BROWN (P.K.)

A STUDY OF THE BLOOD IN SEVENTY-THREE CASES OF BONE TUBERCULOSIS IN CHILDREN WITH REFERENCE TO PROGNOSIS AND TREATMENT.

BY

PHILIP KING BROWN, A. B., M. D.,

SAN FRANCISCO, CAL.

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A STUDY OF THE BLOOD IN SEVENTY-THREE CASES OF BONE TUBERCULOSIS IN CHILDREN WITH REFERENCE TO PROGNOSIS AND TREATMENT.

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Why mesoblastic tissue in children is peculiarly susceptible to tuberculosis and the epithelial structures are correspondingly free I do not know, but the fact remains that glands and large bones are the usual seat of the tuberculous processes in children in contra-distinction to the commoner location in the lungs of adults. It is quite the rule to find enlarged cervical glands as the sole tubercular lesion in a child, pointing to a direct passage of the bacillus tuberculosis from the mucous surface to the gland. This is more clearly shown in the cases of tuberculosis of the mesenteric glands without primary tubercular ulceration of the intestines, which is rather the rule in children, while the condition occurs rarely if ever in adults. Attention has been called also to the infection of the bronchial and tracheal glands without a sign of pulmonary tuberculosis. The location of tubercular processes in children points therefore to infection through the blood and lymphatic circulation by the organisms taken up from the gastro-intestinal and respiratory tracts. It is not unreasonable to consider that there exists in the blood of children a condition which renders it less destructive to organisms than is the blood of adults, and to infer that the element of the blood destructive to organisms does not have its origin directly through the lymphatic system. What the different condition in children's blood is, and how it may account for the greater susceptibility of children to contagious diseases in general, I shall point out in a later paper, confining myself now to the changes in the blood caused by tubercular processes in bone and their chief complication, septicæmia. Secondary infection of the wound is almost unavoidable where a case comes to operation, so that it is necessary to consider that we have the changes of a mild septicæmia to deal with in considering the cases examined in the period following an operation.

HIP-JOINT TUBERCULOSIS, WITH ABSCESS.

A.—Operative Interference.

Case No.	Sex.	Age.	Duration.	Family History.	Location and Extent.	Treatment.	Date of Count.	Hæmoglobin Per cent.	Erythrocytes.	Leucocytes.
1	F.	13	2¼ yrs.	Negative.	Tub. of R. hip, with large abscess opened several times.	Excision of hip in '96. Presisting sinus, Sept., '96.	Sept. 22, 1896	68	4,750,000	12,760
							March 15, 1897	87	4,680,000	9,375
3	F.	9	3 yrs.	Father died phthisis.	Tub. of R. head of ulna with abscess	Excision, etc., '95. Sinus nearly healed April 1, '97.	Sept. 22, 1896	55	3,960,000	12,500
							April 1, 1897	76	4,760,000	5,940
4	F.	6⅔	2 yrs.	Negative.	Tub. of L. hip, with abscess.	Excision head of femur 1 yr. ago. Sinus nearly healed.	Sept. 22, 1896	50	3,720,000	14,150
							March 26, 1897	98	4,400,000	7,500
6	F.	11	7 yrs.	Twin sister had hip disease.	Tub. of hip, with psoas abscess.	Abscess opened and curetted in '92. Traction, sinus.	Sept. 27, 1896	40	3,960,000	26,850
							March 25, 1897	47	4,360,000	14,060
8	F.	12	3½ yrs.	Negative.	Tub. of hip; sinuses lungs involved	Curettings and excisions.	(Died)	15	2,800,000	9,650
9	F.	11½	8½ yrs.	Negative.	Tub. of hip, with abscess.	Excision of head of femur 4 yrs. ago. Curettings. Sinus discharging.	Sept., 1896	87	5,140,000	13 300
							March 3, 1897	85	5,140,000	15,625
10	F.	11½	8½ yrs.	Negative.	Tub. abscess outside hip joint in sac iliac syn.	Incision and curetting Oct., '96. Sinus presisting.	Jan., 1897	66	4,600,000	31,250
							March 26, 1897	95	5,920,000	14,075
12	F.	11½	4 yrs.	Pat. grandmother, uncles and aunts died of phthisis.	Tub. R. hip & L. wrist; abscess opened 4 yrs. ago; curetted Jan., '96.	Curetting. Traction.	Jan., 1897	38	4,133,600	6,650
							March 26, 1897	68	5,680,000	6,595
13	F.	5	3 yrs.	Negative.	Tub. of heads both femurs, with abscesses.	Abscess opened and bone excised 1 yr. ago, before entering Ch. hosp. Sinuses.	Feb., 1897	55	4,900,000	16,650
							March 29, 1897	55	4,480,000	15,625
16	F.	7	2 yrs.	Mat. grandmother died of phthisis.	Tub. disease of R. hip, with abscess in R. lumbar region.	Abscess opened 1 yr. ago.	Jan., 1897	75	4,200,000	6,650
							March 30, 1897	100	5,040,000	12,500
23	M.	7	1½ yrs.	Aunt had phthisis.	Tub. disease of hip-joint, with abscess above and behind joint.	Traction. Abscess opened Feb. 13, '97.	Feb. 4, 1897	68	5,260,000	11,250
							Feb. 13, 1897			12,185

