Public Health Reports

Vol. 59 • OCTOBER 27, 1944 • *No. 43*

PATHOLOGIC REACTION TO THE VIRUS OF LYMPHOCYTIC CHORIOMENINGITIS IN GUINEA PIGS [1]

By R. D. LILLIE, *Senior Surgeon*, and CHARLES ARMSTRONG, *Medical Director*, *United States Public Health Service*

In the course of our studies of this virus (*1*, *2*, *3*, *4*, *5*), a series of guinea pigs was inoculated and their organs submitted for study at varying intervals after inoculation. As certain unexpected and interesting findings were revealed it seemed worthwhile to make a brief report.

The material comprised tissues from 18 guinea pigs killed 4 to 30 days after inoculation. Most of this material was obtained in 1936 and 1937.

The gross post-mortem findings were generally inconsiderable. A slight splenic enlargement was noted in four of the animals. Congestion or areas of consolidation of the lungs were found in five, not differing materially from the common pneumonia of guinea pigs. Peritoneal congestion, following intraperitoneal inoculation, necrosis at the site of subcutaneous inoculation, and slight enlargement of lymph nodes adjacent to the subcutaneous inoculation were noted in one guinea pig each. Material for histologic examination was fixed in Orth's fluid, imbedded in paraffin, sectioned, and stained by our Romanovsky method (*6*) and by Weigert's iron chloride hematoxylin with van Gieson's picrofuchsin.

The brains of 2 animals killed 4 days and 1 killed 30 days after inoculation showed no lesions. Twelve killed 6 to 27 days after infection showed scattered nodules or patches of lymphocyte infiltration in the pia mater of the base or major fissures or in the chorioid plexus of one or more ventricles, or in both. Generally the pia mater over the convexity was normal, and usually the chorioid plexus in at least one of the ventricles was uninvolved. Altogether chorioid plexus was found in sections in 37 ventricles and foci or patches of

[1] From the Pathology Laboratory and the Division of Infectious Diseases, National Institute of Health.

lymphocyte infiltration of the plexus in 15. In a few guinea pigs lymphocyte infiltration of the sheath of one or more intracerebral vessels was found, and in 1 there was a fairly definite "node" of mesoglia cells in the medulla.

Altogether the reaction is strikingly less marked than in monkeys or mice (*1*, *2*). Perhaps it is more evident between the eighth and twenty-first days after inoculation.

In mice, Rivers and Scott (*3*) noted only slight gross cerebral congestion. Microscopically, there were meningeal congestion, occasional hemorrhages, and lymphocyte infiltration. Slight subependymal and chorioid plexal lymphocyte infiltration was noted. Intracerebral perivascular lymphocyte infiltration was rare in the acute stages, but perhaps considerable in amount in chronic cases and in animals killed 2 or 3 weeks after recovery.

In guinea pigs inoculated intracerebrally they noted a mild meningeal reaction characterized by infiltration by mononuclear cells. The brain and cord showed little or no reaction. After subcutaneous inoculation the meningeal reaction was minimal.

In naturally infected mice Traub (*4*) found a slight, predominantly lymphocytic meningitis in 1 of 12 mice. After intracerebral inoculation a more or less pronounced meningeal infiltration was constantly present. The infiltrating cells were chiefly lymphocytes, next in numerical importance were mononuclear cells, and a small percentage were polymorphonuclear leucocytes. The meningitis was usually most marked at the base of the brain, and often extended also to the cord. Thirteen of 17 mice showed round cell infiltration of the chorioid plexus, more marked in the third and fourth than in the lateral ventricles. Some round cell exudation into the ventricles was noted; ependymal infiltration and subependymal gliosis were frequent. Extension of the round cell infiltration into the sheaths of submeningeal and subependymal vessels was noted in 10 of 15 mice. Areas of round cell infiltration of the brain stem, small clumps of oligodendroglia in the basal cortex, pyknotic and shrunken Purkinje cells, and occasional degenerated anterior horn cells surrounded by oligodendroglia and microglia were noted.

Intraperitoneal inoculation of mice gave a slight meningitis in one mouse, without plexal involvement. Moderate meningoplexal infiltration resulted in some mice inoculated intravenously.

Guinea pigs inoculated by various routes by Traub (*4*) inconstantly showed a meningeal and slight plexal infiltration by lymphocytes and mononuclears of much less intensity than in mice. Slight perivascular lymphocyte and mononuclear cell infiltration was rarely seen within the brain. Traub described single and multiple eosinophilic intranuclear inclusions in some of his guinea pigs. These occurred in pia cells, mononuclears in pia, vascular adventitia cells and subpial glia

cells. These inclusions were absent in the lungs of guinea pigs and in mice. Traub is doubtful of their significance. Findlay and co-workers (7) discount them and state that Thompson (8) has recently shown them to be due to a virus found in salivary glands of mice. Findlay et al. saw them in plexal epithelium in a few mice.

Rats inoculated intracerebrally by Traub (4) showed marked plexal round cell infiltration in the lateral ventricles and a meningitis intermediate in intensity between those seen in mice and guinea pigs.

Findlay, Alcock, and Stern (7) noted in intracerebrally inoculated guinea pigs an intense basilar leptomeningitis in which the exudate was predominantly lymphocytic with some admixture of plasma cells and polymorphonuclears. Similar exudate distended the villi of the choroid plexus and filled the ventricles. A little subependymal gliosis was the only intracerebral reaction. We have found a chronic proliferative ependymitis to be a quite common condition in guinea pigs in a variety of experimental and nonexperimental conditions. Mice showed a similar intense reaction, monkeys a less striking one, in contrast to our findings (1, 2).

The heart has constantly shown lesions of greater or less extent and intensity. The lesions consist of focal infiltration by lymphocytes, of focal proliferation of fibroblasts, interstitially, beneath the endocardium and about small vessels, and of combined foci of lymphocyte infiltration and fibroblast proliferation. Concentric endothelial proliferation in small vessels accompanies the other manifestations in some cases.

These lesions occur constantly in the myocardium of atria and ventricles, are almost constant and more extensive, more marked, and more frequent beneath the mural endocardium, and usually are found in the epicardium, particularly in the atrioventricular sulcus. Valve lesions were found in 13 of the 18 guinea pigs, involving the mitral leaflets in 8 of 12 animals, the tricuspid in 4 of 11, the aortic cusps in 6 of 12, and the pulmonic in 2 of 3 animals. The method of longitudinal sectioning used did not regularly show all of the valves.

In one case (11 days) the only alteration of the valves was a focal interstitial exudation of metachromatic mucoid material with consequent thickening in both mitral leaflets, the base of a tricuspid leaflet, an aortic cusp, and the septal endocardium below the aortic insertion. A similar myxomatoid swelling of the aortic cusps was present in another guinea pig with proliferative alterations in the mitral valve (17 days) and in one guinea pig (8 days) myxomatoid swelling of the mitral leaflets was associated with focal proliferation of small fusiform fibroblasts just beneath the atrial surfaces. This patchy proliferation of small fusiform cells within the valve substance was the usual valve lesion, occurring usually beneath the atrial surfaces of the mitral and tricuspid valves and beneath the ventricular or, less often, arterial

surfaces of the semilunar cusps. In six valves or pairs of valves it was the only alteration. In one there was an admixture of lymphocytes. In two fibroblasts proliferation was mixed with lymphocyte and monocyte infiltration with an occasional leucocyte and a little karyorrhexis. In five valves there was associated with and overlying the focus of fibroblast proliferation a stratifying proliferation of the endocardial endothelium, reaching perhaps four layers of fusiform cells. One guinea pig (17 days) showed a small concentric nodule of loosely disposed fusiform fibroblasts in a myxomatous stroma in the base of a mitral leaflet.

The more conspicuous mural subendocardial foci more often showed mixture of lymphocytes with the proliferating fusiform fibroblasts, but stratifying endocardial proliferation was absent. Concentric endothelial and adventitial proliferation of small subendocardial vessels was associated in a number of instances. In one case (6 days) mixed focal subendocardial infiltration by lymphocytes and pseudoeosinophil leucocytes was present, in one (7 days) some monocytes were mixed with the fibroblasts and lymphocytes, and in a third (9 days) besides the fibroblasts and lymphocytes there were plasma cells, monocytes, pseudoeosinophil leucocytes, and a little pyknotic nuclear debris in some foci. In one guinea pig (17 days) with rather marked focal subendocardial lymphocyte infiltration and less fibroblast reaction, there was a small fibrinoid thrombus adherent to the endocardial surface over one of the focal lesions.

Myocardial foci occurred both in atria and ventricles. They were apparently more frequent and larger in the papillary muscles than elsewhere. In one case (25 days) a diffuse focus of lymphocyte infiltration and fibroblast proliferation in the ventricular septum near the base lay partially in the bundle of His. In another (6 days) there were a few small foci in the muscle or adjacent to arterioles, composed of interlacing, or less often fasciculated, large fusiform fibroblasts, some of which showed some lymphocyte infiltration. In about half of the animals some of the ordinary lymphocytic and fibroblast lymphocytic foci were perivascular in location, in some of them obvious adventitial proliferation of the small vessels occurred, and in four there was a concentric stenosing endothelial proliferation in small vessels.

Epicardial reactions were mainly in the form of interstitial and perivascular lymphocyte infiltration, fibroblast participation was less frequent, vascular adventitial and endothelial proliferation were infrequent. Interstitial serous exudation was occasionally seen. Plasma cell, monocyte, and polymorphonuclear admixture were recorded in one case (9 days). In one guinea pig (7 days) there was a nodule in the atrioventricular sulcus composed of large fusiform fibroblasts and infiltrated by lymphocytes and fewer monocytes and pseudoeosinophil

leucocytes. In one animal (17 days) there was some mesothelial swelling and desquamation.

