother a special nurse should be appointed to look after the patient and her laundry in isolation, and to look

after nobody else. All materials used in the nursery should be kept scrupulously clean, and tests should be made periodically of the efficiency of the sterilizers and the packing of drums supervised. The same rigid care must be observed in the theatre.

The management of laundry is all-important. The infants' laundry should be done in the department and not come into contact with articles from elsewhere. Rubell (1931) advised that upon delivery from the laundry the various garments should be sorted into separate bundles, labelled and put through the operating room sterilizers, the nurses using sterile forceps to remove the articles needed. The nursery should be kept at moderate temperature, and the child's skin should not be allowed to become overheated. Above all, the management of the department should be essentially one person's concern. These general rules are rudimentary, but it is the failure to observe them that results in epidemics starting and spreading as they so often do.

Specific prophylactic measures are less effective, and it is difficult to estimate their true value. Chadwell (1928) advocated thorough inunction of the child's body with 5% ammoniated mercury ointment, but a series of reactions caused most people to give this up. Even when using 2% ointment redness and swelling of body and face occurred. In a large number of hospitals in the United States a blend of vegetable and mineral oils with chlorbutanol was found to be effective. The child was cleaned with oil and anointed in the delivery room before transference to the nursery, and the anointment

was there performed daily. The buttocks were cleaned with soap and water and bathed with oil at each change of the diaper (Truett Gandy, 1935). This method seems more reasonable than the first. Winder (1934) used a bacteriophage prepared from Gratia H strain of Staphylococcus albus, washing the entire surface of the infant including the scalp with the bacteriophage after an initial oil bath, and gently massaging the surface for a few minutes. No drying of the skin was necessary as evaporation was rapid. The procedure was repeated five days after delivery. Of 56 girl babies so treated none developed impetigo; of 61 male babies used as controls 21 developed the disease. Epidemics are, however, not so common as to allow one to form an opinion of the true value of such methods.

Sanford (1937) studied a large series of cases under strict conditions of isolation and technical precautions, the only variable factor being the local treatment which was altered as different prophylactic measures were instituted. No seasonal variation was detected, but with progressively less prophylactic treatment the incidence of all pyodermal lesions dropped in proportion. Finally, with the practice of leaving the skin of the new-born entirely untreated, other than to remove cautiously any excess of blood at delivery, the incidence of impetigo was reduced practically to zero. Similar observations were made by Beane (1936).

Generally speaking, the more active the local prophylactic measures instituted the less effective have they been in lowering the incidence of impetigo in

the newly-born , and in some series of cases it has seemed that the measures taken to prevent infection have only served to increase it.

CONTROL OF AN EPIDEMIC.

As soon as the first case occurs, be it even one single lesion in a child otherwise fit and well, that child should be isolated and nursed by a special nurse, gowned and gloved, who nurses no other children. The laundry should be done in isolation. Should a second case occur it will be wiser to close the nursery for a fortnight, wash the walls and put all the staff off duty for several days. Everything in the nursery should be thoroughly washed and stoved. The healthy infants should be transferred to a different ward, and careful watch kept for the reappearance of further lesions. Should further cases occur it is wiser to shut the whole department and stop all admissions for 14 days. This is no easy matter in a department busy, crowded and overworked, but failure to deal drastically with the first cases leads to continuance of the epidemic. All nurses and mothers should be examined for sepsis, and the laundry and sterilizing arrangements overhauled. Visitors should be excluded. The affected babies are best kept away from the mother and fed on expressed breast-milk. The children should be isolated until the lesions are dry and healed and then should be discharged straight home.

