vitamin B₁₂. We have termed this lesion of pernicious anaemia and subacute combined degeneration of the cord "gastric atrophy," in contradistinction to superficial gastritis and atrophic gastritis.

Case 5.—A chocolate manufacturer aged 47 was found to be suffering from typical pernicious anaemia with haemoglobin of 7.5 g. which responded to intramuscular "campolon." Stomach biopsy was performed before treatment and after three, six, and fifteen months' treatment. He was then given a four-months course of treatment with 80 µg. of vitamin B₁₂ weekly. The biopsy was repeated. The gastric mucosa was found by Funder to have remained unchanged. Serial test-meal examinations showed histamine-fast achlorhydria.

What is the correlation between the histological findings and the clinical picture? Of 41 patients showing "gastric atrophy" in the gastric mucosa, 39 had pernicious anaemia and/or subacute combined degeneration of the cord. Thus we have two patients who perhaps may be expected to develop pernicious anaemia and/or subacute combined degeneration of the cord during the next few years. On the other hand, of 66 cases with "atrophic gastritis" only five had pernicious anaemia and/or subacute combined degeneration of the cord.

We have found three cases of probable subacute combined degeneration of the cord with a normal peripheral blood picture and bone marrow and with changes in the gastric mucosa identical with those of pernicious anaemia.

Moreover, we have found gastric biopsy to be of value in diagnosing those patients with possible subacute combined degeneration of the cord or pernicious anaemia who unfortunately have had treatment with liver before an adequate blood examination has been performed and whose nervous lesions are not entirely typical.

When does this gross atrophy of the gastric mucosa occur (Doig, Motteram, Weiden, and Wood, 1950; Doig, and Wood, 1950)? We do not know. Perhaps we have a slight clue. We did test meals on 134 apparently healthy medical students and found only one with persistent histamine-fast achlorhydria. This young man, whom we have called subject X, had suffered no dyspepsia. His mother had hypochlorhydria to gruel when aged 44, and his maternal grandmother had died of pernicious anaemia at the age of 70. Gastric biopsy showed considerable atrophy of the chief and parietal cells but with little inflammatory reaction. However, the change was not as pronounced as in pernicious anaemia. We shall watch his future with great interest.

Atrophic gastritis can be produced artificially by applying deep x-ray therapy to the gastric mucosa. This has been shown by Palmer, in America, and by Holman, in Tasmania, to be of benefit in some cases of duodenal ulcer with high gastric acidity. Thus, a textile worker, aged 36, with duodenal ulcer which did not respond to medical treatment had a normal gastric biopsy prior to irradiation. He was then given a three-weeks course of 2,000 r by Kaye-Scott. In our unit, Doig, Funder, and Weiden studied his progress in considerable detail over a period of 15 months and found that the inflammatory reaction and atrophy became pronounced, with the development of achlorhydria and greatly diminished pepsin secretion one month after the course of treatment (Doig, Funder, and Weiden, 1951). The mucosal changes remained thus for one month and then gradually

returned to a normal appearance with rising acid and pepsin secretion. Normal findings were observed after the lapse of one year. This close correlation between the test-meal findings and the appearance of the gastric mucosa is in keeping with our general experience (Wood, Doig, Motteram, Weiden, and Moore, 1949).

Conclusion

I should like to emphasize that in our opinion diffuse lesions of the mucosa of the body of the stomach occur and may produce symptoms. Superficial gastritis with flatulent dyspepsia may heal or progress to atrophic gastritis. Atrophic gastritis causes recurrent flatulent dyspepsia and shows little or no tendency to heal. Severe gastric atrophy without inflammatory changes is associated with pernicious anaemia and/or subacute combined degeneration of the cord, and is probably not the end-stage of chronic gastritis. It may well be a slow atrophic process of different aetiology. Much has to be learned of the aetiology, course, and prevention of superficial gastritis, atrophic gastritis, and gastric atrophy. If we discovered how to prevent gastric atrophy perhaps we could prevent the onset of pernicious anaemia or the more tragic disease, subacute combined degeneration of the cord.