24	M.	8	3 yrs.	Negative.	Tub. of L. hip, with abscesses which opened spont. 1 year ago.	Curetting. Traction. Abscesses now healed.		80	4,800,000	8,780
30	F.	13½	9 yrs.	Negative.	Tub. of L. hip.	Excision Dec.,'89. Curetting Jan., '97. Healed March, '97.	March 3, 1897	68	4,480,000	12,500
33	M.	14	3 yrs.	Negative.	Tub. of L. hip and elbow; multiple abscess.	Amputation at thigh.	Feb. 28, 1897	54	3,435,000	11,850
34	M.	9	6 yrs.	Negative.	Tub. of R. hip-joint, with abscess.	Excision and curetting '92. Many curettings since. Wound healed March, '97.	Feb., 1897	78	4,125,000	10,330
36	M.	3¼	2 yrs.	Negative.	Tub. of. L. hip, with abscess in inguinal region.	Traction, etc. Abscess opened Dec., '96.	March 4, 1897 April 6, 1897	57 68	4,132,800 4,500,000	29,564 19,365
37	M.	7¾	2 yrs.	Negative.	Tub. of hip, with abscess in joint.	Excision May, '96. Sinus presists.	March 6, 1897 April 10, 1897	58 68	5,328,000 5,400,000	12,185 13,000
38	M.	8	6 yrs.	Negative.	Tub. of L. hip, with abscess which opened spont. 4 yrs. ago	Discharging from 3 sinuses.	March 6, 1897 April 25, 1897	48 37	5,120,000 4,120,000	25,000 13,800
41	F.	8	1 yr.	Mat. gr. gr. mother died of phthisis.	Tub. disease of joint, with abscess opened 4 mos. before.	Head of bone excised March 14, '77. Sinuses, etc., curetted.	March 10, 1897	54	5,000,000	10,937
42	M.	7	2¼ yrs.	Negative.	Tub. of head of femur and acetab. abscess penetr'ing to pelvic cavity	Excision and curetting in '95. Numerous curettings since; last on March 14, '97.	March 10, 1897	68	4,560,000	9,357
44	F.	7	2½ yrs.	Maternal uncle had phthisis.	Tub. of hip, with abscess.	Exsection in '95, and abscess opened. Discharged in Jan., 97. Sinuses nearly healed.	March 11, 1897 (sinus not quite healed)	85	4,600,000	9,062
45	F.	21	6 yrs.	Negative.	Tub of L. hip.	Traction 3 mos.; 3 curettings in next 4 yrs. Excision of head of bone Feb., '96.	March 11, 1897 (sinus nearly healed)	92	5,640,000	9,062
46	F.	15	8 yrs.	Negative.	Extensive abscess of hip-joint; lumbar abscess opened Aug., '95.	Excision Feb., '95. Curetting Feb., '96.	March 11, 1897	96	4,440,000	9,680
54	M.	3½	1½ yrs.	Negative.	Tub. of hip, 2nd stage, with abscess	Opened Dec., '96. Wound healed March 31, '97.	March 22, 1897	67	5,840,000	8,595
55	M.	8½	4½ yrs.		Tub. of both hip-joints, with double psoas abscess.	Double exsection. Sinus presisting.	March 24, 1897	76	5,600,000	10,000
57	M.	15	6 yrs.	Negative.	Tub. of head of femur, with abscess which opened spont. 6 yrs. ago.	Excision in Oct., '92. Sinus presisting.	April 1, 1897 (2 hrs. p. c.)	81	4,800,000	10,250

HIP-JOINT TUBERCULOSIS, WITH ABSCESS.

B.—No Operative Interference.

Case No.	Sex.	Age.	Duration.	Family History.	Location and Extent.	Treatment.		Date of Count.	Hæmoglobin Per cent.	Erythrocytes.	Leucocytes.
11	F.	6	3 yrs.	Negative.	Tub. head of femur L. abscess all around joint, increasing in size (Feb. 3.) Mar. 16, stationary since.	Traction.		Feb. 1, 1897	72	5,168,000	14,370
								March 29, 1897	85	4,640,000	9,685
21	M.	2¼	4 or 5 mo.	Brother with hip disease. Case 31.	Tub. of head of L. femur, with abscess all around trochanter.	Traction.		Feb. 4, 1897	84	4,937,600	13,000
								April 2, 1897	65	4,560,000	13,437
23	M.	7	1½ yrs.	Aunt has phthisis, otherwise neg.	Tub. of hip-joint, with large abscess above and behind, gravitating down.	Traction. 13, '97.	Abscess opened Feb.	March 4, 1897	67	5,100,000	16,875
								March 6, 1897			15,450
								March 9, 1897			23,237
								March 10, 1897			22,250
								March 11, 1897	105		25,045
26	F.	3½	7 mos.	Negative.	Second stage hip dis. with abscess which began to show April 3, '97.	Traction.		March 4, 1877	65	4,852,800	18,800
								April 3, 1897	62	5,000,000	12,190
29	M.	5½	16 mos.	A sister had white swelling.	Tub. of head of L. femur, with large abscess (Pentroch.)	Traction.		March 3, 1897	80	5,200,000	10,000
								April 6, 1897	90	4,720,000	9,390
64	M.	9½	1½ yrs.	Negative.	Tub. of head of L. femur, with abscess which opened spont. 24 hrs. before exam.	Traction.		April 3, 1897	85	4,804,000	8,330

HIP-JOINT TUBERCULOSIS, WITHOUT ABSCESS.

No Operative Interference.

