V O L U M E II.

ANAPHYLAXIS and SENSITISATION,

with special reference to the skin and its diseases.

T H E S I S

submitted by

ROBERT CRANSTON LOW, M.B. Ch.B.

for

the degree of M. D. Edinburgh University.

March 1924.

C O N T E N T S .

VOLUME II.

DERMATITIS VENENATA.

Under Dermatitis venenata are included all the forms of dermatitis due to external irritants. This form of dermatitis only affects certain individuals. A chemical substance or plant, which causes a dermatitis in one individual, may be handled by another individual with impunity. This special susceptibility of certain individuals to certain substances used to be put down to an "idiosyncrasy" on the part of the individual. It will be shown that this idiosyncrasy is really a sensitisation of the skin. It explains why one person is affected by a substance which has no effect on another and why a man may work, for example, as a joiner for many years and quite suddenly suffer from a dermatitis every time he handles certain forms of wood such as teak. I do not propose to describe the appearances of the eruption as these are well known, but will confine myself to the mechanism of the production of the sensitiveness, the nature of the substances involved, whether it is local or general, and whether desensitisation or immunity can be produced. Before discussing these questions I will describe a series of experiments which I did on myself and others to see whether /

whether individuals could be artificially sensitised. I used the Primula obconica (Plate 23.) because it is one of the plants which most frequently causes a dermatitis venenata.

Before starting to experiment with myself I could handle the Primula and rub the leaf on my skin with impunity. I scraped the skin of the back of the left forearm with a scalpel over a small area of about one inch by half an inch till a moist oozing surface was produced. Into this area the crushed leaf of Primula Obconica was vigorously rubbed and the juice allowed to dry on. About twelve hours later the part was thoroughly washed with soap and water. The area crusted over and healed completely in about two weeks leaving a very superficial scar. At no time was there any itching nor inflammatory reaction beyond what is normal in a traumatic lesion.

Three weeks after this inoculation the unbroken skin of the same area on the left arm was again rubbed with the crushed leaf of the same Primula and the juice allowed to dry on. This was done at 8 p.m. and on going to bed at 11 p.m. there was no change on the skin. I was wakened at about 4 a.m. with intense itching of the arm and the skin of the rubbed area was red and slightly swollen. Next morning the whole rubbed area was redder, more oedematous and still very itchy. The centre showed numerous

minute/

minute vesicles with a wide erythematous area around. This erythema gradually spread and Cast (16.) Plate (24.) shows the condition after 36 hours better than any description I can give. Cast (17.) Plate (25.) shows the reaction at six days, Cast (18.) Plate (26.) at ten days, and Cast (19.) Plate (27.) shows how the eruption slowly faded. At the end of ten days it was at its maximum after which it gradually subsided.

I would also draw attention specially to Cast (17.) which shows a central very inflamed area, then a ring of less inflamed skin with a further more inflamed area outside that. This type of reaction did not occur so markedly every time I rubbed on the Primula but it has occurred more than once. It is probably due to the same cause as is suggested to explain the corymbiform arrangement of the rash in some cases of syphilis, viz. that there is an area of antianaphylaxis or antisensitisation round the central zone.

The itching was very marked and came on at intervals usually being worst in the foremoons, evenings, and when I became warm in bed at night. If the area was rubbed (and one couldn't help doing so) the lesions flared up and became very red again. It was only after 28 days that the skin went completely back to normal.

Later/

Later the skin of the other forearm and of the thigh was rubbed with the leaf of Primula Obconica with the same result except that the reaction was slightly less intense and did not last quite so long (only 20 days). Cast (20.) Plate (28.) shows the reaction on the second day on the right forearm. The right forearm was rubbed before the reaction on the left arm had completely subsided and within 24 hours after rubbing the right arm the original area on the left arm became distinctly redder and itchier for a day or two than it had been i.e. a focal reaction had occurred. This focal reaction did not occur when the thigh was rubbed but by that time the arm reaction had completely disappeared. Thus I succeeded in sensitising my skin to the P. Obconica. The sensitisation was a general one although the original inoculation was only made on a very small area.

The second experiment was done on my brother, who also was not previously sensitive to the Primula. I wished to try to immunise him by inoculating him several times at intervals of 24 hours on the same principle that antitoxine is now given by repeated small doses so as to avoid anaphylactic symptoms. His left forearm was scraped in the same way as my own and the crushed leaf of Primula Obconica rubbed in and allowed to dry on. A smaller area was scraped, only one quarter inch in diameter. Similar small areas were/

were scraped and rubbed with the Primula on five successive days at intervals of 24 hours and twice again at intervals of 48 hours i.e. seven times in nine days. The scarified areas were all placed quite close to one another all within an area of 1½ inch square. None of these areas showed any itching or reaction beyond slight redness round them. Then I waited for three weeks, as in my own case and at the end of that time rubbed the inside of the left forearm with the Primula. For three days nothing happened and I was congratulating myself that I had succeeded in producing an immunity, but on the fourth day the skin of the rubbed area became very red and itchy, and a reaction exactly the same as was produced on my own arm appeared and ran an exactly similar course for nearly four weeks with redness, itching etc. Thus I had produced a sensitisation a second time, but the extraordinary thing about it was the long incubation period of four days before the reaction began. In my own case the reaction came on within 12 hours and evidently the different method used for the inoculations was the cause of the delayed reaction. Six weeks later I again rubbed my brother's arm with the Primula and again he reacted as before but this time the reaction began to appear in about 12 hours and a year later, on again testing him, he gave a similar reaction within 12 hours.

so/

so that now his skin is in the same condition of sensitisation as my own.

Since doing these experiments on my brother and myself, other six individuals (all medical men or students) were inoculated in the same way as I was myself, but in no case did I succeed in producing any sensitisation. In three of these cases the experiment was done twice at intervals. One of these individuals had a distinct family history of asthma and epilepsy, another had suffered some years ago from serum sickness after a second injection of serum given one year after the first. In a third I saw the mother with a typical dermatitis venenata which was proved to be due to Primula Obconica. In the fourth there was a family history of asthma. In the other two there was no history of asthma, urticaria or eczema to be obtained. In my own case there is a history of asthma on my mother's side, and my mother suffered many years ago from a Primula dermatitis. Therefore out of eight cases altogether two of whom sensitised there was a distinct family history of asthma or other sensitisation phenomenon in six of them.

Sensitisation once produced seems to last for a very long time, probably for life. It is now over two years since I became sensitised and I still react as before.

My/

My brother also reacted more than a year after he was sensitised. HALL[1059.] mentions a patient, sensitive to Primula Obconica, who avoided contact with the plant for over 20 years and on touching the plant accidentally at the end of that time suffered again from a dermatitis. Therefore once an individual's skin becomes sensitive to any substance, it probably remains so for the rest of his life unless he is artificially desensitised. One attack of dermatitis does not make the individual any less sensitive. On the contrary he seems to become more so, especially if the attacks follow each other closely.

So far as I can trace, the only other experiments on artificial sensitisation to the Primula were done in 1904 by NESTLER[1099.] in Germany. He rubbed his own skin with different parts of the plant to see if they irritated. He does not state definitely that he could handle the Primula with impunity before he began his experiments, but leads one to infer that he was not affected by it. He rubbed the unbroken skin of the left forearm repeatedly at unstated intervals with the leaves and stalks of the plant and left the juice on for three days. He also fixed a part of the plant on to the skin with sticking plaster for some hours at a time, and it was only after 14 days of experimenting thus that he got a marked reaction to the juice of the plant/

plant. In this last application the dermatitis started 40 hours after the plant was applied and from his description was very similar to that which I produced on my own arm. He also at the same time developed a dermatitis of his fingers because he was still handling the plant as he used to do.

Therefore there can be no doubt that he had sensitised himself, and the interesting point about it is that the sensitisation took place through the unbroken skin. The other interesting fact is that it was only after 14 days of rubbing in the plant at intervals that he became sensitive. That corresponds fairly accurately with the time taken to sensitise animals to a protein viz. about 10 days.

NESTLER also describes several cases of persons who had handled the Primula with impunity for years and who suddenly became affected. Among these was a young girl into whose left forearm he rubbed the juice of the primula leaf. As there was no reaction, 6 days later the same area was again rubbed as before and 10 days after that a severe itchy dermatitis with blistering occurred and lasted several days. NESTLER does not give details but I presume the juice of the leaf was left in contact with the skin and not washed off during that time. Here again it took 16 days from the time of the first rubbing before a reaction began/

began. In yet another case in a man, a dermatitis occurred after the sixth rubbing with the primula leaf at intervals of several days. These three sets of experiments of NESTLER'S therefore have definitely proved that the juice of the primula can penetrate sufficiently through the unbroken skin to cause sensitisation. Also if the juice of the plant can penetrate in sufficient quantity through the unbroken skin of a sensitised individual to produce a reaction, then there is no reason why it should not penetrate the skin of a normal individual in sufficient quantity to produce sensitisation.

NATURE OF THE POISON PRODUCING THE DERMATITIS.

NESTLER found that the dermatitis was due to the yeelowish-green fluid on the fine hairs of the leaves and stalks. He found that this fluid contained numerous rhomboidal crystals but did not investigate its chemical composition.

//33.

SIMPSON in America extracted the leaves of Primula Obconica and found that in addition to protein they contained an oleo-resinous mixture. The protein part, when isolated and rubbed on a susceptible skin, produced absolutely no reaction. Whereas the oleo-resinous/

resinous compound, free of protein, gave a very marked reaction on susceptible skin and had no effect on normal skin. SIMPSON also found that the leaves contained two active poisons.

1. Needle-shaped crystals which when powdered and dissolved in water and applied to the susceptible skin produced a rapid reaction in 30 minutes, increasing in intensity for eight hours, being still well marked after 36 hours and then gradually disappearing.

This substance produced no reaction on normal skins. SIMPSON thinks it is a crude glucoside. It did not give the tests for alkaloids and is probably the crystalline substance seen by NESTLER in the juice from the hairs on the stems and leaves.

2. The oleo-resinous mixture already referred to which produced on susceptible skins a reaction, which did not commence till after 12 hours, was very intense, spread beyond the area of application and lasted longer than the reaction produced by the crystalline substance. SIMPSON thinks that the active poison of the oleo-resinous mixture is an acid.

These results agree with what has been found in the various plants of the Rhus family. The most/

most recent authoritative work shows that the active principle of the poison ivy is a substance of a glucosidal nature and not a protein. TOYAMA[1152.] and others have shown that this substance in the Rhus plants is not volatile. Many cases are reported where a Dermatitis has occurred without touching the plant but all the evidence goes to show that these are cases of indirect contact either with objects which had been in contact with the plant or persons who had handled it. McNAIR[1083.] showed that the cases of Rhus dermatitis produced by the smoke of burning Rhus leaves, were due to the solid particles in the smoke because, if screened through muslin, the smoke had no effect on the skin.

How does the poison get into the skin? The poisonous substance in Dermatitis Venenata first enters by the hair follicles and glands of the skin. McNAIR[1087.] found in Rhus poisoning that microscopically the inflammation was most marked and began in these areas, but it may, if well rubbed in and left on for some time, penetrate through the horny cells to the deeper layers below. This I confirmed on myself by rubbing the Primula juice well into the skin and washing it off with soap and water a few minutes later. That produced a reaction which consisted almost entirely of minute red follicular lesions and not a diffuse erythema/

erythema such as occurs if the juice has time to penetrate through the whole horny layer.

GROUP REACTIONS.

Another point of interest is whether the reactions to plants like the Primula are specific. It would seem that they are specific up to a point but they appear to be of the nature of group reactions My skin is very sensitive to the Primula Obconia but gives a reaction lasting about a week to Primula Sinensis. Plate (29.). A lesser but quite distinct reaction to the common wild primrose Plate (30.) lasting 2-3 days and a less reaction still to the coloured hybrid primrose. Plates.(30 & 31.).

I give no reaction to the Primula Polyanthus, Plates (32 & 33.), Primula Auricula, Plates (34 & 35.) Primula Veris (cowslip). Plates (36 & 37.) and Primula Malacoides, Plate (38.).

I have also seen an individual whose skin is very sensitive to the jonquil, but who also gives a lesser reaction to the daffodils. There is also the well known Rhus group all of which are irritating and in certain individuals produce a dermatitis viz. Rhus toxicodendron (poison ivy) Rhus Diversiloba (poison oak) and the Rhus vernicifera, which produces the lacquer dermatitis in Japan. The individual/

individual reacts most severely to the plant to which he is originally sensitised and less so to the other plants of the same family. There is evidently some common chemical substance contained in all plants of the same group. This subject will be referred to later in discussing the group reactions which occur in the protein skin tests.

KANNGIESSER[1054.] in 1911 described cases of dermatitis due to Primula Obconica and also to P. Officinalis and P. Auricula. Including 44 cases of his own of dermatitis due to P. Obconica he analysed the reports of 191 cases and found that 121 were in females and 70 in males. About one third of these cases were in gardeners and of the females nearly all were over 40 years of age. He also noted that fair haired persons were more often affected than dark haired. KANNGIESSER also reports two cases where a bronchial catarrh (Asthma?) was caused by the Primula Obconica being in the room.

DERMATITIS VENENATA DUE TO CHEMICALS OTHER THAN THOSE CONTAINED IN PLANTS.

We have seen that the dermatitis due to plants is not due to proteins and that, therefore brings them into line with the dermatitis due to pure chemicals. In all respects these cases of dermatitis are/

are the same as those due to plants but as the worker in chemicals is always handling the substances and gets them all over his clothing, the eruption is usually much more widely distributed.

In Edinburgh we have an opportunity of seeing a considerable number of these cases as there are three large firms who manufacture morphine and its compounds, strychnine and other alkaloids. These firms are constantly having trouble with their workers developing a dermatitis. Some of them develop it within a few weeks of starting work, others have handled the chemicals for 10, 15 and 20 years without developing a dermatitis. In extracting alkaloids such as morphine and strychnine the crude substances are first put into strong acid usually sulphuric acid and this might cause a traumatic dermatitis but I have satisfied myself by rubbing a case of morphine dermatitis with the crude opium that the skin reacts to that substance. Some years ago Sir Norman Walker had a case of dermatitis in an opium worker in his ward. This man had a very extensive dermatitis of head, face, body and limbs, and it was only after about 8 weeks hospital treatment that the eruption cleared up. After he was practically well, with the patient's permission, I rubbed the forearm with some crude opium made into a paste with water. This was done/

done at 11 o'clock in the forenoon and about six hours later he developed a very severe dermatitis on the area rubbed and within 24 hours all the previously inflamed areas of the skin reacted and he developed an extensive dermatitis very similar to the one which he had on admission, and it took another 3 or 4 weeks before he was able to leave the ward.

In other two cases of opium dermatitis I did cutaneous tests with solutions of the alkaloids with which these men worked. 10% solutions of codein phosphate, heroin hydrochloride, morphine hydrochloride and morphine alkaloid were used. The one case (Plate 39.) gave, after 30 minutes, Plate (40.) a reaction with wheal formation and erythema round all the areas but a much more marked wheal round the codein phosphate than round the three others. The other case also gave a very marked reaction to codein phosphate and a slightly less reaction to heroin hydrochloride and morphine hydrochloride, Plate (41.). These results coincided with what the patients themselves thought as they both said that codein phosphate irritated their skins more than the other alkaloids and they were always worst after handling it.

Although these are only two cases, I think the method is worthy of further use as it might enable an employer to tell what substances his workers could safely/

safely handle.

Cast (21.) Plate (42.) shows the hand and forearm of a strychnine worker but in him the skin test to a solution of strychnine hydro-chloride was negative. The cast shows where the test was applied just above the area of dermatitis. Other two cases of dermatitis of the hands and arms in strychnine workers were also tested with the skin test but were both negative. This might be due to the fact that a 1% Solution of Strychnine had to be used for the test as stronger solutions cannot be obtained. The positive reactions with morphine were obtained with 10% solutions and the reactions to quinine already referred to under drug rashes also only occurred when 10% solutions were used. Another possibility is that PIRQUET'S method is not a good one to use. In a case of iodoform dermatitis, to be referred to later, the Pirquet test with iodoform was negative whilst rubbing the skin with iodoform gave a marked reaction. In the next case which I see of Strychnine dermatitis I shall rub a larger area of the skin with the strychnine solution or rub it in as an ointment as in MORO'S test. Of course there is always the possibility of the dermatitis being due to irritation from the strong acids used in the preparation of the strychnine, but I think that can be ruled out by the fact that all three/

three cases showed a marked oedematous dermatitis of the face as well as of the hands etc.

I have also tested three cases of Dermatitis of the face due to instilling atropin into the eye and in each case a positive skin test was obtained with a 10% solution of atropin sulphate.

Therefore I think we are justified in concluding that these cases of dermatitis from chemical substances are sensitisation phenomena of the same nature as the dermatitis due to plants.

Into the same group also fall the large group of dermatitis due to drugs like iodoform, formalin, hair dyes etc., and in this connection there is the interesting dermatitis which occurred during the war from handling T.N.T. and similar substances in explosive bombs.

Two types of case occurred.

(1) The dermatitis in workers handling Trinitrotoluol (T.N.T.)

(2) The dermatitis caused by the yellow powder scattered by bursting German bombs.

1. The Dermatitis due to T.N.T. was specially reported on by CRIPPS, [1014] PANTON [1103] and RUXTON. [1112] The latter found that 32% of the workers were affected with a dermatitis, if no precautions were taken. A few persons were found to be very susceptible to T.N.T. and these were probably cases of true sensitisation to the chemical/

chemical but the majority only suffered from a dermatitis if no care were taken to protect the worker. CRIPPS showed that the dermatitis varied directly with the amount and alkalinity of the sweat. Patients accustomed to take alcohol also suffered more acutely than others. The period between first contact and the onset of symptoms varied greatly. There was found to be no natural immunity to T.N.T. The sweat seemed to make the T.N.T. more irritant either by dissolving it or the T.N.T. possibly destroyed the fat in the sebaceous secretion and so lessened the protection of the skin. The dermatitis, therefore, in most cases of T.N.T. workers is due to a mechanical irritation which can be eliminated if precautions are taken, so that these cases should be classified as Dermatitis Traumatica as they are not true sensitisations. The T.N.T. besides causing dermatitis, by its absorption in some cases, caused abdominal symptoms, irritation of the bronchi, haemorrhages from mucous surfaces, headache and giddiness. Cases with purpuric rashs were also recorded, 3 of which ended fatally.

2. Dermatitis from explosive bombs. Cases of this type were recorded by Ruxton[1112.], MacLEOD[1081.], SEQUEIRA[1126.], CRIPPS[104.], TYSON[1154.], ADAMSON[976], THIBIERGE[1149.] and PANTON[1103.]. When the bomb exploded it scattered a fine yellow powder over/

over everything. This stained the skin yellow at the time and about nine days later an eruption of closely set vesicles rather like cheiro-pompholyx came out. All the cases reported showed an interval of some days before the rash appeared. These cases seemed to be true sensitisations and the rash appearing after an interval is probably due to the same cause as the rash which occurs in serum sickness. During the nine days interval the person becomes sensitised and some of the chemical still circulating by absorption from the skin causes the rash to appear. SEQUEIRA states that the substance in the bombs which causes the symptoms is probably Hexa-nitro-diphenyl-amine, which is the same substance called "Aurantia" and which CROCKER 1015. described in 1903 as causing a dermatitis in workers who used it to stain cheap leather goods. During the war also many cases of dermatitis occurred from wearing helmets and hats lined with a leather substitute which was stained yellow with the aurantia. Such cases were recorded by BETTMANN 991., PONTOPIDDAN 1106., SCHEMEL 1120., GANS 1035., BAER 985., APPEL 981. and HELLIER 1042.. FEILCHENFELD 1021. also in 1909 described a similar dermatitis produced by wearing gloves dyed with aurantia. In all these cases sweating predisposed to the production of the eruption.

At/

At the Dermatological section of the meeting of the British Medical Association at Newcastle in 1921, BARBER[1074.] mentioned some experiments which he had done on himself with mustard gas. He rubbed it on his left arm and it caused a dermatitis. Later he rubbed it on the right anticubital fossa and it produced a slough and a septic lymphangitis ensued. After rubbing the right anticubital fossa the area on the left arm, where the mustard gas had been previously applied, became red and vesicular. This was evidently a focal reaction in the previous area from absorption of the mustard gas from the left arm.

Innumerable other substances such as chemicals, dyes, plants etc. have been reported as causing a dermatitis in certain individuals. Space will not permit me to give a full list of all the possibilities but probably any substance may lead to a dermatitis under certain circumstances. I shall briefly refer to some of the commoner ones, and record some cases due to substances not previously known to cause dermatitis.

Amongst plants the Primulae and especially the P. Obconica are probably the commonest in this country. Other plants which commonly cause trouble are the Virginia creeper, (Ampelopsis Hoggii) common ivy, Humea elegans, daffodils, hyacinths and other bulbs. The following have also been recorded within recent years.

Eucalyptus/

Eucalyptus plant and oil (GALEWSKY)[1033]

Quercus Robur (Oak) (SPILLMANN).[1135.]

Geranium (ANDERSON).[980.]

Cotton seed (NIXON)[1100.]

Chrysanthemum (WAUGH).[1159.]

Satinwood (BIDIE,[992.] CASH,[1008.] JONES,[1049.] SIEGHEIM,[1132.] WECHSELMANN).[1160.]

Lyco persicum exulentum (tomato) (LAIN,[1063.] WASHBURN).[1158.]

Anthemis Cotula (May-weed) (SEQUEIRA).[1128.]

Lillies (WALSH)[1156.]

Bitter orange (MURRAY).[1096.]

Arundo Donax (Reed) (TIMPANO).[1150.]

In America the Rhus plants are the commonest irritants and belonging to the same family as the Rhus Vernicifera, which causes the lacquer dermatitis of Japan. This lacquer is used for varnishing boxes, walking-sticks etc. Ragweed (SUTTON,[1143.] J.C. WALKER)[1155.] Cactus (SUTTON)[1142.], Timothy grass pollen, flax-seed and many others have been reported in America as causing dermatitis.

PARDO-CASTELLO[1104.] also collected more than 40 species of plants chiefly of the genera Anacardiacea Legumenosae, Euphorbiaceae and Urticaeae which cause different degrees of Dermatitis in the Tropics.

From time to time what might almost be called epidemics of dermatitis occur due to chemical irritants in clothing etc. Of these the commonest are the following.

HAIR/

HAIR DYES. The commonest hair dye to cause dermatitis is paraphenylene diamine. It is sold under the name of "Inecto hair dye". I have seen several cases due to it and BARKER[988], BUNCH[1003], BURKE[1004], BROCQ[1002], CATHELINEAU[1009], HARRINGTON and GOLD[1041], MACKAY[1078], MEWBORN[1093], SHALEK[1129], TISSOT[1151] and WOOD[1172] have reported cases. This hair dye was sold widely in America some years ago as "Mrs Potters Walnut juice Hair Stain". In most cases the dermatitis only occurs after several applications but occasionally it irritates on the first application. The "Inecto" dermatitis is evidently a true sensitisation of the skin. In some cases, by absorption, general symptoms of poisoning have been produced.

DYED FUR DERMATITIS.

Within recent months very numerous cases of dermatitis on face, neck and wrists have occurred in Edinburgh and elsewhere due to an artificial beaver fur (dyed coney) worn on the collars and wrists of ladies' coats. SEMON[1124], ROXBURGH and CASTLE[1111] have recorded cases. In a report which I received on this kind of fur from the manufacturers, they stated that the dye used was quite harmless as it only consisted of paraphenylin diamin! It is a question whether these cases/

cases are true sensitisations as they occur very readily if the fur becomes wet either by rain or by perspiration from the neck or wrists. Some seem to be true sensitisations but many seem to be the same type of dermatitis as occurs in T.N.T. workers.

MATCH-BOX DERMATITIS.

This form of dermatitis was first described by RASCH[1108] in 1918. It occurs chiefly in men and usually begins on the front of the upper part of the left thigh owing to carrying a match-box in the trouser pocket. It may also occur on the right thigh, if the box is carried on that side. It may spread on to groins, genitals and even the lower abdomen and by handling the boxes the hands may be affected and the face, through the hands. The lesions are those of an erythematous and oedematous dermatitis.

CHRISTIANSEN[1010] also described cases in Sweden, FREI[1028], STRANDBERG[1137] and STRANZ[1138] in Germany, and WHITE[1168] and FOX[1027] in America. So far as I can ascertain no cases have been recorded in this country. This condition did not occur till 1918 when the match-box manufacturers in Sweden and Denmark added Phosphorus Sesquisulphide (P_4S_3) to the amorphous Phosphorus usually used on the striking surfaces of match boxes. It was added during the war as a substitute for some of the ordinary phosphorus and seems to cause the dermatitis by/

by the heat of the body vapourising the sesquisulphide either pure or in some other form. The reaction is a quantitative one rather than a qualitative one. FREI did a great many experiments by scraping the substance off the side of the match boxes and testing his own skin and that of persons who had had the dermatitis and also those not previously affected. If this substance was applied for 24 hours to the skin of anyone who had previously had the dermatitis, it produced redness and irritation with swelling of the skin. In 140 healthy persons or persons suffering from other diseases, in only two did he get an irritation of similar extent to that of the previously affected cases. In the great majority of cases there was no irritation or only a slight redness after 24 hours application. But in a good many cases, if the substance were continuously applied for several days, an irritation was produced. FREI found on experimenting on himself that areas which were previously inflamed were more easily irritated on a subsequent application. Therefore this form of dermatitis does not seem to be due to a true sensitisation of the skin. Most individuals seem to get a reaction if the substance is applied for sufficiently long time or rubbed sufficiently well in. It therefore should be classed as a Dermatitis/

Dermatitis Traumatica. A previously inflamed area reacts more readily to subsequent application so that there is a local increased sensitiveness such as occurs in all forms of dermatitis traumatica.

INSECTICIDE POWDERS. REEB[1109], FUJITANI[1030], McDONNELL[1077], McCORD, KILKER and MINSTER[1076] report cases of dermatitis due to powdered Pyrethrum used as an insecticide. The latter observers report it in the workers making the powder, many of whom suffered from a mild dermatitis especially in Summer when they perspired freely but some of the cases were apparently real sensitisations.

TAR DERMATITIS. Apart from the chronic dermatitis caused by tar, dermatologists frequently find that tar irritates certain skins. In 1921 I saw a case where sensitiveness to tar developed in a patient with psoriasis. He was treated for psoriasis in April 1920 with crude coal tar on the body and Pix carbonis in acetone on the scalp. The tar caused no irritation. In February 1921 he was seen again with an acute dermatitis on the scalp, face, and arms where he had again applied the tar as the psoriasis had recurred. He was tested by painting a small area of one arm with crude tar and reacted violently with a marked dermatitis which lasted several days. During the first course of treatment this man had evidently/

evidently become sensitised to the tar and reacted some months later when it was reapplied.

Other chemical substances reported as causing dermatitis are,

Rubber adhesive plaster containing Agathis Damara (KNACK).[1056]

Procain used by Dentists (LANE).[1065]

Oil of Citronella used as preventative of mosquito bites (LANE).[1067]

Anilin dyes in workers and from clothes dyed with these (LANDOWZY and BROWARDELL,[1064] BLASCHKO,[994] BALZER,[986] WILSON,[1171] SACHS,[1116] WHITE,[1166] SCHARLACH R. (LOMBARDO).[1072]

Formalin (GALEWSKY).[1033]

Phenyl Hydrazin (HALL).[1038]

Hairy Caterpillars of the Moth Euproctis Edwardii (CLELAND).[1012]

Copra (McLEOD).[1080]

Printer's Ink (McCONNELL).[1075]

Dinitro benzol (BERNSTEIN).[990]

Hemamethylamine (SHEPHARD and KRALL).[1130]

Various other forms of dermatitis many of them of the nature of folliculites are recorded in metal workers using irritating oils, (McLACHLAN)[1079] and in linen spinners (LELOIR)[1070] but these do not appear to be sensitisation phenomena.

ADAMSON[975] and COOKE[1013] also report cases of dermatitis in the napkin area of infants and in children with enuresis due to ammoniacal urine. COOKE isolated

a/

a gram-positive bacillus which caused fermentation of urea with the production of ammonia. The dermatitis seems in these cases to be due simply to the irritation of the ammonia.

In my own practice within recent years I have seen cases of dermatitis due to the Primula, daffodils, Inecto hair dye, dye used by wool-worker, dyes in furs, morphia, tar, iodoform, lysol formalin, Kresol, atropin, Humea elegans, enamel used to glaze porcelain and guinea-pig's hair. To the above list I should like to add three cases of dermatitis due to substances which have not hitherto been reported as causing irritation.

(1) Last Autumn I saw a medical man with a very marked Dermatitis Venenata of hands and face. He was on holiday at the time in the Highlands. I told him to test himself by rubbing the arm with the different plants with which he had been in contact. He found that he gave a very violent reaction to Bell Heather but not to the ordinary heather.

(2) Another case was a farmer who developed a severe dermatitis after handling sheaves of oats. He gave no reaction on rubbing oatmeal into the skin. Later he had another attack after handling sheaves of barley but he gave no reaction when tested with barley. So I told him to look out for weeds in the sheaves and/

and he found that milfoil (Achillea Millefolium) Plate (43.) was present and it, when rubbed on the skin, gave a very marked reaction.

(3) The third case was in a working housekeeper who had had a dermatitis of hands and face every year for 15 years beginning in October and lasting till about February. She was completely free all the rest of the year. I asked her to keep a look out next year when the dermatitis began and she found that it was due to plucking grouse. As soon as she stopped touching the grouse feathers the dermatitis rapidly disappeared. This patient also showed a group reaction as her skin reacted slightly to the feathers of other game birds such as partridges, and pheasants but not of the common fowl.

GENERAL AND LOCAL SENSITISATION.

In these various forms of Dermatitis Venenata already referred to, it has been seen that the phenomenon is a general one affecting the whole skin surface. This however does not apply to the mucous membranes. I can rub the Primula leaf on the mucous membrane of the mouth and nose without producing any reaction. Jadassohn[1047] also found, in iodoform dermatitis cases, that iodoform had no effect when applied to/

to mucous surfaces. I saw a case in the gynaecological wards some months ago, where after an operation on the uterus the vagina was packed with iodoform gauze. This patient next day showed a very marked dermatitis of vulva, groins and lower abdomen and on testing her later, on the skin of the arm, she reacted markedly to iodoform. Yet the mucous membrane of the vagina showed no change although the iodoform had been in contact with it for over 24 hours. Therefore the absence of reaction on the mucous surface was not due to the moisture of the surface keeping the chemical from getting into proper contact, as might be the case, if it were only applied for a short time.

Also many persons with poison ivy dermatitis have chewed the leaves and swallowed the juice without suffering any inconvenience.

BLOCH[996.] repeated JADASSOHN'S[1] experiments on a patient sensitive to iodoform. He found that subcutaneous injection of iodoform oil (avoiding the skin) internal administration of iodoform or application to mucous surfaces for 24 hours had no effect, but as soon as a trace was applied to the skin a dermatitis resulted. Therefore he concludes that the cases are not a true general anaphylaxis, not a humoral condition but a cellular Hypersensitiveness. To confirm this he transplanted a piece of skin from this iodoform dermatitis/

dermatitis case and also a piece from his own skin on to another person with a healing burn. When both pieces had "taken", he dusted the area with iodoform and obtained a very marked reaction only on the piece of skin from the patient who was sensitive to iodoform. The rest of the skin including the piece of BLOCH'S own skin showed no reaction. BLOCH and MASSINI[849.] also did similar experiments with extract of Ringworm fungus. BLOCH who was himself sensitised to an extract of the Ringworm fungus (Trichophytin) and whose skin gave a positive Trichophytin reaction transplanted on to a patient with chronic leg ulcer a piece of his own skin and a piece from a normal individual not sensitive to Ringworm. After the two pieces had taken, he did cutaneous tests with Trichophytin on this man on the normal skin, on the piece transplanted from BLOCH'S own arm and on the piece transplanted from the normal man's arm, and obtained a positive reaction only on the piece of skin transplanted from his own arm.

These experiments prove conclusively that in sensitisation of the skin, the condition is a cellular one of the cutaneous epithelium and does not depend on the blood or other organ.

IS/

IS DERMATITIS VENENATA A TRUE ANAPHYLAXIS?

Most workers on Anaphylaxis hold the view originally stated by RICHET that true anaphylaxis can only be produced by a protein. FORD[1025] claims to have produced it by a glucoside but doubt is thrown on the purity of his antigen by STEVENS, McNAIR[1084], and WARREN.[1157] ZINNSNER[273] states that in true Anaphylaxis not only are specific antibodies present in the blood but the production of passive anaphylaxis in animals shows that the antibody originating and residing in the fixed tissue cells, must necessarily circulate in the blood stream. SIMPSON[1133] holds that although in primula dermatitis we are dealing with an example of hypersensitiveness to a chemical, the requisites of anaphylaxis are not fulfilled, and has shown that it is not possible to sensitize an animal actively to the poison of the plant or passively with the serum of a susceptible individual. He also found that no specific amboceptors are present in the blood of susceptible persons and that the protein of the plant has nothing to do with the reaction. That being so it is as impossible to produce an antitoxine to the poison of the plant as it is to produce a condition of specific sensitisation, not dependent on circulating toxines."

Antibodies/

Antibodies, if concerned in the reaction, are present only in the sensitive cells. These antibodies, however, must be carried by the blood stream in order to sensitise the cells. This was proved by the fact that by rubbing a small area of my skin with the P. obconica I sensitised my whole skin.

BRUCK[999] did a number of experiments with iodoform to try to show that iodoform dermatitis was a true anaphylaxis. He injected 3 guinea-pigs with 5 cc. serum from a patient who showed a dermatitis when iodoform was applied to his skin. Twenty-four hours later 0.3 grm. of iodoform was injected into each guinea-pig, and within 5 minutes two of them showed typical anaphylactic symptoms and the other showed some dyspnoea only. Control animals injected first with normal human serum and horse-serum and then with iodoform were all negative. Therefore he concluded that passive anaphylaxis could be produced. But by using the serum of another case of iodoform dermatitis BRUCK did not succeed in causing anaphylaxis in guinea-pigs. BLOCH and others have questioned the validity of BRUCK'S results as the symptoms in the guinea-pigs might not be due to true anaphylaxis but to iodoform poisoning which causes symptoms of an anaphylactoid nature. BLOCH repeated BRUCK'S experiments but failed to produce passive/

passive anaphylaxis in guinea-pigs with the serum of cases of iodoform dermatitis, so that there is no definite evidence yet that in dermatitis, such as that produced by iodoform, there is a real general anaphylaxis.

The only reference (apart from Rhus Toxicodendron cases) which I can find to any fatal result in Dermatitis Venenata is a report by BROWN.[997] In his case the patient accidentally scratched her nose whilst smelling a plant of Primula Obconica. The nose rapidly swelled and a carbuncle-like lesion appeared with oedema of the face and head. Death followed in a few days from pneumonia. This fatal result cannot however be attributed to the dermatitis. The nose lesion was scraped under an anaesthetic and it is quite probable that the death was due to septicaemia. BROWN also mentions 2 other similar cases of infection from Primula obconica, one of whom also died and the other recovered after a prolonged illness. It is the usual clinical experience of dermatologists that oedematous dermatitis of the face from the Primula looks somewhat like erysipelas and to an inexperienced observer cases of erysipelas might be mistaken for a dermatitis venenata.

Rhus toxicodendron however is well known to cause serious poisoning. WHITE[1167] reported a case of a boy aged 6 years who was twice poisoned one Summer/

Summer with Rhus Tox. Next year a boy known to be insusceptible to Rhus was employed to tear up some Rhus plants. The boy subsequently washed his hands in hot water and soap and later with vinegar. He was watched to see that the washing was thorough. In the afternoon the boy took the susceptible child to a pond to bathe. Having stripped him he took him into the water, holding him by the arm-pits and afterwards massaging his backwith his open palms. Two or three days later the child was taken ill and grew rapidly worse. Deep ulcers appeared under the arm pits and the skin of the back showed in an aggravated form the usual appearances of poisoning with ivy. The child died at the end of three weeks. Such cases of extreme sensitiveness, however, are rare considering the number of cases of Rhus-poisoning which occur.

LOCAL SENSITIVENESS OF THE SKIN.

In addition to the general epithelial sensitisation there are undoubtedly cases where you get a local sensitiveness of the skin. At Newcastle last year BARBER[1074] mentioned such a case in a baker, whose hands and forearms, but not the rest of his skin reacted on application of flour.

DE JONG[1050] in 1923 drew attention to the fact that/

that dermatitis in bakers is often due to the strong salt solution which is added to the flour before baking. The heat in the bake house causes the salt to crystallise on the arms and in kneading the bread the crystals are rubbed into the skin and so produce a dermatitis. These cases are not therefore cases of true flour dermatitis and should be separated from them. MARKLEY 1092. also reports a dermatitis of face, neck, chest, arms and hands from handling a guinea-pig. The patient's skin only reacted when guinea pig's hair was rubbed on these areas and nowhere else.

Recently I saw a lady with a similar dermatitis on the neck where a pet guinea-pig was in the habit of lying, but I had no opportunity of testing this patient to see whether the skin was sensitive all over or only on the neck.

There is also the well known experiment with Tuberculin by PIRQUET on his own arm. He found that the skin of the left arm which he habitually used for experiments was sensitive to a tuberculin of a dilution of 1 in 1000 whilst the skin of the right arm was negative to Tuberculin ten times as concentrated. It may be that in these cases the skin is sensitive all over but the degree of sensitiveness may vary in different places. On the other hand/

hand it may not be a sensitisation phenomenon at all but merely a mechanical irritability of certain areas of the skin to a repeated or prolonged application of some chemical substance. They are similar to the washer-woman's dermatitis of hands and forearms. In such cases one does not get lesions on the face even although the skin there is frequently touched by the hands. I think a distinction should be made between this group of cases and the true Dermatitis Venenata. Whitfield's term of Dermatitis traumatica seems to be the most appropriate one.

A good deal of experimental work has been done with regard to the tolerance of the skin to local irritation. In 1892 SAMUEL [1114] found that repeated application of croton oil to a rabbit's ear produced a relative immunity, so that the skin after recovering from the first application did not react so violently to a second application of the croton oil. Also such an area of skin after recovering from a croton dermatitis reacted less markedly than normal skin to other irritants such as the application of hot water, etc. FURST [1031] likewise in 1898 by repeated freezings and scalding of the skin at intervals so changed the skin that the same irritants produced after a time practically no inflammation.

SCHAER/

SCHAER[1118.] in 1907 repeated SAMUEL'S experiments on rabbits' ears with silver nitrate, cantharides, turpentine, etc., and obtained the same results i.e. a relative immunity, not only to the substance which produced the original dermatitis but also to other irritants.

So also STEIN[1136.] in 1909 succeeded in producing a skin tolerance to irritants applied in gradually increasing concentrations and also found that the tolerance was not specific for each irritant. STEIN thinks it is due to some alteration in the epithelial cell, whereas SAMUEL put the change down to an alteration in the blood vessels of the skin.

SCHULTZ[1121.] in 1912 tested the effect of different strengths of carbolic acid on the skin in different diseases and found that in eczema the whole skin showed an increased sensitiveness to the carbolic.

In 1920 AUER[983.] found that if a rabbit was sensitised to horse serum, the skin of its ear reacted much more violently to the application of a substance like Xylol than did normal control animals. He explains this as due to the local irritation of the Xylol causing inflammation and an exudation into the part. In a recently reinjected animal the antigen is still circulating in the serum and when the serum passes out into the skin, from the irritation of the Xylol, a local/

local hypersensitive reaction is produced.

FERGUSON SMITH[1213.] repeated AUER'S experiments on sensitised rabbits using cantharides and croton oil instead of Xylol as the irritant. He found that "no dilution, which fails to provoke a visible reaction in a normal rabbit, will provoke one in an anaphylactic animal". This observation tends to throw doubt on AUER'S results, but if AUER'S work is to be taken as valid then his experiments together with SCHULTZ would seem to indicate that in a condition like washer-woman's dermatitis the patient works for years with soap and soda with no ill effect - in fact acquires often a considerable degree of tolerance to these substances.

The same patient, if she becomes sensitised to some antigen, say from internal absorption or elsewhere, possibly not sufficiently so to produce any symptoms, may get a local reaction in the skin of the hands from the soap and soda causing an exudation and thus bringing sufficient circulating antigen through the serous exudate into the skin of these parts to produce a reaction.

Burns of the skin may be included under the heading of D. traumatica, but a number of experiments performed in 1912 by ALHAIQUE[979.] are suggestive that in severe burns a process of sensitisation may occur./

occur. He burned rabbits by applying hot water at a temperature of 80°-100°C. for 3-5 seconds and found that animals which had recovered from a previous burn became more susceptible to the action of a second burn. They often died with symptoms like anaphylaxis from the effects of a second burn which was not of an intensity or extent to cause death usually. Animals sensitised with serum from a previously burned animal showed, on reinjection with the same serum, phenomena which ALHAIQUE attributed to a state of anaphylaxis. He thinks that a burn causes the absorption into the circulation of toxic substances which sensitise the individual and that many of the late deaths from burns can be explained on this theory.

IMMUNITY AND DESENSITISATION TO DERMATITIS VENENATA.

1025.
FORD in 1907 experimented with the Rhus poison and found that he could produce a real antitoxic immunity to it by repeated intravenous injection and thinks that there is a reasonable possibility that the use of an antitoxic serum may acquire a definite place as a practical therapeutic measure in the treatment of severe cases of Rhus dermatitis. 1083. McNAIR says/

says that natural immunity to the Rhus plant is seldom absolute. If sufficiently vigorously applied he always produced a reaction. In my experiments with the Primula in those who did not sensitise, possibly I did not rub the antigen in sufficiently vigorously or over a sufficiently wide area.

In the experiment on my brother by repeated daily inoculation the result was sensitisation with a delayed incubation in the first instance and not an immunity as I had expected. I did not pursue this line of investigation further because, even although I had immunized some one, it would have been impossible to say whether that individual had been made immune or whether he was naturally so. But if I were repeating the experiment I should inoculate the skin at very short intervals, say every half hour for several times in the same way as serum injections are given by splitting the dose and giving at short intervals so as to cause desensitisation.

One attack of Dermatitis Venenata does not produce an immunity or desensitisation to further attacks. There are numerous cases in the literature of poison ivy dermatitis cases, who as soon as they were cured of one attack, immediately got another on touching the plant. Therefore any attempt to desensitise patients by repeated applications of the irritant are not likely to be successful. On the other hand/

hand either ingestion by the mouth or desensitisation by injections are more likely to prove efficacious.

STRICKLER[1139.], DUNCAN[1018.], DAKINS and others quote numerous cases where chewing the leaves of the poison ivy and swallowing the juice each Spring rendered the individuals immune at least for that season. SCHAMBERG[1119.] also confirmed this and found that the administration of a fluid extract of the plant by the mouth was quite successful, if the extract was taken in gradually increasing doses for about a month.

Much experimental work has also been done by STRICKLER[1139.], PETCH and ALDERSON[978.] by injections of extracts of the poison ivy and oak. STRICKLER claims that intramuscular injection of the toxine of Rhus toxicodendron can cure the dermatitis of poison ivy; the inflammation and itching being greatly modified within 24 hours after the first injection. A second injection 24 hours later as a rule was sufficient to cure a case. This result is a desensitisation and not a true immunity as it is only temporary. STRICKLER recommends intramuscular injections during the attack and subsequent administration of the extract by the mouth later on to keep up the desensitisation.

DIFFENBACH[1017.] reports the production of immunity to the poison ivy by drinking milk of cows fed on grass and poison ivy.

A/

A man suffering from a dermatitis due to ragweed pollen was given by SUTTON [1143.] 12 injections of an extract of the pollen in gradually increasing doses. This removed his susceptibility so that he could handle ragweed with safety. HANNAH [1040.] also reports a successful result by the same extract in a similar case.

Therefore desensitisation to plant dermatitis can be successfully accomplished by oral administration or injection of the plant or extracts from it. This desensitisation is not however permanent as the workers with Rhus Toxicodendron showed that the desensitisation only lasts for about two years.

CONCLUSIONS.

1. It is possible by rubbing the juice of a plant such as the Primula obconica into the broken or unbroken skin to sensitise the skin to that plant.

2. The sensitisation in such cases is general over the whole skin.

3. Such sensitisation is not due to the protein of the plant but to a substance of a glucosidal nature.

4./

4. The reactions to plants are relatively specific and are of the nature of group reactions.

5. Sensitisation of the skin may occur to pure chemicals such as morphine etc.

6. Dermatitis due to Bell heather, Achillea. Millifolium and grouse feather are described for the first time.

7. Dermatitis may be true sensitisation, i.e. D. Venenata or simply due to irritation, i.e. D. traumatica.

8. The sensitisation is a sensitisation of the skin cell only and is not a true general anaphylaxis.

9. A true local sensitisation of the skin may occur.

10. Desensitisation has been successfully accomplished to plant dermatitis by oral administration of the plant or injection of extracts from it.

DERMATITIS OTHER THAN D. VENENATA and D. TRAUMATICA.

This large group may be divided into the so-called "eczema" and Seborrhoeic dermatitis.

DERMATITIS - ECZEMA.

This group includes all forms of dermatitis where no definite external cause can be demonstrated. The term "eczema" will be used frequently so as to avoid confusion. Although I do not like the name and prefer the term "dermatitis" for the sake of convenience I shall use it because the word dermatitis to some minds includes Dermatitis Venenata and D. traumatica which are excluded from the eczemas. We are not concerned here with the clinical varieties such as erythematous, papular, vesicular, weeping, crusted, squamous etc. but with the evidence for or against sensitisation being responsible for them. The older theories may be roughly divided into,

(1) Toxic theory. This was strongly advocated by the French school of dermatologists, the gouty, arthritic and other forms of diathesis being held responsible for the condition. There is however no direct evidence of toxines causing the condition although /

although they may indirectly predispose to it.

(2) Neurotic theory. The fact that attacks of dermatitis may result from or be aggravated by anxiety, shock, worry etc. has been held to support its nervous origin but, as in the previous theory, while it may predispose to attacks there is no direct evidence to support this theory. The frequency of dermatitis in infants, where the influence of the nervous system may presumably be to a large extent excluded, is also very much against its acceptance.

(3) Bacterial infection theory. This theory, especially urged by UNNA, is now generally considered as being untenable. All the evidence goes to show that the condition is not contagious and that any organisms present in the lesions are secondary infections.

None of the above theories explain the phenomena satisfactorily. The fact that D. Venenata, which shows a practically identical clinical lesion and is a sensitisation phenomenon at once suggests that all forms of eczema are sensitisations. FORDYCE 1185. was the first to suggest that eczema is anaphylactic. Its association with Asthma, which may be taken as definitely proved to be a sensitisation phenomenon, and the work done on asthma point to a similar origin for the two conditions. Although not an inherited disease/

disease, VAN DER VEER and others have pointed out that in certain families Asthma, urticaria and eczema are much more frequent than in others. As I showed under D. Venenata, only certain individuals have skins which are readily sensitised and therefore the hereditary factor is important.

The actual proof that eczema is a sensitisation phenomenon will necessarily be a difficult one but the more one thinks over the subject the more one is driven to the conclusion that it must be so. The changes in the skin in eczema are identical with those of dermatitis venenata. The eruption flares up and dies down again in the same way as it does in D. Venenata after each application of the irritant. All the conditions which have been shown to be present in the skin cell in D. Venenata may be assumed to be present in eczema.

[219.]

WEIDENFELD made an extensive study of the reaction of the skin of eczema patients to irritants using dilutions of croton oil. He found that the skin of eczema patients shows a greater reaction than normal skin and the more acute the disease the greater the reaction. The strength of the reaction was found to decrease as the disease disappears. The increased sensitiveness lasts for some time after recovery but is always transitory. The normal skin near the eczematous/

eczematous patch showed a greater reaction than that at a distance. The skin of a healed patch was more sensitive than the skin which had never been affected. WEIDENFELD thought that the increased irritability of the skin is due to toxic substances passing from the primary eczema patches into the blood and causing changes in the skin which predispose to eczema. This irritability of the skin in eczema however can be explained on the sensitisation theory. The croton oil by irritating the skin and producing a hyperaemia brings a larger quantity of circulating antigen to the part and a reaction takes place between the antigen and the sensitised skin cells. It is the same phenomenon as has already been referred to under D. Venenata in the experiments which AUER made with xylol (page 362.).

1212. SCHULTZ in 1913 confirmed WEIDENFELD'S results using phenol as an irritant.

1195. JAEGER in 1923 also came to the same conclusion using Formalin, oil of turpentine and Tinct. Arnicae as irritants. He found that in over 50% of the cases of eczema, the skin responds to the external application of these irritants with a more intense reaction than is seen in normal skins. This reaction was found in infantile, professional, seborrhoeic and so-called constitutional eczemas.

1213. FERGUSON SMITH however in 1923 could not confirm/

confirm the results obtained by WEIDENFELD, SCHULTZ and JAEGER. He tested the skin of 50 eczema patients using dilutions of cantharides and croton oil as irritants. He "failed to discover any notable and constant increase of sensitiveness".

Further experiments are therefore necessary to clear up this matter.

HARRIS[1190.] discussing the work of WEIDENFELD and SCHULTZ did not agree that the irritability of the skin in eczema to irritants resided in the epithelial cells as it varies so much. He thought it more probable that the nervous system is responsible. The irritability of the skin can be diminished experimentally by adrenalin applied either externally or subcutaneously. Assuming that eczema was a protein sensitisation and possibly similar to the sensitisation which can be produced with Histamin, (p. 24.) HARRIS made experiments to find out the frequency with which Histamin is found in the stools in cases of eczema. Histamin is a product of pancreatic digestion. Unfortunately HARRIS' death occurred before these experiments were completed.

THE/

THE INFLUENCE OF THE NERVOUS SYSTEM ON ECZEMA.

That the nervous system plays an important part in eczema is undoubted. MORRIS[1205.] and others have reported cases where anxiety, worry etc. have precipitated attacks or aggravated existing eczemas. The itching, which is so constant a feature of the disease, also points in the same direction. SAMUEL[1114.] in his experiments showed that when the sensory nerves supplying the ears of rabbits were cut, scalding did not cause inflammation. BRUCE[335.] showed that cutting the nerves to the conjunctiva prevented inflammation when mustard oil was put in the eye. SPIESS[1214.] noticed that, after tonsillectomy, if the denuded area were anaesthetised the inflammatory reaction and swelling were distinctly less or absent, and the wound healed more quickly than it did if not anaesthetised.

All these facts go to show that nervous reflexes are necessary for the production of inflammatory reactions and that the nervous system is necessary before an inflammation like eczema can occur. It has already been shown that the nervous system plays a part in carrying out the various phenomena of a true anaphylaxis and similarly it does so in all sensitisation phenomena.

Similarly/

Similarly the endocrine glands are intimately bound up with the sympathetic nervous system. EDELMAN[1182.] reports a case of eczema in a child of 3½ years of age. The disease had been present since the age of 4 months. The child showed signs of thyroid deficiency and the eczema rapidly disappeared on the administration of thyroid extract. HARRIS[1190.] suggested that in eczema some toxic substance in the blood neutralised the suprarenal secretion and so affected the nervous reflexes of the skin. In support of this he quotes the frequent association in eczema of low blood pressure and asthma both of which conditions are counteracted by adrenalin.

MORO[1204.] in 1920 published an article on the hyperexcitability of the vegetative nervous system and changes in the endocrine glands at certain seasons of the year. In 237 cases of infantile eczema the incidence of the first eruption was highest in January February and March. From April it gradually decreased till August when it gradually rose again till December. Of these 237 cases 4 died, 3 in March and 1 in April. FEER[1183.] also reports 11 deaths from eczema, 9 of which occurred in February, March and April. The cases which were examined post mortem showed no pneumonia so that the deaths were not due to chills.

MORO/

MORO also quotes HAMBURGER who found that Tuberculin hypersensitiveness was higher in Spring than in Autumn. He also states that cases of Tetany occur most frequently in the Spring. He concludes that in Spring there is a change in the endocrine glands and vegetative nervous system which makes infants more susceptible to attacks of tetany and eczema.

The sympathetic nervous system and the endocrine glands therefore would seem to play a part in eczema as they do in true anaphylaxis.

PASSIVE ANAPHYLAXIS IN ECZEMA.

1440.
BLACKFAN attempted to produce passive anaphylaxis in guinea-pigs by injecting them with the blood serum of patients with eczema due to egg white and cow's milk but failed to obtain any result.
1462.
SCHLOSS however claims to have done it successfully with the blood serum of sensitive patients.

FOODS IN ECZEMA.

It has been known for a long time that in some cases of dermatitis, alteration of the diet has resulted in a cure of the condition. The connection between the diet and the eruption of dermatitis is not so evident as it is in some cases of urticaria. Most of/

of the work done in this connection has been done in infantile eczema where the diet is less complicated and more easily controlled than in adults. Some hold that excess of certain food constituents in the diet will cause eczema, others support the sensitisation theory basing their evidence chiefly on the sensitiveness of the skin to certain foods as demonstrated by the food skin tests. These reactions will be fully discussed later under cutaneous reactions.

FISCHER[1184] thinks that there is an association between gastric and gastro-intestinal derangements and eczema in infants, as acute eczema may follow overfeeding with fats and carbohydrates. He states that eczema is more frequent in bottle- than breast-fed infants. Undigested particles of food give rise to fatty acids and these, when absorbed, cause a toxaemia which results in skin irritation and dermatitis. Feeding with large quantities of cream gives rise to fat indigestion. Similarly excess of sugar and carbohydrates may be factors in the etiology of eczema. He quotes cases of eczema in infants who were given all kinds of food which they could not digest. They all recovered as soon as they were put on a diet of buttermilk, vegetables and fruit juices.

TALBOT and TOWLE[1215] also demonstrated in infantile eczema that there is an incomplete digestion of fats and carbohydrates. MORSE[1207] reports a case of infantile/

infantile eczema associated with constipation and an excess of fat in the diet.

JOHNSTON[1196.] found that in about 40% of cases of eczema in adults hyperacidity was the most common digestive disorder. Indicanuria, which indicates intestinal fermentation, is also frequent and constipation is often present. He found that in eczema there is no constant failure in the synthesis of urea and showed that there is no proof that there is any constant error of metabolism.

WHITE[1220.] examined the stools in 46 cases of eczema and found that 28% had normal faeces, 63% had excess of fat and 8% excess of starch. When both skin tests and faecal examinations were made in the same individual the two methods did not always reveal similar results. In the patients with normal stools one showed a positive skin test to all types of food and another to fat. In those with fat in the stools 10 showed negative skin tests to all foods and two were positive to starch. In the starch-stool group one gave wholly negative skin tests. Therefore mere inability to digest and assimilate certain types of food does not necessarily mean that the individual has become sensitised to these foods. On the other hand sensitisation to a food or foods as shown by skin tests may occur where the digestion is apparently normal.

These/

These observations naturally lead on to the further question as to whether altered digestion does not lead to absorption of undigested proteins into the circulation, so as to cause sensitisation. .A great deal of work has been done in this connection. VAN ALSTYNE [1221.] showed that in dogs egg albumin introduced into the stomach or any part of the small intestine, except the duodenum passed unchanged into the blood stream. The same results were also obtained with ox and beef serum. It is now possible by the precipitin and anaphylactic reactions to distinguish proteins in very small quantities in the circulation. At first it was thought that in digestion the proteins were split into proteoses and peptones and in that form entered the circulation. These substances are soluble and can permeate parchment membranes. Further experiments by UNDERHILL [1614.] showed that peptone and proteoses if introduced direct into the blood were highly toxic and HOWELL [1236.] demonstrated that after a heavy protein meal the blood serum shows no excess of proteoses and that the blood of the portal vein, during the period of maximum absorption after a meal, shows no proteoses or peptones. Recent investigations show that the splitting of proteins before absorption proceeds much further than was supposed. They are split into amino-acids in which form they are found circulating in the blood.

FALLS/

1229.
FALLS quotes ABDERHALDEN as stating that the liver is the buffer between the intestine and the general circulation and protects the latter from the entrance of foreign proteins in an undigested state. The liver is probably rich in ferments (as shown by post-mortem autolysis) and these ferments probably change the absorbed proteins into substances which can with safety be thrown into the general circulation.

1221.
VAN ALSTYNE used the anaphylactic shock test to demonstrate the presence of unchanged proteins in the circulation. The protein was introduced into an isolated loop of intestine and after an interval blood was withdrawn and introduced into a guinea-pig previously sensitised to that protein. If the animal developed anaphylactic shock, it was taken as proof of the presence of unaltered protein in the blood. VAN ALSTYNE showed that conditions which interfere with normal digestion, such as ligaturing a portion of the intestine, increase the amount of protein absorbed.

1243.
NATHAN quotes LESNE'S work in which the latter showed that egg albumin injected into a sensitised animal induces anaphylaxis even after it had been digested with pepsin. But when the egg albumin had been subjected to the action of the pancreatic juice, there were no signs of anaphylaxis. NATHAN reports/

reports a case the same as one recorded by LESNE in which the patient, a boy of 8 years, always had diarrhoea, and an urticarial rash after eating egg. Examination of the stools showed defective functioning of the pancreas and administration of 40cgrm. pancreas daily enabled him to eat eggs with impunity.

ASCOLI[1224.] was able to demonstrate the presence of egg protein in the blood serum of healthy men by means of precipitin tests as early as 1¾ hour after the egg was eaten. ASCOLI and VIGANO[1225.] also demonstrated by precipitin tests in dogs that unaltered alien protein such as egg albumin appears in the blood soon after it was introduced into the stomach. GANGHOFER and LANGER[1230.] found by precipitin tests that the intestinal tract of young animals permitted the passage of heterologous proteins into the blood serum. Also in two debilitated infants they demonstrated undigested egg protein by precipitin tests applied to the blood. LUST[1239.] found that in normal infants undigested egg or beef protein is not absorbed. But in nutritional disorders he found that egg protein could be demonstrated in the serum and urine by precipitin and anaphylactic tests. HAYASHI[1234.] obtained the same results and showed that normal infants do not absorb undigested proteins, whereas infants with nutritional disturbances may do so.

Similar/

Similar results are recorded also by SCHLOSS and WORTEN[1248], HOOBLER[1235], TALBOT[1250], MODIGLIANI, and BENINI[1241], LEWATSCHEK[1238], GREER[1231], GRULEE and BONAR[1232]. But UFFENHEIMER[1252] and HAMBURGER and SPERK[1233] could find no evidence of absorption of undigested protein in young animals. As already mentioned earlier, animals can be sensitised by feeding them on foreign proteins as shown by WELLS, UHLENHUTH, ROSENAU and ANDERSON, RICHET and others. VAUGHAN, CUMMINGS and McGLUMPHY[1253] showed that after feeding rabbits on egg protein, their blood was capable of sensitising guinea-pigs to egg protein. MORO[1242] in one case of an atrophic infant found that the blood serum caused complement deviation with milk protein. HUTINEL[1237] records examples of sensitisation passively transmitted through human milk to nursing infants.

From all the above experiments it is clear that, especially in infants with gastro-intestinal disturbances, unaltered proteins may be thrown into the circulation. It has been shown that this unaltered protein produces a group of symptoms such as vomiting, diarrhoea, skin rashes etc. which are probably an expression of anaphylaxis. As sensitisation has been repeatedly produced by feeding animals on foreign proteins all the evidence goes to support the view that those cases of eczema, where a positive cutaneous test to a food is present and where removal of that food/

food from the diet results in a cure, are due to sensitisation to that food. The cases already referred to, where incomplete digestion of carbohydrates etc. occurs, can also be explained on the sensitisation theory, because the incomplete digestion leads to absorption in an undigested state and subsequent sensitisation. An eczema due to foods may even occur in infants on the breast. I saw a case recently of a child aged 6 months who had been breast fed since birth. On the mother stopping eating eggs the dermatitis completely disappeared within ten days. TALBOT[1471.] reported a case of very severe eczema in a child which cleared up on the mother's discontinuing the eating of chocolate and recurred on her again eating the food.

SHANNON[1464.] has shown that egg protein may appear in the breast milk after its ingestion by nursing mothers. O'KEEFE[1454.] showed that although the infant may give a positive skin reaction to a certain food the nursing mother does not.

These facts account for the cases of infants who sometimes give positive skin reactions to foods which they have never eaten. If the child is on the breast he may become sensitised to something which the mother has eaten or if on cow's milk to something on which the cow has been fed, e.g. turnips. It also accounts/

accounts for the cases of children who develop urticaria the first time they are given an egg and that fact has been quoted as a point against the sensitisation theory.

Although very little is known as to the production of eczema by absorption of proteins other than foods from the bowel, by analogy it may be assumed that in some cases the eczema may be due to sensitisation of the skin to the protein of organisms from the bowel.

DANYSZ[1713.] claims that he obtained very good results by treating eczema patients with autogenous vaccines of the organisms grown from the bowel contents. But whether these act specifically or simply as general protein desensitisers is not very clear.

Focal infections have seldom been shown to cause dermatitis. MORROW[1206.] described a case of long standing very itchy eczema of the face and neck which cleared up completely after the removal of teeth which were associated with multiple dental abscesses and another of generalised erythematous and vesicular eczema in which opening and draining a suppurating joint caused a disappearance of the rash. SUTTON also in a discussion on the subject mentioned a case of vesicular dermatitis of the soles of the feet and another case of dermatitis of the limbs which disappeared when the teeth were put in order.

WHITFIELD/

WHITFIELD records a case of eczema in a child of the flexural type in which the disease disappeared after septic tonsils and adenoids were removed.

Cases of definite foci of infection causing eczema, however, seem to be the exception rather than the rule.

There is a good deal of evidence that infections of the lower bowel may cause eczema. In practice especially in adults, in cases of extensive symmetrical dermatitis where an external cause may be excluded, cutaneous food tests and alterations in the diet may fail to reveal any food as the cause. The only other possibility is some organismal bowel sensitisation and these are the cases where drinking the waters at a place like Harrogate, and high colon lavage by the Plombière douche give such good results. Every dermatologist has seen cases of this type. I can recall a very marked case in a medical man, who had tried all kinds of external applications for years, with very little benefit. This patient experienced relief from itching within 24 hours of having the bowel washed out at Harrogate and a further course of treatment cleared up the dermatitis completely. As soon as the lavage was stopped within ten days or so the eruption reappeared. He learned to wash out his own bowel and found that if he did so, once a week, his skin remained perfectly well. Cases such/

such as that are undoubtedly due to lower bowel absorption and probably sensitisations to the organisms growing in the bowel.

When a person becomes sensitised to a food or organism in the intestine, why does the skin cell become sensitised and eczema result? Why do they not get urticaria or asthma? No explanation has yet been given, but I would suggest that some trauma such as rubbing or scratching of the skin whilst the protein is circulating is necessary. This might break the skin cells and allow the serum from the blood containing the antigen to sensitise the cells just as occurs in D. Venenata. In eczema both the sensitisation of the skin cell and the reaction known as eczema is brought about through the blood stream whereas in D. Venenata these two reactions occur from the outside.

THE/

THE ASSOCIATION OF ORGANISMS IN THE SKIN WITH DERMATITIS.

