the humane effects of the new projectile have doubtless been the controlling factors.

The following tables illustrate the effects of the new armament on the target areas of the human body as compared to the conclusions based upon the results of many battles with the old arm. The Santiago campaign, as far as it goes, shows that wounds of the lower extremities are not nearly so frequent as they were observed in former times. The battle tactics which cause a man to lie down to fire, and to advance by rushes have doubtless added much to these results :

TARGET AREAS OF THE BODY.

Region of Body.	Percentage of the whole Area of Body.
Head, face and neck	8.51
Trunk	28.91
Upper extremities	21.14
Lower extremities	41.41
Total	99.97

REGIONAL DISTRIBUTION OF WOUNDS IN THE PRINCIPAL WARS WITH THE OLD ARMAMENT.

Wounds of :	All Projectiles Included.
Head, face and neck	8.56
Trunk	19.56
Upper extremities	23.56
Lower extremities	48.30
Total	99.98

REGIONAL DISTRIBUTION OF WOUNDS IN THE SANTIAGO CAMPAIGN.

Wounds of :	All Projectiles Included.
Head, neck and face	12.50
Trunk	20.45
Upper extremities	27.95
Lower extremities	39.09
Total	99.99

PURPURA HEMORRHAGICA, OR MORBUS MACULOSUS OF WERLHOF.

BY STEPHEN SMITH BURT, A.M., M.D.,
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THE hemorrhages in the skin which are associated with a variety of conditions, and are essentially symptomatic, will not be considered under the heading of this monograph. So also the less serious form of the disease in question, termed "purpura simplex," even though a mild expression of the more serious affection, will be excluded from present consideration. Reference here is made more especially to the grave malady, in which the cutaneous hemorrhages are often of minor importance, known as Werlhof's disease, or purpura hemorrhagica. We are indebted to Paul Gottfried Werlhof,¹ who was born in 1699, and who died in 1767, for its first comprehensive description, and for the name of "morbus maculosus of Werlhof" in consequence.

This disease is characterized by the occurrence of ecchymoses in the skin, the mucous membranes, the viscera, and the serous membranes, while, at the same time, there is more or less profuse hemorrhage from all the mucous surfaces. Much bleeding is quickly followed by a profound anemia, in which the red blood corpuscles and the fibrin-forming elements are rapidly decreased, the white corpuscles increased, and the percentage of hemoglobin is diminished. After several days of general weakness, or suddenly without previous indisposition, a purpuric eruption appears upon the trunk and limbs, but preferably upon the lower extremities. This is fol-

lowed, or accompanied, or sometimes preceded, by bleeding from the gums and other mucous surfaces. Usually the blood lost is non-coagulable. The spots upon the skin, which may appear in successive crops, vary in size from 1 to 4 millimetres in diameter; the smaller being termed petechiae, and the larger ones ecchymoses. In color they shade from a bright to a dark red, according to the depth of the extravasation, and they gradually fade to a brownish hue during their absorption. Unlike inflammatory congestions, they do not disappear upon pressure. Vicerordt suggests the following test, namely, "through a piece of thin glass pressed upon the spot in question, a hemorrhage becomes more distinct, whereas an inflammatory congestion is obliterated temporarily." While the disease may exist without fever, there is usually a moderate elevation of temperature, and sometimes there is hyperpyrexia. The pulse soon becomes weak and rapid; the mind remains clear in some cases to the end, whereas in others there is delirium which lapses into coma towards an approaching fatal termination. Another group of symptoms, wherein multiple arthritis together with urticaria, as well as purpura, are dominant features, is called peliosis rheumatica. There is another group in which nervous symptoms, and still another in which gastro-intestinal and renal symptoms, are uppermost. These are thought by some authorities to be too closely allied to Werlhof's disease to admit of a separate classification.

"Scurvy," says Lockwood,² "if proved an infectious disease, may be really Werlhof's disease modified by the surroundings and poor condition of the patient, and also by the possibility of the infection being more chronic." Purpura hemorrhagica at present is differentiated from scurvy by the presence of fever and by the absence of swelling of the gums, notwithstanding the bleeding, by the fact that a mixed diet has been previously maintained, and by its failure to respond to an antiscorbutic regimen.