The greater frequency of focal lesions in the heart is quite striking when compared with the lesions in rhesus monkeys (*2*).

Traub (*4*) noted in the majority of his guinea pigs subendothelial infiltrations with round cells, and very small, scattered round cell collections in the myocardium and epicardium. The muscle, as in our animals, was not affected.

The lungs constantly showed perivascular infiltration by lymphocytes of greater or less grade. This finding is so common in guinea pigs that its significance is doubtful when present in slight or moderate grade. However, in about half of the animals it was quite marked. In five guinea pigs it was so marked that secondary follicles or germinal centers were formed in some or most of the perivascular nodules.

As is common in guinea pigs, patches of atelectasis or of carnifying pneumonia, ranging from early fibroblast organization of a serofibrinous exudate through interstitial infiltration by fibroblasts, monocytes, or foamy epithelioid cells to a slight residual interstitial lymphocyte infiltration were found in 13 animals.

Mucoepithelial, mucopurulent, and serous exudates were often seen in the lumina of the bronchi, and hyperplasia of the mucosal lymphoid follicles was present in a few animals.

Possibly the more marked perivascular lymphocyte infiltration with formation of germinal centers is significant, as it was found in some animals without evidence of antecedent pneumonic changes and without purulent bronchitis.

In two animals the pleura showed flattened plaques of mesothelial proliferation. In one of them there was a subjacent irregular lymphocyte infiltration. In the other there was mesothelial desquamation and exudation of polymorphonuclears, monocytes, and phagocytic macrophages (pericardial pleura).

The nodular perivascular lymphocyte infiltration is common to guinea pigs and monkeys (*3*), while the congestion and edema seen in the latter are generally lacking in the former.

In the lungs of mice, Rivers and Scott (*3*) noted frequent areas of discoloration and consolidation which were (culturally?) free from bacteria. Microscopically these were an interstitial bronchopneumonia characterized by perivascular round cell infiltration and, in definitely consolidated areas, dense interstitial and peribronchial infiltration by mononuclear cells, desquamation of bronchial epithelium, irregular atelectasis or alveolar exudation of fibrin and few cells, usually mononuclears. Some sections showed only capillary congestion and a few alveolar hemorrhages.

In guinea pigs also they noted an interstitial bronchopneumonia, of which they published no further description. Their illustration

apparently indicates some interstitial cellular infiltration and extensive intra-alveolar organization.

In Traub's (*4*) intravenously infected mice the lungs, while grossly normal, showed more or less extensive round cell collections about vessels and bronchi, and an interstitial pneumonia in every case. This interstitial pneumonia was inconstant after intraperitoneal inoculation. After intracerebral inoculation slight septal thickening and an inconstant, slight peribronchial round cell infiltration were noted. Natural infection showed in two mice slight peribronchial and perivascular infiltration by round cells and slight septal thickening.

In guinea pigs Traub (*4*) noted frequent more or less extensive consolidation in advanced stages. Cultures were sterile in many cases, while in others various bacteria were cultivated. Histologically he simply notes a "typical virus pneumonia." Marked pulmonary edema was present in many guinea pigs.

Traub (*5*) noted interstitial pneumonia in five of seven mice killed 3 to 7½ months after infection while still carrying virus. He further noted the absence of peribronchial, perivascular, and interstitial round cell infiltration in the lungs of seven normal stock mice killed for histologic controls.

Findlay, Alcock, and Stern (*7*) noted the occasional presence of a virus pneumonia in mice, without giving further particulars, but made no mention of lung changes in guinea pigs or monkeys.

In the larynx, trachea, and extrapulmonary bronchi slight diffuse or denser focal lymphocyte infiltration was often present in the mucosa, and lymphoid follicles were sometimes encountered. Less often the mucosa was recorded as normal. Mucosal lymphocyte infiltration was usually associated with subacute pneumonic processes or purulent bronchitis or both, hence it is probably not significant. However, a similar reaction occurred in some monkeys (*2*).

In 1 animal with a nodular carnifying pneumonia there was an esophagitis characterized by focal lymphocyte infiltration of the corium, leucocyte emigration through and desquamation of the epithelium, and purulent exudate in the lumen. In 13 guinea pigs the esophagus was histologically normal. Similar negative findings were usually recorded in monkeys (*2*).

The submaxillary gland was normal in nine animals. In three there were patches or occasional foci of lymphocyte infiltration and in two an occasional solitary lymphoid follicle with germinal center was included within the gland. Possibly these lymphocytic foci are more frequent than in monkeys (*2*). Traub (*4*) found no lesions.

Thymic tissue was found adjacent to the submaxillary gland in nine animals. Partial involution was present in two, and in a third the cortex was hyperplastic, the cortical reticulum cells contained phagocytosed nuclear fragments, and there was polymorphonuclear

invasion of the parakeratotic Hassall bodies. In six there were no lesions. Monkeys showed no lesions (*2*).

In 10 guinea pigs the stomach was normal, in 2 it showed slight to moderate congestion, in 1 there were scattered clumps of lymphocytes in mucosa and muscularis, and in 3 focal destructive lesions were present in the mucosa. In one of the last the lesions were focal mucosal engorgement and hemorrhage with denudation of epithelium and free and attached masses of bacteria and necrotic epithelial cells on the surface. In another there were multiple hemorrhagic coagulative infarcts of the mucosa with necrosing thrombosed arteries and veins in and beneath the infarcts, edema of the surviving gastric wall, and focal vascular endothelial and adventitial proliferation with perivascular lymphocyte infiltration in the serosa. The last animal presented a small, partially epithelialized, granulating ulcer in the gastric mucosa. The destructive lesions have no parallel in monkeys, but much less diffuse infiltration is present in guinea pigs.

The small intestine was generally normal. Clumps of pigmented macrophages were present in the villi in two animals. In one there was an enteritis with pus in many crypts and infiltration of the villi by intact and fragmenting monocytes and polymorphonuclears; this was the animal with focal gastric hemorrhages.

In two other guinea pigs the colon showed respectively a patchy subepithelial edema and mucosal macrophage infiltration, and an acute colitis with mucosal edema, polymorphonuclear infiltration, pus in crypts, and lymphoid follicle hyperplasia.

Colonic, cecal, and sometimes ileal, lymphoid follicles often showed hyperplasia with accumulation of nuclear debris in the lymph clefts and swollen phagocytic reticulum cells of their germinal centers.

One guinea pig presented a small (1 mm.) shallow, suppurating ulcer in the cecum. In several, mucus secretion in the crypts was quite active. Focal perivascular lymphocyte infiltration and fibroblast infiltration were seen in the cecal submucosa of one animal. Otherwise the intestines were normal.

Summarizing, six guinea pigs presented acute ulcerative, exudative, or destructive processes in the gastrointestinal mucosae, whose relationship to the disease under consideration is questionable. The follicle hyperplasia and phagocytic activity are in agreement with similar processes seen in other lymphadenoid tissues in infections with this virus. The focal proliferative vascular lesions with lymphocyte exudation seen in two guinea pigs are similar to those seen in other organs and tissues and appear significant.

Similar lymph follicle reactions, and nonspecific processes were seen in monkeys (*3*).

The omentum and mesentery were normal in 12 guinea pigs. In

1 there was an omentopancreatic abscess with marginal organization, associated with granulomata and staphylococcus abscesses in the spleen and granulomata in liver and renal pelvis. Foci of perivascular and submesothelial lymphocyte infiltration were present in the remaining 5 animals. In 2 of these there was some associated focal fibroblast proliferation just beneath the surface, and in 1 many patches of stratifying mesothelial proliferation with subjacent capillary endothelial and fibroblast proliferation and lymphocyte infiltration.

After intraperitoneal inoculation of mice Traub (*4*) found a serous pleuroperitonitis in about 20 percent, characterized by mesothelial swelling and slight subjacent round cell infiltration.

The liver showed no lesions in three animals killed 25, 27, and 30 days after inoculation. In three killed 4, 4, and 6 days after inoculation slight congestion was present in one, slight fatty infiltration in one, and both in the third. Eight guinea pigs, killed 6 to 14 days after inoculations, showed slight to moderate periportal lymphocyte infiltration, and two of these (9 and 14 days) and three more (16 to 18 days) showed moderate to rather marked filling of the liver cells by coarse fat vacuoles, more toward the centers of the lobules.

In addition five of these guinea pigs (6, 8, 8, 11, and 11 days) showed scattered small clumps of proliferating Kupffer cells and lymphocytes, grading in two into nodules of epithelioid cells and lymphocytes, which in one (11 days) included a few shrunken coagulated necrotic liver cells and showed marked hemosiderin pigmentation of the epithelioid cells.

In two animals (9 and 21 days) single foci of recent coagulation necrosis without marginal proliferation were found, in two (14 and 18 days) multiple focal coagulative necroses with patent capillaries and surviving or proliferating endothelial cells were present, and in one (7 days) there was a large area of centrally anemic, marginally hemorrhagic, coagulative necrosis with multiple satellite foci of centrolobular, hemorrhagic, coagulative necrosis, and dense lymphocyte infiltration and slight fibroblast proliferation in the abutting surviving hepatic tissue. One guinea pig showed multiple granulomata including a few necrotic liver cells and some leucocytes. This animal also had multiple granulomata and abscesses in the spleen with contained staphylococci, an omentopancreatic abscess, and granulomata in the renal pelvic fat.

Thus focal necroses or proliferative lesions were present in 10 of 11 guinea pigs killed from the sixth to the twenty-first days. The lesions in the last-mentioned animal were probably due to secondary infection.

