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LOW BASAL METABOLISM FOLLOWING THYROTOXICOSIS: *II. Permanent Type without Myxedema*

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LOW BASAL METABOLISM FOLLOWING THYROTOXICOSIS

II. Permanent Type without Myxedema[1]

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INTRODUCTION

As outlined in part I of this study (1), in the course of an investigation of the surprising number of low metabolisms observed following thyrotoxicosis, it was found that approximately half were of the temporary type and half of the permanent type: and moreover, that about two-thirds of the latter group showed no clinical evidence of myxedema.[4] It is our purpose in this paper to present a study of permanent low metabolism without myxedema. The data on temporary and permanent myxedema following thyrotoxicosis, are to be presented later (2).

Although there are a few reports in the literature on low metabolism without myxedema, there are no studies of this phenomenon following thyrotoxicosis.

METHOD AND MATERIAL

For the method used, see part I of this study (1).

Included in this series are 21 patients who, without clinical evidence of myxedema, had a low metabolic rate, presumably of the permanent type, ranging from minus 16 to about minus 25 per cent, after recovery from thyrotoxicosis. In 14 instances the metabolism was followed long enough and closely enough to indicate that it would remain perma-

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[4] The term "myxedema" is used to denote any degree of true thyroid deficiency which is clinically discernible. It is not limited to the full-blown typical picture.

TABLE 1

Twenty-one cases showing permanent low metabolism without myxedema, following thyrotoxicosis

Case number	Description	Date	Basal metabolic rate	Pulse	Weight	Treatment	Clinical notes
			per cent		*kgm.*		
1	Mrs. A. G. (see fig. 2)						
2*	Exophthalmic goiter	December 2, 1924	+51	128	57.4	Lugol's solution M. XV daily	Moderate thyrotoxicosis for 6 months. Goiter +. Slight exophthalmos. Tremor +. Loss of 15 pounds
		December 8, 1924	+18	100	57.4		
		December 10, 1924				*Subtotal thyroidectomy*	
	Mrs. E. C.	December 20, 1924				Lugol's decreased to M. V daily	
	Age 47	December 22, 1924	−6	70	57.4	Lugol's omitted	No thyrotoxicosis
	Lab. No. 2940	January 27, 1925	−10	72	60.6		
		March 3, 1925	−16	64	61.0		Very well
		May 2, 1925	−25	62	58.7	Thyroid extract (Burroughs Wellcome) grains Iss daily	Mild myxedema
		May 14, 1925	−21	68	59.3	Thyroid increased to grains Iss and III on alternate days	Improved
		May 29, 1925	−17	64	58.6	Thyroid increased to grains III daily	
		June 25, 1925	−11	65	59.5		
		August 27, 1925	−19	68	58.8	Thyroid increased to grains IV daily	No myxedema. Well
		October 28, 1925	−14	72	60.6		
		December 31, 1925	−7	68	60.6		
		March 25, 1926	−9	78	61.8		
		June 23, 1926	−9	72	58.5		
		October 28, 1926	−17	74	60.7		Perfectly well

		December 2, 1926	−12	68	60.7		
		January 6, 1927	−15	74	58.7	Thyroid omitted	Perfectly well
		February 19, 1927	−17	76	60.6		
		March 19, 1927	−22	64	60.6		
		April 2, 1927	−18	72	60.3		Just as well as when on thyroid.
		May 14, 1927	−28	66	59.0	Thyroid extract grains I daily	No myxedema
		July 12, 1927	−24	72	58.0	Thyroid decreased to grains ss	No change
		August 15, 1927	−20	68	57.7	daily	No myxedema
		October 22, 1927	−18	68	58.3	Thyroid omitted	Perfectly well
3	Mrs. A. A., (see fig. 1 and case history on page 486)						
4	Mrs. M. H. (see fig. 4)						
5	Mrs. E. W. (see fig. 3)						
6	Exophthalmic goiter	January 27, 1925	+45	112	40.1		Moderate thyrotoxicosis 2 months.
		January 29, 1925				Lugol's solution M. XV daily	Goiter +. Slight exophthalmos.
		February 4, 1925	+22	94	41.0		Tremor +. Lost 17 pounds in
	Mrs. O. McC.	February 6, 1925	+11	84	41.0		6 months
	Age 32	February 7, 1925				*Subtotal thyroidectomy*	
	Lab. No. 3023					Lugol's increased to M. XLV daily	
		February 8, 1925				Lugol's decreased to M. XXX daily	
		February 11, 1925				Lugol's decreased to M. XX daily	
		February 13, 1925				Lugol's decreased to M. XV daily	
		February 16, 1925	−6	72	41.4		

*We wish to thank Dr. J. H. Means for the use of the data on this case and on cases 8 and 19.

TABLE 1—*Continued*

Case number	Description	Date	Basal metabolic rate	Pulse	Weight	Treatment	Clinical notes
			per cent		*kgm.*		
6	Exophthalmic goiter	February 17, 1925				Lugol's omitted	
		February 18, 1925				NaI (saturated solution) M.V every other day	Much improved
	Mrs. O. McC.	March 24, 1925	−19	70	43.4	NaI, M. V daily every other week	No thyrotoxicosis. No myxedema
	Age 32	May 27, 1925	−11	74	45.1	NaI omitted	Well. Slightly nervous
	Lab. No. 3023	November 12, 1926	−18	68	47.5	Lugol's solution M. V daily	No myxedema
7	Toxic adenoma	June 26, 1925	+13	85	40.1	On potassium iodide	Mild thyrotoxicosis for 3 years. Goiter +. Tremor +. Nervousness. Palpitation ++
		July 1, 1925	−3	78	40.0	Iodide omitted	
	Mrs. J. E.	July 10, 1925				*Left hemithyroidectomy*	
	Age 28	September, 1925					No change since operation
	Lab. No. 3338	December 1, 1925	−22	76	42.1		
		December 14, 1925	−20	72	42.4	Thyroid extract (Armour's) grains Iss daily	No myxedema. Nervous
		December 24, 1925	−13	68	43.2	Thyroid increased to grains III daily	? Improvement
		February 8, 1926	−21	82	44.9	Thyroid decreased to grains Iss daily	
		March 30, 1926	−8	76	46.5	Thyroid decreased to grains I daily	Very nervous. Pregnant
		April 15, 1926	−5	84	47.7		
		June 21, 1926	+9	87	51.1		Well
		August, 1926				Thyroid omitted	Parturition
		September 24, 1926	−13	74	45.1	Thyroid grains Iss daily	

		October 15, 1926				Thyroid omitted	
		November 5, 1926	−1	68	46.4		Nervous
		December 31, 1926	−16	72	48.8		No myxedema
		April 1, 1927	−19	64	49.3		
		June 24, 1927	−28	56	49.2		No myxedema
8	Exophthalmic goiter	May 21, 1920	+10	94	48.0		Thyrotoxicosis for 1 year. Eyes stary. Goiter +. Palpitation ++. Perspiration ++
	Mrs. P. T.	April 5, 1923	+66	120	54.0		Persistent thyrotoxicosis
	Age 26	April 25, 1923	+38	126	55.5		
	Lab. No. 615	April 27, 1923				*Double ligation* of superior thyroid	
		May 3, 1923	+37	106	52.5	arteries	
		May 11, 1923	+30	115	51.5		
		May 17, 1923	+32	112	52.2		
		May 18, 1923	+7	88			
		May 19, 1923	+26	110			
		May 22, 1923	+20	93	53.0		
		May 23, 1923				*Subtotal thyroidectomy*	
		May 26, 1925	−28	76	66.7		Very well. No myxedema
		March 10, 1927	−14	74	63.1		Very well. No myxedema
9	Exophthalmic goiter	1913 to 1915					Goiter +. Exophthalmos +. Tachycardia. Tremor. Weakness
	Mrs. L. W.						
	Age 49	March 31, 1923	−22	65	72.0		Goiter and exophthalmos. Weakness chief complaint. No myxedema
	Lab. No. 1927	April 2, 1923	−35	65	72.0		
		April 5, 1923	−29	60	72.0		
		April 6, 1923				Thyroid extract grains IX daily	
		April 20, 1923	−5	72	71.0		