REFERENCES

- Doig, R. K., Funder, J. F., and Weiden, S. (1951). *Med. J. Aust.*, **1**, 828.
 ——— Motteram, R., Robertson, E. G., and Wood, I. J. (1950). *Lancet*, **2**, 836.
 ——— Weiden, S., and Wood, I. J. (1950). *Ibid.*, **1**, 948.
 ——— and Wood, I. J. (1950). *Med. J. Aust.*, **2**, 565.
 Wood, I. J., Doig, R. K., Motteram, R., and Hughes, A. (1949). *Lancet*, **1**, 18.
 ——— Weiden, S., and Moore, A. (1949). *Gastroenterology*, **12**, 949.

VITAMIN B₁₂ IN MEGALOBlastic ANAEMIA OF PREGNANCY AND TROPICAL NUTRITIONAL MACROCYTIC ANAEMIA

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The value of vitamin B₁₂ in Addisonian pernicious anaemia, subacute combined degeneration, nutritional macrocytic anaemia, and tropical macrocytic anaemia has been thoroughly acknowledged (West, 1948; Berk *et al.*, 1948; Ungley, 1948a; Hall and Campbell, 1948a, 1948b; Spies *et al.*, 1948a, 1948b; 1949; Patel, 1948, 1950; Das Gupta, 1950). Reports of its use in megaloblastic ("pernicious") anaemia of pregnancy, however, are contradictory. Ungley (1948b), Bethell *et al.* (1948), Day *et al.* (1949), and Das Gupta (1950) used it without any response, while Patel and Kocher (1950) reported its successful use in five cases of anaemia of pregnancy and the puerperium. Patel (1950) also reported that a higher dosage was required in tropical macrocytic anaemia and in megaloblastic anaemia of pregnancy than in pernicious anaemia. A report of 21 cases of megaloblastic anaemia in women patients admitted under my care at Lady Hardinge Hospital, New Delhi, and treated with vitamin B₁₂, is here presented.

TABLE I.—Haematological Responses to Injection of Vitamin B₁₂

Case No.	Daily Dosage (μg.)	Maximal Retic.	Day of Treatment	Hb (g./100 ml.)	R.B.C. (mil./c.mm.)	P.C.V. %	M.C.V. (μ ³)	M.C.H. (μg.)	M.C.H.C. (%)	Weekly R.B.C. Rise (mil./c.mm.)	Expected R.B.C. Rise (mil./c.mm.) (Della Vida and Dyke)
1	5	15.1%	0	3.4	0.96	10.0	104	35	34.0	0.49	0.73
		14th day	54	12.6	4.82	40.0	83	26	31.5		
2	7	17.5%	0	2.0	0.82	9.0	112.5	25	22.0	0.36	0.75
		15th day	53	10.0	3.50	31.0	88	32	32.0		
3	4	20.2%	0	3.4	1.28	13.0	100	26	26.0	0.90	0.66
		13th day	25	12.3	4.50	32.0	71	27	38.7		
4	4	15.0%	0	5.7	2.15	22.0	102	27	26.0	0.40	0.68
		9th day	15	8.1	2.95	—	—	—	—		
5	4	3.5%	0	3.4	1.03	10.0	99	33	34.0	—	0.72
		4th day	15	4.08	1.20	12.0	100	34	24.0		
6	9	15.0%	0	2.0	0.9	9.0	100	22	2.0	0.51	0.73
		19th day	35	10.0	3.5	30.0	86	28	33.0		
7	5	20.8%	0	2.7	1.07	12.0	112	27	23.0	0.83	0.71
		11th day	22	8.5	3.50	24.0	70	24	35.0		
8	7	11.5%	0	3.4	1.20	13.0	108	28	26.0	0.91	0.67
		7th day	12	5.0	2.60	18.5	71	19	27.0		
9	4	25.0%	0	4.08	1.20	15.0	125	34	27.0	0.77	0.67
		7th day	20	10.0	3.50	30.0	86	28	33.0		
10	4	22.5%	0	3.7	1.02	12.0	117	36	30.0	0.80	0.72
		9th day	30	10.5	4.30	38.0	89	24	27.0		
11	10	15.1%	0	3.70	1.10	12.0	109	34	31.0	1.06	0.70
		9th day	22	10.54	4.30	32.0	74	25	33.0		
12	11	21.5%	0	3.70	1.02	12.0	117	36	30.0	0.93	0.72
		5th day	20	9.18	3.81	29.0	76.5	24	31.0		
13	8	9.1%	0	8.5	2.38	23.0	99	37	37.0	0.76	0.42
		6th day	12	11.2	3.60	33.0	91	31	34.0		
14	10	13.0%	0	3.4	1.28	9.0	70	26	38.0	1.30	0.65
		9th day	12	6.8	2.10	18.0	90	32	38.0		
15	10	11.5%	0	3.4	1.50	15.0	100	23	23.0	0.60	0.60
		7th day	24	10.0	3.78	28.0	80	26	35.0		
16	8	15.1%	0	2.0	0.65	8.5	130.7	30	23.5	0.70	0.80
		10th day	32	10.0	3.90	36.0	92	26	28.0		
17	4	22.5%	0	6.8	2.10	21.0	100	32	32.0	0.77	0.47
		7th day	20	13.26	4.50	40.0	90	30	33.0		
18	5	17.5%	0	6.4	2.00	19.5	97.5	32	32.0	1.25	0.50
		7th day	15	11.28	4.50	35.0	77	25	32.0		
19	2	13.5%	0	6.8	2.00	20.0	100	34	34.0	0.58	0.50
		6th day	30	11.5	4.50	35.0	77	25	32.0		
20	4	15.3%	0	3.4	1.55	15.0	99	22	22.0	0.37	0.61
		9th day	30	10.5	3.10	29.0	96	34	36.0		
21	10	14.1%	0	2.7	1.0	10.0	100	27	27.0	0.75	0.72
		7th day	26	10.8	3.80	33.0	87	28	33.0		