5	F.	7	5 mos.	Negative.	Tub. R. hip, limited abduction; pain in knee, limited rotation inward.	Traction.	Sept. 22, 1897	63	4,260,000	20,000
							March 25, 1897	90	4,480,000	9,060
14	F.	13	5 wks.	Mother died pul. tub. 3 yrs ago.	Tub. of head of L. femur; joint rigid from spasm.	Traction.	Dec. 30, 1896	73	5,000,000	7,250
							Feb., 1897 (Symp. of pul tub. develped.)			
							March 29, 1897 (Died Ap. 6,'97, of pul. tub.)	67	4,120,000	12,185
25	F.	5	2½ yrs.	Sister had vent. tub.	Tub. of L. hip.	Traction.	March 4, 1897	84	5,264,000	6,875
28	F.	6	14 mos.	Negative.	Tub. of head of R. femur.	Traction.	Feb. 28, 1897	78	3,665,000	10,330
							April 6, 1897	91	4,560,000	9,687
61	M.	10	1¼ yrs.	Pat. uncle phthisis	Tub. of head of R. femur. Abscess healed some months ago.	Traction.	March 29, 1897	75	4,332,000	6,250
62	F.	18	4 yrs.	Negative.	Tub. of R. hip.	Traction; splint.	March 29, 1897	55	3,040,000	9,375
68	F.	2½	1 yr.				April 6, 1897	66	4,600,000	15,000
					Tub. of R. hip, 1st stage.	Traction.	April 18, 1897	62	5,600,000	12,400
72	F.	3	6 mos.	Negative.	Tub. of R. hip.	Traction.	April 12, 1897	75	4,700,000	10,312
73	F.	5	2 yrs.	Negative.	Tub. of R. hip.	Traction; splint.	April 10, 1897	63	5,360,000	8,545

VERTEBRAL TUBERCULOSIS, WITH ABSCESS.

A.—Operative Interference.

Case No.	Sex.	Age.	Duration.	Family History.	Location and Extent.	Treatment.	Date of Count.	Hæmoglobin Per Cent.	Erythrocytes.	Leucocytes.
7	F.	4	1 yr.	Negative.	Kyphosis 11th and 12th D. V. Abscess in R. iliac region.	Abscess incised and curetted Feb., '96. Sinus presisting Feb., '97.	Feb. 1, 1897	55	4,080,000	12,500
							March 29, 1897	72	5,120,000	10,310
20	F.	3¾	2 yrs.	Negative.	Tub. disease 11th and 12th D. V. Psoas abscess increasing in size, Feb. 1, '97.	Jacket and traction to Feb., '97. Abscess incised Feb. 15.	Feb. 3, 1897	50	4,400,000	14,700
							Feb. 13, 1897	55		16,250
							Feb. 27, 1897	45	4,160,000	15,625
							March 4, 1897	53		23,248
							March 6, 1897			25,030
							March 18, 1897	47	4,026,400	11,800
							March 26, 1897	50	4,200,000	17,500
56	M.	4	1½ yrs.	Consumption in gr. mother, 2 aunts and father.	Vert. tuberc., with psoas abscess; presisting sinus.	Abscess opened Feb.'96. Traction.	March 24, 1897	50	4,440,000	13,125
							April 16, 1897	58	4,820,000	11,600
59	M.	11	5 yrs.	Negative.	Vert. tub. with psoas abscess, opened in '92 and '95. Many presisting sinuses.	Abscess opened 3 times and curetted. Traction.	March 24 1897	33	4,040,000	13,750
							April 15, 1897	32	4,040,000	19,200

B.—No Operative Interference.

19	F.	4	1½ yrs.	Negative.	Tub. of lower lumbar vert., with abscess behind L. trochanter, increasing in size. Noted 6 weeks before Feb. 3.	Jacket; no traction.	Feb. 3, 1897	55	5,850,000	8,125
							March 16, 1897 (Abscess smaller.)			
							March 30, 1897	85	5,520,000	7,500
20					(*See Case 20A.*)	Abscess incised & curetted Feb. 15.				
22	M.	8½	3 yrs.	Negative.	Scoliosis and kyphosis tub. of 9-12 D and 1st lumbar V. Abscess in L. iliac region getting larger, Feb.'97.	Traction.	Feb. 4, 1897	80	4,640,000	7,150
							April 2, 1897	85	5,140,000	8,905
66	F.	4	2 yrs.	Negative.	Vert. tuberc., with large psoas abscess.	Traction.	April 6, 1897 (12 hrs. p. c.)	89	5,280,000	10,520
69	F.	7½	10 mos.	Negative.	Kyphosis upper dorsal, with abscess in infrascapular region; 3 mos. duration.	Traction.	April 10, 1897	78	5,120,000	11,250
							April 20, 1897	73	5,600,000	10,000

VERTEBRAL TUBERCULOSIS, WITHOUT ABSCESS.

2	F.	10	5 yrs.	Aunt died of phthisis pulm.	Tub. disease middle dorsal region.	Splint traction.	Sept. 22, 1896	70	3,145,000	10,812
							March 28, 1897	85	4,720,000	8,750
17	F.	2½	1 yr.	Negative.	Tub. disease middle dorsal region.	Rest in bed; no traction.	Jan. 2, 1897	72	4,900,000	15,600
							March 30, 1897	85	4.960,000	7,500
18	F.	4	16 mos.	Negative.	Tub. disease 5th and 6th dorsal vert.	Splint traction.	Jan. 2, 1897	90	6,536,000	6,250
							April 2, 1897	75	4,280,000	5,625
31	M.	3¼		Brother, Case 21.	Tub. disease 5th dorsal vertebra. Tub. disease L. hip; incipient.	Splint traction.	March 6, 1897	43	5,040,000	13,125
							April 18, 1897	73	5,280,000	15,600
35	F.	6	3 yrs.	Negative.	Kyphosis 5th, 6th & 7th dorsal vert. No active disease now.	Brace and Jacket.	March 4, 1897	76	4,532,800	7,500
40	M.	2¾	6 mos.	Negative.	Tub. disease 11th & 12th dorsal vert.	Splint traction.	March 8, 1897	52	5.080,000	17,440
							April 17, 1897	62	5,200.000	12,400
52	M.	7	2½ yrs.	Negative.	Tub. disease 7th & 12th dorsal vert.	P. P. jacket and brace.	March 23, 1897	57	5,600,000	14,375
							April 18, 1897	73	5,360,000	9,900
65	M.	5½		Negative.	Tub. disease lower dorsal vertebra.	Rest in bed; no traction.	April 1, 1897	82	4,400,000	10,625
67	M.	2⅓	9 wks.	Mother had pulm. tub.	Tub. disease lower dorsal vertebra.	Rest in bed; no traction.	April 6, 1897	70	3,800,000	10,000

TUBERCULOSIS AT ANKLE-JOINT.