That Werlhof's disease is infectious seems to be well established. Adults as a rule are less likely to succumb to the infection than young children. The latter are very prone to die of the disease, and likewise women attacked during pregnancy. Four of the 5 cases complicating the pregnant state, cited by Lockwood,³ had a rapid and fatal termination. "In children the affection nearly always proves fatal," says Dr. Holder,⁴ who had seen a good many cases on Randall's Island. Of the 30 examples of various ages and different sexes included in Lockwood's⁵ paper there was a fatality of 25.

It is a disease of early life ordinarily, and is somewhat less frequent in the male than the female. It is also a treacherous disease, for it may begin with apparent mildness and rapidly become grave and exceedingly dangerous. There are some evidences which point to its communicability, yet this same proof might serve for an argument in favor of a common source of infection. Dr. Holder,⁶ in discussing a paper by Dr. Bremner on the subject, says, "The disease seemed to have a tendency to recur in groups and to affect especially a single ward at a time, so that there was little doubt of its contagiousness." The latter agreed with his colleague, and expressed similar sentiments as a result of a like experience. Moreover, Grüning⁷ has reported 3 cases of purpura simplex in children, three, four and sixteen years old

respectively, 2 of whom occupied the same room, and all of whom lived in the same house and came into frequent contact with one another. All presented high fever and profound constitutional phenomena. Furthermore, in an instance of my own, to be related, the mother of the child stated that 2 other children in her family had previously developed purpuric eruptions upon the lower extremities.

The grave forms of purpura hemorrhagica, at least in this country, fortunately are not of very frequent occurrence. During one year, stated Dr. Lewis,⁸ there were only 3 cases of this form of the disease out of 20,000 general medical cases in nineteen of the hospitals of New York. Among 63,834 medical cases, in sixteen and a half years at the London Hospital, cited by Mackenzie,⁹ there were but 200 examples of purpura of all descriptions. Of these 61 were associated with rheumatism, and 68 were unexplained, and hence Werthof's disease, inferentially.

The *New York Medical Record*, during the past ten years, has published anumber of papers upon this subject, and notably one by Lockwood, already quoted, who gathered 30 cases, 19 of which were from German, 4 from English, 3 from American, 2 from French, and 2 from Spanish sources; also one by Lewis,¹⁰ who reported 3 cases, 2 of which proved fatal. In the *Transactions of the Association of American Physicians*, during this same period, is a paper by Musser,¹¹ who described 5 cases, including 2 deaths. The *New York Medical Journal*, in 1897, published a paper by Weber,¹² who reported 3 cases that recovered, but which were manifestly mild in character. They were of interest in illustration of infection, probably through bacteria, which gained entrance into the system from gangrenous foci in the mouth and the intestines.

The following is a history of a patient who came under my care and observation during my service at the New York Post-Graduate Hospital.

William S., age eleven, admitted to the wards of the hospital December 29, 1898.

Family history.—Father and mother living and in good health. Upon the maternal side two uncles died of consumption. No other record of tuberculosis. A sister and two brothers alive and well, except that two of the children recently had a mild purpuric eruption upon the lower extremities.

Previous history.—The patient has had the usual diseases of childhood. One year ago he was operated upon for enlarged glands in the neck said to be tubercular. Last Tuesday morning (December 27th) he, being apparently in his usual health, began to bleed from the gums, and shortly afterwards the mother noticed a number of small hemorrhagic spots scattered over his body, but more especially upon the lower extremities.

Present condition.—Patient anemic; pulse weak and rapid; temperature 101°; blood oozing from the gums, which are not swollen; a purpuric eruption, moderate in extent and amount, chiefly upon the legs and thighs, the spots varying in size from a mere point to a quarter of an inch in diameter, and in color from a bright red to a faded brownish tinge. These ecchymoses do not disappear upon pressure. Both heart and lungs are normal, no swelling or tenderness of the joints, no edema, no pain, no soreness of the throat and no gastro-intestinal disturbances.

Examination of the urine.—Specific gravity, 1.012;

reaction, acid; color, yellow; transparency, clear; sediment, slight in amount and nebulous. Chemical analysis: Albumin, a trace; no sugar.

Microscopical examination.—Abundant hyaline and finely granular casts; moderate amount of cellular and coarsely granular casts. Blood, few red and white cells. Bacteria present.