Numerous attempts have been made to prove that the so called eczemas are due to organisms. UNNA[1278.] is the chief upholder of this theory and some years ago described the "Moro coccus" which he thought caused eczema. Practically everyone is now agreed that the moro-coccus is the staphylococcus epidermidis albus which is present on every normal skin. This question involves especially the dermatitis known as Seborrhoeic eczema or dermatitis. In seborrhoea capitis and seborrhoea corporis three organisms are now generally admitted always to be present, viz. the seborrhoea bacillus (? acne bacillus), staphylococcus epidermidis albus and the spore of Malassez. It is still undecided which of these organisms is the causal factor or whether a combination of them is responsible. The same three organisms occur in the lesions in seborrhoea corporis or seborrhoeic dermatitis. In seborrhoea corporis the lesions start as papular, papulo-vesicular and vesicular lesions and end as scaly and crusted areas of a yellowish-red colour usually spreading peripherally, sometimes clearing up in the centre and typically seen on the sternal and interscapular regions. There are also the/

the similar inflamed lesions which may occur on the scalp and spread behind the ears on to the neck, in the axilla, groins etc. There is no difficulty in assuming the parasitic origin of the more superficial forms of seborrhoea corporis where there are circinate lesions spreading slowly outwards. The ease with which these cases are cured by local applications, the slight degree of itching present and the gradual slow spread without marked variation from day to day, which one sees in other forms of dermatitis, are all in favour of a simple infection with one or all of the seborrhoeic organisms. But when larger areas are affected such as the whole scalp, behind ears, axillae, groins and round umbilicus the condition does not look quite so like a parasitic one. One must take into consideration the possibility of these cases being sensitisation phenomena. This applies more particularly to the cases where the eruption, although starting on the typical seborrhoeic areas, spreads more or less acutely and becomes more or less generalised. The eruption flares up and dies down again repeatedly and is often intensely itchy as in other forms of dermatitis. These cases also are not so amenable to local treatment as the simple circinate cases. Are they cases of sensitisation to some protein by internal absorption in which the seborrhoeic virus is implanted on/

on the top or are they due to the skin or sebaceous glands becoming sensitised to the protein of the seborrhoeic virus from the outside? This arises particularly in the infantile eczemas which some dermatologists consider as seborrhoeic because of the constant implication of the scalp. face, behind ears, flexures etc. As already mentioned such cases are often associated with sensitisation to food proteins. If that be so, then the probability is, that the lesions are secondarily infected with the seborrhoeic virus. By scratching there is no reason why the child should not sensitise himself to the protein of the seborrhoeic virus and so produce a double internal and external sensitisation eruption. The same remarks apply equally well to extensive dermatitis in adults especially in cases affecting chiefly the flexures. The dermatitis may start as a sensitisation to food or bowel organismal protein and as a result of scratching end as a sensitisation to the skin organisms.

All dermatologists are familiar with the stubborn cases of seborrhoeic dermatitis met with during the war. I refer especially to the cases where there was a moist dermatitis of the scalp, eyebrows and often the beard region, which resisted all local treatment and tended to relapse,. Were these cases of sensitisation to the seborrhoeic virus or some other/

other organism? Autogenous staphylococcal vaccines were often very helpful in treatment. That would suggest that these were cases of sensitisation to the staphylococcus which had secondarily infected a seborrhoea.

MEDALIA[1203.] in 1915 published the results of treatment of 50 obstinate cases of eczema with autogenous vaccines. The staphylococcus was the most prevalent organism found, but the streptococcus also was found in a few cases. The results obtained by these vaccines were in a great measure successful.

STRICKLER, KOLMER and SCHAMBERG[1240.] did complement-fixation tests with the sera of patients suffering from seborrhoeic dermatitis using the acne bacillus (which some consider as identical with the seborrhoea bacillus) B. Coli and staphylococcus. Of the 10 cases of seborrhoeic dermatitis, which were examined, 30% reacted positively with the antigen of B. Acne and 25% with the staphylococcal antigen. With the antigen of B. Coli from acne patients, fixation occurred in 40% of the sera and in 11% with the antigen of B. Coli from normal persons. These results show the presence of antibodies to skin organisms in seborrhoeic dermatitis and support the view that a sensitisation to these bacteria may take place. The percentage of cases which gave a positive reaction to B. Coli points to the possibility of intestinal absorption playing a part in the sensitisation of the patient.

A/

A local sensitisation of the skin to the seborrhoeic infection, is another possibility. It is well known that seborrhoeic eruptions recur in the same areas of skin each time. It is possible that these areas have become sensitive to the virus and therefore the eruption always recurs in these places.

There is also the type of case of seborrhoeic dermatitis of the head and face in adults, in which, quite suddenly, for no apparent reason, the whole face becomes affected with an acute erythematous oedematous dermatitis with swelling of the eyelids so much so as to suggest erysipelas. Is that an acute streptococcal infection with an organism allied to the erysipelas streptococcus on the top of an ordinary seborrhoeic dermatitis or is it a sensitisation phenomenon? The suddenness with which it comes on, the absence of temperature and the general appearance of the face suggests a D. Venenata. It is possible that the patient is sensitised to the seborrhoeic virus and by scratching the skin of the face, rubs in the virus, living or dead, and so produces a local D. Venenata.

There is another variety of eczema which was first described in 1902 by ENGMANN as "infectious eczematoid dermatitis". This form of dermatitis is frequently preceded by scabies, impetigo, furunculosis, wounds, traumatisms etc. BENDER, BOCKHART and GERLACH showed/

1173.

showed that it was possible to produce a dermatitis by rubbing filtrates of bouillon cultures of staphylococcus into the skin. FORDYCE[1185] in 1911 suggested that discharges from ulcers, sinuses etc. containing the chemical products of the staphylococcus and other organisms might produce a sensitisation dermatitis.

Bearing on this question also is the observation of WHITFIELD[394] who noticed, in a case of vesico-bullous eczema of the legs in a gouty individual, that when the bullae burst and the serum ran down across the legs there appeared in a few minutes a red streak which was followed in about 10 minutes by an urticarial wheal. This was succeeded by a line of vesicles and then a long narrow bulla. When the patient's serum was applied to WHITFIELD'S own arm it caused no reaction. WHITFIELD suggests that this patient was sensitised to his own tissue products. Recently FERGUSON SMITH[1213] has confirmed WHITFIELD'S observation.

Some further observations on the sera of eczema cases have been made. BRUCK and HIDAKA[1177] studied the cocci found in eczema biologically but were unable to demonstrate an increase in agglutinins or haemolysins in the sera.

KREIBICH[1199], BROCK and VEILLON[1176], LEWANDOWSKY[1201], DOHI[1181], FREDERIC[1186] and COLE[1180] found staphylococci or streptococci or both in the vesicles of eczema. But that alone is not sufficient to show whether they were the/

the primary cause of the eczema or only secondary contaminations. COLE criticised BENDER, BOCKHART and GERLACH'S results in producing a dermatitis by rubbing in staphylococcal filtrates claiming that the result was due to the alkalinity of the filtrates. He concludes that there is no proof that staphylococcal toxines as such produce eczema, although they may play some part in its course. COENEN[1179.] also did some work by rubbing staphylococcal filtrates into the skin, but the lesions produced were pustular and did not resemble eczema. RAJKA[1211.] did experiments to see if allergy was present to skin organisms. Autogenous vaccines were made from the skin of each case. The vaccines were exposed to active serum for a time to set free the anaphylatoxin. He used the serum of normal individuals, the serum of eczema cases and that from blisters of eczema cases. With the vaccines treated with eczema serum he obtained a more marked local reaction by intracutaneous injection than with vaccines treated with normal serum. Focal but not general reactions were found to occur on injection of these vaccines. RAJKA thinks that the local and focal reactions prove definitely the existence of a skin-allergy to pyogenic organisms in eczema.

From/

From all these observations, therefore, there is some evidence, although rather scrappy, that sensitisation to the skin organisms occurs in dermatitis, but there is not sufficient evidence yet to show whether that alone may produce a dermatitis or whether the sensitisation is from other sources and the skin organisms complicate the condition.

Before leaving the subject of Dermatitis, it would be well to summarise the results arrived at. Broadly speaking all cases of Dermatitis (including D. Venenata, D. Seborrhoeica, and the so called eczemas) may be divided into 4 groups.

(1) D. Venenata due to sensitisation to plants, chemicals etc.

(2) Dermatitis due to sensitisation to the skin organisms, e.g. D. Seborrhoeica, and infectious eczematoid dermatitis.

(3) Dermatitis due to food sensitisations.

(4) Dermatitis due to sensitisations to organisms in the bowel, or some focal infection.

In the first two groups the antigen acts on the sensitised skin cell from the outside and in the last two from the inside. Assuming that, in all, the skin cell is sensitised the same mechanism would account for the eruption whether the antigen which provokes it is applied externally or brought to the skin by the blood stream.

DERMATITIS EXFOLIATIVA (PITYRIASIS RUBRA).

Very little is known definitely with regard to the etiology of general exfoliative dermatitis. But since the arseno-~~and~~ benzol preparations have come extensively into use numerous cases of a general red scaly eruption indistinguishable from exfoliative dermatitis have been recorded. Some have ended fatally, others recover completely in 2-3 months if the use of the drug is discontinued. These cases are undoubtedly due to the drug and are to be looked upon as a sensitisation phenomenon. They usually occur after several injections have been given and may or may not be associated with other symptoms. The above cases at once suggest that all cases of exfoliative dermatitis are dueto sensitisation to some chemical substance. In exfoliative dermatitis the eruption comes out all over the body and limbs quite suddenly either in previously healthy individuals or in persons with psoriasis or other eruption. It is well known that over-treatment of psoriasis, especially if it is in an inflamed condition, is apt to bring on an exfoliative dermatitis, which may last for months or years. The cases which occur on the top of psoriasis or seborrhoeic dermatitis suggest that it may be due to a special form of sensitisation to the organisms which probably cause these conditions. But in cases where no previous disease

has/

has existed it is possible that the antigen comes from the bowel infection. Foods have never been shown to be associated with exfoliative dermatitis, but chronic rheumatism often accompanies it and the two conditions may have a common origin. Focal infection may in some cases be the cause. HEIMANN [323] reported a case od pityriasis rubra in a man of 60 years of age whose teeth were found to be in a very bad condition. All the carious stumps were extracted and in two weeks the patient was well and has remained so for three years.

POLLARD [285] described a case with tuberculous glands and lupus vulgaris in which exfoliative dermatitis developed. He considers that in some cases at least the rash is a toxi-tuberculide. Doubtless the cause is not the same in all cases as drugs, and bacterial infections of various organs may produce the same type of eruption just as some of the trichophytides are indistinguishable from tuberculides. A short time ago a female patient was admitted to Sir NORMAN WALKER'S ward suffering from psoriasis and whilst in the ward she developed an exfoliative dermatitis all over the body and limbs. It spread, rather slowly for such cases, till the whole skin was affected after 4-5 weeks. Then I gave her 6 injections of sterilised milk intramuscularly using the milk/

milk as a general desensitiser. Within a fortnight the exfoliative dermatitis disappeared leaving the original psoriasis as before. I have tried the same method in other more old standing cases but without much result. It is an isolated observation and may have been a coincidence, but I have never seen another case in which exfoliative dermatitis lasted so short a time. Possibly the fact that she was injected so soon after the rash developed had something to do with the result. These cases will be further referred to under non-specific protein therapy.

The fact that the exfoliative dermatitis after drugs like Arsphenamin clears up in some weeks after the drug is stopped and that the so-called idiopathic cases may go on for years, points to the probability that in the latter cases the antigen is still continuously being absorbed and so the condition is kept up.

Allowing that it is a sensitisation phenomenon, then it is a different form of sensitisation from the so-called eczema. Clinically the eruption is different. It never goes on to vesiculation and the mechanism of its production must be different from that of dermatitis. Further light is necessary before any more definite opinion can be arrived at.

PSORIASIS/

PSORIASIS.

I do not propose to enter into a discussion as to the cause of Psoriasis. Personally I hold the view that it is probably an infection with an organism and probably spread through the blood stream. No definite organism has been described, but the fact that the spots spread peripherally, healing in the centre, and do not itch much, suggests a simple infection rather than a sensitisation. The fact that it runs in families and only affects certain people suggests that a special condition of the skin is necessary before an individual can be infected. There are also numerous cases recorded and I have seen two cases myself where psoriasis first developed at the seat of a vaccination against smallpox and spread from there to other parts.

SELLEI [1284.] made an extract of psoriasis spots by rubbing up excised psoriasis patches with salt solution so as to produce a milky emulsion. This emulsion was used for injection in doses of 0·1 cc. and 0·2 cc subcutaneously. Eight cases of Psoriasis were injected and in all he noticed a local reaction at the seat of injection. This reaction occurred more quickly and strongly the oftener the injections were repeated at short intervals. Even after quite small/

small injections in most cases a general reaction with rise of temperature to 38°-38·6° C. followed. In six of the cases a fresh psoriasis eruption of small papules, on the top of or around old spots, appeared. This happened in all cases where a general psoriasis was already beginning to disappear. As a control a similar extract of normal human skin was made and similarly injected into psoriasis patients but without producing either a local or a general reaction. Therefore SELLEI concludes that in psoriasis there is a sensitisation to the virus.

STOKES [1509.] tested three cases of Psoriasis with an emulsion from psoriasis lesions. He obtained a local reaction with this emulsion by the intradermal method only and not by the Pirquet method.

Further confirmation of these results is however necessary.

I have tried to desensitise psoriasis cases with peptone and milk injections. Many others have also tried vaccines, and various protein desensitisers, but as will be seen later, when desensitisation is discussed, the results have not been very encouraging. If psoriasis is due to an infection with an organism it cannot be one which produces any virulent toxine as the general health is often quite good and there is no rise of temperature. Therefore if patients are sensitised to psoriasis they seem to remain in the sensitive/

sensitive stage and do not go on to immunity. That would account for the cases always relapsing after apparent cure. From the way in which the eruption in the average case slowly spreads, not flaring up and dying down as in dermatitis, I am inclined to think that patients with psoriasis are not highly sensitised to the infection.

ACNE/

ACNE VULGARIS.

It is now generally accepted that acne vulgaris is due to the acne bacillus. The simple infection of the sebaceous gland follicles leads to the comedo but when the infection goes deep into the corium, the large swollen lesion results. This lesion microscopically is a granuloma with giant cells. These lesions resemble structurally tubercle and tertiary syphilis in both of which cases a bacterial sensitisation is present. The more highly the patient is sensitised probably the larger the lumpy lesions are. An acne, if left untreated, remains for some years in this sensitive stage, lesions coming and going till it goes on to complete immunity, and the disease dies out. No cutaneous tests have been done in these cases with acne cultures but vaccines are often very beneficial in treatment.

Complement-fixation tests were done by STRICKLER, KOLMER and SCHAMBERG.[1290.] 57 cases were tested and well marked positive reactions were obtained to the antigen of B. Acne in 84·2% of the cases. As a rule the more severe the infection the higher were the percentages and degree of complement-fixation. With the antigen of staphylococci from acne cases positive reactions were found in 64%. Similar results were also obtained using the antigen of staphylococci from boils. This indicates that the staphylococcus found/

found in acne does not differ from the ordinary staphylococcus of furuncles and other lesions and is probably a secondary infection. With a polyvalent antigen of B. Coli from the intestine of acne cases positive reactions were obtained in 63·1% of the cases, and the control antigen of B. Coli from other sources only yielded 32% positive reactions. This would indicate that the Colon Bacillus may have something to do with the pathogenesis of acne. Complement-fixation tests, using acne, staphylococcal and B. Coli antigens in Syphilitics with positive Wassermanns and in other chronic diseases, in 26 patients were uniformly negative. These results show therefore that in acne vulgaris there is a sensitisation to the acne bacillus and also to the staphylococcus and B. Coli. The character of the lesion and the results of vaccine treatment also support the sensitisation theory.

STAPHYLOCOCCAL/

STAPHYLOCOCCAL INFECTIONS.

These include BOCKHART'S Impetigo, Furunculosis and Sycosis.

In BOCKHART'S Impetigo and furunculosis there is evidence that in some cases at least the patient becomes sensitised and remains for some time in that state. In most cases the disease is either cured by local treatment and removal of the virus or after a number of lesions have been produced, desensitisation or immunity results. Vaccines are well known to be beneficial in furunculosis but whether a true sensitisation occurs in these cases there is no evidence to show. Possibly it does occur in these bad cases of multiple boils which are so difficult to treat and in cases which have been over vaccinated.

Sycosis is definitely a staphylococcal infection of the hair follicles, Here again vaccines are beneficial. These cases may go on for years and the whole affected area become red and moist like a dermatitis. Sycosis suggests a sensitisation to the staphylococcus. If it is, it is probably local and not general. BESREDKA 1294. from experiments on animals concludes that staphylococcal vaccination depends on the production of a local immunity of the skin. He got better results by applying the vaccine cutaneously than/

than by injecting intra- or sub-cutaneously.

ALTMANN and BLUHDORN[1292.] obtained positive complement fixation tests in staphylococcal infections in animals.

SCHREUS and GOEHL[1296.] in two cases of Pyodermia found marked positive complement-fixation with staphylococcal vaccine.

LEDERMANN and WASSERMANN[1295.] worked with a preparation called "Histopin" which was an immunising fluid made by shaking living staphylococci for a long time in water. This extract was made into an ointment in strengths of 25-50% and applied to staphylococcal impetigos, boils etc. They found that the development of fresh lesions could be prevented and existing lesions healed rapidly. They think that the histopin influences the local immunity. BECK[1293.] confirmed these results to some extent but found that in furunculosis, sycosis,& staphylococcal folliculitis the results were not uniform. The best results were obtained in superficial lesions, Occasionally BECK observed a local reaction around the area where the Histopin was applied.

Astonishingly little work has been done on the question of sensitisation to the staphylococcus considering how frequently it complicates skin conditions but what evidence there is points to a low degree/

degree of sensitisation of the skin which in some cases, as in sycosis, is probably local and not general.

SKIN ERUPTIONS IN GONORRHOEAL INFECTIONS.

According to BUSCHKE [1299.] the usual skin complications of gonorrhoea are,

(1) Scarlatiniform erythema.

(2) Urticarial rashes.

(3) Bullous and haemorrhagic rashes.

(4) Rashes resembling erythema multiforme.

(5) Lesions like Erythema nodosum.

He thinks that the eruption is partly due to circulating gonococci and partly to toxines. In some of the nodular rashes the lesions suppurated and the gonococcus was obtained from them. All the above rashes are of the type which we have already seen to be hypersensitive phenomena. HODARA [1308,] and others have demonstrated the gonococcus in the blood in such cases and it is probable that the individual is sensitised to the gonococcus and the rash results from the reaction between the sensitised skin and the organism brought to the skin by the circulation.

There is also the rare condition known as Keratodermia Blenorrhagica which most commonly affects the feet but may appear elsewhere. The lesions begin as/

as vesicles, soon become pustules whose wall becomes keratinised forming round hard lesions. Opinion is divided as to whether this eruption is due to the presence of the gonococcus or to toxines acting directly on the skin or through the nervous system (tropho-neurosis). LEES[1311.] considers it to be a toxic local manifestation of the general systemic infection as shown by the accompanying cachexia, muscle wasting and anaemia. JACQUET, CHAUFFARD and FIESSINGER[1300.] succeeded in reproducing the lesion in a ~~keratolic~~ patient by inoculating the skin under a watch glass with serum from a keratotic lesion. McDONAGH[1313.] found a lymphocytosis of the cerebro-spinal fluid and considers the eruption to be due to meningeal irritation of the trophic fibres of the posterior nerve roots caused by the gonococcal toxines. The gonococcus has frequently been found in the blood but only once in the lesions in these cases. It may however be a sensitisation rash similar to the others which occur in gonorrhoea but with the addition of possibly staphylococcus or other organism which causes the curious keratotic growths.

CUTANEOUS TESTS IN GONORRHOEA.

Numerous workers have performed cutaneous tests with gonococcal vaccines and toxines. BRUCK,[1298.] EISIG,[1303.] GIORGIS,[1306.] IRONS,[1309.] KOHLER,[1310.] LONDON,[1312.] FUCHS,[1305.] FINKELSTEIN,[1304.] and GERSCHUN,[1301.] DECASTRO all obtained positive reactions in/

in the majority of cases. SAKAGUCHI and WATABIKI,[1314.] BRANDWEINER and HOCH,[1297.] SIMON[1315.] and SOMNER[1316.] found positive reactions in only a small percentage of cases. The cutaneous and intracutaneous methods were used. Some observers obtained positive reactions in control cases but these were not very numerous. FUCHS[1305.] using NEISSER'S gonococcal vaccine (gonococcal broth) for intradermic tests, found that as a rule the reaction appeared about six days after the disease developed and that it was present for some months after the disease was healed. The reaction was not associated with any special clinical form of gonorrhoea. Treatment with gonococcal vaccine did not produce any change in the reaction. FUCHS considers that the test can be used to determine whether a case is definitely cured.

Positive complement-fixation reactions have also been shown to occur in gonorrhoea especially in general infections and treatment with vaccines has been very successful, especially in the complications of the disease. Focal and general reactions, also occur in gonorrhoeal infections after intravenous or intramuscular injection of gonococcal vaccine.

From the above facts I think we are safe to conclude that the gonococcus is capable of producing a true bacterial sensitisation.

FOCAL/

FOCAL INFECTION.

The term focal infection is rather a wide one. It includes the local effect of the invasion of an organ with organisms and the method of spread of the infection from the primary focus by lymphatics or blood stream to other organs there to produce similar lesions. But the term is also used to designate the changes which take place in organs at a distance from the primary focus due to the absorption of toxines or other chemical substances from that focus. In diseases such as syphilis and tubercle we have seen that various skin rashes are produced by the organisms which come by the circulation from a focus or foci, reacting on the sensitised skin and in a sense these diseases are focal infection diseases, but under this section will be discussed a series of diseases of the skin which are probably due to the action on a sensitised skin of various toxic substances absorbed from a focus of infection. The possible sources of absorption are the gums in pyorrhoea, teeth in apical abscess, tonsils, lymph glands of naso-pharynx, neck, mediastinum, abdomen etc. mastoid, maxillary antrum and other accessory sinuses, bronchi, endo-cardium, gastro-intestinal tract, appendix, gall bladder, genito-urinary/

urinary tract such as kidney, bladder, prostate, seminal vesicles, uterus or Fallopian tubes. Infected lymphatic glands are secondary to the primary focus and secondary foci in the glands etc. may persist long after the primary focus has healed. The organism most commonly associated with these focal infections is the streptococcus. The flora of the mouth, teeth and tonsils include chiefly the haemolytic and non-haemolytic streptococci and the staphylococcus. Sir WILLIAM WILLCOX (1931.) writing on infections of the teeth and gums refers especially to the haemolytic group of streptococci which cause severe toxaemias and rashes of various kinds especially the purpuras. The organism may grow in an organ like the tonsil and produce very little local effect but the breaking down of the organism by ferments or other means leads to the production of toxic substances which may act on the sensitised skin.

The tissues which are most often associated with focal infections are the teeth and gums, nasopharynx, tonsils and the lymphatic glands connected with these tissues. In many cases there is obvious pyorrhoea alveolaris or carious teeth are present. In these cases there is no difficulty in the diagnosis but there may be abscesses at the root of the teeth which cause no symptoms and in such cases an X-ray photograph of the teeth is necessary to demonstrate the/

the lesion. The fact that a patient has no visible teeth does not necessarily exclude that source of infection as infected stumps from previous imperfect extractions may be present and can only be revealed by X-rays. It is also very difficult to tell by looking at a tonsil, whether it is the seat of an infection. Many small tonsils with old fibrosis and pockets of pus are less conspicuous than the large tonsil with wide crypts but no deep infection. The removal of a septic focus in the jaw or tonsil may lead to a temporary aggravation of the associated skin condition by opening up the lesion and causing a fresh absorption. The fact that the lymphatic glands may be the seat of secondary foci also makes treatment more difficult as the symptoms may persist after the primary focus is removed. Infections of teeth, gums and tonsils are so common that, as RAVITCH and STEINBERG[1329.] pointed out, "we must be careful not to accept a certain number of coincidental cases as proof". Out of 100 cases of skin diseases with focal infections, SUTTON[1342.] found that 75% improved markedly or recovered after finding the focus and removing it. In the other 25% he was unable to find any direct relation between the focal infection and the skin disease.

The question of focal infection has already been referred to under Urticaria, Erythemata, Eczema, Purpura/

Purpura, D. Herpetiformis and Exfoliative dermatitis, but in these diseases a focal infection is only one of many sources from which the eruption may originate. There is still a group of diseases in which it is probable that the skin rash is always due to absorption of toxic substances from a focal infection. Into this group fall Lupus erythematous, alopecia areata, Prurigo and possibly also Pruritus ani and vulvae and generalised Pruritus. HERPES ZOSTER has been mentioned by several writers as an example of focal infection. In the sense that there is a focus of inflammatory infection in the ganglia, producing a skin eruption in the distribution of the sensory nerves associated with these ganglionic cells, HERPES ZOSTER might be called a focal infection, but in these cases there is no sensitisation of the skin and therefore H. ZOSTER does not come into the group which we are discussing.

Scleroderma and Vitiligo have also been mentioned by CHIPMAN [1319.] and others as possibly associated with focal infection, but very little indeed is known of the etiology of these diseases. Recent work rather points to some endocrine gland change in scleroderma but whether the gland change is due to absorption of toxines from some focus is not yet decided. Similarly Vitiligo may have some association with a toxic nerve lesion. Further information on these diseases is necessary before they can be elucidated.

LUPUS ERYTHEMATOSUS.

see Cas/s 22 & 23 Plates. 44 & 45.

[1332.] BARBER in 1915 published a severe case of Lupus erythematosus of the face, neck, ears, scalp, hands and left elbow in which plates prepared from her faeces gave a practically pure culture of a streptococcus longus. She developed later what was apparently a streptococcal septicoemia with high fever and rigors and the rash spread over the back. She was extremely ill for some days after which the temperature gradually fell to normal and the rash all disappeared except a few of the older patches on her face. At this stage no streptococci were to be found in her faeces. No blood culture was taken during the febrile stage, but BARBER came to the conclusion that the eruption of L. erythematosus was due to absorption from/

from the streptococcal focus in the intestines and the fever to a general infection of the circulation with the streptococci. During the febrile attack sufficient antibodies had been formed to destroy the streptococci in the blood and intestines.

In 1919 BARBER [333] reported another case of Lupus erythematosus of discoid type on the face, ears and scalp. In this case examination of the faeces failed to show any streptococcus longus. The tonsils were considerably enlarged and appeared septic and the glands at the angle of the jaw were enlarged. A swab from a tonsillar crypt gave a pure growth of streptococcus longus. The tonsils were removed and the operation was followed by a rise of temperature to 99·6°F. An autogenous vaccine was made and a dose of 5 millions given the day after the operation. This caused a definite focal reaction in the patches of Lupus Erythematosus which swelled up, became very irritable and showed signs of spreading. This reaction lasted about 24 hours. Two days later another similar dose was given and the temperature rose to 99·8° and again a focal reaction occurred. Eight days later 10 millions of vaccine were given and that produced considerable constitutional reaction with a temperature and an intense focal reaction in the skin lesions which became congested and spread considerably.

An/

An old healed lesion flared up again also. The pyrexia lasted some days and gradually subsided, and the patches showed signs of healing. But an acute attack of inflammation in the remaining lymphoid tissue of the throat was immediately followed by a very severe focal reaction in the skin lesions. The complete removal of all the infected lymphoid tissue and the use of a sensitised vaccine caused all the lesions to retrogress completely. BARBER also mentions another case of Lupus eryth. on face, and hands with infected tonsils. Their removal caused a febrile attack of several days duration and a focal reaction in the skin patches. An autogenous streptococcal vaccine was made from the tonsils and when it was given the skin lesions entirely disappeared.

Since BARBER'S cases were published several other observers have recorded similar results. WHITFIELD[394.] reports cases of Lupus erythematosus where treatment for pyorrhoea caused the eruption to disappear and others where enucleation of the tonsils and the administration of an autogenous vaccine produced the same result. LESLIE ROBERTS[1315.] also reports improvement in a case of Lupus erythematosus after tonsillectomy. HARTZELL[1334.] recorded a case where removal of a "capped tooth with an abscess at the root caused marked improvement in a Lupus Eryth. although much previous local treatment/

treatment had had very little effect.

1335.

In 1920 along with LOGAN and RUTHERFORD, I published the post mortem report of a case of generalised Lupus Eryth. This case was first seen in 1915 with an acute Lup. Eryth. of face, ears and left hand accompanied by pyrexia (100°F.) This subsided under treatment with quinine and rest in bed. The eruption persisted for two years on face and hands as a Lupus Eryth. of discoid type. Then it entirely disappeared for nearly a year. In 1918 she had another acute attack in which the eruption reappeared on face, ears, neck, hands and forearms and thighs, and the temperature ran between 100° and 103°F. The patient became extremely ill and emaciated after the temperature and rash had continued for 3 months. Then the temperature gradually fell and the rash disappeared entirely. But 17 months later she had another recurrence of the temperature and rash the latter being even more extensive than previously. She died after this attack had lasted about 2 months. Cultures from the heart-blood post mortem yielded a pure growth of streptococcus longus. Unfortunately the case was complicated by the finding of an unsuspected laryngeal diphtheria. It could not be decided whether the streptococcus had gained access to the blood shortly before death as the result of the diphtheria or whether it was there previous to that.

The/

The fact that a blood culture made a month before death was negative cannot be held as conclusive that no organisms were present at that time. There was also a subacute pericarditis evidently not tuberculous and it is possible that the pericardial lesion was the focus from which the streptococci arose. A vaccine was made from the streptococcus obtained from the above patient's blood and used to treat another case of Lupus Eryth. of face, scalp and hands in a girl, aged 14 years. This case had apparently no tonsillar lesion. Several bad teeth were removed and then the vaccine treatment was commenced. As she improved markedly, the treatment was continued, after the special vaccine was finished, with a stock polyvalent streptococcal vaccine. She was given an injection once a week beginning with 5 million and gradually increasing up to 150 million. The treatment extended over nearly 9 months and at the end of that time nearly all the eruption had disappeared. A month after the vaccine was stopped there was a recrudescence of the eruption on several of the old areas. The presence of an enlarged tender gland under the right side of the lower jaw suggested that there was still some undiscovered focus in the mouth or throat. The vaccine was resumed and has been continued weekly till now. She has again improved but evidently the vaccine is not capable/

capable of causing a complete cure.

Another case of Lupus Eryth. of left cheek and scalp. Mrs. K. age 38, was treated with an autogenous vaccine of streptococcus longus obtained from an inflamed right tonsil. At the end of 5 months the face lesion has entirely healed and the scalp lesion is much better.

A third case of Lupus Eryth. Mrs. H. aged 42 was treated with streptococcal vaccine. No focal infection could be found in the throat and all her teeth had already been removed. After 3 months streptococcal vaccine treatment the eruption entirely disappeared, but 6 months later there was a slight recurrence of eruption. After one of the vaccine injections she showed a focal reaction in the skin lesion similar to that recorded by BARBER in his cases.

In 1920 with RUTHERFORD I published a case 1336. of Lupus Eryth. in a woman age 56 years, affecting the face and ears. She had bronchitis with streptococcus longus and FRIEDLANDER'S pneumo-bacillus present in the sputum. Her teeth were extremely bad and the upper and lower gums showed marked pyorrhoea. All the bad teeth were extracted and she was given a vaccine of pneumo-bacillus and streptococcus longus made from the sputum. The bronchitis soon subsided and the Lupus erythematosus improved greatly, healing completely in several areas.

Three/

Three months later she had another attack of bronchitis with staphylococcus aureus and streptococcus longus in the sputum. At the same time the Lupus erythemotosus relapsed and showed eruption very similar to what she exhibited on the first attack. She died of heart failure and bronchitis a month later. As the post-mortem examination was not made till 24 hours after death no cultures were made from the blood or internal organs, but a very thorough search of all the organs both naked eye and microscopically failed to reveal any trace of tuberculosis. I do not propose to enter into a discussion as to whether Lupus Eryth. is a form of tuberculosis or not, but even one case where tubercle could be absolutely excluded is, I think, sufficient proof that tubercle is not the cause of the condition. The appearance of the rash both naked eye and microscopically is against tuberculosis. On the other hand BARBER'S suggestion that it is due to the absorption of some substance produced in a focus of streptococcal infection acting on a sensitised skin gives a much better explanation of the condition than any other. It has long been recognised that the eruption of Lupus Eryth. is closely related to the erythemata which we have seen may be caused by focal infection. The good results obtained by removal of infected tonsils and sepsis of the teeth and gums and the use of an autogenous vaccine also support this contention./

contention. Therefore in the ordinary cases of Lupus Eryth. of face and ears etc, the probability is that a streptococcal focus in teeth, gums, adenoids, tonsils bowel or bronchi is the cause of the condition. There is some evidence also that exposure to light had something to do with the localisation of the eruption on the nose, cheeks, ears and hands, which are the common situations for it. BARBER mentions a case of SEQUEIRAS where Lupus Eryth. appeared after the application of Finsen light and poultices. Sunburn has also been held responsible for its appearance in some cases.

In the cases of generalised Lupus erythematosus, as BARBER suggested, there is probably, in addition to the original focus, a blood infection with the streptococcus. The streptococcus circulating reacts with the skin cell to produce the rash. As BARBER'S and my cases show the patient may become desensitised and recover both from the septicoemia and the focal infection. The generalised cases resemble very closely malignant streptococcal endocarditis both clinically in the way the temperature remains persistently high and in the fact that there is a focus of infection in the heart valves from which streptococci are shed into the circulation. In malignant endocarditis there is probably no sensitisation of the skin and therefore no rash occurs.

ALOPECIA AREATA.

The true nature of alopecia areata is still in doubt. Three theories have been supported by different sets of observers,

(1) Microbic.

(2) Nervous

(3) Toxic theory.

1. MICROBIC THEORY. This theory was strongly supported by SABOURAUD and others. The seborrhoea bacillus, spores of Malassez, staphylococcus epidermidis albus have all been held responsible. If organismal, it should be transmissible, but the experiments of JACQUET and others on man and animals have all been negative. The fact that epidemics have been described does not necessarily support this theory as a toxine in food might explain epidemics in institutions.

2. NERVOUS THEORY. This theory probably has the most support from dermatologists at present. It is well known that the disease may follow mental strain and shock. Local injuries may also cause it. I have seen a patch of typical alopecia areata develop on the side of the head following a severe smash which resulted in fracture of the zygomatic arch and severe contusion. In this case the patch was rounded and showed typical exclamation-mark hairs at the margins. This/

This case could be explained on the assumption of a traumatic neuritis. The fact that alopecia areata spreads at the edges is not necessarily proof of its parasitic origin as the trophic nerve to a given area might be affected and as the neuritis spreads up the nerve it implicates the smaller branches which come off further up and so, as the neuritis spreads up the nerve, the spot of alopecia enlarges at the edges (Plate 46.) JACQUET'S theory that it is due to reflex irritation from carious teeth had some supporters, but as will be seen later there is another explanation of how the teeth may cause the condition. WHITFIELD'S[1338.] cases where removal of eyestrain cured the alopecia also support the nervous theory.

3. TOXIC THEORY. This theory is chiefly supported by the fact that injection of various bacterial toxines into the skin of animals has often resulted in bald patches. Staphylococcal pustules on the scalp as a rule cause the hair to fall out in these areas, presumably from the effect of local toxines. Acetate of thallium, if taken internally, will also cause the hair to fall out but as it is a nerve poison the effect of this drug rather supports the nervous theory.

Each of the above theories has something to recommend it in given cases, but none of them seem to explain all cases. Since attention has been drawn to the/

the effects of focal infection such an infection has been suggested as the cause. This theory fits in with the nervous and toxic theories because a toxine absorbed from a focal infection may act on the trophic nerves to the scalp and so produce the lesion. CHIPMAN[1319.] in 1917 reported focal infections of teeth in cases of alopecia areata. In 1921 BARBER and ZAMORA[1337.] published some very suggestive cases where removal of septic foci, containing the streptococcus longus, in teeth, gums, tonsils or nasopharynx and the administration of an autogenous vaccine led to recovery or marked improvement in the alopecia areata. In their series of nine cases, they found infected tonsils with or without adenoids in 62%, oral sepsis alone in 5%, oral and tonsillar sepsis in 25%, chronic otitis media and naso-pharyngitis in 2%, chronic naso-pharyngeal catarrh in 4% and severe ethmoidal suppuration in 2%. In 11 cases of alopecia areata LESLIE ROBERTS[1324.] found evidence of bacterial collections in the tonsillar crypts and six showed no evidence of tonsillar infection. Of the five with cryptic collections three had tonsillectomy performed with good result in two of the cases. Of course as BARBER and ZAMORA point out, one must be careful in drawing conclusions in a disease such as alopecia areata, which runs such an indefinite course. Because the hair grows in again after a certain form of treatment, that is no proof that it was "propter hoc"/

hoc" and not "post hoc". In all cases of alopecia areata I now look for focal infections in teeth, tonsils etc. but in cases where visible foci have been removed I cannot say that the results have been very startling. However the theory that a toxine is absorbed from some focal infection and acts on the sensitised nerves to the scalp resulting in the fall of the hair has much to recommend it and time and careful observation in a large series of cases should enable us to prove or disprove it.

LICHEN PLANUS.

As in alopecia areata the microbic, nervous and toxic theories have had their supporters from time to time; although no microbe has been found, the supporters of this theory base their belief on the fact that the eruption often occurs in the line of scratch marks suggesting a local inoculation. Arsenic, antimony and mercury, all drugs which are useful in Syphilis, are also useful in Lichen planus. Sudden shock, worry and anxiety also frequently precede the eruption. The severe itching also suggests the implication of the nervous system and THIBIERGE and RAVAUT /344. and PERNET /339. report the marked effect of lumbar puncture on the itching. The eruption in some cases appears to follow the line of nerve distribution. In the/

the acute cases the sudden appearance of the eruption like an exanthem has been held to support the toxic theory but it also supports a microbic cause.

In a discussion on focal infection in 1917 1342. SUTTON mentioned a case of lichen planus, in which removal of several apical abscesses in teeth, was followed by a cure. CHIPMAN also states that Lichen planus is frequently associated with dental abscesses but WHITFIELD cannot corroborate this. LESLIE ROBERTS also records a case of Lichen planus with marked pyorrhoea in which removal of all the teeth resulted in the eruption completely disappearing about 20 days later. The material on which to form an opinion is still too meagre and as in alopecia areata so in Lichen planus, more evidence is necessary before it can be determined whether or not focal infection is the causative factor.

PRURIGO (of Hebra).

Prurigo is a disease which suggests a sensitisation phenomenon. It begins with an eruption like Urticaria papulosa and continues more or less all the life of the patient. Eosinophilia in the blood is also quite regularly present. Alternating attacks of asthma may also occur with Prurigo and I think that practically/

practically all the so-called eczemas alternating with Asthma are really cases of Prurigo.

In 4 cases of Prurigo in which I did cutaneous tests to all the common foods, 3 were absolutely negative but one gave positive reactions to egg-white, egg yolk and cod-fish. Removal of these articles from the diet caused no improvement in the eruption and feeding the patient on them did not aggravate the condition. These cases will be referred to again later in discussing cutaneous food tests. SCHWARTZ 1345. thinks that in Prurigo there is evidence of protein putrifaction as shown by the presence of indicanuria and states that attacks of eruption follow a high protein intake. As sensitisation may be produced by the alimentary route by overfeeding with protein, SCHWARTZ thinks that food protein plays an important part in the production of Prurigo.

The disease in sheep known as "scrapie" is somewhat like prurigo. The animals rub and scrape themselves especially on head, neck and limbs producing thickening of the skin and enlarged glands as in prurigo. McGOWAN showed that scrpaie is due to a sarcosporidium encysted in the muscles of the limbs near their insertions into bone. The toxine circulating seems to cause the skin itching. In a case of prurigo Mr. D.P.D. WILKIE kindly removed a piece of muscle /

muscle from the elbow region for me, but no sign of sarcosporidium was found.

1325.

LESLIE ROBERTS reports a case of long standing Prurigo who had septic collections in the crypts of the tonsils. Tonsillectomy followed by the administration of an autogenous vaccine of staph. aureus. and streptococcus longus caused an extraordinarily rapid improvement in the skin condition, so that in three months the case was practically cured.

In 1920 in a typical case of Prurigo of 11 years duration in a boy aged 14 years, under Sir NORMAN WALKER'S care enlarged tonsils and adenoids were removed but with no result on the eruption and now (1923) his skin is in much the same condition as previously. Some other focus, which was not detectable, may however have been present.

Prurigo suggests the circulation of some toxine acting on a sensitised skin. LESLIE ROBERTS succeeded in removing the focus of infection in his case but the tonsil is evidently not the only possible source of toxine. Further investigation of these cases on the theory of focal infection will probably disclose its true nature.

PRURITUS/

PRURITUS ANI and VULVAE and GENERALISED PRURITUS.

It is a question whether pruritus, general or local, should be included under focal infections. There is no direct evidence for or against it, but it is known that in certain blood diseases such as leucaemia, in toxaemias like diabetes, gout, rheumatism, etc. severe itching may occur. Certain foods produce itching on some individuals such as shell-fish, mustard, coffee etc. Jaundice is also another well known cause. These are probably examples of toxines using the word in its widest sense, irritating the nerve-endings in the skin and so causing itching. As the itching only occurs in certain individuals it suggests that there is some change in the nerves possibly of the nature of a sensitisation. In Dermatitis venenata where the skin is sensitised itching is one of the most prominent features. Many cases of generalised pruritus occur in elderly persons with arterio-sclerosis, which would suggest that the same toxaemia which caused the arterio-sclerosis also causes the pruritus. I have seen several cases markedly benefited by a course of internal treatment at Harrogate. In the local forms (pruritus ani and vulvae) it has been suggested that the lesion is due to an infection with an organism in that region. MURRAY 1346. in 19 cases of Pruritus ani found the streptococcus in the skin in all cases/

cases and WINFIELD[1347] in 50 cases found streptococcus and B. Coli alone or mixed in 40 of them. Both observers obtained remarkably successful results with autogenous vaccines. It is possible that in these cases a local toxine is produced to which the nerve-endings become sensitised.

SENSITIVENESS/

SENSITIVENESS of the SKIN to LIGHT.

There are two diseases in which the eruption is due to the effect of the actinic rays of light, viz:-

(1) Hydroa Vacciniforme or Aestivale, and

(2) Pellagra.

HYDROA VACCINIFORME OR H. AESTIVALE.

Under this head we do not include the ordinary Dermatitis Solare seen normally from exposure to strong sunlight. This dermatitis is followed by a protective pigmentation of the skin. Nor does it include the chronic atrophic condition of the skin with freckle-like pigmentation and atrophy seen in white persons who are exposed for years to strong sunlight such as occurs in the Tropics.

Hydroa aestivale occurs in two forms. The severe form shows vesicles and bullae on face, ears and hands on exposure to the light. From their appearance BAZIN named the disease H. vacciniforme. The lesions leave scars which may contract later and cause deformities.

The mild form of the disease was described first by Sir JONATHAN HUTCHINSON under the name of Summer prurigo. The lesions consist of small papules resembling/

resembling those in a papular dermatitis. Both forms usually begin in childhood and recur every summer till adult life when it tends to disappear. Cases have been recorded and I have myself seen one, which did not develop till adult life. The eruption is due to the actinic rays of the light as can be demonstrated by exposing the skin to sunlight or artificially produced ultra-violet rays.

One of the most recent articles on the subject is by SENEAR and FINK[371], who review all the 80 hitherto published cases. Males are more often affected than females in the proportion of two to one. Haematoporphyrinuria occurs in a certain number of cases. SENEAR and FINK state that in all the 80 published cases it was only present in 17·5% of them. But as it may only be present temporarily or in other forms it is probably a much more constant factor than those figures ihdicate.

Some two years ago when going through the literature on urticaria and its relation to sensitisation I came across an article by H.L. SMITH[372] in which he reported a case of urticaria, angio-neurotic oedema and vomiting in a boy, whenever he ate buck-wheat. He gave a local cutaneous reaction to buck-wheat grain applied to a scarified area. In this article SMITH refers to buck-wheat posioning or Fagopyrismus which occurs/

occurs in white or white spotted animals that have been fed on common buck-wheat (Fagopyrum esculentum). The disease is commonest in swine and sheep but occurs occasionally in cattle and goats and very rarely in the horse. Clinically the milder form of the disease is associated with an itching erythema chiefly of head and face, constipation and digestive disturbances. The more serious cases show a vesicular, pustular or gangrenous dermatitis with fever or urinary phenomena. White or spotted animals are said to be exclusively affected. Those that are black or artificially blackened escape the disease, and in black and white animals only the white parts are affected by the dermatitis, the black areas remaining quite normal. The worst cases of buck-wheat poisoning are seen in animals that have been fed on the buck wheat plant when in bloom but the disease may develop after eating the grains, bran chaff, straw or stubble. Sunlight is the exciting cause. If the animals fed on buck wheat are kept under shelter or allowed out under cloudy skies they seldom develop the disease, and if they do only in the mild form. But if the animal is put out in a strong sun it rapidly develops the symptoms including the dermatitis on the white parts of the skin only. The interesting fact of this disease is/

is that buck wheat contains phyloporphyrin, a derivate of chlorophyl, which closely resembles chemically haematoporphyrin and mesoporphyrin. It shows an almost identical absorption spectrum with these two haematin derivatives. When phyloporphyrin and haematin are reduced by concentrated hydrochloric acid they both yield haemopyrrol and haemopyrrol is converted into hydrobilirubin by the action of sunlight.

It is obvious that these facts throw some light on the causation of Hydroa vacciniforme in which haematoporphyrin occurs in the urine and presumably in the blood. EHRMANN[1350.] in 1909 seems to have been the first to suggest that Haematoporphyrin in Hydroa vacciniforme acts as a sensitising substance and is the cause of the skin eruptions. SACHS and SACHAROFF[1370.] had previously found that red blood corpuscles in solutions of photodynamic substances were rapidly haemolysed whilst control suspensions kept in the dark remained unchanged. PFEIFFER[1369.] confirmed this observation. HAUSMANN[1357.] had also previously shown that, under the influence of light, rapid haemolysis occurs when the extracts of chlorophyl plants are added to red blood corpuscles but when kept in the dark no haemolysis takes place. He also found that haemolysis results when bile is allowed to act on red blood corpuscles in the presence of light and haematoporphyrin has /

has even greater sensitising properties than bile when activated by light. HAUSMANN also injected white mice with pure hydrochloride of Haematoporphyrin and found that on exposure to sunlight, they scratched themselves, their skin become red and oedematous; they become restless and in some cases died. Therefore in Fagopyrismus the phyloporphyrin and in Hydroa vacciniforme the haematoporphyrin circulating in the blood acts as a sensitiser, like photodynamic substances, when exposed to sunlight.

A good deal of work has been done on photodynamic substances. TAPPEINER[1375.] showed that acridin hydrochloride, which is a substance which shows marked fluorescence, in the presence of day-light is able to kill Infusoria, whereas in the dark it is absolutely harmless. The same results were obtained with other fluorescent substances. TAPPEINER called these substances "photodynamic sensitisers". HERXHEIMER and NATHAN[1360.] group eosin, erythrosin, carboneol (tar preparations) and other fluorescent substances together with haematoporphyrin as photodynamic substances. Painting the skin with these substances has been used to increase the effect of light in the treatment of skin diseases where a marked reaction was desired.

SOBERNHEIM/

SOBERNHEIM[1373] in 1892 showed that after prolonged administration of Sulphonal in man haematoporphyrinuria appeared and NEUBAUER[1366] also demonstrated the same fact in rabbits.

PERUTZ[1368] in 1917 fed a rabbit on increasing doses of sulphonal till haematoporphyrin appeared in the urine and then exposed its ear to the Kromayer lamp for 3 minutes only. This resulted in redness, swelling and blistering of the skin, whilst control animals showed absolutely no reaction at all. This experiment was repeated several times and always with the same result. The reaction areas crusted over and healed with scars. PERUTZ fed the same animal again on sulphonal till haematoporphyrin appeared in the urine and then exposed the ear scarred from a previous reaction and also the other ear to the Kromayer lamp and obtained a reaction on the second ear but not on the scarred one. This is explained by the absence of circulation in the skin of the scar. GOTZL[1353] also found that by injecting Lead triathyl he could produce haematoporphyrinuria in animals and these animals were very sensitive when exposed to ultra-violet rays. MEYER-BETZ[1365] experimented on himself. As haematoporphyrin is not absorbed when taken by the mouth he injected 0·2 haematoporphyrin intravenously into himself and then exposed an area of skin on his right arm to the Finsen light. This produced a reaction which resulted in haemorrhage/

haemorrhage into and sloughing of the skin, which only healed with scarring after several weeks. On the day after the injection he exposed himself for a short time to the sun which caused oedema and redness of hands and face which lasted several days and left marked pigmentation.

Haematoporphyrinuria is a symptom of liver-insufficiency. Under certain conditions a reduction product of haematoporphyrin called mesoporphyrin may appear. Mesoporphyrin has nearly the same spectrum absorption bands as haematoporphyrin, and FISCHER thinks the two substances should be combined under the one name "Porphyrine". FISCHER, BARTHOLOMAUS and ROSE[135] succeeded in finding the mother substance of porphyrin. They called it porphyrinogen. By oxidation porphyrinogen becomes mesoporphyrin. It is known that in many cases of Hydroa vacciniforme and Aestivale the haematoporphyrin was only present in the urine at certain times, but PERUTZ showed that if one oxidises the urine with Potass. permang. it gives the spectrum of haematoporphyrin showing that the urine has porphyrinogen in it. Porphyrinogen is colourless and therefore in all suspected cases of light dermatitis the urine especially if it is not dark in colour should be tested for porphyrinogen as well as haematoporphyrin.

GUNTHER/

GUNTHER[1355] distinguishes different causes for haematoporphyrinuria. In the cases appearing early in life there seems to be some congenital functional or organic defect of the liver which allows the haematoporphyrin to circulate and in the cases appearing later in life the defect in the liver may be acquired. KONIGSTEIN and HESS[1363] report a case of Hydroa vacciniforme in a boy due to congenital syphilis of the liver leading to haematoporphyrinuria and GUNTHER[1356] records an acquired case due to alcohol affecting the liver. There are also the cases due to sulphonal and lead poisoning. The eruption in Hydroa vacciniforme is provoked by the ultra-violet rays of the spectrum. MARTENSTEIN[1364] found that all the ultra-violet part of the spectrum produced the eruption but not the α, β, or γ rays of X-rays.

As showing the effect of ultra-violet rays on the serum, BARONI and JONESCO[1348] showed that these rays could increase the antisensitising properties of horse serum. DOERR and MOLDOVAN[1349] found that the rays could also disturb the faculty of the serum to produce precipitins and passive anaphylaxis. They also demonstrated that if antigens and antibodies were exposed to ultra-violet rays, a diminution occurred in their reactive properties. WHITE[1376] and KANOKI[1362] both found an eosinophilia in the blood in cases of Hydroa vacciniforme. That fact supports the sensitisation theory.

During/

During the last two years four cases of Hydroa aestivale have been seen in Sir NORMAN WALKER'S department at the Royal Infirmary and in only one was haematoporphyrin present in the urine. In one the urine was mixed with sheeps red blood corpuscles and exposed to sunlight but no lysis occurred. In one private case of Hydroa vacciniforme in a girl age 6 years, no haematoporphyrin was found in the urine. Some blood was withdrawn from a vein and Dr. HEDLEY WRIGHT kindly tested it for me at the Royal College of Physicians Laboratory. The serum and red blood cells were separated and the latter washed. The serum was divided into quantities of 0·2 cc. undiluted and in dilutions of 1 in 5, 1 in 10, and 1 in 20 and mixed with 0·4 cc. of a suspension of the washed red blood cells. As a control similar dilutions of syphilitic serum and normal saline were used. One set of tubes was exposed to sunlight and the other kept in the dark. No haemolysis occurred in any in 12 or 24 hours but a slight " pinking" of the patient's serum dilutions occurred in 3 days; probably bacterial. This result was negative but the tubes used were ordinary glass ones and possibly did not allow sufficient ultra-violet rays through to affect the serum. As suggested by the cases of buck-wheat poisoning in animals, I went into the question of diet in this child. As there was no buck-wheat/

buck-wheat in the diet it was thought advisable to exclude ordinary wheaten flour and oatmeal from the diet. The child was given rice bread and scones and biscuits made from rice flour. The parents state that when on that diet the skin was certainly distinctly less sensitive to light than previously. This is only an isolated case but I think it is worth investigating in similar cases whether alteration in the diet may not cause improvement in such cases. A substance similar to the phyloporphyrin found in buck wheat may occur in ordinary wheat or oatmeal and be responsible for some cases of light dermatitis or Hydroa.

SUMMARY.

1. Hydroa vacciniforme or Aestivale is associated with Haematoporphyrin or porphyrinogen in the circulation and urine.
2. From analogy with buck wheat poisoning, this Porphyrinogen acts as a sensitiser and leads to the eruption.
3. The presence of a blood eosinophilia, haemolysis of red blood cells by photodynamic substances, the effect of ultra-violet rays on antigens and antibodies all favour the sensitisation theory.

4. /

4. The haematoporphyrinuria is probably due to some congenital or acquired defect of the liver.

5. The effect of certain substances in the diet requires further investigation in eruptions due to sunlight.

PELLAGRA/

PELLAGRA.

In Pellagra the skin eruption is produced by exposure to the sun. The disease is by no means rare in Scotland. Several cases are at present in Scottish Asylums and every now and then cases are seen at the Edinburgh Royal Infirmary. A few months ago a very typical case was admitted to Sir NORMAN WALKER'S Ward in the last stages of the disease. Casts (24, 25 & 26.) Plates (47 & 48.), show the head and arms of a typical case which was admitted to the Royal Infirmary some years ago.

I do not propose to go into the much discussed subject of the causation of Pellagra. Recent work supports the theory of a dietary insufficiency. There is evidence however that in Pellagra some toxine is circulating which when exposed to light causes the skin eruption. Experiments have been performed in Italy to try to demonstrate anaphylactic phenomena by injecting Pellagra patients and animals with extracts of maize. VOLPINO /383. in 1912 showed that injection of a watery extract of spoiled maize caused a definite reaction of hypersensibility with rapid pulse, rise of temperature, dyspnoea, diarrhoea, and aggravation of preexisting eruption. This reaction was not produced by injection of extracts of sound

maize /

maize nor by injection of spoiled maize in normal individuals. VOLPINO, MARIANI, BORDINI and ALFRAGO [1384] failed to produce passive sensitisation of guinea-pigs by injecting them with serum of Pellagra patients. CESABIANCHI and VALLARDI [1377] also found that guinea-pigs chiefly or entirely fed on maize, sound or spoilt, show after a certain time a very marked sensitiveness to injections of maize extracts. They found it to be a specific reaction only occurring in animals fed on maize. RONDONI [1381] also found a heightened sensitiveness in pellagra patients to injection of maize extracts.

Therefore there is some evidence that in pellagra a sensitisation may occur to some toxine in the food. From analogy with the effects of buck-wheat in animals, it is probable that in Pellagra some photo-dynamic substance is circulating which acts as a sen-sitiser of the skin but at present there in no evidence as to what that substance is.

At the meeting of the Dermatological section of the British Medical Association at Newcastle in 1921 Dr. KENNETH WELLS mentioned cases of old people, who were toothless and could not masticate and who lived chiefly on tea and bread and butter. These persons developed a scaly eruption on the hands when exposed to the sun.

I have also seen an eruption very like Pellagra /

(Cast 27, Plate 49.)

Pellagra (Pseudo-pellagra) in alcoholics. These cases suggest that excessive carbohydrate diet or excess of alcohol cause photodynamic substances to circulate and produce a skin reaction with a resulting eruption. In true Pellagra similar substances are probably responsible for the eruption but at present the exact cause of its production is largely speculative.

TESTS/

TESTS FOR CUTANEOUS HYPERSENSITIVENESS.

CUTANEOUS REACTIONS.

It is generally accepted now that the presence of a specific Cutaneous reaction to any given substance is a sign of hypersensitiveness to that substance. The different diseases in which these tests have been applied will be dealt with first and then the mechanism & significance of the reaction will be discussed.

The cutaneous reactions in Tubercle, Syphilis, Ringworm, Favus and other fungus infections, Dermatitis Venenata, Psoriasis and Gonorrhoea have already been dealt with under these diseases, but there still remains a considerable number of others.

CUTANEOUS REACTIONS IN TYPHOID INFECTIONS.

TYPHOIDIN TEST.

A great deal of work has been done on this subject and the results are rather conflicting as to the specificity of the reaction and its value in diagnosis. The results are not all comparable as different strains of bacilli, methods of preparation and time of reading the reaction have been used by different workers. ZUPNIK 1409. in 1908 was the first to apply the Pirquet test/

test using Typhoid antigen. Four cases of Typhoid fever all gave positive reactions and two cases who had had the disease many years before, gave slight positive reactions. Since then many others have used this method. A preparation called Typhoidin, analagous to Old Tuberculin, has been chiefly employed. GAY and FORCE[1392.] consider the reaction specific and indicating an existing immunity against typhoid and allied fevers. PULAY[1406.] supports GAY and FORCE. He obtained no reactions in healthy individuals but definite ones in typhoid cases and those convalescent from it. In persons inoculated against typhoid he obtained reactions but they were often slight. Patients who had had typhoid reacted more strongly than those only inoculated against it. KILGORE[1394.] also thinks the reaction specific and an indication of immunity. He considers the degree of reaction to be an indication of the degree of immunity present. LINK[1399.] also supports these views. There are many others, however, including CHAUFFARD and TROISIER[1387.], GOODMAN and SUTTER[1393.], ENTZ[1389.], SZONTAGH[1408.], ROLLY[1407.] and KRAUS[1396], who do not consider the reaction specific. CHAUFFARD and TROISIER obtained reactions in both typhoid cases and normal persons but the reaction was greater in the typhoid cases. KRAUS and STENITZER[1398.] by animal experiments showed that a toxine exists in the typhoid bacillus and also in filtrates of cultures.

ENTZ/

ENTZ[1389.] found that a high percentage of normal individuals react to various bacterial toxines including the typhoid toxine. SZONTAGH[1408.] tested 72 children suffering from different diseases, doing Pirquet tests with typhoid toxine, and only obtained marked positive reactions in 4 cases one of which was a case of typhoid, another of epityphlitis and two were cases of scarlet fever. The question has been much discussed as to whether the Typhoidin skin reaction is a sign of immunity or not. GAY strongly maintained that the reaction was evidence of immunity. GAY and CLAYPOLE[1391.] consider the reaction to have a distinct relation to protection against typhoid fever. They did comparative intradermal tests on normal rabbits and human beings with sensitised and unsensitised typhoid vaccines. They obtained, in most cases, greater reactions with the sensitised vaccines. This they attribute to an interaction between antigen and antibody similar to what occurs in the Schick test for diphtheria. Further experiments with rabbits indicate that in the condition of artificial immunisation against typhoid, the antibodies which combine with the antigen to produce the local reaction, are in the circulation as is shown by the passive transfer to a normal rabbit, by means of serum from an immune rabbit/

rabbit of susceptibility to this reaction. Also withdrawal of blood from an immunised rabbit and replacement with the blood of a normal rabbit leads to a loss of the reaction. They do not however regard these experiments as indicating the circulatory nature of the antibody in persons recovered from typhoid who almost invariably react to typhoidin.

MEYER, [401] and MEYER and CHISTIANSEN [402] found that in the rabbit which is an animal, which does not naturally suffer from typhoid, a positive typhoidin skin reaction does not indicate that the animal will resist a subsequent intravenous injection of typhoid bacilli. They found no definite relationship to exist between the presence of agglutinins and complement-fixing antibodies and cutaneous hypersensitiveness. Allergy to bacterial proteins may be demonstrated in rabbits even in the absence of demonstrable immune body. They think the cutaneous reaction to typhoidin in rabbits is due to sensitisation to the bacterial proteins and that it is not an antigen-antibody reaction.

KOLMER and BERGE [395] also found that agglutinins and complement-fixing antibodies are present in the majority of persons reacting positively to the skin test but that there is no definite relationship between them. Cutaneous reactions were found to persist for a longer time among those who had had typhoid than among those/

those actively immunised by a vaccine. They conclude that while the typhoidin reaction indicates sensitisation to typhoid protein, there is not yet sufficient evidence to warrant its acceptance as an index of immunity. NICHOLS (1404.) agrees with this because he found positive typhoidin reactions in soldiers vaccinated with paratyphoid vaccine and it is well known that paratyphoid vaccine will not immunise against typhoid. Therefore he accepts the protein sensitisation theory of the reaction.

AUSTRIAN and BLOOMFIELD (1385.) also failed to confirm GAY'S work that a positive skin reaction means immunity. The typhoidin test has also been used for the detection of typhoid carriers. MEYER found cutaneous hypersensitiveness to typhoidin to be most marked in rabbits infected with the typhoid bacillus. Carriers of bacilli in the gall bladder and liver develop skin reactions which apparently vary directly with the degree of the inflammatory process in the organ. McKENDRICK (1400.) in a recent article gives the results of a series of intracutaneous tests with suspensions of B. typhosus B. paratyphosus A. and B. These results show that positive reactions were very constantly obtained in persons suffering from typhoid fever and in chronic carriers. The test was found to be highly specific the cases only reacting to the type of organism causing the infection. Only 2 out of 360 control cases gave/

gave positive reactions to B. typhosus and none to B. paratyphosus. No relationship was found between the skin reaction and the presence of pyrexia in acute cases or between the skin reaction and Widal reaction in carriers. McKENDRICK'S results indicate that the skin reaction becomes negative early in convalescence from enteric fever. He suggests that patients convalescing from typhoid fever should be examined for the skin reaction. A positive reaction probably indicates persistence of the infection which may result in relapse or the carrier state. McKENDRICK considers a positive skin reaction in apparently healthy persons as suggestive of their being typhoid carriers .

PNEUMONIA.

140. CLOUGH in 1915 studied allergy in pneumonia. He employed the dried and ground residue of extracts of washed pneumococci before and after precipitation with absolute alcohol. Cutaneous and intracutaneous tests with these extracts on persons suffering from pneumonia yielded variable and inconstant results. CLOUGH thought the results were due to the irritant qualities of the extracts and was of opinion that it was impossible to demonstrate a state of hypersensitiveness to pneumococcus protein by these tests.

141. STEINFIELD and KOLMER did intradermal tests with/

with pure cultures of pneumococci shaken and emulsified with salt solution. 0·1 cc. of emulsion was injected and positive reactions consisted in the formation of a definite papule with an area of erythema of more than 1 cm. in diameter. The reaction persisted for 4-5 days. Positive reactions were observed in 30% of 19 cases of lobar pneumonia. True reactions were not observed among normal persons or those suffering from various chronic diseases. The presence of pneumococci in the upper air passages during health does not sensitise the individual so far as could be detected by skin tests.