Treatment of the actual lesion is less important. Provided that the infant does not rub affected areas on normal skin, and that the lesions are kept clean and dry, the tendency is for healing to take place. This natural

tendency has been responsible for many vaunted cures by a vast number of substances. Our own experience was the same as that of Poole and Whittle (1935) and Normark (1936) that the actual substance used mattered little, if at all. Substances used are Milian's solution (an alcoholic solution of brilliant green and crystal violet), 5% aqueous solution of glycerine (Kellert 1929), mercurochrome, potassium permanganate baths, 2% ammoniated mercury ointment (after touching with 95% alcohol), various dusting powders, boric baths, silver nitrate paints, hydrarg. perchlor. 1 in 6000 ex. aq., liniment. calaminae et ichthyol and countless others. Perhaps the best way of treating the lesion is first to aspirate some fluid for culture, then mop up the rest with a sterile swab to prevent auto-inoculation. Remove dead skin, dry the exposed area and dress with a mild antiseptic, followed by a dry dressing. Keep the flexures dry, but do not powder moist, exuding surfaces. Splint the arms, and try to stop the child from infecting itself afresh, though this will be difficult where flexures are involved. An alternative way is to dry and clean the area affected, apply a mild antiseptic, then cover with a firm adhesive dressing and le ave it covered. Cole and Ruh in 1914, and Swendson and Lee (1931) incriminate the wet treatment by ointments, which they consider unless and even harmful in that it favours spreading of the lesion. They favour cleaning out the vesicle and keeping the lesion dry,

A.K. Bowman (1936) advocates the following line of treatment, which he first used in the case of his own child, in a spreading acute phase with systemic upset

when other methods had failed. Remove all clothing from the child, rupture the blister and evacuate the contents. Swab gently with ether, and paint the bullae and the skin around for 1 in. with 3% mercurochrome. Immerse the baby bodily in 8% boracic acid and remove the dead skin by a firm movement of a sterile cotton tipped applicator. When all lesions have been dealt with, remove the child from the bath and wrap in an absorbent towel. This dries the surface rapidly without friction. A second painting with mercurochrome follows and the stained areas are covered with collodion. In the next 48 hours deal with any new lesion arising by painting with mercurochrome on its first appearance, allowing the lesion to progress to the bullous stage, then dealing with it as above. The child is bathed after a few days. The collodion over the unhealed lesions remains adherent; over those which have healed it comes away.

Additional help may be given by ultra-violet light (Gregorson, 1928), which seems to hasten healing and prevent or slow down further spread. We are inclined to think it helped in some of our cases. In response to a questionnaire, the Society of the Lying-in Hospital of New York stated that exposure to the Alpine sun lamp gave the most satisfactory results of many modes of treatment (Rubell, 1931). Vaccines have been used, both autogenous and stock vaccines, but there are few who advocate them now.

DIFFERENTIAL DIAGNOSIS

(a) Bullous congenital syphilide: The child is an ill, miserable one, very different from the (usually) healthy baby with pemphigus. Palms and soles and areas

round mouth and nose are affected. The Wassermann reaction is positive in mother and child.

(b) Drug eruptions - particularly the halogen group: These are not evanescent.

(c) Epidermolysis bullosa - an hereditary lesion following trauma - has been described, but it is rarely recognizable in the infant.

(d) Hydroa vacciniforme: A rare disease the result of exposure to light and almost entirely confined to males. It is rarely seen in infants.

(e) Dermatitis herpetiformis: The occurrence of this disease in the infant is disputed by many authorities. The bullous fluid in such cases is said to be sterile.

(f) Kaposi's varicelliform eruption. A rare disease occurring commonly on a pre-existing cutaneous condition, and complicating it. There is marked systemic disturbance, and bullae give a mixed growth on culture; streptococci are nearly always found. It is rarely or never found in the first few days of life.

The only conditions at all likely to be met with in differential diagnosis are (a) and (b). The rest are very rare.

CASE-NOTES OF TWO EPIDEMICS.

Epidemic 1.

First signs noticed on 13.iii.31, when 4 cases were reported.

1. Baby T-, aged 7 days. Normal delivery. Small bullous eruption containing turbid fluid on finger of right hand.

2. Baby S-, aged 6 days. Premature baby - approximately 34 weeks. Weight 5 lb. Bullous eruption with turbid

fluid content on the thumb.

3. Baby E-, aged 6 days. Normal delivery. Private ward. This child had had a nasal discharge for 3 days, and then developed an eruption on the left nostril.