During the first four days the patient continued to bleed more or less from the gums, especially at night, and finally from the nose, despite temporary relief from plugging the nostrils and the application of astringents. His mind remained clear, and his strength and appetite fairly good. The temperature varied between 99½° F. and 101¼° F., and the pulse between 110 and 132 per minute. On the fifth day he bled freely from the nose, and vomited considerable blood, and, what is more, passed blood from the bowels, at first in the form of black stools, and then in clots of pure blood. His mind was still clear. He had an exsanguinated appearance; his temperature fell to 97° F. and his pulse rose to 160 per minute. Of course he was in bed. His condition becoming alarming from sheer loss of blood, transfusion of a normal saline solution was performed in his left arm, and stimulants were administered. By midnight he was delirious. The following day his temperature ranged between 99° F. and 100° F., and his pulse between 120 and 140. He had two small black stools. On the seventh day he had six bloody stools, and vomited blood. He was in a stupor with intervals of delirium. His temperature was from 100° F. to 101° F., and his pulse from 138 to 160 in the course of the twenty-four hours. On the eighth day he had a small stool of blood and mucus; his pulse was weak, rapid and irregular; he was delirious. On the ninth day his temperature ranged from 99½° F. to 102° F., his pulse from 140 to 150, and his respirations from 34 to 40 per minute. Finally, upon the tenth day, which was the 7th of January, with temperature 101½° F., respiration 42 and pulse 156 per minute, he died towards evening.

These comprise the essential facts which have been abstracted from the daily records. Besides plugging of the nostrils and transfusion, numerous remedies, including ergot, digitalis, hemostatics and stimulants, without avail were employed. In view of the mortality of the disease, and the contradictory statements in regard to the efficacy of remedies, one is inclined to be sceptical about the value of all medication, certainly in the virulent forms of purpura hemorrhagica. Dawson¹³ reported a case, which, however, was evidently not of a grave nature, where one deep injection of 5 minims of ergot worked a cure immediately. On the other hand, Mackenzie¹⁴ states that ergot may be tried, but that it has not in his hands proved very efficacious. Arsenic, turpentine, ergot, benzoate of sodium, nitrate of silver, iron preparations, all have their advocates. Meanwhile, like several other diseases, the mild cases are likely to get well under a diversity of treatment, and the severe cases are very prone to die, drugs to the contrary notwithstanding. This, at all events, is certainly true in regard to young children. Dr. Holder¹⁵ states that, "therapeutic experience on Randal's Island had rather fostered the idea that there is no specific medication for these hemorrhagic conditions in children." Blood serum, it is said, in doses of 250 cubic centimetres has been employed successfully. It is not impossible that the se-

rum of some naturally immune animal, which has been inoculated with a sterilized culture of the bacillus described by Tizzoni and Giovannini, may eventually find useful therapeutic application. Meantime, the discovery of the cause of this not infrequently malignant infection should exert a direct influence upon the prevention as well as the treatment.

Mackenzie¹⁶ remarks that "bacteria or their products must play an important part in the production of the cutaneous hemorrhages," and also that "it is certain the cutaneous hemorrhages are sometimes determined and in all probability primarily caused by nervous influences." Stengel¹⁷ says in connection with the subject under consideration that "organisms have been found in the blood by a number of observers. No definite and specific bacteria have, however, been isolated, and it is doubtful whether the infection in all cases is the same." Letzerich,¹⁸ in 1889, described a long bacillus having sharp angles and edges which he considered peculiar to this affection. These bacilli can be cultivated "from the blood of the petechia, and pure cultures in gelatine injected into rabbits give rise to hemorrhages spontaneously. A hyaline thrombus full of bacteria and spores was always found at the site of the bleeding."

Lapin¹⁹ mentions 2 cases of purpura hemorrhagica, 1 of which ended fatally, and in both of which micrococci were found in the blood. In the fatal case streptococci were present. In his opinion the infecting principle enters the body by the mouth and the pharynx. The proximate cause of purpura hemorrhagica is believed to be a hyaline and amyloid degeneration of the walls, both intima and adventitia, of the blood vessels, with ultimate hemorrhage, and by rupture of the vessels rather than by diapedesis. It seems probable that any unusual pressure of the circulation against the vessels, previously weakened by degenerative changes, would be sufficient to precipitate the extravasation. What is known regarding the part taken by bacteria or their products in the causation of these degenerations will be shown in the following extracts from Flügge's²⁰ "Die Mikroorganismen."

