The Bryson symptom in exophthalmic goître : with a report of forty cases / by Hugh T. Patrick.

THE BRYSON SYMPTOM IN EXOPHTHALMIC GOITRE.*

WITH A REPORT OF FORTY CASES.

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Five years ago Dr. Louise Fiske Bryson† first called attention to a condition in Graves's disease that has since been called the Bryson symptom—viz., diminished chest expansion or vital capacity. She cites it as one of the "distinctive, fixed conditions" prevailing in this disease which "afford the only rational basis for prognosis." Other fixed conditions are not specified and no measurements or cases are given. Dr. Græme M. Hammond‡ considers it "of the greatest importance in regard to the prognosis" and "also of assistance in locating the seat of the lesion." He says further: "Dr. Bryson states that where the expansion is found to be reduced to half an inch or less the termination of the case is invariably fatal." He found the symptom present in the eight cases reported in his paper, and as recovery took place the expansion increased. Dr. A. B. Pope# has reported a case of exophthalmic goître in a man with chest expansion of an inch and a half, and in the discussion Dr. E. Le Fevre said the symptom was "owing to some nervous influence similar to that which gave rise to the rapid heart action."

Three years after her first paper Dr. Bryson‖ resumes the theme, giving particular prominence to the respiratory symptoms, and emphasizing especially diminished chest expansion, but she seems to have receded somewhat from her former positive position as to its pathognomonic importance, as of twenty cases the Bryson symptom was present in only thirteen. She considers the disease, however, to be "a disorder of nutrition *respiratory in its first manifestations*,△ and even makes this assumption the foundation of a therapy consisting of a series of respiratory gymnastics executed by means of Taylor's respirator. These views have been quoted at home and abroad,* and the Bryson symptom would seem to be about to take its place among the important signs of Graves's disease as bearing upon the diagnosis, prognosis, pathology, and treatment. Hammond,† indeed, distinctly calls it the fourth cardinal symptom, a distinction, by the way, which belongs to Marie's symptom—tremor.

At the time when Dr. Bryson's first article appeared I had under observation a case of exophthalmic goître which I measured several times, finding the chest expansion not materially diminished. After this I measured a number of cases, finding the expansion sometimes diminished and sometimes about normal, and I presently noticed that diminished expansion seemed to go with a diminution in general vitality and muscular strength. I finally, then, began comparing the chest expansion with the hand grasp as recorded by the dynamometer, this affording the most convenient, if only approximately accurate, index of the general muscular condition. It is the result of these measurements that I wish to present. I may premise that only well-developed, in a measure typical, cases are comprised in this report, no *formes frustes* being included, and that the diagnosis was in every instance confirmed by some well-known neurologist. I have therefore deemed it unnecessary to lengthen my paper by giving the symptoms of each case in detail. With the exception just noted, the cases have not been selected, but embrace all that I was able to measure from November 1, 1892, to May 1, 1894.

The patients were all women, and it may be worthy of note that of some sixty cases seen since 1891 only two were in men.

* Read before the Chicago Medical Society, October 1, 1894.

† A Preliminary Note on the Study of Exophthalmic Goître. *N. Y. Med. Jour.*, December 14, 1889.

‡ A Contribution to the Study of Exophthalmic Goître. *N. Y. Med. Jour.*, January 25, 1890.

N. Y. Med. Jour., April 19, 1890.

‖ Exophthalmic Goître: A View of Thirty Cases. *The Post-graduate*, July, 1892.

△ Italics mine.

* Frank S. Johnson, *Trans. Ill. State Med. Soc.*, 1893, p. 193; J. Madison Taylor, *Med. News*, 1893, pp. 673, 711; William Oliver Moore, *Internat. Clinics*, 1893, i, p. 92; Edmund Lee Tompkins, *Amer. Jour. of Obstet.*, November, 1893; W. H. Thomson, *N. Y. Med. Jour.*, June 3, 1893; William A. Hammond, *Dis. of the Nerv. Syst.*, 1891, p. 790; Landon Carter Gray, *Treat. on Nerv. and Ment. Dis.*, p. 551; Mannheim, *Der Morbus Gravesii*, Berlin, 1894, p. 33; Oppenheim, *Lehrb. der Nervenk.*, Berlin, 1894, p. 829; Grasset et Rauzier, *Mal. du syst. nerv.*, Montpellier and Paris, 1894, ii, p. 302; Debove et Achard, *Manuel de méd.*, Paris, 1894, art. by Létienne, vol iv, p. 397; Blocq, *Études sur les mal. nerv.*, Paris, 1894, p. 173.

