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OCULAR SYMPTOMS AND SIGNS ASSOCIATED WITH DEFICIENCY OF VITAMIN B COMPLEX

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This is a report of our observations on a series of 590 adult patients, who were treated in the Free Dispensary of the Philippine General Hospital from January 2, 1942 until September 30, 1943. These patients complained of dimness of vision of both eyes, and they were found to be suffering from multiple vitamin deficiencies. These ocular symptoms and signs together with the associated lesions, however, disappeared upon administration of well-balanced and liberal diet, especially rich in the particular vitamins manifestedly lacking.

The number of cases was small at the beginning, but it soon increased and assumed the nature of an epidemic, reaching the peak of incidence about the end of 1942; but it subsided by the end of September, 1943. Previous to this period, similar cases had come to our attention, but they were few and far between. A preliminary report on this clinical investigation was presented to the Staff Clinical Conference of the Philippine General Hospital on January 16, 1943, in conjunction with the presentation of a case of retrobulbar neuritis with co-existing corneal lesion associated with multiple hypovitaminoses.

The patients were divided into three groups depending upon the ocular changes, namely: (1) those with retrobulbar neuritis, 467 cases; (2) those with corneal lesion, 79 cases; and (3) those with retrobulbar neuritis and corneal lesion co-existing, 44 cases. A few of the patients were hospitalized for a detailed study and observation. The case histories of five of these are included in this report. The rest were treated in the outpatient service.

Inquiry into the dietary history of each of these patients revealed that, for several weeks or months prior to the onset of the defect in sight, they had been sub-

sisting on very deficient diet—deficient not only in quantity but also in quality. This lack of adequate food was the inevitable result of the war. Wars have always been linked with hunger and disease. The majority of these patients were poor; many had lost their jobs soon after the outbreak of the war. Food had become scarse in Manila and neighboring provinces due to lack of transportation facilities, and consequently prices had soared beyond the reach of the common people. There was a time when many were thankful if they could eat twice a day. As a strong evidence that this epidemic was caused by dietary deficiency, the cases almost disappeared as soon as adequate supply of food had reached the masses and economic conditions had approached normalcy.

The history and the physical examinations disclosed that these patients were suffering, not from any systemic disease, but from vitamin deficiencies. Those patients who complained of dimness of vision but might be suspected of being afflicted by latent syphilis, multiple sclerosis, cardio-vascular conditions, or other diseases or disorders that might complicate the avitaminosis or which might render the diagnosis of vitamin deficiency not very obvious, were excluded from this series. Likewise excluded were other pathologic conditions obviously known to cause faulty absorption and utilization of foods. However, pregnant and lactating women who contracted this eye affection were included in the series.

The assistance of the internists was secured to establish the diagnosis in some cases. It is unfortunate that, due to lack of laboratory facilities, no tests for the estimation of the content of the principal vitamins in the blood or urine could be made. Similarly, it is to be regretted that synthetic vitamins were not available for administration to the charity patients. Tests for visual acuity and for dark adaptation, fundus examination, scotometry, and other important examinations were repeatedly made, not only of hospitalized patients, but also of many of these from the free-dispensary.

Besides the ocular syndrome, a great majority of these patients presented angular stomatitis, glossitis, and symptoms of multiple peripheral neuritis—varying in degree of severity and in duration and in the combinations of signs and symptoms. The diagnosis of hypovitaminosis B complex was given to these cases.

RETROBULBAR NEURITIS

The type of retrobulbar neuritis we observed was probably similar to that described by Elliot[1] in his *Tropical Ophthalmology*, where he cited many Japanese investigators who had studied the ocular lesions in beriberi (*kakke* in Japanese). However, there was one striking difference between the cases of retrobulbar neuritis described by Elliot and those under our study; namely, those were apparently found in frank cases of beriberi; ours were associated with vitamin B complex deficiency.

In collaboration with Professor S. Miyashita of Osaka, Japan, Elliot wrote a brief and excellent review of the literature on retrobular neuritis of beriberi, citing Japanese investigators who blazed the trail in the study of this ocular disease in Japan. According to Elliot:

> In 1895, Kono published 6 cases of beriberi associated with amblyopia, central scotoma, but presenting no fundus changes; three years later Komoto discovered that the cause of amblyopia and other central scotoma was to be sought in a retrobulbar neurities and noted that the scotoma be-

gan centrally and spread centrifugally; Yamamoto in 1903 first drew attention to an atrophic condition of the temporal side of the disk; Kagoshima, in 1918, made an anatomical examination of five cases in which *kakke*-scotoma was present, and found in all of them a distinct and limited atrophy of the fibers in the outer section of the optic nerve. Miyashita, citing Tshizu (1917) and Fugita (1918) said that the scotoma is connected with the blind spot by a narrow bridge giving it, he said, a "characteristic pestle-shaped form"; and he further stated that the color sense is disturbed in many of the cases, especially for green. Miyashita said that central vision gradually falls till it reached the low limit of 6/60 but that it seldom goes lower than this; that a certain degree of photophobia is observed and that some of the patients can see better in the house than out of doors; that Nakamura (1919) noted some interference with dark adaptation; that Yamaguchi found no marked contraction of the white field but that those for red and green are shrunken; while Ichihara said that contractions of the visual field occur, but are transitory in nature; that Miyashita cited Captain Ishiza who found (1918) that in the earlier stages of *kakke* there was a distinct decrease of sensibility of the cornea and conjunctiva in more than half of all the cases he examined; that Yamamoto stated that fundus examination may reveal: (1) no evidence of any fundus lesion; (2) partial atrophy of the papilla, limited to its temporal quadrant (stressed also by Ichihara); or (3) the whole of the optic disk may be involved in the atrophy.

The eye symptoms in beriberi according to Elliot may appear early in the disease, but as a rule they do not put in an appearance until the general signs of the disease have been recognized for several months. With regard to pathology Elliot cited Wright as saying that the affected nerves show a chronic degeneration, Wallerian in appearance. Miyashita said that complete blindness need never be anticipated. Seasonal incidence: Miyashita stated that in Japan beriberi is most frequent in summer. The ocular symptoms as a rule appear after an interval of one to three months from the commencement of the trouble; hence these eye complications are mostly found in November, December and January. The ophthalmic symptoms are more common in males than in females, a marked exception being found in the latter during pregnancy and after the lying-in-period. Since the papillo-macular bundle is selected for invasion, this disease should be differentiated from tobacco-alcohol amblyopia, lead poisoning, and other intoxications, although the ocular symptoms are the same.

The treatment consists in substituting for the dangerous forms of polished rice, some such food as a bean known as *azuki* (*Phaseolus radiatus*) or this bean combined with barley. Food should also contain a sufficiency of nitrogenous and fatty elements and should not be too bulky. Injections of pilocarpine and strychnine are useful adjuvants. With regard to the frequency of eye symptoms of beriberi in Japan, Ichihara found that in 6,000 out-patients there were 32 cases of amblyopia (0.53 per cent) and that of these 32, no less than 6 were due to beriberi (18.75 per cent).

We have quoted Elliot extensively to show that much of our knowledge about

this eye disease had already been acquired about 25 years ago. But we are greatly impressed by the nearness of our observations in this series to the findings of these Japanese investigators. Our findings corroborate theirs, except that no mention is made in Elliot's review of any associated corneal lesion in some instances and the concomitant clinical syndrome of angular stomatitis and glossitis.

During the period covered by this report we saw at least 467 cases; 215 were males (46%) and 252 were females (54%). As to age incidence, about 60% ranged from 26 to 45 years, the oldest being 65 years, and the youngest, 17 years.

The patients complained of foggy and dim vision, sometimes very marked (20/200 or even worse), but with neither pain in, nor tenderness of, the eyeball. As to duration of the complaint of foggy vision when first seen in the clinic, we obtained the following history: about 1 week in duration, 8% of the cases; over 1 week to 1 month, 41%; over 1 month to 6 months, 38%; over 6 months to 1 year, 6.5%; and over 1 year, 6.5%.

Regarding the amount of defect in the acuity of vision our records showed: vision less than 20/200, 17%; from 20/200 to 20/100, 46%; from 20/70 to 20/50, 27%; and from 20/40 to less than 20/20, 10 per cent.

"SUPERFICIAL VASCULARIZING KERATITIS"

In the second group of patients we attributed the dimness of vision to the corneal lesion, which we would term "superficial vascularizing keratitis". For we believed the lesion to be similar to that peculiar type of keratitis described by Sydenstricker and his associates.[2, 3] But those cases in which the visual defect was very marked and out of proportion to the corneal opacities present and which scotometry revealed central or centrocecal scotoma, were placed in the third group of patients, i.e. those with retrobulbar neuritis and corneal lesion co-existing.

The usual complaints of patients with corneal lesions were: mild photophobia, smarting sensation in the eye, dim, foggy vision, more so when the patient was in the sunlight; no history of night blindness. Oblique illumination showed that the central portion of the superficial layers of the cornea occupying an area about 5 mm. in diameter was steamy. Sometimes this haziness was so slight as to be easily missed. Sometimes this cloudiness or nebula took the fluorescin stain, sometimes not.

Fundus examination: With the ophthalmoscope at a distance, haziness of the red reflex was observed. The fundus appeared normal except for the haziness of the details caused by the corneal opacities. Slit lamp examination revealed this haziness to consist of fine opacities, irregularly scattered, sometimes coalescing and forming patches of denser opacities, sometimes arranged in linear formations. Optical section showed these opacities to be confined in the epithelial layer of the cornea. Sensibility of the cornea was usually impaired not only in the area where the cloudiness was, but also in the whole corneal surface.

When the corneal microscope was focused on the limbal area, the limbal plexus was seen to be engorged. New capillary loops were formed and from them sprang very fine capillary twigs advancing under the epithelial layer towards the center of the cornea. At the start of this investigation, we failed to focus our attention on this vascular engorgement of the limbal plexus, as the eye did not present any circumcorneal injection on mere inspection; and we therefore missed this very important lesion

in some of the cases. No associated iris or pupillary changes were observed. We are sure many cases of the mild type passed unrecognized in a busy clinic.

What was this corneal lesion? What was its pathogenesis? Considering the history of dietary deficiency, the presence of angular stomatitis and glossitis in many of the patients, the similarity of the corneal lesion to the ocular manifestations of ariboflavinosis described by Sydenstricker and his associates, and the prompt improvement following the administration of balanced and liberal diet particularly rich in riboflavin, we are of the opinion that we were dealing here with "superficial vascularizing keratitis" due mainly to riboflavin deficiency. If we did not observe in our cases marked photophobia, marked ciliary injection, extensive new corneal vascularization and opacities involving also the deeper layers of the cornea, and the involvement of the iris, it is probably because we were dealing here with a mild degree of vitamin B_2 deprivation. If this vitamin deficiency were allowed to progress, it is very probable that we would have met with ocular changes very similar to those described by Sydenstricker and his associates.

We believe it pertinent to describe here the ocular symptoms and signs of ariboflavinosis first reported by Sydenstricker and his associates. In June 1940 they published a report of their findings in a series of 47 patients with ocular symptoms and signs of ariboflavinosis. Photophobia and dimness of vision were the most frequent symptoms in their patients. The earliest and most common sign of ariboflavinosis, according to them, is circumcorneal injection. Frequently, it can be seen with a hand lens or ophthalmoscope, and it is always obvious on slit lamp inspection as marked congestion and proliferation of the limbic plexus. Actual invasion of the cornea by capillaries arising from the apices of loops of capillaries in the limbic plexus can be readily seen by retro-illumination just beneath the epithelium. Later extensive superficial vascularizations are produced, and these are termed "superficial vascularizing keratitis" by Sydenstricker and his associates.