TABLE II.—Plasma-protein Levels in Cases Treated with Vitamin B₁₂

Case/No.	Body Weight (lbs.)	Home Diet. Prot. and Cal. Intake/day	Hosp. Diet. Prot. and Cal. Intake/day	Protein			
				Total (g./100 ml.)	Albumin (g./100 ml.)	Globulin (g./100 ml.)	A/G Ratio
1	—	Prot. 38 g. Cal. 800	Prot. 68.2 g. Cal. 1,600	4.30 5.08	1.90 3.63	2.40 1.45	0.8:1 2.5:1
2	—	Prot. 32 g. Cal. 848	Prot. 68 g. Cal. 1,633	4.39 4.55	2.08 2.85	2.31 1.70	0.9:1 1.6:1
3	93	Prot. 28 g. Cal. 720	Prot. 71 g. Cal. 1,771	6.08 7.00	1.80 3.80	4.28 3.20	1:2.6 1.2:1
4	85	Prot. 32 g. Cal. 848	Prot. 69 g. Cal. 1,664	4.10 —	2.00 —	2.10 —	1:1
5	75	Poor intake due to diarrhoea	Prot. 74 g. Cal. 1,684	6.20 —	1.48 —	4.72 —	1:3
6	—	" "	Prot. 89 g. Cal. 2,284	5.25 6.30	2.80 4.00	2.45 2.30	1.2:1 1.7:1
7	81	" "	Prot. 71 g. Cal. 1,771	3.67 5.20	2.40 2.80	1.27 2.40	1.8:1 1.2:1
8	92	" "	Prot. — Cal. —	3.28 —	1.99 —	1.29 —	1.5:1
9	88	Prot. 36 g. Cal. 784	Prot. 89 g. Cal. 2,176	3.80 5.90	1.30 2.90	2.50 3.00	1:1.7 1:1
10	107	Prot. 35 g. Cal. 840	Prot. 68 g. Cal. 1,839	3.30 6.30	1.30 3.90	2.00 2.40	1:1.5 1.6:1
11	85	Prot. 42 g. Cal. 1,081	Prot. 71 g. Cal. 1,811	4.48 6.10	2.10 4.10	2.38 2.00	1:1.1 2:1
12	107	Prot. — Cal. —	Prot. 68 g. Cal. 1,771	3.30 6.80	1.30 3.40	2.00 3.40	1:1.5 1:1
13	91	Prot. 68 g. Cal. 1,687	Prot. 68 g. Cal. 1,687	8.10 6.80	3.90 5.30	4.20 1.50	1:1.1 3.5:1
14	—	Prot. — Cal. 1,545	Prot. 59 g. Cal. 1,545	4.90 —	2.10 —	2.80 —	1:1.3
15	93	Prot. 63 g. Cal. 1,727	Prot. 63 g. Cal. 1,727	7.00 6.70	2.70 4.00	4.30 2.70	1:1.6 1.5:1
16	—	Prot. 77 g. Cal. 1,152	Prot. 82.5 g. Cal. 1,990	4.70 6.80	2.60 5.30	2.10 1.50	1.2:1 3.5:1
17	—	Prot. — Cal. —	Prot. 78.5 g. Cal. 1,930	3.40 8.40	2.40 5.30	1.00 3.10	2.4:1 1.7:1
18	135	Prot. 37.5 g. Cal. 778	Prot. 83 g. Cal. 1,795	6.41 6.80	4.01 5.10	2.40 1.70	1.6:1 3:1
19	—	Prot. 32 g. Cal. 848	Prot. 59 g. Cal. 1,545	4.20 6.90	1.60 2.50	2.60 4.40	1:1.6 1:1.7
20	—	Prot. 37.5 g. Cal. 778	Prot. 76 g. Cal. 1,871	4.10 5.80	2.60 3.20	1.50 2.60	1.7:1 1.2:1
21	103	Prot. 32 g. Cal. 848	Prot. 63 g. Cal. 1,701	6.70 7.80	2.40 4.00	4.30 3.80	1:1.8 1:1.1