WEISS and KOLMER [414] used the endocellular haemolytic toxin of the pneumococcus freshly prepared for each test. Intracutaneous injections in doses of 0·1 cc. led to a local erythema and haemorrhagic oedema. These reactions were seen on the 5th-13th days of the disease, i.e. two days before and six days after the crisis, but patients recovering by lysis reacted as late as the 32nd day. In children the reaction became negative immediately or two days after the crisis. In no case did any control react positively. In general, the reaction was positive in all active cases of pneumonia.

WEIL [412] also did a series of intracutaneous tests using a much weaker extract than did CLOUGH. The/

The injection was immediately followed by a cutaneous blush which was present both in pneumonia cases and controls. This he considered an irritation reaction similar to that seen by CLOUGH. The true reaction occurred after 24 hours as a papule with an area of erythema round it. WEIL obtained no true positive reactions during the course of the disease but in a considerable percentage of cases reactions were found after the crisis. The reaction could be induced exceptionally within 24 hours after the crisis, but usually only appeared after an interval of two or three weeks. Normal individuals sometimes gave a positive reaction presumably from a previous mild or unidentified attack. Contrary to the experience of STEINFIELD and KOLMER, WEIL suggests also that these reactions in normal individuals might be due to the presence of the pneumococcus in the upper air passages. WEIL thinks that two types of skin reaction occur from pneumonia vaccines. The one (as shown in CLOUGH'S work) is due to the toxine and similar to the Schick reaction in diphtheria. A positive reaction of this type indicates a deficiency in the mechanism of defence. The other is a true reaction, which is a sensitisation phenomenon, the same as in the Tuberculin skin reactions. He thinks that the absence of a true reaction during the disease is not due to absence of antibody but to the coexistence of sufficient antigen in/

in the cells to prevent the reaction. After the crisis when the antigen has disappeared, antibody becomes available for the production of the reaction. He considers the absence of the reaction during the febrile stage of the disease as analagous to the absence of the Pirquet reaction in acute miliary tubercle and as it does not occur till after the crisis it has no value in diagnosis.

PERTUSSUS/

PERTUSSUS (WHOOPING-COUGH).

MODIGLIANI and DE VILLA [1415] in 1921 did a series of intracutaneous tests using a solution of Bac. Pertussis in sterile distilled water. Autolysed B. Coli were used as control. In 38 children suffering from whooping-cough all reacted positively. The intensity of the reaction was greatest in the early stages of the disease. In 58 immune children there were no positive reactions.

ORGEL [1416] did similar intracutaneous tests with a vaccine of B. Pertussis containing two billion organisms to each cc. He obtained reactions, in cases of whooping cough, which he considers specific.

RIESENFELD [1417] in 1923 used a Pertussis vaccine of the Bordet-Gengou bacillus. In 60 cases of whooping-cough 53 reacted positively. 39 cases of whooping cough were also tested intracutaneously with staphylococcal vaccine and 35 of them reacted positively. He found that the reaction to B. pertussis vaccine was not specific as it was positive in some children who developed the disease later. The test was found also not to be a reliable guide as to a natural or acquired immunity to the disease.

LEPROSY.

STEIN [1419.] in 1916 published a very good record of the work done on sensitisation in Leprosy. BABES and SCOLTZ and KLINGMULLER [1419.] made an extract from leprous nodules called Leprin similar to Tuberculin. Injection of this extract caused no reactions in cases of leprosy. Then BABES and others used Tuberculin as the Tubercle bacillus is allied to the Leprosy bacillus. Injection of Tuberculin subcutaneously lead in some cases to a general reaction with temperature but he found that it required a larger dose of Tuberculin to produce a temperature reaction than in tubercle. In no case has a focal reaction in Leprosy been seen after injection of Tuberculin although local and general reactions may occur. Many workers such as STEIN and AINING [1419.] consider the local and general reaction to Tuberculin in Leprosy cases to be due to a concomitant tuberculosis. ROST, DE BEURMANN and GOUGEROT [1419.] used cultures of a glycerin extract of Leprosy bacilli (Leprolin) which on injection caused general and focal reactions in both forms of leprosy. DYKE, MUCH [1419.] and others made similar injections of Nastin (extract of streptothrix leproides) and obtained general reactions. But as these general reactions might be due to toxines contained in the extracts, it is doubtful if these reactions indicate sensitisation. PHOTINOS and MICHAELIDES [1418.] did Tuberculin Pirquet tests in/

in 204 Leprosy cases and obtained positive reactions in 118. NICOLLE and TEAGUE[1420.] obtained negative cuti-reactions using a leproma extract. Therefore STEIN "concludes that, as regards allergic reactions in leprosy, cutaneous and subcutaneous inoculation with Leproma extracts produces no reaction. Inoculation with Tuberculin, Leprolin and Nastin often leads to marked reactions on later injection but their specificity is doubtful".

STEIN also reports numerous cases of the so-called Leprosy erysipelas. These cases show a sudden high temperature, with headache. In a few hours a bright red area appears on the skin. It is sharply defined and spreads rapidly. The redness diminishes and is replaced by a paler flat infiltration like erysipelas but with a less defined margin. If it persists, blisters (which contain no leprosy bacilli) appear on the surface. Some of them change into lesions like furuncles, with ulceration and loss of substance. The pus from these lesions contained countless acid-fast bacilli. The infiltration gradually disappears from the lesions and scars may be left but STEIN never saw leprosy nodules develop on these areas. One erysipeloid attack was followed by another. STEIN did intradermal tests using an extract from leprous lymph glands and two cases gave marked positive reactions. These/

These reactions were not present after the attack. He therefore concludes that the so-called leprosy erysipelas is due to an allergic condition of the skin and corresponds to the erythematous and papulo-necrotic tuberculides of tubercle.

The fact that in the ordinary case of nodular leprosy the lesions are teeming with bacilli is very much against the patient being sensitised. If he were sensitised the presence of the bacilli in the skin would cause a more marked reaction, which would inhibit the growth of the organism. In tubercle of the skin very few organisms are present in the lesions because the sensitisation reaction tends to check their growth and spread. Similarly in tertiary syphilis the gumma contains very few spirochaetes.

Therefore from the negative results of attempts to produce cuti-reactions with leprous extracts and the presence of large numbers of bacilli in the lesions, if any sensitisation occurs in leprosy it must be very slight. The various reactions in leprosy from Tuberculin are not a reliable guide as it is admitted that many cases of leprosy are also infected with tubercle.

HYDATID/

HYDATID DISEASE.

The rashes produced in cases of hydatid disease from rupture of a cyst have already been referred to p. (79.). The fact that these rashes occur show that the skin is sensitised. It is, therefore, to be expected that such cases will give a skin reaction. SERRA[422.] recommends the intradermic test, introduced by CASONI, as an accurate method of diagnosing hydatid disease. 0·5 cc. of clear fluid from a hydatid cyst is injected intradermally into the skin of one arm and the same quantity of normal saline solution as a control into the other arm. In positive cases within a few hours an extensive erythema with oedema appears round the area. There is also usually considerable itching. The reaction takes several days to subside. It has never been found positive in any other condition than hydatid disease, but it may be absent, if the cyst wall is abnormally thick or calcified, so that there is not sufficient absorption from the cyst to sensitise the patient. The test is extremely useful in the diagnosis of doubtful cases. A positive result may be taken as proof of the disease but a negative result does not absolutely exclude it.

INFECTIOUS DIARRHOEA.

1424.
BAKER in 1917 did intracutaneous tests with the organism of infectious diarrhoea in 33 cases. The reaction was positive in 85% of the cases and negative in all the controls.

ULCERS DUE TO PENICILLIUM GLAUCUM.

1429.
REBAUDI and PODESTA report a case of multiple ulcers of the legs in a girl, 14 years of age. From the ulcers a culture of Penicillium glaucum was obtained. A filtrate of the fungus culture gave a positive intradermic reaction.

MENINGOCOCCUS INFECTIONS.

122.
KARSNER and ECKER quote GAY and MINAKER'S work on the intracutaneous reaction for the detection of meningococcus carriers. They used an emulsion of powdered meningococci of five strains. They obtained reactions in 64·5% of known carriers and in 26·4% of non-carriers. They do not consider the reaction useful in diagnosis but suggest that in carriers there is some degree of acquired resistance.

CUTANEOUS/

CUTANEOUS REACTIONS TO VACCINE VIRUS.

In his work on vaccination JENNER noticed that in persons who had been previously vaccinated, a second vaccination produced a mild local reaction only. Pirquet[1504.] investigated the matter thoroughly and showed that on revaccination the patient did not develop the usual vaccine vesicles but showed a reaction with papule formation and erythema which subsided in a few days. This is an example of allergy. An attack of small pox or vaccinia by vaccination alters the reactivity of the skin so that it becomes sensitised and any further attempt at revaccination produces a reaction analagous to the Tuberculin Pirquet reaction. These are the cases of vaccination which do not "take". The reaction is often called an immunity reaction but it is really a sign of hypersensitiveness. The reaction is protective as it prevents a reinfection with vaccinia.

MALIGNANT DISEASE.

[1423.] RAVENNA in 1912 took a non-ulcerative Carcinoma of the breast, cut it up and pounded it in a mortar and added physiological salt solution 1 in 4. This was shaken for 24 hours, filtered in a wide-pored filter/

filter and the filtrate used for skin tests. Twenty-four patients with Carcinoma of lip, larynx, aesophagus, nose, rectum, breast, stomach or pancreas were tested by the Pirquet method. Only two gave positive reactions. The other 22 cases, although the test was repeated, were negative. In 20 control cases suffering from diseases other than Carcinoma the tests were all negative. In 21 cases of Carcinoma tested by the intradermal method 6 reacted positively and 12 control cases were all negative.

Further evidence is necessary before any conclusions can be drawn as to the value of the test.

ULCUS MOLLE. SOFT SORE.

1430.
ITO in 1913 did a number of experiments with DUCREY'S bacillus. Rabbits were injected every five days for three times with an emulsion of killed strepto-bacilli. A fortnight later intracutaneous injection of the strepto-bacillus emulsion gave positive reactions which did not occur in control animals. In man similar injections of strepto-bacillus vaccine in an individual who had never had soft sore caused the appearance of a marked reaction on testing the skin intradermally ten days later. In eleven cases of ulcus molle with buboes all gave a strong positive reaction to the intradermic test. ITO considers the reaction/

reaction specific and an aid in confirming the diagnosis of soft sore.

ITO found that by previous treatment with strepto-bacillus vaccine no active immunity can be produced, but, as already shown, by previous treatment with strepto-bacillus vaccine or by a previous soft sore infection hypersensitiveness may result. Passive anaphylaxis can be produced both by the serum of guinea-pigs sensitised to the strepto-bacillus and by the serum of patients with buboes. The precipitation, agglutination and complement-deviation tests both with patient's serum and the serum of previously vaccinated animals did not yield any noteworthy results.

CUTANEOUS TESTS IN PREGNANCY.

ENGLEHORN[1425] and WINTZ in 1914 did cutaneous tests with extracts of placenta in cases of pregnancy. They claimed to obtain favourable results as to the specificity of the reaction. ESCH[1426] and DE JONG[1428] made similar tests but could not confirm these observations. Likewise FALLS and BARTLETT[1427] made cutaneous and intracutaneous tests with whole placenta and various fractions of placenta. Like ESCH and DE JONG they failed to find any evidence of specific sensitisation. They conclude that the pregnant woman is certainly/

certainly not a sensitised woman in the usual sense. STOKES explains the non-specificity of these reactions on the anti-ferment adsorption theory of sensitisation. He quotes EGGSTEIN and PETERSEN'S experimental work on the antiferment-adsorption capacity of the placenta. In pregnancy there is known to be a rise in antitryptic-titer in the serum which also supports the theory of antiferment-adsorption and might explain the want of specificity in the reaction.

CUTANEOUS REACTION IN CANINE DISTEMPER.

KOLMER, HARKINS and REICHEL [431] in 1916 did intracutaneous tests in dogs with distemper using the Bac. Bronchisepticus (Ferry, M'Gowan) which is the probable cause of Distemper. The highest percentage of positive reactions occurred among dogs suffering from distemper when the tests were made and amongst those known to have had distemper. These observers think that the test may prove of value in the diagnosis of distemper and as an index of previous infection in an animal. The test has probably no value as an index of immunity as an animal showing a positive reaction is still susceptible to a relapse of the disease.

SCHICK/

SCHICK TEST FOR DIPHTHERIA.

The Schick test is not really comparable with the cutaneous tests being discussed, as it is not a sensitisation reaction but depends on the presence or absence of the diphtheria antitoxine. The so-called pseudo-reaction in this test was first described by PARK, ZINGHER and SEROTA. /434. It consists in a small area of redness and infiltration at the site of injection within 24 hours. Injection of bouillon alone, gives this reaction. They consider it an allergic reaction due to the proteins in the broth used to prepare the diphtheria toxine. KOLMER and MOSHAGE /433. and WEAVER and MAHER /435. agree with this view. These pseudoreactions can be detected by doing control tests with the broth only.

CUTANEOUS PROTEIN TESTS IN ECZEMA, URTICARIA ETC.

Before discussing these tests I propose to put on record my own experiences with them. I used the Arlington Chemical Company's products in powder form. A small rounded burr was used to scarify the skin short of drawing blood; a drop of deci-normal sodium hydrate solution was placed on this with a glass rod, then a few grains of the protein powder on a thin glass rod were mixed with the sodium hydrate and/

and gently rubbed in. The powder dissolves immediately in the alkaline solution. The tests were done as a rule on the front of the forearms, the control, into which the sodium hydrate solution only was rubbed, being placed near the elbow and the others lower down in two or three rows at intervals of about an inch. The control was placed near the elbow, as I can confirm the observation of other workers, that that area of skin is slightly more sensitive to irritation than the rest of the arm. Fifty different proteins in all were used. Most of the cases were tested to about 30 of these, a selection being made of the substances most likely to react according to information obtained from the patient. Patients were not tested to foods which they said they never ate. Although the tests are quite easy to carry out, the chief difficulty which I had, was to know what constitutes a positive reaction. No reaction was considered positive unless it was at least twice the size of the control. Reactions only very slightly larger than the control were marked as doubtful and looked upon as probably negative. Some of these doubtful reactions were repeated and in every case turned out to be negative. When a positive reaction was present there was no doubt about it (Cast 28.) (Plate 50.). An irregular wheal was produced with a wide area of erythema spreading out/

out irregularly in all directions for a considerable distance. A reaction was not considered positive unless both wheal and erythema were present. A wheal alone or erythema alone was not considered positive. The reaction was usually quite distinct in 10 minutes, at its maximum in 15-20 minutes and was fading in a little over half an hour. I have never seen delayed reactions as described by FREEMAN 1448 and others although the cases were carefully examined for them. Each worker has his own standard of what constitutes a positive reaction. C.J. WHITE 1475. considered a papule sufficient but as he often found this on the control it is obviously a false standard. STRICKLER and GOLDBERG 1470. insist on the reaction lasting 48 hours before they consider it positive, but these were intradermic and not the ordinary cutaneous tests. SCHLOSS, 1462. TALBOT, 1471. SMITH and others considered the reaction positive even although lasting about half an hour or so. FOOTE 1446. thinks the reaction is not positive unless it is three times the size of the control. But most of those, who have used the tests extensively and especially in asthma are agreed that the reaction is a wheal at least half as large as the control surrounded by a zone of erythema.

My results are shown in tabular form. 34 cases were tested, consisting of 21 cases of chronic dermatitis (eczema) 4 of Prurigo, 8 of Urticaria including urticaria papulosa and one of Dermatitis Herpetiformis.

DERMATITIS/

TABULATED RESULTS

of

CUTANEOUS PROTEIN TESTS

in

34 CASES of SKIN DISEASE.

POSITIVE REACTIONS are marked +
NEGATIVE " " " −
DOUBTFUL " " " ±

Spaces indicate that no test was made to that particular protein.

SEX and AGE. / NAME.	DERMATITIS. (ECZEMA)																					PRURIGO.				URTICARIA.								
	M 15/12 K.G	M 5/12 J.A.	F 3 M.A.	F 18/12 W.	M 9/12 M.NS.	M 5 H.H.	F 7 N.S	F 10 D.P	M 11 G.W.	M 14 J.M.	M 9 J.F.	M 7 H.B.I.	F 22 M.C.	F 22 A.S	F 22 M.L.	M 27 A.B.	M 32 R.G.	M 40 B.B.	M 55 Mc F.	F 60 Mrs D.	M 22 E.A.	M 8 D.B	F 18 H.W.	M 32 R.B.	M 42 W.M.	F 2 9/12 R.M.	M 3 A.S.	M 4 J.T.	F 26 C.L.	M 30 A.S.	F 35 Mrs K.	F 65 R.D.	F 89 Mrs B.	M 56 W.T.
CONTROL	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
EGG-WHITE	−	−	−	+	−	−	+	±	−	−	±	−	−	−	−	−	−	−	−	−	−		+	−	−	−	−	−	−	−	−	−	−
EGG-YOLK	−	−	−	−	−	−	+	±	−	−	−	−	−	−	−	+	−	−	−	−	+	−	+	−	−	−	−	−	−	−	−	−	−
MILK (COW'S)	−	−	−	−	−	−	±	±	−	−	−	−	−	−	−	−	−	−	−	−	−		−	−	−	−	−	−	−		−	±	−	−
LACTALBUMIN	−				−									−																				
CHEESE							−	−	−	−	−	−	−	−	−	−	−	−	−	−	−		±	±	−				−		−	−		−
CHICKEN			±			+	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+	−	−		±		−	−	−	−	−	−
VEAL							−		−	−	−	−	−	−	−	−	−	−		−	−		±	−	−			−	−		−	±		±
BEEF	−	−	−	−	−	±	+	−	−	−	−	−	−	−	−	+	−	−	−	−	−		−	−	−	−	−	−	−	−	−	−	−	−
PORK		−	−					−	−	−	−	−	−	−	−	−	−	−	−	±	−		−	−	−	−	±		±	±	−	−	−	−
LAMB		−				±	−		−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−			−	−	−	−	±	−	−
HERRING						−	−		−	−	−	−	−	−	−	−		−	−	−			−	−	−				−		−	−		
HADDOCK	−			−		−	−	−	−	−	−	−	−	−	−	−		−		−	−	−	−	−	−		−		−		−	−		−
COD										−		−	−		−	−	−				−	−			−						−	−		−
SALMON							−	−	−	−	−	−	−	−	−	−	−		−	−		−	+		−				−		−	−		
SOLE								−	−	−	±	−	−	−		−	−			−				−	−						−	−		
MACKEREL									−					−						±		−			−									
LOBSTER														−											−						−			
CRAB								−	−		−			−															−		−			
OYSTER														−																	−	−		−
WHEAT	−		−	+	−	±	−	−	−	−	−	−	−	−	−	+	−		−	−	−	−	−	−	−	−	±	−	−	−	−	±	−	
RICE	−			−		−		−	−	−	−	−	−	−	−	−	−	−	−	−	−			−	−		±		−		±	−		
BARLEY				−			−	−	−	−	−	−	−	−	−	−	−		−	±			±	−	−		−	−	−		−			±
OAT	±	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	±	−			−	−	−	−	±		−		−			−
PEA								−	−	−	±	−	−	−	−	−	−			−	−		−	−	−		±		−		−	−		
BEAN							−	−	−	−	−	−	−	−	−	−	±			−	−		−	−	−		±		−		−	−		
LENTIL						−			−	−	±	−	−	−		−	−		−		−	−	−	−				−						
POTATO	−		−	−		−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−		−	−	−		±	−	−		−	−	−	+
BEETROOT																	−			−	−													
CABBAGE				−			−	−	−	−	−	−	−	−	−	−	−		−	−			−	±	−		±		−		−	−		
CARROT				−			−	−	−	−	−	−	−	−	−	−	−		−	±	−		±	−	−		±		−		−	−		
CELERY								−				−	−	−			−														−			
CUCUMBER								−						−					−	−					−						−			
STRAWBERRY						−		−	−	−	−	−	−	−	−		−			−	−				−				−					
TOMATO								−	−	−		−	−	−	−	+	−			−	−		−	−					−		−			
BANANA					−			±	−	−		−	−	−	−	−	−			−					−			−	−		−			
ORANGE					−				−		−	−					−				−	−									−			
ALMOND								−	−			−		−	−					−														
BRAZIL NUT														−	−						−													
COCOA							−	−	−		−	−	−	−	−		−				−	−	−		−	−					−			
CATTLE HAIR				−	−							−						−			−				−									
CHICKEN FEATHER				−	−		−			−		−	−				−	−	−		−				−									
DOG HAIR									−		−	−					−	−			−				−									
GOOSE FEATHER																			−		−													
HORSE DANDER				−							±	−					−	−			−				−									
RABBIT HAIR												−								−	−				−									
SHEEP'S WOOL					−				−			−	−		−		−	−		−	+				−									
TURNIP															−										−									
ONION															−								−		−									
TEA															−				−				−	−					−		−			−
SUGAR																							−								±			

DERMATITIS CASES.

1. Five of these cases were children under 3 years of age, with the ordinary infantile eczema. Two gave an entirely negative result. 2 gave doubtful reactions and one was positive to egg white and wheat. Cutting these articles out of the diet of the last case however had no effect on the dermatitis.

2. Of the seven cases between the ages of 5 and 14 years, 3 were entirely negative, 2 gave doubtful (probably negative) reactions and 2 gave definite positive reactions. Of the negative cases one (J.M. age 14) a very chronic and extensive dermatitis, later recovered completely after a course of intestinal lavage at Harrogate so that he was probably not a food case but a sensitisation to a bowel infection. Another of the negative cases (H. B-J) age 7 years, also suffered from Asthma. He had his tonsils and adenoids removed but with no improvement on the eruption. A sister of this patient however, who also had suffered from dermatitis and asthma, had recovered completely after her tonsils and adenoids were removed. Of the positive cases H.H. age 5 years, gave a very marked reaction to chicken but removal of chicken from the diet and also feeding him on it had no effect on the eruption. The case N.S. aged 7 years is the one from which the Cast (No. 28.) was taken. In this case/

case there was a marked reaction to egg white and egg yolk; also to beef and chicken but these reactions are not shown in the cast as they were done on the other arm. This case was a most successful one. Removal of egg, beef and chicken from the diet caused the eruption to disappear very rapidly although the child had suffered since infancy.

3. Of 9 adults between the ages of 22 and 60 years, only two gave positive reactions. One reacted to egg yolk, beef, wheat and tomato, the other to egg yolk and sheep's wool. Both these cases also suffered from asthma. In the first case dieting on the lines suggested by the eruption had no effect upon it. Similarly cutting eggs out of the diet had no effect on the second case and rubbing wool on the skin did not produce a dermatitis nor did inhalation in close proximity to wool produce an attack of asthma.

These results are rather disappointing when compared with those recorded by others. RAMIREZ[1456.] in 78 cases of eczema in children under 2 years of age had 30 positive results.

O'KEAFE[1453.] in 70 cases of eczema found 41% positive and in breast-fed infants out of 41 cases found 61% were positive to one or more proteins. BLACKFAN[1440.] in 27 cases in infants and adults found 22 reacted positively. C.J. WHITE[1476.] found 30% were positive but as already mentioned I do not consider his results reliable. SCHLOSS[1462, 1463.] in 77 cases of eczema in children found/

found that 50 were positive. TALBOT[471] in 16 cases of eczema in infants and children found that 87% gave positive reactions to egg-white. FOX and FISHER[447] tested 80 cases by the cutaneous method in adults and obtained 19 positive reactions. ENGMANN and WANDER[444] obtained positive reactions in 78% of cases of infantile eczema and in 38% of chronic extensive eczemas in adults. STRICKLER[467] did intradermic food tests in 46 cases of eczema and obtained positive results in 80%.

The published results of others therefore show on the whole a larger proportion of positive results than I obtained but all agree that the younger the patient the more likely is he to react to a food protein and multiple reactions are the rule rather than the exception.

PRURIGO (OF HEBRA)

Four typical cases of Prurigo were tested and only one gave any reaction and that to egg-white, egg-yolk and cod. Exclusion or inclusion of these foods in the diet did not affect the eruption in any way. I can find no other record of food tests in cases of Prurigo.

URTICARIA/

URTICARIA.

Out of 8 cases not a single positive reaction was obtained. Five gave doubtful, probably negative reactions. One of these cases has already been mentioned under urticaria and eventually recovered completely after two teeth were extracted.

ENGMANN and WANDER[1444.] obtained positive reactions in 79% of his 19 cases of urticaria. SCHLOSS[1462.] in 60 cases of urticaria only obtained positive reactions in 10 and in 14 cases of angio-neurotic oedema only two gave positive reactions. These two cases both suffered from the eruption when fed on the protein, to which they reacted. STRICKLER[1469.] using the intradermic method, obtained 8 positive reactions in 10 cases.

The results of others are more encouraging than in my cases. One of the chief difficulties in urticaria is that many cases react so markedly to the mechanical irritation produced in doing the test that it is impossible to gain any information from the tests.

DERMATITIS HERPETIFORMIS.

I have only tested one case and this case gave doubtful reactions to veal and barley. ENGMANN and WANDER state that in D. herpetiformis the number of cases reacting positively is negligible. 11 cases were examined/

examined and only one gave a slight reaction and that to horse-dander.

In several other skin diseases the protein skin tests have been applied in a few cases. ENGMANN and WANDER found that in erythema multiforme and pemphigus the number of cases reacting was very small. SCHLOSS tested 7 cases of E. Multiforme and in one case which reacted to pork the elimination of that article from the diet cured the case. This patient was later immunised by injections of pig's blood serum.

COKE[1442] also reported two cases of Pruritus ani one of which reacted to potato and the other to pork. On removing these articles from the diet or feeding the patient on them the condition disappeared and reappeared accordingly.

ASTHMA and HAY-FEVER.

Cutaneous protein tests have been used extensively in Asthma and hay fever by WALKER[1474], RAMIREZ[1457] and others. The tests have been found on the whole to be even more reliable than in skin diseases. The results with food proteins, pollens and animal hairs give very useful information as to the cause of these conditions. In chronic cases of asthma however the condition /

condition is complicated by secondary infection of the respiratory tract with organisms so that removal of the food from the diet does not always cure the asthma.

OTHER METHODS OF PERFORMING SKIN TESTS.

Instead of using a burr to make the scarification for the cutaneous tests some make a series of small scratches with a needle whilst others make short superficial cuts with a sharp knife. The latter method has the advantage of eliminating the effect of trauma from the reaction.

The intradermic method by which the dissolved protein is injected with a sharp needle into the skin has been used especially in asthma.

Most of the work in protein tests in skin diseases has been done by the cutaneous method. CHANDLER, WALKER and ADKINSON[474.] report that the intradermic test is much less specific than the cutaneous one. It is too sensitive, is more difficult to do and may cause the patient considerable discomfort. It is also not practicable where many proteins have to be tested. It gives reactions which do not separate closely related proteins and therefore the ordinary cutaneous tests are to be preferred.

I used powdered extracts of the proteins dissolved in alkali applied to the skin but numerous firms/

firms have now put on the market proteins in solution in capillary tubes. Besides being more expensive where many tests have to be applied the method is much more cumbersome than the use of the powders. To get over this difficulty tests have been made with groups of proteins containing several allied substances, in one solution. If a reaction occurs to one group, then one must test again with each member of the group.

THE VALUE OF CUTANEOUS PROTEIN TESTS IN SKIN DISEASES.

In the 34 cases which I tested only 6 reacted definitely positively viz; 5 cases of dermatitis and one of Prurigo. That of course does not necessarily mean that all the negative cases were not sensitised to something. One cannot test every case to every possible protein and there is always the possibility that the patient might be temporarily desensitised. The reaction seems to vary very much from day to day and there are numerous cases recorded where it was present one week and absent the next.

MULTIPLE REACTIONS raise many interesting points. As in the plant dermatitis there seem to be group reactions. A guinea-pig sensitised to sheep serum will also react but less violently to goat serum. WELLS and OSBORNE have shown that preparations of/

of legumen from the pea and vetch are very similar, if not identical and also the gliadin contained in wheat and rye. They have also obtained cross reactions between gliadin from wheat and rye and hordein from barley. HOLOBUT[108.] has found the same group reactions in bacterial proteins. He found that reactions could be produced in typhoid cases with B. Coli and Cholera vibrio as well as with B. Typhosus. FREEMAN[1448.] has shown that an asthmatic sensitive to horse serum will give a marked skin reaction to that serum and also a distinct but lesser reaction to the sera of all the equidae.

WELLS is of opinion that multiple reactions are the result of common groups in the protein molecule even though the proteins may appear to be chemically distinct. These facts may account for some of the multiple skin reactions where a patient reacts to wheat, oatmeal and barley or other cereal.

On the other hand these multiple reactions may be due simply to a multiple sensitisation because, if a person can be sensitised to one protein he may be sensitised equally easily to several. The fact that a case of dermatitis gives a positive skin reaction to a certain food does not necessarily imply that that food is causing the dermatitis. The reaction persists long after the food has ceased to cause symptoms. In cases of infantile eczema, where multiple/

multiple skin reactions occur, the disease may not always be due to the same protein all through its course. These cases notoriously have remissions and exacerbations and these could be explained by the child becoming sensitised say to egg and having a dermatitis from egg for several weeks. If continuously fed on egg, the child would become desensitised and the eruption improve or disappear the skin test, however, still remaining positive. Meantime he might become sensitised to another food such as wheat and so the process goes on till at about 3 years of age, when these cases usually recover, the child has become desensitised to all the common foods.

ARE THE PROTEIN SKIN REACTIONS SPECIFIC?

BLACKFAN[441.] in 43 persons (infants and adults) who had never had eczema found that only in one did he get a positive skin reaction. This case might however have previously had some other condition, such as urticaria, which might have accounted for the reaction.

BAKER[438.] also did a series of food tests in a large number of normal children and found that "in the normal child the incidence of protein sensitisation is negligible. Therefore we may assume that a positive reaction to any protein means that the patient is or has been sensitive to it.

VALUE/

VALUE OF THE REACTIONS.

To be of any value from the therapeutic point of view positive reactions should enable us so to regulate the diet as to cure the condition. In my five positive cases in only one did cutting out the foods indicated by the skin tests cure the condition and that in ten days or so. In the other four cases dieting had no effect, not only so but feeding the patients on foods to which they reacted did not make the skin eruption any worse.

RAMIREZ [1456] found in 30 cases of eczema as a result of dieting based on the skin reactions that ten were cured, twelve improved and eight unaffected. ENGMANN and WANDER [1444] found the tests of great therapeutic and diagnostic value in urticaria and eczema but the method requires time and patience. In every instance where the diet could be absolutely controlled as in infants, the results were excellent, but in adults it is very difficult to control the diet properly, because the patient, sensitive to a food such as eggs, may stop eating eggs, yet continue to take them in small quantities in other foods such as cakes, puddings etc. STRICKLER [1467] found that in 46 cases of eczema showing positive reactions in 70%, 50% of these were in greater or less degree benefited by change of diet as indicated by the reactions. In urticaria only the/

the acute cases benefited by suitable dieting.

Positive reactions have been obtained by various observers to practically all kinds of foods both animal and vegetable. Egg however seems to be the commonest food. O'KEEFE[1454] obtained a reaction to egg in 41% of his cases. TALBOT, SCHLOSS and others agree with this, but RAMIREZ and FOX and FISCHER found that the proteins of cereals and vegetables were very frequently the cause of positive reactions.

THE HISTOPATHOLOGY OF POSITIVE CUTANEOUS REACTIONS.

STRICKLER[1512], and STRICKLER and ASNIS[1513] examined the skin microscopically in Tuberculin Pirquet tests, food and Luetin tests. They found that in food and Tuberculin reactions there was a mononucleated cellular reaction in the more superficial part of the corium with a polymorphonuclear cell infiltration in the deeper parts. In Luetin reactions there was found a polymorphonuclear cell infiltration with congestion of the blood vessels and necrosis. They are unable to suggest any cause for the absence of mononucleated cells in the Luetin reaction.

Some years ago KLINGMULLER in Breslau examined the site of injection of Old Tuberculin given subcutaneously and found it contained typical giant-cell tubercle nodules.

THEORIES/

THEORIES OF CUTANEOUS REACTIONS.

The cutaneous reactions are all based on what is known as the "Arthus phenomenon". ARTHUS showed that if a rabbit receives 5 cc. horse serum subcutaneously every 6 days, after the first three injections there is no reaction at the seat of injection, but after the fourth injection a local reaction occurs with redness and swelling. After subsequent injections a progressively more severe reaction which may even go on to gangrene, occurs each time. The animal is sensitised by the injections and the more highly sensitised it becomes, the greater the local reaction. This serum skin reaction is used now to test patients before injection of antitoxic sera to see if they are sensitive.

BIBERSTEIN and OSCHINSKY [1483.] describe cases of a local reaction on the first injection of rabbit, guinea-pig and sheep serum into the skin of human beings. This reaction develops after an incubation period of 7-8 days. They regard these reactions as "local examples" of serum disease.

KRAUSE [1494.] did a series of experiments to determine the time which elapses between infection and the appearance of cutaneous hypersensitiveness. He injected guinea-pigs with two strains of Tubercle bacilli, one virulent causing general tuberculosis, the/

the other benign causing only local gland changes from which the animals recovered rapidly. Animals injected with virulent cultures showed increasing cutaneous hypersensitiveness from the 11th day on till the 46th day when the disease was well advanced. In every case the reaction was more intense than in the animals inoculated with the non-virulent cultures. In the animals injected with the non-virulent cultures the reaction increased as the disease developed but as the disease came to a standstill and began to heal the skin reaction became milder but never entirely disappeared. The local cutaneous reactions in Tubercle, syphilis etc. have already been described and need not be further mentioned.

These various reactions may be explained according to any of the theories which are held with regard to anaphylactic shock. If one believes in the production of a poison (anaphylatoxin) in anaphylaxis then one explains the skin reaction as the reaction between the antigen and the antibody to produce this poison in the skin., leading to a dilatation of the vessels, cell infiltration and oedema. On the other hand many explain the phenomenon on the physical theory. This theory explains better than any other the non-specific reactions.

SELLEI/

1508.
SELLEI did a number of experiments by doing cutaneous tests with skin emulsions. He obtained a more marked reaction on injection of a skin emulsion into the person from whom the skin was taken than could be obtained from a skin emulsion from another normal individual. This he believes to be due to selective hypersusceptibility of the normal skin to its own proteins and suggests the name "Homaesthesia" for it.

1509.
STOKES like SELLEI found that normal skin reacts to intradermal injection of emulsions of normal skin with a reaction similar to the luetin reaction. No constant specific character could be found in normal persons for the response towards their own skin as compared with that to other skin emulsions. The skin emulsion possessed no antigenic properties in a haemolytic complement-fixation cycle. Attempts to sensitise guinea-pigs passively to emulsions of skin by means of serum from the donor of the emulsion or from another person were unsuccessful. The injections of skin emulsions into animals showed no evidence of active anaphylaxis to the proteins of the emulsions.

1510.
STOKES also made intradermal tests with a 0·5% suspension of agar in physiological salt solution. This suspension of agar produced in two normal individuals, who had not been taking Potass. Iodid. reactions/

reactions like the luetin reaction. Intradermal injection of 20% suspension of Bismuth. subnitrate in salt solution in the same individuals gave a slight reaction.

STOKES explains the reactions such as those to normal skin, agar, luetin reactions in persons taking iodides etc., as, in part at least, due to the introduction of antiferment adsorbents, the activity of which uncovers ferments normally present in the individual. He thinks that these proteases split up the proteins of the body with the formation of anaphylatoxins which in turn cause focal necrosis and inflammation. This explains the non-specificity of the cutaneous reactions. The agar skin reactions correspond to the anaphylaxis produced by agar, Kaolin and similar substances already described. STOKES points out "that the only value of such non-specific reactions from the clinical stand-point, is to measure the enzime balance or liability and the amount and intensity of action of non-specific proteases in the body".

Similarly HIFT[1489.] obtained intradermal reactions to colloidal silver preparations. These reactions never occurred on the first injection. These can be interpreted as the result of a disturbance of colloid equilibrium the preparation acting as an adsorbent.

The question as to whether cutaneous reactions are an index of resistance or immunity has been frequently/

frequently raised. KOLMER[1491.] believes that there is no experimental support for the theory that allergic skin reactions may be taken as an index of resistance or immunity. Positive skin reactions do not run parallel with the presence of circulating antibodies; FLEISCHNER, MEYER and SHAW[1486.] showed that guinea-pigs showing a high degree of acquired immunity to an organism as evidenced by strongly positive agglutination and complement-fixation reactions and complete resistance to a subsequent infection with living organisms, will never give specific positive cutaneous reactions. A state of anaphylactic hypersensitiveness can exist without the least cutaneous hypersensitiveness. As already mentioned under the Tuberculin reactions cutaneous sensitiveness only occurs in the presence of infection. The mere injection of bacterial proteins will not produce skin sensitiveness. BALDWIN[1482.] confirmed this by placing in the abdominal cavity of guinea-pigs capsules of porous Berkefeld filter clay, some filled with living tubercle bacilli and others with filtered watery extracts of the Tubercle bacillus. Cutaneous tests were negative on the 15th and 57th days afterwards. He therefore concluded that cutaneous hypersensitiveness does not develop through the mere presence of Tubercle bacilli in the body even although more or less accessible to the body fluids. Similarly injections/

injections of the fatty or waxy substances in the Tubercle bacilli and extracts of the bacilli themselves did not cause cutaneous hypersensitiveness.

Persons who have had typhoid fever or been inoculated against typhoid will react positively to paratyphoidin. Yet it is known that these individuals are not immune to paratyphoid.

The cutaneous food reactions persist long after the patient ceases to have symptoms. The Tuberculin Pirquet reaction persists for life even although the disease is apparently healed. The skin reaction in pneumonia often only appears after the crisis when presumably immunity has been established. Therefore all the evidence goes to show that a positive cutaneous reaction is an indication of sensitiveness but not of immunity.

Another interesting point is the difference in the kind of reaction occurring in different conditions. Why is the reaction due to food proteins, and drugs all over within almost three quarters of an hour, whereas the Tuberculin and similar reactions do not begin for 24 hours and persist for days. It cannot be a question of the degree of sensitiveness otherwise they would all begin after the same interval, but some would last longer than others. It would point to there being some fundamental difference between the sensitisation which occurs in bacterial infections /

infections and that which occurs in food and drug sensitisations. The whole question of the cutaneous reactions requires further work before many points of importance can be clearly understood.

DESENSITISATION.

Desensitisation or antianaphylaxis has already been discussed, (page 10.) in its relation to anaphylaxis in general. Its importance in the treatment of disease requires that it be further dealt with from that point of view. Desensitisation may be specific or non-specific.

SPECIFIC DESENSITISATION has already been fully described under the different diseases. It has been referred to in the prevention of Serum Sickness, in Urticaria, drug rashes, plant dermatitis, food eczemas, etc. In each of these conditions the desensitisation is brought about by the injection or ingestion of the same substance which caused the sensitisation. Autogenous vaccine therapy in diseases where an organismal sensitisation occurs such as in tubercle, certain forms of ringworm, gonorrhoea etc. has also been mentioned under the different diseases.

NON-SPECIFIC DESENSITISATION, however, still requires to be dealt with. In it, some substance either resembling or differing widely from that, which caused the sensitisation is used. These substances may be roughly divided into three groups.

(1) Non-specific Protein desensitisers such as bacteria, peptone, milk etc.

(2) /

(2) Auto-and hetero-serum treatment, and

(3) Non-specific vaccines.

NON-SPECIFIC PROTEIN THERAPY.

This method of treatment is also known as intravenous protein therapy, protein shock therapy and pyrogenic therapy, (AULD). Intravenous injection of foreign proteins gives rise to a fairly definite train of symptoms quite irrespective of the protein injected. Many proteins have been used, chiefly bacterial emulsions, especially of the coli-typhoid group. Peptone, milk, auto and hetero-sera have also been largely used.

A great deal of work has been done, especially in America, by injecting non-specific bacterial suspensions intravenously. It has been shown that the same results can be obtained by bacterial vaccines, e.g. B. Typhosus or B. Coli in typhoid fever as by non-bacterial substances, e.g. normal serum, Sod. Nucleinate and colloidal metals. The injection of these substances causes a febrile reaction (103-104°) with a rigor which is thought to be essential if benefit is to result. If large doses are given nausea and vomiting may result. Before the rigor there is usually a leucopenia followed by a marked polymorphonuclear leucocytosis (about 40,000) reaching its maximum 2-12 hours after the rise of temperature.

There/

There seems to be no special relation between the degree of leucocytosis and the beneficial results following the injections. Most of the cases so treated were cases of typhoid fever and arthritis but pneumonia, gonorrhoeal complications, diphtheria and sepsis were also treated. In typhoid fever cases the temperature falls by crisis after 2 or 3 injections. Similarly the temperature in pneumonia may come down by crisis or lysis after a single injection of typhoid vaccine.

The reaction produced in these cases is known as the "protein shock reaction" and is independent of the vaccine used, i.e. it is not specific.

KRAUS and MAZZA[1531.] found that they obtained the same results with intravenous injection of typhoid vaccine in both typhoid and paratyphoid and also in typhoid with Coli vaccines. Numerous other observers lay stress on the non-specificity of the reaction. It is supposed to be due to the protein in the bacteria as distinct from the toxine. It acts by strengthening the defensive mechanism in the body. As WILLIAMS[1553.] especially emphasises, in every specific infection there is probably a non-specific element due to the protein of the organism. The toxines are specific for each disease but the protein sensitisation is the same in all, hence the symptoms common to all specific infections. MILLER[1534.] has shown that diphtheria can be successfully/

successfully treated with normal horse serum if given early enough so as to counteract the organism before it has had time to produce its toxine and anti-diphtheritic serum probably acts both by the anti-toxine neutralising the toxine and the horse serum combating the diphtheria bacillus.

DAVIS and PETERSEN[1522], by experiments on dogs with thoracic duct fistulae, showed that recovery following protein shock therapy is due to changes in lymph rather than the blood serum. There is an increase in the flow of lymph. Fluids rich in antibody are forced into the lymph chammels. DAVIS and PETERSEN state that bacterial infections not confined to lymph spaces will not be influenced to the same effect by shock therapy. De CASTELLO supports this view as he obtained a rapid fall of temperature by lysis or crisis in typhoid patients after the shock reaction but injection of a similar dose of vaccine in typhus fever was without effect. DAVIS and PETERSEN also attribute the benefit of protein shock to the great increase in the antiferment which enters the circulation through the lymph stream. Bacteria proliferate best where the antiferment is absent and after protein shock any increase in the antiferment would inhibit the growth of the invading organism.

BULL/

1516.

BULL in a series of experiments on rabbits found that the intravenous injection of typhoid vaccine does not cause specific stimulus. Unlike ordinary vaccine treatment, there is an immediate increase in antibodies. There is no preliminary negative phase. There is a rapid mobilisation of normal antibodies, thus increasing their concentration in the blood, to be followed later, as in other forms of inoculation, by the production of so-called acquired antibodies.

VACCINE/

VACCINE PROTEIN SHOCK IN SKIN DISEASES.

It is well known that certain skin diseases such as psoriasis, mycosis fungoides and leprosy, benefit by intercurrent infections in which a high temperature occurs. ENGMANN and McGARRY [1524] employed intravenous injections of typhoid vaccine in doses of 75 to 500 millions. All cases showed a definite rise of temperature (100°-105°F.) and all showed a slight rise in the number of leucocytes after each injection. In all cases the skin lesion, especially in Lupus erythematosus became much redder and sometimes itched for 1-3 hours after the injection and Herpes labialis was a common occurrence. Five cases of chronic Psoriasis were treated. Injections, beginning with 100 millions, were given every three days. This caused the skin lesions to clear up, scaling diminished, and brown areas were left where the spots had been. But even in the most favourable cases, relapse occurred in a short time.

Six cases of chronic Lupus erythematosus were treated similarly. All were cases which had resisted numerous forms of treatment including X-rays and CO_2 Snow. All improved markedly. A case of Parapsoriasis was also markedly improved by the injections and one of Dermatitis Herpetiformis to a slight degree. In a case of Darier's disease one injection stimulated the formation of new lesions. Cases of Syphilis in

Various/

were treated
various stages and in some, especially nodular lesions, the eruption disappeared and left pigmented areas. A case of Acute exfoliative dermatitis showed great improvement.

1541.
SCULLY treated 8 cases of chronic psoriasis with intravenous injections of typhoid vaccine combined with suitable local treatment. After the first or second injections the lesions became less inflammatory but not smaller in size. When this was followed by the local application of chrysarobin ointment the lesions entirely disappeared in 8-16 days, i.e. much more rapidly than occurs in treatment with chrysarobin alone.

1544.
SUTTON treated cases of Psoriasis similarly. He used a mixed B.Coli vaccine intravenously every 2-5 days, simultaneously with the local application of 20% chrysarobin ointment. The disappearance of the eruption was very rapid, usually in seven days on an average. 1517.
CAPELLI and SIGNORELLI used a cholera vaccine in physiological salt solution intravenously injected. Each case received only one injection which was followed by a rise of temperature for a few hours. In a case of Pemphigus vegetans the lesions showed a definite diminution for some days followed by a slight recurrence of eruption. In two cases of pustular eczema there was a diminution of inflammation and suppuration, but an attack of Influenza caused a recrudescence of the inflammation. In a

case/

case of Lupoid Sycosis the result was nil. In four cases of secondary syphilis a partial or complete resolution of the eruption took place. From all these results therefore it is evident that by injection of non-specific bacterial emulsions an improvement in or disappearance of the eruption occurs in almost all the diseases treated. It would seem that a temporary desensitisation takes place, so that local treatment, as in psoriasis, has a much more rapid effect than usual. The improvement, however, seems to be only temporary so that the method is on the whole of rather doubtful value, except as an aid to local treatment.

TREATMENT OF OTHER DISEASES WITH INTRAVENOUS INJECTIONS OF VACCINE.

GOW [1526.] has found good results in the treatment of local streptococcal infections such as cellulitis with intravenous injections of a sensitised streptococcal vaccine. Similar good results were obtained in streptococcal septicoemia. Arthritis was also benefited by intravenous vaccine injections provided the treatment was given early in the case.

PETERSEN [1538.] records marked improvement in a case of acute multiple arthritis from intravenous injections of typhoid vaccine. CECIL [1518.] treated 40 cases of acute rheumatic, toxic and gonorrhoeal arthritis with/

with a similar vaccine. The result was a rapid recovery in most of the rheumatic and toxic cases usually after two doses. The gonorrhoeal cases were not much benefited. MILLER and LUSK[1535.] and COWIE and CALHOUN,[1520.] also report good results in both acute and chronic arthritis.

COWIE and BEAVAN[1519.] used a typhoid vaccine by intravenous injection in cases of Influenzal pneumonia. It caused a termination of the acute symptoms in 1-3 days. The vaccine must be given before the third day of the disease.

MILLER and LUSK[1535.] treated cases of typhoid fever with typhoid vaccine intravenously and obtained the same beneficial results as they obtained with albumose.

ROBERTS, DUDLEY and CAREY[1539.] in cases of Infleenzal pneumonia treated with intravenous injection of vaccine, had a mortality of 11·3% as compared with 31% in cases not so treated. SQUIER also reports astonishing and rapid improvements in 2 cases of influenza pneumonia from injection of typhoid vaccine. WELLS similarly reports good results. ICHIKAWA,[1527.] LUDKE,[1540.] RUMPF and BIEDL,[1515.] all record similar satisfactory results with intravenous vaccine therapy in typhoid fever. It did not matter whether the vaccine consisted of B. typhosus, B. Coli or B. pyocyaneus the results, being the same in all cases.

PEPTONE/

PEPTONE THERAPY.

Peptone has been used extensively in the treatment of disease by intravenous injection in the same way as bacterial suspensions.

SCHMIDT and MULHEIM [1610.] in 1880 were the first to study the effects of injecting digestion products such as peptone. FANO [1578.] in the following year also worked on the subject. These observers noticed that injection of peptone caused an intoxication characterised by an alteration in the coagulability of the blood. BRIEGER [1567.] thought the effect of the injections was due to production of a toxic substance which he called "Peptotoxin". Similarly PICK and SPIRO [1606.] considered the result due to a hypothetical substance which they called "Peptozyme". But later CHITTENDEN, MENDEL and HENDERSON [1569.] and UNDERHILL and HENDRIX [1627.] showed that the previous workers had been using impure peptones and that much of the result was due to the presence of proteoses.

In 1907 DE WAELE [1617.] pointed out the striking analogy between the effects of peptone injections and anaphylaxis. VAUGHAN'S work on the cleavage of protein in Anaphylaxis seemed to support the suggestion that anaphylactic shock and peptone shock were the same thing. The work already described on agar intoxication by NOVY and DE KRUIF [1599.] showed the production of agar anaphylatoxin. By injecting guinea-pigs with peptone they/

they showed that that substance produced the same effect as was produced by anaphylatoxin from agar or in specific anaphylactic shock. They found that the injections caused dyspnoea, spasms, convulsions, loss of control of the sphincters, low blood pressure, death by stoppage of respiration, the heart continuing to beat for some time. They also claim that the post-mortem findings in peptone shock are the same as in typical anaphylactic shock, viz. distension of lungs, absence of clotting of the blood etc. The lethal dose varied, the minimum being 0·5 grm. per kilo body weight. A certain immunity or tolerance is established after a non-fatal injection of peptone. Opinion is divided as to whether the shock produced by peptone injections is identical with anaphylactic shock or not. Certainly there are many points of similarity between the two reactions. RUSZNYAK 1609. was the first to notice in protein shock a change in the antiferment in the serum. He concludes that, because in anaphylaxis the antitryptic power of the serum is enormously increased, "anaphylaxis is a condition produced by albuminoid substances which result from an abnormally rapid parenteral fermentation (peptone poisoning). NOVY agrees with the view as he found that when normal serum is digested with Witte's peptone, a toxic substance results which he believes to be identical with that which occurs in the body cells and fluids during anaphylactic shock.

Much/

Much work has been done on the blood changes in peptone shock. Fall of blood pressure, leucopenia hyperviscosity, decrease of coagulability all occur as in anaphylaxis. BRODIN and RICHET[1568.] did experiments to show whether peptone could prevent or attentuate anaphylactic shock. Dogs were sensitised to horse serum and one hour before the toxogenic dose was given peptone was injected intravenously. This had the effect of causing only very slight symptoms, whereas in control animals, where no peptone was given the animals showed very marked anaphylactic shock. LARSEN, HAIGH, ALEXANDER and PADDOCK[1589.], however, were not able to confirm this. BESSAU, OPITZ and PREUSSE[1564.], also from their experiments on guinea-pigs proved that, in doubly sensitised animals, antianaphylaxis, if produced, is a non-specific process as regards the antigens previously used. Similarly the disappearance of precipitin from a serum was found to be non-specific. These results support the argument that peptone may act similarly to any antigen.

WEIL[1620.] has shown that if the normal liver of the dog be perfused with peptone solution it quickly presents the swelling and capillary congestion seen in anaphylactic shock in these animals. He does not think that anaphylactic shock is due to the production of peptone-like bodies in the circulation, but to a reaction on the sensitised liver cells and so in peptone shock the effects are due to the change in the liver/

liver cells produced by the peptone. HISANOBU[1582.] confirmed WHIPPLE and VAN SLYKE'S[1623.] results which showed that the injection of proteoses into the blood causes a great and rapid increase in autolysis of the body proteins resulting in a marked increase in urea nitrogen and in non-urea and amino-nitrogen in the blood serum. He found the changes in the nitrogen constituents of the blood in anaphylaxis to be similar to those in peptone intoxication but more intense. Both anaphylaxis and peptone intoxication lead to an abnormally rapid autodigestion of tissue protein. The causative factors as yet undetermined, are probably the same in both cases.

WHAT SUBSTANCE PRODUCES THE PEPTONE SHOCK?

The question arises as to whether peptone shock is not due to the Histamine which is present in commercial peptones and proteoses. Histamine shock has already been referred to p. (24). HANKE and KOESSLER[1580.] found that WITTE'S peptone contained not more than 0·00335 grm. in 100 grm. peptone. AULD[557. et seq.] calculates that this would mean that in an average dose of peptone (1 cc. of a 2% solution) there is 1/1500 mgrm. of Histamine. He found that this dilution gave a positive skin reaction in many persons but intravenous injection of 0·3 cc. had either a very slight or negative/

negative effect. When this amount of Histamine, however, was mixed with the usual doses of histamine-free peptone, the injections give the Histamine effect in many persons (i.e. flushing, headache, salivation, palpitation, cyanosis and dyspnoea). AULD therefore thinks that the peptone reinforces the action of the histamine. The intensity of the effect depends on the rapidity of injection. The reaction to histamine comes on at once and is not like that of peptone delayed for some hours, and is not associated with a rise of temperature. AULD states that Histamine itself is quite useless in the immunising process if not actually harmful. HANKE and KOESSLER found that histamine-free peptone produced the typical peptone shock.

The skin test can be used for the detection of histamine in peptone. All individuals gave a wheal reaction to histamine. If a peptone gives a marked reaction with the skin test it probably contains histamine unless the patient is already sensitive to peptone. Intracutaneous tests to Histamine are dangerous as they may lead to histamine shock. This is apt to occur if an intravenous injection is made soon afterwards. AULD therefore recommends Armour No. II. peptone, which never gives the histamine effect.

Commercial peptones also contain numerous

other/

other bodies which may be the cause of symptoms on injection. As already shown VAUGHAN found that heating of protein with alkali in alcohol produces a very toxic substance of the nature of a proteose. Pure peptone has very little toxic effect, and the effect of Witte's peptone on injection is chiefly due to the proteoses and albumoses in it. If peptone is heated with alkali or acid in alcohol a toxic substance like VAUGHAN'S protein-poison is produced. POPIELSKI [1607] called this substance "Vaso-dilatin". CLARK [1570] found by injecting mice that "1 grm. of Witte's peptone yielded 0·3 grm. vaso-dilatin and that the minimal lethal dose of the peptone was 4 mgrm. per gram of mouse whilst the minimum lethal dose of the vaso-dilatin was 0·1 mgrm. per gram. From that he concludes that the vaso-dilatin cannot be preformed in the peptone although it appears to be formed more readily from peptone than protein".

The effects produced by peptone, histamine and other products of protein breakdown, as we have seen, are very similar but they give different reactions in different species of animals just as different effects are produced in different species of animals in true anaphylactic shock. These effects of protein shock in animals are shown by a rise of temperature with increased nitrogen metabolism, increased secretion of/

of glands (e.g. salivation) contraction of non-striped muscles and increased permeability of capillaries. Experiments on animals are not very satisfactory, firstly from the differences in different species, and secondly because rabbits, guinea-pigs, rats and mice are not very sensitive to protein shock. In human beings, however, treatment by the protein shock method has had considerable success. It has long been known that sudden absorption of products of breaking down tissues in the human body may lead to alarming symptoms. As an example one may cite the rise of temperature and other symptoms of absorption seen by the too intensive X-raying of the tumours in mycosis fungoides. The patient may be poisoned by his own broken down tumours. There is also the evidence regarding traumatic shock which is apparently due to the breaking down of tissue protein. WHIPPLE also showed that in acute intestinal obstruction the toxaemia is due to absorption of tixuc proteoses.

TECHNIQUE.

1579.
As GOW emphasises, intravenous injections of peptone in human beings must be made very slowly. A very fine needle should be used and someone should watch the pulse-rate. GOW recommends that the injection should be stopped temporarily if the pulse exceeds 35 beats per quarter minute. The patient may complain/

complain of giddiness, pain in stomach, tickling in the throat and may cough. These symptoms disappear almost immediately the injection is stopped. There is an immediate fall in blood pressure and the leucocytes disappear from the circulation into the pulmonary capillaries to appear later in increased numbers in the blood. The temperature should be taken 5 or 6 hours after the injection. There is often a rise up to 101°F. or over. AULD[1562.] recommends the name pyrogenic therapy because of the temperature; but he advises that in the treatment of asthma, at first, a reaction should be avoided and just sufficient dose given to get near the reaction point. He recommends that if the temperature rises 1°F. the next dose should not be increased. But if after eight injections, no improvement occurs, a larger dose should be given so as to produce a definite rise of temperature. In cases of asthma which are very sensitive to peptone, AULD recommends the addition of a few drops of Lugol's Iodine to the peptone. He usually begins with 5 m. (0·3 cc.) and gives injections twice a week increasing each time by 0·2 cc. till 1·3 cc. (20 m.) is reached. This latter dose should be given six times and the treatment continued for about six months increasing the intervals between the injections later on.

There/

There is some doubt as to whether a rise of temperature is an essential for successful treatment. Chronic conditions such as arthritis benefit most by a definite temperature reaction. AULD states that the effect of the pyrexia is,

(1) physico-chemical, and

(2) Vital.

The former causes increase of enzyme action and of dissociation of adsorption compounds. This leads to dissociation of oxy-haemoglobin with a supply of more oxygen to the tissues. The viscosity of the blood is reduced and its flow made easier and the dilatation of the blood vessels makes them more permeable to the serum. The Vital changes are expressions of a marked stimulation of the reproductive capacity of certain cells. At first there is a leucopenia rapidly followed by a leucocytosis. Alterations in the leucocytes, blood pressure, phagocytosis of red cells by the large mono-nucleated cells of the blood, increase of blood platelets, increase in circulating antibodies, in non-protein nitrogen content of the blood, in fibrinogen, globulin, thrombokenase and blood sugar, have also been described. Generally speaking the injections cause, as CLARK states, "a washing out of the tissue fluids into the blood and this process causes a number of changes in the composition of the blood. Unfortunately the evidence at present is insufficient to indicate which of the changes observed is really of chief/

1570.

chief clinical importance".

THE KINDS OF PEPTONE USED.

AULD 1559. recommends Armour's No. 2 Peptone (5%) and Witte's peptone (2%). WITTE peptone contains primary and secondary proteoses in the relation of 1 to 2. Whereas in Armour's No. 2 the proportion is only 1 to 7. AULD also uses a mixture of 3 parts of Witte peptone and 5 parts of peptone practically devoid of primary proteose. Armour No. 2 may also be used intramuscularly without causing irritation. Peptone siccum may be used but requires the addition of some primary proteose. The solutions should be freshly prepared as they are then more active.

TREATMENT OF CASES WITH PEPTONE.

In 1921 I treated six cases of skin disease with injections of Armour's No. 2. Peptone (5%). It was prepared according to AULD'S formula as follows:-

Dissolve the peptone as far as possible by agitation in hot (56°C.) normal saline. Take the reaction to litmus (usually acid) and add very carefully normal or seminormal Sod. hydrate or carbonate until the solution is faintly yet distinctly alkaline to litmus. The peptone will now be dissolved except for some insoluble residue. Make up to volume with normal saline and place in a water bath at 56°C. for half/

half an hour. Filter while hot through ordinary filter paper and add to the filtrate 0·5% phenol.

If bottled, sealed rubber caps or well-fitting glass stoppers or rubber stoppers should be used. Wait for 3 days and then smear a loopful of the fluid on an agar slant and incubate for 16 hours at 37°C. If negative the peptone may be used.

The details of the cases treated are as follows:-

CASE I. PRURIGO (Of Hebra).

H. W. Female, age 18, unmarried. Admitted to Ward 2., R.I.E. on June 25, 1921. A very typical case. Disease had lasted as long as patient could remember. Eruption worst on arms and legs, but a certain amount on face and body. Typical papular lesions scaly and crusted. Very itchy, skin of limbs, especially extensor aspects, indurated and thickened. Glands in groins and axillae enlarged and hard. Whole skin had pale pasty appearance. No history of Asthma or bronchitis.

FAMILY HISTORY. Father and Mother alive and well. 2 sisters and 4 brothers. One sister dead - cause unknown. One brother had skin trouble in infancy. No history of Asthma in family so far as known.

SKIN TESTS to FOOD EXTRACTS. June 28, 1921.

(See/

(See also under Cutaneous Tests). Tests were done on anterior aspect of left arm.

RESULTS. NEGATIVE to cow's milk, oatmeal, pea, pork, potato, haddock, wheat, herring, lamb, lentil, cabbage, cocoa, bean, beef, tea.

POSITIVE (lasting over 1¼ hour) to salmon, egg white, egg yolk and chicken.

DOUBTFUL (lasting less than 1 hour and not so marked reaction as above) to veal, carrot, cheese and barley.

Patient was given later salmon (tinned) eggs, and chicken, veal, carrot, cheese and barley to eat but in no case was the eruption affected one way or another.

July 12. Unbroken skin of chest rubbed with white and yolk of raw fresh egg on different places and although left in contact with skin for some hours no reaction produced. Skin of right forearm also rubbed with some of patient's own blood but with no effect.

July 12. 5 minims Peptone (Armour's) solution (5%) intravenous, right wrist.

July 16. 8 " " " "

July 19.11 " " " "

July 23.14 " " " "

July 26.17 " " " "

July 30.20 " " " "

Aug. 2, Aug. 6, Aug. 9, Aug. 13, Aug. 16 and Aug. 20, patient received 20 minims Peptone Solution intravenously.

All/

All injections were given into vein in front of right or left wrist, as it was impossible to find any vein in front of elbow owing to smallness of the vessels and thickening of the skin.

The injections caused no local irritation or reaction even although when the 3rd injection was given some of the peptone solution escaped into the subcutaneous tissues.

On July 19 (i.e. after 2 injections) skin test done with peptone solution on right forearm. Result negative.

The temperature was taken 4 hourly for 24 hours after each injection, but at no time showed any alteration from normal. No symptoms of any kind occurred after any of the injections. The skin eruption did not seem to vary after injection nor was there any definite effect on the itching. No rise of temperature after injections. Locally the skin was treated with a daily warm bath and the application of plain vaseline and later of 1% Ichthyol paste.

Patient had an ordinary diet and internally Parrish's syrup ʒi.t.i.d. Whilst under treatment the eruption gradually became less and when patient left hospital on July 20 there were only a very few isolated papules to be seen here and there on arms and legs. The face and body were free of eruption. The skin, however, still remained thickened and indurated.

Patient/

Patient was given 1% Ichthyol paste to apply at home. During stay in hospital her weight increased from 5 st. 12½ to 6 st. 6½ lbs.

RESULT. Very much the same as would have occurred had no peptone been given.

CASE II. DERMATITIS.

K.M. Male, age 59. Occupation - diver. Patient was first seen in December 1920 with a dermatitis of face, neck, arms and groins of two months duration. He was seen at intervals and on September 20, 1921 he was admitted to Ward 2. R.I.E. under Sir NORMAN WALKER. On admission the face, neck, arms and groins were the seat of a very scratched dermatitis. The skin was indurated, leathery and wrinkled especially on fore-head and round eyes. The itching was intense. The general appearance of the dermatitis suggested at first a dermatitis venenata. He had been working with plaster making a cast of dock gates. But stopping work had no effect on the dermatitis. He had marked arteriosclerosis.

Oct. 8, 1921 Peptone (intravenously) 5 m. into right arm.

Oct. 11. " " 8 m. " "

Eruption gradually spreading but the spread had begun before he was given the injections.

Oct/

Oct. 15	Peptone	Pirquet ±	Peptone	11 m.
Oct. 18	"	" -	"	14 m.
Oct. 21	"	" -	"	17 m.
Oct. 25	Peptone 20 m. Itching worse for 2 nights after this injection.			
Oct. 29	Peptone	20 m.		
Nov. 1	"	20 m.		
Nov. 5	"	20 m.	skin less scratched.	
Nov. 8	"	20 m.	skin improved.	