TABLE 1—*Continued*

Case number	Description	Date	Basal metabolic rate	Pulse	Weight	Treatment	Clinical notes
			per cent		*kgm.*		
9	Exophthalmic goiter	April 23, 1923				Thyroid omitted	
		May 9, 1923	−21	67	70.0	Thyroid extract grains IX daily	
		June 5, 1923	+8	100	68.0	Thyroid decreased to grains III and	Palpitation and trembling
	Mrs. L. W.	August 7, 1923	−1	80	68.5	IVss on alternate days	
	Age 49	November 1, 1923	±0	65	71.0	Thyroid increased to grains IVss	
	Lab. No. 1927	January 3, 1924	−6	64	71.0	daily	
		March 7, 1924	+8	92	70.0	Thyroid decreased to grains III	Weak spells
		April 5, 1924	−9	64	69.0	daily	
		May 29, 1924	+2	80	69.5		
		June 28, 1924	±0	68	69.5		
		September 6, 1924	+1	68	71.0		Weak and listless
		October 29, 1924				Thyroid omitted	
		November 8, 1924	−10	70	74.0	Thyroid extract grains III daily	
		February 7, 1925	−7	64	71.8		
		June 7, 1925	−7	64	73.5		
		August 29, 1925	−8	76	74.0		Weak spells
		December 19, 1925	−10	72	76.7	Thyroid increased by grains Iss every third day	
		March 20, 1926	+1	72	74.9	Thyroid decreased grains Iss every other day	Better than ever before
		April 24, 1926	−13	76	76.1	Thyroid increased to grains III daily	Tired and dopy, but no myxedema
		October 7, 1926				Thyroid omitted	

		October 30, 1926	−5	64	77.9		
		November 13, 1926	−7	96	77.9		
		December 3, 1926	−10	76	77.3		
		December 18, 1926	−18	78	77.1		No change. No myxedema
		January 8, 1927	−2	74	78.0	Thyroid extract (Armour's), grains	
		February 12, 1927	+17	92	76.6	III daily	No change, except minded cold less
		April 2, 1927	+5	112	73.7	Thyroid decreased to grains Iss	
		June 11, 1927	−5	114	74.7	daily	
10	Exophthalmic goiter	March 25, 1925	+55	96	56.6		Mild thyrotoxicosis for 2 years. Goiter +. Tremor +. Exophthalmos +. Lost 34 pounds in 6 years
		March 26, 1925				Lugol's solution M. XV daily	
		April 3, 1925	+25	86	57.4		
	Mr. E. J.	April 6, 1925				*Subtotal thyroidectomy*	
	Age 30					Lugol's increased to M. LX daily	
	Lab. No. 3135	April 9, 1925				Lugol's decreased to M. XV daily	
		April 22, 1925	−4	70	58.0	Lugol's omitted	Much improved
		May 22, 1925	−5	72	60.3		
		April 27, 1926	−14	68	66.7	Lugol's M XX daily	No thyrotoxicosis
		May 11, 1926	−16	72	67.4	Lugol's decreased to M. X daily	No myxedema
						Started thyroid extract (Armour's) grains III daily	
		May 27, 1926	−6	83	65.1	Thyroid decreased to grains Iss daily	
						Lugol's decreased to M. V daily	
		June 28, 1926	−17	72	66.9	Thyroid omitted	Doubtful improvement on thyroid
		July 26, 1926	−29	75	67.7		
		September 13, 1926	−13	74	65.7	Thyroid grains III daily	
		October 23, 1926	−12	68	62.3	Thyroid omitted	Doubtful improvement on thyroid
		December 18, 1926	−14	59	62.1		
		January 15, 1927	−15	63	63.7		

TABLE 1—*Continued*

Case number	Description	Date	Basal metabolic rate	Pulse	Weight	Treatment	Clinical notes
			per cent		*kgm.*		
10	Exophthalmic goiter	February 12, 1927	−9	80	63.4		Well
		March 12, 1927	−23	74	63.4		Well. No myxedema
		April 23, 1927	−14	70	65.3	Lugol's omitted	
	Mr. E. J.	May 27, 1927	−9	72	65.0		No change since omission of Lugol's
	Age 30						
	Lab. No. 3135	July 5, 1927	−12	64	65.1		Well
11	Exophthalmic goiter	February 6, 1922	+43	103	45.5		Moderate thyrotoxicosis about 1 year. Goiter +. Exophthalmos +. Tremor +. Lost 17 pounds in 2 months
		February 13, 1922				*First x-ray treatment*	
		February 15, 1922	+45	100	45.0		
	Mrs. E. G.	March 8, 1922				*Second x-ray treatment*	
	Age 48	March 29, 1922				*Third x-ray treatment*	
	Lab. No. 1311	March 30, 1922	+32	101	51.0		Improved
		April 26, 1922				*Fourth x-ray treatment*	
		April 27, 1922	+13	85	49.5		
		May 17, 1922				*Fifth x-ray treatment*	Mild thyrotoxicosis
		June 6, 1922	+16	83	49.0		
		July 14, 1922	+19	82	48.0		
		July 19, 1922				*Sixth x-ray treatment*	
		August 16, 1922				*Seventh x-ray treatment*	
		September 6, 1922	+27	114	50.0	*Eighth x-ray treatment*	
		September 21, 1922	+11	76	50.0		
		October, 1925					Headaches. No menses for 5 months

		November 4, 1925	−17	64	62.3		Improved
		November 12, 1925	−16	64	62.5		No myxedema, but tired all the time
		November 15, 1926	−15	72	64.6	Lugol's solution M. X daily	No myxedema
		November 30, 1926	−6	66	64.3	Thyroid extract (Armour's) grains IVss daily Lugol's decreased to M. V daily	
		January 6, 1927	−4	74	62.5	Thyroid increased to grains VI daily	
		February 7, 1927	+3	68	61.6	Thyroid decreased to grains Iss daily	? Improvement
		March 21, 1927	−6	72	63.3	Thyroid increased to grains III daily	
		May 17, 1927	±0	68	65.0	Thyroid omitted	No change on thyroid. No myxedema
12	Toxic adenoma	October 7, 1919	+50	116	39.0	*First x-ray treatment*	Moderate thyrotoxicosis 2 years Goiter +. Tremor +. Marked weight loss
		October 27, 1919				*Second x-ray treatment*	
	Mrs. M. B.	November 19, 1919				*Third x-ray treatment*	
	Age 38	December 15, 1919	+52	100	42.0	*Fourth x-ray treatment*	Much improved
	Lab. No. 348	January 5, 1920				*Fifth x-ray treatment*	
		January 26, 1920				*Sixth x-ray treatment*	
		February 25, 1920	+18	80	47.0		
		September 29, 1920	−3	66	48.5		Well
		February 25, 1921	−6	64	50.5		
		August 29, 1921	−35	40	48.0	Thyroid extract grains IVss daily	Myxedema
		September 15, 1921	+2	61	45.5		Normal again
		October 27, 1921				Thyroid omitted	
		October 31, 1921	−13	60	46.0	Thyroid extract grains IVss daily	
		January 10, 1922	−1	63	46.0		
		March 7, 1922	±0	57	46.0		Perfectly well