Abscess and Operation.

47	M.	5		Father died of pht.	Tub. of R. ankle joint; curetted.	Curetted May, '95; Feb.,'96. Mar., '97. Sinus presisting.	March 25, 1897	100	5,160,000	5,900

TUBERCULOSIS OF HIP-JOINT AND VERTEBRAL COLUMN COMBINED.

					No Abscess.	*No Operation.*				
49	M.	7	5 yrs.	Negative.	Tub. of hip and kyphos.	Traction, splint and jacket.	March 23, 1897	67	5,040,000	6,875
					Abscess and	*Operation.*				
43	M.	6	2 yrs.	Negative.	Spina-ventosa and osteo arthritis, hip.	Abscess opened Jan., '96. Curetted March, '97.	March 11, 1897	48	4,000,000	9,850
							April 15, 1897	47	4,280,000	9,200
60	M.	14	10 yrs.	Negative.	Tub. of middle dorsal vert. and hip.	Numerous operations over Cyrs. Pat. died April 4, '97.	March 25, 1897	47	2,830,000	5,100

TUBERCULOSIS AT OR NEAR KNEE-JOINT.

Case No.	Sex.	Age.	Duration.	Family History.	Location and Extent.	Treatment.	Date of Count.	Hemoglobin Per Cent.	Erythrocytes.	Leucocytes.
					No Abscess.	*No Operation.*				
15	F.	9¾	20 mos.	Negative.	Tub. synovitis L. knee, with effusion.	Inclined plane and traction.	Dec. 30, 1896 March 25, 1897	75 66	4,080,000 4,360,000	11,350 7,500
58	M.	11½	10 yrs.	Negative.	Flexion of knee from tub. disease.	Traction; extension.	March 23, 1897	100	5,200,000	8,812
70	F.	5¾	20 mos.	Pat. aunt died of phthisis.	Tub. synovitis, both knees.	Aspiration; rest in bed.	April 11, 1897	84	4,920,000	12,250
					No Abscess.	*Operation.*				
27	F.	1	3 mos.	Both mat. gr. parents died of pht.	Tub. R. knee and R. elbow (elbow incised by quack) reason unknwn		March 4, 1897	53	4,600,000	15,625
32	F.	2½	7 mos.	Aunt had hip disease.	Tub. focus in upper epiphysis of L. tibia.	Epiph. removed Jan., '97. Union by 1st intention, Mar. 16. Despite healing there is evident tub. of synovial membrane.	March 6, 1897 April 15, 1897	70 73	5,162,200 4,600,000	10,515 12,200
39	M.	4½	3¼ yrs.	Negative.	Slight kyphosis of 7th and 8th D. V. Tub of L. knee; very little motion, swelling around femr'l epiphyses.	Condyles trephined and foci removed; no pus.	March 5, 1897 April 18, 1897	70 73	5,040,000 5,600,000	12,500 6,200
63	M.	2	3 mos.	Negative.	Limp, pain and tenderness in L. knee; no marked enlargement.	Trephining.	March 29, 1897	72	5,200,000	8,650
71	M.	8	2 yrs.	Negative.	Tub of knee.	Removal of tub. granulations from synovial membrane. Curetting.	April 11, 1897 (17 hrs. p. c.)	82	5,680,000	11,875
					Abscess and	*Operation.*				
48	M.	7	4 yrs.		Tub. of knee and fingers; enlarged cervical glands.	Amputation above knee and of several fingers. Innumerable abscesses now.	March 23, 1897 April 18, 1897	46 50	3,880,000 5,600,000	11,875 14,800
50	M.	7	1½ yrs.	Negative.	Tub. of knee, with abscess in epiphysis of femur & popliteal space.	Curetting Jan., '97.	*March 21, 1897	100	5,172,000	14,155
51	M.	6¼	2 yrs.	Mat. grandfather died of phthisis.	Tub. synovitis of knee, with abscess.	Tub. focus removed at op., May, '96. Dischrgd Mar. 30,'97, cured.	March 21, 1897	87	4,680,000	9,065
53	M	10	2½ yrs.		Tub. of knee, with abscess which opened spontaneously 2 yrs. ago.	Epiphysiotomy in Apr.,'96. Sinus discharging.	March 23, 1897	68	4,920,000	7,500

* Child has been running very high temperature for some days; once subnormal (March 20); not much food for some days.