Injections stopped as patient was not feeling well. No rise of temperature after injections. Whilst injections were being given he was treated successively with starch poultices, calamine lotion, weak tar lotion, ammoniated mercury paste 1% and mercury and carbolic ointment.

RESULT Improved but no more than would be accounted for by rest in bed and local treatment.

CASE III. FURUNCULOSIS & SYCOSIS.

D. K. age 25. Admitted to Ward 2 on July 1, 1921 with a marked sycosis of upper lip and beard region. The area was very red, swollen and pustular. On axillae, pubic regions and thighs a series of papulo-vesicular lesions were present. His complaint began 5 years ago. Has had furuncles off and on for many years. Has had vaccine inoculations six times; was X-rayed/

X-rayed twice and hair brought out with temporary improvement only. Wassermann reaction negative.

July 16, 1921 Peptone (intravenously) 5 m. right arm

July 19, " " 8 m. " "

July 23, " " 11 m. " "

This injection was followed about 8 hours later by a reaction in the skin. The whole skin became very itchy especially on the left axilla and pubic region. The face lesions became very red and remained so for two days or so.

July 27, Peptone 11 m. Peptone Pirquet slight +.

July 30, " 11 m. Skin very much better in all areas. Patient thought his skin was better than it had been for years.

Aug, 2, Peptone 14 m. This injection made the eruption worse.

Aug, 6, Peptone 11 m.

Aug, 9, " 11 m. Skin slightly improved.

Aug, 13, " 14 m. This was followed by an increase in the itching.

Aug. 16, Peptone 8 m.

No rise of temperature occurred after any of the injections. Starch poultices and 1% Ammoniated Mercury paste were applied locally to the lesions.

RESULT. Reaction in the skin after injections. At first eruption worse, later increased itching after each injection. Improved considerably on the whole. Patient left the ward suddenly without leave and has not been seen since.

CASE/

CASE IV. PSORIASIS VULGARIS.

J. R. age 15. Psoriasis vulgaris.

HISTORY. Treated for psoriasis in skin wards from January to May 1918. At that time had an acute nephritis from which she made a good recovery. Psoriasis cured when she left the hospital in May 1918. Fresh outbreak of Psoriasis in February 1919. Readmitted to Ward and discharged almost well on May 6, 1919, after treatment with 1% Sulph. salicyl. ointment and strong chrysarobin, salicylic and carbolic ointment on legs. She remained much the same for four months when psoriasis returned again and is still there.

There is a history of psoriasis in the paternal grandmother.

Oct. 8, 1921. Patient shows an extensive psoriasis of scalp and body and limbs. Most of the lesions in fairly large patches of usual appearance.

Oct. 8, 1921 Peptone (intraven). R. forearm 5 m.

Oct, 11, " " " " 8 m.

Temperature rose the same evening to 100·8F. and fell to normal next day.

Oct. 15, Peptone Pirquet definitely +.

Peptone (intravenous) 5 m. No temperature.

Oct. 18, Peptone Pirquet again +.

Peptone (intravenous) 8 m.

Oct./

Oct. 21. Peptone (intravenous) 11 m. Peptone Pirquet negative. No temperature.

Patient vomited the same evening. Fresh spots of psoriasis appearing.

Oct. 25. Peptone (intravenous) 11 m. Vomited the same evening - Psoriasis spreading.

Oct. 29. Peptone (intravenous) 14 m. No sickness. Old spots of psoriasis have all faded. Fresh eruption no longer spreading.

Nov. 1. Peptone (intravenous) 17 m. no reaction.

Nov. 5. " " 20 m. no ""

Whilst the injections were being given local treatment was carried on with a daily Sulphur bath, oil of sesame and later 1% and 2% Sulphur salicylic ointment.

RESULT. disappointing. Did not improve any more quickly than if no injections had been given and fresh eruption came out during the injections. Rise of temperature after second injection. Vomiting after 4th-5th injections.

CASE V. PSORIASIS VULGARIS.

J. H. female, age 9 years. Psoriasis of eighteen months duration. Numerous small lesions scattered all over body and limbs.

Sept. 6, 1921. Admitted to Ward. Treated with daily Sulphur bath and 1% Sulphur Salicylic ointment.

Oct./

Oct. 8. Very little improvement. Peptone (intravenous) 5 m.

Oct. 11. Peptone (intravenous) 8 m.

Oct. 15. " " 8 m.

Oct. 18. " " 11 m.

Oct. 21. " " 14 m. Peptone Pirquet negative. Small new spots of psoriasis coming out on body and limbs.

Oct. 25. Peptone (intravenous) 14 m.

Oct. 29. " " 17 m. No change in eruption.

Local treatment with Sulphur baths and Sulphur Salicylic ointment continued whilst injections were being given. No rise of temperature or other reaction occurred after any of the injections.

RESULT disappointing. As in case IV. no more rapid result than would have occurred had no peptone been given. Fresh spots appeared during the course of the injections.

CASE VI. PSORIASIS VULGARIS.

J. McE. female, age 17 years. Large patches of Psoriasis on scalp and trunk of 7 years duration.

Oct. 11, 1921. Peptone (intravenous) 5 m.

Oct. 15, " " 8 m. Peptone Pirquet negative.

Oct. 18. Peptone (intravenous) 11 m. Peptone Pirquet negative.

Oct. 21, " " 14m. "

Psoriasis in statu quo.

Oct/

Oct. 25, Peptone (intravenous) 17 m.

Nov. 1, " " 20 m.

No temperature or other reaction occurred after any of the injections.

Whilst having the injections local treatment was carried out with 2% Sulphur Salicylic ointment. As soon as injections were stopped patient was put on Nov. 2, on chrysarobin vaseline 4% for the body and 4% Sulphur Salicylic ointment for the scalp. The chrysarobin ointment caused a very severe and immediate reaction and two days later (Nov. 4) the temperature rose to 101°.6 F. Chrysarobin zinc paste (2%) was substituted for the 4% ointment and the temperature fell next day. Patient left the Ward cured on Dec. 1.

RESULT. Injections had no effect at the time but subsequent application of chrysarobin produced a much more rapid and severe reaction than usual.

DISCUSSION OF CASES.

Six cases of skin diseases were treated with injections of peptone given intravenously beginning with 5 m. of a 5% solution and gradually increasing to 20 m. In only one case was there any rise of temperature after the injection and in the same case vomiting occurred twice. In Case 3 the eruption was made worse at first and the itching was increased, but/

but improvement set in later. Apart from these cases the injections caused no disturbance whatever. Of the six cases, one suffered from Prurigo, one from a fairly extensive dermatitis, one from Furunculosis and sycosis and three from Psoriasis.

The cases of Prurigo and of Dermatitis did not improve any more than all such cases do with hospital treatment. The case of Furunculosis and sycosis improved somewhat and the cases of Psoriasis were not affected, except that in one the subsequent use of chrysarobin caused a more severe reaction than normal. On the whole the results were so disappointing that the method was given up.

I have only been able to find two other references to the use of injections of peptone in skin diseases. VON ALSTYNE[1555.] treated 4 cases of psoriasis with injections of an alkaline mixture of proteoses and peptones. They caused no reaction and the eruption disappeared in some weeks without local treatment. AMBROSOLI[1556.] used Armour's peptone (5-10%) giving it intramuscularly twice a week and MERCK'S deutero-albumose (2-5%) given intravenously. Practically no result was obtained in cases of eczema, Lichen and Dermat. Herpetiformis. Peptone has been given successfully by PAGNIEZ and VALLERY-RADOT[1600] by the mouth in cases of urticaria. He found that in cases of urticaria, due to eggs, shell fish etc. a small amount of the/

the offending protein taken half an hour before a meal would prevent symptoms. A tablet of 0·4 or 0·5 grm. Peptone similarly taken before the meal had the same effect. The continuous use of the peptone for some time cured the condition. They also quote a case of RAMOND'S of general pruritus in a female which was cured by the ingestion of peptone. JOLTRAIN 1585, records 3 cases, two of urticaria and one of asthma due to eating eggs or other food, which were completely cured by taking peptone before meals. I have tried this method in a few cases of chronic dermatitis, but without any beneficial result.

RESULTS IN OTHER DISEASES.

AULD 1557 et seq. has obtained good results in the treatment of Asthma with peptone injections. He found the most suitable cases were those who showed intervals when they were free of bronchitis and who had not much emphysema. Cases of continuous asthma did not do well, possibly because the anti-anaphylactic mechanism had broken down. This might also account for the lack of result in chronic skin diseases. AULD also records a good result in a case of recurrent migraine with injections of peptone and PAGNIEZ and VALLERY RADOT 1601. report a cure in 5 cases of migraine from the administration of 0·5 grm. peptone by the mouth half an hour before meals.

NOLF/

[1596, 1597.]
NOLF obtained satisfactory results in the treatment of typhoid fever, strepto- and staphylococcal septicoemias with injections of a 10% solution of peptone. He also found this method efficaceous in obstinate cases of acute rheumatism combined with salicylate treatment.

[1591.]
MATTHES studied the effect of injection of deutero-albumose and peptone as compared with Tuberculin in Tuberculosis and came to the conclusion that the effect of the Tuberculin was the same as that produced by these substances.

From a study of all these cases the best results from the use of peptone therefore seem to have been obtained in Asthma. Whether the results are permanent is open to doubt. I have seen one case of asthma which benefited greatly at the time but relapsed some months later. In skin diseases the results with peptone on the whole are disappointing.

MILK THERAPY.

As the results were so disappointing with peptone injections, I tried the effect of intramuscular injections of sterilised milk in various skin diseases.

The effect of milk injections is very much the same as that of intravenous vaccine. A form of shock/

shock is produced resulting in a rise of temperature some hours later and a marked leucocytosis. Up to the present 11 cases have been so treated. Details of these cases are as follows.

CASE I. DERMATITIS.

B. B. Male, age 40 years. The patient was first seen on Nov. 7, 1921. He had a history of eczema in infancy. He remained well till eight years ago, when he had a slight recurrence of the eczema lasting about a month. This attack began about six months before he was seen. He had an extensive itching dermatitis of scalp, face, neck, axillae, arms, upper part of body and behind knees. Food tests to all the common foods were negative. As he was no better in March 1922, milk injections were given.

March 22, 1922. Injection 2·5 cc. boiled milk into R. buttock followed by no reaction.

March 25, 1922. Injection 5 cc. boiled milk into L. buttock. This was followed by a temperature of 101·4°F. for some hours.

March 28, Injection 5 cc. boiled milk into R. buttock, followed by rise of temperature to 99·5°F.

April 1, Injection 6 cc. boiled milk into L. buttock.

Almost immediately afterwards he complained of a constricted feeling round the chest and fluttering of the heart. The pulse was regular. He was slightly/

slightly cyanosed and had a short cough such as occurs in pneumonia. I immediately injected 3 minims Adrenalin (1-1000) hypodermically. This produced no result. The cough and constricted feeling in chest lasted about 20 minutes when the symptoms gradually subsided and in an hour he felt quite well again. This attack was evidently not an asthmatic one, but a fat embolus due to some of the milk getting direct into a vein and blocking some of the vessels in the lung temporarily. Whilst having the injections he was kept in bed and treated with 1% Ichthyol paste and 1% tar paste. The eruption improved very much.

RESULT. Satisfactory. He left the home on April 13, and on April 27 reported that the skin was well.

CASE 2. DERMATITIS SEBORRHOEICA.

J.J.C. Male, age 26 years.

This was a private case sent to me by Dr. GIBSON. He showed a typical seborrhoeic dermatitis of scalp, face, neck and flexures of elbows. As dieting and removal of septic teeth had no effect and local treatment only caused a temporary improvement 2 years after he was first seen, I recommended boiled milk injections. Dr. GIBSON gave him 5 injections at 3 day intervals in doses of 2·5, 5·0, 7·5, 7·5, and 7·5cc. None of the injections caused any rise of temperature. The skin eruption became worse after the fourth injection and after the fifth injection he complained/

complained of, a severe shooting pain in the small of the back which lasted for a few minutes.

RESULT disappointing. He improved slightly but not more than could be accounted for by the rest in bed.

CASE 3. DERMATITIS SEBORRHOEICA.

A.F. Male, age 55 years.

This patient was first seen in January 1921 with a seborrhoeic dermatitis of scalp, ears, neck, axillae, fronts of elbows, backs of arms, behind knees, groins and genitals. This patient was tested to all the common foods but gave no reaction to anything. He was an alcoholic and nearly every local application except lanolin caused irritation. He improved and relapsed for about a year, when milk injections were given.

March 4, 1922. Injection 2·5 cc. boiled milk into R. buttock.

March 7, " 5 cc. " " " L.

March 10 " 7·5 cc. " " " R.

Each injection was followed by a temperature of about 99°. The injections were stopped as he complained of pains all over and a feeling of general malaise.

RESULT. Nil.

CASE/

CASE 4. NEURO-DERMATITIS.

J. P. Male, 54 years.

This patient had suffered from Asthma for years. The asthma suddenly stopped about four months before he was seen. As soon as the asthma stopped a general itching of the skin began. Scratching lead to the production of a dermatitis and when seen on March 11, 1922 he had a general itching all over the skin with a scratched, red, scaly, indurated skin. There was also a general sweating of the skin. Milk injections were recommended and he was given six bi-weekly injections of boiled milk by Dr. PRATT of Whitehaven. As there was a history of Asthma, the first dose was 1 cc. which was increased gradually to 10 cc. These were not followed by any rise of temperature but there was a very slight return of the Asthma.

RESULT. The injections had very little effect on the itching and the dermatitis. Dr. PRATT reported "perhaps a slight improvement".

CASE 5. XANTHORRYTHRODERMIA PERSTANS.

G. S. Male, age 53.

This patient showed large areas of bluish-red eruption affecting the skin of the body and limbs. It corresponded very closely to the condition described/

described by CROCKER as Xantho-erythrodermia perstans He had been in numerous hospitals during the last few years and all kinds of treatment had been used without improvement. He was admitted to the skin wards on January 27, 1922. X-rays and Finsen light were tried but had no effect.

Feb. 11, 1922. Injection boiled milk 2.5 cc. into right buttock.

Feb. 14, 1922. " " " " 5 cc. " left buttock.

Feb. 18, " " " " 5 cc. " right buttock.

Feb. 21, " " " " 7.5 cc " left buttock.

Injection given at 12 noon.

Leucocyte count at 2 p.m. 9,600

" " " 5 p.m. 11,200

Feb. 22, " " "10 a.m. 5,600

Feb. 25, Injection boiled milk 10 cc. into right buttock.

Feb. 28, Leucocyte count at 10 a.m. 7,800.
12 noon. Injection boiled milk 10 cc. into left buttock.

1 p.m. Leucocyte count 8,400

3 p.m. " " 11,200

5 p.m. " " 10,400.

March 4, Injection boiled milk 10 cc. into right buttock.

March 7, Injection boiled milk 10 cc. into left buttock.

Each /

Each injection was followed by a slight rise of temperature for 8-12 hours, 100°. 6 F. being the highest. When the blood was examined before and after each injection, a marked leucocytosis was present after the injection.

RESULT. Absolutely nil as regards the effect on the skin eruption.

CASE 6. PSORIASIS & DERMATITIS EXFOLIATIVA.

Mrs. S. age 50 years. Patient was admitted to Ward on Oct. 19, 1921, with Psoriasis on scalp, flexor aspects of both forearms, sacral region and legs. Before admission the eruption was spreading. At first patient was treated with 2% Sulphur salicylic vaseline on the scalp and plain vaseline on body and limbs. Internally thyroid gr.iinight and morning was administered till Nov. 22, then vin. antimoniale m. iii t.i.d. was given till Dec. 12, when Quin. Sulph. gr.ii was substituted. During that time the eruption changed in character, becoming much redder and spreading all over the body and limbs till by the beginning of December the patient showed the eruption of a typical exfoliative dermatitis (Pityriasis Rubra). The eruption covered the whole of the skin except the central part of the face, the fingers, and toes and the palms and soles. During the spread of the eruption/

eruption the temperature which was 98°F. every morning, went up every night usually to about 100°, but on two occasions it was 101°F. When the eruption stopped spreading the temperature slowly came down almost to normal. After the eruption had been stationary for about six weeks on Jan. 5, 1922 treatment with milk injections was commenced.

Jan. 5, 1922. Injection 2·5 cc. boiled milk intramuscularly into right buttock. The injection caused no local pain or discomfort. No effect on temperature.

Jan. 10. Eruption distinctly better. Distinct areas of paler skin beginning to appear. Injection 5 cc. Boiled milk into left buttock. No rise of temperature.

Jan. 17. Eruption still becoming paler. Injection 5 cc boiled milk into right buttock.

Jan. 21. Injection 10 cc. boiled milk into left buttock.

Jan. 24. Injection 10 cc. boiled milk into right buttock.

Jan. 28. Injection 10 cc. boiled milk into left buttock.

Exfoliative eruption has almost entirely disappeared. Psoriasis spots still visible.

Jan. 31, 1922 Injection 10 cc. boiled milk into right buttock. Skin now shows no sign of exfoliative dermatitis, but small psoriasis spots have appeared here and there on body and limbs.

Feb. 4, 1922 Injection 10 cc. boiled milk into left buttock. Injections were then stopped. Under 1% Ichthyol paste/

paste the psoriasis spots rapidly subsided and on Feb. 18 patient was discharged absolutely cured.

RESULT. Most satisfactory. The exfoliative dermatitis began to improve immediately after the first injection. 8 Injections were given and by the time the sixth injection had been given (i.e. in about 3 weeks) the exfoliative dermatitis had entirely disappeared. The injections did not have the same immediate effect on the psoriasis, but with mild local treatment after the injections were stopped the psoriasis disappeared much more rapidly than usual. None of the injections caused any pain or discomfort and no temperature or other reaction was seen.

CASE 7. DERMATITIS EXFOLIATIVA GENERALISATA.

C. J. age 60 was admitted to the skin ward on March 30, 1921 suffering from a dermatitis of face, neck, arms, groins, and thighs; very itchy. Treated with tar paste, ichthyol paste, liq. alumin, acetat. etc. No treatment had much effect. He left the ward in June 26, still having a very red cracked dermatitis of arms, groin, scrotum and inside of thighs. He was seen by Sir NORMAN WALKER in Chalmers Hospital on Oct. 12, when he showed the clinical picture of a general exfoliative dermatitis. He was transferred later to the Longmore Hospital for Incurables. There he received boiled milk injections in doses/

doses of 2·5, 5·0, 7·5, 5·0, 7·5 and 7·5 cc twice a week. He had a rise of temperature after each injection. The injections had no effect on the eruption but his general condition was never good. He suffered from marked arteriosclerosis and died some months later. Some months before death he became very thin and the skin showed a marked brown pigmentation suggestive of Addison's disease.

CASE 8. DERMATITIS EXFOLIATIVA GENERALISATA.

W. M. Male, age 64 years.

Patient was first seen on Nov. 23, 1922 with a typical red dry scaly exfoliative dermatitis all over head, face, trunk and limbs. The eruption began on the hands and feet and gradually spread till the whole body was affected. Milk injections were suggested and given by Dr. MACLAGEN, AYTON. He was given six injections of boiled milk at intervals of 3 days beginning with 2·5 cc and ending with 10 cc. This caused a diminution in the redness of the skin and a marked diminution in itching. He was seen again in April 1923, when the slight improvement was maintained. but the skin, though paler, was still red and scaly all over. There was no reaction of any kind after the injections.

CASE 9. DERMATITIS EXFOLIATIVA GENERALISATA.

A. S. Male, age 40.

The eruption began as a dermatitis on the legs about a year ago and gradually spread all over till the whole skin was affected showing a general exfoliative dermatitis of the seborrhoeic type. There was a good deal of itching. Milk injections were recommended and given by Dr. HERON, MARKINCH. He had eight injections of boiled milk beginning with 2·5 cc. and gradually increasing up to 10 cc. The result was disappointing as no improvement took place.

CASE 10. DERMATITIS EXFOLIATIVA.

J. R. Male, age 33 years.

Admitted to Ward 1 Royal Infirmary, Sept. 6. 1923. His skin eruption began 9 years ago on the hands evidently as a dermatitis. He suffered off and on from a dermatitis of hands and arms with intervals of freedom from eruption till September 1922. At that time the soles of the feet became affected. The eruption disappeared and reappeared in January 1923 on hands and feet gradually spreading up on to the limbs and body and when admitted in September, the whole skin was affected except the upper parts of face and chest. The eruption was bright red and scaly all over with

an/

an abrupt edge. The patient was treated with a daily sulphur bath. A half per cent sulphur salicylic ointment was applied to the scalp and a paste of zinc oxide and liquid paraffin to the body and limbs. The latter was replaced later by cold cream. Salicin gr. X. tid. was given internally.

Nov. 14, 1923. Blood sugar 114 mgrms. % Blood pressure $\frac{118}{80}$.

Nov. 15, Injection sterilised milk (intramuscular) 2·5 cc. Leucocyte count 2 hours later was 13,430. Temperature on same night 99°. 2F.

Nov. 19, Injection sterilised milk 5 cc. Leucocytes 2 hours later = 8,125. Temperature same night 100°F.

Nov. 22, Injection sterilised milk 7·5 cc. Temperature same night 101°. 6F.

Nov. 26, Injection sterilised milk 5 cc. Temperature same night = 101·4. During the night the patient was very restless with slight delirium. The skin slightly paler especially on the back.

Nov. 29, Injection sterilised milk 2·5 cc. Temperature same night = 99°F.

RESULT. Skin slightly paler all over. Improvement most marked on head and upper part of back.

CASE 11. PSORIASIS.

Mrs. R. age 55 years.

This patient was first seen in 1915 with a psoriasis of scalp and limbs of 4 years duration and of rather an inflamed type. She was treated in a nursing home with Sulphur salicylic ointment in increasing/

increasing strengths and crude coal tar latterly and the eruption disappeared in about 8 weeks. The eruption recurred about 6 weeks later. She was again treated similarly in bed with the addition of X-rays to the more resistant areas. She relapsed again some months later and had a course of sea-water plasma injections subcutaneously. She improved and went to Harrogate where she had a course of sulphur-bath treatment. During the next six years she was never quite free of eruption, so a course of milk injections was recommended in addition to local treatment with 1% Sulphur Salicylic ointment and crude coal tar.

July 29, 1922.	Injection	2.5 cc. boiled milk into right buttock.
Aug. 1,	"	5 cc. left buttock.
Aug. 4,	"	5 cc. right buttock.
Aug. 8,	"	6 cc. left buttock.
Aug 11,	"	7.5cc. right buttock.
Aug. 15,	"	10cc. left buttock.

There was no rise of temperature after the injections except after the third injection (99.5F.) She complained of a feeling of general discomfort and nervousness for about 48 hours after each injection.

RESULT. The psoriasis lesions became paler but did not disappear. On the whole a disappointing result. She/

She relapsed later and is still uncured. The injection apparently had no effect in accelerating the effect of local treatment.

Briefly summarised, 11 cases of skin disease have been treated with milk injections. The cases were dermatitis (1), Seborrhoeic dermatitis (2), Neurodermatitis (1), Xantho-erythrodermia perstans (1), Dermatitis exfoliativa (5) and psoriasis (1).

TECHNIQUE.

In all cases ordinary fresh dairy milk was used. The injections were made as early in the forenoon as possible because it is important that the milk should be used before many bacteria have had time to multiply in it. Some of those who have used this method state that the rise of temperature following the injections does not occur if the milk is absolutely fresh. FORD[1634.] has shown that Bac. Welchii, growing in milk, gives rise to toxic products which, if injected into rabbits and guinea-pigs, can produce sudden death. The result is due to toxines produced by the bacteria because the same results are obtained when the bacteria themselves are removed by filtration. It is not necessary to get the milk in specially sterilised vessels. I found that fresh morning's milk was quite suitable up to 11 o'clock in the forenoon/

forenoon. A small jam jar (which holds ½ lb.) is half filled with milk and a teaspoon is placed in it. This is placed in a pan of hot water and covered with a lid. The water in the pan is then boiled for 15 minutes. The jam jar is taken out and the "skin" removed from the top of the milk by the teaspoon. A large record syringe with a long needle of large bore is filled with the milk which is injected deeply into the gluteal muscle in the usual way. Care should be taken that the milk is not injected direct into a vein, otherwise a fat embolism is produced as occurred in Case 1. If the piston is withdrawn slightly, before injecting, and no blood comes into the syringe, it is quite safe to give the injection. For adults the first dose is 2·5 cc, then 5 cc, 7·5 cc and 10 cc, the last being repeated several times if necessary. For children smaller doses should be given, 1 cc. at first increasing by 1 cc. till 5 cc. are reached. Injections are given twice a week into right and left buttock alternately. The injections should be given slowly and the patient kept in bed. The method is contra-indicated in milk asthmatics.

RESULTS IN MY CASES.

One case of chronic more or less generalised dermatitis of unknown origin did extremely well. He had 4 injections with a rise of temperature after three/

three of them. This case had milk embolism after the last injection, but made a good recovery. In two cases of seborrhoeic dermatitis, in one there was slight improvement, in the other the result was nil. In one case of Neurodermatitis (general pruritus) in an asthmatic, the result was disappointing, both as regards the effect on the itching and the dermatitis. One case with an eruption corresponding to what CROCKER described as Xantho-erythrodermia perstans had 8 injections with no result. Five cases of exfoliative dermatitis were treated. In one the result was astonishingly good, improvement beginning after the first injection. I think the result must be attributed to the injections, as these cases are notoriously slow and unresponsive to treatment. After the exfoliative dermatitis disappeared the psoriasis which had been previously present, remained, but it too responded more readily to local treatment than normal. In the other 4 cases of exfoliative dermatitis, two improved slightly but the result was negative in the other two cases. In the one case of psoriasis treated, the result was disappointing.

In nearly all cases a slight rise of temperature occurred after each injection and if above 100°F. the dose of the next injection was not increased. Apart from the fat embolism already mentioned, no alarming/

alarming symptoms were produced. One case was slightly delirious with a temperature of 101°·4F. and another case complained of malaise and nervousness for two days after each injection. In all cases, where the blood was counted, there was a marked leucocytosis 3-5 hours after the injection. The results therefore on the whole are encouraging and the method seems worthy of more extended trial especially in exfoliative dermatitis and similar conditions which do not yield well to any known treatment.

RESULTS OBTAINED WITH MILK INJECTIONS IN SKIN DISEASES BY OTHER WORKERS.

1556.
AMBROSOLI had the same experience as I had. After trying peptone injections and finding the results disappointing, he gave them up in favour of milk injections. He reports good results in 2 cases of eczema of nipple, one of chronic eczema of scalp and flexures, 3 cases of seborrhoeic dermatitis and 2 cases of eczema in babies. He obtained improvement in a case of lichen planus but no result in 4 cases of psoriasis. A case of chronic pemphigus was benefited and a case of furunculosis was cured after 2 injections. 1641. MORINI treated 12 cases of soft sore and buboes. The milk injections acted very beneficially on the buboes but had no effect on the soft sores./

sores. CATTANEO[1633] also obtained exactly similar results. GUSZMANN[1637], ANTONI[1631] and BERNDT[1632] on the other hand report a good effect on the soft sores themselves. AHLSWEDE[1630] recommends the injection of a germ and toxine-free solution of milk albumin in all local and general staphylococcal skin lesions (furunculosis, carbuncles pyodermia etc.) in superficial and deep trichophytosis, in buboes, and gonorrhoeal complications. MÜLLER[1643] obtained satisfactory results in sycosis, impetigo contagiosa, soft sore and gonorrhoeal complications. He puts the effect of the injection down to the leucocytosis and not to the rise of temperature which does not occur if the milk is not infected.

GAWALOWSKI[1636] used a modified form of treatment. He gave intracutaneous injections of a milk preparation called "Lactin". 16 cases of deep Tinea Barbae were healed with good result, but cases of staphylococcal sycosis were not benefited. KRUGER and PFEILER[1639] report the result of treating animals suffering from skin diseases with subcutaneous injections of "Yatren" a protein similar to that obtained from Casein. He obtained cures in alopecia areata in a dog, 2 cases of Tinea in dogs, dorsal eczema with alopecia and furunculosis in a dog, itchy eczema in a horse and eczema in a pig. VARNEY[1648] obtained marked improvement in cases of infantile eczema as the result of giving a small rectal injection of cow's milk every 2-3 days.

The/

The first effect was a marked aggravation of the eruption within 24 hours, but rapid improvement soon set in, in 50% of the cases.

MILK INJECTIONS IN OTHER DISEASES.

MÜLLER[1644] and THANNER used milk injections with success in Arthritis, conjunctivitis and iritis. FRIEDLANDER[1635] in Trachoma, and UDDGREN[1647] in parenchymatous keratitis. SCHMIDT[1646] also treated cases of arthritis with milk injections and SCHMIDT[1646] and SAXL[1645] obtained good results in cases of typhoid fever. ZIEMBOWSKI[1649] found the method good in cases of sepsis and tuberculosis especially of bone. LEVI[1640] found improvement in ~~the~~ three cases of vomiting of pregnancy after a course of milk injections. The method is still on its trial, but is one which might be tried in many conditions where a sensitisation of unknown cause is probably present. With ordinary care there is no risk from the injections.

AUTO- & HETERO-SERUM THERAPY OF SKIN DISEASES.

That homologus and heterologous normal blood has toxic properties was demonstrated by the accidents which occurred when transfusion of blood was performed in the early days of surgery. The earliest work was done/

done usually with heterologous blood. DOERR[1659] was the first in 1910 to correlate the toxicity of normal blood with anaphylaxis. According to him both the symptoms and post-mortem findings, after normal serum injections, correspond very closely to those of anaphylaxis. DE KRUIF[1677] did a large number of experiments on animals by withdrawing blood and reinjecting it. The blood, which is initially non-toxic, becomes toxic just prior to the appearance of coagulation. The speed of poison-production corresponds to that of the production of anaphylatoxin with substances like agar. The poison-production is caused by changes incidental to the clotting of the blood. KÖHLER[1674] injected a rabbit intravenously with its own previously defibrinated blood and produced acute symptoms and death. He found thrombi in the heart and large vessels and concluded that the injected blood was toxic because of its high fibrin ferment content. STUHMER[1700] did experiments on the toxicity of homologous serum for rabbits and guinea-pigs. He found that this toxic condition only persists for a short time after drawing off the blood. SCHULTZ[1693] found that freshly drawn homologous blood, if applied to normal smooth muscle, produced no response until clotting set in. When that occurred a violent contraction of the muscle resulted. From the above facts, therefore, it is evident that some change takes/

takes place in the toxicity of blood when it clots and the toxine produced is similar to the anaphylatoxin produced by injecting agar and similar substances.

The injection of auto-blood, auto-serum after defibrination and hetero-serum has been extensively used in the treatment of skin and other diseases. There is still much to be learned as to its exact mode of action. It is a question how much of the action of anti-sera used to treat diseases like diphtheria is due to the horse serum and how much to the anti-toxine. BINGEL[1652.] in 1918 treated 466 cases of diphtheria with normal horse serum and he claims that the results were as satisfactory as in 471 controls who received anti-toxic serum. KASTENMEYER[1673.] found that animals, receiving 100 times the fatal dose of diphtheria toxine, were saved by injection of normal serum. MEYER[1687.] also found that by injections of normal horse serum, 33$\frac{1}{3}$% of guinea-pigs were saved after receiving a lethal dose of diphtheria bacilli as compared with 100% saved after antitoxic serum. It is probably that, if injected early before the bacilli have had time to elaborate toxine, the injection of normal serum is successful, whereas if toxine is already formed the antiserum is better.

TECHNIQUE/

TECHNIQUE.

Blood is withdrawn from the anticubital vein into a sterile centrifuge tube. 50 cc. are withdrawn in children and 100 cc. in adults. The blood is shaken for 3 minutes with glass balls. It is then centrifuged thoroughly. It requires a centrifuge which makes at least 4000 revolutions a minute and takes half an hour to separate the serum thoroughly. The serum is then drawn off and injected into the patient again usually into the vein of the opposite arm. The quantity of serum thus obtained is about 40-45% of the original quantity of blood withdrawn. If there is difficulty in getting the serum into the vein it may be injected subcutaneously. Some have withdrawn the blood and reinjected it immediately without defibrinating, but this is difficult to do and there is danger of embolus. The patient's own blood or the blood of another individual either healthy or suffering from the same disease may be used. Usually 30-40 cc. of serum are injected. This treatment is repeated 2-6 times at intervals of 3-5 days.

1655.

BRONFENBRENNER and SCHLESINGER think that the injections should not be given at regular intervals. They think that the blood should be withdrawn, not during the periods of freedom from symptoms when the patient's/

patient's blood is probably free from circulating antigen, but immediately preceding, during or immediately after the anaphylactic reaction. They suggest that the presence of antigen in the blood be determined either by titration of its complement or by the antitryptic index and that the serum containing the antigen be properly preserved and be injected into patients between attacks of the disease.

DANGERS AND DISTURBANCES.

GOTTHEIL and SATENSTEIN [1667] have given over 250 injections of autoserum without any symptoms, except in one case. This was a bad case of gangrenous radiodermatitis in a patient with leucaemia. He had a reaction with rise of temperature after the 5th and 6th injections. This was put down to serum sickness due to sensitisation from the previous injections. STUMPKE [1699] noted headache, rise of temperature, transient diffuse erythema or urticaria with dyspnoea in some cases. The blood showed an eosinophilia. Epileptiform convulsions are reported by FOCKLER [1663] and acute dilatation of the heart by ARNOLD and HOLZEL [1651] in cases treated with intravenous injection of antigenococcal serum.

NICOLAS, GATE and DUSASQUIER [1688] after autoserum/

serum injections, record, apart from the usual leucopenia, fall in blood pressure, and diminution of blood platelets, acute pain in the lumbar region, marked evening rise of temperature, severe arthralgia and even non-suppurative arthritis.

But on the whole there seems to be very little danger from the use of auto- or hetero-serum provided the injections are not given at too long intervals.

EFFECT OF INJECTIONS ON THE SKIN.

SPIETHOFF[1697] found by the use of autoserum and auto-blood, a change in the skin so that it showed a diminished sensitiveness to external irritation. A case of dermatitis, which relapsed every time that Tumenol zinc paste was applied, stood that application well after injections of auto-blood. Similarly a case of Psoriasis after several injections of autoserum stood chrysarobin quite well although previously that drug reacted with intense inflammation of the skin. Reactions which had previously occurred after Salvarsan injections, did not occur if the patient were previously treated with auto-serum or auto-blood injections.

THEORIES/

THEORIES OF THE ACTION OF AUTOSERUM ETC.

Very little is known as to how these injections act. They seem to have a similar effect to the injection of other protein substances such as peptone, milk etc. GOTTHEIL and SATENSTEIN[1668.] think that it may act in some diseases by stimulating some deficiency in the endocrine glands. They tried the effect of withdrawing blood and not reinjecting, but obtained no results, therefore it is not due to the blood letting, as has been suggested by WILE and others. ACHARD and FLANDIN[1650.] think that in conditions in which sensitisation is evident the serum acquires cryptotoxic properties and can be used for purposes of desensitisation. PRAETORIUS[1689.] favours ABDERHALDEN'S suggestion that it is the injection of ferments in the serum which act on the metabolism of the tissues or through the internal secretions. The results are not due to the production of a leucocytosis as SPIETHOFF showed that cases healed where no leucocytosis occurred.

THE/

THE TREATMENT OF SKIN DISEASES WITH HOMOLOGOUS AUTOSERUM, AUTO-BLOOD, & HETEROSERUM & WITH HETEROLOGOUS SERA.

PSORIASIS.

In this disease autoserum treatment does not have much direct effect on the eruption, but makes the lesions much more amenable to local treatment especially with chrysarobin. GOTTHEIL[1666.] and GOTTHEIL and SATENSTEIN[1667.] found that it cut down the time required for local treatment from weeks to days, and postponed relapses for a long time. They gave a series of injections of Autoserum first and then began vigorous local treatment with chrysarobin. SPIETHOFF[1695.], HOWARD FOX[1665.] and FORDYCE[1664.] also obtained similar good results by the combined injection and local treatment. WINFIELD[1707.] reports good results with autoserum injections alone without any local treatment. STUMPKE[1699.], TRIMBLE and ROTHWELL[1703], and SCHAMBERG report no results from the injections.

URTICARIA & ANGIONEUROTIC OEDEMA.

Good results from autoserum injections are reported by HEUCK[1671.], GOTTHEIL[1666.], ACHARD and FLANDIN[1650.] and LINSER[1680.]. SPIETHOFF[1695.], GOTTHEIL and SATENSTEIN[1667.], ULLMANN[1704.] and LUX[1684.], however, only found improvement after the injections./

injections. FORDYCE[1664.] did not have any success.

GENERAL PRURITUS.

HEUCK[1671.], SPIETHOFF[1695.], GOTTHEIL[1666.], NICHOLAS, GATE and DUPASQUIER[1688.] and ULLMANN[1704.] all report good results. Linser[1682.] records 4 cures out of 8 cases treated and LUX[1684.] saw benefit from the injections.

PRURIGO.

SPIETHOFF[1696.] records cure after injections of inactivated autoserum, but 5 weeks later the eruption recurred but disappeared again on further injections. LINSER[1680.] found marked improvement with disappearance of the itching in 12 cases. ULLMANN[1704.] obtained improvement in one case treated. STUMPKE[1699.] also reports good results.

DERMATITIS HERPETIFORMIS.

In this disease autoserum therapy has been beneficial in the majority of cases. FORDYCE[1664.], HOWARD FOX[1665.], SPIETHOFF[1695.], GILCHRIST, SCHAMBERG and HERDINGSFELD[1670.] all report very good results, whilst HEUCK,[1671.] BRECHET[1654.], PUSEY and CORLETT[1656.] record marked improvement. ULLMANN[1704.], LUX[1684.] and FISCHER[1662.], however, obtained disappointing results.

PEMPHIGUS/

PEMPHIGUS.

PRAETORIUS[1689.] reports complete cure of a bad case of Pemphigus of 2 years duration after one intravenous injection of 20 cc. of non-defibrinated normal human blood. LINSER[1680.] treated 6 cases, all long standing ones, two of which had mucous membrane lesions. Two cases were cured by the injections and have remained so for six months. Two were so much improved that they could work. One case died of delirium tremens soon after the treatment commenced and one recovered completely after 4 weeks, but relapsed six weeks later. A second course of injections, however, caused improvement but not a complete cure. One case of Pemphigus foliaceus was improved by the injections.

FISCHER[1662.] treated a case of Pemphigus chronicus in a girl of 20, who had had attacks off and on since childhood. She was given an injection of 20 cc. of blood from a healthy adult female. Next day the temperature was 42°C, new lesions appeared and others became like Pemphigus Foliaceus. Five days after the blood injections she was given 0·45 grm. neosalvarsan intramuscularly. Next day the temperature fell to normal and the skin lesions gradually healed up. She was discharged well 19 days after admission and was still well three months later.

HEUCK/

HEUCK[1671] and STÜMPKE[1699] report improvement in cases of pemphigus after autoserum injections but GOTTHEIL and SATENSTEIN[1668] in 2 cases saw no result.

DERMATITIS & ECZEMA.

GOTTHEIL[1666], SPIETHOFF[1695], McDONELL and LUX[1684] record good results in some cases of eczema. GOTTHEIL and SATENSTEIN[1667] found improvement and HEUCK[1671] obtained no result. LINSER[1682] found that autoserum injections healed most cases of infantile eczema, but in 15 cases of widespread, long standing eczema in adults, only 3 were cured by the treatment. ULLMANN[1704] in two cases of generalised eczema in children obtained no results from the injections, but exceptionally good and quick results as soon as local treatment was instituted. TREUBEL[1702] found that cases of eczema with temperatures did exceptionally well on injections of autoserum or autoblood.

EXFOLIATIVE DERMATITIS.

LINSER[1681] records a cure of one case in 4 weeks after 5 injections of autoserum in 8 days. McDONNELL also reports a remarkable cure following autoserum treatment.

LICHEN/

LICHEN PLANUS.

Good results are reported by STUMPKE[1699] but GOTTHEIL[1666] found autoserum injections ineffective.

Isolated cases of other skin conditions were treated with autoserum by various observers. GOTTHEIL found the method of some value in bad pustular acne, in furunculosis and other pus infections, but useless in leprosy and syphilis. LUX[1684] had no success from its use in a case of mycosis fungoides.

SKIN DISEASES OF PREGNANCY.

Special mention must be made of the work done with serum injections in pregnant women. LINSER[1682] cured a case of Herpes Gestationis (Dermat. Herpetiformis of pregnancy) in a short time by injections of serum from healthy pregnant women. He also treated cases of other dermatoses in pregnancy such as eczema with the serum of healthy non-pregnant and pregnant women. He found that in the diseases, which are due to a toxaemia of pregnancy (e.g. Herpes Gestationis) he did not get as good results from injections of serum of non-pregnant as from the serum of pregnant women. He thinks that the unpleasant results sometimes caused by hetero-serum treatment of non-pregnant cases are due to the use of serum of pregnant

or/

or recently delivered women.

VEIRL[1705], STUMPKE[1699], MEYER[1687] and RUBSAMEN[1691] also report cures of cases of Herpes gestationis with injections of serum of healthy pregnant women or with autoserum. RONGY[1690] has recorded a good result from injections of foreign serum in the pruritus of pregnancy.

ULCUS MOLLE.

TREUBEL[1702] treated two cases of gangrenous soft sore, the one with auto-serum and the other with auto-blood. Both cases healed rapidly without local treatment. MARIANI[1685] found that ordinary soft sores improved with auto-serum treatment, but local treatment was required as well, before the lesions healed completely.

COMPARISON BETWEEN THE DIFFERENT KINDS OF ASPECIFIC PROTEIN THERAPY.

In a recent article MARIANI[1685] gives comparative results from the use of the different forms of aspecific protein therapy. He treated 150 cases comprising 43 of skin diseases of various type, 76 of gonorrhoea with complications and 31 of adenitis associated/

associated with venereal ulcers. He used specific hetero-sera and hetero-vaccines, auto-serum, auto-plasma, auto-blood, aspecific hetero-sera and hetero-vaccines and protein substances such as milk and peptone. Symptoms after a first injection were sometimes seen with hetero-sera, peptone, hetero- and auto-vaccines, but rarely with milk. Anaphylactoid reactions (8-12 days after the beginning of treatment) were fairly frequent after the use of hetero-sera, occasionally after milk, rarely after peptone and hetero-vaccines and never after autogenous vaccines. He sometimes obtained cross reactions e.g. anaphylactoid reactions occurred with milk after hetero-serum injections, with peptone after milk injections and so on. In one case a marked anaphylactoid reaction occurred with an injection of egg albumin given some hours after a peptone injection. On the whole aspecific protein therapy was found to be of very little use in dermatoses of unknown cause and which are probably not infective, although the symptoms might be somewhat ameliorated. In pyogenic dermatoses, hetero-protein therapy had very little effect, but specific autogenous vaccines were undoubtedly advantageous.

NON-SPECIFIC VACCINE THERAPY.

It is not proposed to enter into a discussion on vaccine treatment as a whole. The effect of intravenous injection of bacterial suspensions has already/

already been considered and shown to be non-specific the results being due to Protein Shock. There is still, however, to be studied the effect of giving subcutaneously or by the mouth, vaccines made from the contents of the bowel in cases of alimentary anaphylaxis. This subject has been specially investigated by DANYSZ [1713.] who claims that satisfactory results can be obtained in such diseases as urticaria, eczema, psoriasis, asthma, neurasthenia, gastro-intestinal disorders, arthritis etc. He treated his cases with saline suspensions of four or five varieties of bacteria cultivated from the stools. The vaccine is sterilised by heating for one hour at 70°C. The vaccine usually contained B. Coli, streptococci, enterococci, diplococci and in some cases also an anaerobe. Each cc. of vaccine contained about $\frac{1}{200}$ mgrm. of the dried bodies of the bacteria. He gave 1 cc. for the first dose, then 0·5 cc. 24 hours later and continued to give daily injections for a week increasing the dose by 0·1 cc. each time up to 1 cc. He also treated cases by giving them daily the same vaccine by the mouth in dose of 1 cc. in about 150 cc. of ordinary water.

DANYSZ started on the thoery that the albuminoid substances from the microbes in the intestine pass into the circulation and act as antigens and induce sensitisation. He clains that this occurs in/

in all the chronic non-contagious diseases. At first he used autogenous vaccines from the bowel of each case but later on he found that the same bacteria occurred in the same proportions in nearly all cases he used a polyvalent heterogenous preparation. He obtained successful results in cases of urticaria and chronic dermatitis in which the cure was premanent for some years. He also treated cases of Psoriasis in which he obtained cures in 60%. In some there was a recurrence in 3-6 months but these yielded promptly when the vaccine was resumed. SEMON [1718.] has treated 7 cases of severe psoriasis by DANYSZ method and only obtained a response in one case. BARBER [1718.] found that in some cases it failed completely but admits that it is of distinct value in others.

ERSETTIG [1715.] treated 14 syphilitics with subcutaneous injections of typhoid, paratyphoid and cholera vaccines giving an injection once a week. He found that the injections were capable of turning a strongly positive Wassermann reaction into a negative. They also caused a gradual modification of the syphilitic lesions ending in their complete disappearance even although no antisyphilitic treatment was given. BIACH [1708.] found that injections of old Tuberculin in doses of 0·01 cc. in cases of primary, secondary and late nerve syphilis caused a marked improvement which/

which ran parallel with the fever reaction. The injections had no effect on the Wassermann reaction.

Pus vaccines have also been used by NES - septocoemia, pneumonia,
1716. FIELD and others in the treatment of sycosis, acne and other pyodermias. A few drops of pus are mixed with sterile water and carbolic acid is added to sterilise the suspension. After 24 hours the suspension is injected similarly to a vaccine. I have found benefit in some cases of acne and sycosis from this method but the results are not any better than can be obtained from ordinary vaccines.

In all infections it is a question whether treatment with non-specific vaccines is advisable. A specific vaccine may do good in infections by virtue of its containing the specific antigen and by raising the patient's resistance to that, but in non-specific therapy in general, including all the methods of non-specific desensitisation, it is doubtful whether the method is advantageous in the long run. If one believes in the theory that, in infection, part of the symptoms are due to sensitisation to the protein of the organism and part to the toxines, then removal of the sensitisation to the protein may have a harmful effect on the patients resistance, because these sensitisation reactions are probably protective in nature. In non-infective/

infective conditions, however, such as sensitisations to some non bacterial protein, treatment by non-specific desensitisation is a method to be recommended.

A great deal of work still requires to be done before the method of action of non-specific therapy is fully explained. In all, whether due to intravenous vaccines, peptone, milk, autoserum, or heterologous vaccines, there is a common reaction with rise of temperature, leucocytosis, alteration in the ferments in the blood and in the permeability of the capillaries etc. Which of these changes are essential is not decided, but there is no doubt that the parenteral administration of various albuminoid bodies causes a definite local reaction on pathological tissues which leads to their absorption. For that reason these methods have been found useful therapeutically.

In conclusion, I should like to express my thanks to SIR NORMAN WALKER for the encouragement which he has given me in carrying out my investigations and for the generous way in which he has placed the material in his department at my disposal.

I am also greatly indebted to the staff of the Royal College of Physicians' Library for the amount of trouble they have taken in enabling me to make the bibliography as complete as possible.

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

ANAPHYLAXIS.

1. ADAMSON, H.G. Goulstonian Lectures on modern views upon the significance of skin eruptions. London, 1912.

2. ALPHEN, A. J. S. Anaphylaxie. Centralblatt f.Bakt. etc. 1910 1 abt. Origin. Vol.57, p. 242

3. ALTMANN K. & SCHULTZ, J.H. Verwendung von Bakterien-Antiformin - extrakten als Antigene bei der Komplimentbindung. Zeitsch. f. Immunitätsforch. etc. 1909. Bd. 3 p.98

4. ANDERSON J.F. Maternal Transmission of Immunity to Diphtheria Toxin and hyper-susceptibility to Horse serum in the same animal. Bull. No.30.Hyg.Lab. U.S. Pub.H. & M.H.S.

5. ANDERSON J. F. & FROST W. H. Studies upon Anaphylaxis, with special reference to the antibodies concerned. Journ. Med Research 1910. Vol.xxiii p.31.

6. " " " Studies in Anaphylaxis Bull. No. 64. Hyg.Lab. U.S.Pub.H. & M.H.S.

7. ANDERSON & SCHULTZ The cause of serum anaphylactic shock and some methods of alleviating it. Proc. Soc.Exper.Biol. & Med. vii 1909-10. p.32.

8. ANDRÉ & COURMONT. Folia Haematologica 1904. p.389.

9. APPELMANS, R. The place of the Thyroid gland in Anaphylaxis. C. R. Soc. de Biol. Dec.9 1922. Abstr. B.M.J. Jan.20,1923.

10. ARMAND-DELILLE L'Anaphylaxie et les réactions anaphylactiques. Monographies Cliniques sur les questions nouvelles. No. 56 Jan.21, 1910.

11. ARMIT, H. W. Hypersensibility to pure egg albumen. Zeitschr. f.Immun. forch. & exper. Therapie, 1910. p.703.

12. ARNOLDI, W. & LESCHKE E. Sessile receptors in Anaphylaxis and the role of the autonomic nervous system in the anaphylactic syndrome. Deutsch.Med.Woch.1920. Vol.46, p.1018.

13. ARTHUS Injections répétées de sérum de cheval chez le lapin. Compt. rendu du Soc. Biol. 1903, Vol.55.p.817.

14. " Le sero-anaphylaxie du Lapin. Arhhiv. Inter. de Physiol. Vol.9. pp.156 & 178.

15. " Maurice, Anaphylaxis and Immunity. 1921.

16. AUER J. P. & LEWIS, PAUL A. Acute Anaphylactic death in guinea-pigs. Journ.Amer. Med.Ass. liii.1909, p.458.

17. " " " The physiology of the immediate reaction of Anaphylaxis in the guinea-pig. Journ. Exper.Med. 1910. xii. p.151

18. " " " Demonstration of the cause of acute anaphylactic death in guinea-pigs. Proc.Soc.Exper.Biol.& Med.Vol.vii p.29.

19. AUER & VAN SLYKE a contribution to the relation between proteid cleavage products and anaphylaxis. J.exper.med.1913. Vol.18.p.210.

20. BARDUZZIE. On the clinical value of Anaphylaxis in Dermatology. Giorn.Ital.d.malven e. della pelle Bd.53.1912.

21. BANZHOF E. J. & FAMULENER L. W. The influence of Chloral hydrate on serum-anaphylaxis. Journal Infect.Dis. 1910. Vol.7, p. 577.

22. " & STEINHARDT, Vaughan's split products and unbroken proteins. A comparative study of their effects. Journ. Med. Res. 1910, Vol.23. p.5.

23. BAUER, Ueber die biologische Differenzirung von Körperflüssigkeiten derselben Thierart. Zeitsch. Exp. Path. u. Ther. 1910, Vol. 7 p.417.

24. BECHT F. C. & LEUCKHART A. B. The origin of the antibodies of the Lymph. Amer. Journ. Physiol. 1916. Vol.40, p.366.

25. BEDFORD E. A. The epinephrine content of the blood in conditions of low blood pressure and shock. Amer. Journ. Physiol. 1917 Vol.43 p.235.

26. BERGER H. C. Eosinophilia occurring in Infants following the Ingestion of a foreign protein. Arch.Pediat. 1916, Vol.33, p.743.

27. BATARELLI E. Die Verwendung der biologischen Methode zur Auffindung und Diagnose der Hülsenfruchtmehle mit besonderer Berücksichtigung der Wicke. Centralbl.f. Bakt.2 Abt.) 1904. Vol.II. p.8 & 45.

28. BESREDKA A. A. Comment empêcher l'anaphylaxie. Compt rendu Soc.Biol. 1907. No.62 p.1053.

29. " Anaphylaxis & Antianaphylaxis, translated by Gloyne.

30. " Toxicité des sérums therapeutiques, sa variabilité et son dosage. Ann. d'Institute Pasteur, 1907 xxi.p.777.

31. " The preventive treatment of anaphylaxis 16th Internat. Med. Congress, Budapest Aug.-Sept. 1909. Report J.A.M.A. 1909. Vol.53. p.965.

32. " Le procédé des petites doses et les injections sub-nitrantes. Ann. de l' Inst. Pasteur 24, 1910.p.879.

33. " Antianaphylaxis. Transactions of Inter-Congress of Med. London 1913.

34. " & STEINHARDT. De l'anaphylaxie et de l'antianaphylaxie vis-á-vis du sérums de cheval. Ann. de l'Inst.Pasteur 1907 Vol.21 pp.117 & 384.

35. " & STRÖBEL De l'anaphylatoxine typhique. Compt.rendu Soc.de biol. 1911 Vol.71 p.413.

36. BETTMANN S. Die praktische Bedeutung der eosinophilen Zellen.. Volkmann's Sammlung Klin. Vorträge. 1900 No.266 p.1570.

37. BEZANCON & LABBÉ Haematologie

38. BIEDL & KRAUS Experimentelle studien über Anaphylaxie. Wiener Klin. Woch. 1909. Vol.22 p.363

39. " " Die Serum anaphylaxie bei Meerschweinschen. Wiener Klin.Woch.1910. Vol. 23, p.385.

40. " " Ueber passive Anaphylaxie (Serumanaphylaxie). Zeit.f.Immunitatsf. 1909-10. (Orig. p.115.

41. BORDET. Le mécanisme de l'anaphylaxie. Compt.rendu Soc. de Biol. 1913 Vol. 74 p.225.

42. BORDET & ZUNZ Production d'anaphylatoxine dans le sérum traité par de l(agar épuré de son azote. (pararabine). Zeitsch.f.Immunitätsfor. 1914.15. Vol. 23 pp. 42 & 49.

43. BOUGHTON T.H. Vascular Lesions in chronic protein intoxication. Journ.Immunol. 1917 Vol.2 p.501.

44. " " Kidney Lesions in chronic anaphylaxis. Journ.Immunology. 1916, Vol.1. p.105.

45. BRAUN H. Zur Frage der Serumüberempfindlichkeit. Zeit.f.Immunitätsf. 1910. Vol.IV. p.590.

46. BRODIN, RICHET & ST. GIRONS. Une nouvelle méthode d'antianaphylaxie. Révue de Méd. 1920, Vol.37,p.7.

47. BRONFERBRENNER J. The Nature of Anaphylatoxin. Journ.exper.med. 1914, Vol.21 p.480.

48. " & Schlesinger, M.J. On the action of Anaesthesia in Anaphylaxis. Proc. Soc. Exper.Biol. & Med. 1915, Vol.12, p. 110.

49. BRUCK. Die biologische Differenzierung von Affenarten und menschliches Rassen durch spezifische Blutreaktion. Berlin Klin.Woch. 1907, Vol.44.p.793.

50. BURNS & WATSON A.M. The effect of Thyro-parathyroidectomy on the heart and circulation. Journ. of Physiol. 1918-19 Vol. 52 p. 88. & 1919-20 Vol.53,p.387.

51. CATASTINI G. Sulle precipitine de funghi. Bull della R. Acad. Med. di Roma 1905-06. Fase 1-3. Abstr in Centralbl. f. Bakt. (Ref) 1907. 40 p. 83.

52. CAULFIELD A. H. A note on the desensitization of rabbits and guinea-pigs. Proc. Soc. Exp. Biol. & Med. 1915, Vol.12, pp.170 and 171.

53. CLARET. Treatment of anaphylactic symptoms of alimentary origin. Bull. Soc. de Therap. Oct.12, 1921. Abst. B.M.J. Jan.21,1922

54. COCA, A. F. The desensitization of guinea-pigs sensitized to dogs' serum. Journ.Immun. 1917, Vol. 2, p.439.

55. " " The Site of reaction in Anaphylactic shock. Zeitsch. f.Immunf. 1914, Vol. 20 (Orig.) p.622

56. " " The perfusion experiment in the study of Anaphylaxis. Journ. Immunol. 1919, Vol.IV. p.209.

57. " " Hypersensitiveness; anaphylaxis & Allergy. Journ. of Immunology, 1920. Vol. V. p.363.

58. " " & KOSAKAI M. Studies in Anaphylaxis. Journ. Immun. 1920 Vol.5.p.297.

59. CORPER H. J. Effect of Thorium on Active Anaphylaxis in guinea-pig. Journ. Infect.Dis. Chicago, 1919, XXV. No.3.p.248.

60. DA COSTA. Haematology. London, 1902. p.198.

61. COULLIE, A. G. The relation of various pathological states in man to sensitization to foreign proteins, with special reference to diseases of the skin. Thesis Edin.Univ. 1921.

62. CUNNINGHAM, A. R. Review of the literature of the past five years on Anaphylaxis and related phenomena. Amer.Journ.Dis. Child. 1920.p.393.

63. COWIE D. M. & CALHOUN H. A study of the changes in the blood consequent on the intravenous injection of typhoid protein. Arch. Internat. Med. 1919. Vol.23, p.69.

64. CUNNINGHAM W. P. Ductless glands and Dermatology. New York Med.Journ. 1918, Vol.107, p.97.

65. DALE H. H. The Anaphylactic Reaction of Plain muscle in the guinea-pig. Journ. Pharm. & Exper.Therap. 1912, Vol.4, p.167.

66. DALE H. H. Herter Lectures. Bull. Johns Hopkins Hosp. 1920-21. pp.257, 310, 373.

67. " " Croonian Lecture. Proc.Roy.Soc, Series B. 1920. Vol.91, p.126.

68. " " Anaphylaxis. Johns Hopkins Hosp.Bull.1920. Vol. 31, p.310.

69. " " Anaphylatoxin. B.M.J. 1921, Oct. p.689.

70. " " Specific Sensitiveness and Anaphylaxis. B.M.J. 1922 p.45.

71. " " & KELLAWAY, C.H. Anaphylaxis and Anaphylatoxins. Philosoph. trans. Royal Soc. of London 1922. Series B. Vol.211 p.273

72. " " " Anaphylaxis & Anaphylatoxins, B.M.J. p.268.

73. " " & LAIDLAW P.P. Histamine Shock. Journ. of Physiol. 1918-19 Vol.52, p.355.

74. DEAN H. R. The Mechanism of the Serum Reactions. Lancet, 1917 Vol.1, p.45.

75. DOERR R. Ueber Anaphylaxie. Wiener Klin. Woch. 1912 Vol. 25 p.331.340.

76. " " Kolle & Wassermann. Handb.d.path.micro-organismen, 1913. Vol.2.p.947

77. DOLD, H. Ueber die Wirkung des Serums auf die wasseriges organextraktgifte. Berlin Klin. Woch.1912 Vol.49.p.2310.

78. DOERR, R. & RAUBITSCHEK, H. Toxin und anaphylaktisierende Substanz des Aalserums. Berlin Klin.Woch.1908 No.33.p.1525.

79. DOERR & RUSS. Studien über anaphylaxie. Zeitsch.f. Immunitf. II. Heft I.1909, p.109.

80. " " Der Anaphylactische Immunenkörper und seine Beziehung zum Eiweiss-Antigen. Zeitsch. f.Immunitätsforschung. 1909 III p.181. Originale.

81. DOLD, H.C. & AOKI K. Weitere Studien über das Bakterienanaphylatoxin. Zeitsch. f.Immunitatsfor. 1912. Orig. Vol.15.p. 171

82. " " " Über sogenanntes Desanaphylatoxieren von Bakterien.

83. DOLD, H. & AOKI, K. Beitrag zur Frage der Identität des in vitro darstellbaren Anaphylatoxins mit dem in vivo entstehenden anaphylaktischen Giften. Zeitsch.f.Immunitätsf.1912-13 Orig. Vol.16.p.357

84. DOLD, H. & OGATA S. Weitere Beiträge zur Kenntnis der wässerigen Organextraktgifte. Zeitsch.f. Immunitätsf.1912. Vol. 16, p.475.

85. DUPREZ. The anti-anaphylactic action of Lipoids. Compt. rendu Soc.Biol.Feb.4.1922 Abstr. B.M.J. Apr.8, 1922.

86. EPPINGER & HESS. Die Vagotonie. Sammlg.Klin.Abhandl. über Path.e.Ther.d.stoffwechseln u. Ernährungstörungen. 1910.

87. EWING J. Clinical Pathology of the Blood. London, 19 1901, p.137.

88. FENYVESSY & FREUND. Ueber den Mechanismus der Anaphylaxie. Zeitsch.f.Immunitätsfor. 1914 Vol.22 p.59.

89. FRIEDBERGER E. Kritik der Theorien über die Anaphylaxie. Zeitschr.f.Immun. 1909.II. (Orig.) 208.

90. " " Weitere Mitteilung über Anaphylaxie. Zur Theorie Friedbergers über anaphylaxie. Zeitsch.f.Immun.d.1909,III. p.692.

91. " " Weitere Untersuchungen über Eiweissenanaphylaxie. IV. Zeitsch.f.Immun. Orig.IV. Heft 5 p.636. 1909-10.

92. " " Die Anaphylaxie. Deutsch.Med.Woch.1911 No.11

93. " & HARTOCH Ueber das verhalten des Komplements bei der aktiven und passiven anaphylaxie. Zeitsch.f.Immunität. (Orig.) 1909 Vol.3. p.581

94. " & MITA. Versuche über aktive und passive Bakterien-anaphylaxie. Centr.f.Bakt. 1911.Bd.50. p.58,

95. " " & KUMAGAI T. Die Bildung eines akut wirkenden Giftes (anaphylatoxin) aus toxinen. Zeitsch.f.Imm.1913 Vol.17 p.506.

96. FRIEDBERGER, E. & TSUNEOKA R. Weitere Beiträge zur Wirkungsweise des Kaolins und anderer Chemischendifferenter und unlöslicher inorganischer kolloidaler Substanzen. Zeitsch.f.Immunitätsforsch.u.exper. Therap. Vol.20, p.405.

97 GASIS, D. Ueber die Untersuchung verschiedenen Pflanzeneiweissarten mit Hilfe spezifischer Sera. Berlin.Klin.Woch.1908 Vol.45.p.358.

98 GAY & SOUTHARD On serum anaphylaxis in the guinea-pig. Journ.Med.Res.1907 xvi.p.143.

99. " " On the mechanism of Serum Anaphylaxis and Intoxication in the guinea-pig. Journ.Med.Research 1908, xviii p.407.

100. " " Further Studies in Anaphylaxis. Journ.Med. Res. 1908, xix.p.1

101. HAYMAN & FAY. Studies on albuminuria and eosinophilia in scabies.

Arch.f.D. & S. 1921, Vol.III. p.32.

102. HEKTOEN, L. Further studies on the effects of the Roentgen ray on Antibody-production. Journ. Infect.D.Vol.xxii.1918, p.28.

103. HEMPL, H. The disappearance of Agglutinin from the blood of anaphylactic and normal animals. Journ.Immunol. 1916, Vol.II. p.157.

104. HERRICK, W. W. Experimental Eosinophilia with an extract of an animal parasite. Its relation to Anaphylaxis and certain clinical problems. Archiv.of Int.med. 1916.Vol.ii. p.165.