TABLE 1—*Continued*

Case number	Description	Date	Basal metabolic rate	Pulse	Weight	Treatment	Clinical notes
			per cent		*kgm.*		
12	Toxic adenoma	June 10, 1922				Thyroid omitted	
		July 3, 1922	−23	61	47.0	Thyroid grains IVss daily	Myxedematous again
	Mrs. M. B.	August 15, 1922				Thyroid omitted	
	Age 38	September 7, 1922	−23	60	46.0	Thyroid grains IVss daily	Myxedematous
	Lab. No. 348	February 8, 1926	+3	72	43.3	Thyroid decreased to grains III daily	Excellent condition. No myxedema
		November 10, 1926	−11	64	45.5	Thyroid omitted	
		November 30, 1926	−8	58	45.9		
		December 21, 1926	−8	68	46.0		
		January 4, 1927	−13	62	45.6		
		January 27, 1927	−19	56	46.3		No myxedema. Just as well as when on thyroid
		February 15, 1927	−17	60	45.9		
		March 8, 1927	−23	56	46.5		Perfectly well
		April 26, 1927	±0	68	47.1		
		June 7, 1927	−12	64	45.7		
		June 21, 1927	−8	64	45.7		
		July 19, 1927	−15	64	45.7		
		August 9, 1927	−8	60	46.0		Perfectly well. No myxedema
13	Exophthalmic goiter	December 9, 1926	+88	130	49.9	Lugol's solution M. XLV daily	Moderate thyrotoxicosis 1½ years. Goiter +. Exophthalmos +. Tremor +. Lost 40 pounds in 1 year
		December 15, 1926	+37	105	49.9		
		December 19, 1926				Lugol's decreased to M. XXX daily	
	Mrs. L. E.						
	Age 43	December 23, 1926				Lugol's decreased to M. XV daily	
	Lab. No. 4383	December 24, 1926	+13	80	47.5	*Subtotal thyroidectomy*	

		January 11, 1927				Lugol's omitted	
		January 21, 1927	−16	61	54.7		? Mild myxedema.
		February 1, 1927	−7	62	56.9	Lugol's M. V. daily	Mild myxedema. No thyrotoxicosis
		February 11, 1927	−12	68	57.4	Thyroid extract (Armour's) grains	Mild myxedema
		February 21, 1927	−3	80	58.4	III, daily	No change
		March 14, 1927	−5	70	58.8		Improved
		April 18, 1927	−2	68	60.4	Thyroid omitted. Lugol's con-	Well
		May 17, 1927	−17	68	64.0	tinued	
		June 3, 1927	−22	58	62.9	Lugol's omitted	
		August 2, 1927	−16	64	62.6		No myxedema
		August 12, 1927	−23	56	62.9		
		October 26, 1927	−19	63	65.5	Thyroid extract grains Iss daily	No myxedema. Perfectly well
14	Mrs. A. G. (see fig. 5)						
15	Exophthalmic goiter	May 19, 1926	+94	138	29.8		Moderate thyrotoxicosis 4 years Exophthalmos +. Goiter +. Tremor ++. Lost 53 pounds in 5 years
		May 24, 1926	+72	117	29.5	Lugol's solution M. XLV and	
		May 26, 1926	+69	122	29.7	thyroid extract (Armour's)	
	Mrs. D. C.	May 28, 1926	+57	108	29.7	grains Iss daily	
	Age 32	May 29, 1926	+63	108	29.6		
	Lab. No. 3980	June 1, 1926	+69	108	29.3		
		June 3, 1926	+63	108	29.7		
		June 5, 1926	+58	112	29.7	Thyroid omitted	
		June 8, 1926				*Right hemithyroidectomy*	
						Lugol's increased to M. LX daily	
		June 9, 1926				Lugol's decreased to M. XXX daily	
		June 14, 1926	+51	117	29.5		
		June 17, 1926				Lugol's decreased to M. XX daily	

TABLE 1—*Continued*

Case number	Description	Date	Basal metabolic rate	Pulse	Weight	Treatment	Clinical notes
			per cent		*kgm.*		
15	Exophthalmic goiter	June 19, 1926				Lugol's decreased to M. V daily	Improved
		July 28, 1926				Lugol's increased to M. XXX daily	
		July 29, 1926				Lugol's increased to M. XL daily	
	Mrs. D. C.	July 30, 1926				*Left hemithyroidectomy*	
	Age 32					Lugol's increased to M. LX daily	
	Lab. No. 3980	July 31, 1926				Lugol's decreased to M. XXX daily	
		August 7, 1926	+9	115	35.3	Lugol's decreased to M. XV daily	Much improved. No thyrotoxicosis
		September 22, 1926	−21	73	43.9		? Mild myxedema
		October 22, 1926	−15	74	45.5	Lugol's decreased to M. V. daily	
						Thyroid extract (Armour's) grains IVss daily	? Mild myxedema
		November 6, 1926	+10	92	44.3	Thyroid decreased to grains Iss daily	Myxedema practically gone
		December 4, 1926	−5	76	44.3	Thyroid omitted. Lugol's continued	Perfectly well
		December 30, 1926	−7	72	45.6		Perfectly well
		February 5, 1927	−16	84	47.2	Thyroid extract grains III daily	? Mild myxedema, less marked than in September, 1926
		February 19, 1927	−11	88	46.5		No myxedema
		April 2, 1927	−9	96	46.9	Thyroid decreased to grains Iss daily	Well
		May 7, 1927	−11	68	48.0	Thyroid omitted. Lugol's continued	

		June 11, 1927	−18	78	48.9	Lugol's omitted	
		July 2, 1927	−12	86	47.9		
		July 23, 1927	−12	70	48.5	Lugol's M. V daily	Better than ever in her life
		August 6, 1927	−18	72	48.7		No myxedema
		August 20, 1927	−24	72	49.1		
		October 15, 1927	−22	74	51.1	Lugol's omitted	Perfectly well. No myxedema
16	Exophthalmic goiter	July 23, 1925	+38	103	45.7		Moderate thyrotoxicosis 3 months. Slight exophthalmos. Goiter +. Tremor +. Lost 30 pounds in 9 months
		July 28, 1925	+39	96	45.5	Lugol's solution M. XXX daily	
		July 30, 1925	+39	84	44.6		
	Mrs. M. F.	August 2, 1925	+18	84	44.9		
	Age 33	August 6, 1925				Lugol's omitted	
	Lab. No. 3399	August 7, 1925				*Subtotal thyroidectomy*	
		August 22, 1925	+6	76	46.0		
		September 8, 1925	−7	68	46.9		
		October 13, 1925	±0	74	49.2		
		April 20, 1926	−10	74	52.0	Lugol's M. X daily	
		May 7, 1926	−16	70	53.1	Lugol's increased to M. XX daily	No myxedema. Slight residual thyrotoxicosis
		May 18, 1926	−18	60	53.9	Lugol's decreased to M. X daily	Less nervous
		May 25, 1926	−13	55	54.3	Thyroid extract (Armour's) grains Iss daily	
		June 7, 1926	−12	61	53.7	Thyroid increased to grains IVss daily	
		June 28, 1926	−5	59	51.5		Definite improvement
		July 12, 1926	+4	66	50.9	Lugol's omitted	
		July 27, 1926	+2	72	50.6		
		September 1, 1926				Thyroid omitted	
		November 9, 1926	−7	68	52.5	Lugol's M. XX daily	Well
		December 3, 1926	−12	61	53.6	Lugol's decreased to M. V. daily.	
		January 18, 1927	−7	65	50.5	Thyroid extract grains IVss daily	Severe cold for 3 weeks
		February 25, 1927	+6	72	50.3		Well
		April 21, 1927	+2	76	48.5	Thyroid omitted. Lugol's continued	Tired
		June 7, 1927	−14	66	51.7		Well. Less tired
		July 11, 1927	−17	72	51.5	Thyroid grains Iss daily	Easily tired. No myxedema