Monkeys showed a similar incidence of focal necroses but comparatively little tendency to proliferative focal reactions (*2*).

Rivers and Scott (*3*) noted in mice some engorgement of capillaries, a definite Kupffer cell increase, and a few small areas of focal necrosis.

Traub (*4*) frequently noted in mice small collections of round cells about blood vessels, scattered single and clumped lymphocytes in the

parenchyma, and patchy Kupffer cell hyperplasia in naturally infected mice. In intracerebrally inoculated mice similar but less marked changes were noted, while after intraperitoneal inoculation perivascular lymphocyte and mononuclear cell inoculation was more marked, and after intravenous inoculation necrosis of some liver cells adjacent to round cell infiltration was noted.

In guinea pigs Traub (*4*) saw frequent fatty vacuolation of liver cells, often small perivascular clumps of round cells, and necrosis of groups of adjacent liver lobules in about 20 percent of the animals. Traub questions the significance of this necrosis.

Findlay, Alcock, and Stern (*7*) noted the presence of fatty degeneration, focal necrosis, and accompanying round cell infiltration in the livers of guinea pigs and monkeys (*M. irus* and *rhesus*), and of Kupffer cell swelling in mice.

Splenic enlargement was infrequent, congestion inconstant and usually of minor grade, and follicles were usually moderately hyperplastic. Moderate numbers of or numerous polymorphonuclear leucocytes were present in the pulp in all animals killed 4 to 11 days after inoculation, very few or none thereafter. After about 6 days a moderate to marked pulp reticuloendothelial swelling appeared and was present in all animals up to the latest studied (30 days). Free macrophages were present in the pulp in a few of these. Erythrophagia was common from the sixth to the eleventh day and was accompanied by moderate hemosiderosis which apparently became more marked from about the eleventh to the eighteenth day, diminishing thereafter. A moderate focal lymphocyte infiltration of the pulp was noted in 9 of 16 animals killed 6 or more days after inoculation. The reticulum cells of the splenic follicles were swollen and contained ingested nuclear debris in greater or less quantity, sometimes also leucocytes in 11 of the 16 guinea pigs killed after the fourth day.

In one guinea pig granulomata and granulomatous abscesses containing staphylococci were present. This animal also had granulomata in the renal pelvic fat and liver and a granulating omentopancreatic abscess.

In general, splenic reactions are of similar nature but more intense in grade than those seen in monkeys (*2*). The latter species did not show the polymorphonuclear reaction seen in the earlier stages in guinea pigs.

Rivers and Scott (*3*) stated that in mice the spleen showed nothing particularly characteristic and that the other organs (besides liver, lungs, and brain) showed no characteristic changes.

Traub (*4*) noted in mice after intraperitoneal inoculation a 50 to 100 percent enlargement of the spleen, after intravenous inoculation, up to six times. Miscroscopically there were follicle hyperplasia, pulp reticuloendotheliosis, and, with more marked enlargement,

pulp infiltration by lymphocytes and mononuclear cells. After intracerebral inoculation and in natural infections, mice showed no changes in the spleen.

In guinea pigs Traub (*4*) found no definite lesions in the spleen. Splenic corpuscle hypertrophy was present in 2 of 7 virus-carrying mice (*5*) dead respectively 7½ and 6 months after infection. The first also showed pulp megakaryocytosis, the second splenic enlargement and lymphocyte infiltration of the pulp.

The femoral marrow was one-third to one-fourth fatty in 2 guinea pigs (14, 30 days) and solidly cellular in the other 12 studied. Congestion and small nodules of reticulum cells were noted in 1 animal (8 days), a little erythrophagia in another (18 days), and foamy epithelioid cell granulomata with a few central polymorphonuclears in the animal with splenic abscesses, omental abscess, and renal and hepatic granulomata. Otherwise there were no focal lesions.

Pseudoeosinophil myelocytes were generally the predominant cell type; in three animals killed 17, 18, and 21 days after inoculation there were appreciable to considerable numbers of nongranular promyeloid cells. Polymorphonuclear leucocytes were generally numerous in animals killed 4 to 14 days after inoculation, few thereafter. Similarly, erythropoietic activity seemed greater in the first 2 weeks than later, while megakaryocytes were relatively reduced in numbers from the ninth to the eleventh day.

The guinea pigs showed an early leucocytic reaction not observed in monkeys (*2*).

According to Trab (*4*), three of seven intravenously inoculated mice showed marked leucocytosis, up to 55,000 per cu. mm. Monocytosis (4 to 23.5 percent) was present in six mice, lymphocytosis (62.5 to 80 percent) in five. Naturally infected mice, and those inoculated intracerebrally and intraperitoneally showed no blood changes.

Lymph nodes were studied in 14 guinea pigs, cervical in 8, mediastinal in 7, abdominal in 6, pelvic in 2 and unidentified in 1. A few nodes were recorded as normal, a few showed congestion or slight edema, and a few follicle hyperplasia of slight grade. More often there were a more marked swelling and hyperplasia of the follicles with more or less swelling and ingestion of pyknotic nuclear fragments by the intrafollicular phagocytic reticulum cells. Sometimes this was accompanied by swelling of reticulum cells in the pulp cords, and more often by slight to moderate swelling of the sinus reticuloendothelium. Erythrophagia and hemosiderosis were found in one group of abdominal nodes (21 days), slight macrophage exudation in another (25 days).

The reactions are quite similar to those seen in rhesus monkeys (*2*).

In guinea pigs Traub (*4*) found no lesions of lymph nodes. In mice inoculated intravenously he noted small lymph nodes with reticuloen-

dothelial hyperplasia in some. Naturally infected mice and those inoculated by other routes showed no lesions. One of Traub's (*5*) virus-carrying mice dead 6 months after infection presented a huge mediastinal lymphosarcoma.

The kidneys were studied in 17 guinea pigs. All showed usually minor grades of parenchymatous degeneration. The cortical convoluted and loop tubules were usually swollen, their epithelium finely granular and sometimes marginally frayed. Basal striation and rod borders were discernible in a few animals in some tubules. Foamy or granular albuminous exudate was present in the tubules in 5, hyaline casts in 2 guinea pigs.

In 12 guinea pigs foci of interstitial or perivascular lymphocyte infiltration were more or less numerous in the renal cortex, perhaps more often in the arcuate zone about the larger vessels. Concentric vascular endothelial proliferation of small cortical vessels was seen in one of these and in one other guinea pig. Usually few foci of glomerular endothelial proliferation were seen in 4 guinea pigs, associated with cortical interstitial lymphocyte infiltration in 2, and with periglomerular fibroblast proliferation in 1 of them. Periglomerular proliferation was seen also in another animal with cortical lymphocyte infiltration but no endothelial proliferative changes. Vascular and exudative changes were absent in 2 animals. Moderate infestations with *Klossiella cobayae* were present in 2 guinea pigs.

In two animals elongate calcified masses were present in the pyramids, between the tubules. The significance of these is not clear.

The renal pelvis showed focal diffuse and perivascular lymphocyte infiltration in eight guinea pigs, in the fatty tissue in four, in the mucosa in three, and in both in one. In one animal the pelvic fatty tissues contained small concentric, fibrosing nodules of epithelioid cells, some with multinucleate giant cells, some with peripheral lymphocyte infiltration. These were undoubtedly part of a subacute disseminated granulomatous process in which staphylococci were present in the splenic granulomatous abscesses.

In general the kidneys showed a greater tendency to exudative inflammatory reaction and less parenchymatous degeneration than in rhesus monkeys (*2*).

Traub (*4*) found no lesions in the kidneys of guinea pigs. Only after intravenous inoculation did he note in mice collections of round cells in the kidneys. In one mouse there was pronounced unilateral renal enlargement with cortical atrophy and huge round cell collections in cortex and pelvic tissues. Virus-carrying mice killed 3 to 7½ months after infection also showed an interstitial nephritis constantly (*5*).

Findlay, Alcock, and Stern (*7*) noted in monkeys a glomerular

swelling, more the result of endothelial swelling than of round cell infiltration, and occasional slight intertubular lymphocyte infiltration.

The urinary bladder was normal in nine guinea pigs, two showed focal lymphocyte infiltration of the mucosa, two more lymphocyte infiltration with focal proliferation of fibroblasts and vascular endothelia, in one of these also in muscularis and serosa, and in one the mucosa showed congestion, vascular endothelial swelling and small hemorrhages. Changes are slight compared with monkeys (*3*).

The seminal vesicles were normal in 11 guinea pigs. In 2 the muscularis and adventitia presented focal vascular endothelial swelling or proliferation, some adventitial proliferation, and, in 1, lymphocyte infiltration. In 1 guinea pig the subvesicular fascia showed a slight diffuse leucocyte infiltration of uncertain significance.

Ureters and penis were normal in the few specimens studied, except that in one case the ureter near the renal pelvis showed a focal lymphocyte infiltration of its mucosa, similar to that often seen in the renal pelvis.

Testis and epididymis were normal in 5 of the 17 male guinea pigs. In the other 12 the testis showed more or less intercellular edema and reticulation of the germinal epithelium, with desquamation of many epithelial cells, occasionally only Sertoli cells remaining. In 3 there was active mitotic regeneration, in 1 with epithelial giant cells in the lumina of the seminiferous tubules. In the 3 guinea pigs showing the slightest degenerative testicular changes the epididymis was normal. The rest showed usually empty tubules in the upper pole, rounded necrotic germinal epithelial cells in the upper part of the lower pole, grading to intact or necrotic spermatozoa in the large muscular tubules near the origin of the vas. In one of these there was a frankly purulent, subacute bilateral epididymitis with squamous metaplasia of tubules, interstitial infiltration by leucocytes and lymphocytes and concentric vascular endothelial proliferation. One other guinea pig showed some pus mixed with necrotic germinal epithelium and spermatozoa in the tubules, with sparse focal interstitial and perivascular lymphocyte infiltration in epididymis and cremaster. Two other animals showed a few foci of perivascular lymphocyte infiltration, 1 in the cremaster, 1, with vascular endothelial swelling and proliferation, in the epididymis.