105. TANNER HEWLETT, Manual of Bacteriology 7th Edit.

106. HIGHMAN. Interstate Med.Journ. xxiv.p.5.

107. HOFFMANN R. Anaphylaxie und interne sekretion. Berlin Klin.Woch.1910, p.1925.

108. HOLOBUT. Zur Frage der Bakterienanaphylaxie. Zeitsch.f.Immunität.(Origin), 1909, Vol.3.p.639.

109. HOWARD, CAMPBELL P. The relation of the eosinophile cells of the blood, peritoneum and tissues to various Toxins. Journ. Med.Research 1907, Vol.17. p.237.

110. HUXLEY, JULIAN S. Chromosome Theory of Heredity. Science Progress 1921. Vol.16. p.249.

111. INOMATA V. Ueber die durch Pflanzensamen hervorgerufene ueberempfindlichkeit. Zeitsch.f.Immun. 1910. Referate Teil.ii. p.760.

112. INOUYE Ueber alimentare Albuminurie. Deutsch.Archiv. f.Klin.Med. 1903. Vol.75,p.378

Ueber Ricin Immunität.

113. JAKOBY Hofmeister's Beiträge 1902. Vol.i.p.51.

114. JANNEY N.D. & ISAACSON v.I. The Blood sugar in Thyroid and other endocrine Diseases. Archiv.Int.Med.1918.Vol.22.-p. 160.

115. JOBLING J.W. & PETERSEN W. The mechanism of Anaphylatoxin formation. Studies on Ferment action. Journ.Exper.Med.1914.Vol.xx.p.37.

116 " " " The Therapeutic Action of Iodin. Archiv.Int.Med.1915.Vol.xv.p.286.

117. " " " & EGGSTEIN A.A., The mechanism of Anaphylactic Shock. Journ.Exper.Med. 1915. Vol.xxii.p.401.

118. " " " " Serum proteases and the mechanism of the Abderhalden reaction. Journ.Exper.Med. 1915 Vol.21.p. 239

(The Myth of Anaphylaxis.)

119. JOUSSET Med. Presse 1919. Sept.9.p.250.

/20. KAHN & McNEIL. Complement Fixation with protein substances.
Journ.Immunology, 1918.Vol.III. p.277.

/21. KARASAWA, M. Ueber Anaphylaxie erzeugt mit pflanzlichen Antigen. Zeitsch.f.Imm. 1910 p.509

/22. KARSNER, H. F., & ECKER, E.E., The Principles of Immunology, 1921. Chap.X.

/23. KATO & YOSHIO. Zur Kenntnis ueber die Anaphylaxie mit Serum eiweiss.
Mittteilungen aus der mediz.Fakultät der K.Univ.zu Tokyo. Band xviii. 1917 p.195

/24. KEPINOW, L. & METALNIKOW, S. The Thyroid gland and Sensitivity to Tuberculin.
Compt.rendu. Soc.Biol.1922. June 24th, p.210.

/25. KEYSSER & WASSERMANN. Ueber Toxopeptide.
Zeitsch.f.Hyg.u.Infektionskr. 1911, Vol.68.p.535.

/26. KLINKERT D. Eosinophilia, Anaphylaxis and the nervous system. Nederlandsch.Tijdschrift voor Geneeskunde. 1917, 1 No.4.pp. 201-272.

/27. KOLMER A practical Text-book of Infection, Immunity & Specific Therapy. Chapter xxvii. p.530. "Anaphylaxis".

/28. KOPACZEWSKI W. & MUTERMILCH S. Sur l'origine des Anaphylatoxines.
Zeitsch.f.Immunitätsf.u.exper. Therap. 1914 (Origin.) Vol.22.- p.539.

/29. KOPACZEWSKI L'Anaphylaxie. Annales de médicine, Paris 1920, 7.p.361.Abs.J.A.M.A.1920, Vol.75 p.507

/30. " Antianaphylaxis. Annales de Méd.1920, Vol.8.291. Abs.J.A.M.A.1921 Vol.76 p.551

/31. KOWARSKI, ALB. Ueber den Nachweis von pflanzlichen Eiweiss auf biologischen Wege.
Deutsch Med.Woch.1901, Vol.27, p.442.

132. KRAUS, R. & AMIRADZIBI, F.S. Ueber Bakterienanaphylaxie.
Zeitsch.f.Immun. 1910, Originale Vol.4.p.607.

133. KRAUS, DOERR & SOHMA, Ueber Anaphylaxie hervorgerufen durch Organextrakte (Linsen).
Wiener Klin.Woch. 1908, Vol.21 p.1084.

134. KRITCHEWSKY, A Contribution to the Theory of Anaphylactic Shock. Journ.Infect.Dis. 1918 p.101.

135. DE KRUIF & J. GERMAN, Anaphylatoxin and amino-nitrogen.
Journ.Infect. Dis. 1917, Vol.20 p.833.

136. LARSON & BELL E. T., The Perfusion experiment in the study of cellular Anaphylaxis.
Journ. Infect.Dis. 1919. Vol.24. p.185

137. LEWIS J. H. Slow intravenous injection of anti-serum to prevent acute Anaphylactic shock
Journ. A,M,A, 1919 Vol.72. p.329.

138. LOEFFLER, F.C. Das Komplement als ausschlaggebender Factor für das Zustandekommen des Anaphylactisches Anfalles.
Zeit.f.Immunitätsf. viii.1910. p.129

139. LUMIÈRE & CHEVROTIN Sodium Hyposulphite & Anaphylaxis. Acad.des Sciences. Paris. 18:10:1920.

140. McEWEN, E. L. The relation of nerve impulse to cutaneous inflammation.
J.A.M.A. 1906, Vol.47.p.8.

141. McNEIL C. Critical Review. Anaphylaxis in man. Its bearing upon Hay-fever, animal and food idiosyncrasy and Asthma.
Edin.Med.Journ. March 1921.

142 MANWARING, W. H. Der physiologische mechanismus des Anaphylaktischen Shocks.
Zeitschrift f.Immunit. 1910, Vol. 8, p.1. & 589.

143. " " Intestinal & Hepatic reactions in Anaphylaxis.
J.A.M.A. 1921 Vol.77.Sept.10.

144. MANWARING W. H. & CROWE H. E., Passive cellular Anaphylaxis.
Proc.Soc.Exper.Biol. & Med. 1916-17, Vol.14, p.173.

145. " " " The Role of Hepatic Tissue in Anaphylaxis.
Proc.Soc.Exper.Biol.& Med.1916-17, Vol.14, p.174.

146. " " " Absorption of foreign protein by the Anaphylactic lungs.
Proc.Soc.Exper.Biol.& Med.1916-17, Vol.14 p.129

147. " " " Role of hepatic tissues in the Acute Anaphylactic reaction.
Journ.Immunol. 1917, Vol.2.p.517.

148. " " " Types of Anaphylactic Reaction
Proc.Soc.Exper.Biol.& Med. 1917, Vol.14, p.173.

149. " " " The role of hepatic tissues in the acute anaphylactic shock.
J.A.M.A.1917. Vol.xix.p.772.

150. " W.H. & KUSAMA Y. Analysis of the Anaphylactic & Immune reactions by means of the isolated guinea-pig lungs.
J.Immunol. 1916. Vol.2.p.157.

151. " " " & CROWE H.E. Fate of the foreign protein in the acute anaphylactic reaction.
Journ.Immunology 1917. Vol.2, p.511

152. " " " " Abst. J.A.M.A. Vo.lxix. p.1297

153. MAJOR R. H. Ueber den Einfluss der Anaphylaxie auf den stickstoff-stoffwechsel bei kaninchen. Deutsch. Arch.f.Klin.Med.1914. cxvi.p.248

154. MAJOR & NOBEL. Ueber die empfindlichkeit der kindlichen Haut gegenueber Dysenterietoxin und Tuberculin.
Zeitschr.f.exper.med.1913 ii.p.9

155 MARBE S., & RACHEWSKI, Etudes sur l'anaphylaxie.
Compt.rendu Soc.de Biol. Tome69. 1910 pp.529 & 531.

156. MATSUURA & NISHIURA, Japanische Zeitsch.für Derm. & urologie, 1910 Bd.10, Heft 6.

157. METALNIKOW. Anaphylaxis & Chemiotaxis.
Compts rendus Soc.Biologie. May 21, 1921

158. METCHNIKOFF, Elie. L'immunité,
Paris 1901. p.124.

159. MICHAELIS & OPPENHEIMER C. Ueber Immunitat gegen Eiweisskörper.
Arch.f.Anat. und Phys. 1902, Supplement p.336.

160. MOLDOVAN. Ueber die Wirkung intravaskulärer Injektionen frischen defibriniertes Blutes und ihre Beziehungen zur Frage der Transfusion.
Deutsch.Med.Woch. 1910. Vol.36 p.2422.

161. MOORE W. H. On the mechanism of the anaphylactic reaction in smooth muscle.
Proc.Soc. Exper.Biolog. & M. 1915, Vol.12. p.175.

162. " " Comparative Physiology of immune and anaphylactic smooth muscle.
Proc.Soc.Exper.Biol. 1914-15. Vol.12.p.176.

163. MOSCHOWITZ, E. Eosinophilia and Anaphylaxis.
New York Med.Journ. 1911. Vol.93 p.15.

164. MUIR R. Studies on Immunity. London 1909.

" " & RITCHIE J. Manual of Bacteriology. 7th Ed. p.595.

165. NATHAN. Ueber Anaphylatoxin- bildung durch Stärke.
Zeitsch.f.Immunitätsfor. 1913, Vol.18.p.636.

166. NEUSSER. Klinisch-hämatologische Mittheilungen.
Wiener Klin.Woch. 1892. Vol.5. pp.41 & 64.

167. NICOLLE, M. & POZERSKI E. Une exception generale des anticorps et de leurs effets.
Ann.de l'Inst.Past.1908.xxii. pp.26, 132 & 237.

168. NOVY F. G. & de KRUIF, P.H. & NOVY R. L. (Anaphylatoxin and Anaphylaxis. Trypanosome anaphylatoxin.)
J.Infect.Dis.1917, Vol.20. p.499.

169. NOVY F.G. & De KRUIF P.H., Agar anaphylatoxin - guinea-pig serum.
Journ.Infect.Dis.1917. Vol.xxx. p.536.

170. NOVY F. G. & De KRUIF P.H. Agar anaphylatoxin. Rabbit serum.
Journ.Infect.Dis. 1917 Vol.20, p.566.

171. " " " " Agar anaphylatoxin. Rat Serum.
Journ.Infect.Dis. 1917. Vol.20. p.589

172. " " " " Effect of intravenous Injection of Agar.
Journ.Infect.Dis. Vol.20.p.629.

173. " " & " " Specific Anaphylactic Shock.
Journ.Infect. Dis. 1917, Vol.20 p.776.

174. " " " " Anaphylatoxin & Anaphylaxis. J.A.M.A. May 26, 1917. p.1524.

175. NEUSSER.
Wiener Klin.Woch.1894 Vol.7.p.737

176. OBERMAYER, F. & PICK E. P. Ueber die chemischen Grundlagen der Arteigenschaften der Eiweisskörper.
Wiener Klin.Woch.1906.No.12.p.327

177. OTTO Zur Frage der Serum Ueberempfindlichkeit.
Munch.Med.Woch. 1907 p.1665

178. PARK E. A. A case of hypersensitveness to cow's milk.
Am.Journ.Dis.child. 1920 Volxix. p.46.

179. PEARCE, R. H. & EISENBREY, A. B. The Physiology of Anaphylactic Shock in the dog.
Journ.Infect.Dis. 1910. Vol.7 p.565.

180. PESCHI. Researches on the theory of Anaphylaxis.
Ann. de l'Inst.Pasteur. May 1921

181. PFEIFFER. Versuchstechnische Bemerkungen zum Nachweis des anaphylaktischen Temperatursturzes.
Wiener Klin.Woch. 1909 xxii,1227

182. PFEIFFER H. Ist der Temperatursteigerung als Kriterium bei der passiven Uebertragung Tuberculoseüberempfindlichkeit anzusehen.Zeitsch.f.Immun.1910 Vol.4

183. PICK E. P. & YAMANOUCHI T. Studien ueber Anaphylaxie.
Wiener Klin.Woch. 1908 No.44. p.1513.

184. POLLITZER S. Skin Diseases in relation to the Nervous System.
New York Med.Journ. 1912. Vol.96 p.574.

185. POTTENGER, F. M. Relationship of the Syndrome of Anaphylaxis to the vegetative nervous system.
Journ.Immunol. 1917, Vol.2.p.452.

186. REBAUDI A. Sui fenomeni anafilattici nel campo dermatologico.
Genova. 1921.Artistici Tipografici.

187. REEDE, Edward H. The Role of the Vegetative nervous system in Diseases of the Skin.
Journ. cut.D. 1918, p.505.

188. RELANDER, L. K. Kann man mit Präzipitinreaktion Samen von verschiedenen Pflanzarten und Abarten von einander unterschieden?
Centralbl.f.Bakt.1908 (2 abt.) Vol.20 p.518.

189. RICHET C. L'anaphylaxie crée un poison nouveau chez l'animal sensibilisé.
Compt.rendu. Soc.Biol.1909, Vol. 66. p.810.

190. " " Anaphylaxis. Translation by J.Murray Bligh. 1913. Constable & Co. Liverpool Univ.Press.

191. RITCHIE JAMES. Anaphylaxis as a factor in Disease in Man.
Northumberland & Durham Medical Journal Vol.xxiii. No.87.

192. " " & MILLER J. An enquiry into the question of whether lipoids act as antigens.
Journ.Path. & Bact. 1913. Vol.17. p.429.

193 RITZ & SACHS Ueber das Anaphylatoxin.
Berlin.Klin.Woch.1911. Vol.48 p.987

194. ROSENAU & AMOSS. Organic matter in the expired breath.
Journ.Med.Research 1911-12,Vol. 25.p.35.

195. ROSENAU & ANDERSON A Study of the cause of sudden death following the injection of horse serum.
Bull. No.29 Hygienic Lab.1906 April.

196. " " " Further Studies upon hypersusceptibility and immunity.
Hyg.Lab.Bull.No.36.U.S.Pub.H. & Mar.Hosp.Ser.Washington, 1906.

197. " M. " " J.F. Further Studies upon the phenomenon of anaphylaxis.
Bull.No.50 Hyg.Lab.U.S.Pub.Health & Mar.Hosp.Serv.Washington, 1909.

198. " " " The Specific Nature of Anaphylaxis.
Hyg.Lab.Bulls.36 U.S.P.H.& M.H.S. April 1907.

199. " " " The Specific nature of Anaphylaxis.
Journ.of Infect.D.1907 Vol.IV. p.552.

200. " " " Further Studies upon hypersusceptibility and immunity.
Journ.Med.Research, 1907.Vol.16. p.381.

201. " " " Further Studies upon Anaphylaxis.
Bull. No.45.Hygiene Lab. 1908.

202. " " " The Specific Nature of Anaphylaxis.
Hyg.Lab.Bull.U.S.P.H.& Mar.H.Ser. June 1908, p.65.

203. " " " Further studies upon Anaphylaxis.
Journ.Med.Res. 1908. Vol.xix.p.37.

204. " " " Anaphylaxis (Harvey Lecture).
Archiv.Int.Med.1909 Vol.3.p.519.

205. " " " Further studies on the phenomenon of Anaphylaxis. Bull.Hyg.Lab.No.50. U.S.A.P.H. & M.H.S. Washington, April 1909.

206. ROSENSTERN J. Exudative Diathese und Eosinophilie.
Jahrbuch für Kinderheilkunde Vol. 69, p.631.

207. SALECKER P. Blutuntersuchungen bei Asmatikern .
Münch. Med. Woch. 1907, Vol.54 p.358.

208. SCHEPPEGRELL. Pub.Health Rep. 31, p.1907 (July 31) 1916. Ibid.32.p.1135 (July 20) 1917.

209. SCHENK, Ferd. UEber den Uebergang der Anaphylaxis von Vater und Mutter auf das Kind. Münch.Med.Woch.1910 No.48.p.2514.

210. SCHIASSI. Auto-anaphylaxis in Paroxysmal Haemoglobinuria. , (Il Policlinico.Sez.Med.,Fasc.9 and 10). Abstr.in B.M.J.April 23, 1921.

211. SCHLECHT H. Ueber Einwirkung von Seruminjektionen auf die Eosinophilen und Mastzellen des Menschlichen und tierschen Blutes. Deutsche. Archiv. f.Klin.med.1910 Vol.98,p.308.

212. " " Ueber experimentelle Eosinophilie und basophile Leucocytose. Verhandl. d.Kongr.f.inn.Med. Wiesbaden. 1910. Vol.27. p.483.

213. " " Ueber experimentelle Eosinophilie nach parenteraler Zufuhr Artfremden Eiweisses und ueber Beziehungen der Eosinophilie zur Anaphylaxie. Arch.f.exper.Path.u.Pharm. 1912. Bd.67. p.137.

214. " " Ueber lokale Eosinophilie beim anaphylaktischen Versuche. Verhandl. 2 Kong.f.Inn.Med. Wiesbaden 1912.Vol.29.p.416.

215. SCHLECHT G. & SCHWENKER. Ueber lokale Eosinophilie in der Lunge Anaphylaktischer meerschweinchen. Archiv.f.exp.Path.u.Pharm. 1912. Bd.68.

216. SCHULTZ, W. H. Reaction of smooth muscle of the guinea-pig, sensitized with Horse Serum. Journ.Pharm. & exper.Therap. 1910 Vol.1.p.549.

217. " " Reaction of smooth muscle from various organs of different animals to protein. Bull.Hyg.Lab.No.80, 1912.January.

Abstr.Amer.J.Dis.Child, 1920.p.394

218. " " & JORDAN H. E. A microscopic study of the anaphylactic lung of the guinea-pig and mouse. Journ.Pharm & Exper.Therap.1911 Feb.II. p.375.

219. SCHWENKER G. & SCHLECHT, H. Ueber den Einfluss sympathekc und Autonomotroper Substanzen auf die eosinophilen Zellen. Zeitsch. f.Klin.Med.Vol.76.p.77.

220. SCOTT, W. M. On Anaphylaxis and the behaviour of Complement. Journ. Path. & Bact. 1910, Vol.14 p.147.

221. SICARD J. A., & PARAF, J. Prophylaxis of Anaphylaxis by Sodium Carbonate intravenously. Bull.et Mem.Soc.Med.d'Hop. de Paris. Jan 28, 1921. Abt.J.A.M.A. April 2. 1921 Vol.76 p.965.

222. SIMONDS. Anaphylactic Shock in Dogs. J.Infect.Dis. 1916, Vol.19. p. 746.

223. SKINNER, E. F. Blood Transfusion. B.M.J. 1923, i. p.750

224. SLATINEANO & CIUCA, Pouvoir toxique du sérum normal de cobaye et réactivation par un colloid de ce sérum ayant perdu sa toxicité en vieillissant. Compte.rendu Soc, de Biol. 1913 Vol. 74. p.631.

225. SMITH, G. H. & COOK, M. W. The Specificity of Intracutaneous Absorption. Journ. Immun. 1918. Vol. 3, p. 35.

226. SMITH, M. J. & RAVITZ, S. Epinephrine Content of Suprarenal Glands in Anaphylaxis. Journ.Exper.Med.1920, Vol.32. p.595.

227. SOULA, L. C. Essai doctrinal sur l'anaphylaxie. Presse médicale XXIV. Oct.23.1916. No.59 p.471

Abstr. in Journ.A.M.A.1916, p. 1794.

228. STAUBLI C. Zur Kenntnis der lokalen Eosinophilie. Münch.Med.Woch. 1905. Bd.52, p. 2072.

229. " " Ueber Eosinophilie. Volkmann's Klinische Vorträge 1909-10 No.543,p.43.

230. " " Die Klinische Bedeutung der Eosinophilie Ergebn.d.inn.Med.u.Kinderheilkd. 1910. Bd.6.p.209.

231. STCHASTNYI, S.M. Ueber die Histogenese der eosinophilen granulationen im Zusammenhang mit der Haemolyse.
Ziegler's Beiträge zur Path.1905. Vol.38 p.456.

232. SUTTON R. L. Anaphylaxis or Allergy. Diseases of Skin. 3rd Edit. 1919, p.51.

233. SVESTKA V. Eosinophilia and its relation to Skin Diseases. Ceska Dermatologie 1919. Nov.3.& 4.
Abstr. B.J.D., 1921.p.351.

234. TANIGUCHI T. Studies on heterophile Antigen and antibody.
Journ.Pathol. & Bact. 1921. Vol.24 p.217. & p.456.

" " Journ. " 1922. Vol.25.p.77

235. TERREROS, C.S. De Los, Diathesis & Anaphylaxis. Plus-Ultra, Madrid. Feb.1919. II. No. 8. p.84
Abstr. J.A.M.A. 1919 lxxiii.p.374

236. THIELE F. H. & EMBLETON D. On the Role of Lipoids in Immunity.
Zeitsch.f.Immunitatsfor. & Exper. Therap.1913, Vol.16, p.160.

237. THOMSEN O. "Ueber die Spezifizität der Serumanaphylaxie und die Möglichkeit ihrer Anwendung in der medikoforensischen Praxis zur Differenzierung von Menschen-und Tierblut (in Blutflecken etc.).
Zeitschr. f.Immunität.(Orig.) 1908-09. Vol.1.p.741.

238. THOMSON J. The Clinical Study and Treatment of Sick Children. 3rd Edit.1921.

239. TURK, W. Vorlesungen ueber klinische Haematologie, Wien, 1904.

240. UHLENHUT. Neuer Beitrag zur spezifischen Nachweis von Eisreiweiss auf biologischen Wege.
Deutsch. med.Woch. 1900, Vol. 26. p.734.

241. UHLENHUTH & HAENDEL. Untersuchungen ueber die praktische Verwertbarkeit der Anaphylaxie zur Erkennung und Unterscheidung verschiedener Eiweissarten.
Zeitsch.f.Immunol. 1909-10,Vol 4.p. 761.

242. VAUGHAN. Protein split products in relation to Immunity & Disease.
Amer.Journ.Dis.child. 1913. p.245

243. VAUGHAN, V. C. & WHEELER S. M. The effects of egg white and its split products on animals; A Study of Susceptibility & Immunity.
Journ.Infect.Dis. 1907. IV. p.476.

244. WEIL, RICHARD. The Nature of Anaphylaxis and the Relation between Anaphylaxis & Immunity.
Journ.Med.Research Boston, 1912-1913. Vol. 27. p. 497.

245. WEIL, R. W. Desensitisation; Its theoretical and practical significance.
Journ.Med.Research 29, 1913. p.233.

246 " Studies in Anaphylaxis. On the relation between Precipitin and sensitizin
Journ.Immunol. 1915. Vol. 1 p. 1.

247. " " " " Equilibrium in precipitation reactions. Equilibrium in combination.
Journ.Immunol. 1916. Vol.1. p. 19.

248. " " " " Equilibrium in precipitation reactions - Dissociation.
Journ.Immunol. 1916. Vol.1. p. 35.

249. " " " " On the coexistence of Antigen and antibody in the body.
Journ. Immunol. 1916 Vol.1. p. 47.

250. " " " " The mechanism of delayed shock.
J. Immunol. 1916. Vol. 2. p. 95.

251. " " " " Simultaneous injections of antigen and antiserum. The anaphylatoxin theory of Anaphylaxis.
Journ.Immunol. 1916. Vol.2. p.109.

252. " " " " The reciprocal relations of Antigen and antibody within the cell.
Journ.Immunol. 1916.Vol.2. p.469.

253. " " " " Anaphylaxis in dogs. A study of the liver in shock and in peptons poisoning. J.I. 1917. Vol. 2. p.525.

254. WEIL R. & EGGLESTON C. Studies in Anaphylaxis. Anaphylactic reactions of the isolated dog's liver.
Journ.Immun. 1917.II.p.571.

255. WEINBERG M. & SEGUIN. Anaphylaxis et Eosinophilie.
Compt. rendu Soc.de Biol. Vol.76.

256. WEISS HUGO, & TSURU I. Ueber den Einfluss der anaphylaktischen Shocks auf das Blut.
Zeitsch. fur Immun.1910.P.516.

257. WELLS, H. G. Studies on the chemistry of Anaphylaxis.
Journ.Infect.D. 1908. Vol.5.p.449.

258. " " " " "
Experiments with isolated proteins.
Journ.Infect. Dis. 1911.Vol.9.p. 147.

259. " " The"present status of the Problems of Anaphylaxis.
Physiological Review 1921. Jan..p. 1.& 44.

260. " " & OSBORNE, T. B. The biological reactions of the Vegetable proteins.
Journ. Infect.Dis. 1911. Vol.8.- p.66.

261. " " " " Is the specificity of the anaphylaxis reaction dependent on the chemical constitution of the proteins or on their biological relations. The biological reactions of the vegetable proteins.
J.Infect.Dis. Vol.12.1913.p.341.

262. " " " " The Anaphylactogenic Activity of some vegetable proteins.
J.Infect.Dis. 1913.Vol.14.p.377.

263. WENDELSTADT & FELIMER. Beitrag zur Kenntniss des Immunisierung durch Pflanzeneiweiss.
Zeitsch.f.Immun. 1910.viii. p.43

264. WHIPPLE & COOKE. Proteose intoxications and injury of body protein.
I. The metabolism of fasting dogs following proteose injections.
Journ. exper.med.1917. Vol.25.p. 461.

265. " " VAN SLYKE, Proteose intoxications and injury of body protein.
Journ.exper.med.1918, Vol.28,p. 213.

266. WHITE BENJAMIN, Some experiments in Anaphylaxis with the Lipoids of the Tubercle Bacillus. Journ. Med. Research, 1914. Vol.30 p.393.

267. WILE. The role of Anaphylaxis in the relation of the skin to disorders of the gastro-intestinal system. Med.Rev. of Rev. 1912. xviii.p. 379.

268. WILENKO, M. Ueber das Präzipitationsvermögen pflanzlischer Eiweiss-stoffe. Zeit.f.Immunitätsfor.1910.p.91.

269. WITZINGER, von. Zur anaphylactischen analyse der Serumkrankheit. Zeitsch.f.Kinderheilk.1911-12. Vol.3, p.211.

270. WYARD, S. The phenomena of Anaphylaxis. Lancet 1917, Vol.1.p.105.

271. ZADIK, P. Experimentelle Studien ueber Toxopeptide. Folia serol. 1911 Vol.7.p.865.

272. ZAPPERT, J. Ueber das Vorkommen der eosinophilen Zellen im menschlichen Blut. Zeitsch.f.Klin.Med. 1893, Vol.23 p.227.

273 ZINSNER, HANS. Infection & Resistance. New York 1914.

274. " Identity of Antibodies. Journ. Immunol.Sept.1921. Abstr. B.M.J. Jan., 7th, 1922.

275. " " LIEB, C.C., & DWYER, J.G., On the action of Sodium Chloride in the prevention of proteotoxin shock. Proc.Soc.Exper.Biol.& Med. 1915, Vol.12, p.204.

276. " " & PARKER, J.T., Studies on Bacterial anaphylaxis & Infection. Journ.experim.Med. 1917, Vol.26. No.3.p.411.

SERUM SICKNESS.

277. BOUGHTON, T. HARRIS, Anaphylactic Death in Asthmatics
Journ.A.M.A. 1919 lxxiii.p.1912.

278. BRODIN, P. Improved Technique for Serotherapy.
Presse Med. 1920 Vol.28.p.807.

279. BROWNING, C.H.,
Brit.Journ.of Surgery, 1916, Vol. 4, p.13.

280. BURROWS, H. Modified Tetanus.
Lancet 1917, Vol.1.p.139.

281. DAVIDSON, W.T.G., An investigation into the phenomena of Serum Disease. The relation between its various forms and the proteins of Horse Serum.
Glasgow Med.Journ.1919, pp.75 & 129.

282. DEAN, H.R. Fatal Anaphylaxis in Man.
Journ. of Path. & Bact.1922.p.305

283. FIÈVEZ, J. Serum Anaphylaxis. Progrès médical, 1918. Vol.33.p.318.
Abstr. in J.A.M.A. Vol.71.p.1946

284. JOUSSET, Massive doses in Serotherapy.
Bullet.de l'academie de Med. Paris. June 1918. 79. No.22. p.432.
Abstr. in Journ.Amer.Med.Assoc. 1918 p.605.

285. KER, C.B. Serum Sickness. Infectious Diseases. Chap.11.

286. LEISHMAN, Sir W.B. & SMALLMAN A.B., Recent Cases of Tetanus in the British Expeditionary Force, with special reference to their treatment by Antitoxine.
Lancet 1917. Vol.1.p.131.

287. LEMANN, J.J. Prophylactie Injections of Serum and the theory of Anaphylaxis.
New Orleans Med. & Surg.Journ. Aug.1909.
Abstr.J.A.M.A.1909, May 22, p. 1687.

288. LEWIS, J.H. Slow intravenous injection of Anti-serum to prevent Anaphylactic Shock.
Journ.Amer.Med.Assoc. 1919.lxxii. p.329.

289. LEWIS, J.H. The route and rate of Absorption of subcutaneously injected serum.
J.A.Med.A.1921, Vol.76.p.1342.

290. MACKENZIE, G.M. Serum Desensitization.
J.A.M.A. 1921. Vol.76.p.1563.

291. MARTIN Danger of Anaphylaxis with Antitoxic Serums.
Rev.d'Hygiène. Nov.1919.
Abstr.in B.M.J. Jan.17, 1920.

292. PÉHU & BERTOYE. Death from Anaphylaxis.
Journ.de Med.de Lyons. Aug.5, 1921
Abstr.B.M.J.Sept.17 1921.

293. VON PIRQUET & SCHICK. Die Serumkrankheit.
Leipzig, 1905.

294. ROSENAU, M.J. & ANDERSON, J.F. A Study of the cause of sudden death following the injection of horse serum.
Bull.Hyg.Lab. No.29. U.S.P.H.& M.H.S. Washington, 1906, [illegible].

295. SCHAMBERG. Discussion on Toxic Dermatoses.
Journ.Cutan.Dis. 1912.Vol.30.p.163

296. SEQUEIRA. Serum Eruptions. Diseases of the Skin. London. 3rd Edit.1919, p.350.

297. STEWART, F.E., Serum Disease, Anaphylaxis and Allergie.
New York Med.Journ. 1917, Vol.106. p. 644.

298. SUMNER, F.W., Sudden Death from Anaphylactic Shock.
B.M.J. 1920. Vol.I.p.465.

299. UNTVEDT. Are Serum Disease and Anaphylaxis Identical?
Norsk.Mag. for Laegevidenshaben, July 1920.
Abstr. B.M.J. Nov.13.1920

300. WEIL, RICHARD, Serological analysis of a case of serum sickness in a human being.
Proc.Soc.Exper.Biol. & Med. 1915, Vol.12.p.37.

301. " " Further studies in Serum Sickness.
Proc.Soc.Exper.Biol.& Med.1916-17. Vol.14.p.60.

302. WOODYATT, R.T., The method of timed intravenous injections.
Journ.Biol.Chem.1917.Vol.39 p.355.

303. WYARD, S. Precipitins and the etiology of Serum Sickness.
Journ.Pathol.1920, p.191.

EXANTHEMATA.

304. ARLOING, F., DUFOURT A., & LANGERON, L. Therapeutic anaphylactic Shock.
Bull.de l'acad.Med.1921.Vol.85. p.241.
Abstr. J.A.M.A. Vol.76,p.1048.

305. BARASCH J. H. Vaccination and local Anaphylaxis.
Journ.A.M.A. 1913.Vol.60.p.569.

306. BOUCHE G. & HUSTIN A., Allergic Phlegmons.
Journ. de Chirurgie. 1921. No.5
Abstr.J.A.M.A. 1921 Vol.76.p.1711.

307. COOKE, R.A. & VANDER VEER, ALBERT jr.
Journ. of Immunology Vol.1. No.3.
June 1916.p.201.

308. DUKE, W.W., Multiple Infections - A Study of the relation of one infection to another.
Journ. Amer.Med.Assoc. 1918.lxxi. p.1703.

309. FERRY, N.S. The phenomena of Anaphylaxis. Its Clinical Significance & practical utilization.
Therapeutic Gazette 1916,Vol.40. p.843.

310. FORCE, J.N. & BECKWITH, H.L. A laboratory method for the diagnosis of smallpox.
J.A.M.A.1915.p.588.

311. HEKTOEN L. Allergy or Anaphylaxis in experiment, and disease.
J.A.M.A. 1912. lviii.1081

312. HOFFMANN, E. Protective function of the skin (Esophylaxis).
Deutsch.Med.Woch.1919, Vol.45. p.155.

313. JOHNSTON, J.C. Some toxic effects on the skin of disorders of digestion and metabolism.
Journ.Cut.Dis. 1912. Vol.30. p.136.

314. KEYSSELITZ & MAYER. Ueberempfindlichkeitsprüfungen bei Variolarekonvaleszenten. Arch.f.Schiffs.u.Tropenhyg. 1908 p.775.

315. KOLMER, J.A., Complement fixation in Varicella. Journ.Immun.1916, Vol.1.p.51.

316. " " Complement fixation in Vaccinia and Variola. Journ.Immunol.1916,Vol.1.p.59.

317. Von PIRQUET. Eine Theorie des Blatternexanthems. Wien.Klin.Woch. 1907.p.9.

318. " Zur Theorie der Vakzination. 1903.

319. " Klinische Studien über Vakzination und vakzinale allergie. Leipzig & Wien Deuticke. 1907.

320. " & SCHICK. Zur Theorie der Inkubationszeit. Wiener Klin.Woch.1903, Vol.26.p. 45.

321. ROGER, G.H. Nouveau Traité de Médicine 1920. Fasc.I. Maladies Infectieuses.

322. ROLLESTON, H. Asthma and allied disorders. B.M.J. 1921.ii., 231.

323. ROSENFELD, J. Successive Cow-pox vaccination. J.A.M.A.1912 Vol.59.p.16.

324. SUGAI, T. Ueber der Komplementbindungsversuch bei variola vera. Centralbl.f.Bakt.etc. 1909.Bd.49 Originale. p.650.

325. TIECHE Einige weitere Notizen ueber die Variola-epidemien und Virus. Korrespondenzblatt f.Schweizer Aerzte. 1915. p.1291

326. VAUGHAN, V.C. Protein sensitization and its relation to some of the Infectious Diseases. Zeit.f.Immun. I. 1909. p.251.

327. ASAMI, Oedema cutis circumscriptum acutum und Anaphylaxia. Japansche Zeitschrift für Dermatologie & urologie. 1919. Feb.14, p.134.

328. BARBER, H. W. A case of Urticaria of eight years duration associated with hyperthyroidism. Guy's Hosp.Gaz.1920.Ap.17.

329. " " Chronic Urticaria and Angioneurotic Oedema due to bacterial sensitisation. B.J.D.& S., 1923, p.209.

330. BARLING, J. E. & WELSH, D.A., The Leucocytosis of Hydated Disease. Austral. Med.Gaz. Aug.20, 1906.

331. " " " " Symptom-complex in ruptured abdominal Hydatid. Lancet. 1910. Vol.2.p.1001.

332. BEDDARD. Periodicity of Gnat bites. B.M.J. 1922, ii. p.951.

333. BOYCOTT, A. E. The Reaction to Flea-bites. Journ. of Path. & Bact. 1912-13 Vol.17 p.110.

334. BRAMWELL, Wm. Periodicity of Gnat Bites. B.M.J. Nov.25, 1922.

335. BRUCE, A. N. Ueber die Beziehung der Sensiblen Nerven digungen zum Entzundungsvorgang. Arch. f.Exp.Path. u.Pharm. 1910 Vol.63. p.424.

336. BRUCK C. Experimentelle Beiträge zur Aetiologie und Pathogenese der Urticaria. Arch.f.Dermat. und Syph. 1909. Bd.96.p.241.

337. CHAUFFARD, BORDIN & LAROCHE. Anaphylaxie hydatique experimentale. Compt. rendu.Soc,Biol.1909. Vol. 67.p.32.

338. CHIARI R. & JANUSCHKE H., Hemmung von Transsudat- und Exudatbildung durch Kalziumsalze. Wiener.Klin.Woch.1910. Vol.23. p.427.

339. CLARKE, J.A. & MEYER, G.P. A Case of Hypersensitiveness to Silk.
J.A.M.A. 1923.Vol.80.p.11.

340. Von COPPOLINO. Anaphylaxis-phenomena in the skin.
Giorn.Ital.d. mal.ven.e.della pelle. 1911. Fasc.V.

341. CROWDER, J.R. & T.R., Five Generations of Angioneurotic Oedema.
Archives of Internat.Medicine 1917 Dec.p.840.
Abstr. in Journ.Cut.D. 1918, p. 266.

342. DAVIDSON, A. The relation of fruit ingestion to Cutaneous Diseases.
Journ.Cutan.Dis. 1917, Vol.35. p.665.

343. DÉVÉ, F. Anaphylaxie hydatique post-operatoire mortelle.
Comptes rendus Soc.de Biol. de Paris. 1910.ii.p.400.

344. EDGERLEY, E.T. & LUSH, F.B., Angioneurotic oedema.
J.A.M.A.1919 Vol.72, p.1816.

345. FISCHER, MARTIN. Oedema & nephritis. 1915.

346. FORDYCE, J.A., The Influence of Anaphylaxis in the toxic dermatoses.
Journ.Cutan.Dis. 1912, Vol.30. p.128.

347. " Discussion on Protein Sensitization.
Archiv.of D. & S. 1920. Vol.2. p.571.

348. FOX, H. & FISCHER J.E. Discussion on Protein Sensitisation.
Arch.of D. & G. 1920, Vol.2,p. 572.

349. FREEMAN, J. Toxic Idiopathies.
Lancet 1920.Vol.2.p.229.

350. FROST, L. C. A Case of intense food Anaphylaxis.
Med.Record 1915. Sept.8

351. GILCHRIST T. C. Experimental urticaria.
Duhieng's Text Book on Cutaneous Medicine. 1895, Vol.I.p.129 & Vol. II. p.293.

352. " " Exper.urticaria.
John Hopkins Bulletin Vol.vii. No.64.p.141. (1896)

353. GILCHRIST. Some experimental Observation on the histopathology of urticaria factitia.
Trans. VI. Internat.Cong.1907 ii. p.905.

354. GILCHRIST T.C. Do. Do. Do.
Journ.Cutan.Dis. 1908, p.122.

355. HARTZELL. Discussion on Protein sensitization.
Archiv.f.D.& S. 1920, Vol.2, p.571.

356. HAZEN, H.H., Syphilis as a cause of Chronic Urticaria
Amer.Journ. of Syphilis. Oct. 1917, p.750.
Abstr. in Journ.Cut.D.July 1918, p.418.

357. " Discussion on Protein Sensitization.
Arch.of D.& S. 1920, Vol.2,p.573

358. HEIDINGSFELD,M.L. Formaline Urticaria.
Journ.Cut.Dis.1916, Vol.34, p.303.

359. HIGHMAN W. J. & MICHAEL, J.C. Protein sensitization in Skin Diseases; and Urticaria and its allies.
Archiv.of.D.& S., 1920, Vol2. p.544.

360. HODARA, M. Untersuchungen über die Histologie der urticaria factitia (Dermographigmus) und der urticaria chronica, sowie über die mastzellen bei beiden Affektionen.
Dermat.Woch.1913, Bd.57, p.971.

361. HOFFMANN, R. Anaphylaxie und interne sekretion.
Berlin.Klin.Woch.1910 p.1917.

362. HOLDEN, F.N. Angioneurotic Oedema.
S.African Med.Record, 1920, July 24.

363. JOERG O. Urticaria.
N.Y. Med.Journ.1917, Vol.106, p.647.

364. KREIBICH, Die Angioneurotische Entzündung.
Wien.1905.

365. " C. Ueber nervöse überempfindlichkeitder Haut.
Archiv.f.D. & S., 1908. Bd.93, p.59.

366. LEVI & ROTHSCHILD. Hypothyroïdie et urticaire chronique.
Gaz.des hôpitaux. 1906.No.79,p. 944.

367. LEVI, L. & ROTHSCHILD Un Cas d'instabilité vasomotrice provoquée par le traitement thyroidien.
Compt.Rend.Soc.de Biol.1909.
Thoma 68.- p.104.

368. LONGSCOPE & RACKEMANN. Severe Renal insufficiency associated with attacks of Urticaria in hypersensitive Individuals.
Journ. Urol.1917, Vol.I.p.351.

369. LOUSTE A. Urticaria.
Paris Medical 1920 Vol.10? p.198

370. McBRIDE W.L. & SCHORER, E.H., Erythema and urticarial erythema resulting from sensitization to certain foods.
Journ.Cut.Dis. 1916. Vol.34.p.70.

371. McKAY, H. Focal Infection and Chronic Urticaria.
Canad.Med.Assoc.Journ.July 1919. p.603.

372. MOORE, W. An interesting reaction to Louse bites.
Journ. Amer.Med.Assoc. 1918, Vol.lxxi. p.1481.

373. NEISSER. Ueber Vitiligo mit lichenoider Eruption.
Verhandl.d.deutsch.dermat.Gesellschaft IV. Kongress. p.435.

374. PAGNIEZ, Ph. & PASTEUR VALLERY-RADOT. Etude physiopathologique et therapeutique d'un cas d'urticaire géante; Anaphylaxie et anti-anaphylaxie alimentaires;
La Presse médicale 1916.p.529.
Abstr.in Annales de D.& S., Tome vii. Nos.5-6.July 1919).

375. " " " " Digestive Antianaphylaxis.
Bull.de la Soc.med. des Hôpitaux Paris 1919, 43, No.19, p.549.
(Abstr. in J.A.M.A. Vol.73, No.7 p.562)

376. PHILIPPSON. Angioneurosen und hematogene Entzündungen
Rapports officiels. Sect.xiii. 16th Cong.Internat. de Med. Budapest. 1909, Fasc.1.p.1.

377. PONTARO. Il Policlinico. Jan.15, 1921.

378. RAVITSCH, M.L. The Thyroid as a factor in Urticaria Chronica.
Journ.Cutan.Dis. 1907. Vol.25. p.512.

379. " " Focal Infection in Relation to certain Dermatoses.
J.A.M.A. 1916. Vol.67.p.430.

380. SAMBERGER. Die entzündliche und urtikarielle Hautreaktion.
Dermat.Woch.1915, Vol.Vi.p.739.

381. " Weitere Erfahrungen über die lymphatische Hautreaktion.
Dermat.Woch.1917.Vol.65.p.623.

382. SCHAMBERG. Discussion on Protein sensitization.
Archiv.f.D. & S. 1920.Vol.2 P.572

383. SHATTUCK, H.F. Studies in Protein Intoxication . 1 Blood Coagulation.
Arch.Int.Med.1917. Vol.20.p.167

384. STEINER, K. Das Moskitofieber.
Wien.Klin.Woch.1905.Vol.19.pp.37 & 309.

385. STOKES J.H. A Clinical, Pathological & Experimental Study of the lesions produced by the bites of the Black Fly. (Simulium venustum).
Journ.Cutan.Dis. 1914. Vol.32. pp.751 & 830.

386. " " Discussion on Protein sensitization.
Arch.f.D. & S. 1920, Vol.2.p.575.

387. STRASSBERG. Intravenous Treatment of Pruriginous Skin Diseases.
Wien.Klin.Woch.Dec.8.1921.
Abstr.B.M.J.March 25, 1922.

388. TUROK. Die Angioneurotische und die hematogene Entzündungen
Rapports. Spec.Sect.viii.16th Cong.Inter. de Med.Budapest. 1909. Fac.1.p.B.

389. " & HARI. Experimentelle Untersuchungen ueber die Pathogenese der Urticaria.
Arch.f.D.& S. 1903. Vol.65.p.21.

390. THOMSON, J. Clinical Types of Convulsive Seizures in very Young Babies.
B.M.J. 1921, Oct. p.679.

391. TRACY, E.A. Normal Reaction of Skin to stroking.
Boston Med. & Surg. Journal. Clxv. No.6. Aug.10, 1916.
Abstr. in Journ.A.M.A., 1916, Vol. lxvii. Sept.2.p.771.

392. TURNBULL, F.M. An etiological factor in Angioneurotic oedema.
J.A.M.A., 1921. Vol.77.p.858.

393. WARD, E. The occurrence of Urticaria after Specific Fevers.
B.M.J. 1920, Aug.21.p.279.

394. WHITFIELD, A. Some points in the etiology of Skin Diseases.
Lancet 1921 II. p.122.

395. WEIDENFELD, S. Ueber Mechanische Reizbarkeit der Haut (Dermographismus) Zugleich eine Studie ueber Adrenalinwirkung.
Archiv.f.Dermat.1910.Bd.99.p.229.

396. WIDAL, ABRAMI, BRISSAUD, & JOLTRAIN, Reactions d'ordre anaphylactique dans l'urticaire. La crise hemoclastique initiale.
Soc. med.des Hop. 1914.Feb.13.

397. WILLIAMS, D. Immunity from Snake-bite.
Nature, 1897. Vol.55, p.415.

398. WINKLER, F. Studien ueber das Zustandekommen der Juckempfindung.
Archiv.f.D. & S. 1910. Vol.99. p.273.

399. WOLFF-EISNER. Ueber die urticaria vom Standpunkte der neueren Erfahrungen.
Dermatol. Zeitschr.1907 Vol.14. p.312. & p.164.

400. WRIGHT, A.E. & PARAMORE W.E., On certain points in connexion with the exaltation and reduction of blood coagulability by therapeutic measures.
Lancet 1905, Vol.II. p.1096.

401. AZUA, JUAN de, Local anaphylaxis?
Aetas dermo.sifiliograficas, 1912 Oct.-November.

402. BALDWIN H.T. Discussion on Asthma.
Boston Med.& Surg.Journ. 1918, Vol.179.p.298.

403. BAUGHER A.H. & VAUGHAN, R.T. Blood Findings after Salvarsan Injections.
Tr.Chicago Path.Soc.1911. Vol.8. p.176.

404. BEESON, B.B. Dermatitis exfoliativa, polyneuritis plus dermatitis exfoliativa following neo-arsphenamin.
Tr. Sect.Derm. & Syph. A.M.A. 1920, p.74.

405. BERMAN, L. The Nitritoid Crises after Asphenamin injections.
Archiv.Int.Med. 1918, Vol.22, p.217.

406. BOERNER, FRED. A Skin reaction to Quinine. J.A.M.A. March 24, 1917. p.907.

407. COLE, H.N. Drug exanthemata in relation to Anaphylaxis
Cleveland Med.Journ. 1911, Vol.10 p.442.

408. COOKE, R.A. Allergy in Drug Idiosyncrasy.
Journ.A.M.A., Vol.73, No.10. Septr.6, 1919, p.759.

409. DANYSZ, J. Les propriétés physicoclinique des produits du groupe des Arsénobenzénes, leurs transformations dans l'organisme.
Ann. de l'Inst.Pasteur. 1917. Vol.21.p.114.

410. DÜRR - Ueber Anaphylaxie.
Wiener Klin.Woch.1908.

411. EDLAVITCH, B.M. Cutaneous Reaction to Quinine in Quinine Idiosyncrasy.
Journ.Amer.Med.Assoc. 1919.lxxiii p. 1933. Abstr. in B.M.J. Feb.28, 1920.

412. EICKE, H. Ueber Nierensperre im verlauf der kombinierten Quëck-silber Salvarsanbehandlung.
Deutsch.med.Woch.1921. Vol.47. p.412.

413. EMERY & MORIN, Paris Med. Jan.24, 1920.
Abstr.in B.M.J. Apr.24,20.

414. FFRENCH, E.G. Exfoliative dermatitis occurring during Arsenical treatment.
Lancet 1920, p.1262.

415. FRIEDBERGER, E. & TETSUTA, J. Die Idueberempfindlichkeit des Meerschweinchens.
Zeit.f.Immünitatsforsch. u.Exp. Therap. 1912, Vol 12. p.241.

416. GLASER A. Ueber einen Fall von Vergiftung nach Formalintabletten.
Med.Klin. 1908. No.25.

417. GLOMBITZA. Spatexantheme nach Salvarsan-Natriuminjektionen; zugleich ein Beitrag zu visceralen Fruhlues.
Deutsch.Med.Woch.1917. Vol.43. p.1452.

418. HANZLEK, P.L. & KARSNER H.T., Anaphylactoid phenomena from the intravenous administration of various colloids, Arsenicals and other agents.
Journ.Pharm. & Exper.Therap. 1920, Vol.14. p.379

419. HARNACK & GRUNDLER (Quoted by Volk)

420. HEIDINGSFELD, M.L. Formalin urticaria.
Journ.Cutan.Dis. 1916. Vol.34. p.303.

421. HERAN & ST. GIRON'S. Un cas d'anaphylaxie à la quinine chez un paludéen. Intolerance absolue et urticarre, antianaphylaxie, par voie gastrique. Guérison.
Paris Méd. 1917.p.161.

422. " " Montpelier Med. 1917 Vol.39.pp.21 & 609

423. IWASCHENZOW, G. Ueber anaphylaktoide Erscheinungen bei wiederholten intravenösen Salvarsan-injektionen.
Münch.Med.Woch.1912, No.15.

424. KLAUSNER, E. Ein Fall von Idiosynkrasie gegen Iodoform und Iodkali.
Archiv.f.D.& S., Bd.98, 1909. p.323.

425. " " Arzneiexantheme als Ausdruck von Idiosynkrasie und Anaphylaxie.
Münch.Med.Woch. 1910, p.1451.

426. " " Arzneiexantheme und Überempfindlichkeit
Münch.Med.Woch.1910.No.38.p.1983.

427. KOLMER J.A. & LUCKE B. A study of the Histologic changes produced experimentally in rabbits by Arspenamin.
Arch.D.& S., 1921. Vol.3.p.483.

428. KYRLE, J. Zur Frage der Arzneiüberempfindlichkeit.
Archiv.f.D. & S., 1912.Vol.113. p.541.

429. LAROCHE, RICHET & ST. GIRONS. Alimentary Anaphylaxis. Bulletin Medical. 1920. Vol.34. p.625.
Abstr.J.A.M.A., 14/8/1920.p.508.

430. LATHAM, J.R. Exfoliative Dermatitis due to Arsphenamin.
J.A.M.A.1919. p.14.

431. LEONARD, L.G. Severe Dermatitis during treatment with Novarsenobillon.
B.M.J. 1919, Vol.2.p.773.

432. LIEBERTHAL, D. Toxic erythemas & their bullous manifestations.
Journ.Cutan.Dis. 1918.p.568.

433. McBRIDE W. L. & DENNIE, C.C. Treatment of Arsphenamin dermatitis and certain other metallic poisonings.
Arch.of D. & S. 1923.p.63.

434. McDONNELL, W.C. Death after Salvarsan.
B.M.J. 1912.May 18.

435. MACKEE, G.M. A comparison of the results obtained by the Intravenous Administration of Acid and Alkaline solutions of Salvarsan.
New York Med.Journ. 1911.Vol.94 p.825.

436. " " A Study of the Blood after intravenous Injections of Salvarsan.
Journ.Cutan.Dis. 1912 Vol.30. p.199.

437. MANDILOW, E.O. Ueber die Idiosynkrasie gegenueber Brom und Chinin als Erscheinungen der Anaphylaxie.
Charkowskij jMedicinskij Journal 1911 Bd.12.No.7.
Abstr.Wien.Klin.Woch.1912.No.3.

438. MARCHALKO, Th. & VESZPREMI. Histologische und experimentelle Untersuchungem ueber das Salvarsan tod.
Deutsch.Med.Woch.1912. No.26.

439. MILIAN, G. Les Intolerants du 606.
Bull.Soc.franc.de dermat. et de syph. 1912. Vol.23.p.520.

440. MILIAN G. L'erytheme arsenical oedemateux desquamatif. Bull. et mem. Soc.med.d.Hôp.de Paris, 1919. Vol.43.p.1055.

441. MOLESWORTH, E. H. A case of Nodose Bromide eruption in a breast-fed infant. B.J.D. 1917 Vol.29.p.30.

442. MOOK, Wm.H. Skin Reactions to Apothesin and Quinin in susceptible persons. Archiv.of Derm. & Syph. Vol.38, No.6. June 1920, p.651.

443. MOORE, J.E., & FOLEY, F.E.B. Serious reactions from the Salvarsan and Diarsenol brands of Arsphenamin. Archiv. of D.& S. 1920. Vol.1. p.25.

444. MOORE, J.E., & KEIDEL, A. Dermatitis and allied reactions following the Arsenical treatment of Syphilis. Arch.Int.Med.1921.Vol.27.p.716.

445. MORRIS, Sir MALCOLM, Diseases of the Skin, 6th Edit.

446. MULLER, P.T., Ueber den Bakteriengehalt des in Apotheken erhältlichen destillierten Wässers. München.Med.Woch.1911. Vol.58. p.2739.

447. NÄGELI, O. Neosalvarsan exanthem cured with epinephrin. Correspondenz-Blatt für Schweizer Aerzte. 1917. Vol.47, p.1291. Abstr. in J.A.M.A., 1917 Vol.ii. p.1743.

448. OBERMAYER & PICK. Ueber die chemischen Grundlagen der Arteigenschaften der Eiweisskörper Wiener.Klin.Woch.1906. Vol.19.p.327

449. O'MALLEY, J.J. & RICHEY D.G., Cutaneous reaction and desentisization in quinine idiosyncrasy. Archivof.Int.Med.1919. Oct. p.378.

450 OPPENHEIM, M. Ein durch Eukalyptusbonbons hervorgerufenes exanthem. Dermat.Woch.1912.Bd.54.p.224.

451. PELLIZARI, C. Nuovo contributo allo studio delle eruppzioni iodiche . Lo sperimentale 1884.p.233.

452. PIGNET, G. L'urticaire anaphylactique.
Annales de D.& S. 1921.No.4.
p.184.

453. PINKUS F. Ueber die hyperaemische Hautreaktion nach Salvarsan namentlich Frühreaktionen
Derm.Zeitsch.1911.Vol.18. p.672

454. PISTORIUS, H. Beiträge zur der acuten Arsenvergiftung
Archiv.f.Exper.Path.u.Pharmakol. 1883. Vol.16.p.188

455. POMARET. The Nitritoid Crisis.
La Medécine Nov.1921.
Abstr.B.M.J. Jan.7, 1922.

456. PUSEY, W.A. Universal exfoliative Dermatitis from Sodium cacodylate.
Archiv.of D.& S., 1920, Vol.1
p.57.

457. RAVAUT P. & WEISENBACH, R.J. Symptoms of intolerance resembling anaphylaxis after Salvarsan injections.
Gaz. des Hôp. 1911.No.18.

458. RAVITCH. A Case of veronal eruption.
J.A.M.A., 1912. Vol.58, p.2026.

459. RICKER G. & KNAPE W. Mikroskopische Beobachtungen am lebenden Tier ueber wirkung des Salsalvarsans & neosalvarsans auf die Blutstromung.
Med. Klin. 1912 p.1275.

460. ROTERS. Aerztl.Zentralanzeiger.1907 No.47

461. SCHAMBERG, J.F., KOLMER, J.A., RAIZISS, G.W. & WEISS, C.W. Laboratory and Clinical Studies bearing on the causes of the reactions following intravenous injections of Arsphenamin & neo-Arsphenamin.
Archiv.of D. & S., 1920. Vol.38
p.235.

462. " " Clinical commentary on studies of histological changes in organs induced by arsphenamin, by Neo-arsphenamin and by mercury.
Archiv.of D. & S. 1921. Vol.3.
p.571.

463. SMITH, M. J. Relation of certain drugs to anaphylactic reaction and bearing thereof on mechanism of anaphylactic shock.
J. Immun. 1920. Vol. 5. p. 239.

464. STOKES, J.H., Atropin and induced antianaphylaxis as a protection against acute Arsphenamin reactions.
Journ.Amer.Med.Assoc. 1919. Vol. 72, p. 241.

465. " " & CATHCART, E.P., Contributory Factors in Postarsphenamin Dermatitis with special reference to the influence of focal and intercurrent infection.
Archiv. of D.&.S., 1923. p. 14.

466. " " RUEDEMANN, R.J., & LEMON, W.S., Epidemic infectious jaundice and its relations to the therapy of syphilis.
Arch.Int.Med. 1920. Vol. 26. p. 521.

467. SWIFT, H.F. Anaphylaxis to Salvarsan.
J.A.M.A. 1912. Vol.59. p.1236.

468. TRIMBLE, W.B., Drug Eruptions.
J.A.M.A., 1912. Vol.58. p. 2026.

469. TZANK. Passive Anaphylaxis in the guinea-pig.
Compt. rendu.Soc.Biol. 1921. Nov. 12. B.M.J. 1921 Dec. 24.

470. VOLK, R. Das Ueberempfindlichkeitsproblem in der Dermatologie.
Archiv. f.Dermatol. 1911. Bd. 109. p. 163.

471. VÜRNER, H. Durch Eukalyptusöl horvorgorufenes Exanthem das später periodisch rezidivierend wird.
Dermat.Zeitsch. 1907.Bd.14. p.678

472. WECHSELMANN. Ueber die anaphylaktoidin Erscheinungen bei wiederholten intravenösen Salvarsaninjektionen.
Deutsch.Med.Woch. 1912. No.25.
Arch.f.der.u.Syph.1912.Bd. 111.

473. " Ueberempfindlichkeit bei intravenöser Salvarsaninjektion.
Heft. 1.

474. " W. Ueber die Pathogenese der Salvarsantodesfälle. Vienna. 1913. p. 88.

475. WIDAL, F., ABRAMI, P., & JANCOVESCO, N. L'épreuve de l'hémoclasie digestive dans l'étude de l'insuffisance hépatique.
Presse méd.1920. Vol.28.p.1920.

476. " ", & VALLERY RADOT, P. Anaphylaxis to acetylsalicylic acid.
Presse med.1920 Vol.28.p.93.
Abstr.J.A.M.A., 1920. Vol.74.p. 1055.

477. " " " " " Antipyrin and anaphylaxis.
J.A.M.A., 1920 Vol.74.p.538

478. WILE, U.J. & WRIGHT, C.S., & SMITH, N.R. A preliminary study of the experimental aspects of Iodid and Bromid exanthems.
Arch.of D. & S. 1922.p.529

479. WOLFSOHN, G. Über thyreotoxische Symptome nach Iodmedikation.
Deutsch.med.Woch.1911, No.5.

480. WOOLEY, P. G. Veronal Dermatitis.
J.A.M.A. 1907 p.2153.

481. ZEISLER, JOSEPH. Some uncommon and often unrecognised forms of Toxic dermatitis.
J.A.M.A., June 20 1912. p.2024.

482. ZIELER, K. Ist die Idiosynkrasie gegen Arzneistoffe als echte Anaphylaxie aufzufassen?
München.med.Woch.1912. Vol.59. p.401.

483. " " Zur Frage der Idiosynkrasie gegenüber Salvarsan, insbesondere sind Hautimpfungen mit Salvarsanlösung zur Feststellung einer vorhandenen Idiosynkrasie brauchbar?
München.med.Woch.1912.No.30.

ERYTHEMATA.

484. ANTHONY, H.G., The Toxic origin of Erythema multiforme
Journ.Cut.Dis.1912.Vol.30.p.152

485. BARTHELEMY. Annales de D. & S. 1888.

486. BASS, M.H., Erythema multiforme as a rheumatic manifestation in Children.
New York.med.Journ.1919.p.427.

487. CHRISTIAN, H.A., Visceral Disturbances in patients with cutaneous lesions of the erythema group.
J.A.M.A., 1917. Vol:69.p.325.

488. CIUFFINI, P. Polymorph erythema and Tuberculosis.
La.Clin.med.Ital.1912 No.12.

489. CORLETT, W.T. Erythema exudativum multiforme, its present significance - with a report of a case of erythema circinatum bullosum et haemorrhagicum.
Journ.Cut.Dis. 1908, xxvi. p.7

490. DUERING, L. von. Beitrag zur Lehre von den polymorphen Erythemen.
Archiv.f.D.& S., 1896.Vol.35.p. 211.

491. ENGMAN. Discussion on Toxic Dermatoses.
Journ.Cutan.Dis.1912.Vol.30.p.166

492. FOX. H. Dermatitis Herpetiformis following vaccination.

493. GALLOWAY, Sir.J. Cutaneous affections in Rheumatic Conditions.
Practitioner, 1912. Vol.88.p.67.

494. " " Erythemata as indicators of Disease.
B.J.D. 1903.Vol.15.p.235.

495. " " Skin Diseases in relation to Internal Disorder.
Lancet 1921.Feb.p.364.

496. GILCHRIST. Discussion on Toxic Dermatoses.
Journ.cutan.Dis. 1912. Vol.30. p.165.

497. GIRODE, J. Phlébite dans l'erythème polymorphe.
Annales de D.& S., 1888.p.791.

498. HAZEN, H.H. Severe erythema multiforme with Anaphylaxis due to oyster protein.
J.A.M.A., 1914, Vol.62.p.695.

499. HERXHEIMER, K. Eine epidemie von Erythema exudativum.
Arch.f.D. & S., 1894. Vol.29.p. 118.

500. LESIEUR & MARCHAND. Erythema multiforme.
Prov.med.1912.No.3.

501. LIEBERTHAL, DAVID, Toxic erythemas and their bullous manifestations.
Journ.Cutan.D.1918.p.568.

502. LYONNET & MARTIN. Erytheme polymorphe et Tuberculose
Lyon medicale. Bd.118.No.10.

503. MARANÓN, G. A constant sign of Hyperthyroidism.
Rev.Espan.de Med.y cir.Nov.1919.
Abstr.B.M.J. Jan.31.1920.p.17.

504. MERKLEN & ACHPISE. Erythème maculo-papuleux consécutif à la vaccination antityphique.
Annales de D. & S. 1919.p.318

505. OSLER, Wm. On the visceral complications of erythema exudativum multiforme.
A.J.M.Sc. 1893.Vol.110 p.629.
A.J.M.Sc.Dec.1895

506. " " Visceral lesions of the erythema group.
Brit.Journ.Derm.1900 Vol.12.p.227

507. " " On the Visceral Manifestations of the Erythema group of Skin Diseases.
A.J.M.Sc.1904.Vol.127.p.1.

508 " " On the Surgical importance of the visceral crises in the erythema group of Skin Diseases.
A.J.Med.Sc.1904.Vol.127 p.751.

509. WALKER, N. Erythema multiforme & Vaccination.
Brit.Med.Journ.1901 .p.1201.

ERYTHEMA NODOSUM.

510. ALAMARTINE H. Erythema nodosum of tuberculous origin. Contribution to the study of inflammatory Skin Tuberculosis.
Gaz.des Hop. 1912 No.69

511. ANDERSON, K. & COOPER, N.C., Erythema nodosum.
B.M.J. 1921 ii.p.863.

512. BEURMANN & CLAUDE. De l'érythème noueux d'origine syphylitique.
Annales de D. & S., 1896.p.485.

513. BRIAN, O. Untersuchungen ueber die aetiologie des Erythema Nodosum.
Deutsch.Arch.f.Klin.Med.1911. Heft 3 & 4.

514. BRONSON, E. Erythema Nodosum associated with Tuberculosis.
Brit Jour.Child.Dis. 1918, Vol.5 p.191.

515. CALDEROLA, P. A case of multiple cutaneous tuberculous gummata simulating at first the eruption of erythema nodosum.
Giorn.Ital.d.mal.ven.d.della pelle. 1922. Fasc.II.p.654

516. CHAUFFARD & LE CONTE, Erythème noueux et syphilis
Annales de Méd.1915.- p.563.

517. " & TROISIER, Erythème noueux expérimental par injection intradermique de tuberculine.
Soc.méd.des hôp. de Paris 1909. Jan.15.