TABLE 1—*Concluded*

Case number	Description	Date	Basal metabolic rate	Pulse	Weight	Treatment	Clinical notes
			per cent		*kgm.*		
17	Exophthalmic goiter	June 11, 1925	+62	124	53.8		Moderate thyrotoxicosis 6 months. Goiter +. Exophthalmos +. Tremor +
		June 13, 1925	+63	110	53.2		
		June 14, 1925	+49	118	52.3	Lugol's solution M. XXX daily	
	Miss I. C.	June 17, 1925	+33	96	51.8		
	Age 23	June 19, 1925	+37	96	52.0		
	Lab. No. 3303	June 21, 1925	+20	82	52.2		
		June 23, 1925				*Subtotal thyroidectomy* Lugol's increased to M. LX daily	
		June 26, 1925				Lugol's decreased to M. XXX daily	
		July 3, 1925	−9	61	52.5	Lugol's omitted. Started NaI	Much improved
		July 24, 1925	−13	79	55.6	(saturated solution) M. V every	
		August 6, 1925	−15	59	59.2	other day	
		August 22, 1925	−16	63	63.5		
		September 16, 1925	−19	70	60.7		No thyrotoxicosis. No myxedema
		October 14, 1925	−19	65	61.5		
		November 1, 1925				NaI omitted	
		November 13, 1925	−12	80	61.7		
		November 20, 1925	−18	61	60.9	KI M. V daily	
		December 1, 1925	−17	67	62.3		No myxedema
		December 10, 1925	−16	72	62.3	Thyroid extract (Armour's) grains IX daily	
		December 15, 1925	−15	67	62.5	Thyroid increased to grains XVIII daily	No change
		December 17, 1925	−6	80	60.5	Thyroid decreased to grains III daily. KI continued	? Improvement. No thyrotoxicosis. Well

18	Exophthalmic goiter	February 5, 1919	+40		63.0		Mild thyrotoxicosis about 1 year. Goiter +. Slight exophthalmos. Tremor +. Loss of 20 pounds in 1 month
		February 13, 1919				*Subtotal thyroidectomy*	
		October 25, 1919	−5	78	72.5		
	Miss C. A.	June 4, 1920	−10	76	71.7		
	Age 36	October 18, 1921	−8	67	72.5		Well
	Lab. No. 200	January, 1922				Double oophorectomy for ovarian	
		August 2, 1923	−19	68	80.0	cyst	Well. No myxedema
19	Exophthalmic goiter	June 8, 1922	+34	85	49.5		Moderate thyrotoxicosis for 3 years. Exophthalmos +. Tremor +. Rapid weight loss. Goiter not visible
		July 7, 1922	+66	80	48.4	(In another hospital)	
		July 8, 1922				*Subtotal thyroidectomy*	
	Mr. W. A. H.	July 14, 1922	+47	70	47.0		
	Age 64	September 22, 1922	+8	48	57.5		No thyrotoxicosis
	Lab. No. 1518	January 27, 1923	+6	50	59.0		Doing well
		May 26, 1927	−25	52	59.0		No myxedema. No thyrotoxicosis. Moderate arteriosclerosis
20	Exophthalmic goiter	January 20, 1919	+16	84	52.5		Mild thyrotoxicosis 6 months. Goiter +. Tremor +. No eye signs. Lost 14 pounds in 6 months
	Mrs. D. B.	January 28, 1919				*Left hemithyroidectomy*	
	Age 30	November 10, 1919	−8	80	53.0		Much improved. No thyrotoxicosis
	Lab. No. 202	February 21, 1922	−8	70	50.5		
		September 21, 1922	−17	66	48.0		No myxedema
		1903				Double oophorectomy	
21	Exophthalmic goiter	April 24, 1920	+27	96	57.0		Moderate thyrotoxicosis about 6 months. Goiter +. Exophthalmos +. Tremor +. Lost 35 pounds in 6 months
		April 29, 1920				*Subtotal thyroidectomy*	
	Mrs. M. R.						
	Age 44	May 17, 1920	+9	100	56.5		Very well since operation
	Lab. No. 596	October 26, 1921	−17	81	79.0		No myxedema

nently at a low level without medication. In 3 others, iodine was administered throughout most of the period of observation, thus rendering the permanency of the low metabolism somewhat uncertain. In this group of 17 patients there are 6 who, shortly after treatment, had signs and symptoms either suggestive of or actually diagnostic of myxedema. These manifestations were only transient. At present, without medication, these patients show no evidence of myxedema, although they have low metabolic rates. They therefore have been listed as cases of permanent low metabolism without myxedema. There are in addition to these 17 cases 4 others, not on iodine, whose low metabolism determinations were too few to establish their permanency, but on account of the considerable length of time they were observed after treatment they probably belong to this group.

Table 1 gives an outline of the basal metabolic and clinical histories on those of the 21 cases on whom the data is not charted.

Time of onset

In 7 cases the onset of the permanent low metabolism was within 3 months after treatment: in 4 cases it was within 3 months to 1 year, and in 3 cases it was over 1 year after treatment. In 7 cases the time of onset was unknown.

Absence of myxedema

The main issue with regard to permanent low metabolism is whether it is normal or abnormal for the patients concerned. This must be decided upon the basis of the accompanying clinical picture. The striking thing about the patients presented in this study was the absence of signs and symptoms of myxedema, as illustrated by a typical case history:

Case 3[5] *(fig. 1)*. Lab. No. 819. Age 44 in 1920. In 1912 she developed typical exophthalmic goiter, for which a partial thyroidectomy was performed in another hospital in March, 1913. After operation her symptoms were exaggerated, but

[5] The early data on this and 3 other cases have been reported before as follows:
Cases 3 and 12: Holmes, Am. Jour. Roent., 1921, viii, 730; and Means and Holmes, Arch. Int. Med., xxxi, 303, 1923.
Case 4: Holmes, ibid.
Case 5: Richardson and Means, Arch. Surg., 1924, ix, 237.

began to improve about 2 years later. In December, 1920, when first seen in this hospital, her basal metabolic rate was plus 50 per cent and her pulse 100. Her thyroid was diffusely enlarged. She had slight exophthalmos, marked tremor, palpitation and dyspnea. She was hoarse, her mucous membranes were slightly pale, her skin was dry and she had brown pigmentation of hands, axillae, groins and genitals. The diagnoses were post-operative exophthalmic goiter and cardiac decompensation. She was given 5 x-ray treatments during the period December, 1920, to March, 1921, and her basal metabolic rate dropped to normal. In October she was reported as having done well all summer. In November, 1921, there was some recurrence of thyrotoxicosis with a basal metabolic rate of plus 31 per cent. Three more x-ray treatments were given November, 1921, to January, 1922. Her basal metabolic rate again dropped to normal and she was symptom-free.

She felt well until September, 1924, when she developed symptoms from uterine fibroids, for which she had a hysterectomy and double salpingo-oophorectomy performed during the same month. No metabolisms were done between January, 1922, and April, 1925. On the latter date, 3 years after her x-ray treatment for toxic goiter and 6 months after her pelvic operation, her basal metabolic rate was minus 21 per cent, and continued at about this level during the following two months. She was prevailed upon to enter the hospital for study. She had gained 3 kgm. since 1922. Her hair was slightly dry and her skin was pigmented, as previously described when she had thyrotoxicosis in 1920. Her tongue was not particularly thick. Her memory was good. Her appetite was good. Her speech was rather slow, but the patient said this was natural for her. There was no abnormal weakness or fatigue, no marked palpitation or dyspnea on exertion, no headaches and no dizziness. The palms and soles of her feet sweated profusely. Her ankles swelled only if on her feet all day. She had a large dilated heart. There was no clinical evidence of myxedema. She felt well and insisted that she did not need to be in a hospital.