In the single female guinea pig included in this series, the uteri and tubes were normal. The intraepithelial saccular glands of the vagina were not infrequently filled with pus. Inoculation was intravaginal.

The male genitalia showed acute degenerative reactions not seen in monkeys (*3*). However, the former were sexually mature, the monkeys juvenile. Exudative inflammatory changes assignable to virus reaction were comparatively infrequent in guinea pigs.

The pancreas was normal in 14 animals. The mucous glands of the ducts were hyperactive in 1 and the ducts distended with mucus. In 1 animal the pancreas bordered on an omental abscess, but portions distant from the abscess wall were normal.

Similar negative findings were recorded in monkeys (*2*) and in guinea pigs by Traub (*4*).

Lymphocyte infiltration varying from a few small clumps of lymphocytes to fairly dense foci was seen in four of the eight adrenals studied. One other showed two small capillary hemorrhages in the cortex. Similar changes occurred in monkeys (*2*), and, in addition, occasional focal necroses.

Traub (*4*) found no lesions in the adrenals of guinea pigs.

Findlay, Alcock, and Stern (*7*) noted focal round cell infiltration in the adrenals of monkeys, particularly in the cortex.

Sympathetic ganglia containing masses of chromaffin cells were encountered in three guinea pigs (16, 21, 25 days). One (16 days) showed focal lymphocyte infiltration, the other two were normal. A pararenal ganglion (7 days) showed marginal tigroid clumping. Other cervical, cardiac, abdominal, and pelvic ganglia showed no significant lesions.

The spinal ganglia of monkeys (*2*) usually showed sheath cell proliferation, lymphocyte infiltration, or both.

In 12 animals the thyroid was normal. In 1 there were moderate interstitial edema with scattered monocytes in the widened tissue spaces and thin watery colloid in the alveoli. In another there were foci of interstitial fibroblast proliferation and lymphocyte infiltration with concentric vascular endothelial proliferation.

No lesions were found in monkeys (*3*).

Normal parathyroids were found in three animals. Focal perivascular lymphocyte infiltration was seen in a few monkeys (*3*) and in one a focal necrosis.

Skeletal muscles were recorded as normal in 10 of 17 guinea pigs, and in 1 more one of two blocks studied was normal. One guinea pig (6 days) showed a single focus of vascular endothelial and adventitial proliferation with some lymphocyte infiltration, another (21 days) a single focus of perivascular lymphocyte infiltration. A few similar foci of both types were seen in another animal (11 days). A fourth (14 days) presented a mixed lymphocyte and monocyte or fibroblast reaction about occasional small vessels. In a fifth (27 days) there were marked focal interstitial and perivascular lymphocyte infiltration and vascular adventitial fibroblast proliferation, endothelial swelling, and hyaline thrombosis in a few vessels. In the remaining 2 (18, 30 days), occasional vessels showed respectively concentric endothelial swelling and perivascular lymphocyte infiltration, and in both scattered or single degenerating or necrotic muscle fibers, hyalinized and oxyphil, in the one with peripheral nuclear accumulation in basophilic

cytoplasmic masses, in the other with invasion of the necrotic fiber by fibroblasts, macrophages, and lymphocytes.

Only one monkey showed a sparse focal lymphocyte infiltration (*3*).

Sections from the site of subcutaneous inoculation were made in two guinea pigs. One such lesion 9 days after inoculation contained necrotic fragmenting leucocytes and fibrin, its margin was densely packed with fragmenting pseudoeosinophil leucocytes and granular debris, its wall was composed of concentrically disposed fusiform fibroblasts proceeding from between the abutting muscle fibers. In the later lesion, 14 days after inoculation, the wall was a fibroblastic granulation tissue with areas of organizing hemorrhage and hemosiderin pigmentation, numerous multinucleate giant cells, and oxyphil hyaline masses included in the free margin.

SUMMARY

Lymphocytic choriomeningitis infection in guinea pigs presents a relatively mild and chiefly basilar lymphocytic meningitis and focal lymphocyte infiltration of the chorioid plexus. There is also striking focal lymphocyte infiltration and fibroblast proliferation in the heart muscle, endocardium and epicardium. Focal lymphocyte infiltration with secondary follicles are seen in the lungs and respiratory passages. Focal necrotic, proliferative, and mixed lesions appear in the liver, together with evidence of fatty changes in some. An early polymorphonuclear infiltration and later reticuloendotheliosis and lymphoid cell infiltration are seen in the spleen pulp. Follicle hyperplasia and intrafollicular phagocytosis of nuclear fragments are noted in spleen, lymph nodes, and intestinal lymphoid follicles. In the kidneys there are mild parenchymatous degeneration and focal interstitial lymphocyte infiltration. Focal lymphocyte infiltration of the mucosa of the renal pelvis and bladder occur in less than half of the animals. Germinal epithelial degeneration is seen in the testis. Focal lymphocyte infiltration occurs in some animals in epididymis, adrenal medulla, skeletal muscle, thyroid, and submaxillary gland, in some with concomitant vascular endotheliosis. Muscle fiber degeneration and necrosis are found in occasional animals.

REFERENCES

(*1*) Armstrong, C., and Lillie, R. D.: Experimental lymphocytic choriomeningitis of monkeys and mice produced by a virus encountered in studies of the 1933 St. Louis encephalitis epidemic. Pub. Health Rep., **49**: 1019 (1934).

(*2*) Lillie, R. D.: Pathologic histology of lymphocytic choriomeningitis in monkeys. Pub. Health Rep., **51**: 303 (1936).

(*3*) Rivers, T. M., and Scott, T. F. McN.: Meningitis in man caused by a filterable virus. II. Identification of the etiological agent. J. Exp. Med., **63**: 415 (1936).

(*4*) Traub, E.: An epidemic in a mouse colony due to the virus of acute lymphocytic choriomeningitis. J. Exp. Med., **63**: 533 (1936).

(5) Traub, E.: Persistence of lymphocytic choriomeningitis virus in immune animals and its relation to immunity. J. Exp. Med., 63: 847 (1936).
(6) Lillie, R. D., and Pasternack, J. G.: Romanowsky staining with buffered solutions. II. Current modification. J. Tech. Meth. and Bull. of Inter. Assn. Med. Mus., 15: 65–70 (1936).
(7) Findlay, G. M., Alcock, N. S., and Stern, R. O.: The virus aetiology of one form of lymphocytic meningitis. Lancet, 230: 650 (1936).
(8) Thompson, J.: Salivary gland disease of mice. J. Infect. Dis., 58: 59 (1936). (Cited after Findlay, Alcock, and Stern.)

MEDICAL CARE: A PRIVATE ENTERPRISE OR A SOCIAL SERVICE? [1]

By JOSEPH W. MOUNTIN, *Medical Director, United States Public Health Service*

One of the very hopeful signs of the time is the increasing tendency of producers and consumers to meet for frank discussion of ways and means of extending and improving medical service. The issues involved are manifold and not simple of solution. While physicians occupy a central position in the provision of this service, it should be borne in mind that other groups also have their parts—dentists, nurses, and a variety of technicians. Persons representing these several skills cannot be fully effective without access to hospitals, clinics, and related facilities which serve as their workshops. Clearly it is the province of the medical and allied professions to determine the content of the medical care program. Likewise, it is their responsibility to foster the development of new and improved methods for preventing, detecting, and alleviating human ailments. In carrying out these purposes the professions concerned are entitled to every aid that can be placed at their disposal.

On the other hand, disease has its social implications. It is the people at large who suffer the discomforts of illness and the economic consequences thereof. If the illness is minor in character, the wage loss and the costs of care probably will not be a source of serious embarrassment to an individual in moderate circumstances. A more protracted illness or one entailing expensive forms of treatment may drain off the patient's resources to a point where assistance is necessary. Any general impairment of health affects national vitality.

The increasing public interest and concern about questions involving medical care can be traced to two distinct but somewhat related factors: First, the measurable benefits that now may be derived from good medical service; and second, the increasing proportion of the population that is denied full participation in these benefits. The situation today presents a striking contrast to that which obtained when the prevailing pattern of medical practice took form.

[1] Delivered at the Forum on National Health Insurance, South Agriculture Auditorium, Washington, D. C., April 1944.

608128°—44——3

Even as late as the beginning of the present century, most people spent the greater part of their lives in the same area where they were born or first established residence; hence, a general feeling of neighborliness prevailed. The sick were cared for in their own homes by members of the family under the general direction of a local physician. For diagnosis the physician relied on his powers of observation; his remedies likewise were simple. Figuratively speaking, he practiced medicine out of a black bag which contained little more than a stethoscope, a clinical thermometer, a bistoury, and a limited assortment of pills and potions. Dentistry, nursing, and hospitalization were practically unknown to the great masses of the population. Under such circumstances, as one might expect, the costs of illness were not high. And because the physician knew personally his entire clientele, their social habits, and their economic position, it was possible for him to extend credit, vary fees, or remit payments, according to individual circumstances. In many respects this kind of relationship afforded a desirable type of health supervision, but it may be questioned if the primitive methods of diagnosis and treatment in use could have influenced favorably the course of a very high proportion of the illnesses so attended.

Industrialization of itself was sufficient to render obsolete this highly individualistic form of medical practice. A high degree of mobility in the population always accompanies industrialization. The factory worker has less economic security than the owner of a farm or a small business. Industrialization also places a high value on physical fitness. The net result has been the creation of problems of health, security, and happiness.