4. Baby M-, aged 10 days. Normal delivery. Room 8. Bullous eruption both thumbs.

5. On 15.iii.31 Baby P-, aged 6 days. Normal delivery. General ward. Bullous eruption on fingers of the right hand.

By this time the eruption had become worse in babies T-, S- and E-. In Baby M- it had not spread, and this baby was discharged home.

On 16.iii.31 the eruption had spread extensively on Baby S-, the skin had peeled off, leaving raw areas which involved hands, arms, legs, scrotum and parts of the face and body. The child died.

Baby T- had now both hands infected, and Baby E- had scattered patches on face and legs. These 2 cases began to improve the next day.

6. On 18.iii.31 Baby I-, aged 7 days. Normal delivery. Room 8, and -

7. Baby B-, aged 7 days, presented slight lesions, and the next day (19.iii.31)-

8. Baby D-, aged 6 days, Normal delivery. General ward, also developed slight lesions, and on 23.iii.31-

9. Baby B_2-, aged 15 days. Cæsarian section - general ward, developed slight lesions.

Two days later all these cases were improving rapidly and no further cases occurred.

TREATMENT.

When first seen the lesions had been fomented - a bad form of treatment. Later ung. hydrarg. ammon. dil. was changed to 4-hourly potassium permanganate baths and a zinc-calamine-starch powder. All infected infants and their mothers were isolated and no new patients were admitted. A separate nursing staff was provided day and night, and separate gloves, gowns, soap and other articles kept strictly apart for each baby. Umbilical cords were sealed over with collodion dressing, and the thermometers completely immersed in spirit.

INVESTIGATIONS.

The nasal discharge of Baby 3 gave a pure growth of Staph. aureus as also did the swabs from the bullae of Babies 1, 2 and 6. Throat swabs were taken from all members of the staff and cultured. Only one, Nurse D-, grew Staph. aureus.

A daily inspection was made of the hands, arms and face of the nursing staff. On 16.iii.31 a very small septic spot was discovered on the finger of Nurse C-. On 21.iii.31 Nurse R- developed a whitlow. Both nurses were taken off duty immediately, and Nurse R- sent away on holiday.

NOTE.

No apparent source of sepsis was present at the onset but for the rhinorrhoea of Baby 3. No other cases of pemphigus were in the district. The nursing staff was fit and free from sepsis. The course of events seems to have been:

10.iii.31: Baby 3 commenced nasal discharge (S. aureus, pure culture).

13.iii.31: Babies 1, 2 and 4 developed small bullae, and Baby 3 a nasal bullous lesion.

15.iii.31: Baby 5 developed lesions. Babies 1, 2 and 3 worse. Baby 4 discharged home.

16.iii.31: Baby 2 died. Babies 1 and 3 worse. Nurse C- developed a small septic spot on her finger.

17.iii.31: Cases begin to show improvement.

18.iii.31: Babies 6 and 7 developed mild lesions.

19.iii.31: Baby 8 developed a mild lesion.

21.iii.31: Nurse R- developed a whitlow.

23.iii.31: Mild lesions in Baby 9.

No further cases.

EPIDEMIC 2.

There was no sign of any skin-lesion, on patients or attendants, and no evident source of sepsis until Mrs. C- on her 10th. day developed a slight mastitis, her temperature rose, and she became obviously ill. Her baby (No. 1) was taken from the breast, but 4 days later developed a bullous impetigo on the forehead with a rise of temperature, and signs of systemic disturbance (crying, refusing feeds, etc.). Culture from the breast and from the baby's lesion gave a pure heavy growth of Staph. aureus.

The eruption on the child appeared also on thumbs and extremities, and was worst in the flexures. Fresh crops of bullae, containing clear, serous fluid, developed over a period of 5 days. At the end of this 5-day period the fever abated and the child's general condition

began to improve on the 7th. day, at the time of the commencement of the ultra-violet light therapy.

There was no crusting; the skin came away in large flakes, leaving clean new pink skin beneath. Healing took 7 days, and the child was perfectly well by the 15th.day. The course of the eruption was divided into two 7-day periods, one of recurrent bullous eruption, each lesion tending also to spread by direct extension, attended by fever and malaise, and one of desquamation and improvement. The improvement was rapid when once it had started.