She was put on thyroid extract, 6 grains daily. In 5 days' time her rate was minus 17 per cent. Thyroid was decreased to 3 grains daily, on which dose her metabolism remained practically unchanged. Thyroid was increased to 6 grains daily on several subsequent occasions, but each time caused palpitation, sweating, insomnia and hot flashes, and had to be decreased. There was no significant effect on metabolism, and no clinical improvement occurred. Her basal metabolic rate remained at about minus 21 per cent up to January, 1926. Thyroid was omitted in February, 1926. In November, 10 months later, her rate was minus 25 per cent. She looked and felt exceptionally well. She was strong, not easily fatigued and not sensitive to cold. Her appetite was very good. Her hair was slightly dry. Her speech was somewhat slow but not thick. There was no edema. In short, there was no evidence of hypothyroidism. Thyroid extract, 3 grains daily, was started again. She took it for about one month with no clinical results, except that she experienced occasional hot flashes. In April, 1927, when 4 months

off thyroid, she was apparently quite normal, with no symptoms of myxedema, although her metabolism was minus 24 per cent. She stated that she had hot flashes only when on thyroid. Ovarian extract, grains V, three times daily, was given although there was no indication for it clinically. It produced no change.

Most of our cases were like the one just presented during the period of their low metabolism, e.g., figures 2 and 3. They were alert, ener-

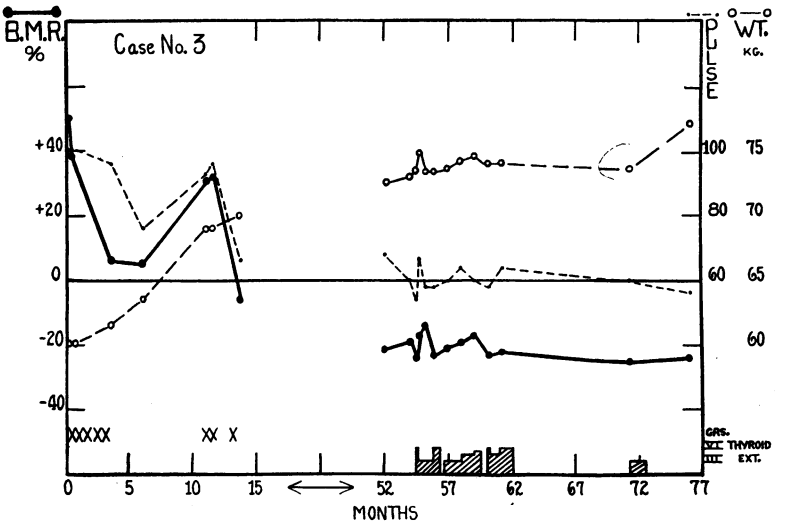

FIG. 1. MRS. A. A., AGE 44. LAB. NO. 819. PERMANENT LOW METABOLISM WITHOUT MYXEDEMA, FIRST OBSERVED THREE AND ONE-QUARTER YEARS AFTER TERMINATION OF X-RAY TREATMENT (*X*), AND TWELVE YEARS AFTER HEMITHYROIDECTOMY FOR EXOPHTHALMIC GOITER

Double oophorectomy performed 6 months before low metabolism was observed. Six grains of thyroid extract daily did not significantly affect the basal metabolism, but caused symptoms of thyroid intoxication. (In this and subsequent figures cross-hatched areas denote thyroid therapy.)

getic individuals who did not tire easily and were not sensitive to cold. Their skin was soft and smooth, there was no edema and their hair was not dry. Mrs. E. C. (case 2, table 1), for example, worked daily from 6.00 a.m. to 9.30 p.m. Besides doing all the housework in an eight-room house, she was able to take care of a small garden and do most of the work in connection with about 300 chickens, as well

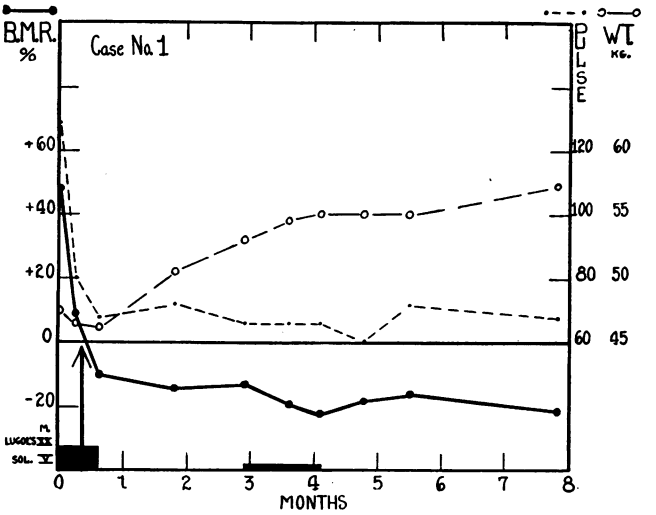

FIG. 2. MRS. A. G., AGE 42. LAB. NO. 4549. PERMANENT LOW METABOLISM WITHOUT MYXEDEMA FOLLOWING SUBTOTAL THYROIDECTOMY (ARROW) FOR EXOPHTHALMIC GOITER

The metabolism remained low after omission of iodine, in contrast to what happens in cases of temporary low metabolism, as shown in figures 4 and 5 in Part I of this study (1). (In this and subsequent figures, black areas denote Lugols' therapy.)

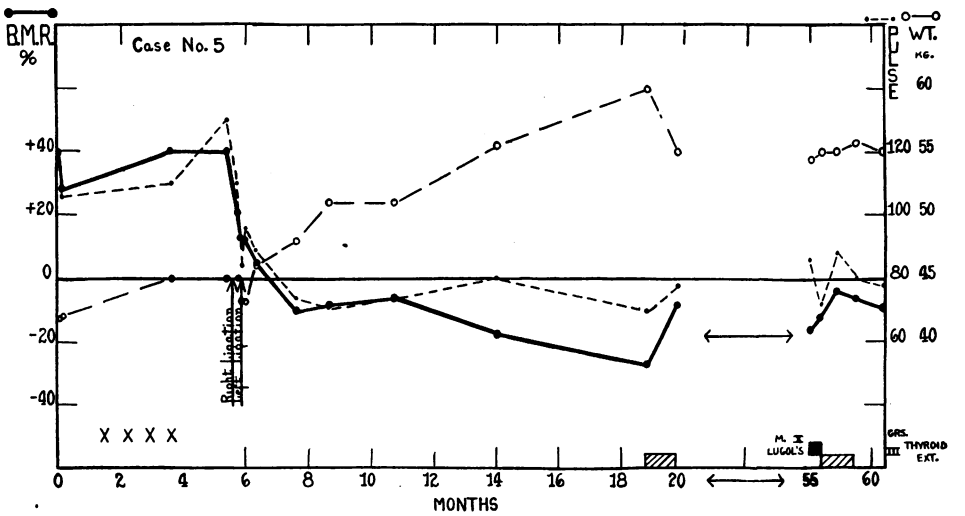

FIG. 3. MRS. E. W., AGE 22. LAB. NO. 1432. PERMANENT LOW METABOLISM WITHOUT MYXEDEMA FOLLOWING X-RAY TREATMENT (*X*) AND DOUBLE LIGATION FOR EXOPHTHALMIC GOITER

Thyroid therapy did not produce any clinical change

as serve on several church and civic committees. All these activities produced only slight fatigue, which was not increased by omission of thyroid extract. This medication in moderate doses raised her metabolism only slightly.