The superficial plexus is always more extensive and complicated than the posterior, in contrast to the predominant posterior vascularization in syphilitic interstitial keratitis. Meanwhile superficial nebulae, seen grossly as "steaminess" of the cornea and with the slit lamp as a fine diffuse superficial opacity, usually developed.

Sydenstricken and his associates were the first to report on the slit-lamp examination of the cornea and iris in human ariboflavinosis. Associated with these changes is a tendency to marked mydriasis (spastic mydriasis) and to the production of pigmented spots on the anterior surface of the iris. The eye lesions respond rapidly and satisfactorily to the administration of riboflavin. Superficial opacities disappear more slowly than interstitial nebulae.

The order in which these various lesions appears, evident in cases allowed to relapse, is as follows: The first sign to appear is conjunctival injection, followed by photophobia and impairment of visual acuity. Corneal opacities are encountered 7 to 10 days after re-instituting a deficient diet. Cheilosis and glossitis seldom recur before the end of the second week.

In our consideration of the diagnosis of the corneal lesion, we ruled out "prexerosis corneae" described by A. Pillat[4] in xerophthalmia in adults. Unlike prexerosis corneae we did not observe any accompanying loss of lustre of the conjunctiva or cornea in our cases; no "drying-out" phenomenon (i.e. the cornea becomes dry after exposing it from 5 to 10 seconds by retracting the lids); no "characteristic exfoliation

of corneal epithelial cells" which Pillat says are well shown by the slit lamp. Xerosis epithelialis corneae rarely occurs alone; it is often associated with one of the various types of xerosis of the bulbar conjunctiva. As a matter of fact, we did not observe xerosis of the conjunctiva in any of the cases in this series nor did we elicit history of night blindness. It is generally considered that "night blindness is the commonest and most easily recognizable manifestation of A avitaminosis." Another point against xerophthalmia is the engorgement of the limbal plexus and the presence of new vascularization.

In some of the patients in this series biophotometry revealed poor dark adaptation. This, however, was not a constant finding in those with still a fairly good vision. Because of this finding, vitamin A deficiency might be assumed. But poor dark adaptation may be produced by conditions other than lack of vitamin A in the retina. In our cases, this may be attributed to A hypovitaminosis, to the corneal opacities, to the retrobulbar neuritis, or to all the three conditions combined. Furthermore, as Gordon and Servinghaus[5] say, "There is the widest possible disagreement as to the reliability of the light sense in detecting this deficiency, the frequency of A avitaminosis in the population generally and the accuracy of any of the instruments now in existence for adequately measuring dark adaptation ... The subjective elements of cooperation and training are of the greatest significance in obtaining reliable results. Repeated tests on the same subject tend to produce a certain facility in performance which is not proportional to any true improvement in efficiency of adaptation. Vitamin A requirements of a single individual may deviate from the average requirements of large groups arrived at by statistical analysis... It may therefore be concluded that some degree of caution is necessary in the interpretation of results."

Our consideration of whether this corneal epithelial lesion might be attributed to hypovitaminosis A brings us to a very pertinent question. Do we have evidence that keratomalacia in adults is found in the Philippines? We have never seen it or heard of it or read reports that it has been encountered here. Xerophthalmia in the form of xerosis conjunctivae and night-blindness in adults are accasionally found, but not a single one of the 590 patients in our series had these symptoms. The worst economic and climatic conditions in the Philippines that we have ever had have not been so unfavorable as in certain parts of North China where, Pillat reports, there are frequent outbreaks of keratomalacia in adults due to improper diet, severe winter, hard physical labor, and other influences.

Unless the supply of vitamin A is severely cut off for a long period of time, the liver in adults—provided it is not diseased—is capable of maintaining to a minimum level the vitamin A content of the blood. For this organ is a good storage of this substance. We believe that in the epidemic that we had it was the water-soluble factors which are known to be readily excreted that were depleted first and to a greater degree than the fat-soluble vitamin A.

In children, however, cases of avitaminosis A occur rather frequently. During the last epidemic, these cases increased about three-fold. In adults, it was the B complex; and in young children, the Vitamin A that we observed to be predominantly deficient. From January 1940 to September 1941, there were 69 cases (0.8%) of xerophthalmia and 18 cases (0.22%) of keratomalacia out of a total of 8627 eye cases, as against 197 cases (2.50%) of xerophthalmia and 49 (0.63%) of keratomalacia out of a total of 7771 eye cases for the corresponding period from January 1942 to

September 1945. Seventy-four per cent of the cases of keratomalacia were in children under three.

With regard to age incidence of those patients with corneal lesion, we found the following: 70% were from 21 to 35 years old; the oldest in the series being 58 years old and the youngest 14 years.

As to sex incidence, 58 per cent of the patients were females and 42 per cent males. Pregnant and lactating women swelled the number of the former.

REPORT OF CASES

The following case reports are illustrative:

(Service of Dr. Fernando)

Case 1: C. T., 55 years old, male, married, plumber, admitted July 24, 1942 complaining of dimness of vision of both eyes. This had started two months before, as gradually progressive dimness of vision, accompanied by redness of the eye, slight photophobia and smarting pain in the eyes. He said he never suffered from night blindness. Complained of occasional vague pains in the lower extremeties. No numbness around the mouth. Angular stomatitis appeared after the dimness of vision.

He had been unemployed since the outbreak of the war. He had a large family, and had no savings. The family had been subsisting on very deficient diet, consisting most of the time of gruel, small dried fish, and some vegetables.

The important findings in physical examination follow: Fairly well developed and fairly nourished. Heart and lungs apparently normal. No seborrhea on the face; angular stomatitis present in both corners of the mouth; the lips slightly reddened, but not swollen. The tongue appeared glazed due to atrophic papillae, and without sensation of burning. Many carious teeth; several were missing; some exudate appeared on pressure of the gum (dentist's report, July 28: "advanced pyorrhea of all remaining teeth".) No tenderness of calf muscles. . Knee-jerk, normal. The eyes on inspection showed angular blepharitis, outer canthus; pupils normal; no ciliary injection. On oblique examination, haziness was visible in the central portion of the anterior layer of the cornea, the rest of the cornea appearing normal; no drying-out symptom or loss of lustre in the conjunctiva or cornea. Both cornea were hyposensitive, the right eye more than the left. The lesion in the cornea stained slightly with flourescin. Slit lamp examination showed that the opacities were confined to the anterior epithelium, the central portion corresponding to the pupillary area. The optical section showed definitely that the opacities were confined to the epithelium. At the limbus, many very fine new capillaries came from congested limbal loops, and crept under the epithelium towards the center of the cornea, but hardly reaching 2 mm. from the limbus. Vision: O.D. = 10/80 and O.S. = 10/120 and not improved with lenses. The patient could not read any of the Jaeger test types even with the presbyopic correction. Biophotometer test showed poor dark-adaptation (after exposure 4.64 M.C.L.) After ten minutes—0.948 M.C.L. Perimetry, showed slightly contracted field, temporal side, for form; contracted for green. Small absolute central scotoma was present in both eyes. Fundus examination: apparently normal fundus on both eyes; Ophthalmoscope at a distance showed slight haziness of tbe red-reflex. There was small subcortical opacity of the lens, more dense in the right eye than in the left and situated below the central portion of the lens, so that it does not interfere with the vision. Blood pressure—110 systolic; 64 diastolic.

Laboratory examinations:

Blood: R.B.C. = 4,350,000. W.B.C. = 7,520 cu. mm. Differential: neutrophile 69%; lymphocytes, 29%; monocytes, 2%.

Urine: normal. Feces: Ascaris, one plus and Trichuris one plus.

Basal metabolic rate — plus 12.5 (7/30/42).

Conjunctival smear — staphylococcus aureaus and diptheroids.

Blood chemistry (7/30/42) sugar — 77.5; uric acid — creatinine — 1.20.

Cerebro-spinal fluid (8/6/42) — Normal.

A dietary regimen was started Aug. 1st, consisting of a balanced and liberal diet, expected to give a minimum of 5 mgm of B_2 daily. As the source of this vitamin would also contain plenty of vitamin A, the patient was also receiving liberal supply of vitamin A (estimated to be at least 25,000 International Units.) Liver, whole eggs, camote, string beans, spinach, papaya, pork, etc. constituted most of his diet. No medicine was administered.

The vision began to improve on Aug. 4; it was O.D. = 10/80; O.S. = 20/60. On Aug. 13, V = O.D. 20/40; O.S. = 20/60. On Aug. 20 V = O.D. 20/30; O.S. = 40 and for near = Jeager No. 6 with plus 3.00 sph. On Aug. 25, V = O.D. 20/25; O.S. = 20/25 and near vision = Jaeger No. 1 with plus 3.00 sph.

Record of biophotometric readings: July 25, after exposure — 4.64 and after 10 minutes 0.948; Aug. 15, 4.98 and 0.400; Aug 29, 0.948 and 0.301; Sept. 16, 0.533 and 0.210.

The visual field was much improved by Aug. 4th, and on Aug. 29, normal visual field for form.

Slit-lamp examination on Sept. 15, showed: Vascular changes in the cornea had almost completely regressed. The lens opacity in either eye had remained the same, but this did not interfere with the line of vision because of its location; i.e. peripheral to the free margin of the iris.

Discharged on Sept. 17, 1942, recovered, near vision being Jaeger No. 8 in either eye, but with plus 3.00 sph. it was Jaeger No. 1. Central scotoma completely disappeared.

Case 2: — Ward 4 — Bed 23. — Service of Dr. A. S. Fernando

A case of Retrobulbar Neuritis with Associated Corneal Epithelial Opacities Secondary to Multiple Hypovitaminosis.

E.L., 38 years old, housekeeper, admitted for the first time on Dec. 18, 1942, with the chief complaints of dimness of vision of both eyes and numbness of the lower extremities. The present illness had started 3 months before admission as numbness and occasional cramp-like pains of both lower extremities usually occurring at night. Two months later she noticed a gradual diminution of vision to such an extent that close work like reading and sewing were impossible. The dimness of vision started in the left followed by the right eye weeks later. She consulted the E.E.N.T. dispensary where a series of thiamine chloride injections was given — 1 mgm. daily for 10 days. The treatment relieved her of the numbness around the mouth but no subjective improvement in the vision. She had history of receiving a trauma over the left eye two weeks before admission. No lachrymation or photophobia.

Diet.—Food at breakfast consisted of a cup of coffee and some rice cakes. At

lunch, meat was served about 4 times a week; first three times a week. Vegetables were usually mixed with the fish or meat, but vegetables were seldom cooked alone. Sometimes bananas or sweets. At supper same thing was served as their food was cooked once daily to economize on fuel. According to her, her appetite was not good, so that on many occasions she took very little supper or not at all. She was also nursing a child.

The most important findings on admission were: a slight angular stomatitis; tongue red; knee jerk absent in the left and very weak in the right.