Two days before the onset of mastitis in the mother a small spot at the side of the child's nostril had been noted, but no rhinorrhoea. At this time the child was perfectly well. The lesion was not bullous, was small and not apparently septic, and was not in a site later covered by bullae. It was reported at the time by the nurse but its significance overlooked.

Treatment: Ultra-violet light- daily exposures, commencing with 1 minute - pulv. acid boracis with calamina, flavine and spirit and fissan dusting-powder. All bullae were snipped with sterile scissors and the contents mopped up. This child was isolated, its hands were bound, and attendant gloved and gowned.

Further cases. - Baby 2 had been discharged fit and well and on the breast the day Mrs. C- 's temperature rose, with the onset of mastitis. 48 hours later she returned - aged 13 days, with bullous lesions on the arm spreading to the forehead. Healing occurred in 10 days, and there was no systemic upset. She had been born the same day as Baby 1, in the same theatre.

Baby 3, aged 6 days, nursed by the same nurse as

Baby 1, developed bullous discrete lesions 4 days after Baby 1, on buttocks, back and arms. The lesions remained discrete and did not spread. There was no systemic upset and no fever. Healing was complete in 14 days. It had occupied the cot next to Bab y 1.

Baby 4, a twin, aged 11 days, developed a bullous eruption on the fingers 48 hours after Baby 3. The other twin, sharing the same nurse and in close contact, remained unaffected. The nurse had also nursed Baby 3, and the babies had been in adjacent cots. There was no fever, and healing was complete in 14 days.

Baby 5, aged 6 days, 72 hours after Baby 4 developed on the arm a bullous eruption which did not spread. No fever. This case was nursed in a private nursery by a private nurse, who never saw any other infants and only met the other nurses at lunch, when the nurse of Baby 4 sat next to her. All washing of woollens was done in the private nursery, and no utensils or clothes were used from elsewhere. Healing occurred in 11 days on pot. permang. baths and dusting-powders.

The nursery wards were now shut down, stoved, scrubbed, well cleaned out and the walls repainted. All the staff were put off duty for 24 hours. The main wards were closed to admission for 10 days.

Baby 6: A premature child, born when the department was closed as it was impossible for the mother to be transferred, as she had arrived without warning in labour. When aged 7 days - 8 days after the onset in Case 5 - his temperature rose to 100.6°, he went off his feeds and

became generally ill. The next day blisters appeared on the abdomen between cord and pubis, then spread round the attachment of the cord and down to the pubis and into the flexures. This case was the youngest of the series and the only one whose cord became infected. There was no nursery contact. His garments had never been used before and had never been anywhere near the other garments. Case 5 was, however, still in hospital in isolation, and was still unhealed. The two nurses only met at meals. After a long and stormy course, marked by fever and desquamation, the child made a good recovery.

Baby 7: Mrs. E- was admitted on re-opening, and baby was born the same day. Eight days later a typical bullous eruption appeared on chest, axilla and arm. There was no further spread and the lesions cleared up in 9 days. This was 12 days after the onset of the last case, which was, however, still unhealed, but in a far distant part of the hospital. There seemed no contact but for an occasional meeting of the nurses at meals.

Meantime, in the children's ward, in an entirely different part of the hospital, with its own staff and laundry, two cases of staphylococcal bullous impetigo had occurred, The nurses only met the other nurses at meals and off duty.

Case 8: A girl, aged 5 weeks, on her 7th. day in hospital (3 days after the onset in Baby 5) developed a typical lesion on both hands. There was no spread, no fever or upset, and recovery was complete in 9 days.

Case 9: A girl, aged 5 months, 72 hours after the onset in Case8, developed bullous lesions on the dorsum of both feet. The lesion did not spread, and cleared up in 5 days. She was in a cubicle next to Case 8, but separated completely by a glass and wood partition. The same nurse had been looking after both children at the onset of the lesions in Case 8.

These were the only 2 cases under 1 year of age in the children's ward at the time. There was no other case of skin-disease in the ward at the time Case 8 became affected, and no other Staph. aureus infections so far as can be ascertained. Sister had not noticed any such lesions before and none have been observed since.