A few of the cases, viz., cases 4 (fig. 4), 7, 9, 10, 11 and 16, while they did not have myxedema, were slightly different from those just described.

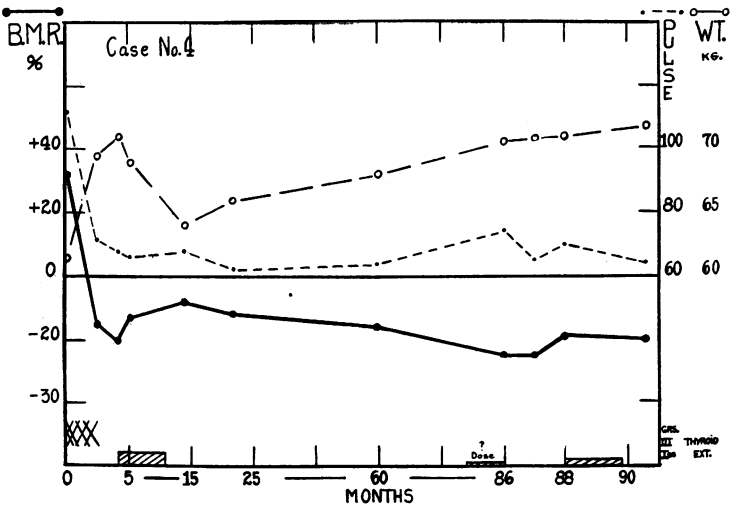

FIG. 4. MRS. M. H., AGE 37. LAB. NO. 575. PERMANENT LOW METABOLISM FOLLOWING X-RAY TREATMENT (*X*) FOR EXOPHTHALMIC GOITER

Question of mild myxedema during first 3 months after termination of treatment. No myxedema with a low basal metabolic rate, 2, 5, 7 and 7½ years after treatment.

Case 4 (fig. 4) is fairly typical of the group. At the time of her low metabolism, which ranged for the most part between minus 20 and minus 25 per cent, she was nervous and excitable, tired easily, and was sensitive to cold. Her skin, however, was soft and warm, her hair was not dry and there was no edema. She was definitely not myxedematous. Thyroid extract in doses of 1½ grains daily produced no effect on her metabolism, but, while taking it, she thought she tired a little less easily and was less nervous. No objective changes were discernible. The other patients of this type on whom thyroid

extract was tried felt a little better at times while taking it, but at other times no improvement was noted. This whole group resembled patients with very mild thyrotoxicosis rather than those with myxedema.

Relation of permanent low metabolism to some pathological entities other than myxedema

Myxedema is of course not the only abnormality associated with low metabolism. The latter may be observed for example in conditions of starvation, in chronic nephrosis, and in deficiency of endocrines such as anterior pituitary or adrenal cortex. These disorders were not factors in any of our cases.

A low metabolism is often attributed to hypogonadism because the two are sometimes found to be associated. Inasmuch as this idea is prevalent, those of our cases are cited below in which there was a history of either double oophorectomy or of the occurrence of the menopause. They appeared to be normal individuals and showed no symptoms known to be associated with ovarian deficiency.

Case 18 (table 1), had a thyroidectomy in February, 1919. Her metabolism was minus 10 per cent in 1920. A double oophorectomy was performed in January, 1922. The only metabolism determination thereafter was minus 19 per cent in August, 1923.

Case 21 (table 1) had a double oophorectomy in 1903 and a thyroidectomy in April, 1920. Her basal metabolic rate was plus 9 per cent in May, 1920, and minus 17 per cent in November, 1921.

Case 3 (fig. 1) had several x-ray treatments for toxic goiter ending December, 1921. In January, 1922, her basal metabolic rate was minus 6 per cent. A double oophorectomy was performed in September, 1924. There were no further determinations of metabolism until April, 1925, when her rate was minus 21 per cent.

Case 11 (see table 1) had x-ray treatment for toxic goiter ending September, 1922, when her basal metabolic rate was plus 11 per cent. The menopause occurred in May, 1925. No metabolism determinations were made until November, 1925, when her rate was minus 17 per cent.

In case 18 there was a tendency to a low metabolism before the oophorectomy was performed. In case 21 it is impossible to say

whether the oophorectomy had anything to do with the low rate. In cases 3 and 11 there is no record of the metabolism for about 3 years after x-ray treatment for thyrotoxcosis, so that it is not known whether it was low before the ovarian deficiency occurred. In short, in all these cases, one cannot preclude the possibility of a low metabolism which may have been present even before thyrotoxicosis occurred.

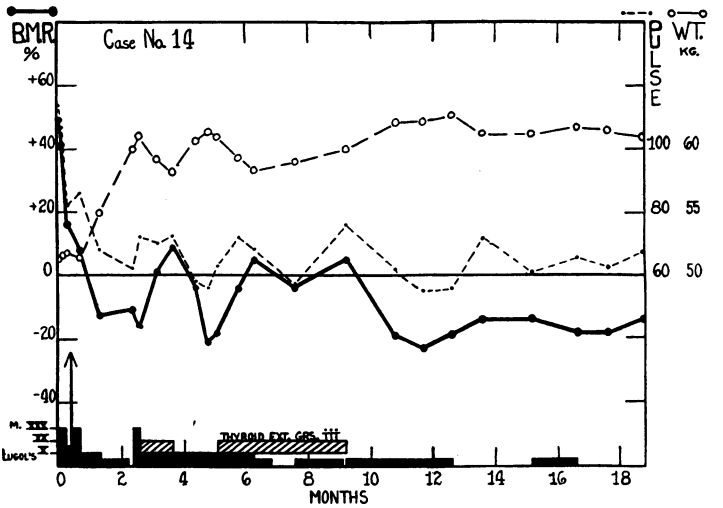

FIG. 5. MRS. A. G., AGE 24. LAB. NO. 3768. PERMANENT LOW METABOLISM FOLLOWING SUBTOTAL THYROIDECTOMY (ARROW) FOR EXOPHTHALMIC GOITER

No myxedema except mild temporary type during first 6 months after operation. The metabolism remained low after omission of iodine. Note well-marked rise in metabolism due to thyroid therapy, in contrast to the effect of thyroid shown in figure 1.

Influence of type of treatment employed for thyrotoxicosis

Permanent low metabolism without myxedema occurred spontaneously in 1 case: after subtotal thyroidectomy in 13 cases: after hemithyroidectomy in 2 cases: after surgery and x-ray combined in 2 cases: and after x-ray alone in 3 cases. Two patients (one treated by hemithyroidectomy and one by x-ray) had toxic adenoma; the remainder had exophthalmic goiter.

In 8 cases iodine was used at the time of subtotal thyroidectomy. In 5 of these 8 cases, although iodine was continued at intervals after operation, the data show that it was not the cause of their metabolic depression, as their metabolism remained low when iodine was omitted. In the other 3 instances (cases 15, 16 and 17; see table 1) iodine was continued practically throughout the period of the low metabolism. It is impossible to tell just what rôle this medication played in these instances until iodine is omitted for a sufficient length of time. They may be low rate cases of the temporary type (1), but are just as likely to be of the permanent type like case 14 (fig. 5), case 1 (fig. 2) and case 13 (table 1), in which the metabolism remained low after iodine was omitted.

It is evident that in the very great majority of instances of permanent low metabolism without myxedema, iodine therapy was not a factor which need be considered, in striking contrast to the important rôle it played in temporary low metabolism (1).