518. COURMANT, SAVY & CHARLET, Six cases of erythema nodosum, - Discussion as to its tuberculous nature.
Lyon med.Bnd. 117, No.53.

519. CRAIG, J. C. Erythema nodosum.
B.M.J. 1911 I. p.1175

520. EMRYS-ROBERTS, E. A case of 'Erythema Nodosum' with note of Blood cultures.
Journ. of Path,& Bact.1921. Vol.24.p.477.

521. ERNBERG, H. Erythema Nodosum. Its nature and significance.
Jahrb.f.Kinderheilk. 1921.Bd.95.
Heft.1 & 2.

522. FORDYCE, J.A., Notes on Drug Eruptions.
Journ.cut.Dis.1895 p.496.

523. GUEISSAZ? Erythema Nodosum.
Rev,Med.Suisse Rom.Oct.& Nov.1921
Abstr.Brit.Med.Journ.1922 Jan.21.

524. HALLAM, H. Erythema Nodosum.
B.M.J. Jan.26.1921.

525. HOFFMANN, E., Ueber Aetiologie und Pathogenese des erythema nodosum.
Deutsch.Med.Woch.1904. Vol.30 p. 1877

526. HALLOPEAU & GRANDCHAMP, Diagnostic des lépprides erythemateuses et de l'erythème noueux.
Annales de D. & S., 1905 p.444.

527. " & TEISSEIRE. Cas d'iodisme avec nodules intradermiques localisés aux derniers plis articulaires des deux annulaires.
Annales de D. & S., 1905.p.257.

528. HAUG, K. Erythema nodosum & Tuberculosis. Tidsskr.f.d. Norske Laegsforening Jan.1.1920.
Abstr.B.M.J. March 17.1920.

529. HOFFMANN Venenerkrankungen im Verlauf der Sekundärperiode der Syphilis.
Arch.f.D. & S. Vol.73.p.39.

530. HOFFMANN E. Beitrag zur Frage des akuten nodosen Syphilids. (Erythema nodosum syphiliticum)
Arch.f. D. & S., 1912 Bd.113.
p.437.

531. HOLLAND W. Erythema Nodosum & Tuberculosis. Norsk. Mag. f.Laegendenskaben. 1922.p.526.
Abstr. in B.M.J. Dec.9th 1922.

532. HOYER, W. The Blood Count in Erythema nodosum.
Acta Medica Scandinavica Feb.22. 1923.
Abstr. B.M.J. March 24 1923.

533. JOINT, E. P. Erythema nodosum following measles.
B.M.J. 1911. Vol.I.p.867.

534. LANDOUZY, LAEDERICK & RICHET Erythème noueux d'origine bacillo-tuberculeuse.
Presse Med.1913.p.1045.

535. LEVINSOHN, S.A., Case of erythema Nodosum treated by streptococcus vaccine.
Med.Record 1920, Vol.98.p.859

536. MARFAN - Erythema Nodosum.
Medical Press, 1918, July 3rd p.6
Abstr.Urol.& Cutan.Review 1919 p.11

537. " Erythème noueux et tuberculose.
La Presse med.1909.p.457.

538. MEARA, F.S. & GOODRIDGE, M., The confusion between Erythema nodosum and Tuberculosis.
Amer.Journ.Med.Sci. 1912 March.

539. NEAVE, T. The aetiology of Erythema Nodosum.
B.M.J., 1912. Apr.20.

540. NETTER, A. Hypersensibility to Tuberculin in Erythema Nodosum.
Bull. et Mem. de la Soc,Med.des Hop. 1917, p 280.

541. OCHME, E. Nodosum followed by Tuberculous Meningitis
Arch.der Heilkunde.1877.p.426

542. PERIGAL, A. F. Erythema nodosum following measles.
B.M.J. 1911.ii.p.163.

543. PIC, A. Erythema nodosum & tuberculosis.
Lyon.med.Bd.117.No.53.

544. POLLAK, R. Erythema Nodosum und Tuberculose.
Wien.Klin.Woch.1912.No.32

545. POLLOCK, G., Erythema nodosum.
B.M.J. 1911 ii. p.214

546. VAN PRAAGH, H.J. Erythema Nodosum.
B.M.J. Jan.26.1921.

547. ROSENOW, E.C., Aetiology of Erythema Nodosum.
Journ.Cutan.Dis. 1915. Vol.33. p.408

548. SCHIDACHI, T. Ueber nodose Iodexantheme.
Med.Klin.1907 p.169.

549. STEFANO, E. Nodosum and Tuberculosis.
La Pediatria Nov.1919.
Abstr.B.M.J. Jan.17,20.

550. STOKES, J.H. Fatal Case of Erythema Nodosum and Tuberculosis.
Arch.of D.& S., 921.p.29.

551. STUMPKE, G. Ueber die Beziehungen zwischen Erythema Nodosum und Lues.
Archiv.f.D. & S. 1917. Bd.124. p.671.

552. SYMES, J. O. Erythema Nodosum.
B.M.J. 1921.ii.p.741.

553. THIBIERGE, G. Eruptions médicamentuuses.
La Pratique Dermatologique. Vol.2 p.487

554. THIBIERGE, J. & GASTINEL, Reproduction experimentelle de certaines dermatoses, de la série des erythèmes, par l'injection intradermique de tuberculine et de divers sérum.
Soc.Méd.des Hôp.de Paris. 1909 Apr.23.

556. VETLESEN, H.J. Erythema Nodosum and Tuberculosis.
Tubercle.1922.p.433.

557. WARD, E. Erythema Nodosum & Tuberculosis
B.M.J. 1919, Dec.20.p.811.

558. WIBORG, A. The Etiology of Erythema Nodosum.
Norsk Mag.f.Laegevidenskaben. Feb.1923.
Abstr.B.M.J. March 31 1923.

559. WILLIAMS, D. O. Erythema nodosum.
B.M.J. 1911 Septr.2.

PURPURA.

560. BEDSON, S.P. Blood platelet anti-serum, its specificity and role in the experimental production of Purpura.
Journ.Path.& Bact. 1921, Vol.24. p.469.

561. " " Do. " " "
Journ.Path.& Bact. 1922. Vol.25 p.94.

562. DIXON, M. Purpura treated by Injection of Human Blood
B.M.J. 1923.p.16

563. GOTTLIEB, Experimental purpura.
Journ.Immunol. 1919, Vol.4.p.309.

564. HANNS & WEISS. Anaphylactic Purpura.
Revue de médicine. Vol.40.p.104.
Abstr.B.M.J. 1923.Apr.28.

565. HESS, A. F. A consideration of the reduction of blood platelets in Purpura.
Proc.Soc.Exper.Biol. & Med. 1916-17, Vol.14.p.96.

566. LEDINGHAM. The experimental production of Purpura in Animals.
Lancet 1914.I.p.1673.

567. " & BENSON. Experimental Purpura.
Lancet 1915.I.p.311.

568. LEE & ROBERTSON. The effect of antiplatelet serum on Blood platelets and the experimental production of Purpura Haemorrhagica.
Journ.Med.Res.1916.Vol.32.p.323

569. MUSSER & KRUMBHAAR. The resistance of erythrocytes in normal rabbits and guinea-pigs and the changes produced in experimental purpura.
J.A.M.A. 1916.Vol.67.p.1894.

570. NOBÉCOURT & TIXIER. The treatment of congenital Haemophilia and of Purpura with Peptone (Witte).
Gaz.des Hop. 1911.No.6.

571. WATABIKI. Studies on experimental purpura haemorrhagica produced by anti-bloodplatelet. serum. Kitasato Archiv. of experimental Med. 1917. Vol. I. p. 195.

572. WILSON, S.J. A case of Henoch's Purpura cured by treatment with human serum. Med. Record. 1912. Aug. 10.

DERMATITIS HERPETIFORMIS & PEMPHIGUS.

573. LE CALVÉ, J. Choc hémoclastique et oedème. Gaz. des Hôpitaux. 1921. No. 96. p. 1533.

574. HARTZELL, M.B. Dermatitis Herpetiformis Its Etiology & relationship to certain members of the bullous group of Diseases especially to erythema multiforme & pemphigus. Journ. Cutan. d. 1918. p. 487.

575. " " Dermatitis Herpetiformis J. Cutan. D. 1918 Vol. 36. p. 497.

576. LOW, R. CRANSTON Pemphigus Foliaceus. B. J. D. April & May 1909.

577. " " A further case of Pemphigus foliaceus with remarks. B. J. D. Jan. 1911

578. LOWENBERG. 84 Versammlung Deutscher Naturforscher und Aertze, Münster 1912.

579. TOMMASI, L. A contribution to the study of the etiology of Pemphigus. Giorn. Ital. d. mal. vené, della pelle. 1918 Fasc. 3.- p. 146.

TUBERCULOSIS.

580. ARLOING. Agglutination du bacille de la Tuberculose. Compt. rend. de l'acad. des Sciences 1898 Vol. 5. p. 16.

581. BANG. Das Geflügeltuberculin als diagnostisches Mittel bei der chronischen pseudotuberkulosen darmentzundung des Rindes (Johnes Disease. Centralbl. f. Bakt. 1909. I.O. Vol. 51. p. 450

582. BAUER, J. Die passive uebertragung der Tuberkulos-ueberempfindlichkeit.
Münch.med.Woch.1909 No.24.p.1218.

583. BESSAU, G. Ueber die biologischen Vorgänge bei der Tuberkulinbehandlung.
Münch.Med.Woch.1915. Vol.62.P.323

584. HAZEN, H.H. Multiple Disseminated Lupus vulgaris.
Journ.Cutan.Dis. 1919.Vol.37.p.89

585. HELMHOLTZ, H.F. Ueber passive Uebertragung der Tuberkulin-empfindlichkeit bei meers-schweinchen.
Zeitsch.fur Immunitätsforchung. 1909.Bd.III.p.371.

586. ICHOK. Diagnosis of Tuberculosis by the Reaction of Fixation.
Archiv.Med.Belges.Oct.1921
Abstr.B.M.J., Feb.18.'22.

587. KOLMER, J. A. The Tuberculin Reaction. Therapeutic Gazette. 1922.p.381.

588. LOW, R. CRANSTON. Tuberculin as an aid to diagnosis and treatment.
Scottish Med.& Surg.Journ. May 1905

589. " " Emploi de la Tuberculine comme moyen de diagnostic and de traitement.
Revue pratique des malades cutanées Avril 1906.

590. PETRUSCHKY. Die spezifische Diagnostik und Therapie der Tuberkulose.
Ergebn.d.Inn.Med.und kinderheilk. 1912 Vol.9 p.537.

591. SACHS, O. Ueber das Verhalten der Wassermanschen Reaktion bei Tuberkuliden.
Arch.f.D.& S. 1916. Bd.123.p.838

592. SCHAUMANN, J. Séro-réaction de Wassermann positive dans deux cas de tuberculides.
Annales de D.& S. 1918-19.Tome 7 p.8.

593. SCHÖNFIELD, W. The question of the occurrence of positive Wassermann Reactions in Tuberculosis of the skin and the Tuberculides.
Arch.f.D. & S., 1919. Vol.126, p.702.

594. SLATINEANU & DANIELOPOLU Sensibilisation à l'infection tuberculeuse par une injection prealable de tuberculine.
Soc.de Biol.de Paris.1908 I.p.418.

595. " " " & CINCA. Sensibilisation de l'organisme humain normal aux injections répetées de tuberculine.
Soc.de Biol.de Paris.1909.1.p.652.

596. SMITH, A. NIMMO, The complement-fixation Reaction in Tuberculosis. Critical Review.
Edin.Med.Journ. 1922. Vol.28 p.174

597. STOLL, H. F., & NEUMAN, L. A complement-fixation test in the diagnosis of tuberculosis.
J.A.M.A. 1919. Vol.72.p.1043.

SKIN REACTIONS IN TUBERCULOSIS.

598. BASS, M.H. Cutaneous and Intracutaneous Tuberculin Tests in Infants and Children.
Am.J.Dis.Child. 1918. Vol.15.p.313

599. BESSAU, Die Tuberkulinüberempfindlichkeit und die durch Tuberkulin-darreichung zu erzielende Tuberkulinempfindlichkeit.
Jahrb.f.Kinderheilk.1915, Bd.81.

600. " & SCHWANKE. Wiederholte Tuberkulinreaktionen
Jahrb.f.Kinderheilk. 1914.Bd.79.
Heft.2.

601. BONDY, O. Ueber Kutanreaktionen bei Neugeborenen.
Wien.Klin.Woch.1908 No.49.p.1704.

602. BOSCH, E. Die diagnostische Verwertbarkeit der Wildbolzschen Eigenharnreaktion.
München.Med.Woch.1921.p.733.

603. BOUVEYRON. Changes in the Tuberculin Reaction due to the addition of Adrenalin and quinine.
Compt.rend.Soc.Biol.Nov.12.1921.
Abstr.B.M.J.Dec.3.1921.

604. BROWNING, C.C. The value of the Tuberculin Cutaneous Test.
Northwest Med.1919.Vol.18.p.230

605. BUSACCA. An intracutaneous reaction for Tuberculosis of the Skin.
Wien.Klin.Woch.Nov.24.1921.
Abstr.in B.M.J. Feb.25.1922.

606. CATTERMOLE, G.H. Tuberculin Tests in Children in Colorado.
J.A.M.A. 1915. Vol.65.p.782.

607. CEPULIC, N. & PINNER, M. Leitlinien zur Beurteilung der Quaddelprobe.
Med.Klinik 1921.Vol.17 p.162.

608. COLLIVER, J.A., Cutaneous regional variation in the Von Pirquet Reaction.
Archiv.Pediat.1915. Vol.32.p.92

609. DEBRÉ & BONNET. The Intradermal Reaction in experimental Tuberculosis.
Compt.rendu Soc.de Biolog. March 4, 1922.
Abstr. in B.M.J. May 20, 1922.

610. EMMERICH. Ueber die Klinische Bedeutung der Kutanen und perkutanen Tuberkulinreaktion (nach V.Pirquet u.nach Moro) beim Erwachsenen.
Munch.Med.Woch.1908, No.20

611. EPSTEIN. Die lokale Tuberkulin Reaktion.
Prager med.Wochenschrift.1891. No.1 & 2.

612. ESCHERICH. Die Resultate der Koch'schen Injectionen bei Scrofulose und Tuberculose des Kindesalters.
Jahr.f.Kinderheilkunde. 1892. Bd.33, p.369.

613. FISHBERG, M. The cutaneous Tuberculin Test in Children of non-tuberculous parentage.
Arch.Pediat.1915. Vol.32 p.20.

614. FRAZER, T. The significance of the von Pirquet Tuberculin Test.
Med.Rec. 1915.Vol.87.p.57.

615. HAMBURGER, F. Ueber den Wert der Stichreaktion nach Tuberkulininjektion.
Wiener Klin.Woch.1908.p.381.

616. " Ueber Tuberkulinimmunität.
Munch.Med.Woch.1908 No.42.

617. " & MONTI. Ueber Tuberkulinimmunität.
Brauers Beitr.1910.Bd.16.

618. IMHOF, O. Autourine test for Tuberculosis.
Schweiz Med.Woch.1920. Vol.50. p.1033.

619. KEIFETZ, M.N. Cutaneous Tuberculin Tests in School Children.
Abstr,J.A.M.A. 1915.Vol.65.p.1412.

620. LEONI, R. A New Test for Tuberculosis.
Ref.Med.Aug.20.1921 &
Folia Medica No.9.1921.
Abstr. in B.M.J. Nov.5.1921.

621. LEWANDOWSKY, F. Ueber die Hautimmunität bei Tuberkulose.
Dermat.Woch. 1913.Bd.57.p.1443.

622. LOW, R. CRANSTON, The cutaneous Tuberculin reaction in skin Diseases.
Edin.Med.Journ. Aug.1909.

623. LUTZ. Ueber das Verhalten der menschlichen Haut gegen verschiedene bakterielle Giftstoffe.
Wien.Klin.Woch.1908 p.379.

624. McMICHAEL, O.W., Tuberculosis in children.
Illinois Med.Journ.1915 Vol. 28 p.337.

625. McNEIL, C. The cutaneous Tuberculin Test (Pirquet) with reference to its failure in advanced tuberculous disease in childhood.
B.M.J. 1923 Vol.1.p.674.

626. MANNING, J.B., & KNOTT, H.J., A clinical study of 228 Children in relation to Tuberculous exposure controlled by the v.Pirquet reaction.
Am.Journ.Dis.Child, 1915 Vol.10. p.354.

627. MAYR, J.K. & HOFSTADT, H. Die "Eigenharnreaktion" nach Wildbolz bei der Tuberkulose der Haut.
Dermat.Woch.1923 Bd.76.p.165.

628. MONDOLFO & COSCERA. The Regional cuti-reaction.
Sez.Prat.Nov.21.1921. II Policlinico.
Abstr.B.M.J. Feb.4.1922.

629. MONTI, Diagnostischer Wert der intrakutanen Tuberkulinreaktion.
Wien.Med.Woch.1912.No.7.

630. MORO, E. Klinische Uberempfindlichkeit.
Munch.Med.Woch.1908.Vol.4.p.2025.

631. RAISCHEL, F. Vergleichende Bewertung der Tuberkulinreaktion im Kindersalter.
Munch,Med.Woch.1908.Vol.1 pp. 326 & 397.

632. RAMSAY, W.R., The results of von Pirquet Reaction.
Am. Journ.Dis.Child. 1915. Vol.10, p.201.

633. RIEHL, Tuberkulose und Tuberkulide; Lepra.
Verhandlungen der deutschen dermatologischen Gesellschaft XI. Kongress Wien.18-20 Sept.13 A.f.D.Mai 14 p.145

634. ROGERS, O.F. A study of Children with positive skin Tuberculin Reactions.
Boston Med.& Surg.Journ.1915. Vol.172.p.161

635. RÜMER, P.H., & JOSEPH, K. Zur Verwertung der intrakutanen Reaktion auf Tuberkulin.
Beitr.z.Klin.d.Tuberk.1909. Vol. 14 p.1.

636. SALVETTI, G. The significance of the cutaneous Reaction in the Diagnosis of Tuberculosis in Children.
Abstr.Jahr.f.Kinderh.1915.Vol.32 p.173.

637. SCHICK, B. Die diagnostische Tuberkulinreaktion im Kindesalter.
Jahr.f.Kinderkeilk.1905.Bd.61. p.811

638. SOLIS-COHEN, M. Hypersensitiveness to Tuberculin as determined by intracutaneous injection of different dosages.
Journ.Infect.Dis.1917.Vol.20. p.233.

639. TRIMBLE, W.B. The Diagnostic value of the inunction Tuberculin Reaction in cutaneous Tuberculosis.
New York Med.Journ.1909 May 22.

640. VERDER, B.S. & JOHNSTON M.R., The frequency of Infection with the Tubercle Bacilli in Childhood.
A.J.Dis.Child.1915.Vol.9.p478

641. WOLFF-EISNER. Die Ophthalme-und Cutidiagnose der Tuberculose.
Wurzburg 1908.

TUBERCULIDES.

642. AUDRY. Ch. Etude de la lésion de l'erythème induré (de Bazin)
Annales de D.& S. 1898, p.209.

643. BARBAGLIA, V. Lichen scrofulosorum.
Giorn.Ital.d.mal.ven.e.della pelle 1919.fasc.vi. p.473.

644. BRUHNS, C. & ALEXANDER, A. Zur Kenntnis des Lupus pernio und des Boeckschen Sarkoides.
Arch.f.D.& S., 1920 Bd.127 p.833.

645. CALLOMON, F. Hauttuberkulose und Tuberkulide bei Heeresangehörigen.
Derm.Zeitsch.1917, Vol.24.p.718.

646. CAPELLI, J. Contribution to the study of diffuse cutaneous haematogenous tuberculosis, with clinically atypical eruption.
Ann.de D. & S., 1919.Tome.7.p. 257.

647. CIVATTE, A. & VIGNE, P. On the treatment of the Sarcoid Boeck-Darier.
Annales de D.& S.1920 No.6.p.254.

648. FABRY, J. Zur Klinik und Aetiologie des Angiokeratoma.
Arch.f.D.& S., 1916.Bd.123, P. 294.

649. FANO Sarkome und Sarkoide Geschwülste.
Arch.f.D.& S., 1907. Bd.83, pp. 33, 225 & 427.

650. FOX, COLCOTT. Report on the Tuberculides.
B.J.D. 1900. p.383.

651. FRIEBOES, W. Multiples idiopathisches Lymphosarkoma cutis Sarcomatosis cutis Spiegler und sarkoide Tumoren.
Dermat.Zeitsch.1917.Vol.24.p.257.

652. GOUGEROT & LAROCHE Etiology of Skin tuberculides - experimental tuberculides.
Gaz.des Hop. 1912.No.11 & 14.

653. GRANCHER & LEDOUX-LEBARD. Tuberculose aviare et humaine.
Arch.méd.expér.1892.p.1.

654. HABERLANN. Fall von Boeckschem Sarkoid mit nachgewiesener Tuberkulose.
Deutsche med.Woch.1916.No.50. p.1564.

655. HALKIN. Sarcoide de la peau.
Archiv.f.D.& S., 1907.Bd.84.p.227

656. HASLUND, P. Haematogenes tuberkulöses exanthem und dessen Abhangigkeit von elektrischen Bogenlichtbädern.
Arch.f.D.& S. 1916.Bd.123.p.349.

657. HEMPELMANN T.C., The frequency of tuberculides in infancy and childhood and their relation to prognosis.
Arch.of Pediatrics.1917.p.362.

658. HUDELO, CIVATTE & RABUT Granuloma annulare treated by Tuberculin.
Bull.Soc.Franc. de D.&.S. 1920 xxvii p. 12.

659. JADASSOHN "Tuberkulide"
Dermat.Woch.1913.Bd.57.p.1407.

660. " Die Toxicodermien.
Deutsch.Klinik.Bd.10.p.117

661. KERL, W. Zur Frage der Spezifität der Wassermann-Reaktion insbesonders ueber den Ausfall bei Tuberkulose and Tuberkuliden.
Arch.f.D.& S. 1917.Bd.124,p.734.

662. " " & KOCH, H., Ueber Ursachen des Ausbleibens von Herdreaktionen.
Arch.f.D. & S., 1917.Bd.124.p.757

663. KUTZNITZKY & BITTORF. Boecksches Sarkoid mit Beteiligung innerer Organe.
Munch.Med.Woch.1915.p.1349

664. KYRLE J. & McDONAGH, J.E.R. Lichen Nitidus.
B.J.D.& S. 1909 Vol.21.p.339.

665. LEREDDE, M. Tuberculides nodulaires des membres inférieurs.(Erythème induré de Bazin).
Annales de D.& S. 1898.p.893.

666. LEWANDOWSKY, F. Tuberkulose-Immunität und Tuberkulide (experimentelle studien)
Arch.f.D.& S. 1916. Vol.123.p.1.

" " Zur Kenntnis des Lichen Nitidus.Originale.Arch.f.D.& S. 1916.Bd.123.p.494

667. LIEBREICH, E. Zur Kenntnis des Granuloma Annulare und seiner eventuellen Beziehungen zum Lichen ruber planus.
Arch.f.D. & S. 1916 Bd.123.p.180.

668. LITTLE, GRAHAM. A case of Lichen scrofulosorum.
Proc.Roy.Soc.Med.Derm.Section) May 1916.

669. LITTLE, GRAHAM. Granuloma annulare.
B.J.D. 1908.p.83.

670. LOW, R. CRANSTON, On the microscopic changes in Tuberculin exanthemata.
Scott.Med.& Surg.Journ.Sept.1905.

671. " " Tuberculides and their relation to Tuberculosis of the skin and other organs.
Edin.Med.Journ.1920.January.

672. MANTEGAZZA U. Contribution à l'etude de l'erythème induré de Bazin.
Annales de D.& S. 1901.p.497

673. MARTENSTEIN, H. Wirkung des Serums von Sarkoid-Boeck und Lupus-pernio Kranken auf Tuberkulin.
Archiv.f.D.& S. 1921 Bd.136.p.317.

674. MILIAN, G. Eczema as Tuberculid.
Presse med.1920 Vol.10.p.206.

675. PAUTRIER A propos des nouvelles conceptions touchant la pathogenie des sarcoides.
B.S.f.D. 1914.p.253.

676 PEYRI, J. & SOTERAS, J. Experimental production of Toxi-tuberculides of the skin in susceptible and non-susceptible animals.
Revista espanola de Dermatologia y sifilografia. 1912.Vol.14.No.160.
Abstr.in Dermatol.Woch.1912.Bd.54 p.678.

677. PINKUS, F. Lichen Nitidus.
Archiv.f.D.& S., 1907 Vol.85.p.11.

678. POLLAND, R. Relations of certain forms of exfoliative erythrodermias to Tuberculosis.
Dermat.Zeit.Vol.21 p.665.

679. " Ueber sarkomartige Hauttumoren.
Arch.f.D.& S. 1910 Bd.104.p.69.

680. POLLITZER, S. Tuberculosis of the Skin.
Journ.cutan.Dis.1918.
Abstr.Urol.& Cutan.Review 1919.p. 171.

681. PRUDDEN & HODENPYL. Studies on the action of dead bacteria in the living body.
New York Med.Journ.1891.June 20.

682. RASCH. Zur Kenntnis der Sarkoiden Hauttumoren.
Arch.f.D.& S. 1907 Bd.87.p.163.

683. REJSEK, B. Etiology of Sarcoids of Boeck-Darier.
Ceska Dermatologie 1920 No.5.
Abstr.B.J.D.& S. 1921 p.350.

684. SCHAUMANN, J. Recherches sur le lupus pernio et ses relations avec les sarcoides cutanées et sous-cutanées.
Nord.Med.Arkiv.1917. Afd.II.Häft 6, No.17.

685. SCHEER, M. & LANE, J.E., Dermatoses possibly related to Tuberculosis.
Journ.Cutan.Dis.1918?
Abstr.Urolod.& Cutan.Review 1919 p.170.

686. SKINNER, E.F. Notes on a case of multiple pigmented idiopathic Sarcoma (Kaposi)
B.J.D.& S., 1919 Vol.31.p.28.

687. SMITH, J.F. A case of papulo-necrotic Tuberculide.
B.J.D.& S., 1919.p.97

688. STOKES, J.H. Cutaneous Aspects of Tuberculosis.
Am.Journ.Med.Sci.1919.
Abstr.Urolog.& Cutan.Review 1919 p.168. - also p.232.

689. STRAUSS & GAMALEIA. Contributions à l'étude du poison tuberculeux.
Arch.med.exper.1891.

690. SCHWEITZER, S.E., & MICHELSON, H.E., Sarcoid of Boeck and Erythema Induratum of Bazin.
Journ.Cutan.Dis. 1919 Vol.37.p. 98.

691. THIBIERGE G. & RAVAUT, P. Etude sur les lésions et la nature de l'erytheme induré.
Annales de D. & S. 1899. p.513.

692. VISSMANN, - Wirkung der Tuberkelbacillen auf den Organismus.
Virchow's Arch.Bd.129, p.163

693. WEINBERG. Boecksches miliarlupoid und Tuberkulose.
Munch.Med.Woch.1916.p.892.

694. WHITFIELD, A. On the Nature of the Disease known as Erythema Induratum scrofulosorum.
Amer.Journ.Med.Sc. 1901. Vol.cxxii p.828.

695. WILDBOLZ, H. Biologic test for Active Tuberculous focus in the human body by the response to intradermal injection of Autourine. Corresp.Blatt f.Schweiz.Aerzte 1919 Vol.49.p.793.

696. WISE F. Miliary tuberculosis of the skin, lichen scrofulosum, and the papulo-tuberculoses.
Journ.Cutan.Dis.1919.Vol.37.p.105.

697. ZINGALE, M. A contribution to the study of Lichen Nitidus.
Giorn.Ital.d.mal.ven.e.della pelle 1922. Fasc.VI. p.1099.

SYPHILIS.

698. ALMKVIST, J. Beobachtungen ueber die Ursachen der verschiedenen Lokalisation der syphilitischen exantheme.
Arch.f.D. & S. 1916.Vol.123.p. 207.

699. BLASCHKO Ein Beitrag zur Lehre von der Immunität bei Syphilis.
Arch.f.D.u.S. 1911 Bd.106.

700. BETTMANN. Zur Frage der syphilitischen Reinfektion.
Dermat.Woch.1912.Bd.54.p.220

701. BROWN W.H., H. WADE, & PEARCE L., The significance of syphilitic Reinfection.
Journ.Exper.Med.Baltimore, 1921 xxxiii, 553-567.

702. BROWN W.H. & PEARCE L. The resistance (or immunity) developed by the reaction to syphilitic infection.
Arch.of D. & S. 1920 p.675.

703. BUSCHKE & FISCHER. Ueber die Beziehung der Spirochaete pallida zur Kongenitalen Syphilis.
Arch.f.D.& S., Bd.82.p.63.

704. BUSCHKE & HARDER. Ueber die provokatorische Wirkung von Sublimatinjektionen und deren Beziehungen zur Wassermannschen Reaktion bei Syphilis.
Deutsche Med.Woch.1909 No.26.p. 1139.

705. CAPELLI, J. Sifilide Secondaria tardiva e reinfezione.
Giorn.Ital.d.mal.ven.e.della pelle 1921. Fasc.ii. p.77

706. DELBANCO, E. Zur Infektiosität des Gumma.

Monatshefte f.prakt.Dermat.1904 Bd.38p.586.

707. DIDAY, P. De la réinfection syphilitique, de ses degrés et de ces modes divers.
Archives générales de médicine 1862. Vol.II. pp.26. 177 & 179

708. DUCREY, A. Caso singolarissimo di reinfezione sifilitica in una donna.
Giorn.Ital.d.mal.ven. 1888. Vol. 23, p.361.

709. EHLERS. Neue Statistik ueber 1501 Fälle von tertiärer Syphilis.
IV.Kongress d.deutschen dermatol Gesellschaft.1894.p.301.

710. ERSETTIG, M. The effect of Anti-typhoid and anti-cholera injections on syphilitic individuals.
Giorn.Ital.d.mal.v.e della p. 1920 p.187.

711. FINGER, E. Die syphilis als infektionskrankheit von standpunkt der modernen Bakteriologie.
Arch.f.D. & S. 1890.p.361.

712. " " & LANDSTEINER, K. Untersuchungen ueber syphilis an Affen.
I. & II. Mitteilung Wien.1905

713. FOURNIER. III Internat.Congr. of Dermat.1896.p.498

714. FOURNIER. La Syphilis.

Paris 1898-1901.

715. FRIEBOES, W. Ueber Pseudoprimäraffekte nach intensiven Behandlung in Frühstadium der Syphilis
Dermat.Zeitschr.1911.Bd.18.p.542

716. GASCOYEN. Case of syphilitic reinfection with Remarks:
Lancet 1874, Vol.2, p.762.

717. GATÉ & PAPACOSTAS. A new Serum Reaction in Syphilis.

Compt.rend.Soc.Biol.1920. Nov.20.

Abstr.B.M.J. Jan.15.1921.

718. GLASER, F. Die Erkennung der Syphilis und ihrer Aktivität durch probatorische Quecksilberinjektionen.

Berlin.Klin.Woch.1910, No.27.

719. GROSGLUCK, A., Immunotherapeutische Versuche bei Syphilis.

Dermat.Woch.Bd.59.p.960.

720. von GUTMANN, C. Reinfektio syphilitica oder Pseudoprimäraffekt.

Berlin.Klin.Woch.1911,No.28.

721. HALLER, D.A. & WALKER, J.C. Syphilis with Neurologic symptoms simulating other conditions, J.A.M.A. 1916, Vol.67.P.1497.

722. HANOCEK. Reinfektion und Solitarsekundäraffekt nach Salvarsanbehandlung. Dermat.Woch.1912.Bd.54.p.189.

723. HELL, F. Reinduratio Reinfektio Superinfektio and Chankriforme Papeln bei Lues. Archiv.f.D.& S. 1917.Bd.124. p.443.

724. HERXHEIMER & KRAUSE. Ueber eine Syphilitischen vorkommende Quecksilberreaktion. Dermatol.med.Woch.1902.No.50.

725. HOFFMANN, E. Mitteilungen und Demonstrationen ueber experimentelle Syphilis, Spirochaete pallida und andere Spirochaetenarten. Dermat.Zeitschrift Bd.13.p.561.

726. JADASSOHN, J. Syphilidologische Beiträge. Archiv.f.D.& S. 1907, Bd.86.p. 45.

727. KLAUSNER. Ein Fall von Reinfektion nach Salvarsan Munch.Med.Woch.1911.No.44.

728. KLAUDER, J.V. Superinfection in Syphilis. Journ.Cutan.Dis.1918 Vol.36.p. 515.

729. KÖBNER. Ueber Reinfektion mit constitutioneller Syphilis. Berlin Klin.Woch.1872,No.46.p. 549

730. KREIBICH, K. Zur Wirkung des Quecksilbers. Arch.f.D.& S., 1907 Bd.86.p.265.

731. KREMER, H. Zur Frage der Exanthemprovokation im zweiten Inkubationsstadium der Syphilis. Dermat.Zeitsch.1910.Bd.17.p.904.

732. LANDSTEINER, K. Immunität und Serodiagnostik bei menschlichen syphilis. Zentralbl.f.Bakt.1908.Bd.41.p. 785.

733. LANDOUZY [illegible]III.International Congress of Dermatology, 1896.p.478

734. LESSER, F. Ein eigenartiger Fall von Syphilitischer Reinfektion. Muhch.Med.Woch.1914.No.10.p.543

735. McINTOSH, J. Anaphylaxis & its bearing on Medicine, Quart.Journ.of Med.1914. Vol. VII. p.272.

736. MAURIAC, Ch. La syphilis tertiare dermo-epidermique des organes génitaux-urinaires Est-elle contagieuse? Revue general de Clin. et de Therap. 1887, p.194.

737. MERKEL, J. Ein Fall von veralteter knochensyphilis combiniert mit frischem induriertem Schanker und frischer papuloser syphilis. Bayr.ärztl.Intelligenzblatt 1868, p.289.

738. MIBELLI, Di un caso di gomme cutanee sifilitiche sviluppatesi dove erano state praticate da tempo injezioni di calomelano. X Reunione Soc.Ital.Dermat.e. Sifilografia 1908.

739. NAKANO, H. Ueber Immunisierungsversuche mit Spirochaeten Reinkulturen. Archiv.f.Derm. u.Syph. 1913.Vol.116, p.265.

740. NEISSER, A., Versuche zur uebertragung der Syphilis auf Affen. IV.Mitteilung Deutsch.Mediz. Woch.1906 No.13.

741. " Beiträge zur Pathologie und Therapie der Syphilis. Springer - Berlin, 1911.

742. " & BRUCK, C. Immunisierungsversuche. Beitr. z. Path. & Therap.d.Syph.1911.

743. NEUMANN, J. Welches sind die anatomischen veränderungen der luetischen Haut nach Ablauf der Klinischen Erscheinungen. Wien.Med.Woch.1885 No.26.

744. " Ueber die verschiedenen Reproductionsherde der Syph. Wien.Med.Woch.1887,Nos.8 & 9.

745. " Ueber die Klinischen und Histologischen Veränderungen der luetisch erkrankten Tonsillen und Gaumenbogen. Wien.Klin.Woch.1891,No.49.

746. " Beiträge zur Lehre von Syphilis-rezidiv. Wien.Klin.Woch.1902.No.28&29.

747. OKSENOW. Ueber Sklerogommi, welche eine Reinfektion vortäuschen können. Russische Zeitschr.f.Haut& Ven. Krankheiten. 1911.Bd.22. Abstr. in Derm.Woch.1912.Bd.54.p.381.

748. PASINI, A. Sulla permanenza della spirochete pallida in una macula atrofico-pigmentaria residua di una papula sifilitica.
VIII Reunione Soc.Ital.Dermat. e.Sifilografia, 1906.

749. " " Sifilide e Trauma.
Giorn.Ital.d.mal.ven.e.della pelle. 1921. Fasc.II.p.102.

750. PIGHINI - Localizzazione sifilitiche precoci e. frequente del sistema nervosa centrale nei combattenti.
La Reforma medica 1919. No.48.

751. QUEIRAT & PINARD. Résultats de l'inoculation des produits syphilitiques primaires aux sujets atteints d'accidents tertiaires.
Bull.Soc.franc.Derm. et Syph.1909 March 18.

752. RAILLET. Influence des petits traumatismes professionels sur les localisations de la syphilis.
Bull.Soc.Med.1919.

753. RAVAUT, P. Récidives et Réinfections après traitement de la syphilis.recente.
La Presse médicale 1913. No.74, p.749.

754. SACK. Verhandlung des Gesellschaft deutschen naturforscher und Arzte.
Hamburg 1901. p.424.

755. SIMON C. Case of injury followed by tertiary cutaneous syphilide.
Bull.de D.& S. 1921.p.82

756. SWEREW. A Case of syphilitic Reinfection after Salvarsan treatment.
Russki Wratsch.1911.No.38. Abstr in Derm.Woch.1912. Bd.54.p.546.

757. TARNOWSKY. Syphilis & Trauma.
Vierteljahrschrift.f.Dermat.1877

758. TOMASCZEWSKI, E. Ein Beitrag zur Pathologie der Syphilis.
Arch.für D.& S. 1907. Vol.85.p. 177.

759. THALMANN. Die Syphilis,
Dresden, 1906.

760. UNNA. Histopathologie der Hautkrankheiten.
Berlin, 1894. p.516-568.

761. VERESS, F. Veränderungen im Vorlaufe der Syphilis nach intensiven Behandlung Ueber pseudoreinfektion und Frührezidive.
Dermat.Woch.1912.Bd.54, pp.22 & 62.

762. VIGNOLO-LUTATI, K. Klinischer und experimenteller Beitrag zum Studium der Immunität bei syphilis tarda mit besonderer Berücksichtigung des auslösenden Einflusses vom Trauma in der Latenzeit.
Dermatol.Zentralblatt.1912.Bd.15 No.12.

763. " " " Contributo Clinico Spermentale allo Studio della immunita nella sifilide tarda con speciale reguardo all influenza rivelatrice del trauma nel periodo di latenza.
Il Morgagne, 1912.No.4.

764. WASSERMANN, A. Ueber neuere Immunierungsverfahren Schluss aus S. 1938.
Deutsch.Med.Woch.1907.Vol.33. p.1981.

765. WELANDER, E. Ueber die Reaktion der syphilitischen Hautaffektionen (besonders des Roseols) gegen die erste Einführung Quecksilber in den Organismus.
Arch.f.D.& S. 1909 Bd.95.p.75.

766. ALDERSON, H.E., Value of the Tests with commercial Luetin.
Arch. of D.& S. 1922.p.610.

767. ARISTOWA. La luétine-réaction de Noguchi.
Soc. de D. & S. Tarnowsky, St,Petersburg, 14 mars, 1914.

768. " Observations sur la pallidin - réaction dans la syphilis.
Dermatologie (Russe) mars 14 p.322

769. BAERMANN & HEINEMANN. Die Intrakutanreaktion bei syphilis und Frambösie.
Münch,Med.Woch.1913. Vol.60, p.1537.

770. BIACH, M. Reinfectio syphilitica nach drei ein halb Monaten.
Wiener Klin.Woch.1913.Vol.26 p.1363.

771. BLECHMANN et DELORT, La réaction à la luétine de Noguchi dans la syphilis infantile, étude preliminaire.
Soc.de Pediatrie, 7 avril, 1914.

772. BLECHMANN, DELORT & TULASNE, Résultats comparatifs de la réaction à la luétine et de la réaction de Wassermann dans la syphilis héréditaire.
B.S.f D. juin 1914.p.391

773. BOAS, H. & DITLEVSEN C. Untersuchungen ueber Noguchi's Luetin-reaktion.
Arch.f.D.& S. 1913.Vol. 116 p.852.

774. BOAS & STURUP, Untersuchungen ueber Kutanreaktionen mit Organextrakten bei Syphilitikern.
Archiv.f.D.& S., 1914. Vol.120 p.730.

775. BORBERG, N.C. Concerning the luetin reaction and the effect of Iodine.
B.J.D. 1917.p.190.

776. BURNSTEIN, NAST & NICKAU, Ueber eine unspezifische Abbaureaktion in Serum gewisser Syphilitiker.
Arch.f.D.& S. 1914.Vol.120, p. 240.

777. BURNIER, La cuti-réaction dans la syphilis.
Annales des maladies veneriennes Janv.1914.p.1.

778. BURNIER, M. La cuti-réaction à la luétine.
Bull.Soc.franc. de D.& S., 1914
Vol.25.p.31.

779. CALICO. The Luetin Test. Laboratorio. March 1921.

780. CHIEFFI, A. Short note on the intradermic reaction with Naguchi's Luetin in Syphilis.
Giorn.Ital.d.mall. ven.e.della pelle 1918. Fasc.II.p.65.

781. COHEN, M. Noguchi's Cutaneous Luetin Reaction and its application in Ophthalmology.
Arch.of Ophthalm. 1912.Vol.51. p.9.

782. COLE & PARYZEK. The provocation of the luetin test in non-syphilitic patients.
J.A.M.A. 1917.p.1089.
Abstr.Am. de D.& S. 1918-19, p. 43.

783. CUIFFO. Tentativi della cuti-ed oftalmoreazione nella sifilide.
Giornale Ital.d.mal.ven.e.della pelle. 1909.p.170.

784. CURTI. Contributo alle studio del valore clinico della reazione alla luetina (Noguchi)
Riforma medica. 13 Nov.1914, p.1264.

785. DESNEUX. The cutireaction with Luetin in Syphilis.
Soc.Clin.des Hop. de Brux. Nov. 8, 1913.

786. FAGINOLI & FISICHELLA. Weitere Beobachtungen ueber die Intrakutanreaktion mit dem Luetin von Noguchi.
Berlin.Klin.Woch.1914.No.10.

787. FONTANA, A. Ueber die Diagnose der Lues durch die Intradermoreaktion.
Dermat.Wochenschrift.1912. Bd.54.p.109.

788. FOX, H. Experiences with Noguchi's Luetin-reaction.
Journ.Cut.Dis. Aug. 1912.

789. GRADWOHL. The Luetin Test in Syphilis.
Medical Record.1912.No.5.

790. HANES, F.M., The Luetin Reaction in the diagnosis of Tertiary and Latent Syphilis.
Amer.Journ.Med.Sci.1915.Vol.40. p.703.

791. JADASSOHN. Indurierte (tert.) Pseudoschanker.
IXth Kongress der Deutsche derm. Gesellsch. Berne.1906.p.309

792. KAFKA, V., Ueber Noguchi's Luetinreaktion mit besonderer Berücksichtigung der Spätlues des Centralnervensystems.
Berlin Klin.Woch.1915 No.1 p.15

793. KALISKI, D.J. The Luetin Skin Reaction in Syphilis.
New York Med.Journ. 1915. Vol.98 p.24.

794. KAMMERER, H. Diagnostische Intrakutanreaktionen mit Spirochaetenextrakt.
Münch.Med.Woch.1912.No.28.

795. KILGORE. The luetin cutaneous reaction for syphilis.
Journal of the American Medical Association 18 avril, p.1236. 1914.

796 KLAUSNER, E. Ueber eine klinisch verwendbare kutanreaktion auf tertiäre Syphilis.
Wien.Klin.Woch.1913.No.24.

797. " " Zur Technik der Pallidinreaktion.
Munch.Med.Woch.1914.No.2,

798. " " Die Kutireaktionen bei Syphilis mit besonderer Berücksichtigung der Pallidinreaktion. A.f.D. 1914, p. 444.

799. KOLMER, J.A., & BROADWELL, S. Anaphylactic skin reactions in relation to Immunity. II. The relation of the Luetin Skin reaction to Immunity in Syphilis.
Journ.Immunol.1916. Vol.I. p.429.

800. " " BROADWELL S. & MATSUNAMI Y. The effect of Potassium Iodide on the Luetin Reaction.
J.A.M.A. 1916. Vol.67.p.718.

801. " " & GREENBAUM, S.S. Cutaneous Allergy in Syphilis; with special reference to the Luetin Reaction and the necessity for controls in intracutaneous tests.
Journ.A.M.A. 1922 Vol.79.p.2063

802. KOLMER J.A., IMMERMAN, S.L., MATSUNAMI, T., & MONTGOMERY, C.M., The effect of certain drugs on the Skin Reactions.
Journ. of Laboratory & Clin.Med. 1917,No.9. Abstr.J.Cutan. Dis.1917 Vol.35. p. 785.

803. KONIG, H. Ueber den Wert der Luetinreaktion in differential diagnostischer Beziehung.
Arch.f.Psychiatrie 1917.Bd.57. p.91

804. LAGANO, L. & BROUGHTON & ALCOCK, W. The Luetin reaction in Syphilis and other infectious Diseases.
Bull. de la Soc.Med.des Hop.de Paris. 1913. No.37.

805. LUITHLEN, Ueber Veränderungen der Hautreaktion, Dermatologische Sektion der 85. Versammlung deutscher Naturforscher und Aerzte.
Wien.21-27 Sept.13. A.f.D. Mai 1914.p.420.

806. McNEIL, H.L. Experiences with Noguchi's Luetin in Syphilis.
J.A.M.A. 1914.p.529.

807. MARIE, A. & BROUGHTON-ALCOCK. On 100 Luetin reactions.
Bull. de la Soc,Med.des Hop.de Paris. 1913. No.34.

808. MATSURA. Ueber die Noguchische Luetinreaktion.
XIV,Dermat.Urol.Kongress, Tokyo, 3-4 Avril 1914. Japanische Zeitsch. f.D. & U. juin 1914 p.545.

809. MEIROWSKY. Ueber die diagnostische und spezifische Bedeutung der v.Pirquetschen Hautreaktion.
Arch.f.D.& S., 1909.Bd.94.

810. MÜLLER & STEIN. Kutireaktion bei Lues . Dermat. Sektion der 85 Versamml.deutscher Naturforsch. & Aerzte.
Wien, 21-27 Sept.13. A.f.D.Mai 1914.p.424.

811. NAKANO, H. Experimentelle und Klinische Studien ueber kutireaktion und anaphylaxie bei syphilis.
Arch.f.Dermat.1913, Vol.116 p.281.

812. NANU-MUSCEL u. DERSCA. Ueber die Luetin-reaktion nach Noguchi.
Munch.Med.Woch.9 Juin 1914.p. 1271.

813. NEISSER, A. Erforschung der Syphilis.
Arbeiten aus dem Kaiserlicher Gesundheitsampte Bd.37.p.173.

814. NICOLAS, FAVRE & GAUTHIER. Les Cutiréactions dans la syphilis.
Comptes rend.d.l.Soc.Biol. de Paris 1910. Feb.12.

815. NOBL, G, & FLUSS, K. Zur Intrakutanreaktion bei Syphilis.
Wiener Klin.Woch.1912. Vol.25. p.475.

816 NOGUCHI, H. A cutaneous Reaction in Syphilis.
Journ.Exper.Med.1911. Vol.14. p.557.

817. " " Hautallergie bei syphilis - ihre diagnostische und prognostische Bedeutung
Munch.Med.Woch.1911.p.2372.

818. " " The Luetin Reaction.
J.A.M.A. Oct.5, 1912.

819. " " Practical Application of the Luetin Test.
New York Med.Journ.1914. p.349.

820. NONNE. Luetin.
Med.Klin.1914.Bd.2.p.1629.

821. PERKEL. On the intradermal Reaction with Noguchi's Luetin in syphilis.
Arch.f.D.& S. 1915.Vol.121.p.7

822. ROBINSON, D.O., Diagnostic value of the Noguchi Luetin Reaction in Dermatology.
Journ.Cutan.Dis. 1912. Vol.30. p.410.

823. ROEDNER, J. Beitrag zur Frage der praktischen Verwerthung der Palladinreaktion.
Dermat.Woch.Bd.59 p.1021.

824. SCHMITTER, F. The Luetin Test.
Journ.Cutan.Dis. 1913.Vol.31. p.549.

825. SCHERRICK, J.W., The effect of Potassium Iodide on the Luetin Reaction.
J.A.M.A. 1915, Vol.65.p.404.

826. STOKES. A Luetin Reaction in Syphilis produced by agar, with a brief consideration of its mechanism.
J.A.M.A. 1917.p.1092.
Abstr.Annales de D.& S. 1918-19 p.43.

827. TEDESCHI. Ueber kutis- und Ophthalmoreaktion bei syphilis.
Munch.Med.Woch.1908 p.2200.

828 TESCOLA, C. Ueber die Noguchische Kutireaktion mit Luetin.
Soc.Med.Chir.Bologna. Jan.22 1914.

829. WOLFSOHN, J.M. The cutaneous Reaction of Syphilis.
Bull.John Hopkin's Hosp.1912. Vol.23.p.223.

RINGWORM and FAVUS.

830. ADAMSON H. G. Lichen pilaris seu Spinulosus. B. J. D. & S. 1905, pp. 39 & 77.

831. AMBERG, S. The cutaneous Trichophytin Reaction. Journ. Exper. Med. 1910, Vol.XII. p.435.

832. AMBROSOLI, G. A. Coltora di Trichophyton Gypseum del sangue circolante in Tricofizia profunda con Lichen Trichophyticus. Giorn.Ital. d.mal.ven.c.della pelle. 1921 Fasc.III. p.233.

833. ARNOLD, W. Die intradermale Trichophytinreaktion beim Kinde. Arch.f.D. & S., 1921, Bd.136, p. 125.

834. ARZT, L. Die Allgemeinexantheme bei Mikrosporie; Mikrosporide. Dermat.Woch. 1922. Bd.75, pp. 1193 & 1220.

835. ARZT, L. & FUHS, H. Ueber mykotische Allgemeininfektionen bei Trichophytie and Mikrosporie (Trichophytosen und Mikrosporosen) Arch.f.D. & S., 1921. Bd. XXXVI. p.333.

836. AUDRY, Ch. Keratoris circumpilaris (Keratose pilaire engainante) Monatshefte f.prakt.Dermat. 1904. Bd.XXXVIII.p.529.

837. AXAMIT, O. Ueber-empfindlichkeitserscheinungen nach Hefeinjektion. Arch.f.Hyg. 1907 LXII. p.15.

838. BAUM, - Discussion on Immunity to Ringworm & Favus. Dermat. Zeitsch. 1910, Bd.XVII. p.572.

839. BECK, S.C., Ueber Keratosis Spinulosa (Lichen spinulosus Crocker) Dermat.Woch.1912, Bd.LV. p.1458.

840. BLASCHKO, - Discussion on Immunity to Ringworm and Favus. Dermat.Zeitschr. 1910, Bd.XVII. p.572.

841. BLOCH, B. Zur Lehre von den Dermatomykosen. Archiv. f. D. & S., 1908, Bd.XCIII. p.157

842. BLOCH, B. Das Achorion violaceum, ein bisher unbekannter Favuspilz. Dermatol. Zeitschr. 1911, Bd. XVIII. Heft 9.

843. " " Ueber das vorkommen des Mäusefavus beim Menschen und seine stellung im system der Dermatomykosen. Dermatol. Zeitschr. 1911. Vol. XVIII. p. 451.

844. " " Ueber "Ableitende" Verfahren in moderner Beleuchtung. Medizinische Klinik. 1911. No. 16. p. 607.

845. " " Die Trichophytien und verwandte Pilzkrankungen der Haut. Correspondenz-Blatt f. Schweizer Aerzte. 1912. Vol. XLII. p.3.

846. " " Die Allgemein-pathologische Bedeutung der Dermatomykosen. Halle 1913. p. 82.

847. " " Ueber einige Allgemein-pathologische und therapeutische Probleme auf dem Gebitte der Dermatomykosen. Munch. Med.Woch. 1915. p. 737. Nos. 22 & 23.

848. " " Les Trichophytides. Annales de D. & S., 1921. Vol. II. pp. 1 & 55.

849. " " & MASSINI R. Studien über Immunität und Ueberempfindlichkeit bei Hyphomyzetenerkrankungen. Zeitschr. f.Hygiene und Infektionskr. 1909. Bd. 63 p. 68.

850. BLUMENTHAL, F., & HAUPT, ASTA von - Immunisatorische Vorgänge bei der Trichophytie des Menschen. Deutsch. Med. Woch. 1920.No. 2.

851. BODIN, E. Les Champignon parasites des hommes. pp. 32 & 38.

852. BRUCK, C. & KUSONOKI F., Ueber spezifische Behandlung von Trichophytien. Deutsch. Med. Woch. 1911. Vol. XXXVIII. p. 1110.

853. BRUHNS. Untersuchungen über Immunität nach überstandener Trichophytie-Infektion. Versaml. Berlin. Derm. Gessell. Feb. 13. 1912. Dermat. Woch. 1912. Bd. 54 p. 290.

854. BRUHNS C. & ALEXANDER A. Zur Frage der Immunität nach Trichophytie-Erkrankungen. Dermat. Zeitschr. 1910. Bd.17. Heft 10.

855. BRUUSGAARD, E. Haematogenous Infection with Trichophytia. B.J.D. & S. 1922.p.150.

856. BUKOWSKI, - Ein Beitrag zur Kenntnis der experimentellen und klinischen Eigenschaften des Achorion Schönleinii. Arch.f.D. & S. 1900. Bd.51.

857. CALDERONE, - Contributo sperimentale alle biologia de trichophyton e dell' Achorion Schönleinii. Communicazioni alle Soc.Ital. de dermat. 1899 Oct.22.

858. CAMPANA, - Dermic Trichophytosis. Giorn. Ital d.mal. ven e della pelle 1887.July-Aug.

859. CARINI, - Sull' istogenesi del pseudo-tubercolo sperimentale . Lo Sperimentale. 1891. Fasc.V. & VI.

860. CAROL, W. L. L. Die Komplementbindungsreaktion bei Trichophytie. Nederl. Tijdschr.v.Genetsk 1918. Bd.2 p.784. Ref. Berl.Klin.Woch. 1918 p.1006.

861. CEDERCREUTZ, - Recherches sur un coccus polymorphe, hôte habituel et parasite de la peau humaine. Lab. de la ville de Paris à l'hop.St.Louis.1901.

862. CHABLE, R. H. Ueber kerion celsi und lichenoides exanthem bei Audoinscher mikrosporie. Dermat. Zeitschr. 1917, Vol.14.

863. CHAJES, B. Ueber Mikrosporieerkrankung der behaarten Kopfhaut Berlin.Klin.Woch. 1901 Vol.XLV. p.1491.

864. " " Mikrosporie in Berlin. Medizin.Klinik. 1908. 4 Jahrgang.p.905.

865. CHIRIVINO, V. Granuloma trichophyticum Majocchi. Giorn.intern delle scienze med. 1907. No.10.

866. CITRON, J. Ueber das verhalten der Favus und Trichophyton pilze in organismus. Zeitschr. Hyg.u.Infektkht. 1905, Vol.XLIX. p.120.

867. COSTA S. & FAYET, A., Sur l'immunite acquise dans les trichophyties. Comptes rendu. Soc. de Biol. 1911. Tome LXX. p. 553.

868. COHEN, MAX. Der Untergang pathogener Schimmelpilze. Monograph. Bonn. 1887.

869. DARIER, J. & HALLE, J., Sur un cas de Gramulome favigue. Annales de D.& S., 1910. p.129.

870. FARRERA DALLA, Sur les trichophytons de la provence de Parme. Annales de D. & S., 1909.

871. FOLLY, M., Vienna Dermat. Soc., May 11, 1892.

872. FORLANINI, - Studi sulla anatomica, pathologica e la nature del Cherion (celso) e vespaio del capillizio (Dubini). Giorn. Ital. d.mal. ven. e della pelle. 1880.

873. FOX, COLCOTT, Lichen scrofulosus (L. Spinulosus) B.J.D. 1907. p. 130.

874. FUHS, HERBERT, Ueber die therapeutische Wirksamkeit eines durch Verdauung von Trichophytonkulturen gewommenen Priparates (Joannovics). Wien. Klin. Woch. 1920. Jahrgang XXXIII. p. 653.

875. GILETTI, A., Trichophytosis of the buccal mucous membrane. Toreno. Monograph. 1895. Abstr. B.J.D. 1896. p. 59.

876. GLASER, F., Eine Mikrosporie-epidemie. Berlin Klin. Woch. 1908. Vol. XLV. p. 1086.

877. " " Die Mikrosporie und Makrosporie der Kinderkopfe. Berlin Klin. Woch. 1908 Vol. XLV. p. 2013.

878. GUTH, A. Ueber lichenoide (klein papulöse spinulöse) Trichophytie. Archiv. f.D. & S. 1913. Vol. 118 Originale p. 856.

879. HALBERSTADTER, - Eine eigenartige Form von Keratosis follicularis. Archiv. f.D. & S. 1903. Bd. LXVII. p. 133.

880. HANAWA, S. Histologische Untersuchungen über Trichophytie Heilung und Allergie beim meerschweinschen. Dermat.Woch.1913. Bd.LVII. p.939.

881. HELLER, - Discussion on Immunity to Ringworm & Favus. Dermat.Zeitschr.1910.Bd.XVII. p.571.

882. HELLER, JULIUS, Die vergleichende Pathologie der Haut. Berlin, 1910 (Hirschwald). p.633

883. HERXHEIMER & KOSTER, Ueber sekundäre lichenoide Trichophytie. Dermat. Zeitsch. 1914, p. 567.

884. HUBER, - Ueber die mikrosporieepidemie in Schönberg. Medizin.Klinik. 1909, 5 Jahrg. p.762.

885. JADASSOHN, - Die Trichophytie und verwandte Pilzerkrankungen der Haut. Correspond. Blatt f. Schweizer Aertze 1912.Vol.XLII. p.22

886. " Ueber die Trichophytien (allgemein-pathologisches und Klinisches). Berlin Klin. Woch. 1918. Vol.LV. p. 489.

887. JESSNER, M. Zur Pathogenese der Trichophytide. Arch. f. D. & S. 1921. Bd.136 p.456.

888. KOLMER J.A., & STRICKLER, A., Complement Fixation in Parasitic Skin Diseases - Journ. Amer. Med.Assoc.1915, Vol.LXIV. p.800.

889. KUSONOKI, F. Experimentelle und Klinische Studien zur Lehre der Dermatomykosen (Infektion, Prophylaxie, Immunität) Archiv.f.Derm. und Syph. 1913, Vol.ii4, p.1.

890. LEWANDOWSKY, F. Ueber Lichen Spinulosus. Arch. f. D. & S., 1905, Vol.LXXIII. p.343.

891. " Ueber Kerion Celsi verursacht durch Mikrosporin Audoini. Archiv.f.D. & S., 1915, p.561.

892. LITTLE, GRAHAM, A case of Keratosis follicularis (Lichen Spinulosus) B.J.D., 1905 p.363.

893. " " Two cases of Keratosis follicularis in sisters. B.J.D., 1901.p.417

894. LOEB, H. Leukogen (Höchst). Ein spezifikum gegen sykoais parasitaria profund und weitere Erfahrungen über Leukogen. Dermat. Woch. 1918. pp. 377 & 398.

895. LOMBARDO, C., Experiments on Hypersensitiveness and Immunity in certain Dermatomycoses. Ref. Arch. f.D. & S. 1911. Bd. 110.

896. " " Richerche sulla Ipersensibilita ed Immunita in alcune Dermatomycosi. Giorn. Ital. delle mal. sensi e della pelle 1911. Vol. III. p. 70.

897. MACLEOD, J. M. H., Case of Lichen spinulosus associated with seborrhoeic dermatitis. B.J.D. 1908. p. 85.

898 MAJOCCHI, - Sopra una nuovu tricofizia (granuloma tricofitico) Bolletine della R.Academia Med. di Roma, 1883.

899 " Granuloma Trichophyticum, Soc. Ital. di Derm. e. Syph. Rome Dec. 1907. Reported Archiv. f.D. & S. 1908 Bd. 93. p. 235.

900. " D. Clinical contribution to the histology of granuloma Trichophyticum. Giorn. Ital. d. mal. van. e della pelle. 1920. Fasc. III. p. 397.

901. MARTENSTEIN, S. Experimentellen Beiräge zur Frage der überempfindlichkeit des Meerschweinchens nach uberstandener Trichophytie. Archiv. F.D. & S. Bd. 131 p. 180.

902. MARTENSTEIN, H. Weitere experimentelle Untersuchungen über die allergie des Meerschweinchens nach der Impfung mit Achorion Quinckeanum. Arch. f. D. & S. 1923. Bd.142. p. 279.

903. MAZZA, - Ueber das granuloma trichophyticum. Arch. f. D. & S., 1907. Vol. XXXVII. p. 25.

904. MIESCHER, G. Die Trichophytinreaktion im Blutbild, Dermatol. Woch. 1915. Vol. LXI. p. 1011.

905. NAEGELI, L.A. Ueber den Einflussder Pilze auf die Bildung von Riesenzellen mit Wand-standigen keinen. Archiv. f.Exper.Path.und Phar. 1885. p. 101.

906. NATHAN, E. Zur Kenntniss der Immunitätsvorgänge bei der Trichophytie des Menschen. Dermat. Woch.1920. Bd. LXXI. p.439.

907. NEISSER, - Plato's Versuche über die Herstellung und Verwendung vom Trichophytin. Archiv.f. Derm.& Syph. 1902. Bd. 60. p.63.

908. NOVAK, F. V., Injections of Trichophytin in the treatment of Ringworm Infections. Ceska Dermatologie. 1922 III.p.19. Abst. B.J. D. & S., 1922 p.173.

909. PECORI - Sull' immunità nella Tricofizia. Roma, 1914.

910. PEDERSEN, H. B. Ein Beitrag zum studium der Pathogenese der Sekundären Trichophytide. Derm. Zeitsch. 1917. p.731.

911. " " " Ueber die sogenannten sekundären lichenoiden Trichophytien. Hospitalstidende 1916. Heft.20

912. PELLIZZARI - Recerche sul Trichophyton tonsurans. Giorn.Ital d. mal ven e delle Pelle. 1888.

913. " - Del polimorfismo tricofitico ed in particolare di una forma clinica non descritta. Clin. Derm. Firenze. 1890

914. PERSSON, - Preliminary report of a case of Favus treated by bacterial inoculations. J.A.M.A., 1909. p.1663

915. PICCARDI, - Keratosis pilaris e keratosis spinulosa. Ricerche cliniche ed istologiche. Torino 1906.

916. PINI, - Granuloma trichophyticum (Majocchi). Giorn. Ital.d.mal ven e delle pelle 1897.p.710.

917. " Granuloma Trichophyticum (Majocchi) . Derm. Zeitschr. 1902, p.710.

918. PLAUT, H. C., Die Dermatomykosen oder Hautpilze. Kolle & Wassermann's Handbuch d.pathogenen Mikroorganismus. 1903, Bd.1.p.599.

919. PLAUT, H. C., Dermatomykosen in Mracek Handbuch der Hautkrankheiten. 1906.Bd.IV.

920. PRYTEK, - Zur Kenntniss der Immunität nach Trichophytieinfektionen. Arch.f.D. & S., 1912, Vol.CXIII.originale p.821.