The nurses were examined at the onset and visitors excluded. No signs of sepsis were found and no other possible source of infection discovered.

No further cases occurred until nearly 2 months after the onset in the last case (Baby 7), when Baby 10 developed a transitory temperature and a bullous rash on the right ear, then on the right side of the face and forehead. Within the next 4 days Babies 11, 12 and 13 developed typical lesions of a mild variety one after the other unassociated with any general upset, and healing within 10 days. All were being attended by the same nurse in the same nursery.

Baby twin G -, aged 9 days, then developed acute respiratory symptoms and died the next day. Next day Baby twin T-, aged 6 days, developed identical symptoms and died in 30 hours. Both were premature, puny infants, and difficulty with feeding had been experienced. Baby G- had erratic respiration and periods of cyanosis from birth.

Neither had any bullous skin-lesions.

At autopsy Baby Twin G- was found to have a staphylococcal pneumonia and a small tentorial tear with some blood-clot. There was no congenital cardiac abnormality. Baby T - had a similar pulmonary condition. Staph. aureus abounded on section and smear, and the culture growth was pure and very heavy. Both were of a type of pneumonia quite different from the usual, and no such pneumonias had been seen before in the department. Clinically they were of a fulminating type and marked by cyanosis. The pneumonic process consisted of many large deep purple-red patches of consolidation, very firm to the touch, up to 1 in. in diameter, occurring both sides and essentially of a broncho-pneumonic type. Congestion was marked. No naked-eye suppuration was noted, and though the lungs teemed with Staph. aureus and many polymorphonuclear leucocytes, there was no true abscess-formation. Both were considered to be cases of fulminating staphylococcal broncho-pneumonia.

The ward was closed for 14 days. No further cases of pemphigus or pneumonia occurred.

INVESTIGATIONS.

Cultures of the breast of Mrs. C- and her baby's lesion both gave a heavy pure growth of Staph. aureus, and cultures were taken from all subsequent cases unless the bullae had burst and the lesions dried. All gave Staph. aureus on culture. In most of the cases the fluid was withdrawn from the bleb and inoculated directly on to the medium. In 2 cases in addition to this the exudate from the deeper skin layers was cultured - as advocated

by H.G. Adamson - but only Staph. albus grew. No streptococci were isolated in any lesion. Swabs of all the nurses' throats were taken in the later stages of the epidemic, after the ward had been first shut. Only one gave a good growth of Staph. aureus; two several colonies but not a profuse growth. Occasional swabs taken previously from nurses handling the earlier cases had been negative. None had rhinorrhoea nor any septic conditions of face, neck or hands. The only other person entering the wards was the obstetrical house surgeon, whose swabs were negative.

After the two deaths from pneumonia I took cultures from anything and everything in the department, and found that the "sterile" wool from the drums was contaminated with Staph. aureus. I repeated this several times with different drums. Some were negative, but many gave a heavy growth of Staph. aureus. All other drums in other departments were negative but for one in the children's ward which gave one positive culture. The pledglets of wool, as used for cleaning the noses, after being rolled by the nurses' fingers gave no heavier a growth than wool taken direct from the drum.

I then took swabs from the noses of all the newly born children every day for the first 6 days of life, and from all other babies in the department. Every one over the age of 4 days gave an extraordinarily heavy growth of Staph. aureus. Positive cultures began to appear from the second to the fourth day after birth and growth was profuse by the fifth day. The nose in the first 48 hours of life did not give any growth.

The sterilization, packing of drums and all else was overhauled thoroughly. The positive cultures fell away, and 3 months later only 1 out of 3 nasal swabs gave a fair growth - much less than the profuse growth previously obtained. Growth was usually scanty or negative.

Throat swabs varied, but usually gave a few colonies of Staph. aureus or none at all. Several gave growths of haemolytic streptococci, sometimes profuse, usually moderate.

TREATMENT.

A variety of treatments was tried. No particular treatment seemed appreciably better or worse than any other, though it was felt that ultra-violet light might have helped. Flavine and spirit, mercurochrome, pot. permang. baths, dusting powders, etc. seemed of equal value, Provided the blister was snipped, the contents mopped up and the part kept from coming into contact with other parts, the natural tendency was to get well.