Eyes — On the cornea corresponding to the pupillary area there were small opacities varying in size from pin point to pinhead, some clustered and some in linear formation. On oblique illumination, these opacities appeared whitish. On corneal microscopy, the epithelium presented areas of thickening which appeared whitish and glistening. With the corneal prism, these opacities were seen on the anterior surface. The opacities stained with flourescin. The limbal plexus was congested. New capillaries were advancing toward the center of the cornea. Pupils of both eyes were practically normal in size and shape, and reacted well to light and distance. Corneal sensibility — slightly hypersensitive. She could be examined with the slit lamp for a long time with little discomfort and no epiphora.

Vision — F.V. — O.D. — c. f. at 12 ft. chart with white backgound = 3 1/2 ft. (black background) O.S. = 22 ft. — Not improved with lenses. O.S. = c. f. at 7 ft. — Not improved with lenses.

N.V. — O.D. — Jo — Not improved with lenses. O.S. — Jo — Not improved with lenses.

Fundus examination — Some black spots on an orange red reflex were seen on ophthalmoscopic examination at a distance. There was slight pallor of the temporal portion of the disc of both sides, otherwise normal. Perimetry showed slight contracted field for white and for colors (red and blue.) She could not recognize the green. Biophotometer test — poor dark adaptation. There was however no xerosis or dryness of the conjunctiva. Scotometry — Centrocecal scotoma in both sides, larger in the left. Test for color blindness (1) using Ishihara's book — the right could see figures on plate I only, while the left could not see the figure in any of the plates. (2) Worsted's Yarn — Could see the fundamental colors. She distinguished the red well; she recognized the blue better than the green.

Laboratory examination — Blood Wassermann — negative
Blood chemistry — normal
Urine — normal

Patient was referred to the Neurologist who gave the diagnosis of "Polyneuritis secondary to hypovitaminosis."

Discharged Jan. 13, 1943, with V = O.D. = 20/160 O.S. counting fingers 16 ft. Corneal opacities very slight.

Case 3: — (Service of Dr. Vitug) — R. A., 19 years old, female, single, from Orion, Bataan, admitted on February 14, 1943, with the following complaints: inability to walk, dimness of vision in both eyes, numbness and formication of upper and lower extremities, and scabies. The dimness of vision had developed about two months before admission. A month later her legs became weak and numb, and she found it difficult to walk; she complained of numbness around the mouth, of

chest oppression, and of angular stomatitis before admission. For the past eleven months she had been having recurrent attacks of malaria.

The important physical findings were: scabies, angular stomatitis, glazed and reddened tongue, heart apparently normal, knee jerk, absent. Patient could not stand up without support.

Laboratory examinations revealed nothing important, except that there were a few malarial parasites (schiz). Blood Wassermann test—negative. The diagnosis given was Hypovitaminosis B complex, Chronic malaria, and scabies.

Vision : O.D. = 8/200 O.S. = 8/200 Left eye with slight ciliary injection.

Centrocecal scotoma 4 mm, white object at 1000 mm.

Biophotometer test showed: poor dark-adaptation (After exposure — 4.98 M. C. L. and after 10 minutes — 1.18 M. C. L.)

Slit-lamp examination showed fine superficial opacities in the central portion of the cornea; very fine new capillaries arising from the congested limbal plexus, advancing toward the center of the cornea, and situated just under the epithelium.

Observation on the course of the disease:

Anti-beriberi diet was given. Thiamine chloride, 1 cc. by hypo daily. Tikitiki extract, 10 cc. t. i. d. The thiamine was changed to betaxin from March 5 to April 6 for lack of that medicine. Quinine was given for the malaria and sulphur ointment for the scabies.

March 2, 1943 — the vision improved to 10/160 in either eye.

March 5, 1943 — the haziness of the cornea had disappeared. The capillaries were still visible near the limbus, although they appeared smaller.

April 14, 1943 — Patient could walk slowly. Calf muscles as well as those of the palm slightly atrophied. The tongue papillae were already regenerated. Vision was much improved.

Case 4: — D. L., 30 years old, male, married, jobless, admitted on September 16, 1942, for dimness of vision in both eyes which he had noticed two months before admission. Numbness around the mouth and tips of fingers and toes. Had slight photophobia and could see better in the shade. Because of poverty his food was very inadequate, but his appetite was good. Physical examination revealed, some carious teeth, a fairly developed, fairly nourished, strong patient. The neurologist gave a diagnosis of peripheral neuritis due to hypovitaminosis B. Laboratory examination:

Wassermann tests of the blood and cerebro-spinal fluid, normal. Eye examination: Conjunctiva and cornea, normal.

Vision = O.D. 20/200, O.S. = 20/200, and for near, Jaeger No. 11 in either eye, not improved by lenses.

Centrocecal scotoma for 2 mm. white, at 1000 mm., present.

Biophotometer test: September 24: After exposure 2.41 M.C.L. After 10 minutes — 0.464 M.C.L.

September 18 to October 3 — 4 teeth extracted.

Medical diathermy, exposed for 15 minutes daily for 9 days. Adjuvant treatments: strychnine.

Discharged November 21, 1942 with

Vision = O.D. 20/80 J. No. 2
O.S. 20/80 J. No. 2

Case 5. — Ward 6 Bed 8 — (Service of Dr. Gutierrez)—A Case of Hypovitaminosis. — L. R., 28 years old, female, single, housekeeper, Para V, Gravida V, born in Leyte, residing in Cavite, admitted on December 1, 1942, with the chief complaint of being easily fatigued, general numbness, and dimness of vision.

Illness was of 9 months duration, having started as a slight numbness of the hands and feet accompanied by occasional pricking sensations, which became worse when the patient was 7 months pregnant. Two months later, she had normal parturition. The numbness was slow in its progress upwards until after 3 months when it rapidly involved the thighs, abdomen, and chest; and was accompanied by breathlessness on walking short distances. She also had a sensation of thickening around the mouth and linear area of paresthesia in the back for about the same length of time. About a month previously, she had lacrymation and redness of the eyes which lasted two weeks and subsided without treatment. Since then her vision had been impaired. For one week now she was unable to go about on her feet. Except for the occasional vague abdominal discomfort for two months there were no other symptoms.

Socio-economic history—She had come to Manila 9 years before from Leyte Since then she had been living with a common-law husband until the previous year when he had to leave for abroad. Up to then the family had never suffered from want as the husband's salary was adequate for the family of five. After her husband's departure, however, she had to depend on her in-laws, of whom only one was a wage-earner (₱1.50 per day); and he had to support a family of 10. Their diet consisted of rice, fresh or dried fish, and occasional vegetables in the form of kangkong and young sweet potato sprouts. She abstained from meat in the belief that it made her numbness worse. Two of her 3 living children had to be confined for deficiency diseases. She was not a chronic alcoholic but she drunk Chinese wine occasionally.

Important physical findings: Fairly developed and somewhat poorly nourished, unable to walk unaided and unsteady on her feet. No angular stomatitis. Chest symmetrical with lactating breasts. There was a slight enlargement of the heart to the right (area of dullness on percussion was 1-1/2 fingers from the right sternal border). Heart sound rather weak—Rate 110/min. Moderate accentuation of the second pulmonic sound. Lungs normal. Abdomen, liver, and spleen not palpable. Linea gravida present.

Extremities—negative ankle and knee jerks; no adventitious reflexes; no foot-drop; Romberg sign—positive. Impaired sensibility to pain, touch, and temperature —most marked in the hands, forearm, feet and upper abdomen and chest.

Laboratory Examination: Wassermann test—negative.

This case was referred to the oculist and the vision showed 20/200 in either eye, not improved by lenses. Fundus examination revealed media clear as well as fundus details. Nothing abnormal in the fundus. The visual field showed marked contraction mainly in the temporal portion. Impression given was neuritis, retrobulbar, bilateral. Dark adaptation poor. Treatment—cod liver oil, 15 cc. after each meal three times a day with betaxin (2 mgms.) ampules injection once a day.
Note on the progress:—

By Dec. 12, 1942, patient could walk unaided, but there was no improvement in the vision.

COMMENT

We will comment first on the retrobulbar neuritis. The lesion is supposed to be in the papillo-macular bundle of the optic nerve, and our hypothesis is that this nerve is susceptible to attack of possibly a toxic substance in the presence of vitamin B complex deficiency. Apparently this injurious process does not operate so frequently in frank, uncomplicated beriberi, i. e., when the signs of B_1 deficiency are prominent and unmistakable, and without concomitant ariboflavinosis.

This view seems to be supported by our hospital statistics which show that rarely did our uncomplicated beriberi patients complain of poor vision. Our hospital figures show only 8 cases of infantile beriberi were observed in 1942, and not a single case from January to September, 1943. With regard to adult beriberi, there were 112 cases out of a total admissions of 36,891, or 0.3% from January 1940 to September 1941 as against 16 cases out of a total admissions of 20,424, or only 0.078% from January 1942 to September 1943. As to pellagra, 9 cases were recorded in the hospital during the former period, or 0.02%; and 3 cases, or 0.01% in the latter period.

One striking feature noted during the period covered by this report is that there were relatively fewer cases of infantile beriberi, adult beriberi, and pellagra admitted to the hospital, but very frequent occurrence of cases of B complex deficiency in adults was noted in our out-patient service.

What is the nature of this injurious attack to the optic nerve? Veasey, (citing reports of Yudkin, Carrol, Goodhart, Johnson, Laval, Buschke) says that toxic amblyopia occurs by action of certain poisons on the optic nerve when the threshold has been lowered by avitaminosis B and can be cured by taking the latter. Veasey also mentioned Shastid as reporting in 1929 two cases of optic neuritis cured promptly by B complex and Soga (cited by Buschke) who reported an outbreak of the so called beriberi amplyopia (now known as chronic retrobulbar neuritis) in Japan. It is unfortunate that we have no access to the current Japanese medical literature, owing to language difficulties. It is to be remembered that the Japanese pioneered in the investigation of ocular lesions in beriberi.

In connection with this unusual frequency of cases of B complex avitaminosis during the war, we should like to quote Spies and his associates: "During the past ten years, from our studies of deficiency diseases in human beings, we have accumulated evidence which shows that pellagra, beriberi, and riboflavin deficiency are clearcut clinical syndromes which frequently co-exist and often are associated with other nutritional disorders. This is not surprising, since in natural foodstuffs the water-soluble vitamins are often closely associated and a diet deficient in one chemical substance is almost certain to be deficient in others."

With regard to the visual defect observed in this epidemic, it is consoling to state that we did not see a single case of blindness or atrophy of the optic nerve. This is probably due to the fact that the deprivations did not last long.

What is the pathology of the nerve lesion? We cite Vedder who, in discussing the degenerative changes in the peripheral nerves, said that in most cases these degenerative changes are not to be regarded as complete. This would mean the death of the cell, from which there could be no recovery; but they do indicate a very complete exhaustion of the affected parts. Degeneration of the myelin sheath

is constant, according to him, but the majority of the nerve cylinders appear normal, even when the medullary sheath shows advanced degeneration.

At this juncture, we would like to say a word or two about the diminished sensibility of the cornea and conjunctiva in many of our patients. Many also complained of numbness around the mouth which completely disappeared when the patient recovered. Recently we saw a beriberi patient with paralysis of the left vocal cord. She complained of numbness around the mouth, but no complaint in the eyes. But when the corneal sensibility was tested, it was found much diminished. We explain these findings by changes in the terminal branches of the trigeminal nerves supplying those parts, the impairment of function being due not to complete degenerative change but as "complete exhaustion of the affected parts of the nerve."