921. PULVERMACHER, L. Zur Klinik und Pathogenese der Trichophytide. Dermat.Zeitsch. 1919, Vol. XXVII. p.11.

922. QUINCKE, - Ueber Favuspilze. Arch.f.exper.Path.und Pharm.1886 Heft 1 & 2.

923. RADAELLI, F. Pseudoactinomycotic formations produced experimentally with Achorion Schönleinii & Trichophyton Violaceum. Giorn.Ital. d.mal.ven.e.della pelle 1915. Fasc.V. p. 413.

924. RASCH, C. Secondary lichenoid Trichophytides in association with Kerion-Celsi (Lichen spinulosus trichophyticus). B.J.D. 1916. Vol.XXVIII.p.9.

925. " " Clinical Remarks on Trichophytia Profunda (Kerion-celsi) B.J.D.& S., 1920 p.317.

926. REBAUDI, - I fanomeni anafilattici nella tricofizie Genova. 1920.

927. RIBBERT, - Ueber wiederholte Infektion mit pathogenen Schimmelpilzen und über Abschwächung derselben. Deutsch. Med.Woch.1888, Vol. XIV. p.981.

928. ROZSAVÖLGYI, M. Die Behandlung der Trichophytiasis profunda, mit dem Kulturextrakt des Trichophytonpilzes. Dermat. Woch.1921 No.35.p.924.

929. SABOURAUD, R., Les Teignes Paris, 1910. Immunisation et Anaphylaxie. p.234

930. SABRAZES, - Sur le Favus. Paris, 1893.

931. " Pseudo-tuberculoses favique experimentale Soc.franc. de dermat et syph. Sed.Apr.7 1893.

932. SAEVES, J. Experimentelle Beiträge zur Dermatomykosenlehre. Arch.f.D. & S., 1915. Vol.XXI. p.161.

933. " " Experimentelle Beiträge zum Dermatomykosenlehre Korymbiforme lichenoide Trichophytie. Arch.f.D. & S., 1915. Vol.CXXI p.228.

934. SALINIER, - Keratoris spinulosa. Toulouse 1906.

935. SCHOLTZ, W. Ueber die diagnostische und therapeutische Anwendung des Trichophytin Höchst. Münch.Med.Woch.1918.p.509.

936. SCHOLTZ & DOEBEL, Beobachtungen über Favus sowie über Trichophytie des Kopfes, des Bartes und der Nägel. Archiv.f.D.& S. 1908. Bd.XCII.

937. SCHRAMEK, - Favus ohne scutulum-bildung. Wiener Dermat.Gesellschaft.May 1911. Ref.Arch. f.D. & S., 1911. Bd.110 p.286.

938. SEMMER, M. E., Resultate der injectionen von Pilzsporen und Pilzhefen in's Blut der Thiere. Arch.f.Path.Anat. &c. 1870, p.158

939. SEQUEIRA, J. H. A case of Trichophytic granulomata. Brit.Hourn.D. 1912. Vol.XXIV. p.207.

940. SORRENTINO, - Granuloma trichophyticum (Majocchi). Giurn.Ital.d.mal.ven e delle pelle 1907. Fasc.VI.

941. STEIN, A. D., Die Spezifische Behandlung der tiefen Trichophytie. Wien.Klin.Woch.1912, Vol. XXV. p.1817.

942. " " " Die Fadenpilzerkrankungen des Menschen. 1914.

943. " " " Ueber Cutireaktion bei Favus. Arch.f. D. & S., 1921, Vol.CXXXII., p.294.

944. STRICKLER A. The vaccine treatment of Ringworm of the scalp. Journ.Cutan.Dis. 1915, Vol.XXXIII. p.181.

945. " " Differential Blood-counts in parasitic skin diseases. Amer.Journ.Cutan.Dis. 1916, p.757.

946. SUTTER, E. Beiträge zur Lehre von der Immunität und Überempfindlichkeit der Trichophytie-erkrankungen. Dermat.Zeitschr. 1917. Vol.XXIV. p.65

947. " " Zur Kenntnis der Pathogenese der Trichophytide. (Skarlatiniforme, lichenoide und nodöse exantheme,) 1920. Archiv.f. D. & S. 1920. Vol.CXXVII. Orig. p.735.

948 THARDSHIMANJANZ, A. Experimentelle-biologische Untersuchungen über Immunität und Allergie bei Hyphomycetenerkrankungen. Basel 1910.

949. TOMASZEWSKI, - Discussion on Immunity to Ringworm and Favus. Dermat.Zeitschr. 1910. Vol.17 p.570.

950. " E., Kulturelle und experimentelle Untersuchungen über Achorion Schönleinii und Achorion Quinckeanum. Dermat.Zeitschr. 1911 Bd.XVIII. p.886.

951. TRUFFI, M. Ricerche sperimentale sulle Tigne. Giorn. Ital. d. mal.ven.e della pelle. 1902,

952. " " Sulle Tigne 1902. p.142.

953. " " Un caso de Cherion devuto all' Achorion di Schönleinii Granuloma nel tessuto cutaneo provocate dall'achorion. Giorn.Ital. d. mal.ven e della pella. 1902. p.491.

954. " " Sur le Trichophytin. Revue pratique des maladies cutan. et syph. 1903, Vol.X.

955. " " Ricerche sulla Tricofitina. Clinica medica Italiona. 1904.

956. VERROTTI, GUISEPPE. A case of small spored Ringworm of the scalp due to microsporon Lanosum. Giorn.Ital.d.mal.ven.e della pelle 1916. Fasc.II. p.84.

957. VIGNOLO-LUTATI. Case of Granuloma Trichophyticum (Majocchi's) Archiv.f.D. & S., 1908, Bd.XCIII. p.236

958. WANDEL, O. Zur Frage des Tier - und Menschenfavus. Deutsche Archiv.f.Klin.med. 1903. Bd. LXXVI. p. 520.

959. WILLIAMS, C. M. Four cases of lichenoid Trichophytide Arch.f.D.& S. 1921. p.353.

960. ZOLLIKOFER, R. & WENNER, O., Ueber eine St. Galler Mikrosporie-epidemie. Korrespondenzblatt f.Schweizer Arzte. 1908.No.17 Ref.Der. Zeit. 1909. Bd.16. p.121.

SPOROTRICHOSIS & OTHER FUNGUS DISEASES.

961. AUVRAY, - A propos d'une nouvelle mycose observée chez l'homme. Compt. rendues de la Soc. de Chir. 1909. p.186.

962. BEURMANN, CLAIR, & GOUGEROT, Une mycose nouvelle l'Hemisporose. Bull. et.Mem. de la Soc.Méd des Hôpit. de Paris. 1909. p.917

963. BEURMANN & GOUGEROT, L'État de sensibilisation des Sporotrichosiques. Bull. et Mém. de la Soc.Méd. des Hôp. de Paris. Oct.8, 1909.

964. " " " Intradermo-réaction sporotrichosinique. Annales de D. & S. 1910.p.120.

965. " " " ibid. p.121.

966. BEURMANN, RAVAUT, GOUGEROT & VERDUN. Intradermo-réactions Sporotrichosiniques positives chez les malades porteurs de lésions cutanées non sporotrichosiques. Annales de D. & S. 1910.p.121

967. BEURMANN & GOUGEROT, Enclycopedie scientifique des aides-mémoire. Les Nouvelles Mycoses. Paris.

968. " " " Les sporotrichoses. Felix Alcan. Paris, 1912.

969. GOUGEROT & CARAVEN. Hémisporose humaine. Revue de Chir. 1909. December.

970. " " " Mycose nouvelle; l'Hémisporose. Bull. de la Soc. de Biol. 1909 Vol.66.p.474.

971. LANGERON M. Les Sporotrichoses. Nouveau Traité de médécine. Gasc.4.p.462

972. LEBAR & ST. GIRONS. Sporotrichose de de Beurmann. Ulcération cutanée de l'avant bras avec ostéite, du cubitus, Sérodiagnostic et intradermo-réaction positif. Annales de D. & S. 1910.p.121.

973. PAUTRIER & LUTEMBACHER. Sub-cuti-reaction positive obtenu chez deux sporotrichosiques par l'injection sous-cutanée de cultures jeunes de Sporotrichose, broyées, diluées dans du sérum et sterilisées. Soc.de Biol. de Paris 1909.II.p.24

974. WALKER N. & RITCHIE J. A case of Sporotrichosis. B.M.J. 1911 Vol.II. p.1.

DERMATITIS VENENATA.

975. ADAMSON, H. G. On Eruptions of the Napkin region in Infants, with special reference to the diagnosis of the eruptions of congenital syphilis, with certain non-specific Napkin area eruptions of common occurrence.
B.J.Dis.Child. 1908. Vol.5,p.13.

976. " " Dermatitis from explosives used in Air Raids.
B.M.J. 1917. Vol.II. p.45.

977. ADELUNG, VON, E. An experimental study of Poison oak
M.A. Thesis, Univ. of Calif.1912
Arch. Int. Med. 1913, p. 148.

978. ALDERSON, H. E. Notes on Skin Diseases observed at the Letterman General Hosp.
California State Journal Med. 1920. Vol.18. p.353.

979. ALHAIQUE A. Sui fenomeni di Anafilassia nelle Scottature.Pathologica. 1912. Vol.4. p.479.

980. ANDERSON, J. W. Geranium Dermatitis.
Arch. of D.&S. 1923. p.510.

981. APPEL J. Dermatitis frontalis durch. Hutlederersatz.
Derm. Woch. 1920. p.775.

982. ATKINS, W. R. G. Oxydases and their inhibitors in plant tissues.
Sc. Proc. Royal Dublin Soc. 1913 Vol. 14, p.144.

983. AUER, J. The influence of systemic changes on local tissue reactions.
Proc. Soc. exper. Biol & Med. 1920. p.93.

984. " " Local autoinoculation of the sensitized organism with foreign protein as a cause of abnormal reactions.
Jour. exper. med. 1920.Vol.32, p.247.

985. BAER. Dermatitis infolge Gebrauchs einer mit Ersatzleder verschenen Bartbinde. Munch.Med.Woch. 1920. Bd. 67, p. 874.

986. BALZER, Dermatitis eczematiforme des pieds provoquee par la teinture des chaussettes. Ann. de D.&.S. 1898, p. 683.

987. BALZER & GAUCHERY Dermite eczematiforme des pieds provoquee par la teinture des chaussettes. Ann. de S. &. S. 1899. p. 683.

988. BARKER, W. W. Another case of poisoning with Mrs. Pottler's pure walnut juice hair dye. J.A.M.A. 1909. Vol. 52. p. 787.

989. BENTLEY B. & TRIMEN H. Medicinal Plants. London 1880. Vol. 3. p. 153.

990. BERNSTEIN, M. J. Dermatitis produced by Dinitrochlorbenzol. The Lancet. June 8, 1912.

991. BETTMANN, Bemerkungen über eine durch Schweissledererersatz hervorgerufene Dermatitis. Munch.med.Woch. 1920. m. 5. p. 291.

992. BIDIE, G. Satinwoods and Dermatitis. B.M.J. 1905. Vol.I. p. 74.

993. BLACKWOOD, W. R. D. Some thoughts on Rhus poisoning. Philadelphia Med.Times 1880. Vol. 10. p. 618.

994. BLASCHKO, A. Die Hauterkrankungen der Anilinarbeiter Deutsch.Med.Woch. 1891. Vol. 17. pp. 1241 & 1265.

995. " " Gewerbliche Hautkrankehiten. Deutsch.Med.Woch. 1892, No. 7.

996. BLOCH, B. Experimentelle Studien über das Wesen der Iodoformidiosynkrasie. Zeitsch.f. exper.path.u.Therap. 1911. Vol. 9. p. 509.

997. BROWN, W. H. Fatal blood poisoning following a wound by the Primula Obconica.
Lancet, 1906. Vol.I. p.861

998 BROWN, E. D. Experiments on the variability in susceptibility to poison ivy.
Arch. of D.&.S. 1922. p.714.

999. BRUCK, C. Experimentelle untersuchungen über das Wesen der Arznei exantheme.
Berlin. Klin.Woch. 1910. No.12 p.517.

1000. BRUCK, K. Der einfluss der anaphylaxie [illegible] forschung auf Dermatologie und Venerologie.
Beiträge zur Klinik.der Infektionskrankheiten und zur Immunitätsforschung. Bd.1. Heft.3. p.549.

1001. BRUCK, C. Weitere untersuchungen über das Wesen der arzneiexantheme.
Berlin Klin.Woch. 1910.Halbjahr 2. p.1928.

1002. BROCQ, L. Les eruptions eczématiformes provoquées par une teinture pour cheveux à base de chlorhydrate de paraphenylene diamine.
Le Bull. med. 1898. p.237.

1003. BUNCH, J. L. Paraphenylene diamine dermatitis.
B.J.D. 1915. p.348.

1004. BURKE, J. G. Dermatitis from Hair-dye.
J.A.M.A. 1909 p.528.

1005 BURRILL, J. T. Some vegetable poisons.
Amer. Monthly Microscopical Journal, 1882. Vol.3. p.192.

1006. " " Some vegetable poisons.
Amer. Naturalist. 1883. Vol.17 p. 319.

1007. " " Letter to Editor.
Garden & Forest, 1895. Vol.8. p.368.

1008. CASH, J. Th. Satinwood dermatitis.
Brit. Med.Journ. 1911. Oct.7.

1009. CATHELINEAU Note sur 18 cas d'accidents provoquées par une teinture pour cheveux à base de chlorhydrate de paraphenylene diamine.
Ann. de D.&.S. 1898 Vol.9.p.63.

1010. CHRISTIANSEN, J. Reaktioner par de giftige wenske Taendstikaesker.
Saertryk of Ugeskreft Laeger, 1918. No. 14.

1011. CLARKE, F. H. Eczema caused by Primula Obconica.
B.M.J. 1890 II. p.789.

1012. CLELAND, R. B. Rashes caused by the hairlets of caterpillars of the moth. Euproctis Edwardsi.
Med. Journ. Australia 1920. p.169.

1013. COOKE, J. V. The Etiology and treatment of Ammonia dermatitis of the gluteal region of infants.
Amer. Journ. Dis.Child. 1921 Vol. 22. p.481.

1014. CRIPPS, L. The properties of Tetryl (as affecting the human system).
B.J. D.&.S. 1917. Vol.29. p.3.

1015. CROCKER, RADCLIFFE, H. Diseases of the Skin. 1903 p.417.

1016. CURSON, H. H. Saria (Dermatitis of cattle).
Vet. Journ. 1920. Vol.76, p.405

1017. ~~DAKIN~~.
~~Am. J. Med.Sci. 1919? p.1829.~~

1018. DIFFENBACH, W. H. Treatment of Ivy poisoning.
Calif. Pract. 1917. Vol.32. p.91.

1019. DUNCAN, C. H. Autotherapy in Poison Ivy.
N.Y. Med. Journ. 1916 Vol.54 p. 901.

1019. EDITOR (Letter to) Eau sublime and Mrs. Potler's Walnut Tint Hair Stain.
J.A.M.A. 1910, p.1662.

1020. EDITORIAL A Poisonous hair-dye.
J.A.M.A. 1909. Vol.53. p.803

1021. FEILCHENFELD, L. Uber eine durch Handschuhfarbe ver-vorgerufene Hautentzundung.
Deutsch. Med.Woch.1909.p.2065.

1022. FERGUSON, J. The primula Obconica.
B.M.J. 1890 II. p.954.

1023. FOERSTER, O. H. Primula Dermatitis.
J.A.M.A. 1910 Vol.55. p.642.

1024. FORD, W. W. On the presence of hemolytic substances in edible fungi.
Journ. Infect.Dis. 1907. Vol.4. p.434.

1025. " " Antibodies to glucosides, with special reference to Rhus Toxicodendron.
Journ.Infect.Dis. 1907. Vol.4. p.541.

1026. " " Further observations on the Immunization of Animals to the Poisons in Fungi.
Journ. Phar. and exper.Therap. 1910. Vol.2. p.145.

1027. FOX, G. H. Match-box Dermatitis.
Journ. Cutan.Dis. 1918. Vol.36 p.530.

1028. FREI, W. Über Streichholzschachtel dermatitis.
Med.Klin. 1921. No. 16.

1029. FROST, L. C. The bacterial etiology of poison oak dermatitis (Rhus poisoning)
Med. Record.1916, Vol.90.p.121

1030. FUJITANI, J. Chemistry and Pharmacology of Insect Powder.
Arch. f. exper.path.u.Pharmakol 1909. Vol.61. p.47.

1031. FURST, E. Über die Veränderungen des epithels durch leichte warme und Kaltœimwirkungen beim menschen und säugetier.
Ziegler's Beiträge 1898. Bd. 24. p.415.

1032. GALEWSKY Über Eucalyptus dermatitis. Versammlung deutscher Naturforscher und Ärzte Breslau. Sept. 1904.
Dermat. Zeitsch. 1904. Bd.XI. p.752.

1033. GALEWSKY Über berufliche Formalin onychien und Dermatitiden.
Munch.Med.Woch. 1905. No.4. p.164.

1034. " Über Eucalyptus Dermatitiden.
Dermat. Zeitsch. 1905.Bd.12 p.36.

1035. GANS, Fall von Schweissleder Dermatitis.
Deutsch.Med.Woch.1920. Vol.46.
p.1044.

1036. GESSARD, M. C. Antilaccase.
Compt. rend. Soc.biol. 1903.
Vol.55. p.227 & 637.

1037. GREGORSON A.W. & TAYLOR, F.E. On Trinitrotoluene posioning, with records of five cases.
Glasgow. Med.Jour. 1918. p.65.

1038. HALL, A. J. Repeated attacks of Eczema produced by Phenyl hydrazin. Hydrochloride.
Brit. Journ.Dermat.1899. Vol.XI.
p. 112.

1039. " " The Skin and its reactions.
Lancet 1921 No. 1 p. 426.

1040. HANNAH, LOUIS Ragweed Dermatitis. New Treatment.
Journ. Amer.Med.Assoc. 1919.
Vol. 72. p. 853.

1041. HARRINGTON, W. W. & GOLD. J. D. A toxic hair-dye and its analysis.
J.A.M.A. 1909. Vol.52. p.1121.

1042. HELLIER J. B. A Vésicating dye.
B.M.J. 1892. II. p.1107.

1043. HOFFMANN, E. Über die Primelkrankheit und andere durch Pflanzen verursachte Hautentzundungen.
Munch.Med.Woch.1904. Vol.51.
p. 1966.

1044. HUBBARD Poison Ivy.
Med. Brief. 1904. p.884.

1045. JACQUET L. & JOURDANET P. Étude étiologique pathogénique et therepeutique des dermites professionelles des mains.
Annales de D. &. S. 1911 Vol.2.
p.11.

1046. JADASSOHN Immunität gegen Quécksilber.
Verhandlung der deutsch.dermat.
Gesellsch. Graz. 1895.

1047. JADASSOHN J. Zur Kenntiss der medicamentösen Dermatosen .
Verhandlg.der deutschen.dermat.
ges. V.congress. 1896.

1048. JANOWSKY Uber Iodoformexatheme.e.
Arch. f.Dermat.1884. p.495.

1049. JONES, H. E. Acute Dermatitis produced by Satinwood Irritation.
B.M.J. June 25, 1904.

1050. DE JONG, OSCAR. Dermatitis in Bakers.
B.M.J. 1923. p. 23.

1051. " " Aetiology of Dermatitis in Bakers.
Lancet, 1923. Vol.I. p.80.

1052. KALL Akute Dermatitis, hervorgerufen durch Tragen eines neuen Bergstockes.
Munch.Med.Woch.1920.Vol.67. p.1321.

1053. KANNGIESSER, F. Gartenflora, 1909.

" " Vergiftungen durch Pflanzen und Pflanzen stoffe.
Jena, 1910. p.40.

1054. " " Beitrage zur kenntnis der Primeldermatitis.
Korrespondenzblatt f.Schweizer Aertze. 1911. Vol.41. p.1041.

1055. KHITTEL, J. Wittstein's Vierteljahresschrift fur prakt. Pharmacie.
1858. p. 348.

1056. KNACK Uberempfindlichkeit gegen Kautschukheftpflaster.
Dermat.Woch. Bd.59. p.936.

1057. KNOWLES, F. C. Eczema of external origin and its relationship to Dermatitis (based on a study of 6453 cases classed as eczema)
Journ. Am.Med.Assoc. Vol.68. No. 2. p. 79.

1058. " " The external origin of eczema particularly the occupational eczemas, as based on a study of 4,142 cases.
Journ. Cutan. Dis. 1913. Vol. 31.p.11.

1059. KREIBICH C. Uber Nervose Uberempfindlichkeit der Haut.
Arch. f. D. &.S. 1908. Bd.93 p. 59.

1060. " " Uber lokale Unterempfindlichkeit der Haut.
Arch. f. D.&.S.1910 Vol.103. p. 133.

1061. KREIBICH, C. Weitere Beiträge zur abnormen Hautempfindlichkeit.
Arch. f. D.&.S. 1911. Vol.108. p.41.

1062. LABERNADIE Dermatitis due to wearing an M.L. mask.
Annales de D.&.S. 1918-19 p.90

1063. LAIN, E. S. Dermatitis Lycopersicum Esculentum (Tomato Plant)
J.A.M.A. 1918. Vol. 71. p. 1114.

1064. LANDOUZY E. & BROUARDELL G. Empoisonnements non professionelles par l'aniline.
Annales de Hygiene, Bd.44, p.137.

1065. LANE, C. GUY Occupational Dermatitis in Dentists; susceptibility to Procain.
Arch. of Dermat. and Syph. 1921 Vol. 3. p. 235.

1066. " " Occupational dermatitis in Dentists,
Lancet 1921 Vol. I. p.712.

1067. " " Dermatitis caused by oil of Citronella.
Arch. of D. &. S. 1922. Vol. 5. p.589.

1068. LEE, C. N. Eczema caused by Primula Obconica.
B.M.J. 1890. II. p. 790.

1069. LEGGE T. M. Industrial eczema. Industrial poisoning in manufacture of aeroplanes, explosives and dyes.
J. Industrial Hyg. 1920.Vol.2.p.121

1070. LELOIR, H. Dermite professionelle speciale (eczema des fileurs et varonleurs de lin).
Annales de D.&.S. 1885. Vol.6. p. 129.

1071. " " De la folliculité et perifolliculité des fileurs et Rattacheurs (Bouton d'huile)
Ann. de D.&.S. 1889. Vol.10. p.672.

1072. LOMBARDO A case of anaphylaxis to Scharlach R.
Giorn Ital. d.mal. ven. e. della pelle 1912, Bd. 53. Fasc. 1.

1073. LOTTRUP-ANDERSEN Atropine Idiosyncrasy in Ophthalmology.
Hospitalstidende, June 15, 1921.

1074. LOW, R. CRANSTON — Cutaneous Sensitisation. B.M.J. 1921. II. p.559.

1075. McCONNELL, W. J. — Industrial Dermatoses among Printers, U.S. Public Health Service. Pub. Health Reports, May 6.1921 Vol. 36. p.979.

1076. McCORD, C.P., KILKER, C.H. & MINSTER, D.K. — Pyrethrum Dermatitis. J.A.M.A. 1921. Vol.77. p.448.

1077. McDONNELL, C. C. — Insect Powder. Bull. 3824. U.S. Dept. Agricult. June 3, 1920.

1078. MACKAY, J. H. — Hair dye poisoning. J.A.M.A. 1909, Vol.52. p.1579.

1079. McLACHLAN, A. D. — A form of Industrial dermatitis Glasgow Med. Journ. Apr. 1922.

1080. MacLEOD, J. M. H. — Note on a case of acute dermatitis due to Cōpra. B.J.D. 1915, p.118.

1081. MACLEOD, J. M. H. — Dermatitis from handling German Bombs. B.M.J. July 21. p.80.

1082. McNAIR, J. B. — Pathology of Dermatitis Venenata from Rhus Diversiloba. Journ. Infect. D. Sept. 1916, p.419.

1083. " " — The Transmission of Rhus Poison from plant to person. Journ. Infect.Dis. 1916. Vol. 19. p.429.

1084. " " — The poisonous principle of poison oak. Journ. Amer. Chem.Soc. 1916. Vol. 38, p. 1417.

1085. " " — The oxidase of Rhus Diversiloba. Journ. Infect. Dis. 1917. Vol. 20. p. 485.

1086. " " — The Poisonous principle of Poison-oak, non-bacterial. Med. Record. 1917. Vol.91. p.1042.

1087. McNAIR, J. B. Pathology of Rhus Dermatitis.
Arch. of D.&.S. 1921. Vol. 3. p. 383.

1088. " " Lobinol - A determinant from Rhus Diversiloba (Poison Oak).
Journ. Am. Chem. Soc. Jan. 1921

1089. " " Susceptibility to dermatitis from Rhus Diversiloba.
Arch. of D.&.S. 1921. p.625.

1090. " " A contribution to the Chemiotherapy of Rhus Dermatitis and a tentative method of treatment.
Arch. of D.&.S. 1921. Vol. 3. p. 802.

1091. MAISCH, JOHN M. On the active principle of Rhus Toxicodendron.
Proc. Amer. Pharmaceutical Assoc. 1865. p.166.

1092. MARKLEY, A. J. Anaphylactoid dermatitis due to direct contact with animal epidermal structures.
Arch. Dermat. & Syph. 1920. Vol. 2. p.722.

1093. MEWBORN, A. D. A case of acute dermatitis caused by the use of a hair-dye having for its base the hydrochlorate of paraphenylene diamin.
J.A.M.A. 1901, Vol.36. p.1389.

1094. MONTGOMERY, D.W. & COLVER, G.D. Dermatitis caused by Primula poisoning.
California State Journ. Med. Aug. 1914.

1095. MUICHI, Uber Lackdermatitis.
Japanische Zeitsch. f. Derm. & urologie. 1915. p.888.

1096. MURRAY, F. ANDERSON, Dermatitis caused by Bitter Orange.
B.M.J. 1921 Vol.I. p.739.

1097. NEISSER, A. Ueber Iodoform exantheme
Deutsch. med. Woch. 1884. No. 30. p.467.

1098. NEISSER, Über Vitiligo mit Lichenoider eruption.
Verhandlung d. deutsch. dermat Gesellsch. IV Kongress, p.435.

1099. NESTLER, A. Hautreizende Primeln.
Berlin, 1904.

1100. NIXON Cotton-seed dermatitis.
Proc.R.Soc. London Derm. Sect. 1915. p.112.

1101. OLDACRES, C. E. Toxic symptoms produced by handling Primula Obconica.
B.M.J. 1889, II. p.719 & 1890 II. p.870.

1102. PAGE, CALVIN & BUSHNELL. Oil folliculitis.
Journ. Indust. Hygiene. 1921. No. 2. p. 62.

1103. PANTON, P. N. The effect of Trinitrotoluene upon the blood.
Lancet, 1917. Vol.II. p.81.

1104. PARDO-CASTELLO, V. Dermatitis Venenata. A study of the tropical plants producing dermatitis.
Arch. of D.&.S. 1923. p.81.

1105. PFAFF, F. On the active principle of Rhus Toxicodendron and Rhus Venenata.
Journ. Exper. Med. 1897. Vol.2. p. 181.

1106. PONTOPPIDAN. Hat-band Dermatitis. Ugeskrift for Laeger, Sept. 29. 1921.
Abstr. in B.M.J. Nov. 19, 1921.

1107. PUSEY, W. A. Lacquer dermatitis.
Arch. of D.&.S. 1923. p.91.

1108. RASCH, Om en Hudlidelse fremkadt of Taendstikaesker.
Saertryk of Ugeskrift for Laeger 1918. No. 7.

1109. REEB, E. Principes actifs de la poudre insecticide.
Journ. Pharm. Elsass Lothringen 1909. Vol. 35. p.267.

1110. REICHEL — Immunität gegen das Virus von Eiterkokken. Verhandlung der deutsch. Gesell. für chirurgie 1891 p.75.

1111. ROXBURGH, A. C.& CASTLE, W. F. — Dermatitis from dyed fur. B.M.J. 1923. I. p.534.

1112. RUXTON, W. L. — An investigation into the cause and prevention of industrial diseases due to Tetryl. B.J.D.&.S. 1917. Vol.29.p.18.

1113. SABOURAUD — Sur l'éruption artificielle due au contact de certaines espéces de primevères. La Clinique. 1908. Apr. 17.

1114. SAMUEL, S. — Über ein Art von Immunität nach überstandener Crotonentzündung. Virchow's Archiv. 1892.Bd.127.

1115. SACHS, O. — Klinische und Experimentelle Untersuchungen über die einwirkung von Anilinfarbstoffen auf die menschliche und tiersche Haut. Arch. f. D.&.S. 1913. Vol.116. p. 555.

1116. SACHS, — Acute dermatitis in artificial Amberworkers. Wien. Klin.Woch. 1921. July 21

1117. SCHAEFER, W. — Beiträge zum Klinischen Studium und der quantitiven Prüfung der Hautreaktion auf Chemische Reize. Über die chemische Hautreaktion bei peripheren und Zentralen Lähmungen. Arch. f. D.&.S. 1921. Vol.132. p. 87.

1118. SCHAER, G. — Versuche über die gewohnung des Kaninchenohres an entzündungserregende Mittel. Inaug.Dissert. Bern 1907.

1119. SCHAMBERG, J. F. — The desensitisation of persons against Ivy poison. Journ.Amer.Med.Assoc. 1919. Vol. lxxiii p.1213.

1120. SCHEMEL. Über Schweissleder verbrennung der Stirnhaut bei der Sicherherts polizei Königsberg.
Munch. Med. Woch. 1920. Vol.67 p. 700.

1121. SCHULTZ, J. H. Beiträge zum Klinischen Studium und der quantitativen Prüfung der Hautreaktion auf chemische Reize.
Arch. f. D.&.S. 1912. Vol.113 p.987.

1122. " " Die Prüfung der Hautreaktion auf chemische Reize.
Jahrb. f. Kinderh. 1913. Vol. 78. p. 347.

1123. SCHWALBE, On the active principle of Rhus diversiloba (Poison Oak)
Med. Record. N.Y. 1903.p.855.

1124. SEMON, H. C. Dermatitis from dyed fur.
B.M.J. 1923 Vol.I. p.467.

1125. " " Dermatitis and dyed fur.
B.M.J. 1923. I. p.613.

1126. SEQUEIRA, G. W. Dermatitis from handling German bombs.
B.M.J. Vol. 2. p.80.

1127. SEQUEIRA, J.H. Dermatitis due, to explosives used in Air-raids.
B.M.J. 1917, II. p.148.

1128. " " A case of bullous eruption caused by May weed.
Lancet, 1921 II. p.560.

1129. SHALEK, A. Dermatitis Venenata due to a proprietary hair-dye.
J.A.M.A. Vol. 52. 1909 p.557.

1130. SHEPARD, N. A. & KRALL, S. Poisons in the Rubber Industry.
The India Rubber World. 1919. Vol. 61. p.75.

1131. SHIE, M. D. Wound infection among lathe workers.
J.A.M.A. 1917. Vol.69. p.1927.

1132. SIEGHEIM Über Satinholzdermatitis.
Berlin Klin.Woch.1909. No.45. p. 2020.

1133. SIMPSON, C. AUGUSTUS. Primrose Dermatitis and its relation to Anaphylaxis.
J.A.M.A. Vol. lxix. July 14, 1917. p. 95.

1134. SÜLLNER Ein Fall von systematisierter Lichenifikation, als Beitrag zur kenntnis metamerischer Hautaffektionen.
Archiv. f. D.&.S. 1905. Bd.73 p. 147.

1135. SPILLMANN, M. L. Dermatitis Venenata caused by the oak.
Bull. de Derm. & Syph. 1921. p. 33.

1136. STEIN, R. Experimentelle und histologische Untersuchungen über Hautgewöhnung
Arch. f. D.&.S. 1909. Bd.97. p. 27.

1137. STRANDBERG, J. Investigations re the so-called match eczema and its causes.
Acta Dermato. Venereologica, 1920. Vol. I. p.116.
Abstr. B.J. D.&.S. 1921. p.274.

1138. STRANZ, H. Über Streichholzschachteldermatitis.
Munch.Med.Woch. 1921 Vol.68. p. 548.

1139. STRICKLER, A. Treatment of Dermatitis Venenata by vaccines.
J.A.M.A. Vol. 68. No.20. p.1503.

1140. " " The treatment of Dermatitis Venenata by vegetable Toxins.
Journ. Cutan.Dis. 1918. Vol.36 p.327.

1141. " " The Toxin treatment of Dermatitis Venenata.
J.A.M.A. 1921. p.910.

1142. SUTTON, R. L. Dermatitis Venenata.
J.A.M.A. 1918. Vol.71. p.1116.

1143. " " Ragweed Dermatitis.
Journ. Amer.Med.Assoc. 1919. lxxiii, p. 1433.

1144. " " Infectious eczematoid Dermatitis.
J.A.M.A. 1920. Vol.75. p.976.

1145. SYM, ALLAN C. Eczema caused by Primula Obconica.
B.M.J. 1890. II. p.686.

1146. SYME, Some constituents of the Poison Ivy (Rhus Intoxicodendron)
John Hopkins Thesis, 1906.

1147. SYME, W. A. Some constituents of the Poison Ivy Plant.
Johns Hopkin. Univ. Bull. 1906.

1148. THIBIERGE, G. Primula dermatitis.
Bulletin Med. 1911. No. 33.

1149. " " Dermite à type dyshidrosique provoquée par le contact de l'explosif d'une bombe d'avion allemande.
Annales. de D.&.S. 1919. Tome 7. p. 131.

1150. TIMPANO, Reed Dermatitis.
Il Policlinico 1921 p.6. Abstr. B.M.J. 1921.

1151. TISSOT, G. Des teintures pour les cheveux, de leurs dangers, étude historique clinique et medicolegale.
Thèse de Paris, 1898.

1152. TOYAMA, J. Rhus Dermatitis.
Journ. Cutan D. 1918. Vol.36. p. 157.

1153. TURRO, R. & DOMINGO, P. The Nature of Local Immunity.
Compt. Rendu.Soc. Biol. 1923. p. 410.
Abstr. B.M.J. 1923. Apr. 7.

1154. TYSON, J. W. Dermatitis from explosives used in Air raids.
B.M.J. 1917. Vol.II. p. 45.

1155. WALKER, I. C. Causation of eczema, urticaria and angio-neurotic oedema.
J.A.M.A. 1918. Vol.70. p.897.

1156. WALSH, D. Dermatitis among flower pickers in the Scilly Isles. The so-called "Lily-rash".
B.M.J. 1910. Vol.2. p. 854.

1157. WARREN, L. E. The poisonous principle of Rhus.
Am. Journ. Pharm. 1913. p. 85.
531 & 562. also Pharm. Journ. London, 1909. Vol. 29. pp.
1158. WASHBURN, R. G. Dermatitis due to Tomato.
J.A.M.A. 1918. Vol. 71. p.1116.

1159. WAUGH, F. Dermat. Venenata. Discussion on Lain's papers.
J.A.M.A. 1918. Vol. 71. p.1116.

1160. WECHSELMANN. Über Satinholzdermatitis, eine Anaphylaxie der Haut.
Deutsch. Med.Woch. 1909. No.32. p. 1389.

1161. WEISS, LUDWIG Artificial Dermatitis.
J.A.M.A. 1912. Vol.58.p. 2026.

1162. WELANDER. Sur un cas de Vitiligo, de Lichen ruber planus et de Nevrodermite chronique circonscrite.
Annales. de D.&.S. 1894. p.645.

1163. WERNER, R. Experimentelle epithelstudien - über Wachstum, Regeneration, Amitosen, und Riesenzellenbildungen des epithels
Bruhns Beiträge zur Klin. Chirurgie.1902. Bd. 34.

1164. " " Zur lokalen Sensibilisierung und Immunisierung der gewebe gegen die Wirkung der Radiumstrahlen.
Deutsch. Med.Woch. 1905. No. 27 & 28.

1165. WHITE, J. C. Notes on Dermatitis Venenata.
Boston Med. and Surg.Journ. 1897. Vol. 136. p. 77.

1166. WHITE, JAMES Dermatitis Venenata.
Boston 1887. p. 43.

1167. WHITE, JAMES C.
Garden & Forest, May 3, 1888.

1168. WHITE, C. J. Match-box dermatitis.
Journ. Cutan.Dis. 1919 Vol.37. p.125.

1169. WHITFIELD, A. Dermatitis Traumatica et Venenata.
Encyclopedia Medica. Vol.3. p. 417.

1170. WHITFIELD, A. Dermatitis Traumatica.
Sect.Skin.Dis. Bain's Textbook of Med. p.483.

1171. WILSON, ERASMUS. Inflammations of the skin from Anilin dyes.
Journ. Cutan.D. 1869 April.

1172. WOOD, W. W., HANNUM, E. A. & EWAN, E. N. Poisoning from Mrs. Potter's Walnut juice hair stain.
J.A.M.A. 1909. Vol.53.p.809.

ECZEMA.

1173. BENDER, E., BOCKHART, M., & GERLACH, V. Experimentelle Untersuchungen über die Ätiologie des Ekzems.
Monatsheft f. prakt.Dermat. 1901. Bd. 33 p. 149.

1174. BERGER, H. C. The relation of anaphylaxis to asthma and eczema.
J. Missouri M.A, 1920. Vol.17. p. 109.

1175. BOCKHART, M. Untersuchungen über das Staphylotoxin Ekzem.
Monatsheft f. prakt.Derm. 1901 Bd. 32. p.421.

1176. BROCQ & VEILLON Discussion sur l'origine parasitaire des eczémas.
Annales de D.&.S. 1900. p.969.

1177. BRUCK, C. & HIDAKA, S. Biologische Untersuchungen über die Rolle der staphylokokken bei ekzemen.
Arch. f. D.&.S. 1910. Vol.100 p. 165.

1178. CHIPMAN, E. D. Streptococcic Dermatoses.
Arch. of D.&.S. 1921 No.4. p. 534.

1179. COENEN
Beiträge zur Klin.Chir. Bd.60.

1180. COLE, H. N. Bakteriologische, histologische und experimentelle Beiträge zur Kenntnis der Ekzeme und der Pyodermien.
Arch. f. D.&.S. 1913. Vol.116 p. 207.

1181. DOHI, K. & DOHI, SH. Zur klinik und aetiologie der Impetigo contagiosa.
Arch. f. D.&S. 1912 Bd.71. p. 629.

1182. EDELMAN, M. H. Eczema due to deficient thyroid secretion.
New York Med.Journ. 1918. p.450.

1183. FEER — Über plotzliche Todesfälle im kindersalter, insbesondere über dem ekzemtod.
Schweiz, Korr. bl. 1904. No.1.

1184. FISCHER, L. — Acute Eczema due to faulty metabolism of food elements.
New York Med.Journ. 1918. Vol. 108. p.804.

1185. FORDYCE, J.A. — Infective eczematoid Dermatitis - possible influence of Anaphylaxis in Skin Reactions.
Journ. Cutan.Dis. 1911. Vol.29. p.129.

1186. FRÉDÉRIC, J. — Zur Ekzemfrage.
Munch,Med. Woch. 1901. No. 38.

1187. GORDON, A. — Case of chronic eczema cured by vaccines.
Semana Medica 1921. Vol.28. p. 136.

1188. HAHN, H. — Die Durchlässigkeit des magen-Darmkanales ernährungsgestörter Säuglinge für ein heterologes Eiweiss gebundendes Antitoxin.
Jahrb. f. Kinderh. 1913. Vol. 77. p. 405.

1189. HALL, A. J. — An inquiry into the etiology of Infantile Eczema.
Brit.Journ. Dermat. 1905. Vol. 17. pp. 161,203, 247 & 287.

1190. HARRIS, F. G. — The etiology of eczema with a preliminary report of experimental studies.
Arch. of D.&.S. 1921. p.579.

1191. HAZEN, HENRY H. — The etiology of Eczema.
Arch. of. Derm.& Syph. Vol. 38. No. 6. June 1920. p.642.

1192. HEIMANN, W. J. — The Pathology and Pathogenesis of Eczema, and Dermatitis.
Journ.Amer.Med.Assoc. Vol.68. No. 2 p.75.

1193. " " — A critical Review of Eczema and Dermatitis with an analysis of a group of cases.
Journ. Cut. Dis. 1916 Vol.34. p. 259.

1194. JADASSOHN, J. Discussion sur l'origine parasitaire des eczemas.
Annales. de D.&.S. 1900. Vol.1. p. 963.

1195. JAEGER, H. De la nature de l'eczema.
Annales de D.&.S. 1923. pp. 10 & 109.

1196. JOHNSTON, J. C. Speculations as to the causation of eczema.
Journ.Cut.Dis. Vol. 31. 1913. p. 3.

1197. JOHNSTON, J.C. & SCHWARTZ, H.J. Studies in the metabolism of certain skin disorders.
New York Med.Journ. 1909. Vol. 89. pp. 535, 590 & 636.

1198. KERLY, C. G. Eczema in Infants and Young Children
N.Y. State Med.J. 1916. Vol. 14. p. 523.

1199. KREIBICH, K. Zur Eiterung der Haut.
Festschrift fur Kaposi. 1900. p. 447.

1200. LESNE & DREYFUS L'anaphylaxie alimentaire.
Ann. de med. et chir. 1912. Vol. 16. No. 22.

1201. LEWANDOWSKY, F. Uber Impetigo contagiosa s. vulgaris nebst, Beiträgen zur kenntnis der staphylo und streptokokken bei Hautkrankheiten.
Arch. f.D.&.S. 1909. Bd. 94. p. 163.

1202. LOIZAGA, N. S. Vaccine treatment of eczema.
Semana medica Argentina, 1921. Vol. 28. p. 89.

1203. MEDALIA, L. S. The treatment of eczema with special reference to the use of vaccines and the part played by bacteria in its etiology.
Boston Med. and Surg.Journal. 1915. Vol. 173. No.6. p. 187.

1204. MORO, E. Ubererregbarkeit des vegetativen Nervensytems im Frühjahr und Ekzemtod.
Munch.Med.Woch. 1920. Vol. 67. p. 657.

1205. MORRIS, MALCOLM Diseases of skin. 6th Edit.

1206. MORROW Discussion on Focal Infection. Journ.Cutan.Dis.1917. Vol. 35. p. 657.

1207. MORSE, J. L. Constipation and eczema in an infant from an excess of fat in modified milk. M. Clinics. N. Amer. 1920. Vol. 4. p. 585.

1208. NEISSER. THe cause of eczema. Deutsch. derm. gesell. Discussion 1892.

1209. NEISSER M. & LIPSTEIN A. Die Staphylokokken. Kolle & Wassermann Handbuch der path. mikroorganismen. II Auflage. Bd. IV. p. 10.

1210. PETRINI, Discussion on Eczema. Trans. 4th Internat. Cong. Dermat. Paris 1900. p. 63.

1211. RAJKA, E. Untersuchungen über die Ätiologie des Ekzems. Die Rolle der pyogenen mikroorganismen bei der entstehung des ekzemas. Arch. f. D.&.S. 1922. Bd. 141. p. 32.

1212. SCHULTZ, J. H. Die Prüfung der Hautreaktion auf chemische Reize. Jarhbuch f. Kinderhk. 1913. Vol. 78 p. 347.

1213. SMITH, J. FERGUSON Some points in the aetiology of eczema. Glasgow.Med.Journ. 1923. p.160.

1214. SPIESS, G. Die Bedeutung der Anästhesie in der Entzundungstherapie. Munch.Med.Woch. 1906. Vol.53. p. 345.

1215. TALBOT, F. B. & TOWLE, H.P. Infantile eczema and Indigestion. A. Journ.D.Child. 1912.Sept. p. 219.

1216. TORUK Discussion on Eczema. Annales. de dermat. et syph. Vol. 139. 1900.

1217. UNNA, P. G. Eczema and Dermatitis.
Derm. Woch. 1921. p,233.

1218. WALKER, C. Causation of eczema, urticaria and angioneurotic oedema by proteins other than those derived from the food.
J.A.M.A. Vol. 70. 1918. p.897.

1219. WEIDENFELDT, S. Beiträge zur Pathogenese des ekzemas.
Arch. f. D.&.S. 1912 Vol.III.
p. 891.

1220. WHITE, J. C. Two modern methods to be employed in the treatment of chronic eczema.
J.A.M.A. No. 68. p. 81.

ABSORPTION OF PROTEIN BY BOWEL.

1221. VAN ALSTYNE — The absorption of Proteins without digestion.
Archives. Int.Med. 1913 Oct.

1222. VAN ALSTYNE E. & GRANT, J.P. The absorption of Albumin without digestion.
Journ. Med. Research. 1911. Vol.25. p. 399.

1223. ANDERSON, Y.F. & FROST, W.H. Studies upon anaphylaxis with special reference to the antibodies concerned.
Tr. Amer.Cong.Phys. & Surg. 1910. Vol.8. p. 414.

1224. ASCOLI, M. — Über den mechanismus der Albuminurie durch Eiereiweiss.
Munch.Med.Woch. 1902. Vol. 49. p. 398.

1225. ASCOLI M. & VIGANO L. Zur kenntnis der Resorption der Eiweisskörper.
Zeitschr. f. physiol.Chem.1903. Vol. 39 p. 283.

1226. BARNATHON — De l'anaphylaxie alimentaire.
Thèse de Paris 1911.

1227. BOUTEIL, — Des voies d'introduction des substances anaphylactisantes.
Thèse de Paris 1910.

1228. CANNON, W. B. — Recent advances in the physiology of the digestive organs.
Am. J. Med.Sc. 1906. Vol. 131. p. 563.

1229. FALLS, F. H. — Proteolytic Ferments in Portal Blood.
J.A.M.A. 1915. Vol. 65. p. 524.

1230. GANGHOFER & LANGER Über die Resorption genuiner Eiweisskorper im magendarmkanal neugeborener Tiere und Säuglinge.
Munch.Med.Woch. 1904. Vol.51. p. 1497.

1231. GREER, V. D. — The intradermal reactions to Proteins of infants suffering from gastro-intestinal disorders.
Arch.Pediat. 1917. Vol.34.p.810.

1232. GRULEE, C.G. & BONAR, B.E. Precipitins to egg-white in the urine of new born infants. Amer.Journ.Dis.Child. 1921. Vol.21 p. 89.

1233. HAMBURGER F. & SPERK, B. Biologische Untersuchungen über Eiweissresorption vom Darm aus. Wien.klin.Woch. 1904. Vol.17 p. 641.

1234. HAYASHI, J. UBer die Durchlaëssigkeit des Saüglingsdarmes für artfremdes Eiweiss und Doppelzucker. Monatsch. f. Kinderh. 1914. Vol. 12. p. 749.

1235. HOOBLER, B. R. Some early symptoms suggesting Protein sensitisation in Infancy. Am.Journ.Dis.Child. 1916. Vol. 12 p. 129.

1236. HOWELL, W. H. Note upon the presence of Amido acids in the blood and lymph as determined by the B. naphthalinsulphochloride reaction. Am.Journ. Physiol. 1906. Vol. 7 p. 273.

1237. HUTINEL, Intolérance pour le lait. La clinique, Apr. 1908.

1238. LAWATSCHEK Die enterale Resorption von genuinen Eiweiss bei Neugeborenen und Darmkranken Saüglingen. Prag. Med.Woch. 1914. Vol.39. p. 185.

1239. LUST. Die Durchlässigkeit des magendarmkanales für heterologes Eiweiss bei ernährungsgestörten Saüglingen. Jahrb. f. Kinderh. 1913. Vol.77 pp. 244 & 283.

1240. MAYERHOFER, E. & PRIBAM, E. Das Verhalten der Darmwand als osmotische membran bei akuter und chronischer, enteritis. Wien.Klin.Woch. 1909. Vol. 22. p. 875.

1241. MODIGLIANI & BENINI Permeability of Intestinal Tract of Infants for Casein of Cow's Milk. Il. Policlinico. Dec. 1915.

1242. MORO, E. Kuhmilchpräzipitin im Blute eines 4½ monate alten atrophikers. Munch.Med.Woch. 1906. Vol. 53. p. 214.

1243. NATHAN, M. Anaphylaxis from Pancreas Insufficiency. Bulletin Medical. 1920. Vol. 34. p. 59. Abstr. J.A.M.A. Vol. 74. 1920. p. 831.

1244. NEISSER, M. & WECHSBERG, F. Über das staphylotoxin Zeitsch. f. Hyg. 1901. Bd. 36. p. 299.

1245. NOBECOURT, P. Mortalite des lapins soumis a des injections de blanc d'oeuf. Compt. rend.Soc. biol. 1909. Vol. 66. p. 850.

1246. RICHET, Alimentary Anaphylaxis. Trans.Internat.Congress of Med. London 1913.

1247. ROSENAU & ANDERSON Further studies upon Anaphylaxis. Bull.Hyg.Lab. U.S. P.H.S. 1908 No. 45.

1248. SCHLOSS, O.M. & WORTHEN, T.W. The permeability of the gastro-enteric tract of Infants to Undigested protein. Am.Journ.Dis.Child. 1916. Vol. II. p. 342.

1249. STARLING, Principles of Human Physiology. 3rd Edit.

1250. TALBOT, F. B. Idiosyncrasy to cow's milk; its relation to Anaphylaxis. Boston Med. and Surg.J. 1916. Vol. 175 p. 409.

1251. UHLENHUTH, Neuer Beitrag zur spezifischen Nachweis von Eiereiweiss auf biologischeh Wege. Deutsch.Med.Woch. 1900. Vol. 26. p. 734.

1252. UFFENHEIMER, A. Zur Frage der intestinalen Eiweiss Resorption.
Jahrb. f. Kinderh. 1906.
Vol. 74. p. 383.

1253. VAUGHAN, CUMMINGS & McGLUMPHY. The parenteral Introduction of Proteins.
Zeitsch. f. Immunitätsf. 1911
Vol. 9. p. 16.

1254. WELLS The Biological Reactions of the Vegetable Proteins.
Journ. Infect.Dis. 1911. Vol.8.
p. 66.

1255. WELLS, H.G. & OSBORNE, T.B. The biologic reactions of the vegetable proteins.
J. Infect.Dis. 1915. Vol. 17
p. 259.

DERMATITIS SEBORRHOEICA.

1256. AUDRY, Séborrhée - Séborrhéides.
La Prat. Derm. 1904. Tome IV. p. 270.

1257. BARBER, H.W. & SEMON, H.C. The etiology and treatment of Seborrhoeic eruptions.
B.M.J. 1918. Vol. II p. 245.

1258. BEATTY, WALLACE, Seborrhoea.
B.J.D. 1894. Vol.6. p. 161.

1259. BROCQ, Les eczémas séborrhéiques ou seborrhéides.
La presse méd. 1897. p. 162.

1260. BROOKE, H. G. The clinical relationship of seborrhoea.
B.J.D. 1904. Vol. 16 p. 205.

1261. CUMSTON, C. S. Seborrhoea of the scalp.
N.Y. Med.Journ. 1921. Vol.113. p. 156.

1262. DARIER, Le groupe des maladies dites séborrheique. La Kérose.
Ann. de D.&.S. 1907. p. 3.

1263. DUHRING, Seborrhoea corporis.
Cutaneous Medicine. Part II.

1264. FOX, COLCOTT. Discussion on Seborrhoea.
B.M.J. 1901. II. p. 855.

1265. GARDINER, F. Eczema or Seborrhoeic dermatitis in children.
Practitioner, 1920. Vol. 105. p. 47.

1266. HALLÉ & CIVATTE The bacteriology of the Sebaceous glands.
Annales de D.&.S. 1907. p.184.

1267. KUZNITSKY, Experimentelle and Klinische Beiträge zur Frage der Hauttalgsekretion.
Arch. f. D.&.S. 1913. Vol.114. p. 691.

1268. MacLEOD, J. M. H. Seborrhoea and the seborrhoides. Practitioner 1904. p. 755.

1269. MONTGOMERY, D. W. The relation of diets to seborrhoea. Journ. cutan.Dis. 1916. Vol. 34. p. 829.

1270. MUIR & RITCHIE Manual of Bacteriology, 1919, p. 201.

1271. PRINGLE, J. J. Seborrhoea corporis. B.J.D. 1896. Vol. 8. p. 482.

1272. " " A rare Seborrhoide of the face. B.J.D. 1903. Vol. 15. p. 41.

1273. ROBERTS, LESLIE, Recent Researches on Seborrhoea and its consequences. B.J.D. 1897. Vol.9. p. 219.

1274. SABOURAUD, R. Seborrhoea. B.J.D. 1901. Vol. 13. p. 390.

1275. " " La question des séborrhéides. La Presse méd. 1904? pp. 353 & 393.

1276. " " Les maladies séborrhéiques. 1902.

1277. SOUTHWORTH, T.S. Predominence of seborrhoeic eczema in early life. Arch. Pediat. 1920. Vol. 37. p. 338.

1278. UNNA, P. G. Das seborrhoische Ekzem. Volkmann's Klin Vortrage 1893. No. 79. p. 669.

1279. " " Histopathology of the Skin.

1280. " " Was wissen wir von der Seborrhöe? Monatshefte f. prakt. D. 1887 Vol. 6. pp. 698 & 739.

1281. " " Function of the sweat glands in man. B.J.D. 1894. Vol. 6. p. 257.

1282. " " Das seborrheoische eczem. Das Petaloid. Munch.Med.Woch. 1921. Vol.68. p. 547.

1283. SCHWARTZ, LEVIN & MAHNKEN, The alkali reserve in various diseases of the skin. Journ.cutan.Dis. 1919 Vol.38. p. 9.

1284. SWEITZER, S.E. & MICHELSON, H.W. Acidosis in Skin Diseases.
Arch. of D.&.S. 1920. Vol. II. p. 61.

DERM. EXFOLIATIVA.

1285. POLLARD, R. Uber die Beziehung gewisser Formen exfoliativer Erythrodermien zur Tuberkulose.
Dermat. Zeitsch. Bd. 21. p.665.

PSORIASIS.

1286. ALSTYNE, G. V. N. The protein treatment of Psoriasis
New York Med. Journ. 1918. p.326.

1287. LANE, J. E. A case of Post-vaccinal Psoriasis.
Journ. Cutan D. 1916. Vol. 34.
p. 201.

1288. PROWAZEK, S. Von. Notiz zur aetiologie der Psoriasis
Vulg.
Central blatt. f. Bakt. 1912.
Bd. 62. Heft. 1 & 2.

1289. SELLEI, J. Überempfindlichkeit bei Psoriasis
Vulgaris.
Wiener Klin. Woch. 1909. Vol.
22. pp. 1183 & 1216.

ACNE.

1290. STRICKLER, A, KOLMER, J. A., & SCHAMBERG, J. F.
Complement fixation in acne vulgaris
Journ. of Cutan. Dis. 1916.
Vol. 34. p. 166.

1291. SELLEI, J. Uberempfindlichkeit bei Psoriasis Vulgaris.
Wienen,Klin.Woch. 1909. No. 35.
p. 1216.

STAPHYLOCOCCAL INFECTIONS,

BOILS, SYCOSIS, ETC.

1292. ALTMANN, K. & BLUHDORN, C. Komplementbinding bei Staphylokokken und Sarcinen.
Zentralbl. f. Bakt. Abt. I. origin Bd. 57. p. 87.

1293. BECK, S. C. Heilversuche mit der lokalen Immunisierung der Haut nach von Wassermann.
Med. Klinik. 1912. No. 22.

1294. BESREDKA, A. Local Immunity to Staphylococcal Infection.
C.R. Soc. Biol. 1923. p. 1273.

1295. LEDERMANN & WASSERMANN, Über einen Versuch, die lokale Immunitat für die Praxis brauchbar zur machen.
Med. Klinik. 1911. No. 13.

1296. SCHREUS, H. T. & GOEHL, E. Über lichenoide Eruption bei Pyodermic (Lichen pyodermicus) nebst Bemerkungen über Komplement ablenkung im Blute bei staphylokokken und Trichophytie - Erkrankungen mit Berücksichtigung der Einwirkung von Röntgenstrahlen.
Dermatolog. Zeitsch. 1920. p. 273.

GONORRHOEA AND SOFTSORE.

1297. BRANDWEINER & HOCH Mitteilungen über gonorrhoe.
Wien.Klin.Woch. 1913. No.22.

1298. BRUCK, C. Uber spezifische Behandlung gonorrhoeischer Prozesse.
Deutsch.Med.Woch. 1909 No. II.

1299. BUSCHKE, A. Uber exantheme bei gonorrhoe.
Arch. f. D.&.S. 1899. Vol.48. p. 181.

1300. CHAUFFARD & FIESSINGER, Deux cas de keratose blenorragique. Reproduction expérimentelle de la lésion cutanée.
Bull Soc. franc. de dermat. 1909. p. 162.

1301. DECASTRO, Beitrag zur Bestimmung des diagnostischen Wertes der Antigonokokkenvakzine.
Gaz. internat. de med. et.Chir. 1914. No. 10.
Abstr. Derm.Woch. 1915. No. 31 p. 762.

1302. DWIETRIEW, Die Hautreaktion beim Trepper.
Ref. Derm.Woch. 1914. p. 558.

1303. EISING, The disgnostic use of gonococcus vaccine for gonorrhoeal infections.
Med.Record. 1912. June 1.

1304. FINKELSTEIN & GERSCHUN Zur Serologie der blenorrh. Erkrankungen.
Berlin Klin.Woch. 1913. No.39.

1305. FUCHS, H. Hautallergie bei gonorrhoe.
Arch. f. D.&.S. 1917. Vol. 123. p. 331.

1306. GIORGIS, Intradermal Reaction in gonorrhoea.
Gazz. degli. osped. 1912. p.28.

1307. HECHT, H. Gonorrhoisches exanthem vershciedender gestalt und Tendovaginitis beim einem Fall.
Arch. f. D.&.S. 1916. Vol.123 p. 392.

1308. HODARA, OSMAN, IZET & CHEVKIET. Ein Fall von Gonokokkamie und generalisiertem gonorrhoeischem exanthem.
Dermat.Woch.1912. Bd.54 p.397.

1309. IRONS, E. E. Cutaneous allergy in gonococcal Infections.
Journ.Infect.D. 1912. Vol.II. p. 77.

" " A cutireaction in gonorrhoeal Infections.
J.A.M.A. 1912. p. 931.

1310. KÖHLER, Vakzinediagnostik und Therapie bei gonorrhoeischen affektionen.
Wien.Klin.Woch. 1911. No. 45.

1311. LEES, D. Keratodermia Blenorrhagica.
Edin.Med.Journ. 1922. Vol.28. p. 99.

1312. LONDON, La vaccination gonococcique en tout que guide du diagnostic et du traitement.
Journ. d'orolog. 1913. Vol. 3. p. 279;

1313. McDONAGH, Keratodermia Blenorrhagica.
Venereal Diseases p. 335.

1314. SAKAGUCHI. Y. & WATABIKI, Ch. Kutane Reaktion bei Gonorrhöekranken.
Dermat.Woch. 1912. Bd. 54. p. 717.

1315. SIMON, Über arthigonbehandlung der gonorrhoe.
Munch.Med.Woch. 1912. No. 10.

1316. SOMMER, A. Die biologische Diagnose der Gonorrhoe.
Archiv. f. D.&.S. 1913-14. originale Vol. 118. p. 583.

1317. SUTTER, E. Über gonokokken allgemeininfektion.
Zeitsch. f. Klin.Med. Bd. 86. Heft. 1 & 2.

1318. VERSHININ, Dermatoses due to gonorrhoeal infection.
Russk. Jurkozhn; ven. boliezn. 1914. No. 5.
Referred to in J.A.M.A. 1916. Vol. 67. p. 430.

FOCAL INFECTION.

1319. CHIPMAN, E. D. Focal Infection in the Etiology of Skin Disease.
Journ.Cut.Dis. 1917. Vol. 35. p. 647.

1320. " " The Etiology of Lichen planus.
Journ.Amer.Med.Assoc. 1918. Vol. 71. p. 1276.

1321. DAVIS, R. H. Discussion on Focal Infection.
Journ.Cutan.Dis. 1917. Vol. 35. p. 655.

1322. ENGMAN, Discussion on Focal Infection.
Journ.Cutan.Dis. 1917. Vol. 35. p. 656.

1323. HEIMANN, W. J. Discussion on Focal Infection.
Journ.Cutan.Dis. 1917. Vol. 35. p. 657.

1324. LESLIE, ROBERTS H. Focal Infection.
B.J.D.&.S. 1921. pp. 319 & 353.

1325. " " Focal Infection in relation to the Etiology of skin diseases.
B.M.J. 1922. p. 262.

1326. MACKEE, Discussion on Focal Infection.
Journ. Cutan.Dis. 1917. Vol. 35. p. 654.

1327. NOVITZKY, JOSEF. Dead Teeth.
New York Med.Journ. 1918. Vol. 107. p. 548.

1328. McGOWAN, J.P. Investigation into the disease of sheep called "Scrapie".
1914. Wm. Blackwood & Sons.

1329. RAVITCH, M.L. & STEINBERG, S.A. Relationship of Focal Infections to certain Dermatoses. Further observation.
Journ.Amer.Med.Assoc. 1918. lxxi. No. 16. p. 1273.

1330. SCHAMBERG, Discussion on Focal Infection.
Journ.CutanD. 1917. Vol. 35. p. 655.

1331. WILLCOX, Sir Wm. The clinical, pathological and radiological aspects of infection of the teeth and gums.
B.M.J. 1923. p. 53.

LUPUS ERYTHEMATOSUS.

/332. BARBER, H. W. A case of acute Lupus erythematosus. British Journ.Dermat. 1915. Vol. 27. pp. 319 & 365.

/333. " " A case of Lupus erythematosus associated with streptococcal infection of the Tonsils. B.J.D. 1919. p. 186.

/334. HARTZELL, M. B. Lupus erythematosus and focal infection. Arch. f. D.&.S. 1920. Vol.2. p. 441.

/335. LOW, R. CRANSTON, LOGAN, W.R. & RUTHERFORD, A. A fatal case of Lupus erythematosus with Autopsy. B.J.D. 1920. Vol. 32. p. 253.

/336. LOW, R. CRANSTON, & RUTHERFORD, A. Post-mortem report on a case of Lupus erythematosus. B.J.D.&.S. 1920. p. 326.

ALOPECIA AREATA.

1337. BARBER, H. W. & ZAMORA, A. M. Alopecia areata with a note on the estimation of the pathogenicity of the Tonsil. B.J.D.&.S. 1921. p.1.

1338. WHITFIELD, A. A contribution towards the etiology of Alopecia areata. The Lancet, 1904. Vol. 1. p. 651.

LICHEN PLANUS AND PRURIGO & PRURITUS.

1339. PERNET, G. A case of acute Lichen planus. B.J.D. 1913. p. 261.

1340. " " Note sur le lichen plan aigu et la ponction lombaire. Annales de D.&.S. 1913.p.461.

1341. " " Nouvelle Note sur le lichen plan aigu et la ponction lombaire. Annales de D.&.S. 1916. Vol. VI. p. 257.

1342. SUTTON, R. L. Discussion on Focal Infection. Journ.Cutan.Dis. 1917. VOL. 35. p.653.

1343. WELANDER, Sur un cas de vitiligo, de Lichen ruber planus et de Nevrodermite chronique circonscrite. Annales de D.&.S. 1894.p.645.