Concurrent with the socioeconomic evolution profound changes have taken place in the field of medicine, especially within the past half century. By drawing on the several basic sciences medicine has evolved highly complex, yet very effective, bodies of knowledge and skills for preventing, detecting, and alleviating human ailments.

The acquisition of this fundamental information and the varied skills essential for its application entail an arduous process of training. In less complex days young men entered medicine after being apprenticed to older physicians for a short time. Nowadays a person is not admitted to practice until he has completed 6 to 8 years of education following graduation from high school. This lengthy schooling represents a personal outlay approximating $10,000 on the part of each student or his sponsor. To that sum may appropriately be added the cash value of what he would have produced in goods or services if he had followed a gainful occupation instead of acquiring a college and medical education. Since tuition paid by students meets only a small part of the cost of operating medical schools and teaching hospitals, society at large is called upon to make its contribution to medical

education. Because of these factors a physician ready for professional work represents a large investment which is reflected in the value of his services.

Furthermore, in the practice of modern medicine, it is necessary that physicians have ready access to physical facilities represented by hospitals, clinics, and laboratories, and be assisted by the skilled staffs associated with such institutions. While adding to the effectiveness of diagnostic and therapeutic procedures, the full use of such resources materially increases the cost of medical care. Without such facilities, however, the patient cannot hope to obtain all the benefits which medical sciences have to offer.

In one very essential field—namely, organization—medicine has not kept pace with its own scientific advancement or with the social and economic changes in the body politic.

Despite successful demonstration of group practice and prepayment plans, it would appear that the prevailing opinion within the profession sustains the view that the most desirable arrangement is for each physician to operate as a unit and be paid directly by the patient on the basis of fees for service rendered. These principles, literally interpreted and applied, would preclude the introduction of schemes of organization that have operated so successfully in fields of commerce and industry; they would not permit the spreading of risk through the insurance technique; the consuming public would not have any voice in management; and there could be little or no participation on the part of government. Happily this extreme position does not prevail in actual practice.

Experience has demonstrated that such rigid principles cannot be applied to the whole range of medical service—for reasons that are more or less obvious. Even in prosperous times a substantial proportion of the population is not self-supporting, and a much larger group is precipitated into the so-called medically indigent class by the occurrence of even moderately severe illness. Such conditions as mental disease, tuberculosis, and chronic invalidism often require prolonged periods of institutional care. This of itself is very expensive; moreover, these disorders almost invariably have a permanent depressing effect on income. In still another class are the communicable diseases which require special treatment facilities and the use of both law-enforcement and health-education measures. For want of a better term, this broad and variable category of disorders is sometimes referred to as "unprofitable illness," largely because the remedial measures indicated are not adapted to application by private enterprise. Even nonprofit agencies, many of which derive a substantial part of their incomes from endowments and gifts, have not been able to operate extensively in this field. Hence, the job falls to government and the costs are borne out of taxes.

Most of the current controversy centers around that group of human ailments often spoken of as "profitable illness." In the main, these are the short-term illnesses experienced by self-sustaining members of the population. The patient is expected to pay for the care he receives either out of pocket or on credit, coupled perhaps with some adjustment of fees. Up until quite recently, in this country, medical care for profitable illnesses had somewhat the social status of shoes, clothing, groceries, or recreation. That is, it represented a need which should be recognized by the individual and be satisfied according to his wishes within the limit of his purchasing capacity. With this arrangement there could be no disagreement, provided medical care had the same essential characteristics as the articles mentioned and occupied a similar position in respect to human necessity. But few persons would claim the complete identity of medical care with shoes, much less their similarity.

The head of the household can estimate in advance, with a high degree of accuracy, his expenditures for shoes, clothing, groceries, and recreation. In fact, these costs vary little from day to day or month to month, except as the price index fluctuates. By substitution and denial, a family head may adjust expenditures for these commodities without seriously affecting the health or happiness of his household. But the family head is not in a position to meet his illness problems so easily. One family group may live together for several years and experience no serious illness, another may pass from one medical disaster into another, while still a third may have the so-called average illness experience. For an ensuing period the illness patterns of these families may continue unchanged or they may be completely reversed. No one can predict what the illness experience of any family, or of any of its members, will be even during the course of a day. Since treatment must be adapted both to the character of the illness and to the reactions of the patient, advance estimates on costs likewise are not within the range of calculation; hence, budgeting for illness by the individual is not feasible.

Under a system where receipt of service is dependent on ability to pay for it, one naturally expects that service will be apportioned accordingly. Despite all assertions to the contrary, that is what actually occurs. Physicians are known to give generously of their time in ministering to the sick of limited means, but this gratuitous service falls far short of effecting complete equalization in receipt of care among persons of all income groups. A large part of the difficulty arises from the fact that persons in the lower economic brackets have more illness than do those in better financial circumstances. Furthermore, a combination of lowered vitality and a tendency to delay seeking care seems to result in unduly severe illness among the poor.

Thus more prolonged and expensive forms of treatment than otherwise would be indicated are necessary.

Accumulating funds for paying medical bills may be only part of the problem in obtaining care. If no physician is within reach it matters little whether or not the patient can pay the prevailing fee. Regardless of financial considerations, a patient's need for hospitalization cannot be satisfied unless such facilities are available. Perhaps one of the most serious obstacles in the way of adequate care for everyone is the inequable distribution of professional personnel and hospital accommodations. In general, their distribution follows economic lines when State totals are considered.

TABLE 1.—*Number of hospital beds and physicians in States of different income classification, 1940*

State per capita income	General and allied hospital beds, per 100,000 population	Physicians per 100,000 population
Total, United States	385.9	133.0
Under $300	214.1	76.7
$300–$599	331.6	110.0
$600 and over	457.0	160.3

Within the States both physicians and hospital beds tend to be concentrated in centers of population. It is recognized, of course, that centers of population are apt to be centers of wealth also. However, analyses made by the United States Public Health Service show that a hospital of itself exerts a definite influence both in attracting physicians to a community and in retaining their services.

TABLE 2.—*Number of hospital beds and physicians in metropolitan and nonmetropolitan counties*

Character of county	General and allied hospital beds per 100,000 population (1940)	Physicians per 100,000 population (1938)
Metropolitan	507.5	168.9
Nonmetropolitan	246.9	92.1

TABLE 3.—*Number of hospital beds and physicians in counties of different urban character*

Largest urban place in county	General and allied hospital beds per 100,000 population (1940)	Physicians per 100,000 population (1938)
No urban place of 2,500 or over	96.8	66.8
City 2,500–49,999 in county	295.5	100.6
City 50,000 or over in county	538.0	179.2

Since economic factors are largely responsible for the present concentration of professional and hospital resources in centers of population and wealth, there is reason to suppose that payments for service out of a pooled fund derived from taxation or insurance, would do much to reverse current trends. To meet the special needs of low-income and sparsely settled areas, it will probably be necessary also to make direct grants for the construction and operation of hospitals and to supplement the income of professional personnel.

In general it may be said that the amount and character of medical service the people receive are determined by three factors: (1) their financial resources, (2) the availability of properly trained personnel, and (3) the presence of hospitals and related facilities. During different periods in our recurring economic cycles and in different parts of the country, one or the other of these hurdles may be the most prominent obstacle. At no time in the experience of this country have the forces of free enterprise, supplemented by charity, brought about a combination of circumstances under which good medical care is available to everyone. Whether or not it can be accomplished under any other system may be open to question. Nevertheless, a substantial part of the general public and some very thoughtful members of the medical and allied professions believe additional methods should be tried.

In the discussion so far, little mention has been made of disease prevention. Despite its over-all importance from the standpoint of general health, prevention has never become a prominent element of private medical practice. Activities that contribute to disease control and general health promotion, for the most part, are now supported by taxation and administered by agencies of Federal, State, and local governments. While public health agencies have to their credit many outstanding accomplishments, such as the conquest of yellow fever and typhoid fever, and the very substantial reduction in deaths attributable to tuberculosis, diphtheria, and the disorders of infancy, there are still further possibilities in the preventive field of endeavor. To exploit these possibilities traditional health programs need to be strengthened and the geographic coverage of health services, under the direction of full-time professional personnel, should be extended to all parts of this country. In addition, some method should be developed whereby the system established primarily to render medical care to the sick may also serve the interest of preventive medicine.

The blending of preventive and curative forces and their full utilization in a complete health program scarcely seems possible so long as the receipt of service is contingent upon payment by the beneficiary of a fee to cover the cost of the service. Such a system

obviously restricts service to those who are both willing and able to pay for it on such a basis. If a health program is to be comprehensive the primary objective must be to remove this financial barrier. Furthermore, every citizen must be encouraged to utilize the service up to the limits indicated by his need. Variation in need for service should have no weight in determining what payments shall be made by the individual.

In addition to satisfying the foregoing medical needs of the people, a national health program must make provision for research. Unless this is done, stagnation, and eventually retrogression, will set in. Research should be of sufficient breadth and intensity to insure reasonable advancement in every sector of the entire health field. Another and equally essential provision of a health program designed to insure continuous progress is that of professional education. While the present system for instructing medical students has attained a high degree of excellence, it makes no orderly provision for keeping up to date the physician in practice. This defect especially should be remedied in plans for the future.