As we have stated elsewhere, the corneal lesion is probably due to mild B_2 avitaminosis. The circumcorneal injection and the new vascularization in the cornea that came to our attention were not so extensive as those described by Sydenstricker and his associates. No iris changes were observed. Seborrhea of the nasolabial folds and face were not frequent; the burning sensation in the tongue was not frequently complained of.

However, Sydenstricker and his associates did not mention any co-existing retrobulbar neuritis. In our cases, we suspected this whenever the dimness of vision was altogether out of proportion to the corneal opacities present. We noted that case No. 8 in their series[2] had a vision of only 5/200, but no mention was made whether scotometry did not reveal a central scotoma. Likewise, the findings of the Japanese investigators on the cases of retrobulbar neuritis as noted by Elliot did not mention any co-existing superficial vascularizing keratitis. We do not know what the current medical literature of Japan says regarding this point.

With respect to nicotinic acid deficiency in B complex avitaminosis, we can say that some cases of glossitis we encountered might be attributed to the former. It is claimed that if the tongue is scarlet red and denuded and if the papillae are atrophied, the deficiency must be in the nicotinic acid; whereas if the tongue is clean, purplish red or magenta, and frequently fissured, and if the papillae are large, flattened or mushroom-shaped, and with "scalded" feeling at the tip, the deficiency must be in the riboflavin.

We have described in this report an epidemic of eye diseases, the probable cause of which might be ascribed mainly to multiple vitamin deficiencies brought about by very low intake of food. We have not seen a similar epidemic before, nor has one ever been reported in the Philippines. Cases similar to these affections have appeared sporadically in our clinics, but they have been few.

One of us (A. S. F.) treated a case of retrobulbar neuritis of this type a few years ago, the record of the case being as follows:

> Free Dispensary Case No. 379: D. E. 20 years of age, examined on January 13, 1935. Husband, a chauffeur. Complained of foggy and dim vision of 3 months duration, without either pain or redness in the eye; also of numbness of the extremities. These symptoms appeared soon after delivery. First para. Child healthy; breast-fed; was given extract of tikitiki as a preventive measure. The vision was counting fingers 12 feet in the right eye and 10 feet in the left. Fundus, normal. Visual field not contracted. A definite absolute central scotoma in both eyes present. Corneal sensibility, normal. Knee-jerk, normal.

Treatment consisted of balanced diet, but particularly rich in vitamin B_1; also strychnine. Recovered.

Ocular manifestations of avitaminosis have long attracted our attention, and papers on this subject have appeared from time to time. [9, 14] But our work had always encountered a stumbling block: the determination of the specific vitamin deficiency responsible for the particular eye lesion.

The same difficulty impeded our progress in this present investigation. Foremost obstacle was the lack of modern laboratory facilities for the estimation of the different vitamin content of the blood or urine, particularly of the vitamin A. Biophotometry is no longer reliable. The clinical findings should be carefully checked for the amount of the vitamin A in the blood. Etiologic diagnosis is difficult to make with a certain degree of assurance when the deficiency is of mild degree, because the clinical picture is not clearcut in many cases. When B_2 deficiency is too pronounced, it has been claimed by Sydenstricker that it is possible to recognize it more easily than others on account of the specific lesions of the eye which occur early in the period of deficiency. This claim has been confirmed by us in a few cases.

There are probably other factors contributing to the development of these clinical hypovitaminosis, since other members of the patient's family did not necessarily manifest the known clinical syndrome even if all the members of that family ate practically the same kind of food and in proportionately the same amount. It is possible that the others may develop sub-clinical state and that some precipitating factors may cause the sudden appearance of the recognizable characteristic manifestations of the particular disease any time.

With respect to the therapy, we share the general conception that by the administration of a balanced and liberal diet particularly rich in the vitamins in which the patient is supposed to be deficient, we take advantage of the synergestic relation between vitamins—i. e. one influencing others favorably. Patients in our service who could afford it were asked to supplement the prescribed diet with synthetic vitamins, particularly with the vitamin B complex. Extract of tikitiki was also recommended. As adjuvants, medical diathermy was tried, but we are not in a position to assert how much benefit, if at all, was derived from it.

SUMMARY AND CONCLUSION

We have reported here an outbreak of nutritional eye disease associated with multiple vitamin deficiencies. This seems to be the first epidemic of this nature ever reported in this country. The chief complaint was dimness of vision in both eyes, the cause of which was: (1) chronic retrobulbar neuritis or (2) superficial vascularizing keratitis or (3) both affections co-existing. The patients invariably gave the history of having subsisted for some time on very deficient diet—deficient both in quantity and in quality. They were on the whole healthy up to the time of onset of their complaint. Adult males and females were attacked. In females, pregnancy and lactation were predisposing factors.

In many patients associated signs and symptoms of vitamin B complex deficiency were observed—namely, angular stomatitis, glossitis, numbness around the mouth, hyperesthesia or hyposthesia in the extremities, and other manifestations.

The retrobulbar neuritis was probably brought about by several known and unknown causes but mainly by the deficiency of the B_1 factor and especially of the

vitamin B complex: Likewise, the superficial vascularizing keratitis was brought about by similar mechanism—the deficiency of the B_2 factor of the same vitamin B complex playing a predominant role.

The essence of the treatment was the administration of a well-balanced and liberal diet, especially rich in vitamins that were suspected to be insufficient. Prompt response was noted. In those cases in which a let-up in the liberal dietary regime could not be helped owing to economic reasons, relapse occurred.

The outbreak of the epidemic commenced soon after the war, reaching the maximum height of incidence about December 1942 and practically disappeared about the end of September 1943. The cause could be traced to the sudden dislocation in the supply of foods and the poor economic conditions of the masses occasioned by the national emergency. Unfortunately, no laboratory tests were employed for the estimation of the different vitamin content of the blood or urine to check the clinical diagnosis of hypovitaminosis. It was, therefore, found necessary to establish this diagnosis by dietary trial. Since satisfactory results appeared promptly after dietary therapy alone, the presumption of a deficiency disease was considered verified.

We conclude this report by emphasizing that there is a great necessity for continuing the study of nutritional eye diseases in collaboration with the internist, the neurologist, the nutritionist, and the pathologist. There is likewise an imperative need of recommending to the public a balanced diet that is both easily available and within the reach of even the poorest in order to help forestall future outbreaks of serious epidemics of deficiency diseases in the face of sudden and unexpected national emergencies that may disrupt the adequate supply of foods for the masses.

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SIMPLE QUALITATIVE TESTS FOR ADULTERANTS IN FRESH MILK

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INTRODUCTION

Milk is considered to be the best single food. Its high nutritive value is due to the fact that it contains in the right proportions the essential elements of a balanced diet. It is rich in some vitamins. The quality of the protein is especially good for body building. The fat content favors growth. And it has high calcium content in readily usable form. Milk is considered a protective food against certain deficiency diseases when used in combination with other foodstuffs. Furthermore, milk is very palatable and readily digestible.

On the other hand, milk may cause illness and even death when contaminated with pathogenic micro-organisms or with poisonous chemical substances. Contamination of milk with harmful bacteria is rather common, as shown by reports of epidemics of typhoid fever and other diseases traced to contaminated milk. Of all the foodstuffs, milk is perhaps the most difficult to harvest, handle, transport, and deliver in a clean, fresh, and satisfactory state.

Due to the high cost and scarcity of fresh milk in the Philippines, adulteration of milk is still frequently practised by unscrupulous dealers. Adulteration is always objectionable, because it lowers the nutritive value of milk and so changes its composition as it make it less easily digestible. The danger of this practice is great, because of the facility with which milk may be contaminated with pathogenic bacteria during the process of adulteration.

PURPOSE AND SCOPE OF THIS PAPER

This paper was written in response to the request of Dr. M. C. Icasiano, City Health Officer, to provide simple qualitative tests for adulterants in fresh milk, which can be performed easily in the field especially in the provinces where elaborate laboratory equipments are not available.

The simple tests for adulterated fresh milk are based mostly on accepted standard chemical procedures and partly on the past experiences of the author as a chemist analyst of the Food and Drug Laboratory of the government.

FORMS OF ADULTERATION OF FRESH MILK

The most common methods of adulteration of fresh milk as found in samples submitted to the government Food and Drug Laboratory are the following: (1) watering with or without the addition of sugar, (2) watering with the addition of starch and sugar, (3) addition of diluted coconut juice to fresh milk with or without the addition of sugar.

Less common forms of adulteration of fresh milk encountered in laboratory sam-

ples include the following: (1) addition of soya bean milk with or without sugar, (2) use of yellow coloring matter to diluted milk, (3) removal of some of the fat content by skimming, (4) use of chemical preservatives to retard the decomposition of the milk, and (5) use of alkalies to neutralize acidity due to the souring of milk.

The main object of the milk dealers in adulterating fresh milk is to increase the volume for bigger profit and to simulate as much as possible the normal physical characters of fresh milk. The use of impure water, dirty bottles, and cans contaminates the milk and renders it very dangerous to public health, as the adulterated milk is usually not sterilized before it is sold to the public especially in rural communities.

QUALITATIVE TESTS FOR ADULTERANTS IN MILK

1. Addition Of Water Or Watering:

   Watering may be detected because it lowers the specific gravity of the milk. The specific gravity of milk is usually determined by a simple instrument known as the lactometer.

   In its fresh state, the specific gravity of pure cow's milk varies from 1.027 to 1.035. The specific gravity of carabao's milk varies from 1.030 to 1.035 while that of pure goat's milk is from 1.028 to 1.036.

   The determination of specific gravity is useful in detecting the adulteration of milk by simple dilution of the milk with water which lowers the specific gravity. Skimming of milk or removal of butterfat also lowers the specific gravity. However the addition of sugar or starch or any other substance which increases the total solids tends to raise the specific gravity. When milk is diluted or skimmed or when other substances are added to it, specific gravity test alone is not a reliable test for the addition of water.

2. Cane Sugar In Fresh Milk:

   Cane sugar is often used to increase the total solids in milk; and, if it is present to any marked degree, it can hardly fail detection because of the sweet taste imparted to the milk.

   Cane sugar in milk may be detected by boiling for a few minutes 5 to 10 cc. of the sample with about 0.1 gram of resorcin and a few drops of hydrochloric acid. If cane sugar is present, the milk becomes rose-red.

3. Starch In Fresh Milk:

   Heat about 5 cc. of the milk in a test tube to boiling, cool, and add a drop of iodine solution containing potassium iodide. A blue color indicates the presence of starch.

4. Coconut Juice In Fresh Milk:

   Evaporate to dryness about 20 cc. of the sample in a porcelain casserole on a water bath. Remove the casserole from the water bath and heat directly over a flame. A characteristic "copra" odor indicates the presence of coconut juice.

5. Soya Bean Milk In Fresh Milk:

   Evaporate to dryness about 20 cc. or more of the sample in a porcelain casserole on a water bath. While still hot, note the color. A characteristic "soya bean cake" odor denotes the presence of soya bean milk.

6. Yellow Coloring Matter (caramel or burnt sugar, annatto or *achuete*, anilin orange, etc.) In Milk:

   Place about one-half glassful of the sample in a porcelain casserole. Add a

few drops of acetic acid and heat over a flame. Remove the supernatant fluid by decantation. Press the curd to remove as much water as possible and transfer the curd into a flask, add ether and allow it to soak for several hours.