COMMENT ON CASE HISTORIES.

Epidemic 1. - The children's throats and noses were not swabbed, and it is now known whether the wool used in the nursery was as sterile as it was presumed to be. No other children had had rhinorrhoea, and there was no sepsis in the wards in babies, mothers or attendants. It is perhaps permissible to assume that the rhinorrhoea in the first baby caused auto-inoculation in the shape of a bullous impetigo while the same organism was being conveyed from nurse to nurse and so to the other children.

The lesions were all on exposed parts, suggesting infection by the hands of attendants. Two nurses developed septic fingers during this time, but neither was swabbed. It is uncertain what connection this had with the epidemic, if any. Possibly they became infected from the children.

The epidemic has a stage of increment and decline, with the death of Baby S- at the peak. The appearance at death was that of an extensive exfoliation. No autopsy was performed. Baby S- was the only premature child in the nursery at the time.

Staphylococcus aureus only was cultured from the lesions. Streptococci failed to grow. The nasal discharge of Baby E- gave a pure and heavy growth of the same organism. None gave any cultural differences from any of the usual strains of Staphylococcus aureus.

Epidemic 2 - This really consists of two separate waves of infection, with a clear 2 months between. The origin appeared at first sight to be the mastitis in Mrs. C -, but at this time her child had a small spot on the nostril and the laundrywoman had a rash on the neck, for which she was being treated. Presumably also the swabs were contaminated. There was no sign of sepsis among the nurses, but one had a naso-pharyngitis and later gave a fair growth of Staphylococcus aureus from a throat swab. There were, therefore, several potential sources of infection.

Baby 1 possibly became infected from the mother, but by this time Baby 2 had shown lesions. It is possible that (1) the same unknown source started all 3 cases, (2) that the child infected the mother, then the mother the child, or (3) the mother infected the child as first

thought, Baby 2 being infected via the nurse. However, Baby 1 developed his first bullous lesion on the face and was seriously ill for a time. Such a case is often the first one in an epidemic, and probably further infection was from this case. It seems most likely that the mother's infection started the epidemic, and it is possible she was infected by the small spot on the child.

After this the cases were probably from carriage, by nursing staff. Baby 6 almost suffered the common fate of any infected premature child, but recovered. The time-intervals increased, so the infection must have persisted in some carrier form. Two casesof milder type appeared in a different ward in slightly older children. All this time strict isolation, gloving and gowning was carried out, but failed to stop infection.

Two months then elapsed, after closing of the ward, before Baby 10 developed his first lesion with slight systemic upset. Spread to the others was via the nurse, the time-interval 24 hours in each case. 24-28 hours later Baby Twins J- and T- commenced a fulminating staphylococcal pneumonia, which caused death presumably before the stage of frank pus-formation wa s reached. Again the ward was closed, and since then no cases have occurred. The only organism grown throughout was Staphylococcus aureus. There was nothing to suggest a specific strain. The serous exudate from the cleaned area gave a growth of Staphylococcus albus only - no streptococci grew at all.

Several points of interest arise. The only infected wool was in the two wards where cases occurred,

but which were also the only two wards containing children under 6 months of age. The growth of Staphylococcus aureus in the noses of the infants at the time was excessively heavy as compared with swabs from elsewhere and from the same wards 3 months later. I attribute this growth to contamination of the "sterile" wool used in cleansing the noses, and the two deaths from pneumonia to the same cause. C.M. Smith (1935) obtained very similar findings, for four fatal cases of staphylococcal pneumonia occurred in rapid succession - usually after a 24 hour interval - and profuse growths of Staphylococcus aureus were obtained from infants throughout the wards, which Wiseman considered to be most unusual and to indicate a widespread Staph. aureus carrier condition. Throat swabs in his cases, as in ours, gave mixed cultures and less heavy growths. Controls of his cases also gave similar results to ours. Dickie (1938) also obtained similar findings in an outbreak of acute staphylococcal pneumonia in one ward of a London Hospital, all other wards being unaffected. Seven cases occurred with two deaths. All gave a heavy growth of Staph. aureus from the upper respiratory passages and in all cases the dominant symptoms had been nasal obstruction with rhinorrhoea. A control series also gave similar results to ours.