I have separated the 73 cases into groups according to the location of the process, the presence or not of a clinically demonstrable abscess, and the treatment. Two cases were studied carefully before and after operation where a large primary abscess was involved, and are therefore counted twice, making a total of 75, of which 42 came to operation. The cases are divided as follows:

Hip-joint tuberculosis with abscess.		
(*a*) operative interference		26
(*b*) no operative interference		6
Hip-joint tuberculosis without abscess, no operative interference		9
Vertebral tuberculosis with abscess.		
(*a*) operative interference		4
(*b*) no operative interference		5
Vertebral tuberculosis without abscess, no interference		9
Vertebral and hip tuberculosis with abscess and operation		2
Vertebral and hip tuberculosis, no abscess, no operation		1
Ankle joint tuberculosis, abscess and operation		1
Knee joint tuberculosis.		
(*a*) abscess and operation	4	
(*b*) no abscess and no operation	3	12
(*c*) no abscess but an operation	5	
		75

The family history was obtained in full in 69 cases and showed (1) absolutely negative results in regard to tuberculosis in 47 cases or 68 per cent; (2) negative in regard to direct inheritance in 59 or 85 per cent; (3) direct family history in parent, grand- or great-grandparent in 10 or 15 per cent, of which only 2 cases showed a family history in more than one other generation, and in no case were both parents affected. The direct influence of heredity is certainly a very small one.

From the distribution of the lesions and the varying history of injuries, the small value which can be attached to injury as an etiological factor is shown. It is so easy to obtain the history of an injury to the child if stress be laid on the

question, that only in exceptional cases have I been able to attach much import to any particular injury reported by the parent. The fact that in this list of cases the hip is attacked more than twice as often as the vertebral column and more than three times as often as the knee, which is eternally black and blue in a child from injury, and as the list shows no case of primary tuberculosis of wrist-, elbow- or shoulder-joints, it seems to me likely that too much stress has been laid on injury as an etiological factor. I am more inclined to the belief that anæmia from malnutrition and the accompanying lowered resistance of the blood to the invasion of the bacillus tuberculosis, are the most important factors in the etiology of bone tuberculosis. A large proportion of these cases show abundant cause for a secondary anæmia in the history of having been bottle-fed as babies, of having had serious digestive disturbances and of having been delicate always.

Of the technique employed in these examinations I shall say nothing further than to recount the methods employed and standards taken, so that comparisons with subsequent work in this line may be made accurately. The hæmoglobin determinations were made with von Fleischl's instrument, and the Thoma-Zeiss blood corpuscle counting apparatus was used for the counts—a dilution of twenty volumes with 3 per cent solution of common acetic acid being used for leucocytes and Heyeni's solution to 200 volumes dilution for erythrocytes. The differential counts were made from coverslip preparations made at the time of examination and stained with Neusser's modification of Ehrlich's triacid stain and eosin and hæmatoxylin.

The normal number of erythrocytes in male children I have taken at 5,000,000 per cubic millimeter of blood and in females at 4,500,000. Hæmoglobin in males is normal at 90 to 100 per cent, and in females as low as 80 per cent is not incompatible with a perfectly healthy color appearance to the erythrocytes. This great variation in the normal amount of hæmoglobin is not so marked in childhood.

Leucocytes settle down to about 10,000 per c. cm. shortly after birth and slowly diminish until the sixth year when the constant point of 7,500 is reached. An increase of about 2,000 per c. cm. is counted in my paper as leucocytosis. The counts

were all made from three and a half to five and a half hours after meals, at which time the digestive leucocytosis has generally subsided. In referring to abscess formation I mean a clinically demonstrable accumulation of pus.

Laache, among his early observations upon cases of tuberculosis in general, makes this statement: "Tuberculosis in itself in most cases gives no appearance of marked anæmia." The pallor of the skin is in marked contrast to the redness of the mucous membranes and the number of erythrocytes is not diminished, and the hæmoglobin is surprisingly high. The general statement is also made by different authorities that in pure tuberculosis of all organs there is an absence of change in the leucocyte conditions. Many observers fail to distinguish between simple tubercular infection and a combined infection with one of the pathogenic organisms, so that their deductions are valueless in this discussion. The work of Dane, of Boston, on the blood in bone tuberculosis is the only extended observation on the subject that I know of, and it is an admirable effort to put the subject in shape to be of use, and to show that a careful study of the blood, in this form of tuberculosis at least, does show interesting and important changes. I shall make Dane's conclusions a basis of criticism in presenting the results of the study of my cases.