A brown curd indicates the presence of caramel. A bright orange curd denotes the presence of "Anilin Orange," a coal tar dye.

Evaporate the other extract on a water bath to dryness. Allow it to cool. Alkalinize it with a little sodium hydroxide solution, and pour it through small wet filter. On washing off the fat from the filter paper with water, an orange color on the paper indicates the presence of Annatto or *achuette*.

7. Chemical Preservatives In Milk:

(a) Boric Acid:

10 cc. of the milk sample is thoroughly mixed with 6 drops of concentrated hydrochloric acid, after which turmeric paper is moistened with the mixture and dried. A brownish color of the turmeric paper denotes the presence of boric acid.

(b) Benzoic acid and Salicylic acid:

Shake 5 cc. of hydrochloric acid with about 50 cc. of the milk in a flask. Then add one-half glass of ether, cork the flask, and shake it well. Extract the curdled milk with several portions of ether, avoiding the formation of an emulsion. Transfer the ether extract to a separatory funnel and shake with diluted ammonia water to take out the benzoic acid from the fat as ammonium benzoate. Evaporate the ammonia solution in a dish over a water bath till all free ammonia has disappeared. But before getting to dryness, add a few drops of ferric chloride solution.

A characteristic flesh-colored precipitate indicates benzoic acid. A violet color precipitate denotes presence of salicylic acid.

(c) Formaldehyde:

Add about 10 cc. of commercial hydrochloric acid containing a few crystals of ferric chloride to an equal volume of the milk sample in a porcelain casserole and heat slowly over the free flame nearly to boiling, giving it a rotary motion to break up the curd.

The presence of formaldehyde is indicated by a violet coloration varying in depth with the amount present.

Remarks: The use of chemical preservatives or alkalies in milk is not allowed by law. The best milk preservatives are cleanliness and refrigeration at a temperature below 45°F.

8. ALKALIES (Sodium carbonate and Sodium Bicarbonate).

Evaporate to dryness about 25 cc. of the sample in a porcelain casserole on a water bath. Heat the residue over a flame until reduced to ash. Allow to cool and add a few drops of hydrochloric acid. Effervescence denotes the presence of carbonates.

SUMMARY

1. The most common methods of adulteration of fresh milk in the Philippines include the following: (a) watering with or without the addition of sugar; (b) watering with the addition of starch (thickening agent) and sugar; and (c) addition of diluted coconut milk with or without the addition of sugar.

2. Less common forms of adulteration of fresh milk are the following: (1) addition of soya bean milk with or without sugar, (2) use of yellow coloring matter in diluted milk, (3) removal of some of the fat content by skimming, (4) use of chemical preservatives to retard the decomposition of milk, and (5) use of alkalies to neutralize acidity due to souring of the milk.
3. Simple qualitative tests for adulterants in fresh milk based on accepted standard chemical procedures and on the past experiences of the author as a chemist-analyst, are compiled in this paper.
4. These tests which require no elaborate equipments can be performed easily in the field by medical officers, sanitary inspectors, and nurses.

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GALL BLADDER DISEASE: AN ANALYSIS OF CASES IN THE NORTH GENERAL HOSPITAL[1]

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Exactly what constitutes gall bladder disease is in many instances still a very debatable point. For the sake of convenience, we have included under this entity cholecystitis, gall stones, and ascaris in the biliary tract. These conditions are so intimately associated both clinically and pathologically that a discussion of one necessarily involves the others.

Cholecystitis may originate independently of gall stones, or gall stones may cause cholecystitis, but we believe that ascaris in the biliary tract is almost always secondary to a preceding pathologic disturbance in the biliary system.

MATERIAL

The material for this work was derived chiefly from the case records of patients in the Department of Surgery in the North General Hospital from the time of its establishment in February, 1945 to January, 1946. The charts of the surgical patients were carefully reviewed and those that seemed to come within the scope of this study were sorted out. Only those charts where the diagnosis were fully warranted by the presence of definite signs and symptoms of the disease or where the diagnosis were verified by operation were utilized. These patients were admitted for gall bladder disease, cholecystitis, empyema, biliary lithiasis, ascaris in the common bile duct, and in a few as cases acute abdomen.

For purposes of this study, we have decided to divide our cases into: (1) Operated cases and (2) Non-operated cases. It should be mentioned and understood that when a case is admitted to Surgery, there is always the belief or at least the possibility that a surgical intervention is contemplated. Hence a patient with only a mild attack of gall bladder disease is referred to the Medical Department and only those with severe attacks are admitted to Surgery. If a patient is not operated on, it signifies either that the attack has subsided and the doctor thinks that an operation is not necessary for the time being, or simply that the patient has stubbornly refused to submit to an operation in spite of the surgeon's attempts to persuade him. It has been our observation, however, that usually the attacks in patients who refuse to submit to an operation are not so violent, for a really severe attack usually does not give the patient a chance to be stubborn. Instead he usually urges the surgeon to perform the operation.

INCIDENCE

During the twelve months of the hospital's existence, we have admitted 78 patients with gall bladder disease to Surgery—all candidates for surgical intervention.

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[1] Read at the Staff Clinical Conference of the North General Hospital, January 28, 1946.

Of these, 38 were operated on and 40 were not. Of the 38 operated cases, 13 (34%) were males and 25 (66%) were females. In the non-operated cases, 13 (32%) were males and 27 (68%) were females. It will be noted that there have been about 2 times as many women as men in this series. Patients have ranged in age from 14 years to 71 years, although the greater number of cases have been found to be in the third decade of life.

In studies on the occurrence of gall stones, one of the few facts on which almost all authors are agreed is that women are more susceptible to gall stones than men. This conclusion is one of the outstanding features encountered in any review of publications about gall stones.

Fallis and McClure reviewed 320 cases of acute cholecystitis and found that the disease occurred twice as often in females as in males. Judd and Phillips reported 508 consecutive patients which came to operation, 40 per cent of whom were men. Among patients in the Mayo clinics, women's susceptibility to the formation of gall stones when compared with that of men gave the proportion of 1.8 to 1; and, in a combined summary of reports of similar statistics from 22 writers, this proportion was found to be 2.2 to 1. We are all familiar with the phrase "fair, fat and forty" and "flatulent, fecund, female", which has been emphasized by many as being the "characteristic type" in patients with gall bladder disease. Although it is true that we have found a preponderance of these patients in females, we regret to report that we have not seen patients who may come under the classification of "fair". Furthermore, we have found not infrequently the occurrence of this disease in emaciated individuals.

Why female patients in middle life are so prone to have gall stones has baffled the imagination of all investigators. The usual explanation offered for this fact has been that pregnancy favors the occurrence of gall stones (the majority of women in middle life have been pregnant). However, it is also a common observation that one may get gall stones without being fat or pregnant. The literature on the formation of gall stones is top-heavy with hypotheses, and the last word on the subject has not yet been said.

HISTORY AND SYMPTOMS

In the group of operated cases, a history of symptoms which had something to do with the biliary tract and which appeared prior to the attack under treatment was given by 35 patients. Only three definitely denied such symptoms. In the non-operated group, one half of the patients made no mention of any previous biliary symptoms or denied such symptoms. Many of the patients gave a history of gall bladder disease extending for many years, but the great majority could hardly recollect when the symptoms were first noticed. They could say "since childhood" or "even before she was married".

The duration of the present attack from the time of its onset to the time of admission ranged from a few hours to an indefinite period of over a month. The average duration for the operated cases was 4.5 days and for the non-operated cases 2.5 days.

All these cases have a common clinical character. The pain is typical and pathognomonic. It usually begins more or less suddenly in the epigastrium or right upper quadrant of the abdomen and is projected to the right back and shoulder. At

the height of each seizure, the patient doubles up and writhes in agony as the pain increases in ferocity. It is at this stage usually that a patient pleadingly asks that he be operated on. A measure of relief is felt when liberal doses of morphine are given. The pain may cease as suddenly as it began after a few minutes or hours, but the usual sequel is marked residual soreness in the region of the gall bladder for several days. In our series, we have found the pain of the biliary colic to be commonly localized at the right hypochondrium in 50% of the operated and in 67% of the non-operated cases. A large number had localizations in the epigastrium. In 26% of the operated cases, there was a radiation of the pain to the back and right shoulder; this was found in 10% of the non-operated cases.

We have found accompanying chills and fever a rather frequent symptom, being present in 55% of the operated and in 12.5% of the non-operated cases. Such a history was obtained by Steinke in 7% of his group with acute cholecystitis. Nausea and vomiting were present in many cases—in 34% of the operated and in 15% of the non-operated cases. Taylor found that 88% of his series of gall bladder disease experienced either nausea or vomiting. Nausea was noticed by Fallis and McClure in 91% and vomiting in 83% in patients with acute cholecystitis.

A history of jaundice was given in 21% of the operated and in 2.5% of the non-operated cases. A history of passing out ascaris was given by 10% of the patients in both series. The possibility of an ascaris lodging in the biliary tract and causing the acute obstructive symptoms is entertained whenever such a history is elicited.

Tenderness in the right hypochondrium has almost always been present, being elicited in 92% of the operated and in 80% of the non-operated cases. As much as possible, Murphy's method of demonstrating tenderness of the gall bladder has been followed—hook the fingers well up under the liver and tell the patient to take a deep inspiration. On inspiration, the pain becomes acute; and respiration is usually interrupted.

The majority of patients in the operated group had high temperature—38.5 to 39 C.; those in the non-operated group, 37 to 37.5 C.

The white blood cell count in the operated group was high—50% had 10 to 15 thousand; in the non-operated group — 55% had 6-10 thousand.

TREATMENT

In our series, 44 operations were performed—10 cholecystectomies, 12 cholecystostomies, 11 cholecystoduodenostomies, 8 lithotomies, and 3 removal of ascaris.

During the early days of the hospital, most of the operations on the biliary tract were cholecystoduodenostomies. This was due to the influence of one of the consulting surgeons, Dr. Estrada, who advocated the conservation of the gall bladder in the belief that if the gall bladder is saved, it may still regain its normal function, and that the creation of a detour for the flow of bile will obviate the resultant bad effects should there be a subsequent obstruction in the common bile duct.

Our present procedure is to remove the gall bladder. It is our belief that the gall bladder is the mischief-maker in the biliary tract. The troublous gall stones come from it. It is logical to remove the cause of all the trouble. We do not

believe in anastomosis because, first—we repeat—it does not remove the cause of trouble (the diseased gall bladder is more of a liability than an asset); and second, we feel that the bile will always try to follow its natural route and soon the anastomotic opening will contract and finally close up from disuse. In fact, one of patients who came here for empyema of the gall bladder had already had his gall bladder anastomosed to the duodenum, but the opening was no longer working and he had developed multiple stones and cholangiectatic abscesses.[1]

It will be noticed that we have a big number of cholecystostomies, because the condition of the majority of our patients has demanded the adoption of the safest procedure possible. We always reserve cholecystostomies for cases in which excision or removal of the gall bladder would be too hazardous or in which the patient's condition warrants only the simplest procedures possible under the circumstances. This is, of course, not meant to be a substitute for cholecystectomy. As much as possible, we try to perform a subsequent cholecystectomy when the condition of the patient makes it permissible.