Permanent low metabolism without myxedema preceded by temporary low metabolism without myxedema. Possible relationship between the two

Having now presented cases both of temporary low metabolism without myxedema (1) and of permanent low metabolism without myxedema, following thyrotoxicosis, it seems desirable to consider a case that may furnish a connecting link between the two, viz., one which shows both types of low metabolism. Such a case is no. 18 listed in the paper on temporary low metabolism. Her chart is given here (see fig. 6) because this seems to be the logical place for it. She had 3 x-ray treatments for exophthalmic goiter. One month later, without medication, her metabolism was minus 17 per cent. Thyrotoxicosis recurred, with a rate of plus 22 per cent. She had 2 more x-ray treatments, and one month later, without medication, her rate was minus 17 per cent again. There was no hypothyroidism either time. Her metabolism then rose without thyroid therapy to standard normal (ranging from minus 2 to minus 7 per cent) for a period of 14 months, during which time she displayed no clinical evidence of thyrotoxicosis. Her rate then dropped spontaneously to approximately its former low level, without any accompanying hy-

pothyroidism. Thyroid extract, up to 3 grains daily, was prescribed. This affected her metabolism very little and produced only very questionable clinical improvement. On omission of this medication (for 7 months at the date of her last test) her rate was, with one exception, about minus 15 per cent. She claimed that she felt just as well, if not better, than when on thyroid extract, and appeared to be a perfectly healthy individual.

The significance of this case will be elaborated in the discussion.

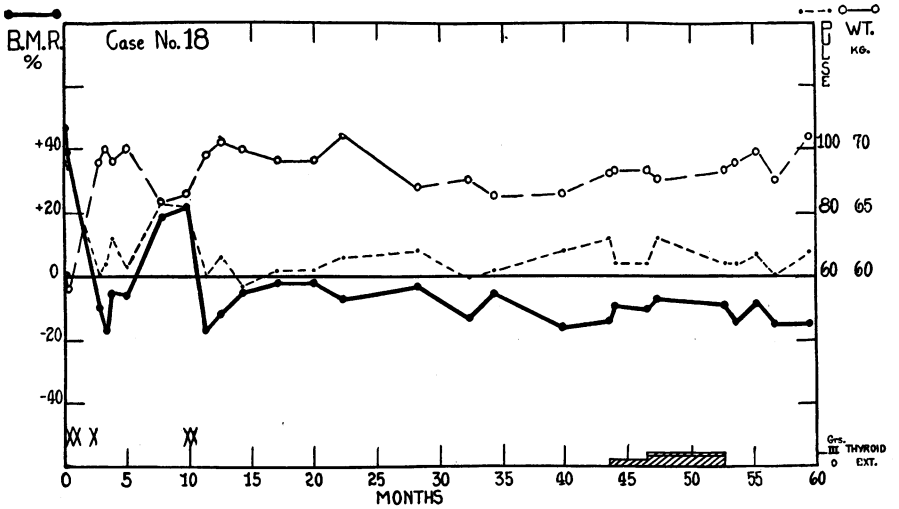

FIG. 6. MISS L. B., AGE 23. LAB. NO. 1510. TEMPORARY LOW METABOLISM WITHOUT MYXEDEMA FOLLOWED BY PERMANENT LOW METABOLISM WITHOUT MYXEDEMA, OCCURRING AFTER X-RAY TREATMENT (*X*) FOR EXOPHTHALMIC GOITER

DISCUSSION

The fact that a permanent low basal metabolic rate may be normal for some individuals, does not seem to be very clearly recognized. There are healthy people who have a metabolism as low as minus 16 to minus 25 per cent. They look and feel perfectly well, and display no symptoms of hypothyroidism. Sturgis (3) reports such a case with a rate of minus 22 to minus 25 per cent. Wishart (4) reports 3 cases with rates of minus 16, minus 21 and minus 21 per cent respectively. We have seen several such cases in our experience.

They differ in no apparent respect from healthy people with a standard normal metabolic rate.

Then again there are those individuals with a low metabolism who have symptoms such as headaches, dizziness, weakness, chronic fatigue and nervousness. In fact the nervousness is often so marked that they suggest mild hyperthyroidism rather than hypothyroidism. They do not *look* myxedematous in the least. The hair and skin are usually not dry. There is no edema. They are not slowed up. One of their chief complaints is that of feeling tired all the time. In this respect only, they suggest myxedema. In general, they present an entirely different picture. Were it not for their low metabolism and susceptibility to fatigue, thyroid extract would rarely be tried on them. There are many persons whose metabolism is within the standard normal zone of plus or minus 10 per cent who have the same complaints, yet because their metabolism is at this higher level and no definite clinical pathology is found, they are often regarded as more or less normal, or at most "neurotic." Certain observers have reported patients of this type, who have a low metabolism, as improved on thyroid therapy and have on this basis diagnosed the condition as a form of hypothyroidism, even though the symptoms are on the whole quite atypical of true thyroid deficiency. It seems much more likely that the explanation lies in some other obscure pathology about which nothing is known at present. The low metabolism often found may be an essential part of this picture. On the other hand, it may be "normal" for the particular individual, just as in the group of cases mentioned in the foregoing paragraph.

Coming now to permanent low metabolism *following thyrotoxicosis*, we know that in a certain proportion of cases it is associated with definite underfunction of the thyroid gland—true myxedema. In another much larger proportion of cases, however, there are no symptoms of myxedema. The patients are of the two types just described, the majority of them being of the first type, i.e., apparently normal individuals. Inasmuch as a surprising number of normal people who have never had thyrotoxicosis have a low metabolism, it is only reasonable to suppose that in a group of patients who develop toxic goiter, a certain number have normally a low metabolism which manifests itself as soon as the thyrotoxicosis disappears. In

fact Plummer (5) suggests that perhaps more cases of exophthalmic goiter are recruited from the group of persons with a low metabolism—many of whom are of the asthenic type—than from any other group. He actually has observed patients with a low basal metabolic rate who later developed exophthalmic goiter.

As previously stated, abnormalities other than myxedema that are at present known to be associated with a low metabolism, were not factors in our cases, with the exception of hypogonadism. The possibility of the influence of this factor must be considered in four cases already described. The knowledge of the effect of ovarian deficiency per se on metabolism is very ill defined, although the occurrence of myxedema about the time of the menopause is well known. King (6) from his own observations and DuBois (7) from a review of the literature, have come to the conclusion that loss of ovarian function has little effect on metabolic rate. Several others, notably Aub (8) and Bailey (9), have concluded, both from the literature and from their own observations, that experimental oophorectomy sometimes depresses the basal metabolism. In the instances where a low metabolism is found after removal of the ovaries or the occurrence of the menopause, the possibility of its having been present before the ovarian deficiency developed, and of its representing the normal metabolic level of the patient, never seems to be taken into consideration. Inasmuch as our four patients appeared to be healthy individuals, we feel that it is just as reasonable to assume that their low metabolism was "normal" for them as to assume that it was due to an ovarian deficiency.

In brief, it is suggested that permanent low metabolism without myxedema following toxic goiter may be a manifestation of any one of the following:

1. A return to a low metabolism which is normal for the individual and which was probably present before the development of thyrotoxicosis, the patients concerned being healthy persons.

2. A return to a low metabolism which was probably present before thyrotoxicosis developed and which may be "normal" for the individual, the patients concerned, however, having symptoms such as headaches, dizziness, susceptibility to fatigue, nervousness, etc.