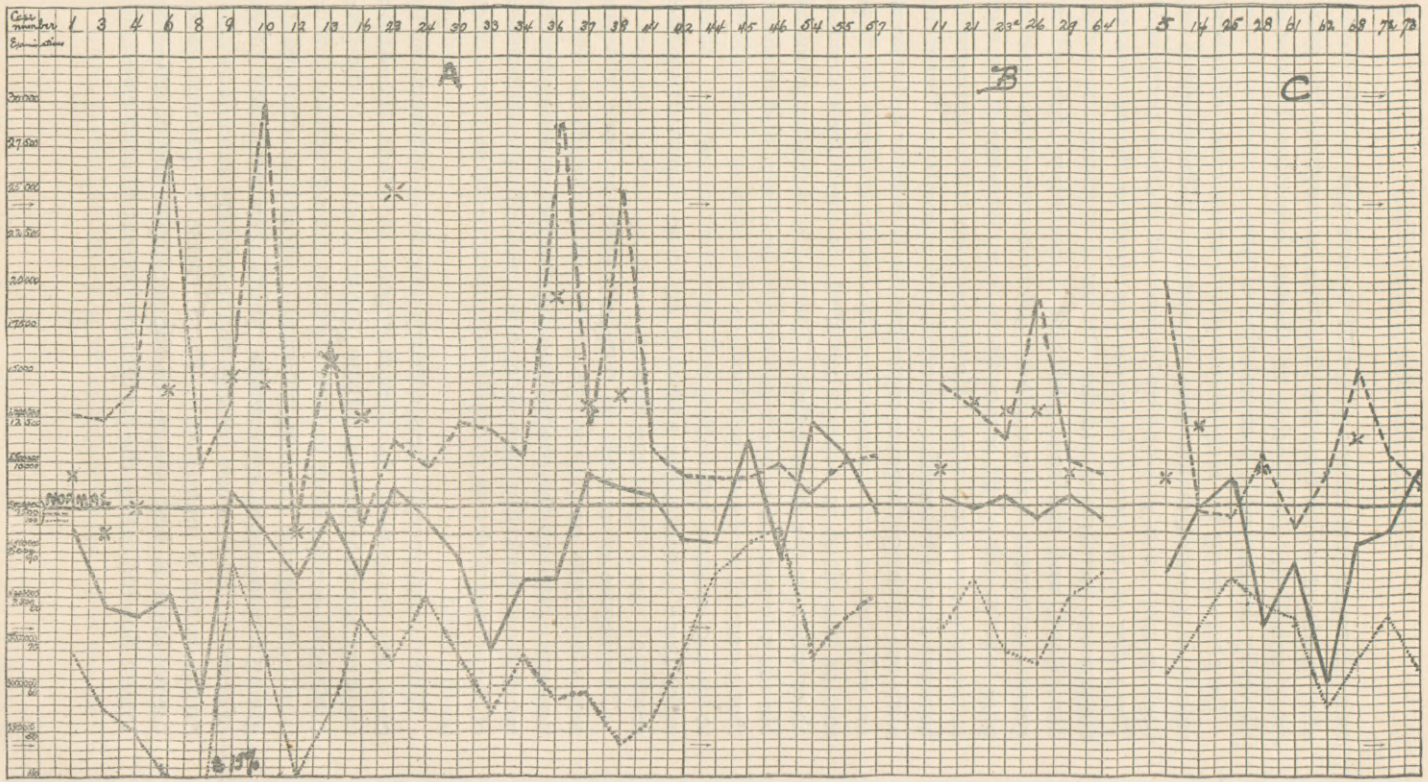
The degree of anæmia in purely tubercular bone disease seems to depend on (1) the age of the child, and (2) the duration and extent of the process, young children showing effects much more markedly than older ones, and a long continuance of the process under unfavorable circumstances telling decidedly on the child.

The resemblance of the secondary anæmia of tuberculosis to chlorosis has caused the Vienna school to adopt the name chloranæmia tuberculosa. Dane's observation that "the percentage of hæmoglobin in bone tuberculosis is generally diminished, giving rise to mild chlorosis," should be changed to giving rise to secondary anæmia, which may reach any grade of severity, for chlorosis belongs to the primary anæmias whose causal factor is unknown. The decrease in percentage of hæmoglobin is best shown on the accompanying charts—the lowest line indicating the percentage present in each case.

A— } Abscess formation.

HIP JOINT TUBERCULOSIS.
A—Operative interference
B—No operative interference.

C—No abscess formation and no operative interference.

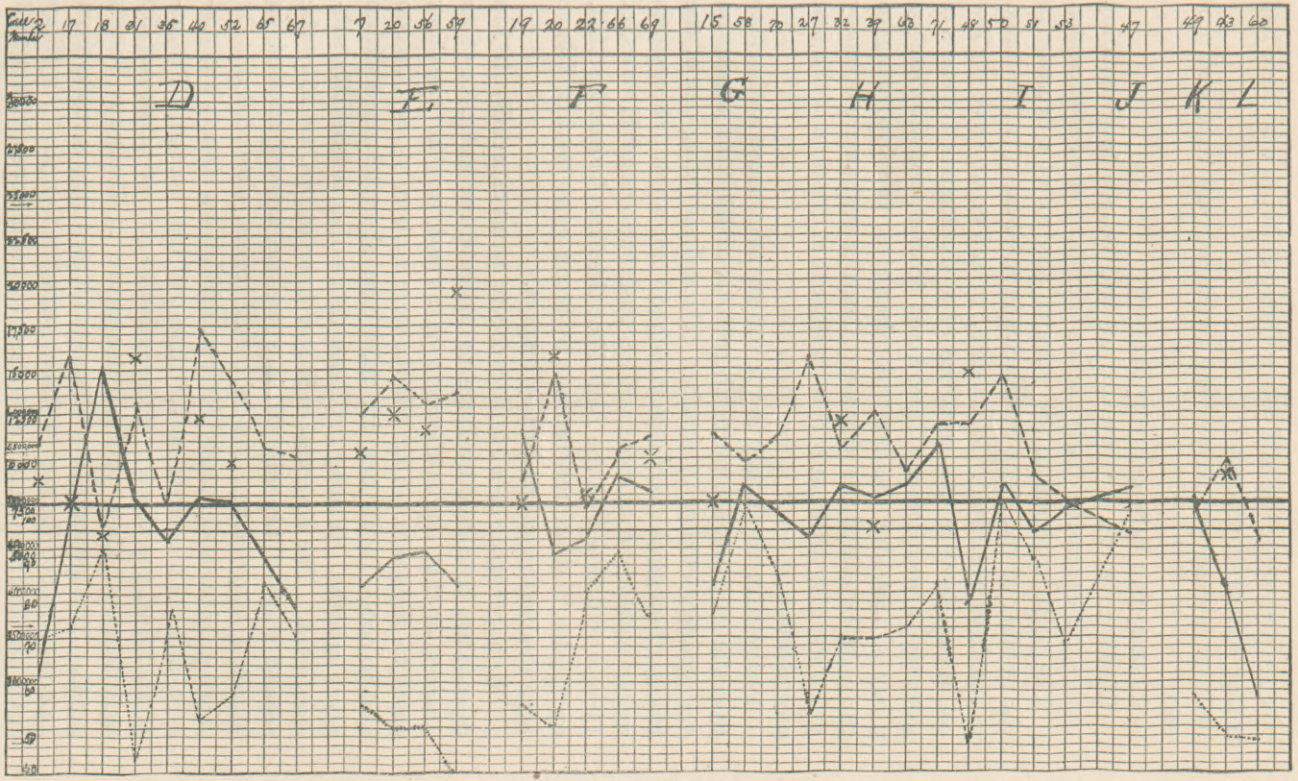


— — — — Leucocytes. ———— Erythrocytes. · · · · · · Hæmoglobin percentage. X Second leucocyte count.