To recapitulate, in treating gall bladder diseases, we first perform a cholecystectomy. Together with this, we always do a choledochostomy—opening and draining the common bile duct—in order to explore the bile duct thoroughly. The surest way to do this is to make an incision in the anterior wall of the supraduodenal portion of the common bile duct and thoroughly to explore the bile ducts from within with scoops, with probes, or even with the fingers. After this, a tube is inserted towards the liver to provide external biliary drainage.

In our operated cases we have found gall stones in 78% of the cases, noncalculous cholecystitis in 26%, and ascaris in 18%. There were three (7%) cases of perforation of the gall bladder. Two (5%) cases were doubtful; but, because of the history of recurring colics, they were labelled as biliary dyskinesia or dysynergia.

A few words should be devoted to the finding of ascaris in the biliary tract. The ascaris, like its distant cousin the schistosoma, has been known to be one of the most erratic creatures inhabiting the human host. It has been reported in places distant from its native habitat. There are reports of its being found in the common bile duct. In our study of the gall bladder disease, we have found an unusual frequency of these visitors in the common bile duct.

This naturally raises the question: Does the presence of the ascaris in the common bile duct necessarily signify a pathologic condition of the biliary tract, or is the ascaris found in the common bile duct simply because it had the urge to get into a "tight hole?" We know that the gateway to the biliary tract is protected by a powerful sphincter—the sphincter of Oddi. We hesitate to imagine what would happen to the poor ascaris should it ever be caught in the grips of the sphincter of Oddi. Naturally, we cannot help thinking that the only time that the ascaris, with the little intelligence that nature has given him, will ever endeavor to get into the common bile duct is only when the sphincter has ceased to be a

[1] On March 1, 1946, we reoperated one of the first patients in this series, a female patient upon whom a cholecystoduodenostomy was done by Dr. Franco after removal of stones in the common bile duct. The gall bladder was distended and still joined to the duodenum. The gall bladder was opened and not even a pin-point anastomotic opening with the duodenum existed. Another stone was found in the c.b.d.

menace. Such a thing is possible when there is a pathologic disturbance in the biliary system; i.e. the opportune moment for the ascaris to invade the biliary system is when the ravages of an inflammatory condition has made the sphincter of Oddi powerless to prevent its intrusion.

This belief has been substantiated by our finding in this series. We have found that in all the cases where an ascaris has been surprised lurking in the common bile duct, there has always been an accompanying pathologic process in the biliary tract ranging from advanced cholecystitis, multiple calculous formations, to slight inflammatory conditions of the gall bladder—where the organ has lost its normal bluish-green translucency. We have also further observed that these cases give histories of recurring biliary colics for durations beyond the usual expected life span of the ascaris.

There has been a wide diversity of opinion regarding the opportune time for operation in the gall bladder disease—unlike in acute appendicitis. Many well-known surgeons advise immediate operation for acute cholecystitis. On the other hand, there are those who advocate delayed treatment. The proponents of immediate operation regard cholecystitis in the same light as acute appendicitis—where the danger of a perforation and peritonitis is ever-present. The advocates of delayed operation contend that such complications in the gall bladder disease is rare, and that after an expectant treatment, the patient becomes a better risk.

We feel that no hard and fast rule can be laid down. In some cases, an early operation is advisable; while in others, delay is clearly indicated. Each case should be considered as an individual problem. When a case comes with an unduly large tense gall bladder palpable, suggesting that perforation is imminent, we lose no time in performing an operation. In our series, we have come across three perforations and in one, we had reason to believe that the perforation had occurred because there had been a delay of one day in the operation. Inasmuch as most of our cases come after 48 hours from onset—in other words, it is already a little late for an early operation—we feel that the proper attitude would be towards a delayed operation, in order to be able to prepare the patient and make him safe for surgery. We have, in some cases, been forced to abandon this attitude, however, because we can almost anticipate that such patients will refuse to submit to an operation the moment there is a relief from his symptoms.

In our series of operated cases, we had three mortalities. All these cases were cholecystostomies and were very poor surgical risks from the time of admission.

SUMMARY

An analysis of 78 patients with gall bladder disease admitted to the surgical department of the North General Hospital was made. These cases were divided into the non-operated cases (40) and the operated cases (38). There was an incidence of twice as many female as male patients.

Cholecystectomy and choledochostomy was the procedure of choice in this series. Unless there were signs of imminent perforation and therefore resultant peritonitis, the patients were usually subjected to a preoperative regimen designed to build them up.

A high incidence of ascaris in the common bile duct was noted. It is believed that the presence of ascaris in the common bile duct signifies a preceding pathology in the biliary system.

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TABLE 1.—*Incidence of Gall Bladder Disease*

Age	Operated Cases (38)				Non-Operated Cases (40)			
	Male (13)		Female (25)				Female (27)	
	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent
10-20	1	2.63					4	10
21-30	3	7.90	6	15.79	5	12.5	9	22.5
31-40	5	13.60	7	18.41	5	12.5	9	22.5
41-50	1	2.63	7	18.41	1	2.5	2	5.0
Above 50	3	7.90	5	13.60	2	5.0	3	7.5

TABLE 2.—*History of Previous Attacks*
(Gall bladder Series N. G. H.)

Occurrence of Previous Attack	Operated Cases			Non-Operated Cases		
	Male	Female	Total	Male	Female	Total
None	1	2	3	8	12	20
½-1 Year	2	8	10	2	6	8
1-2 Years	3	2	5		3	3
2-5 Years		1	1		2	2
5-10 Years	1	3	4	1	2	3
Indefinite	6	9	15	1	3	4

TABLE 3.—*Duration of Attack From Onset to Admission*
(Gall Bladder Series N. G. H.)

Days	Operated Cases (1)			Non-Operated Cases (2)		
	Male	Female	Total	Male	Female	Total
1	1	2	3	7	8	15
2	1		1	2	1	3
3	1	3	4	1	10	11
4	2	8	10		2	2
5	4	4	8			
6					3	3
7	3	4	7			
8-14	1	1	2	1	2	3
15-21		2	2		1	1
Over 21		1	1	2		2

(1) Average duration 4.5 days (2) Average duration 2.5 days

TABLE 4.—*Localization of Pain*
(Gall Bladder Series N. G. H.)

Localization of Pain	Operated Cases		Non Operated Cases	
	No. of Cases	Per Cent	No. of Cases	Per Cent
Right Hypochondrium	19	50.00	27	67.5
Epigastrium	15	39.47	11	27.5
Right Hypochondrium and Epigastrium	3	7.90	1	2.5
Generalized	1	2.63	1	2.5

TABLE 5.—*Signs and Symptoms*
(Gall Bladder Series N. G. H.)

Signs and Symptoms	Operated Cases		Non-Operated Cases	
	No. of Cases	Per Cent	No. of Cases	Per Cent
Chills and Fever	21	55.26	5	12.5
Nausea and Vomitting	13	34.47	6	15.0
Jaundice	8	21.05	1	2.5
Ascaris	4	10.53	4	10.0
Tenderness	35	92.11	32	80.0
Radiation of Pain	26	68.42	4	10.0
Palpable Gall Bladder	18	47.32	4	10.0

TABLE 6.—*White Blood Cell Examination*
(Gall Bladder Series)

White Blood Cells	Operated Cases		Non-Operated Cases	
	No. of Cases	Per Cent	No. of Cases	Per Cent
6-10 Thousand	12	39.47	22	55
10-15 Thousand	19	50.00	13	32.5
15-20 Thousand	3	7.9	5	12.5
Above 20	1	2.63		

TABLE 7.—*Temperature on Admission*
(Gall Bladder Series N. G. H.)

Temperature	Operated Cases			Non-Operated Cases		
	Male	Female	Total	Male	Female	Total
37-37.5	4	7	11	7	13	20
37.5-38	4	6	10	2	6	8
38.5-39	5	12	17	1	3	4
39.5-40	-			3	5	8

TABLE 8.—*Operations Performed (44)*
(Gall Bladder Series N. G. H.)

OPERATIONS	No. of Cases	Per Cent
CHOLECYSTECTOMY	10	22.73
CHOLECYSTOSTOMY	12	27.27
LITHOTOMY	8	18.18
CHOLECYSTO-DUODENOSTOMY	11	25.00
REMOVAL OF ASCARIS	3	6.82

TABLE 9.—*Operative Findings*
(Gall Bladder Series N. G. H.)

OPERATIVE FINDINGS	No. of Cases	Per Cent
Stones		
Gall Bladder	10	26.32
Common Bile Duct	11	28.95
Combined	2	5.26
ASCARIS	7	18.42
NON-CALCULOUS CHOLECYSTITIS	10	26.32
PERFORATION	3	7.9
BILIARY DYSKINESIA	2	5. 26

TABLE 10.—*Comparative Mortality*
(Gall Bladder Series N. G. H.)

AUTHORS	Cholecystostomy	Cholecystectomy	Total
Blomberg and Zisserman	9.1%	20%	29.1%
Judd-Philiphs	48%	7%	55%
Guzman-Morales	6%	0%	6%

INDEX OF PHILIPPINE LITERATURE ON OPHTHALMOLOGY AND OTORHINOLARYNGOLOGY FROM 1892 TO 1941 (ARRANGED CHRONOLOGICALLY)[1]

ANTONIO S. FERNANDO, M. D.
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[1] N. B.—Because of the destruction of the libraries in Manila during the Battle of Liberation, the writer could not check this index and should there be some errors or omissions, it is hoped that the readers will pardon him. As much as possible only those articles published or specifically mentioned in society transactions as read were included.

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OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL CONFERENCES

By Antonio S. Fernando, M.D. and Geminiano de Ocampo, M.D.
Subjects and date of publication.

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Tuberculous, Left, with Osteomyelitis, Temporal Bone and Mandibular Process and Facial Paralysis.
Jl. P.I.M.A., 17: 183, *March*, 1937, *No.* 3

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Jl. P.I.M.A., 17: 315, *May*, 1937, *No.* 5.

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Jl. P.I.M.A., 17: 431, *July*, 1937, *No.* 7.

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Jl. P.I.M.A., 17: 493, *August*, 1937, *No.* 8.

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Jl. P.I.M.A., 17: 781, *December*, 1937, *No.* 12.

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Editorial

THE PHILIPPINE MEDICAL ASSOCIATION AND THE NEWLY ORGANIZED MEDICAL SOCIETIES

The Philippine Medical Association is a pre-war Association. As a matter of fact it was founded in 1903. Before the outbreak of the war it created a special committee composed of well-known members of the profession with the object of studying the creation of sections on Eye, Ear, Nose and Throat; on Obstetrics and Gynecology; and on Surgery.

World War No. II put a stop to the work of the committee. The proposed sections did not materialize. The committee had to be disbanded, but the spirit behind its creation lived and persisted throughout the war. And after liberation, several medi-

cal societies on different specialties were organized or had resumed activities.

Only one of these societies has affiliated itself with the Philippine Medical Association; namely, the Philippine Ophthalmological and Otolaryngological Society. The others like the Manila Roentgen Society and the Philippine College of Surgeons have not done so.

The organization of these various medical societies is a healthy movement towards the promotion of science and arts in the various field of medicine. The Philippine Medical Association would welcome their federation into one strong and solid mother organization. These different medical societies will undoubtedly hasten the rehabilitation of the medical profession. Each society, in its particular field can cater to the needs of its members.

I think the main purpose of the organization of these medical societies is to elevate the standards of the medical profession and of medical practice, and to disseminate medical knowledge among a greater number of physicians. This objective can better be realized, if these newly organized medical societies would affiliate themselves with the Philippine Medical Association as sections on different specialties. The Philippine Medical Association should be looked upon as a mother association to centralize the activities of all medical societies, thus promoting cooperation and harmony among different medical societies.