The hemorrhagic infections of man have not as yet been wholly elucidated, at least they have no uniform etiology. In many cases the process is dependent upon the especial malignant character of an infection otherwise progressing without hemorrhagic diathesis, or upon an abnormal disposition of the affected individual; in others upon specific microorganisms causing fragility of the vessel walls. Cornil and Ranvier* and Babes,† giving examples, distinguish three groups of causes: (1) Infection exciters, which are to be classed along with bacteria or hemorrhagic septicemia in animals; (2) such bacteria as gain access from gangrenous foci in the mouth and intestinal canal (*Bacillus proteus septicus et letalis* of Babes, *Bacillus capsulatus septicus* of Foa and Bonome, Bardon-Uffreduzzi, and pyogenic cocci); (3) violently acting streptococci. Compare Claisse.‡

Bacillus hemorrhagicus septicus. — Cultivated by Babes§ from 3 fatal cases of hemorrhagic sepsis accompanied by stomatitis, angina, bronchitis, purpura, hematuria and fever. In the hemorrhagic pulmonary foci and mesenteric glands there were found dense masses of bacilli. From the spleen and lungs the following bacilli were obtained in pure culture: Non-motile short rods (.3-.4 millimetres thick), surrounded by a capsule. Stain faintly with aniline dyes, still fainter with Gram's method. No

spore formation. Facultative anaërobic. Slight growth on surface of gelatine; along the stab a granular stripe; no liquefaction. Upon agar-agar minute transparent droplets; later, whitish-yellow, indistinctly defined spots. On potato, whitish drops. Bouillon is clouded.

Mice die within a few days, often with manifestations of septic changes (spleen tumor), with hemorrhages upon the serous membranes. Rabbits die in three to eight days with hemorrhages in all the organs, especially in the liver and lungs, and with swelling of the spleen. In those animals which die late the bacilli are often no longer microscopically demonstrable. Sterilized cultures (filtered or heated to 60° C.) also produce multiple hemorrhages. For guinea pigs and dogs it is slightly pathogenic. The cultures rapidly lose their virulence.

Bacillus hemorrhagicus (Kolb). — Isolated in pure culture from the cadavers of three persons who died in three or four days with fever, hemorrhages in the skin and mucous membranes.* Besides pin-head to twenty-five-cent piece sized hemorrhages in all the organs, the early autopsies show no other changes. The bacilli lie in all sections, especially numerous in the spleen, partially in clumps, partially isolated. Non-motile rods, .8 : 1-2 μ , also in long threads with slight capsule, which, however, is inconstant. They are in great part decolorized by Gram's method. No spore formation. Facultative anaërobie. Superficial colonies in gelatine show serrated periphery; no liquefaction. Upon agar-agar smooth, non-serrated layers. On blood serum thin, moist stripe; the same on potato. Bouillon is first clouded, then (on sixth day) simply a sediment. Slight reduction power. Exact biological description is wanting, as in the preceding bacilli.

A drop of bouillon kills mice in two or three days with numerous multiplication of the bacilli, which are found in especially large numbers in the organs; quite numerous small hemorrhages, and spleen tumor. Guinea pigs are susceptible to infection only on administration of large doses. Rabbits frequently die within one to three days after intraperitoneal injection of $\frac{1}{2}$ to 1 cubic centimetre, with production of extensive hemorrhages. Bacilli everywhere. Doves are not susceptible. In dogs hemorrhages are also noted, even after twenty days, if they do not die too quickly, as do rabbits. Sterilized cultures in doses of 3 to 3 cubic centimetres produce in experiment animals phenomena similar to those caused by the living bacilli.

Bacillus hemorrhagicus velenosus. — A non-motile bacillus, isolated by Tizzoni and Giovannini in a case of purpura hemorrhagica, secondary to an impetigo contagiosa, which came to autopsy. The bacillus occurred, associated with staphylococcus pyogenes aureus, in the hemorrhagic foci of the skin occupied by impetigo pustules, also in the liver, and venous blood, but not in the spleen and kidneys. The staphylococcus was found in the pure hemorrhagic foci of the skin and also in the kidneys.

The bacillus is pathogenic for dogs, rabbits and guinea pigs, but not for doves and mice. The bacilli multiply locally only (edema); they cause fever, hemorrhagic nephritis, vomiting, bloody stools, and cutaneous hemorrhages. Spleen normal; coagulation necrosis of the liver and renal epithelia; non-coagulability of the blood. Cultures sterilized at 70° C. produce albuminuria; repeated injections of the same immunize against subsequent infection.

In spite of admixture with staphylococci, this bacillus, as its behavior toward animals proves, is said to be worthy of consideration as the excitant of purpura.†

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EYE STRAIN AS A CAUSE OF GASTRO-INTESTINAL NEUROSES.¹

BY M. P. SMITHWICK, M.D., BOSTON.