Methods

All the patients were in hospital during the period of treatment. They were given no other treatment than vitamin B₁₂. A history of their illness and details of their diet during the previous six months were recorded. Each case was given a thorough examination. Full blood counts; total and differential blood proteins; urine, stool, and gastric analyses; and a blood Kahn test or Wassermann reaction were carried out. Bone marrow was megaloblastic in all cases. The reticulocyte count was done daily from the time of admission until 15 days after the initiation of treatment. When the blood level had become stationary, treatment with vitamin B₁₂ was given. Most patients came to the hospital in a low condition, and, as they had had no previous therapy, treatment was instituted after the first blood examination. Vitamin B₁₂ was given intramuscularly in 20- μ g. doses either every other day or every fifth day, depending upon the condition of the patient (Tables I and II).

All patients were females and did household duties. Their ages varied between 19 and 45 years—the child-bearing age—15 of them being between 19 and 30. The actual distribution is given in Table III.

TABLE III.—Age Distribution

Age in Years	No. of Cases
10-19	1
20-29	10
30-39	9
40-45	1
Total	21

Five of the 21 patients were admitted in the third trimester of pregnancy. Eleven were treated within a period of three months after delivery—nine of these within 15 days of delivery. Treatment of one of the five cases of pregnancy was continued during the puerperium.

The remaining five patients had a history of frequent attacks of diarrhoea and poor nutrition. One of them had bleeding haemorrhoids. Nine cases had macrocytic hypochromic anaemia and 11 had macrocytic normochromic anaemia—types common in this part of the country. The initial erythrocyte level was below 1,000,000 in four cases, below 1,500,000 in eleven, below 2,000,000 in two, and below 2,500,000 in four. The haematological findings before and after treatment are given in Table IV.

TABLE IV.—Haematological Findings Before and After Treatment

	Haemoglobin (g./100 ml.)		R.B.C. (mil./c.mm.)		C.V. %	
	Before	After	Before	After	Before	After
Mean	4.01	9.82	1.35	3.58	13.1	28.7
Maximum	8.5	13.3	2.38	4.8	23.00	40.0
Minimum	2.0	5.0	0.65	1.2	8.50	12.0

The total proteins, particularly albumin, in all cases were low, and although the values increased with treatment they did not reach a normal level (Table II). This hypoproteinaemia was associated with oedema of the feet in 15 patients. Reticulocytosis occurred in all cases after treatment, and varied between 3.5 and 25%; the maximum reticulocytosis usually occurred between the seventh and tenth days. In some cases it was delayed.