If and when all these medical societies become affiliated with the Philippine Medical Association — each society being a component part of the mother association, the Philippine Medical Association — the members of each society would feel at home in attending the scientific meetings of every other society. It must be remembered that the general practitioner, or any other group of physicians, may be interested in certain surgical, obstetrical, or other cases. Without one mother association to bring these newly organized medical societies together, the benefits of one society may not be taken advantage of by members of other societies; and vice versa.

As there are still very few physicians specializing in different lines, we cannot afford to establish societies independent of the Philippine Medical Association, without at the same time detracting from the importance of our national organization. To have one strong and united national organization is very desirable if we in the profession of medicine are to make ourselves heard in any government project affecting our profession, or in the formulation of policies regarding our profession and other medical matters. Therefore the Philippine Medical Association would welcome the affiliation of these newly organized societies for the benefit of all concerned.—V. de D.

Miscellaneous

ABSTRACTS FROM CURRENT LITERATURE

ABSTRACTORS:
Isabelo Concepcion, M. D.
Walfrido de Leon, M. D.
Felisa Nicolas-Fernando, M. D.
Carmelo Reyes, M. D.

The Value of Protein and its Chemical Components (amino acids) in Surgical Repair, by Co Tui, Bulletin of the New York Academy of Medicine 21:631 - 655 (Dec.) 1945.

The author summarized the findings of the team working on this research project as follows:

1. Proteins are essential to wound healing, to the maintenance of tissue integrity; and from present indications, to expeditious convalescence. Protein deficiency endangers all three.

2. A patient can become protein-deficient as a result of inadequate intake and increased nitrogen output or a combination of these two factors.

3. Decreased intake may be due to the patient or neglect on the part of the attending staff.

4. Increased output may be due to increased metabolic loss and to the loss through exudates.

5. The ceiling of nitrogen intake in natural food is naturally low and consequently natural food is often inadequate to replenish the increased protein loss in disease and injury.

6. Studies on cases of burns, of postoperative gastrectomy, cholecystectomy and herniotomy, suggest that convalescence can be shortened, strength and weight conserved by full caloric and nitrogen replacement immediately postoperatively; and that there may be a critical range of nitrogen intake for each disease category.

7. The protein hydrolysates, by raising the ceiling level of nitrogen-intake, are indispensable in many disease conditions and can be used with greater elasticity than natural food.

8. In the course of study on the value of protein hydrolysates, a new treatment of peptic ulcers has been evolved.

All cases of peptic ulcers were treated with protein hydrolysates and high caloric diet. All were treated with from .5 to .6 grams nitrogen per KBW (kilogram body weight), in the form of amigen, and enough dextri-maltose to make up to 40 C per KBW. The mixture is suspended in water, divided into eight to nine feedings, and given at two-hourly intervals. Feedings were continued two or three weeks exclusively, depending upon the clinical response and X-ray findings. No antacids or antispasmodics were given, and wherever necessary, amphojel in 4 cc. doses was given twice a day to control diarrhea. A full complement of vitamins was added to the feeding. The results may be summarized as follows:

Pain and distress stopped in twenty-four to forty-eight hours. Vomiting stopped forty-eight hours after institution of the feedings; rapid roentgenologic healing; positive nitrogen balance, averaging 10 to 16 grams (first ten days); gain in weight, 1 to 8 Kilos in two to three weeks; and rapid gain in strength and morale. However, the treatment does not prevent recurrence on resumption of old dietary habits.

9. Protein hydrolysates for clinical use must fulfill a number of criteria and clinicians using them must be quantitatively-minded.

10. A discussion on the assay of the nutritional course of convalescent patients has been attempted.—F. N. F.

The Surgical Treatment of Essential Hypertension, by Max M. Peet and Emil M. Isberg, M.D., J.A.M.A. 130:467 (Feb. 23) 1946.

Since the first bilateral supradiaphragmatic splanchnicectomy and lower dorsal sympathetic ganglionectomy was performed by one of the authors (M. M. P.) in November 1933, more than 1,500 patients have received this surgical treatment for essential hypertension at the University Hospital, Michigan, as of September 1945. Surgical treatment has long been accepted here as an integral part of the therapeutic armamentarium for hypertensive disease. Its acceptance has been tempered with the realization that its limitations are definite, that its failures are not uncommon. Rarely does surgical treatment result in a cure of hypertensive disease, but it has given remarkable benefit and prolonged the life of many hypertensive patients.

The authors gave the following summary of their experience:

Four hundred and thirty-seven patients with essential hypertension have been followed since receiving the surgical treatment of bilateral supradiaphragmatic splanchnicectomy and lower dorsal ganglionectomy five to twelve years ago.

Two hundred and fifty-one patients, or 57.5 per cent of the series, are living five to eleven years after operation. At the end of five postoperative years 64.8 per cent of the entire series was alive. The hypertensive state in 82 per cent of the patients in this series had already progressed to serious organic disease prior to operation.

Fifty-six per cent of all the males have died, while the female mortality was only 30 per cent.

Ninety-five per cent of hypertensive patients who showed no preoperative evidence of cardiac, cerebrovascular or renal involvement are living five to eleven years after operation.

Approximately one-third of all patients who manifested preoperative evidence of organic heart disease, cerebrovascular disease or impaired kidney function did not survive five to eleven years.

Nineteen per cent of 112 patients with preoperative malignant hypertension are living five to eleven years later.

Fifty-one patients have maintained normal blood pressure levels and 28 show no evidence whatever of hypertensive disease five to eleven years after operation.

Significant reductions in blood pressure, complete and definite symptomatic relief, improvement of eyegrounds and improvement of abnormal electrocardiograms, cardiac enlargement and kidney concentration ability have been maintained for five to eleven postoperative years in a remarkable percentage of patients.

Sixty percent of patients who have previous cerebral accidents suffered no recurrence during the long postoperative period.

Surgical treatment is a measure to be considered in the management of every case of essential hypertension, but to be utilized only when indicated. Evidence of progression and activity of hypertensive disease constitutes indication for surgical treatment.—F. N. F.

Penicillin Therapy in Abdominal Surgery: Result of Prophylactic and Therapeutic Use in 50 Cases. By G. F. Wollgast.—Surgery, Gynecology and Obstetrics, 81:593-710 (Dec.) 1945.

Wollgast observed 5 patients who were critically ill with generalized peritonitis. Treatment with penicillin was started late, and all of these patients died. Later 12 additional patients with peritonitis were treated more adequately. Seven received local treatment and parenteral therapy; 5 received only parenteral therapy. Local administration of penicillin in 3 instances consisted in instilling 50,000 units and in 4 other cases 100,000 units of penicillin into the abdominal cavity just prior to closure. Parenteral therapy was administered intramuscularly at three-hour intervals. Dosage was varied from 10,000 units to 20,000 units every three hours, totalling 80,000 to 160,000 units every twenty-four hours. These patients also received all other accepted therapeutic measures. The postoperative course in 10 of these cases was unexpectedly mild. Twenty-five patients with infected wounds received penicillin both preoperatively and postoperatively as a prophylactic measure. Treatment consisted in parenteral

administration of 10,000 of 15,000 units every three hours intramuscularly for an average of three or four days preoperatively. Treatment was continued postoperatively until the patient had been afebrile for at least three or four days. The average duration of treatment was eleven days. A man aged 31 was operated on in another hospital for perforated gangrenous appendix. On the ninth day a small incisional abscess was drained. On the eighteenth day his temperature rose to 103 F., with the onset of a septic course with daily elevations between 104 and 105 F. Improvement had not been noted over a seven-week period prior to the use of penicillin, but definite improvement followed soon after initiation of this therapy. This serious postoperative complication was cured by penicillin therapy. Seven patients whose wounds became acutely infected following surgery were treated with penicillin. Six of these wounds responded remarkably to treatment.—F. N. F.

Folic Acid Therapy, Editorial, J. A. M. A. 130:496, 1946. In 1935 Day and his associates of the University of Arkansas reported that young rhesus monkeys given a diet consisting of casein, polished rice, whole wheat, salt mixture, cod liver oil and ascorbic acid develop a syndrome characterized by anemia, leukopenia, loss of weight, diarrhea and ulceration of the gums. If the daily deficiency diet is supplemented with either 10 Gm. of dried brewers' yeast or 2 Gm. of liver extract, however, normal body growth and a normal blood picture are maintained over a long period. From these experiments it seemed that yeast and liver extract contain an unknown substance essential to the nutrition of monkeys. For this unknown nutrition factor the Arkansas clinicians proposed the term Vitamin M. It was afterward found that this Lactobacillus casei factor is curative of Vitamin M deficiency in monkeys and necessary for normal growth and hemoglobin formation in chicks. A crystalline folic acid apparently identical with this Lactobacillus casei factor was recently synthesized by Angier and his associates of the Lederle Laboratories and made available for clinical trial.

The similarities of the sprue syndrome in man to the manifestation of Vitamin M deficiency in monkeys led Darby and his associates of Vanderbilt University to test the therapeutic value of synthetic folic acid in 3 human cases,

These patients were given daily intramuscular injections of 15 mg. of synthetic folic acid as the sole therapeutic agent. By the fourth day symptoms of glossitis had disappeared.

The striking and rapid response of these patients led Darby to confirm the belief that synthetic folic acid is identical with, or closely allied to, the substance in liver extract which is effective in the treatment of sprue.

Spies and his associates found that synthetic folic acid given either parenterally or orally is effective in producing significant hemopoietic response in persons with nutritional macrocytic anemia and with macrocytic anemia of pellagra, pernicious anemia, sprue or pregnancy. —F. N. F.

SOCIETY ACTIVITIES

Culion Medical Society:—

The 20th Annual Meeting of the Society was held on January 27, 1946. The scientific program was as follows:

1. Presidential Address, by Dr. J. O. Tiong.
   (He emphasized the necessity of the segregation of lepers, advocated sterilization as a measure to prevent lepers from having children, and urged against sex-seclusion.)
2. *Is There Blood Calcium Deficiency in Leprosy?* By Dr. E. Paras
3. *Potency of Stored Lepromin*, by Dr. Jose O. Nolasco
4. *Observations Bearing on the Incidence of Leprosy in Children of Lepers*, by Dr. C. B. Lara.

Manila Medical Society:—

The society held its monthly meeting on March 30, 1946 in the Sto. Tomas University gymnasium, under the auspices of the Department of Medicine of this institution. The following papers were read:

The Operative Management of Hypertrophy of the Prostate in Patients with Coronary Disease, by Domingo Antonio. Jr., Major MC, F.A.C.S.; Asst. Prof., Dept. of Surgery and Urology, U.S.T.; Faculty of Medicine and Surgery.