An attempt has been made to present some of the social and economic aspects of disease, together with a few of the problems that are involved in its prevention and alleviation. Extensive studies serve to support the well-known fact that illness is highly unpredictable both as to character and time of occurrence. Also it is a matter of common knowledge that a person of limited income has great difficulty securing adequate care for even ordinary illness at a price within his means. In many sections of the country diagnosis and treatment of complicated illness are scarcely possible because the necessary professional personnel and physical facilities are not within reach. While mortality statistics suggest a high level of general health, the results of physical examinations show we are not a robust people. It would therefore seem that many of the splendid accomplishments of public health agencies are being nullified by failures in the application of curative medicine. Ways and means for correcting this defect should receive special consideration in any plan that is designed to improve the health of the individual citizen. Actually, as a people we have not attained a level of health that is possible under the present state of scientific knowledge. Hardly anyone can envision the still higher levels that may be reached should health become one of our chief social objectives when the energies of the people can again be diverted from war to the pursuits of peace.

PUBLIC HEALTH SERVICE PUBLICATIONS

A List of Publications Issued During the Period January–June 1944

The following is a list of publications of the United States Public Health Service issued during the period January–June 1944.

The purpose of the publication of this list is to provide a complete and continuing record of Public Health Service publications for reference use by librarians, scientific workers, and others interested in particular fields of public health work, and not to offer the publications for indiscriminate free public distribution.

Those publications marked with an asterisk (*) may be obtained only by purchase from the Superintendent of Documents, Government Printing Office, Washington 25, D. C., at the prices noted.

Periodicals

*Public Health Reports (weekly), January–June, vol. 59, Nos. 1 to 26, pages 1 to 856. 5 cents a number.

*Venereal Disease Information (monthly), January–June, vol. 25, Nos. 1 to 6, pages 1 to 196. 5 cents a number.

*Journal of the National Cancer Institute (bimonthly), February–June, 1944, vol. 4, Nos. 4 to 6, pages 339 to 600. 40 cents a number.

Public Health Engineering Abstracts (monthly), January–April, vol. XXIV, Nos. 1 to 4, 32 pages each; May–June, Nos. 5 and 6 combined, 32 pages.

National Negro Health News (quarterly), January–March, vol. 12, No. 1, 20 pages; April–June, No. 2, 24 pages.

Reprints From the Public Health Reports

2535. National inventory of needs for sanitation facilities. I. Public water supply. By H. W. Streeter and Ray Raneri. January 7, 1944. 20 pages.

2536. Lesions in rats given sulfathiazole, sulfadiazine, sulfanilamide, sulfamerazine, sulfapyrazine, or acetylsulfadiazine in purified diets. By K. M. Endicott, A. Kornberg, and F. S. Daft. January 14, 1944. 6 pages.

2537. Illness from cancer in the United States. By Harold F. Dorn. January 14, 21, and 28. 46 pages.

2538. Cultivation of *Pasteurella tularensis* in a liquid medium. By Edward A. Steinhaus, R. R. Parker, and Max T. McKee. January 21, 1944. 2 pages.

2539. Sanitation manual for public ground water supplies. February 4, 1944. 41 pages.

2540. National inventory of needs for sanitation facilities. II. Milk pasteurization facilities. By John Andrews and A. W. Fuchs. February 11, 1944. 16 pages.

2541. I. A comparison of light trap and animal bait trap Anopheline mosquito collections in Puerto Rico. II. A list of the mosquitoes of Puerto Rico. By A. Earl Pritchard and Harry D. Pratt. February 18, 1944. 13 pages; 2 plates.

2542. An index of the prevalence of dental caries in school children. By John W. Knutson. February 25, 1944. 12 pages.

2543. Location and movement of physicians—methods for estimating physician resources. By Elliott H. Pennell. March 3, 1944. 25 pages.

2544. The reportable diseases. Diseases and conditions required to be reported in the several States. By William Fowler. March 10, 1944. 24 pages.
2545. Problems created by returning malaria carriers. By Stanley B. Freeborn. March 17, 1944. 8 pages.
2546. Complement fixation in the rickettsial diseases—technique of the test. By Ida A. Bengtson. March 24, 1944. 4 pages.
2547. The action of penicillium extracts in experimental tuberculosis. By M. I. Smith and E. W. Emmart. March 31, 1944. 8 pages.
2548. Entomological phases of the recent dengue epidemic in Honolulu. By Robert L. Usinger. March 31, 1944. 8 pages.
2549. The effect of a synthetic marihuana-like compound on musical talent as measured by the Seashore test. By C. Knight Aldrich. March 31, 1944. 4 pages.
2550. Nomenclature of pneumococcic types. A study of cross reactions among the pneumococcic types and their application to the identification of types. Cross reactions between the several pneumococcic types and their significance in the preparation of polyvalent antiserum. By Bernice E. Eddy. April 7 and 14, 1944. 36 pages.
2551. Hospitals in the public health panorama. By Warren F. Draper. April 21, 1944. 10 pages.
2552. The patch test in contact dermatitis. By Louis Schwartz and Samuel M. Peck. April 28, 1944. 12 pages.
2553. Airplane dusting with paris green for control of *Anopheles quadrimaculatus* Say in water-chestnut covered areas of the Potomac River during 1943. By William C. Murray and Herbert Knutson. May 5, 1944. 12 pages; 2 plates.
2554. The therapeutic efficacy of penicillin in relapsing fever infections in mice and rats. By Harry Eagle and Harold J. Magnuson. May 5, 1944. 6 pages.
2555. Organization of the medical and sanitary program, Alaska highway project. By Edwin H. Carnes. May 12, 1944. 9 pages; 2 plates.
2556. Pathologic changes in sheep resulting from exposure to low barometric pressures. By John W. Miller. May 12, 1944. 4 pages; 2 plates.
2557. The chemotherapy of burns and shock. VI. Standardized hemorrhage in the mouse. VII. Therapy of experimental hemorrhage. By Herbert Tabor, Herman Kabat, and Sanford M. Rosenthal. May 19, 1944. 21 pages.
2558. Studies on trichinosis. XVI. Epidemiological considerations based on the examination for trichinae of 5,313 diaphragms from 189 hospitals in 37 States and the District of Columbia. By Willard H. Wright, Leon Jacobs, and Arthur C. Walton. May 26, 1944. 13 pages.
2559. A strain of typhus rickettsiae isolated from the brain of a wild rat in California. By M. Dorthy Beck, Howard L. Bodily, and Rosemary O'Donnell. June 2, 1944. 12 pages.
2560. Prevalence of poliomyelitis in the United States in 1943. By C. C. Dauer. June 2, 1944. 8 pages.
2561. Sulfarsphenamine in the therapy of syphilis. A comparative study of the toxic manifestations of neoarsphenamine and sulfarsphenamine. By Thomas F. Probey, Edgar W. Norris, Austin V. Deibert, and Eleanor V. Price. June 9, 1944. 20 pages.
2562. The therapeutic efficacy of phenyl arsenoxides in mouse and rabbit Trypanosomiasis (*Tryp. equiperdum*). By Harry Eagle, Ralph B. Hogan, George O. Doak, and Harry G. Steinman. June 16, 1944. 20 pages.

2563. Births, infant mortality, and maternal mortality in the United States—1942. By J. Yerushalmy. June 23, 1944. 14 pages.
2564. A sieve device for sampling air-borne microorganisms. By H. G. duBuy and L. R. Crisp. June 30, 1944. 4 pages; 1 plate.
2565. Production of vitamin K deficiency in rats by various sulfonamides. By A. Kornberg, F. S. Daft, and W. H. Sebrell. June 30, 1944. 12 pages.

Supplements to the Public Health Reports

18. Malaria. Lessons on its cause and prevention for use in schools. By H. R. Carter. Revised by L. L. Williams, Jr., June 28, 1943. 1944. 23 pages.
174. The notifiable diseases. Prevalence of certain important communicable diseases, by States, 1942. 1944. 13 pages.
175. The bedbug—its habits and life history and methods of control. By Willard H. Wright. 1944. 9 pages; 2 plates.
176. Nursing in the United States Public Health Service. By Katharine S. Read. 1944. 6 pages.

Public Health Bulletins

281. The aliphatic alcohols: Their toxicity and potential dangers in relation to their chemical constitution and their fate in metabolism. By W. F. von Oettingen. 253 pages. 1943.
282. Toxicity and potential dangers of penta-erythritol-tetranitrate (petn). By W. F. von Oettingen, D. D. Donahue, A. H. Lawton, A. R. Monaco, H. Yagoda, and P. J. Valaer. 1944. 39 pages.
283. Nursing practices in industry. By Olive M. Whitlock, Victoria M. Trasko, and F. Ruth Kahl. 1944. 70 pages.

Workers Health Series

12. Let's See! 1944. 10 pages.
13. Below the Belt. 1944. 6 pages.

Posters

Community Health Posters. Four colors, each 10 x 14 in.

3. Build out rats—screen ventilators, cover floor holes, install metal flashing, seal openings, lay concrete floors.
4. Don't feed rats.

Workers Health Posters. Two colors, each 10 x 14 in.

18. Cover up coughs and sneezes. A bad cold may be flu! See your doctor.
19. Don't gamble with appendicitis—don't use a laxative—call a doctor.
20. Welding. Guard against: explosives, electric shock, burns, gases, powerful arc lights.
21. Let's see! Better, longer, easier.
22. Be kind to your stomach. Choose food and drink wisely. If stomach upset is violent, see your doctor.
23. See your dentist to prevent pain and control infection.
24. Wear clean clothes—beat the skin game. A shower after work.
25. Know your score—get a physical check-up at least once a year.
26. Beware of carbon monoxide in garages, in foundries, in heating plants.
27. What you don't know can hurt you—get a blood test for syphilis.
28. Night shift. Guard against fatigue by getting plenty of rest and good food.
29. Correct pick-up, avoid hernia.

Community Health Series

*5. Is there a doctor in town? 4 pages. 1944. Out of print. (Obsolete.)