Twenty cases responded adequately to treatment with vitamin B₁₂. One (Case 4) of these was given a mixture containing iron and ammonium citrate—90 gr. (5.9 g.) a day—during the third week of treatment. The response seemed to be better with iron. Megaloblasts were

present in the bone marrow, but the peripheral blood picture was hypochromic. Another patient (Case 5), who had soreness of tongue and marked diarrhoea, gave a poor haematological response to vitamin B₁₂ (60 μ g. in 15 days), though there was a general improvement and the oedema disappeared on the fourth day. The maximum reticulocyte count was 3.5% on the fourth day. Later she responded very rapidly to parenteral folic acid. In most cases the treatment was given throughout the stay in hospital and lasted 12 to 54 days. In some it was given on alternate days or every fourth, seventh, or tenth day, depending upon the severity of the condition. The minimum amount used was 60 μ g. in 15 days, and the maximum 360 μ g. in 53 days.

Roughly, it can be deduced that in mild cases an average of 4 μ g. a day was needed to produce improvement, and in severe cases as much as 10 μ g. a day. It was also found that anaemias associated with pregnancy required higher doses.

Discussion

The place of vitamin B₁₂ in the treatment of macrocytic anaemia in the Tropics is still unsettled (Dameshek, 1949; Minot and Castle, 1950). The suggestion that it could not be effective resulted from an old-standing conception that refined liver extract had little effect. It was taken for granted that vitamin B₁₂ was the most active substance in the refined liver extract. Patel and Bhende (1949), however, showed that refined liver extract gave good results in a large number of cases of tropical macrocytic anaemia. Sometimes only small doses were required. In their report the requirement in tropical macrocytic anaemia was two or three times that in Addisonian pernicious anaemia. It was also shown (Patel, 1948; Patel and Kocher, 1950; Patel, 1950) that vitamin B₁₂ was efficacious in tropical macrocytic anaemia and pregnancy anaemia in doses larger than that required in Addisonian pernicious anaemia. The results of the present investigation also show that vitamin B₁₂ is effective in megaloblastic anaemia of pregnancy and the puerperium as it occurs in India, and in four cases of tropical macrocytic anaemia occurring in women, but that higher doses are required than in pernicious anaemia.

In one woman suffering from tropical macrocytic anaemia the haematological response with vitamin B₁₂ was not satisfactory, even though there was general improvement, with disappearance of oedema. This patient later responded to parenteral folic acid therapy. Such cases have also been noted by Das Gupta *et al.* (1949). Other cases which had initially failed to respond to folic acid subsequently responded to liver extract.

From all available evidence it can be concluded that vitamin B₁₂ is effective in most cases of tropical megaloblastic anaemia and megaloblastic anaemia of pregnancy and the puerperium, but that the dosage required is higher than is needed in Addisonian pernicious anaemia. Our results confirm those reported by Patel and his co-worker.

Summary

Sixteen cases of megaloblastic anaemia of pregnancy and the puerperium were treated successfully with parenteral vitamin B₁₂.

Out of five cases of tropical megaloblastic anaemia treated with vitamin B₁₂, four responded to vitamin B₁₂ and one responded better to subsequent folic acid therapy.

Four micrograms of vitamin B₁₂ a day was the estimated requirement in mild cases of macrocytic anaemia of pregnancy and 10 µg. in severe cases.

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REFERENCES

- Berk, L., Denny Brown, D., Finland, M., and Castle, W. B. (1948). *New Engl. J. Med.*, **239**, 328.
 Bethell, F. H., Meyers, M. C., and Neligh, R. B. (1948). *J. Lab. clin. Med.*, **33**, 1477.
 Dameshek, W. (1949). *Blood*, **4**, 76.
 Das Gupta, C. R. (1950). Remarks in discussion at International Congress of Haematology, Cambridge.
 — Chatterjee, J. B., and Mathen, K. K. (1949). *Indian J. med. Res.*, **37**, 455.
 Day, L. A., Hall, B. E., and Pease, G. L. (1949). *Proc. Mayo Clin.*, **24**, 149.
 Hall, B. E., and Campbell, D. C. (1948a). *Ibid.*, **23**, 584.
 — (1948b). *Ibid.*, **23**, 591.
 Minot, G. R., and Castle, W. B. (1950). *The 1949 Year Book of Medicine*, p. 375. Year Book Publishers, Chicago.
 Patel, J. C. (1948). *British Medical Journal*, **2**, 934.
 — (1950). "Vitamin B₁₂ in Tropical Macrocytic Anaemia." Paper read at International Congress of Haematology, Cambridge. (To be published.)
 — and Bhende, Y. M. (1949). *Blood*, **4**, 259.
 — and Kocher, B. R. (1950). *British Medical Journal*, **1**, 924.
 Spies, T. D., Lopez, G. G., Milanes, F., Toca, R. L., and Silver, B. (1948a). *Sth. med. J.*, **41**, 523.
 — Stone, R. E., and Armaburu, T. (1948b). *Ibid.*, **41**, 522.
 — Suarez, R. M., Lopez, G. G., Milanes, F., Stone, R. E., Toca, R. L., Armaburu, T., and Kartus, S. (1949). *J. Amer. med. Ass.*, **139**, 521.
 Ungley, C. C. (1948a). *Lancet*, **1**, 771.
 — (1948b). *British Medical Journal*, **2**, 154.
 West, R. (1948). *Science*, **107**, 398.