It is not unreasonable, therefore, to connect up these facts. Other staphylococcal lesions are known to co-exist with pemphigus neonatorum; possibly the carrier condition was here behind both. Another point of interest is that when the infection reached the children's ward only two children became infected - the only two

there under six months of age. In both the illness was very mild. The infection was probably carried by the nursing staff. The contamination of the wool here is interesting, but of doubtful import. The children's noses were not swabbed.

This epidemic stresses the value of completely overhauling the whole department when a succession of cases of pemphigus neonatorum occurs, and of shutting it for 14 days. During this time everything from the house officer to the wool used shouldbe thoroughly examined and the wards washed, stoved and if necessary painted. If such measures - together with rigorous overhaul of the laundry - are not instituted the infection may live on for months or even years (Carter and Osborn). The staphylococcus is one of the most resistant non-sporing of organisms. Dried on threads it may retain its vitality for 3 - 6 months and from dried pus it has been cultivated after 2 - 3 months (Topley and Wilson 1936). It is interesting to note that in 1888 Wichmann found organisms isolated from bullae were viable on dry threads after 1½ months. Lastly, if it is really necessary to push pledglets of wool into infants' noses to clean them it should be certain that the wool is sterile, and the nurses' fingers that roll them as clean as possible.

FURTHER OBSERVATIONS.

Since observing these two epidemics I have been allowed to visit the wards of the City of London Maternity Hospital, Queen Charlotte's Maternity and Isolation Hospitals and the Maternity Wing of the Royal Northern Hospital whenever doubtful cases occurred. When

bullous skin lesions occurred in the wards of Paddington Green Children's Hospital, Great Ormond Street Children's Hospital, the Children's Ward of the Royal Northern Hospital or Queen's Hospital, Hackney, the Honorary Staff of these hospitals kindly allowed me to see the cases. As no true epidemics have occurred cases have been relatively few in number and have occurred singly or in groups of two or three, usually in direct or indirect contact with each other. Further bacteriological study has been extremely limited as the bullae are so rapidly broken and contaminated. In some half dozen cases where I have been able to aspirate intact bullae the findings have been as above. Swabbing of broken bullae has resulted in mixed and heavy growth, frequently with heavy growths of streptococci of different types. In general three types of pyodermia are seen in infants admitted to the wards of Queen Charlotte's Isolation Hospital, a folliculitis or frankly suppurative lesion giving a mixed growth similar to that of a broken bulla, a bullous impetigo giving a pure growth of Staphylococcus aureus, and deep seated abscess formation. All have been noted to occur with staphylococcal lesions in the mother - frequently mastitis and breast abscess - but we have not noticed bullous and other lesions all developing from a single source at one time. Little seasonal incidence has been noted as the cases have been scanty and in small groups, but the summer months gave slightly higher figures for admission of infected children. There have been no deaths therefore no autopsies have been available. No cases of so-called "congenital pemphigus"

(Labhardt and Wallart 1908; Reed 1929 and others) have been seen. No artificial infection by bullous fluid has been attempted owing to the danger of spreading infection, but on three occasions when monsters were born we inoculated them onto, into and under the skin with staphylococcus aureus from lesions of mothers then in the wards. In no case were bullae produced, the lesions being small abscesses. when a Staphylococcus aureus was introduced into and under the skin , no lesion being obtained when the organism was externally applied. Further expermental work will be done when conditions are favourable.

As regards the older children; bullous lesions were occasionally seen in children between the ages of 1 - 10 years. The lesions responded rapidly to treatment, there was no spread of infection and the lesion was not associated with systemic upset. A pure growth of Staph. aureus was obtained on every occasion. This absence of systemic upset in children past infancy is illustrated well by case 5 in William Hughes' (1938) paper on staphylococcal infections in Singapore. It seems that the older the subject the less likely is systemic upset likely to occur and the more easily treated are the lesions. The source of infection was always uncertain. Ormsby (1937) notes that in children pyogenic nasal infection is often present but though this question was not investigated fully by us in these older children the observation is of interest in view of the findings in the second epidemic reported. In no case were cases of pemphigus neonatorum reported in the homes.