Unnumbered Publications

Index to Public Health Reports, vol. 58, part 2, July–December 1943. 16 pages.

Index to Journal of the National Cancer Institute, vol. IV, August 1943–June 1944. 7 pages.

National Negro Health Week program. This pamphlet is published annually, usually about the middle of March, for community leaders in an effort to suggest ways and means by which interested individuals and organizations may be organized for a concerted and effective attack upon the community's disease problems. Thirtieth observance, April 2–9, 1944. 4 pages.

National Negro Health Week poster. Thirtieth observance. 1944.

National Negro Health Week leaflet. Thirtieth observance. 1944. 2 pages.

Annual Reports

Annual Reports of the Surgeon General of the United States Public Health Service 1941–42, 1942–43. 194 pages.

Reprints From Venereal Disease Information

213. Serial examinations in the epidemiology of gonococcal infections. By Samuel D. Allison, W. L. Zink, and W. S. Ito. Vol. 25, January 1944. 2 pages.
214. Venereal disease and selective service. By Richard H. Eanes. Vol. 25, January 1944. 6 pages.
215. Improvement of present methods for extrafamilial contact tracing. By Mary A. Burke. Vol. 25, January 1944. 5 pages.
216. Criteria of cure in gonorrhea. By Richard A. Koch, Earl N. Mathis, and Jacob C. Geiger. Vol. 25, February 1944. 8 pages.
217. Progress in the wartime management of gonorrhea. By Percy S. Pelouze. Vol. 25, February 1944. 4 pages.
218. Combined artificial fever and aldarsone in the treatment of neurosyphilis. By A. E. Bennett, W. H. Morrison, and H. C. Modlin. Vol. 25, March 1944. 8 pages.
219. Gonorrhea: the epidemic we face. By P. S. Pelouze. Vol. 25, March 1944. 5 pages.
220. The one-day treatment of syphilis with fever and mapharsen. By Nathaniel Jones, Charles M. Carpenter, Ruth A. Boak, Stafford L. Warren, and Henry Hanson. Vol. 25, April 1944. 5 pages.
221. A study of the amount of active syphilis found in a group of newly inducted soldiers. By Joe W. Still and Eugene Greenwald. Vol. 25, April 1944. 4 pages.
222. Evaluation of positive Kolmer and Kahn tests in leprosy. By G. H. Faget and Hilary Ross. Vol. 25, May 1944. 5 pages.
223. An evaluation of the blood-dye diluent for the transportation of material from gononoccic infections. By S. Edward Sulkin and Joseph C. Willett. Vol. 25, May 1944. 6 pages.
224. Trichomonas urethritis and prostatitis: A preliminary report on incidence and an analysis of 44 cases of this common venereal infection. By Russell B. Roth. Vol. 25, June 1944. 4 pages.

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

The State reports regularly scheduled for this issue will be published in the issue of November 3. This change is necessitated by a new arrangement with the Government Printing Office, whereby the Public Health Reports will, after a brief transition period, appear approximately on its issue date, instead of 2 weeks later, as at present.

WEEKLY REPORTS FROM CITIES

City reports for week ended Sept. 30, 1944

This table lists the reports from 90 cities of more than 10,000 population distributed throughout the United States, and represents a cross section of the current urban incidence of the diseases included in the table.

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
NEW ENGLAND												
Maine:												
Portland	0	0	-----	0	0	0	1	1	3	0	0	1
New Hampshire:												
Concord	0	0	-----	0	0	0	1	0	0	0	0	0
Vermont:												
Barre	0	0	-----	0	0	0	0	0	3	0	0	3
Massachusetts:												
Boston	3	0	-----	0	31	4	11	6	22	0	0	13
Fall River	0	0	-----	0	0	0	2	0	0	0	3	6
Springfield	0	0	-----	0	0	0	0	3	1	0	1	0
Worcester	0	0	-----	0	0	0	7	1	4	0	0	12
Rhode Island:												
Providence	0	1	1	0	0	1	0	0	3	0	0	18
Connecticut:												
Bridgeport	1	0	-----	0	0	0	0	0	0	0	0	1
Hartford	0	0	-----	0	0	0	1	1	0	0	0	2
New Haven	0	0	-----	0	0	1	0	2	0	0	0	15
MIDDLE ATLANTIC												
New York:												
Buffalo	0	0	-----	0	0	1	6	20	0	0	0	5
New York	6	1	1	0	3	5	47	116	35	0	3	76
Rochester	0	0	-----	0	4	1	1	26	0	0	0	5
Syracuse	0	0	-----	0	0	1	1	1	1	0	0	6
New Jersey:												
Camden	0	0	-----	0	0	1	0	1	0	0	0	0
Newark	1	0	1	0	3	0	1	1	4	0	0	19
Trenton	0	0	1	0	0	0	2	1	0	0	0	0
Pennsylvania:												
Philadelphia	1	0	1	0	3	4	5	9	13	0	3	14
Pittsburgh	0	0	-----	0	0	1	6	4	7	0	0	4
Reading	0	0	-----	0	0	0	1	0	0	0	0	0

City reports for week ended Sept. 30, 1944—Continued

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
EAST NORTH CENTRAL												
Ohio:												
Cincinnati	0	0	----	0	0	0	2	9	15	0	0	4
Cleveland	0	0	5	0	2	4	5	15	20	0	1	21
Columbus	0	0	----	0	0	0	2	1	2	0	0	10
Indiana:												
Fort Wayne	0	0	----	0	0	0	2	0	0	0	1	0
Indianapolis	5	0	----	1	0	0	3	1	3	0	0	3
South Bend	0	0	----	0	1	0	0	0	3	0	0	0
Terre Haute	0	0	----	0	0	0	4	0	2	0	0	2
Illinois:												
Chicago	1	0	----	1	7	3	16	14	19	0	2	48
Springfield	0	0	----	0	1	1	0	1	3	0	0	0
Michigan:												
Detroit	3	0	----	0	3	4	15	19	20	0	1	56
Flint	0	0	----	0	0	0	6	0	0	0	0	0
Grand Rapids	0	0	----	0	1	0	2	0	2	0	0	1
Wisconsin:												
Kenosha	0	0	----	0	0	0	0	0	1	0	0	13
Milwaukee	0	0	----	0	1	1	6	0	5	0	0	16
Racine	0	0	----	0	0	0	0	0	0	0	0	1
Superior	0	0	----	0	2	0	0	2	2	0	0	0
WEST NORTH CENTRAL												
Minnesota:												
Duluth	0	0	----	0	0	0	1	5	3	0	0	0
Minneapolis	5	0	----	0	2	0	6	9	8	0	0	4
St. Paul	1	0	----	0	0	0	2	7	2	0	0	21
Missouri:												
Kansas City	0	0	----	0	0	0	6	1	5	0	0	0
St. Joseph	0	0	----	0	0	0	0	0	0	0	1	0
St. Louis	0	0	2	0	0	1	12	15	5	0	0	17
North Dakota:												
Fargo	0	0	----	0	0	0	2	0	1	0	0	0
Nebraska:												
Omaha	0	0	----	0	4	0	0	1	0	0	0	0
Kansas:												
Topeka	0	0	----	0	0	0	0	1	3	0	0	0
Wichita	0	0	----	0	0	0	1	0	4	0	0	0
SOUTH ATLANTIC												
Delaware:												
Wilmington	0	0	----	0	0	0	2	6	0	0	0	2
Maryland:												
Baltimore	2	0	----	0	0	0	6	14	10	0	0	38
Cumberland	0	0	----	0	0	0	0	0	0	0	0	0
Frederick	0	0	----	0	0	0	0	0	0	0	0	0
District of Columbia:												
Washington	0	0	1	0	1	0	1	9	9	0	2	1
Virginia:												
Lynchburg	0	0	----	0	0	0	0	3	5	0	0	1
Richmond	0	0	1	1	0	0	1	3	2	0	0	0
Roanoke	0	0	----	0	0	0	1	2	0	0	0	0
West Virginia:												
Charleston	0	0	----	0	0	0	0	0	0	0	0	0
Wheeling	0	0	----	0	1	0	2	0	0	0	0	2
North Carolina:												
Raleigh	0	0	----	0	0	0	0	0	2	0	0	1
Wilmington	1	0	----	0	0	0	0	0	0	0	0	1
Winston-Salem	1	0	----	0	1	0	1	0	6	0	0	0
South Carolina:												
Charleston	0	0	----	0	0	0	0	1	0	0	1	0
Georgia:												
Atlanta	0	0	9	1	0	0	4	2	4	0	0	1
Brunswick	0	0	----	0	1	0	0	0	0	0	0	0
Savannah	0	0	----	0	0	0	0	0	2	0	0	0
Florida:												
Tampa	1	0	----	0	0	0	2	0	1	0	0	0