AN OUTBREAK OF RESPIRATORY TUBERCULOSIS IN A SCHOOL

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At a time when medical and lay opinion is being focused on the problem of tuberculosis it is opportune to relate the story of a local outbreak. Criticism is often directed at the difficulties associated with the threefold control of our Health Service. It is suggested that there should be a greater integration of the hospital, general practitioner, and preventive medicine services. Tuberculosis is frequently cited as an example of a disease which could be combated more effectively if a closer liaison existed between the various services. The localized outbreak to be described is therefore not only of clinical but also of medical administrative interest.

The first intimation of this outbreak followed an observation by the paediatrician who was investigating three cases of clinical primary respiratory tuberculosis in hospital and, in addition, had made domiciliary visits to two similarly affected children. Noting that they all came from the same area, he informed the County

Medical Officer on July 15, 1950. It was quickly confirmed that all the affected children attended the same school, and the school became the centre of investigations. Inquiries revealed that a member of the teaching staff had been absent from May 23 to June 26 suffering from laryngitis, but on July 17, when the school was visited by the Deputy County Medical Officer, this teacher was again absent, this time with "haemoptysis," which had occurred on the previous Friday (July 14). In due course the diagnosis of respiratory tuberculosis was confirmed.

As a routine the milk supplies at the homes of the affected children were investigated. There was no common supply. The school milk supply was pasteurized and there was no record of inefficient heat treatment.

Future action was decided at a meeting of the paediatrician, the local chest physician, and the County and Deputy County Medical Officers. A letter was sent to the parents of all the children at the school asking their consent to carry out Mantoux skin tests on the children. In addition, the health visiting staff of the local health authority called at the homes of all the children who were absent from school, and arrangements were made for them to be investigated. It was then possible to test immediately 176 of the 186 children on the school register. This excellent co-operation of the parents reflects their keen interest in the medical welfare of their children. Those found to be Mantoux-positive were x-rayed, using large films. The results of the Mantoux tests and x-ray examination are set out in Table I. Mantoux testing consisted in the intradermal injection of 0.1 ml. 1/10,000 old tuberculin followed by 0.1 ml. 1/100 in the negatives.

TABLE I.—July Mantoux and X-ray Tests

(a) Results in Various Ages

Age	No. Tested	Mantoux-positive	Percentage Positive	No. with X-ray Evidence of Tuberculosis
5	17	3	18	1
6	30	9	30	3
7	20	7	35	1
8	20	6	30	0
9	16	7	44	2
10	19	5	26	1
11	22	4	18	0
12	10	4	40	0
13	14	5	36	0
14	8	2	25	0
Total	176	52	30	8

The above figures include the five cases which were already undergoing treatment.

(b) Results in Various School Classes

Class	Age Range	No. Tested	Mantoux-positive	Percentage Positive	No. with X-ray Evidence of Tuberculosis
Infants	5-7	48	13	27	4
Standards I and II	6-9	31	8	26	1
Standard III	8-11	26	12	46	2
Standard IV	9-12	33	8	24	1
Forms I and II	11-14	38	11	29	0
Total	—	176	52	30	8

The schoolteacher who was now the suspected source of infection was in charge of Standard III. Why, then, should the infants' class show the greatest incidence of cases with x-ray evidence of tuberculosis? The explanation was apparent when it was discovered that this teacher was in charge of the infants' class from May 8 to 12 owing to the temporary absence of the infants' class teacher.