CONCLUSIONS.

It seems that pemphigus neonatorum is a form of contagious impetigo. The lesion is essentially at first a bullous one. While in the adult impetigo is said to be nearly always a streptococcal lesion, in the newly born it is due,in most cases at least, to a staphylococcus, usually Staphylococcus aureus. The organism is not a specific one; the condition is essentially a skin,condition and not a specific infective disease, although it may appear so in an epidemic. It is fatal in weak, puny and premature children, and the spreading exfoliative type is the most dangerous. Infections of the cord are usually fatal. Similar staphylococcal bullous lesions occur in older children and have been reported in adults. In this older age group there is less likelihood of a systemic reaction than in infants and the lesions appear more benign and responsive to treatment.

Treatment is essentially of the epidemic and not of the individual case. Concurrent infections may occur, and a carrier condition in the noses of infants may play some part. Articles of clothing and hands of the nursing staff are the most important factors in spread of the infection. Midwives, doctors, visitors and nurses have all been shown to have acted as carriers.

An epidemic starts either from outside, from infection brought in on the hands of one who has been in contact with a case, from within from a source of sepsis on child, nurse, mother or visitor, or from no apparent source for no apparent reason. Its appearance calls

for drastic overhauling of the whole department as mentioned above, and one should not wait until several cases have occurred for the infection spreads rapidly, and case follows case in from 12 to 36 hours, though where contagion has been more indirect the time interval is longer. Although the first lesion in the first case may arise from infection other than pemphigus neonatorum, once the bullous lesion occurs other cases are almost bound to follow if strict measures are not immediately adopted. The bullous lesion appears to be the highly contagious one, and should be the warning sign in a maternity department.

The disease is deceptive, for so many cases are mild that insufficient attention is paid to them until several cases have occurred, possibly with one or two deaths. It is not among the list of certifiable diseases, but the Central Midwives Board have drawn attention to it and ordered every midwife to notify cases at once to the local supervising authority, and to disinfect herself and her appliances to the satisfaction of that authority.

SUMMARY.

(a) Two epidemics are described of pemphigus neonatorum (impetigo of the newly born). The staphylococcus aureus was found in all cases, while attempts to grow streptococci failed. The fluid effusion from the cleaned infected areas grew only Staphylococcus albus in two cases.

(b) One epidemic appeared to be started by an infant with rhinorrhoea, from which a profuse pure growth of Staphylococcus aureus was obtained. The origin

of the second was obscure, but followed a mastitis in the child's mother. This also yielded a pure, heavy growth of Staphylococcus aureus.

(c) The organisms were not of any single strain, and appeared to be indistinguishable from the usual strains of Staphylococcus aureus.

(d) Two cases in a milder form appeared in slightly older children in the Children's Ward in a different part of the building and subsequently other apparently sporadic cases in older children have been seen.

(e) Two deaths occurred at the close of the second epidemic from fulminating staphylococcal pneumonia. The association of this and the skin-condition with heavy nasal contamination by contaminated wool is discussed.

(f) Attention is drawn to the need for thorough overhauling of the ward, nurses and laundry on appearance of the first cases. The carrier condition in infants and nurses is discussed.

(g) The lesion is essentially a bullous one, and the contents of the bullae are highly contagious.

(h) All premature babies and babies under 6 days old arein grave danger in an epidemic. The mortality in such infants is very great.

(i) The lesions usually cause no systemic upset, unless large and spreading and of exfoliative type. The first case in a series arising from it tends to show systemic disturbance.

(j) Treatment is essentially prophylactic, and the control of the epidemic is by general means. The treatment of the lesion is of less importance. Different treatments

are discussed.

(k) Attention is drawn to the fact that the conditions of pemphigus neonatorum, bullous impetigo neonatorum and staphylococcal exfoliative dermatitis appear to be all one, the latter being the most dangerous form.

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BULLOUS CONTAGIOUS IMPETIGO OF THE NEWLY BORN

(PEMPHIGUS NEONATORUM).

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