City reports for week ended Sept. 30, 1944—Continued

	Diphtheria cases	Encephalitis, infectious, cases	Influenza		Measles	Meningitis, meningococcus, cases	Pneumonia deaths	Poliomyelitis cases	Scarlet fever cases	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough cases
			Cases	Deaths								
EAST SOUTH CENTRAL												
Tennessee:												
Memphis	2	0	------	0	1	1	4	0	5	0	2	7
Nashville	0	0	------	0	0	0	2	0	4	0	0	0
Alabama:												
Birmingham	0	0	1	0	0	0	3	0	2	0	0	0
Mobile	0	0	1	0	0	2	1	0	4	0	0	0
WEST SOUTH CENTRAL												
Arkansas:												
Little Rock	0	0	------	0	0	0	1	0	1	0	0	1
Louisiana:												
New Orleans	3	0	2	0	0	2	7	2	5	0	0	3
Shreveport	3	0	------	0	0	0	1	1	0	0	0	0
Texas:												
Dallas	5	0	------	0	0	0	1	0	5	0	0	0
Galveston	1	0	------	0	0	0	0	0	2	0	0	0
Houston	1	0	------	0	0	0	4	1	1	0	2	3
San Antonio	1	1	1	2	1	0	6	0	0	0	1	0
MOUNTAIN												
Montana:												
Billings	0	0	------	0	0	0	0	1	0	0	1	2
Great Falls	0	0	------	0	1	0	0	0	2	0	1	0
Helena	0	0	------	0	0	0	0	0	0	0	0	2
Missoula	0	0	------	0	0	0	2	0	0	0	0	0
Idaho:												
Boise	0	0	------	0	0	0	0	0	0	0	0	0
Colorado:												
Denver	5	0	5	0	1	1	4	1	4	0	0	4
Pueblo	0	0	------	0	0	0	4	0	1	0	0	2
Utah:												
Salt Lake City	0	0	------	0	2	0	0	0	3	0	0	4
PACIFIC												
Washington:												
Seattle	0	0	------	1	0	0	0	4	4	0	0	0
Spokane	0	0	------	0	0	0	1	0	0	0	0	0
Tacoma	0	0	------	0	0	1	0	0	0	0	1	2
California:												
Los Angeles	6	0	3	0	6	2	3	0	11	0	2	11
Sacramento	0	0	------	0	1	2	0	0	5	0	0	0
San Francisco	4	0	2	1	15	1	5	0	6	0	0	1
Total	63	3	38	8	100	46	255	344	330	0	29	506
Corresponding week, 1943	60	------	28	13	253	------	263	------	500	0	29	875
Average, 1939–43	67	------	44	[1] 12	[2] 173	------	[1] 257	------	360	0	36	989

[1] 3-year average, 1941–43.
[2] 5-year median, 1939–43.

Dysentery, amebic.—Cases: Boston, 1; Baltimore, 1; Houston, 1; Los Angeles, 1.
Dysentery, bacillary.—Cases: Buffalo, 6; New York, 9; Rochester, 2; Chicago, 1; Detroit, 6; St. Louis, 4; Baltimore, 1; Charleston, S. C., 11; Atlanta, 1; Los Angeles, 2.
Dysentery, unspecified.—Cases: Richmond, 2.
Rocky Mountain spotted fever.—Cases: Richmond, 1.
Tularemia.—Cases: Cleveland, 1.
Typhus fever, endemic.—Cases: Wilmington, N. C., 3; Atlanta, 3; Savannah, 10; Tampa, 4; Nashville, 10; Birmingham, 1; Mobile, 4; New Orleans, 3; Shreveport, 1; Dallas, 2; Galveston, 3; Houston, 4; San Antonio 1.

Rates (annual basis) per 100,000 population, by geographic groups, for the 90 cities in the preceding table (estimated population, 1943, 34,394,800

	Diphtheria case rates	Encephalitis, infectious, case rates	Influenza		Measles case rates	Meningitis, meningococcus, case rates	Pneumonia death rates	Poliomyelitis case rates	Scarlet fever case rates	Smallpox case rates	Typhoid and paratyphoid fever case rates	Whooping cough case rates
			Case rates	Death rates								
New England	10.5	2.6	2.6	0.0	81	15.7	60.1	36.6	94	0.0	10.5	186
Middle Atlantic	3.7	0.5	1.9	0.0	6	6.5	32.4	82.8	28	0.0	2.8	60
East North Central	5.5	0.0	3.0	1.2	11	7.9	38.3	37.7	59	0.0	3.0	106
West North Central	11.9	0.0	4.0	0.0	12	2.0	59.7	77.6	62	0.0	2.0	84
South Atlantic	8.2	0.0	18.0	3.3	7	0.0	32.7	65.4	67	0.0	4.9	77
East South Central	11.8	0.0	11.8	0.0	6	17.7	59.0	0.0	89	0.0	11.8	41
West South Central	40.2	2.9	8.6	5.7	3	5.7	57.4	11.5	40	0.0	8.6	20
Mountain	39.7	0.0	39.7	0.0	32	7.9	79.4	15.9	79	0.0	15.9	111
Pacific	15.8	0.0	7.9	3.2	35	9.5	14.2	6.3	41	0.0	4.7	22
Total	9.6	0.5	5.8	1.2	15	7.0	38.8	52.3	50	0.0	4.4	77

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended September 16, 1944.—During the week ended September 16, 1944, cases of certain communicable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Chickenpox		5		22	44	6	2	12	21	112
Diphtheria	1	3	2	36	8	4	1			55
Dysentery (amebic)					3					3
Dysentery (bacillary)				30						30
German measles				4	6		1		7	18
Influenza					9				14	23
Measles		1		24	9	4	3	6	8	55
Meningitis, meningococcus			1						2	3
Mumps				21	19	2	1	15	16	74
Poliomyelitis		1	4	4	27	11		11	1	59
Scarlet fever		2	6	44	59	14	4	7	14	150
Tuberculosis (all forms)		42	6	192	62	20			33	355
Typhoid and paratyphoid fever				36	1	1	1		3	42
Undulant fever				9	1			1		11
Whooping cough		23	1	93	69	9	23	27	32	277

JAMAICA

Notifiable diseases—4 weeks ended September 23, 1944.—During the 4 weeks ended September 23, 1944, cases of certain notifiable diseases were reported in Kingston, Jamaica, and in the island outside of Kingston, as follows:

Disease	Kingston	Other localities	Disease	Kingston	Other localities
Cerebrospinal meningitis		1	Leprosy		1
Chickenpox	2	5	Tuberculosis	39	65
Diphtheria	7	6	Typhoid fever	12	84
Dysentery		2	Typhus fever	3	
Erysipelas		2			

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK[1]

NOTE.—Except in cases of unusual incidence, only those places are included which had not previously reported any of the above-mentioned diseases, except yellow fever, during the current year. All reports of yellow fever are published currently.

A table showing the accumulated figures for these diseases for the year to date is published in the PUBLIC HEALTH REPORTS for the last Friday in each month.

(Few reports are available from the invaded countries of Europe and other nations in war zones.)

Plague

Algeria—Algiers.—For the month of September 1944, 29 cases of plague with 5 known deaths were reported in Algiers, Algeria. All precautionary measures have been taken.

Belgian Congo—Stanleyville Province.—Plague has been reported in Blukwa region of Stanleyville Province, Belgian Congo, as follows: Week ended September 16, 1944, 2 deaths; week ended September 30, 1944, 5 deaths.

Bolivia.—For the month of August 1944, 5 cases of plague were reported in Santa Cruz Department, and 3 cases of plague with 1 death were reported in Tarija Department, Bolivia.

Brazil.—Plague has been reported in Brazil as follows: January 1 to March 31, 1944, 89 cases with 17 deaths. For the month of April 1944, 5 cases of plague were reported in Brazil by States as follows: Alagoas, 1 case; Bahia, 1 case; Ceara, 3 cases.

Madagascar—Tananarive.—For the month of August 1944, 11 cases of plague with 9 deaths were reported in Tananarive, Madagascar.

Union of South Africa—Cape of Good Hope Province—Saint Marks.—A report dated October 4, 1944, states that during the past week 13 deaths from plague had been reported among natives in Saint Marks about 160 miles northeast of Port Elizabeth, Cape of Good Hope Province, Union of South Africa.

Smallpox

Bolivia.—For the month of August 1944, 180 cases of smallpox with 57 deaths were reported in Bolivia. Departments reporting the highest incidence are: Chuquisaca, 7 cases, 2 deaths; La Paz, 70 cases, 30 deaths; Potosi, 89 cases, 23 deaths.

Mexico.—For the month of August 1944, 277 cases of smallpox were reported in Mexico. States reporting the highest incidence are: Aguascalientes, 46 cases; Hidalgo, 43 cases; Oaxaca, 58 cases; Vera Cruz, 63 cases.

Turkey.—For the month of July 1944, 78 cases of smallpox were reported in Turkey.

[1] The monthly cumulative table regularly published in the last issue of each month will appear in the next issue.

Typhus Fever

Algeria.—For the period August 11–20, 1944, 12 cases of typhus fever were reported in Algeria.

Bolivia.—For the month of August 1944, 26 cases of typhus fever with 4 deaths were reported in Bolivia. Departments reporting the highest incidence are: Cochabamba, 7 cases; La Paz, 8 cases, 2 deaths; Potosi, 8 cases, 2 deaths.

Hungary.—For the week ended September 9, 1944, 21 cases of typhus fever (including 15 cases in Subcarpathia) were reported in Hungary.

Mexico.—For the month of August 1944, 109 cases of typhus fever were reported in Mexico. States reporting the highest incidence are: Mexico, D. F., 24 cases; Mexico, 22; Nuevo Leon, 17; Queretaro, 11 cases.

Rhodesia (Northern).—For the week ended August 19, 1944, 10 cases of typhus fever were reported in Northern Rhodesia.

Turkey.—For the month of July 1944, 210 cases of typhus fever were reported in Turkey.

Yellow Fever

Belgian Congo—Stanleyville Province—Babeyru.—For the week ended March 11, 1944, 1 fatal case of yellow fever was reported in Babeyru, Stanleyville Province, Belgian Congo.

Gold Coast.—For the week ended July 29, 1944, 1 case of yellow fever was reported in the northern territories of Gold Coast.

Venezuela—Tachira State—San Camilo (vicinity of).—For the period September 6–15, 1944, 3 cases of yellow fever were reported in the jungle area in the vicinity of San Camilo, Tachira State, Venezuela. A telegraphic report, dated September 27, states that 2 additional cases of yellow fever were reported from this same area.