3. Intervening abnormalities other than myxedema, that are known to be associated with low metabolism.

In group 1 thyroid therapy is a useless procedure, except as a therapeutic test to establish beyond doubt the type of patient with which one is dealing. When this is accomplished, it should be omitted. Otherwise the patient may continue taking thyroid extract unnecessarily for years, and even may be made quite uncomfortable at times from efforts to raise the metabolism to standard normal by giving large doses. In view of the possible benefit in group 2, thyroid therapy may be tried on these patients. It is difficult to believe, even in cases which may improve under such treatment, that the underlying pathology is primary under-function of the thyroid. It should be stressed that in patients of this type sufficient thyroid to produce improvement often causes very little increase in metabolism. In group 3 the most effective treatment is that which is directed at the underlying cause of the patient's clinical symptoms. Thyroid therapy is not a routine procedure, but is said to be of value in certain rare instances, e.g. when used in large doses in order to reduce the edema of chronic nephrosis.

The idea is all too prevalent that when the metabolism is below accepted normal standards there must be a depressed function of the thyroid. Consequently when a situation arises where the circumstances are supposedly more propitious than usual for the occurrence of myxedema, viz., after a subtotal thyroidectomy or after several x-ray treatments of the thyroid, such a diagnosis is doubly likely to be made, regardless of the clinical picture. This is as reasonable as saying that because the blood pressure is high, the patient must have chronic nephritis. We should not stress this point were it not for the fact that wrong diagnoses are so common, evidently due to the implicit belief that there is a practically perfect correlation between myxedema and low metabolism. This is the result of thinking of the normal in terms of averages and not taking individual variations sufficiently into account. The need for broadening our ideas of what is normal, to allow for occasional variations, applies to many other phases of medicine besides basal metabolism.

Those who report benefit from thyroid therapy in low rate cases without myxedema, mention that in some instances they were not successful. Our experience in this clinic has been that most of our low rate cases without myxedema have received no benefit from

thyroid medication. One or two have had headaches relieved. A few others thought they felt brighter and a little more energetic at first, although several months after omission of thyroid many of them felt just as well as ever and had no desire to resume the tablets.

The explanations offered for the lack of effect in these instances indicate the current attitude toward the status of the patient. The inference is made that it is due to such various causes as the long-standing nature of the symptoms, with complicating arteriosclerosis (10), lack of absorption from the gastro-intestinal tract (3), loss of potency of the thyroid preparation (3), or insufficiency of dosage (11). Practically no consideration is given to possible biological differences in patients themselves. Several of our cases in which thyroid had no clinical effect were young people who did not have any arteriosclerotic changes. In many others we are sure that potency, absorption and dosage were not at fault, because the metabolism rose to standard normal, whether with varying doses of thyroid extract by mouth or with intravenous thyroxin—yet there was no improvement clinically. That is to say, we got metabolic but not clinical results. In some instances where we did not get even a significant effect on metabolism, we are sure that the thyroid extract was absorbed, because symptoms of thyroid intoxication supervened whenever the dose was increased beyond the patient's tolerance. For example, case 3 (fig. 1) with a metabolism of about minus 23 per cent, suffered from precordial pain and palpitation whenever her dose was increased to 6 grains daily in spite of the fact that her rate remained practically unaffected, i.e., she developed some symptoms of thyroid intoxication with a distinctly subnormal rate. A case dealt with in the paper on myxedema following thyrotoxicosis (2) is somewhat similar in this respect. He developed myxedema a few months after a subtotal thyroidectomy in 1918. Thyroid has never been omitted long enough in the subsequent 9 years to observe whether he still has thyroid deficiency. It would seem at present that, regardless of myxedema, he has normally a low rate in the neighborhood of minus 15 to minus 25 per cent, because he felt perfectly well and his metabolism remained within this range although he was on 6 grains of thyroid daily. This is more than sufficient to raise the metabolism to normal in most cases of myxedema: in fact many show symptoms of thyroid intoxication on

this dose. When he took the large dose of 12 grains daily, his rate rose to minus 2 per cent, but he experienced severe precordial pain and palpitation which disappeared promptly on omission of thyroid, i.e., sufficient thyroid to make his metabolism standard normal produced symptoms of thyroid intoxication.

In general, patients with low metabolism and no myxedema may be divided into two groups with respect to the effect of thyroid extract by mouth on metabolic rate:

1. Those who respond readily to moderate doses with a well-marked rise in metabolism, e.g., case 14 (fig. 5).

2. Those who require large doses to affect the metabolism perceptibly. Moderate doses do not produce any very significant change, e.g., case 3 (fig. 1).

The latter type is fairly common, and in this group, symptoms of thyroid intoxication may supervene with a definitely sub-normal rate. As a general rule, the patient who requires more than 4 grains of thyroid daily to raise the metabolism to standard normal has either not got myxedema, or else if he has that deficiency, has in addition a low rate normally. The maintenance dose for most cases of true myxedema seen in this clinic is $1\frac{1}{2}$ to 3 grains of thyroid extract (Armour's) daily.

Treating the metabolism rather than the clinical symptoms appears to be a prevalent practice. The folly of flooding the body with an excess of a normal product is obvious, and it is very poor therapy.

Since several of our cases of temporary low metabolism without myxedema (1) seem to be best accounted for by the same hypothesis that we have elaborated above for our cases of permanent low metabolism without myxedema, it seems important to consider any possible relationship that may exist between the two. If both types of low metabolism represent depressions to a metabolic level which is normal for the individual, the logical conclusion is that several of the patients who had temporary low metabolism without myxedema may eventually develop permanent low metabolism without myxedema. Case 18 from study I (fig. 6 in this study) thus may be regarded as a connecting link between these two types of low metabolism. In such an instance the intervening period of standard normal metabolism evidently represents a period of mild thyrotoxicosis.

These considerations lead up to a fairly important deduction, which is supported by actual observation, viz., that thyrotoxicosis may exist with a standard normal metabolism. This accounts satisfactorily for the repeated depressions of the metabolism from a standard normal to a low level produced by iodine months to years after operation, coincident with the disappearance of signs and symptoms either diagnostic of, or suggestive of, mild thyrotoxicosis (1).

It follows that in order to judge the true elevation of the metabolic rate of patients who develop thyrotoxicosis, it is necessary to know their normal metabolic level. The clinical significance of this point may be illustrated by referring to case 8 (table 1). When first seen, this patient had definite signs and symptoms of thyrotoxicosis with a rate of plus 10 per cent, but because her metabolism was standard normal, she was given no treatment. In view of her low rates of minus 28 and minus 14 per cent, 2 and 3 years respectively after operation, when she was not receiving any medication and was in perfectly good health, her level of plus 10 per cent in 1920 was probably equivalent to plus 24 or plus 38 per cent in a person with a normal level of about zero.

SUMMARY

Twenty-one cases have been presented showing permanent low metabolism without myxedema, following recovery from thyrotoxicosis.

After recovery, the patients were for the most part apparently normal individuals.

In the cases in which thyroid therapy was tried, no definite beneficial effect was noted clinically, although potency, absorption and dosage of the drug were not at fault.

Many of these patients required much more thyroid extract to raise the basal metabolism to standard normal than do patients with spontaneous myxedema. Moreover, symptoms of thyroid intoxication due to thyroid feeding sometimes occurred while the metabolism was still low.

The importance of treating the patient rather than the basal metabolic rate has been stressed.

CONCLUSIONS

Facts are cited which are consistent with the hypothesis that permanent low metabolism without myxedema following thyrotoxicosis, is, in most instances, a return to a normal metabolic level, which was probably low even before the development of the disease.

Thyrotoxicosis may exist with a standard normal basal metabolic rate.

The interpretation of the degree of basal metabolic elevation in thyrotoxicosis is directly affected by the level of the patient's normal metabolism.

Temporary low metabolism without myxedema may be, in some instances, an initial phase in the eventual development of permanent low metabolism without myxedema.

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