VERTEBRAL TUBERCULOSIS (D. E. F.)
D—No abscess, no operative interference.
E—Abscess, operative interference.
F—Abscess, no operative interference.

KNEE AND ANKLE JOINT TUBERCULOSIS (G.H.I.J.)
G—No abscess, no operative interference.
H—No abscess, operative interference.
I & J—Abscess and operative interference.

HIP AND VERTEBRAL TUBERCULOSIS (K. L.)



— — — — Leucocytes. ——— Erythrocytes. · · · · · · Hæmoglobin percentage. X Second leucocyte count.

CASES 7, 19, 20, 40 and 56 are young children who have had a long continued process.

CASES 27, 49, 52 and 59, long continued and extended process.

CASE 27 is a baby one year old.

CASES 6, 8, 12, 13, 33, 43, 48 and 60 are long-standing cases with extensive disease, and a complication of septicæmia.

The point in regard to the general absence of a decrease in the number of red blood corpuscles in most cases is illustrated in the cases presented. In Cases 8, 33 and 60 we have the low count explained by the presence of a septicæmia in cases of extensive bone disease. Cases 8 and 60 died, and in Case 33, a boy of 14 years, the leg was taken of at the hip owing to the wide extent of the disease. Cases 2 and 62 are of many years standing without proper treatment at the time of the first examination.

I have referred already to the statement of most authorities that the leucocytes in pure tuberculosis of all forms present no very marked change in number, tuberculosis being one of the few pathological processes which do not show leucocytosis under ordinary conditions. The observations of Holmes, of Denver, published in the *New York Medical Journal* last fall, on the diagnosis of tuberculosis by blood examination, I have been unable to confirm in any respect.

The appearance of a high leucocyte count especially in hip disease has been held by Dane to indicate the presence of an abscess, or that one is about to be formed, and he cites four cases to illustrate. The counts were 14,000, 15,000, 20,000 and 30,000. In all but the third case abscess formed within seven months. Among my cases this point is not fully substantiated. The observation is corroborated by Case 26 in the second stage of hip disease in which no abscess showed on March 4, 1897, when the leucocyte count was 18,800. On April 3rd it was definitely determined that an abscess was present, and the leucocyte count that day was 12,190. Pus aspirated from the abscess was negative to culture and coverslip examination. Case 31 may also prove to illustrate Dane's point. The leucocyte count has increased in six weeks from 13,000 to 15,600, but as yet no abscess has formed. Six of my cases on the other hand have presented conditions which would

go to disprove Dane's point. All of them presented a more or less marked leucocytosis at first examination, and at the second examination, from two weeks to six months later, no abscesses had developed, and in every case the count was less.

Case No.	1st. Exam.	2nd. Exam.	Interval.
5	20,000	9,060	6 months
17	15,600	7,500	3 months
39	12,500	6,200	6 weeks
40	17,440	12,400	5 weeks
52	14,375	9,900	1 month
68	15,000	12,400	2 weeks.

I should be inclined therefore to modify very much the conclusion drawn by Dane by saying that a case may go on to abscess formation without any increase in the number of leucocytes, and give every appearance clinically, and through blood examination, of improvement (Cases 19, 22, 29). Undoubtedly purely tubercular abscesses of considerable size may be formed and be absorbed without there ever having been a leucocytosis. When, however, such a case begins to show an increase in the number of leucocytes, one of two things has happened, either a secondary infection has occurred (Cases 20, 23), or there is a considerably increased activity in the tubercular process (Cases 14, 21, 31, 32).

Case 14.—Girl of 13, with tubercular disease of head of left femur; five weeks duration at time of first examination. Leucocyte count January 1st, 7,250; March 29th, 12,185. In February pulmonary tuberculosis developed with hemorrhages from the start. Death early in April.

Case 21.—Boy 2½ years; had tubercular disease of the head of the left femur, with an abscess. The process was of five months duration at the date of first count. February 4th, hæmoglobin, 84 per cent; erythrocytes, 4,900,000; leucocytes, 13,000. April 2nd, hæmoglobin, 63 per cent; erythrocytes, 4,500,000; leucocytes, 13,400. Attempts at cultures on blood serum from pus aspirated April 8th, and from coverslips, were negative.

Case 31.—Boy of 3¼ years, with tubercular disease of fifth dorsal vertebra, and left hip trouble beginning. March 6th, hæmoglobin, 43 per cent; erythrocytes, 5,000,000; leucocytes, 13,125. April 18th, hæmoglobin, 73 per cent; erythrocytes, 5,280,000; leucocytes, 15,600. Clinical note, "Fever, appetite bad, local condition not improving."

CASE 32.—Girl 2½ years old, with a tubercular focus in upper epiphysis of left tibia of seven months duration. Focus removed January 1st, union by first intention. March 6th, hæmoglobin, 70 per cent; erythrocytes, 5,162,000; leucocytes, 10,515. April 18th, hæmoglobin, 73 per cent; erythrocytes, 4,600,000; leucocytes, 12,200. March 16th.—Clinical note, "Despite healing there is evident an active tuberculosis of the synovial membranes."