"A GASTRIC neurosis is the local expression of some constitutional state or nervous irritation arising outside of the stomach." If this definition taken from the "American System of Practical Medicine" is made to include both stomach and intestines, it describes accurately the class of cases on which my remarks are based.

In searching for the cause of a gastro-intestinal neurosis one should avoid "snap" diagnosis. It isn't sufficient to discover astigmatism, deafness, inharmoonious environment, exhausting occupation, or other single cause of nervous fatigue. It is my custom to learn the temperaments of my patient's parents, brothers and sisters; to get a clear mental picture of the early home life, and review carefully the patient's life, endeavoring to see his environment from his point of view. By ascertaining when health commenced to fail it is possible frequently to determine the cause. All this requires time and patience, but often reveals a suitable background for the picture as we see it. Every means of diagnosis that can contribute to an accurate knowledge of the whole organism should be employed. Of course this includes a careful study of the gastric and intestinal functions. The eyes should be examined by an oculist.

One can attempt the removal of all possible causes or commence with the most probable. The latter allows the more intelligent study and is my preference. If, for example, the oculist reports defects to be corrected, and the eyes are probably responsible for the nervous exhaustion, the patient is allowed to wear glasses for a time, only dietetic errors being corrected.

After all possible causes have been considered it might seem easy to determine the real one. A little experience, however, teaches one that digestive neuroses tend to disappear spontaneously for considerable periods, improve temporarily under almost any treatment, and often persist for a long time under treatment that finally effects a cure. It may be impossible to state which of several possible causes was the real cause where all have been removed and the neurosis cured.

Treatment of the digestive tract is frequently necessary, but when once the diagnosis of digestive neuro-

sis is established, our chief aim should be to discover and remove the cause.

The first case which particularly directed my attention to this subject was a barber, thirty years of age, who consulted me in July, 1898, for so-called bilious attacks, from which he had suffered every two or three weeks since he began that work, at sixteen years of age. Previous to that time he was well, and excepting these attacks has always been well. His appetite has always been good, and the only digestive symptoms are moderate constipation and occasionally "sour" stomach. Between attacks headache is present only when he rides on cars, or occasionally when the electric lights are turned on in the shop. An attack may be preceded a day or so by the sensation of a "lump" in the throat, and commences with vertigo, partial loss of sight, nausea, vomiting and, for three or four hours, frontal headache. Symptoms of hyperacidity are more likely to annoy him before an attack.

Physical examination negative, except for exaggerated reflexes. Nothing uniform about gastric analyses. At times delayed motility and hyperchlorhydria.

From July until late in October I dieted and treated him in every way that occurred to me, and then stopped trying; told the patient that I had not helped him at all, and did not know the cause of his attacks. I sent him to an oculist and glasses were ordered for .75 D hypermetropia. The oculist suggested that the attacks might be scintillating scotoma and was not sanguine; I was not, and the patient wore glasses because asked to do so. Some months later he made me a social call, and asked why the attacks had ceased. Apparently he could not believe me when I insisted that glasses and not medicine had done it. Later he broke the glasses and was uncomfortable, but unfortunately for the cause of eye strain the optician repaired them in two days. Since October, 1898, he has had one slight attack and one severe one, the latter in December, 1898.

Early in November, 1898, I was consulted by a man, thirty-two years of age, clerk in a railway office. His work made severe demands on his eyes, and great accuracy was necessary. He was well until June, 1897, when he had an attack of vomiting which he attributed to strawberries and ice-cream soda, eaten after a long bicycle ride. In December, 1897, he was working on dividends, and the coupons were on green paper and caused headache. For a short time he wore glasses fitted by an optician. The attacks were usually worst when his work was most trying, and might be preceded an hour or so by sour taste. Then suddenly would come nausea and intense vomiting. This might last a day or several days. In the first attacks observed by me, there seemed to be no pain, except that at times after vomiting he complained of pain apparently caused by the exertion. Later he seemed to be in great distress. No headache. He was inclined to constipation, and worried much over this symptom, but usually had a dejection every day except Sunday. He was very neurasthenic. There was an indefinite specific history.

Physical examination was negative except for diminished pupillary light reflex, diminished knee jerks, and rather prominent abdomen. No inco-ordination. Several times fecal masses in the ascending and descending colon were palpated. In February, 1899, I referred him to two specialists for an opinion as to

¹ Abstract of a paper read before the Clinical Section of the Suffolk District Medical Society, May 16, 1900.