Recent Advances in the Diagnosis and Treatment of Cardiovascular Diseases, by A. Benedict Schneider, Jr., Major MC, U.S.A., Western Reserve University, Cleveland, Ohio

Tarlac Medical Society:—

The Tarlac Medical Society was organized on the 19th of January by 36 physicians. The following officers were elected:

President: Dr. Gonzalo Santos
Vice-Pres.: Dr. Alfonzo C. Concepcion
Sec.-Treas.: Dr. Jose G. Cruz
Board of Censor Delegates:
Dr. Marcos J. Corpus
Dr. Arsenio C. Regala
Dr. Marcelo S. Agana

The inaugural meeting was held on March 1, 1946. After the opening remarks of Dr. Jose G. Cruz, the invocation was said by Rev. Fr. Benedicto J. E. Arroyo. The officers were installed by Dr. Victorino de Dios, President, Philippine Medical Association, after which Dr. Gonzalo Santos gave the Presidential address. The guest of Honor was Dr. Jose C. Locsin, Secretary of Health and Public Welfare. A reception and ball climaxed the occasion.

Philippine Ophthalmological and Otolaryngological Society:—

The first scientific meeting of the Philippine Ophthalmological and Otolaryngological Society was held on March 30, 1946 under the auspices of the E. E. N. T. Staff of the North General Hospital, composed of Dr. Felisa Nicolas-Fernando, Chief; Dr. Jesus Tamesis, resident; Dr. Caridad Estanislao, extern; and Dr. A. S. Fernando, visiting physician.

A.—SCIENTIFIC MEETING:

1.—Relative frequency of Eye, Ear, Nose and Throat cases in the out-patient service of the North General Hospital.

2.—Presentation of cases:

a.—A case of advanced glioma of the optic nerve (left)

b.—A case of mastoiditis with facial paralysis (The facial paralysis recovered after operation on the mastoid and the use of penicillin parenterally and topically and intramuscular injection of thiamine hydrochloride.)

c.—A case of homonymous hemianopsia, traumatic

d.—A case of cerebellar abscess of otitic origin with necropsy findings

e.— A case of total ophthalmoplegia, bilateral, probably syphilitic.

f.—A case of a child on which a radical mastoid operation was performed and thyrothricin was used for topical application after operation. Satisfactory results.

g.—A case of an old woman, operated on for simple cataract extraction (extra capsular) with irrigation; the base of the iris prolapsed slightly 2 days after the operation. This was successfully replaced. Vision was 20/20 and Jaeger No. 1 with the correcting glasses after several weeks.

B.—BUSINESS MEETING:

The Philippine College of Surgeons held its scientific meeting on March 30, 1946 in the Philippine General Hospital.

The scientific program follows:

Present Status of Surgery in the Philippines, by Dr. Januario Estrada.

Cesarean Section, by Dr. Alfredo Baens.

Surgery of the Autonomic Nervous System, by Dr. Gervasio Cuyugan.

The President is Dr. Nicanor Jacinto and the Secretary-Treasurer is Dr. Carmelo Reyes.

NEWS ITEMS

(Physicians are requested to send in news items of interest to the profession.)

LOCAL:

A critical shortage of medical, dental, and hospital facilities in the Philippines is leaving a large part of the population an easy victim to disease and malnutrition according to the results of a survey by Charles H. Forster, Executive Director of the Philippine War Relief, Inc. The survey revealed that approximately 11,500,000 Filipinos, representing nearly 2/3 of the total population, are without medical, dental, or hospital aid of any kind. In rural and outlying areas populated by almost 12 million Filipinos, there are barely enough medical facilities to provide emergency aid for 500,000. Philippine War Relief is at present engaged in a health and rehabilitation program designed to bring immediate aid to these rural areas. An important part of this agency's health program are mobile dispensaries.

Brig. Gen. Howard F. Smith, Chief Public Health Adviser to the U. S. High Commissioner, announced recently (Mar. 21) that the U. S. Health Service will soon organize additional malaria units to expand its malaria control activities in the Philippines. Those interested may communicate with Lt.-Col. Francisco J. Dy, Customs Building, Port Area.

The North General Hospital celebrated its first anniversary on February 28, with scientific and literary-musical programs. In the scientific program, Lt. Col. Maurice H. Schnitker, Medical Consultant of AFWESPAC, was the guest speaker. He spoke on *Hypertension and Hypertensive Heart Disease*. Drs. Agustin Liboro and Wenceslao Vitug led the discussions. Secretary Jose C. Locsin of the Department of Health and Public Welfare was the guest of honor at the literary musical program.

Thirty-three government hospitals in Manila and the provinces have been reopened to the general public since the liberation of the Philippines last year, Dr. Jose C. Locsin, Secretary of Health and Public Welfare, declared recently. This was made possible by the laboratory equipment and medical supplies, amounting to several million pesos donated by the United States Army to the Commonwealth Government, Locsin said. On its part, the administration set aside ₱3,779,000 for the repair and reconstruction of hospitals. It has also opened charity clinics, maternity houses, and puericulture centers in the towns. Military hospitals have likewise been operating since the liberation of the country; and, in addition to caring for the sick and wounded officers and soldiers, they have also rendered medical treatment to civilians in places where there are no government hospitals.

Santo Tomas University Hospital on Calle España was inaugurated and blessed March 7, 1946 by Mons. Guillermo Piani, Apostolic Delegate, with Mrs. Esperanza Osmeña as sponsor. The Hospital is located in the modern three-story concrete structure on the University campus. It is equipped with 60 private rooms, spacious general wards, two separate surgical operating rooms, and an up-to-date delivery room and nursery.

Mr. C. Leo Wilheim, Manager of the Philippine Red Cross, has plans for extensive expansion of activities.

According to a recent statement of Secretary Jose C. Locsin of the Department of Health and Public Welfare, there is a dearth of physicians in the Visayas, especially Antique, where only two physicians are trying to serve a population of 240,000; and people depend mostly on herbolarios and quacks for medical assistance.

On February 19, Dr. Co Tui, Associate Professor of Experimental Surgery of New York University, came to Manila by plane on his way to China on official mission. During his brief stay in the city, he was feted by his Chinese and Filipino friends, among whom were Mrs. Socorro S. Diaz, Drs. Baldomero Roxas, Jose Albert, Alfredo Baens, Vicente R. Ocampo, and Jose Santillan.

Dr. Miguel Cañizares, Managing Director of the Philippine Tuberculosis Society, stressed recently, in an appeal to the press, radio and other information services, the necessity of mass education in connection with the campaign against tuberculosis. He said that phthisis causes one out of seven deaths in the country and that it is easily curable if detected early.

FOREIGN:

Dr. Thomas Parran, Surgeon General of the U. S. Public Health Service, conferred with President Truman recently on plans for the establishment of an international organization for world-wide control of communicable diseases. The body will function under the United Nations Organization. Dr. Parran will leave for Paris to assist a preparatory committee in planning for a conference in June to set up the health organization, which will coordinate the activities of the International Office of Public Health, the League of Nations Health Organization, and the UNRRA'S present health activities.

According to a United Press dispatch, Dr. A. B. M. Sison emphasized, in his address on February 8 at a meeting of the National Commission on Children in Wartime under the auspices of the Department of Labor, the urgent need of aid to Filipino children. Dr. Sison is in the U. S. on a Rockefeller Foundation grant to continue his studies in nutrition and to obtain equipment and supplies for the rehabilitation of the Medical Center in the Philippines.

In the February 1946 issue of THE JOURNAL of the Kansas Medical Society, the following appeared:

NEED FOR MEDICAL BOOKS IN MANILA

Some months ago the Academy-International of Medicine and Dentistry moved its executive office from St. Paul, Minnesota, to the Liberty Building, Topeka, because of its central location. According to the executive secretary, Mr. J. B. Young, one of the projects of this organization is to attempt to supply the destroyed medical library at the University of Manila with sufficient books to enable the school to operate. It is well known that the Japanese destroyed the University and its library until almost no piece or usable equipment remained.

The Academy-International of Medicine and Dentistry is appealing to the medical profession all over Canada and the United States to donate books that may be sent to Manila. Already 10,903 individual publications are in transit and many more are needed. These books have come from individuals, from medical libraries, medical schools and clinics.

Kansas doctors are invited to assist in this worthwhile undertaking through the contribution of books, periodicals or cash. Doctors willing to donate books should first write to the Academy-International of Medicine, Liberty Building, Topeka, giving the names and authors and edition numbers of the books that are available. In an effort to send only material that is critically needed and to avoid duplication, all gifts should be cleared before they are sent. The donor will then be instructed which of these books are desired. If cash is given, the donor may be assured that all money will be used for the purchase of needed texts, that arrangements have been made with leading publishers to sell books for this purpose at cost, and that they will be forwarded immediately to the School of Medicine, University of the Philippines. Make all checks payable to: Manila Library Fund, A-1, M.

Among the important articles read at the 42nd Annual Congress on Medical Education and Licensure held February 10-12, 1946, in Chicago were the following:

The Development and Protection of High Educational Standards by the Medical Profession, by Ray Lyman Wilbur, M. D.; Medical Progress During the War, by Perrin H. Long, M. D.; Medical Education in Europe, by Wilburt C. Davidson, M. D.; Problems in Specialty Practice, by Paul Titus, M. D.; Research in Undergraduate Medical Education, by Detler W. Bronk, Ph. D.; The People's Health—A National Asset, by Hon. Watson Miller Agency; Why Require Graduates or Approved Medical Schools to Pass a Basic Science Board Examination, by H. M. Platter, M. D.

U. S. DOCTORS BUCK TRUMAN'S HEALTH PROGRAM —American doctors, like their British Colleagues, are refusing to be nationalized. Their arguments are very similar to those expounded by the British Medical Association. The current issue of the Journal of the American Medical Association has denounced President Truman's health medical program as an "attempt to enslave medicine as first among the professions, industries or trades to be socialized." The Journal put forth a strong plea for Congress to defeat a bill which would carry out the President's recommendations. The measure, the Journal states, would "destroy the private practice of medicine," "place the medical profession under the direction of one man," "enforce the hospitalization provisions to destroy the voluntary hospital system," and "require people to take any doctor the surgeon general tells them to."

IMPORTANT NOTICE

Original articles are accepted for publication only with the understanding that they have not been and are not to be published elsewhere. The Editorial Board reserves the right to accept, reduce, or reject all manuscripts submitted for publication.

Twenty-five reprints are given free to the author and any number of additional copies will be furnished at cost price provided that written request be made at the time the article is submitted for publication.

REFERENCES TO LITERATURE CITED SHOULD INCLUDE: FOR PERIODICALS: NAME OF AUTHOR, TITLE OF ARTICLE, FULL NAME OF PERIODICAL, VOLUME NUMBER, INCLUSIVE PAGING, MONTH AND YEAR OF PUBLICATION, ALL IN THE ABOVE ORDER. EXAMPLE FOLLOWS:

Pio de Roda, Alfredo: Typhus Fever in the Philippines: Weil-Felix Reaction of 500 Cases, Journal Philippine Islands Medical Association, 17:147-156 (March) 1936.

REFERENCES TO BOOKS SHOULD INCLUDE: NAME OF AUTHOR, TITLE OF BOOK, PLACE OF PUBLICATION, NAME OF PUBLISHER, DATE OF PUBLICATION, VOLUME NUMBER, AND PAGES CITED, ALL IN THE ABOVE ORDER. EXAMPLE FOLLOWS:

Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Baltimore, Williams & Wilkins Company, 1932, vol. 2, 892-893.

THESE REFERENCES SHOULD BE LISTED ON A SEPARATE PAGE AT THE END OF THE ARTICLE WITH CORRESPONDING NUMBERS IN THE TEXT. ACCURACY AND COMPLETENESS OF LITERATURE CITATIONS IS REQUESTED.

Any other information regarding publication of figures, graphs, etc. will be furnished by the Editor upon request.

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