CASES 20 and 23 showed abscesses at the time of first examination, and showed leucocytosis, the counts being 14,700 and 11,250 respectively on February 3rd. Ten days later the count twelve hours after a feeding stood, 16,250 and 12,185, an increase in each case. On the theory of Gage, of Worcester, that this was an indication of secondary infection, and that the time for opening had come, both abscesses were opened immediately. Cultures on nutrient gelatine and agar-agar, and coverslips were negative in both cases. Commenting on Gage's statement (made in *Boston Medical and Surgical Journal* in 1896), I would say simply it is a well known law that all secondary infections cause leucocytosis, but that a moderate leucocytosis in a case with tubercular abscess probably has its cause in the sudden activity of the tubercular process, as I have already pointed out.

All the cases of primary tubercular abscess showing any degree of leucocytosis were examined most carefully in regard to this point. Pus was aspirated in every case but one and cultures on blood serum were attempted and coverslip preparations examined. In every case, as in Cases 20 and 23, the result was negative.

CASE 11.—February 1st, 14,370; March 29, 9,685; abscess smaller, no aspiration.

CASE 21.—February 4, 13,000; April 2, 13,437; aspirated April 8; negative.

CASE 26.—March 4, 18,800; April 3, 12,190; aspirated April 15; negative.

CASE 29.—March 3, 10,000; April 6, 9,390; aspirated April 8; negative.

CASE 66.—April 6, 10,520; aspirated April 13; negative.

CASE 69.—April 10, 11,250; aspirated April 13; negative.

This concerns also Dane's statement that with abscess formation low leucocyte count indicates absence, and high count

the presence of secondary infection with pyogenic organisms. It becomes a most important question to know what is meant by a high count. In Case 20 the pus at operation was negative, although just previous to operation the count was 16,250, showing an increasing leucocytosis. Two weeks later the count was 15,600. One week after that it was 23,000 and agar-agar cultures showed the staphylococcus pyogenes aureus. Case 23, showing the same condition and operated on at the same time, showed an increase of 4,500 in the leucocyte count three weeks after operation, making the count 16,800, and cultures showed the same organism. The patient developed tubercular meningitis and died five weeks after operation. No autopsy obtained. Judging from these two cases it seems difficult to say what is the lowest count which may be said to constitute the leucocytosis of secondary infection, and I would clear the ground partly by saying that if the known causes of leucocytosis be excluded, a rapid increase of several thousand in the leucocyte count in a case with tubercular abscess is most significant of secondary infection, for Cases 14, 20, 21, 23, 31 and 32 show that other causes—probably an activity in the tubercular process—make an increase in the leucocyte count. From the surgical point of view either cause of the increase might indicate the necessity of surgical interference.

That the leucocyte count bears no direct relation to the temperature, another observation of Dane, I can fully corroborate. The only relation which exists is where the cause of a leucocytosis is also the cause of the fever as in a secondary infection. Even here there is no constant relation between the two. The absence of relation is perhaps best illustrated in the fever following a malarial chill, and in the continued high temperature of typhoid fever where there is a diminished rather than increased number of leucocytes.

The leucocytosis which occurs from the infection of the large open wounds following operations on tubercular bone is fully illustrated by forty of the cases coming to operation. Infection is bound to follow the long continued dressing of these wounds. The leucocytosis in nearly all cases is very high for a period after the infection, and then it gradually falls unless the sepsis is acute and threatens the life of the patient, in which case it may remain high until a crisis is reached. If the resistance of the patient is good and the case progresses

favorably, the leucocytosis slowly disappears and the hæmoglobin percentage increases. If the anæmia was of the second degree of severity and the erythrocytes had greatly decreased, there occurs also an increase in their number. (Cases 1, 3, 4, 16, etc.)

The failure of the hæmoglobin percentage to rise, and of number of red blood corpuscles to increase, provided they were diminished, is a certain warning of an unfavorable condition in spite of the diminution of the leucocytes to the normal or below (Cases 8, 60). Stating the case generally, marked leucocytosis follows secondary infection with a pyogenic organism in children where the recuperative power is good, and the leucocytosis disappears as the recuperation goes on, or as the pyogenic material overcomes the recuperative power. In the first case the anæmia disappears, and in the second case it remains stationary or grows worse.

Before concluding I wish to state that all of the seventy-three cases examined were from the clinic at the Children's Hospital and the private practice of Dr. H. M. Sherman, and I wish to acknowledge my indebtedness to Dr. Sherman for his kindly help and many suggestions in carrying on this study.

RÉSUMÉ.

I. No decrease in erythrocytes except in secondary anæmias of second and third stages, which come (*a*) in long-standing and extensive cases, (*b*) in very young children, and (*c*) in septic infections.

II. Hæmoglobin is decreased in all cases, and in proportion to the same factors which influence the erythrocytes.

III. The return to health is indicated by the tendency of blood to return to the normal.

IV. Abscess formation not necessarily accompanied by leucocytosis. Slowly developing leucocytosis points to activity in the tubercular process. Rapidly developing leucocytosis points to secondary infection with pyogenic bacteria. Abscess may be absorbed without a leucocytosis having developed.

In septic infection of wounds, leucocytosis is marked at first and diminishes as the resistance of the child increases or decreases. If the diminution is accompanied by an increased anæmia, it is a sign of the lowered vitality of the child.

W. A. WOODWARD & CO., 12 SUTTER ST., S. F