1344. THIBIERGE, G. & RAVAUT, P. Influence de le ponction lombaire sur le prurit du lichen de Wilson. Annales de D.&.S. 1905. Vol.6. p. 890.

1345. SCHWARTZ, H. J. Studies in the metabolism of Dermatitis Herpetiformis and Prurigo; their relation to Anaphylaxis. Journ.Cut.Dis. 1913. Vol.31. p. 994.

1346. MURRAY, D.H. Pruritus ani, its probable cause and subsequent treatment. J.A.M.A. 1911. No. 24.

1347. WINFIELD. The infective origin of Ano-genital Pruritus. Archiv. D.&.S. Nov. 1921.

LIGHT HYPERSENSITIVENESS, HYDROA VACCINIFORME.

1348. BARONI, V. & JONESCO, C. Destruction par les rayons ultra violettes de la propr. anti-sensitilisine du serum de cheval. Compt. rendu. hebd. Soc. biol. 1910 Vol. 69. p. 273.

1349. DOERR & MOLDOVAN, Die Wirkung des ultra-violet Lichtes auf das Eiweissantigen und seinen Antikorper. Wien.Klin.Woch. 1911. p. 555.

1350. EHRMANN, S. Weitere Untersuchungen über Lichtwirkung bei Hydroa aestivalis (Bazin) Summereruption (Nach Hutchinson). Arch. f. D.&.S.1909. Bd. 97.

1351. FISCHER, BARTHOLOMÄUS & RÖSE. Zur kenntnis der Porphyrinbildung. Zeitsch f. phys. chemie. 1913. Bd. 84. p. 262.

1352. GARROD, Sir. A. E. Haemato-porphyrinuria. B.M.J. 1922. March 11.

1353. GÖTZL, A. Beiträg zur kenntnis der Haematoporphorinuria bei der Bleivergiftung. Wien.Klin.Woch. 1911. Vol. 24. p. 1727.

1354. GROSZ, Ein Fall von Hydroa vacciniformis Dermat.Woch. 1913. Bd. 57. p. 1448.

1355. GUNTHER, H. Die Haematoporphyrie. Deut. Arch. f. Klin.Med. 1912. Bd. 105. p. 89.

1356. " " Clinical symptoms of Light-hypersensitiveness. Derm.Woch. 1919. Vol. 68. pp. 178, 203, 213, 230, and 243.

1357. HAUSMANN, Die sensibilisierande wirkung tierscher Farbstoffe und ihre physiologische Bedeutung. Wien.Klin.Woch. 1908. Vol. 21. p. 1527.

1358. HAUSMANN, H.
Jahrbücher f. Wissenschaftl. Botanik. 1909

1359. " " Die sensibilisierende wirkung des Hëmatoporphyrins.
Biochem. Zeitschrift. 1911. Bd. 30. p. 276.

1360. HERXHEIMER, K. & NATHAN, E. Über sensibilisierung der Haut durch Carboneol gegenüber Sonnenlicht und eine dadurch bedingte Dermatitis Solaris.
Dermat. Zeitsch. 1917. Vol. 24. p. 385.

1361 HUTYRA, & MAREK Spezielle Pathologie und Therapie der Haustiere.
1906. Vol. II. p. 828.

1362. KANOKI, Ch. Hydroa vacciniforme.
J.A.M.A. 1907. Vol. 49. p. 1774.

1363. KONIGSTEIN & Hess, Zur klinik und Atiologie einer bisher nicht beobacteten Form von Hautgangren.
Dermat. Zeitschr. 1910. Bd. 17. p. 911.

1364. MARTENSTEIN, H. Experimentelle Untersuchungen bei Hydroa vacciniforme.
Arch. f. D.&.S. 1922. Bd. 140. p. 301.

1365. MEYER - BETZ, F. Untersuchungen über die biologische (photodynamische) Wirkung des Haematoporphyrins und andere Derivative des Blut und gallenfarbstoffs
Deutsch.Arch.f.Klin.Med. 1913. Bd. 112, p. 476.

1366. NEUBAUER, A. Haematoporphyrin und Sulphonalgiftung
Arch. f. exp. Path. u. Pharm. 1900. Bd. 43. p. 456.

1367 PERINA, C. Therapy of Hydroa aestivalis.
Ceska Dermatologie, 1921. II. No. 2.
Abstr. B. J.D.&.S. 1922. p. 137.

1368. PERUTZ, A. Über Hydroa aestivale und vacciniforme eine Klinisch. experimentelle untersuchung.
Archiv. f. D.&.S. 1917. Bd. 124 p. 531.

1369. PFEIFFER, H. Über die Wirkung des Lichtes auf Eosin Blutgemische.
Wien.Klin.Woch. 1905. p. 221.

1370. SACHS & SACHAROFF Ueber die haemolytische Wirkung der photodynamischen Stoffe.
Munch.Med.Woch. 1905. Vol. 52. p. 249.

1371. SENEAR, F. E. & FINK, H.W. Hydroa vacciniforme seu Aestivale.
Arch. f.D.&.S. 1923. Vol. 7. p. 145.

1372. SMITH, H. L. Buckwheat Poisoning with report of a case in man.
Arch. Int.Med. 1909. Vol. 3. p. 350.

1373. SOBERNHEIM, G. Ein Beitrag zur Lehre von der Haematoporphorinurie.
Deutsch.Med.Woch. 1892. Vol. 18. p. 566.

1374. SOBOTKA, P. Studien über den einfluss experimentell veränderter örtlicher Bedingungen auf die Lichtreaktion. (Ultraviolettreaktion) der menschlichen Haut.
Arch. F.D.&.S. Bd. 121. 1915-16. p. 45.

1375. TAPPEINER, H. Von. Über die Wirkung fluorescirender Stoffe auf Infusoria nach Versuchen von O. Raab.
Munch. Med.Woch. 1900. No. I.

1376. WHITE, J. C. Hydroa vacciniforme.
Journ.Cutan.Dis. 1898. p. 514.

PELLAGRA.

1377. CESABIANCHI, D. & VALLARDI, C. Maize feeding and Hypersensitiveness to maize extracts.
Pathologica 1912. No. 88.

1378. HINDHEDE, M. Pellagra, and Protein.
J.A.M.A. 1923. Vol. 80. p. 1685.

1379. LOW, R. CRANSTON, & BROWN, R. DODDS, Pellagra.
Edin.Med. Journ. Sept. 1909.

1380. LOW, R. CRANSTON & YELLOWLEES. A case of Pellagra.
Edin.Med.Journ. 1920. p.315.

1381. RONDONI, P. Hypersensitiveness of Pellagra cases to maize.
Lo Sperimentale. Bd.66. No.5.

1382. VOLPINO, Hypersensitiveness of Pellagra cases to extract of maize.
V Ital.Congress for the study of Pellagra, Bergamo. Sept. 9-11, 1912.
Abstr. in Dermat.Woch. 1912. Bd. 55. p. 1383.

1383. VOLPINO, G. Anaphylactic phenomena in Pellagra patients inoculated with a water extract of spoilt maize.
J.A.M.A. 1912. Vol. 59. p. 1480.

1384. VOLPINO, MARIANI, BORDONI & ALFRAGO. First series of experiments on Pellagra.
Rio d'Igiene e Sanita-pubb. 1912. No. 2.

TYPHOIDIN SKIN TEST.

1385. AUSTRIAN, C.R. & BLOOMFIELD, A.L. Observations on the Typhoidin Reaction.
Arch. Int.Med. 1916. Vol.17. p. 663.

1386. CHANTEMESSE, A. L'ophthalmo-diagnostic de la fievre typhoide.
Deutsch. Med.Woch. 1908. Vol. 33. p. 1572.

1387. CHAUFFARD & TROISIER, Reproduction experimentale des taches rosées lenticulaires.
Compt. rend.Soc. de biol. 1909. Vol. 66. p. 519.

1388. DERHAM, S. J. The Typhoid cutaneous Reaction.
Univ. Penn. Med. Bull. 1909. Vol. 22. p. 192.

1389. ENTZ, R. Ueber das Verhalten der menschlichen Haut gegen verschiedene bakterielle giftstoffe.
Wien.Klin.Woch. 1908. Vol. 21. p. 379.

1390. FLOYD, C. & BARKER, W.W. The Typhoid cutaneous reaction.
A.J. Med. Sci. 1909. Vol. 138. p. 188.

1391. GAY & CLAYPOLE, An experimental study of methods of prophylactic Immunization against Typhoid fever.
Arch. Int.Med. 1914. Vol. 14. p. 699.

1392. GAY, F.P. & FORCE, J.N. A skin reaction indicative of Immunity against Typhoid Fever.
Archiv. Int.Med. 1914. Vol. 13. p. 471.

1393. GOODMAN, E.H. & SUTTER, C.C. The cutaneous reaction of Link in Typhoid Fever.
Univ. Penn. Med.Bull. 1909-10. Vol. 22. p. 81.

1394. KILGORE, E. S. Quantitative studies of the cutaneous Test of Typhoid Immunity.
Arch. Int.Med. 1916. Vol. 17. p. 25.

1395. KOLMER, J.A. & BERGE Jr. J.H. Anaphylactic skin reaction in relation to Immunity. I. The relation of the Typhoidin skin reaction to Immunity in Typhoid fever.
Journ.Immunol. 1916. Vol. 1. p. 409.

1396. KRAUS, R. Erwiderung auf L. Zupnik's Artikel.
Wien.Klin.Woch. 1907. Vol. 20. p. 346.

1397. KRAUS, LUSENBERGER & RUSS. Ist die ophthalmoreaktion nach Chantemesse zur diagnostischen Zwecken bei Typhus verwertbar.
Wiener.Klin.Woch. 1907. Vol.20. p. 1385.

1398. KRAUS, R. & STENITZER, R. Über Toxine der Typhusbacillus.
Wiener.Klin.Woch. 1907. Vol. 20. p. 344.

1399. LINK, R. Ueber Hautreaktionen bei Impfungen mit abgetöteten Typhus, Paratyphus und Kolikulturen.
Munch.Med.Woch. 1908. Vol. 55. p. 730.

1400. McKENDRICK, W. Cutaneous hypersensitiveness in enteric infections with special reference to enteric carriers.
Journ.Path.& Bact. 1923. Vol.26. p. 535.

1401. MEYER, K. F. & CHRISTIANSEN, C. R. The intracutaneous "Typhoidin" Reaction. I. The preparation and properties of the antigen.
Journ.Infect.Dis. 1917. Vol. 20. p. 357.

1402. " " " The intracutaneous "Typhoidin" Reaction. III. The relation of cutaneous hypersensitiveness to experimental Immunity and Infection.
Journ.Infect.Dis. 1917. Vol.20. p. 424.

1403. MEYER, K.F. & CHRISTIANSEN, C.R. The intracutaneous "Typhoidin" Reaction. II. The Nature and specificity of the Reaction.
Journ.Infect.Dis. 1917. Vol.20. p. 391.

1404. NICHOLS, H. J. Observations on Antityphoid vaccination.
Journ.exper.Med. 1915. Vol. 22. p. 780.

1405. PAISSEAU & TIXIER L'intradermo-reaction dans la fievre typhoide.
Compt. rend.Soc. de biol. 1909. Vol. 66. p. 877.

1406. PULAY, E. Diagnostische Hautreaktion bei Typhusrekonvaleszenten u.s.w.
Wiener.Klin.Woch. 1915. Vol. 28. p. 1189.

1407. ROLLY, Fr. Ueber die Beeinflüssung der durch Bakterientoxine hervorgerufenen Hautreaktionen.
Munch.Med.Woch. 1911. Vol. 58. p. 1285.

1408. VON SZONTAGH Ueber Diphtherie und Typhuscutanreaction.
Arch. f. Kinderhk. 1912. Vol. 58. p. 326.

1409. ZUPNIK, Ophthalmoreaktion bei Typhus.
Munch.Med.Woch. 1908. Vol.55. p. 148.

PNEUMONIA.

1410. CLOUGH, P.W. Some observations on hypersensitiveness to Pneumococcus protein with special reference to its relation to Immunity.
Bull. Johns Hopkin's Hosp. 1915. Vol. 26. p. 37.

1411. STEINFIELD, E. & KOLMER, J.A. Allergic skin reactions in pneumonia to type strains of pneumococci.
Journ.Infect.Dis. 1917. Vol. 20. p. 344.

1412. WEIL, R. Note of a skin reaction in Pneumonia. Journ.Exper.Med. 1916. Vol. 23. p. 11.

1413. WEIL, R. & TORREY, J.C. Immunological studies in Pneumonia. Journ.Exper.Med. 1916. Vol. 23. p. 1.

1414. WEISS, C. & KOLMER, J.A. A skin reaction to Pneumotoxin. Journ.Immunol. 1918. Vol. 3. p. 395.

PERTUSSIS.

1415. MODIGLIANI, E. & DE VILLA, S. The intracutaneous reaction for the early diagnosis of Pertussis. Pediatria. 1921. Vol. 29. p. 337.

1416. ORGEL, S.Z. A method for the early diagnosis of Pertussis. J.A.M.A. 1922. Vol. 79. p. 1508.

1417. RIESENFELD, E.A. Intracutaneous Reactions in Pertussis. J.A.M.A. 1923. Vol. 80. p. 158.

LEPROSY.

1418. PHOTINOS, G. & MICHAELIDES, N. The Wassermann and Pirquet skin reactions in Lepra. Lepra. Bd. 12. Heft. 4.

1419. STEIN, R.O. Die Kutireaktion bei Lepra und ihre Beziehung zum Lepraerysipeloid. Arch. f. D.&.S. 1916. Bd. 123. p. 908.

1420. TEAGUE, R.L. The cutaneous reaction in Leprosy. Abstr. in Zt. f. Immun. Ref. 1910 II. p. 712.

HYDATID/

HYDATID SKIN REACTION.

1421. PONTARO, Intradermo and Subcutaneous Reactions in Hydatid Disease.
B.M.J. April 16. 1921.
Il Policlinico, Sez.Med. November 1, 1920.

1422. SERRA, The Intradermo Reaction in the Diagnosis of Hydatid Disease.
Il Policlinico, 1921. Jan. 15.
Abstr. B.M.J. 1921. April 23.

CUTANEOUS REACTIONS IN OTHER DISEASES.

1423. RAVENNA, F. Ricerche Sull'anafilassi attiva e passiva dei tumori maligni.
Pathologica 1912. No. 84. p.243.

1424. BAKER, H.M. Intracutaneous Reaction in Infectious Diarrhoea.
Journ.Immunol. 1917. Vol. 2. p. 453.

1425. ENGELHORN & WINTZ. Über eine neue Hautreaktion in der Schwangerschaft.
Munch.Med.Woch. 1914. Vol. 61. p. 689.

1426. ESCH, P. Über eine neue Hautreaktion in der Schwangerschaft.
Munch.Med.Woch. 1914. Vol. 61. I. p. 1115.

1427. FALLS & BARTLETT. On the specificity of placental proteins in skin reactions of the human body.
Chicago Path.Soc. 1915. Vol. 9. p. 249.

1428. DE JONG, Intradermale und Konjunktivale Schwangerhaftsreaktion.
Munch.Med.Woch. 1914. Vol. 51. ii. p. 1502.

1429. REBAUDI, U. & PODESTA, G.B. On a dermatosis of ulcerative type of probable penicillary origin.
Giorn.Ital. d. mal ven. e. della pelle. 1922. Fasc. IV. p. 871.

/430. ITO, TETSUTA. Klinische und bakteriologisch-serologische Studien über ulcus molle und Ducreyshe Streptobazillen.
Archiv. f. D.&.S. 1913. Vol. 116 p. 341.

/431. KOLMER, J.A., HARKINS, M.J., & REICHEL, J. A cutaneous reaction in canine Distemper.
Journ.Immunol. 1916. Vol. I. p. 501.

DIPHTHERIA. SCHICK TEST.

/432. KOLMER, J. A. An allergic skin reaction to Diphtheria bacilli.
Proc.Soc. Exper.Biol & Med. 1916. Vol. 13. p. 89.

/433. KOLMER, J.A. & MOSHAGE, E.L. A note on the occurrence of pseudo-reactionson the skin.
J.A.M.A. 1915. Vol. 65. p. 144.

/434. PARK, W.H.,ZINGHER A. & SEROTA, H.M. The Schick reaction and its practical applications.
Arch. Pediatr. 1914 Vol. 31. p. 481.

/435. WEAVER, G.H. & MAHER, L.K. The diagnostic value of Intracutaneous Injections of Diphtheria Toxin (Schick reaction).
Journ.Infect.D. 1915. Vol. 16. p. 342.

/436. ZINGHER, A. Methods of using diphtheria toxin in the Schick test and of controlling the reaction.
J.A.M.A. 1922. Vol. 78 p. 490.

CUTANEOUS FOOD TESTS.

/437. AYRES, SAMUEL Jr. The application of anaphylactic skin tests to General Medicine.
Boston Med. & Surg.Journ. 1918. clxxviii p. 697.

/438. BAKER, H.M. Incidence of protein sensitisation in the normal child.
Amer.Journ.Dis. Child. 1920. Vol. 19. p. 114.

1439. BAKER, H.M. & FLOYD, C. Protein extracts in states of hypersensitisation.
Boston Med. & surg.Journ. 1916. Vol. 175. p. 199.

1440. BLACKFAN, K.D. Cutaneous Reaction from Proteins in Eczema.
Amr. Journ.Dis.child. 1916. Vol. 11, p.441.

1441. " " A consideration of certain aspects for Protein hypersensitiveness in children.
Amer.J. Med.Sc. 160, 1920. p.341

1442. COKE, F. Discussion on Pruritus ani at sub-section on Proctology of Royal Soc. Med.
B.M.J. April 23, 1921.

1443. DUKE, W. W. Food allergy as a cause of abdominal Pain.
Archiv. Int.Med. (Chicago) 1921. Vol. 28. p. 151.

1444. ENGMAN, W.F. & WANDER, W.G. The application of cutaneous sensitisation to Diseases of the skin.
Archiv.of Derm. & Syph. 1921. Vol. 3. p. 223.

1445. FERRY, Methods of preparing pure proteins for skin tests.
J. Lab. & Clin.Med. 1917. Vol.2. p. 655.

1446. FOOTE, J. Egg sensitisation of hidden origin in eczema of infant.
Internat. Clinics 1920. IV Series 30. p. 212.

1447. FOX, H. & FISHER,J.E. Protein sensitisation in eczema in adults.
Journ.Amer.Med.Assoc. 1920. Vol. 75. p. 907.

1448. FREEMAN, J. Toxic Idiopathies.
Lancet. July 31, 1920. p.229.

1449. FREEMAN, J. Toxic Idiopathies. Relationship between hay and other pollen fevers animal asthmas, food idiosyncrasies, bronchial and spasmodic asthmas etc.
Proc.Royal. Soc.Med. 1920. Vol. 13 p. 129.

1450. GERSTENBERGER, H.J. & DAVIS, J.H. Report of a case of Anaphylaxis following an intradermal protein sensitisation test.
J.A.M.A. 1921 Vol. 76. p. 721.

1451. GOULD, A. G. Protein Sensitisation.
J.A.M.A. 1923. Vol. 80. p. 394.

1452. NOBL, Kutireaktion mit wässrigem und Alkoholischem extrakt.
Archiv. f. D.&.S. Bd. 99. p. 427.

1453. O'KEEFE, E. S. The relation of food to Infantile Eczema.
Boston M.&.S. J. Vol. 183. 1920. p. 569.

1454. O'KEEFE, E. S. Eczema in the breast-fed baby and protein sensitisation.
Boston Med. & Surg.J. 1921. p. 194. Vol. 185.

1455. RACKEMANN, F.M. Skin tests with foreign proteins in various conditions.
Am.Journ.Med.Sci. 1922. Vol. 163, p. 87.
Abstr. B.M.J. March 4, 1922.

1456. RAMIREZ, M.A. Protein Sensitisation in eczema.
Arch. of D.&.S. Sept. 1920. p. 365.

1457. " " Protein sensitisation with special reference to bronchial asthma, hay fever and eczema.
New York Med.Journ. 1921. Vol. 114. p. 320.

1458. RAVITCH, M.L. & STEINBERG, S.A. Eczema in infants and the thyroid gland.
Journ.Cutan.Dis. 1919. Vol. 37. p. 313.

1459. SABATINI, G. Saggi di cutireazione con proteini specifiche asmogene.
Il policlinico. Apr. 16, 1921. p. 539.

1460. SCHLOSS, O. M. Allergy to common Foods - Prelimin. Report.
Trans.Am. Pediat. Soc. 1915. Vol. 27. p. 62.

1461. SCHLOSS, O.M. A case of allergy to common Foods. Am. Journ.Dis.Child. 1912. Vol.3. p. 341.

1462. " " Allergy in Infants and Children.
Amer.Journ.Dis.Child. 1920. Vol. 19. p. 433.

1463. " " Allergy in Infants and Children.
Cornell Univ.Med.Bull. 1921 July.

1464. SHANNON, W. R. Demonstration of food proteins in human breast milk by experiments on guinea pigs.
Amer.Journ.Dis of child. 1921. Vol. 22. p. 223.

1465. " " Anaphylaxis to food proteins in breast-fed infants and its probable relation to certain diseases of the nursing infant especially exudative diathesis.
Minnesota State Med.Assoc. Aug. 1921.
J.A.M.A. 1921. Vol. 77. p.964.

1466. STOKES, Discussion on papers by Towle and Higman and Michael.
Arch. D.&.S. 1920. Nov.

1467. STRICKLER, ALBERT Anaphylactic food reactions in Dermatology with special,Reference to Eczema.
Amer.Assoc. of Immunologists, 3rd Annual meeting in Washington, May 11-12, 1916.

1468. " " Anaphylactic Food reactions in Dermatology with special reference to Eczema.
Proc.Amer.Assoc. Immunol. Journ. Immunol. 1916. Vol. I. p. 475.

1469. " " Anaphylactic food reactions in skin diseases.
New York Med.Journ.104. 1916. p. 198.

1470. STRICKLER & GOLDBERG Anaphylactic Food reactions in Dermatology.
Journ.Amer.Med.Assoc. 1916. lxvi. p. 249.

1471. TALBOT, F. B. The relation of food idiosyncrasies to the diseases of childhood.
Boston Med. & Surg.Journ. 1918. Vol. 179. p. 285.

1472. TALBOT, F. B. Eczema in childhood.
Med.Clin N. America. Jam. 1918. p. 985.

1473. TOWLE, HARVEY P. Protein sensitisation in the production of skin disease.
Arch. of D.&.S. Nov. 1920. Vol. 2. p. 531.

1474. WALKER, J.C. & ADKINSON, J. A comparison between the cutaneous and the intradermal tests in the sensitisation of asthmatic and hay fever patients.
Journ.Med.Res.1917 Vol.37.p.287.

1475. WHITE, C. J. The anaphylactic phenomenon in eczema and the recent progress in our knowledge of the etiology and treatment of the disease.
Journ.Cut.Dis. Feb. 16, 1916. Vol. 34. p. 57.

1476. WHITE, C. J. Two modern methods to be employed in the treatment of chronic eczema.
J.A.M.A.No.68. Jan 13, 1917. p. 81.

1477. WHITE, C. J. The treatment of eczema in childhood
Boston Med. & Surg. Journ. 1918. Vol. 178. p. 5.

1478. WODEHOUSE, R. P. Preparation of vegetable food proteins for Anaphylactic tests.
Boston.Med.& Surg.Journ. 1916. Vol. 175. p. 195.

1479. WODEHOUSE, R. P. & OLMSTED, J.M.D. Preparation of vegetable proteins.
Boston Med. & Surg.Journ. 1917. Vol. 176. p. 467.

ALLERGY/

ALLERGY - GENERAL.

1480. AMBERG, S. & KNOX, J.H.M. The influence of Sodium Iodoxybenzoate and sodium cyanide upon an allergic Reaction of Inflammatory character.
Journ.Pharm. & Exper. Therap. 1911-12. Vol. 3. p. 223.

1481. ARTHUS, M. & BRETON, M. Lesions cutanées produites par les injections de serum de cheval chez le lapin anaphylactique par et pour ce serum.
Comptes rendus hebdom. des sceances et mémoires de la Soc. de Biol. de Paris. 1903. Tome. 55. p. 1478.

1482. BALDWIN, E. R.
Journ.Nat.Assoc. Study & Prev. Tubere.7th annual meeting 1911. p. 351.

1483. BIBERSTEIN & OSCHINSKY, Experiments on the Susceptibility of Human Skin to animal sera.
Arch. f. D.&.S. 1923. Vol. 142. p. 353.

1484. BLOCH, B. Problems of metabolism and Immunity in Dermatology.
Correspondenz Blatt. für Schweizer aertze, Aug. 4, Vol. xlvii, p. 993.
Abstr. in J.A.M.A. Sept. 29, 1917. Vol. lxix. p. 1118.

1485. FLEISCHNER, E. C. Certain aspects of cutaneous Hypersensitiveness.
Amer.Pediatric Soc. 31st Annual meeting. Atlantic City. June 16-18, 1919.
Abstr. in Journ. A.M.A. 1919. lxxiii, p. 217.

1486. FLEISCHNER, E.C., MEYER, K.F. & SHAW, E.B. A resume of some experimental studies on cutaneous hypersensitiveness.
Am.Journ.Dis.Child. 1919. Vol. 18. p. 577.

1487. FUKUHARA, Y. Ueber die Kutanreaktion bei der Serumanaphylaxie. Zertschr. f. Immunitats. forch 1911. Vol. 11. p. 640.

1488. GOURD, F. B. Focal reactions to Horse-serum. Arch. of Surgery. 1921. p.419.

1489. HIFT, R. Zur nicht-proteinogenen Allergie Wiener Klin, Woch. 1913. Vol. 26. p. 1546.

1490. KNOX, MOSS & BROWN, Subcutaneous reaction of rabbits to horse serum. Journ.Exper.Med. XII. 1910. p. 562.

1491. KOLMER, J. A. Concerning allergic skin reactions as an Index of Immunity. Proc.Amer. Assoc. Immunologists Journ. Immunology 1916. Vol. I. p. 472.

1492. KOLMER, JOHN A. Allergic skin reactions as an Index of Immunity. 3rd Annual meeting of Amer. Assoc. of Immunologists, Washington May 11-12, 1916.

1493. " " " The mechanism and clinical significance of anaphylactic and pseudo-anaphylactic skin reactions. Bull. JohnsHopkin's Hosp. 1917. Vol. 28. p. 163.

1494. KRAUSE, A. K. Experimental studies on the cutaneous reaction to Tuberculo-protein etc. Journ.Med. Res. 1916. Vol. 35. pp. 1, 25 and 43.

1495. LANDMANN, P. Ein seltener Fall von Idiosynkrasie gegen Huhnereiweiss nebst Beitrag zur Wurdigung des "Fleischsaft" Puro. Munch, Med.Woch. 1908. Vol. 55. p. 1079.

1496. LEARY, T. DUNBAR, W.H. & WATSON, J.W. Anaphylactic Reactions to normal serums. Boston Med. & Surg.Journ. Nov. 1. clxxvii, No. 18. p. 617. Abstr. J.A.M.A. Vol. lxix. p. 1829.

1497. LUCAS, W.P. & GAY, F.P. Localised Anaphylactic intoxication in children following the repeated injection of antitoxin. Journ.Med.Res. 1909. p. 251.

1498. LUCKIE, J. B. Allergies in chronic Diseases. Medical Rec. 1920. Vol. 98. p. 733.

1499. MOSS, W. L. A cutaneous anaphylactic reaction as a contra-indication to the administration of antitoxin. Journ.Med.Assoc. lv. 1910. p. 776.

1500. PARHON & SATINI Essais sur les cuti-réactions glandulaires. Révue neurologique 1914. Vol. 27. p. 875.

1501. PESTALOZZA, C. The regional cuti-reaction in children. La Pediatria. 1920. Feb. 15. Abstr. B.M.J. June 12, 1920.

1502. VON PIRQUET, C.F. "Allergie". Berlin, 1910.

1503. VON PIRQUET, C. Ist die Vakzinale Frühreaktion spezifisch ? Wiener Klin.Woch. 1906. Vol. 19. p. 1407.

1504. VON PIRQUET, Allergie. Munch,Med.Woch. 1906. Vol. 53. p. 1457.

1505. " " Der diagnostische Wert der kutanen Tuberkulinreaktion bei der Tuberkulose des kindesalters auf Grund von 100 sektionen. Wien.Klin,Woch. 1907. No. 38.

1506. " " The cutaneous Tuberculin Test. Arch. Pediatrics, New York, 1910. Vol. 27. p. 161.

1507. RACHMILEWITSCH, E. Hautreaktionen von kindern mit exudativer Diathese. Jarhb. f. Kinderh. 1913. Vol. 77. p. 176.

1508. SELLEI, J. Die Empfindlichkeit des organismus gegen die Körpereigener Eiweisskörper.
Berlin Klin.Woch. 1910. Vol. 47, p. 1836.

1509. STOKES, J. H. Intradermal reactions to emulsions of normal and pathological skin.
Journ.Infect.D. 1916. Vol. 18. p. 402.

1510. " " An intradermal reaction to,agar and an interpretation of intradermal reactions.
Journ.Infect.Dis. 1916. Vol. 18. p. 415.

1511. STRICKLER, ALBERT Studies concerning the influence of Arsenical preparations on cutaneous Tests.
Arch. of D.&.S. 1921 Vol. 4. p. 177.

1512. " " A histopathologic study of Positive cutaneous tests.
J.A.M.A. 1922. Vol. 78. p. 1287.

1513. STRICKLER, A. & ASNIS, E.J. The Histopathology of Cutaneous tests.
Arch. Dermat. & Syph. 1923. vii. 379.

INTRAVENOUS VACCINE THERAPY.

1514. AULD, A. G. Pyrogenic Therapy.
B.M.J. 1918. Vol. 1. p. 195.

1515. BIEDL, A. Therapeutische Verwendung von Typhusimpfstoffen bei menschen.
Prag.med.Woch. 1915. Vol. 40. p. 53.

1516. BULL, C. G. The influence of Typhoid Bacilli on the antibodies of normal and Immune rabbits.
Journ. exper. med. 1916. Vol. 23, p. 419.

1517. CAPELLI & SIGNORELLI. Results of Hetero-vaccine Therapy in certain skin affections and syphilis.
Giorn.Ital. d. mal. ven e. della Pelle 1920 Fasc. II. p. 181.

1518. CECIL, Report on forty cases of acute arthritis treated by intravenous injection of foreign protein.
Arch. Int.Med. 1917. Vol. 20. p. 951.

1519. COWIE, D.M. & BEAVEN, P.W. Non-specific protein therapy in Influenzal pneumonia.
J.A.M.A. 1919 Vol. 72. p. 1117.

1520. COWIE, D.M. & CALHOUN H. Non-specific Therapy in Arthritis and Infections.
Arch. Int.Med. 1919. Vol. 32. p. 69.

1521. DAVIS, B.F. & PETERSEN, W.F. A comparative study of serum and lymph. ferments after feeding.
Journ.Exper.Med. 1917. Vol. 26. p. 693.

1522. DAVIS, B.F. & PETERSEN, W.F. A comparative study of lymph. & serum ferments during protein shock reactions.
Journ.Exper.Med. 1917. Vol. xxvi, p. 699.

1523. EGGERTH, Über die Behandlung des Typhus abdominalis mit Typhus vakzine.
Wien.Klin.Woch. 1915. Vol. 28. p. 209.

1524. ENGMAN, M.F. & McGARRY, R.A. The Treatment of certain Diseases of the Skin by the Intravenous injection of a foreign proteid.
Journ.Amer.Med.Assoc. Dec. 9. 1916. Vol. lxvii p. 1741.

1525. GAY, F.P. & CLAYPOLE, E.J. Specific hyperleucocytosis.
Archiv. Int.Med. 1914. p. 662.

1526. GOW, A note on certain phenomena associated with the Protein Shock Reaction and Intervenous vaccine Therapy.
Quart.Journ.Med. Oxford, Vol. xiii. No. 49. p. 82.

1527. ICHIKAWA. Abortive treatment of Typhoid and Paratyphoid.
Sei-i-Kwai Med.Journ. 1913. Vol. 33. p. 73.

1528. JOBLING, J.W. & PETERSEN, W. The non-specific Factors in the treatment of Disease.
Journ.Am. Med. A. Vol. lxvi. 1916. p. 1753.

1529. KRAUS, R. Über Bakteriotherapie akuter Infektionskrankheiten. Heterobakteriotherapie.
Wiener.Klin.Woch. 1915. Vol. 28, p. 29.

1530. KRAUS, R. & DOERR. Über anaphylaxie.
Centralbl. f. Bakt. 1909. Referate. No. 42. Abt. 1. p. 36.*

1531. KRAUS & MAZZA Zur Frage der Vakzinetherapie des Typhus abdominalis.
Munch.Med.Woch. 1914. Vol. 61. p. 1967.

1532. McWILLIAMS, HELEN J. Is the hyperleucocytosis following the injection of Typhoid bacilli into immunized rabbits specific?
Journ. Immunol. 1916. Vol. 1. p. 159.

1533. MILLER, J.L. The non-specific character of vaccine therapy.
J.A.M.A. 1917. Vol. 69. p. 765.

1534. " " Foreign Protein Therapy in the acute Infections.
J.A.M.A. 1921. Vol. 76. p. 308.

1535. MILLER, J.L. & LUSK, F.B. The treatment of arthritis by the intravenous injection of foreign protein.
J.A.M.A. 1916. Vol. 66. p.1756.

1536. MÜLLER, E. F. Die allgemeine Protoplasmaaktivierung Weichards.
Med. Klin. 1918. No. 46.

1537. PETERSEN, W. F. Serum changes following protein shock therapy.
Arch. of Int.Med. 1917. Vol. 20. p. 716.

1538. PETERSEN, W. F. The non-specific Reaction. J.A.M.A. 1921. Vol. 76. p.312.

1539. ROBERTS, DUDLEY & CAREY, Bacterial protein injections in Influenzal Pneumonia. J.A.M.A. 1919 Vol. 72. p.922.

1540. RUMPF, Die Behandlung des Typhus abdominalis mit abgetöteten kulturen des Bacillus Pyocyaneus. Deutsch.Med.Woch. 1893. Vol. 19. p. 561.

1541. SCULLY, F. J. The reaction after intravenous injections of foreign Protein. J.A.M.A. July, 7, 1917. Vol. lxix, p. 21.

1542. SCULLY, F. J. The reaction after intravenous injections of foreign Protein. J.A.M.A. 1917. Vol. 69. p. 1684.

1543. SLADEK & KOTLOWSKI, Zur vakzine Therapie des Typhus abdominalis. Wien.Klin.Woch. 1915. Vol. 28. p. 389.

1544. SUTTON, R. L. The treatment of Psoriasis. Arch. of D.&.S. 1921. Vol.IV. p. 633.

1545. SWIFT, H.F. & KINSELLA, R.A. Active immunization with sensitised and non-sensitised bacteria. Proc.Soc. exper.Biol.& Med. 1916-17. Vol. 14. p. 120.

1546. TEAGUE, O. & McWILLIAMS, H.J. The Bacteriolytic power of normal and immune rabbit serum for typhoid bacilli and the influence of the intravenous injection of vaccine upon the same. Journ.Immunol. 1917. Vol. 2. p. 167.

1547. TEAGUE, O. & McWILLIAMS, H.J. Experiments with a possible bearing upon treatment of typhoid fever with typhoid vaccine administered intravenously. Journ.Immunol. 1917. Vol. 2. p. 185.

1548 THOMAS, H. B. Arthritis and Foreign Protein. J.A.M.A. 1917. Vol. 69. p. 770.

1549. WASSERMANN, A. Über neuere Immunisierungsverfahren. Deutsch.Med.Woch. 1907. Vol. 33. p. 1936.

1550. WEICHARDT, W. Ueber unspezifische Leistungs-steigerung (Protoplasmaaktivierung) Munch.Med.Woch. 1920. Vol. 67. p. 91.

1551. WEISS, A. Ueber intravenöse Vakzinebehandlung gonorrhoischer Komplikationen. Wien.Klin.Woch. 1916. Bd. 29. p. 619.

1552. WELLS, C. W. Intravenous injections of foreign protein in Influenzal Pneumonia. J.A.M.A. 1919. Vol. 72. p.1813.

1553. WILLIAMS, H. S. The present status of non-specific protein therapy. Medical Record 1921. Vol. 100. p. 529.

PEPTONE THERAPY.

1554. ABDERHALDEN & WEICHARDT, Über den gehalt des Kaninchenserums, an peptolytischen Fermenten unter verschiedene Bedingungen. Zeitsch. f. physiol. chemie. 1909. lxii. p. 120.

1555. VAN ALSTYNE, N. The non-specific protein treatment of Psoriasis. Med.Record. 1917. p. 538.

1556. AMBROSOLI, G. A. La proteinoterapia non bacterica in alcune malattie della pelle. Giorn.Ital. d.mal. ven. e. della pelle. 1921. Fasc. II. p.128.

1557. AULD, A. G. Preliminary note on a new treatment of Bronchial Asthma. B.M.J. 1917. Vol. I. p. 580.

1558. AULD, A. G. Treatment of asthma by peptone injections.
B.M.J. 1917. Vol. I. p. 749.

1559. " " Further Remarks on the Treatment of Asthma by Peptone.
B.M.J. July 20, 1918. p. 49.

1560. " " Results of the Peptone treatment of Asthma.
Brit.Med.Journ. 1920. April 24, p. 567.

1561. " " The use of Peptone in Asthma and its Congeners.
B.M.J. 1921. May 14. p. 696.

1562. " " Pyogenic Therapy ("Protein Shock")
B.M.J. 1921. p. 822.

1563. BESSAU, G. OPITZ, H. & PREUSSE O. Über die Spezifität der antianaphylaxie.
Zentralbl. f. Bakt. 1914. Pt.I. Vol. 74. p. 162.

1564. BESSAU, G, OPITZ, H. & PREUSSE O, Precipitin schwund und Antianaphylaxie.
Zentralbl. f. Bakt. 1914. Vol. 74. p. 310.

1565. BIEDL & KRAUS Über Kriterien der Anaphylaktischen Vergiftung.
Centralbl. f. Bakt. Ref. 1910. Vol. 47. Beiheft. p. 35.

1566. BIEDL & KRAUS Die Wirkung intravenös injezierten Peptons beim meerschweinchen.
Zentralbl. f. Physiol. 1910. Vol. 24. p. 258.

1567. BRIEGER, L. Ueber Ptomaine.
Berlin, 1885. p. 14.

1568. BRODIN & RICHET Prevention of anaphylactic shock.
Compt. Rendu Soc.Biol. Feb. 12 1921.
B.M.J. April, 30. 1921.

1569. CHITTENDEN, MENDEL & HENDERSON. A chemico-physiological study of certain derivatives of the Proteids.
Am.Journ.Physiol. 1899. Vol. 2. p. 142.

1570. CLARK, A.J. The scientific basis for non-specific Protein Therapy. B.M.J. 1923. Vol.I. p. 315.

1571. COKE, F. Discussion on Asthma. B.M.J. 1921 II. p. 236.

1572. COOKE, J.V. & WHIPPLE, G.H., The metabolism of dogs with sterile abscess, pancreatitis and pleuritis. Journ.Exper.Med. 1918. Vol. 28. p. 223.

1573. CROSS, R. Foreign Protein injections in the treatment of human infections. Lancet 1917, 37, 23, p. 764.

1574. CULVER, H. Intervenous protein injections in urology and dermatology. J.A.M.A. 1921. Vol. 76. p. 311.

1575. DALE, H.H. & LAIDLAW, P.P. The physiological action of B iminazolylethylamine. Journ.Physiol. Vol. 41. p.337.

1576. DALE, H.H. & RICHARDS, A.N. The vasodilator action of Histamine and of other substances Journ.Physiol. 1919. Vol. 52; p. 110.

1577. DALE & LAIDLAW. Histamine Shock. Journ. of Physiol. Vol. 52 (1919) p. 355.

1578 FANO, Das Verhalten des Peptons und Tryptons gegen Blut und Lymphe. Arch. f. Anat. u. Physiol. 1881. p. 277.

1579. GOW, Intravenous Protein Therapy. B.M.J. 1920. p. 284.

1580. HANKE, M.T. & KOESSLER, K.K. The relation of Histamine to peptone shock. Journ.Biolog. Chem. 1920. Vol. 43. p. 567.

1581. HIRSCHFELDER, A. D. Another point of resemblance between anaphylactic intoxication and poisoning with Witte's peptone. Journ. Exper.Med. 1910. Vol. 13. p. 586.

1582. HISANOBU, K. On the distribution of the non-protein nitrogen in cases of anaphylaxis and peptone poisoning. Amer.Journ.Physiol. 1920. Vol. 50. p. 357.

1583. ILVENTO, A. Anatomic basis for anaphylaxis. Il policlinico 1920. Vol. 27. p. 889.

1584. JOBLING, PETERSEN & EGGSTEIN, The effect of protein split products on the serum ferments and antiferment. Journ. Exper.Med. 1915. Vol. 22. p. 597.

1585. JOLTRAIN, Treatment of alimentary anaphylaxis. Bull. de la Soc.Med. des Hop. de Paris. 1919. 43 No. 19. p. 556. Abstr. Journ. Amer.Med.Assoc. 1919. lxxiii. p. 562.

1586. KRAUS, R. Anaphylaxis, Allergy and Treatment. Revista Medica de Chile, Santiago, May 1919, 47. No.5. p. 191.

1587. KUMAGAI, T. Vergiftung durch Witte-pepton. Zeitsch. f. Immunitatsf. 1913. Vol. 17. pp. 626 & 636.

1588. LAROCHE, G., RICHET, C, Jr. & St. GIRONS, Alimentary anaphylaxis. Bull. Med. 1920. Vol. 34. p. 625.

1589. LARSEN, HAIGH, ALEXANDER & PADDOCK. Failure of Peptone to protect against anaphylactic shock. Journ. Immunol. 1923. p. 409. Abstr. B.M.J. Oct. 27, 1923.

1590. LÜDKE, H. Die Behandlung des abdominal typhus mit intravenösen Injektionen von Albumosen. Munch.Med.Woch. 1915. Bd. 62. p. 321.

1591. MATTHES, M. Über die wirkung einiger subcutan einverleibter Albumosenauf den thierischen insonderheit auf den tuberculös inficirten organismus. Deutsch.Archi. f. Klin.Med. 1895. Vol. 45. p. 39.

1592. MILLER, J.L. & LUSK, F.B. The use of Foreign Protein in the treatment of Arthritis. J.A.M.A. 1916. Vol. 67. p.2010.

1593. MITTLANDER, Budapest Letter. J.A.M.A. 1916. p. 1320.

1594. MUCH, H. Non-specific Immunity. Deut.Med.Woch. 1920. Vol. 46. p. 483 & p. 791.

1595. NOLF, Ambulance de l'ocean 1917. Vol. I. p. 197.

1596. NOLF, P. Du traitement des arthrites aigües par le salicylate de soude associé aux injections intraveineuses de peptones. Presse med, Par., 1918, xxvi, p. 485.

1597. NOLF, Des injections de peptone dans le traitement de la fievre typhoide et des autres etats infectieux. Arch.Med.Belges. 1917.Vol.70.p.114.

1598. NOLF, P. Proteose therapy by the intravenous method. J.A.M.A. 1919. Vol. 72. p.1901.

1599. NOVY, F.G., DE KRUIF, F.H., & NOVY, F.O. Peptone anaphylatoxin. Journ.Infect.Dis. 1917. Vol.20. p. 657.

1600. PAGNIEZ & VALLERY-RADOT, Etude physio-pathologique et therapeutique dun cas d'urticaire géante. Anaphylaxie et anti-anaphylaxie alimentaire. La presse med. 1916. Nov. 23.

1601. PAGNIEZ, P. & VALLERY-RADOT, P. & NAST, A. Migraine. Presse médicale, 1919. Vol. 27. No. 19. p. 172.

1602. PAGNIEZ & VALLERY-RADOT, Nouvelles observations d'anti anaphylaxie digestive. Soc.Med. des Hop. 1919. June 6.

1603. PAGNIEZ, P. & VALLERY-RADOT, P. Digestive anti-anaphylaxis. Ann. de Med. 1920. Vol. 8. p. 303.

1604. PAGNIEZ & VALLERY-RADOT. Anaphylaxie Digestive. Traitement de certaines urticaires et dermatoses.
Annales de D.&.S. 1920. Tome I. No. 10. p. 436.

1605. PFEIFFER & MITA. Studien ueber Eiweiss-anaphylaxie
Zeitsch. f. Immunitatsf. 1909 Vol. 4. p. 439.

1606. PICK & SPIRO, Ueber gerinnungshemmende Agentien im Organismus höherer wirbelthiere
Zeitschr. f. Physiol. Chem. 1900. Vol. 31. p. 235.

1607. POPIELSKI, L. Ueber die physiologischen und chemischen Eigenschaften des Peptons Witte.
Pflügers Archiv. 1909. p. 483.

1608. RITZ, H. Ueber die Rolle hypertonischer Salzlosunger bei der anaphylaxie.
Zeitsch. f. Immunitatsf. 1911 Vol. 12. p. 654.

1609. RUSZNYAK, S. Die Aenderung des antitryptischen Titers des Serums bei der Anaphylaxie.
Deutsch.Med.Woch. 1912. Vol. 38. p. 168.

1610. SCHMIDT-MÜLHEIM, A. Beiträge zur Kenntniss des Peptons und Seiner physiologischen Bedeutung.
Arch. f. Anat. u. Physiol. 1880. p. 33.

1611. STARKENSTEIN, E. Proteinkorpertherapie und Entzundungshemmung.
Munch.Med.Woch. 1919. Vol. 66. p. 205.

1612. THOMPSON, W. H. Contributions to the physiological effects of peptone when injected into the circulation. The local vascular influences of peptone and proteoses.
Journ. Physiol. 1899. Vol.25. p. 1.

1613. THOMPSON, W.H. The influence of peptone and albumoses on the urinary secretion. Journ. Physiol. 1899. Vol. 25. p. 179.

1614. UNDERHILL, F. P. New experiments on the physiological action of the proteoses. Am.Journ. Physiol. 1903. Vol. 9. p. 345.

1615. VAUGHAN, Protein split-products in relation to Immunity and Disease. Philadelphia, 1913.

1616. VAUGHAN, V. C. & PALMER, G.H. Non-specific Immunity. Military Surgeon, 1920. Vol. 46. p.1.

1617. DE WAELE, H. Contribution à l'étude de l'anaphylaxie. Bull de l'acad. roy. de méd. de Belgique. 1907. Vol. 21 p.715.

1618. WEICHARDT, W. Ueber Proteinkörpertherapie Munch.Med.Woch. 1918. Vol. 65. p. 581.

1619. WEIL, R. Anaphylaxis in the Dog. Proc.Soc. Exper.Biol.&.Med. 1916-17, Vol.14. p. 117.

1620. WEIL, R. A study of the liver in shock and in peptone poisoning. Journ. of Immunology, 1917. Vol. II. p. 525.

1621. WELLS, H. G. Studies in the chemistry of anaphylaxis. Journ.Infect.Dis. Sept. 1909. Vol. 6. p. 506.

1622. WERBITSKI, Contribution à l'étude de l'anaphylaxie. Compt. rend.Soc.Biol. 1909. Vol. 66. p. 1084.

1623. WHIPPLE, G.H. & VAN SLYKE, D.D. Proteose intoxication and injury of body protein. Journ.Exper.Med. 1918. Vol.28. p. 213.

1624. WICK, Behandlung des gelenkrheumatismus mit Einspritzung von Kollargol ins Blut.
Munch.Med.Woch. 1916. Vol. 63. p. 350.

1625. WILLIAMS, H. S. A year of Proteal Therapy.
Med.Record. 1919. Nov. 22.

1626. " " Theory and Practice of Proteal Therapy.
Med.Record. 1919. Dec. 20.

1627. UNDERHILL & HENDRIX Studies on the physiological action of some protein derivatives.
Journ.Biol.Chem. 1915. Vol. 22. p. 443.

1628. ZUBIZARRETA, A. The cure of Digestive anaphylaxis.
Semana.Medica (Buenos Aires) 1920. Vol. 27. p. 513.
Abstr. J.A.M.A. 1920. Vol. 76. p. 277.

1629. ZUNZ, Contribution à l'étude de la digestion et de la resorption des proteins dans L'estomac et l'intestin grêle chez le chien.
Mem. Curonnées, 1908. Vol. 20. p. 3.

MILK INJECTION THERAPY.

1630. AHLSWEDE, E. Non-specific protein therapy.
Arch. of D.&.S. 1922. Vol. 5. p. 586.

1631. ANTONI, Die a[illegible]olanbehandlung des weichen Schankers und entzündlicher Bubonen.
Munch.Med.Woch. 1919. Bd.lxvi. p. 746.

1632. BERNDT, F. Über Milchbehandlung bei Entzündlichen Prozessen speziell beim Ulcus Molle.
Med.Klinik. 1921. Vol. 17. p. 164.

1633. CATTANEO, L. La Proteinoterapia aspecifica nelle malattie veneree.
Giorn.Ital. d. mal.ven e. della pelle 1921. Fasc. II. p. 123.

1634. FORD, W. W. Observations on the toxic properties of heated and decomposed milk and of milk cultures of B. Welchii
Am.Journ.Child.Dis. 1919. Vol. 18. p. 199.

1635. FRIEDLÄNDER. Therapeutische Erfahrungen bei parenteraler Injektion von Protein Körpern und ihren Spatzproduction in die Augenheilkunde.
Wien.Klin.Woch. 1916. Vol.29. p. 1321.

1636. GAWALOWSKI, Intracutaneous injections of "Lactin".
Ceska Dermatologie 1922. Vol. 3. p. 147.
Abstr. in B.J.D.&.S. 1923. p. 79.

1637. GUSZMAN, J. Untersuchungen über die Wirkung der Milchinjektionen auf des Verlauf des weichen Schankers.
Derm.Woch. 1918. Bd. 67. p.808

1638. GUSZMAN, J. Provokatorische Versuche mit Milchinjektionen im latenten stadium der Syphilis.
Dermat.Woch. 1921. p.1173.

1639. KRUGER & PFEILER. Protein-activation therapy.
Dermat.Woch. Feb. 1922.

1640. LEVI. Protein Therapy in Pregnancy Toxaemias.
Annali di Ostet. e.Gin. Feb. 28, 1922.
Abstr. in B.M.J. Apr. 8, 1922.

1641. MORINI, L. Circa l'efficacia della proteinoterapia aspecifica nella Cura del bubbone venereo.
Giorn.Ital.d.mal. ven. e. della pelle. 1921. p. 43.

1642. MÜLLER, E. F. Die myeloische wirkung der Milchinjektion.
Med.Klinik. 1918, XIV, 440.

1643. " " Weitere Mitteilungen zur kenntnis der Milchinjektions-wirkung.
Med.Klinik. 1918. XIV. 688.

1644. MULLER & THANNER Über parenterale eiweiss Injektion.
Med.Klin. 1916. Vol.12 p. 1120.

1645. SAXL, Über die Einwirkung pyrogener substanzen und Fieber.
Abstr.Munch.Med.Woch. 1916. Vol. 63. p. 571.

1646. SCHMIDT, R. Über Proteinkorpertherapie und über parenterale Zufuhr von Milch.
Med.Klin. 1916. Vol. 12. p.171.

1647. UDDGREN, Milchinjektionen und Wassermann'-sche Reaktion.
Berlin Klin.Woch. 1918. Jg lv. p. 354.

1648. VARNEY, Discussion on protein sensitisation.
Arch. of D.&.S. 1920. Vol. 2. p. 574.

1649. ZIEMBOWSKI, D. Über den therapeutischen Wert parenteraler Milch Zufuhr.
Med.Klin. 1916. Vol. 12 p. 1174.

NON-SPECIFIC SERUM THERAPY.

1650. ACHARD & FLANDIN Autosero therapy in Hay fever, urticaria etc.
Bull de la Soc.Med. des Hop. Paris. 1920, May 21. p. 723.

1651. ARNOLD, A. & HÖLZEL, H. Über den Wert intravenöser Arthrigonin injektionen bei Gonorrhoischen Prozessen.
Munch.Med.Woch. 1914. Vol. 61. p. 1967.

1652. BINGEL, A. Über Behandlung der Diphtherie mit gewohnlichen Pferdeserum.
Deutsch. Arch. f. Klin.Med. 1918. Vol. 125. p. 284.

1653. BRAND, A. T. Autoserum in the treatment of Disease.
B.M.J. 1921. Vol.2. p. 418.

1654. BRECHET, Case of Dermatitis Herpetiformis treated with autogenous serum.
Journ.Cutan.Dis. 1916. Vol.34. p. 134.

1655. BRONFENBRENNER, J. & SCHLESINGER, M. J. Some suggestions for rational auto-serum therapy.
Proc.Soc. Exper.Biol.& Med. 1916-17. Vol. 14. p. 61.

1656. CORLETT, W. T. Discussion on Autoserum treatment.
J.A.M.A. 1914. Vol. 63. p. 1194.

1657. DAVIS, D. H. Vaccine Therapy.
J.A.M.A. 1917. Vol. 68. p. 159.

1658. DELAFIELD & PRUDDEN Text Book of Pathology, 11th Edition.

1659. DOERR, R. Der gegenwärtige Stand der Lehre von der anaphylaxie.
Zeitsch. f. Immunitatsf. Ref. 1910. Vol. 2. p. 117.

1660. DOERR & RAUBITSCHEK, Toxin und anaphylaktisierende Substanz des Aalserums.
Berlin Klin.Woch. 1908. Bd. 45. p. 1525.

1661. DUNCAN, C. H. Autotherapy in the prevention and cure of purulent infections.
Practitioner, 1914. p. 551.

1662. FISCHER, A. Contribution to the therapy of Pemphigus chronicus and of Dermatitis herpetiformis (Duhring)
Dermat.Woch. 1916. Vol. 62. p. 25.

1663. FÖCKLER, Zur Vakzinebehandlung der gonorrhoea.
Dermat.Woch. 1912. Vol. 55. p. 1395.

1664. FORDYCE, J. A. Discussion on autoserum Treatment.
J.A.M.A. 1914. Vol. 63. p. 1193.

1665. FOX, HOWARD, Discussion on Autoserum Treatment.
J.A.M.A. 1914. Vol. 63. p. 1193.

1666. GOTTHEIL, W. S. The value of Autoserum injections in skin diseases.
New York Med.Journ. 1916. Vol. 103. p. 1209.

1667. GOTTHEIL, W.S. & SATENSTEIN, D.L. Autoserum Injections in certain obstinate Dermatoses. Med.Rec. New York. 1914. Vol. 85. p. 620.

1668. GOTTHEIL, W. S. & SATENSTEIN, D.L. The autoserum treatment in Dermatology. Journ.Amer.Med.Assoc. 1914. Vol. lxiii, p. 1190.

1669. HAREN, P. Ueber die Giftigkeit arteigenen serums und die Anaphylatoxinbildung aus Agar und Gelatine. Zeitsch. f. Immunitatsf. 1913. Bd. 20. p. 673.

1670. HEIDINGSFELD, M. L. Discussion on Autoserum Treatment. J.A.M.A. 1914, Vol. 63. p. 1194.

1671. HEUCK, W. Erfahrungen über Behàndlung Hautkranker mit Menschenserum. Munch.Med.Woch. 1912. Vol.lix. p. 2608.

1672. HILARIO, J.S. A contribution to the Autoserotherapy of certain diseases of the skin. Journ.Cutan.Dis. 1914. Vol.32. p. 780.

1673. KASTENMEYER, B. Über den einfluss normalen antitoxinfreien Pferdeserum auf experimentell erzeugte Diphtherie. Deutsch.Med.Woch. 1919. Vol. 45. p. 1338.

1674. KÖHLER, (Quoted by de Kruif).

Inaug. Dissertation Dorpat. 1877.

1675. KOLMER, A practical Text book of Infection, Immunity and Specific Therapy. 1915, p. 775.

1676. KRAUS, R. Non-specific Treatment of Anaphylaxis. Revista del Instituto Bacteriologico.Buenos Aires. March 1919. 2. No. 1. p. 1.

Abstr, in Journ. A.M.A. Vol. 73. Vol. 7. 1919 p. 567.

1677. DE KRUIF, P.H. The primary toxicity of normal serum.
Journ.Infect.Dis. 1917. Vol. 20. p. 717.

1678. LINSER, P. Über Hauterkrankungen bei Schwangerschaft und deren Heilung.
Dermat.Zeitsch. 1911. Bd. 18. p. 217.

1679. LINSER, P. Über einige mit Serum geheilte Falle von urtikaria.
Med.Klin. 1911. No. 4.

1680. LINSER, P. Über die Behandlung der juckenden Hautkrankheiten mit normalem, menschichem serum.
Dermat.Woch. 1912. Bd. 54. p. 365.

1681. " " Über die therapeutische Verwendung von normalem, menschlichen serum bei Haut und innerlichen Krankheiten.
Arch. f. D.&.S. 1912. Vol. 113. p. 701.

1682. LINSER, P. Über neuere Bestrebungen zur Heilung von Hautkrankheiten durch Blutverbesserung.
Dermat.Woch. 1913. Bd. 57. p. 1239.

1683. LUTHLEIN, F. Zur kenntnis der Wirkung der Vakzens
Wien.Klin.Woch. 1916. Vol. 29. p. 253.

1684. LUX, Fr. Die Behandlung juckender Dermatosen mit Ringerscher Lösung und Eigenblut.
Dermat.Woch. Bd. 59. pp. 1247 & 1273.

1685. MARIANI, G. Aspecific protein Therapy in the treatment of skin and venereal diseases.
Giorn.Ital.d.mal. ven e. della pelle. 1922. Fasc. II. p. 739.

1686. MAYER, A. Normales schwangerenserum als Heilmittel gegen schwangerschaftsdermatosen im besonderen und Schwangerschaftstoxikosen überhaupt.
Zentralblatt. f. Gynäk, 1911. Bd. xxxv. pp. 350 & 1299.

1687. MEYER, S. Experimentelle Untersuchungen über der einfluss normalen Pferdeserum & die infektion der Meerschweinchen mit lebenden Diphtheriebazillen.
Munch.Med.Woch. 1919. Vol. 66, p. 873.

1688. NICOLAS, GATE & DUPASQUIER. Deux cas de prurigos rebelles guéris par l'antohemotherapie.
Annales de D.&.S. 1921. Tome II. p. 127.

1689. PRAETORIUS, G. Pemphigus malignus durch einmalige intravenöse Blutinjektion geheilt
Munch.Med.Woch. 1913. Vol.I. p. 867.

1690. RONGY, A preliminary Report on the treatment of toxaemias of pregnancy with placental serum.
New York State Journ.Med. Jan. 1914. Vol. 14. p. 21.

1691. RUBSAMEN A further contribution to the serum therapy of Pregnancy.
Deutsch.Med.Woch. 1913. No. 20.

1692. SCHENK, F. Ueber gesteigerte Reaktionsfähigkeit gravider Tiere gegen subkutane Gewebsinjektionen.
Munch.Med.Woch. 1910. Bd. 57. p. 903.

1693. SCHULTZ, W. H. Physiological studies in Anaphylaxis.
Bull.Hyg.Lab. U.S. P.H.S. 1912. p. 80.

1694. SMITH, L. D. The value of anaphylaxis in the treatment of gonorrhoeal complications.
J.A.M.A. 1916. Vol. 66. p. 1758.

1695. SPIETHOFF, B. Zur Behandlung mit eigenserum und Eigenblut.
Med.Klin. 1913. No. 24.

1696. " " Zur therapeutischen Verwendung des eigenserums.
Munch.Med.Woch. 1913. Vol.I. p. 521.

1697. SPIETHOFF, B. Die Herabsetzung der Empfindlichkeit der Haut und des Gesamtorganismus durch Injektionen von Eigenserum, Eigenblut und Natrium nucleinicum. Dermat.Woch. Bd. 57 No. 42. p. 1227.

1698. SQUIER, Michigan Med.Soc. Journ. 18. 6. p. 328.

1699. STÜMPKE, G. Über Serumbehandlung von Hautkrankheiten. Deut. .Med.Woch. 1913. Vol. 39. p. 1447.

1700. STÜHMER, A. Die Giftwirkung arteigener Eiweissstoffe. Deutsch. Med.Woch. 1912. Bd. 38, p. 2123.

1701. TCHERNOROUTZKY, Sur l'anaphylatoxine de Bordet. Compt. rend. Soc. de Biol. 1913. T. 74. p. 1213.

1702. TREUBEL, W. Die Behandlung des Ulcus Molle gangrenosum und anderer Ansteckungskrankheiten mit Eigenstoff, Eigenserum oder Eigenblut. Med. Klin. 1915. No. 33.

1703. TRIMBLE, W.B. & ROTHWELL, J.J. The treatment of Psoriasis with Autogenous serum. Journ.Cutan.Dis. 1915. Vol. 33. p. 621.

1704. ULLMANN, E. Über die therapeutische anwendung von Normalserum bei juckenden Dermatosen. Arch. f. Dermat. 1913. Vol. 118. p. 125.

1705. VEIRL, A contribution to the serum treatment of Pregnancy Dermatoses. Munch.Med.Woch. 1912. No. 35.

1706 VELDEN, R. VON DEN, Beiträge zur parenteralen Protein-Körpertherapie Berlin.Klin.Woch. 1919. Vol. 56. p. 481.

1707. WINFIELD, J.M. Discussion on autoserum Treatment. J.A.M.A. 1914. Vol. 63. p. 1193.

NON/

NON-SPECIFIC VACCINE THERAPY.

1708 BIACH, M. Die Tuberkulinbehandlung der Frühlues. Wien.Klin.Woch. 1915. Vol. 28. p. 1345.

1709. DANYSZ, J. Paris Med. 1918. Vol. 9. p. 3.

1710. DANYSZ, J. Antianaphylaxis in treatment of Asthma certain skin diseases and gastro-intestinal mischief. Presse.Med. 1918. Vol. 26. p. 367.

1711. DANYSZ, J. Treatment of acute and chronic anaphylactic Disturbances. Paris medical. 1919. Vol. 9. p. 329.

1712. " " Immunité et Anaphylaxie. Journ.Infect.Dis. 1918. p. 427.

1713. " " Origine, évolution et traitement des Maladies chroniques non contagieuses. Paris. Baillière et fils. 1920.

1714. " " Therapeutic anti-anaphylaxis. Bull.Med. 1920. Vol. 34. p. 155.

1715. ERSETTIG, U. The effect of antityphoid and anti-cholera injections on syphilitic individuals. Giorn.Ital. d.mal. ven e. della Pelle. 1920. Fasc. II. p. 187.

1716. NESFIELD, V. B. Sterilised pus for treatment of infections and sterilised cancer inoculations. Indian.Med.Gaz. 1913. p. 307.

1717. NESFIELD, V. B. The treatment of suppuration by pus inoculations, and the treatment of pneumonia by subcutaneous injections of the patient's own blood. Indian.Med.Gaz. 1914. p. 471.

1718. SEMON, H. C. Psoriasis treated by Danysz's method. B.J.D.&.S. 1922. p. 18.