† Græme M. Hammond, *loc. cit.*

Table I.—GRAVES'S DISEASE.

No.	Age.	Duration of disease.	Date of examination.	Chest expansion.	Average.	Dynamometer. R.	Dynamometer. L.	Dynamometer. Average.	Remarks.
				Centimetres.					
1	29	4½ years.	Nov. 1, 1892.	4½	....	40	30	.....	Following fright.
			Dec. 8, 1892.	4½	....	47	40	.....	
			Feb. 25, 1893.	4	....	46	37	.....	
			Mar. 2, 1893.	4½	4·31	50	45	41·8	
2	30	2 years.	Nov. 1, 1892.	5½	....	55	45	....	
			Feb. 17, 1893.	4	4·75	60	57	54·2	
3	32	4 years.	Nov. 30, 1892.	1¾	....	2½	2½	.....	At last examination she had been two weeks confined to bed, following mental worry. May 17th, much better; had gained over twenty pounds.
			Dec. 14, 1892.	1¾	....	12	16	.....	
			Dec. 21, 1892.	2¼	....	26	16	.....	
			Jan. 28, 1893.	1¼	....	..	..	....	
			May 17, 1893.	2	1·8	16	16	10·7	
4	37	1 year.	Nov. 9, 1892.	3	....	37	47	.....	Is right-handed, but has always been stronger in left.
			Nov. 30, 1892.	3¼	3·25	40	50	43·5	
5	31	Probably 4 years.	Oct. 15, 1892.	7	....	67	47	.....	Much worse since a confinement nine weeks ago.
			Dec. 2, 1892.	5½	6·25	49	48	52·75	

No.	Age.	Duration of disease.	Date of examination.	Chest expansion.	Average.	Dynamometer.			Remarks.
						R.	L.	Average.	
				Centimetres.					
6	23	1 year.	Feb. 20, 1893.	4½	4·5	22	15	18·5	
7	17	1½ year.	Nov. 19, 1892.	5	5	65	55	60	
8	40	12 years.	Nov. 1, 1892.	4	....	30	25	.....	Goitre from youth.
			Jan. 5, 1893.	3½	3·75	30	18	25·75	
9	55	8 years.	Nov. 2, 1892.	3½	3·5	37	30	33·5	No treatment the past year, as treatment the two preceding years almost cured her.
10	35	8 years.	Nov. 3, 1892.	2	2	20	10	15	Much worse since influenza two years ago.
11	30	2 years.	Nov. 9, 1892.	6	6	67	63	65	Developed after hemiplegia. Cerebral apoplexy.
12	21	3 years.	Nov. 12, 1892.	7	7	60	50	55	
13	18	2 years.	Nov. 14, 1892.	6½	6·5	50	50	50	
14	28	4 months.	Nov. 12, 1892.	5½	5·5	44	44	44	
15	51	4½ years.	Nov. 16, 1892.	5½	....	45	55	.....	Locomotor ataxia for the last ten years.
			Mar. 2, 1893.	5	5·25	42	40	45·5	
16	24	1 year.	Nov. 19, 1892.	5	....	30	45	.....	Stigmata of hysteria; right hand excluded from estimate, as low figure is due to psychic inhibition.
			Dec. 14, 1892.	4	4·5	3	62	45·6	
17	19	3 years.	Nov. 22, 1892.	3½	3·5	45	45	45	
18	26	9 months.	Nov. 29, 1892.	4	....	62	57	.....	
			Dec. 14, 1892.	3½	....	62	55	.....	
			Jan. 10, 1893.	4	3·83	67	53	59·33	
19	35	1 year.	Dec. 7, 1892.	4	4	62	56	59	Was much worse eight months ago; all symptoms were more pronounced.
20	48	2 years.	Dec. 20, 1892.	5	5	15	10	12·5	
21	20	9 months.	Dec. 20, 1892.	4	4	47	37	42	
22	21	Uncertain,	Jan. 5, 1893.	4½	....	48	47	.....	Patient thinks an operation on the nose for
		1 to 4 years.	Jan. 20, 1893.	3½	....	50	35	.....	catarrh aggravated the disease.
			Feb. 16, 1893.	5	....	47	37	.....	
			Mar. 2, 1893.	5	....	42	40	.....	
			Apr. 27, 1893.	4½	....	40	30	.....	
			July 10, 1893.	4½	4·45	35	35	40·5	
23	39	8 years.	Jan. 10, 1893.	3½	....	60	60	.....	
			Feb. 17, 1893.	3½	3·5	50	50	55	
24	25	.....	Jan. 13, 1893.	3	3	16	16	16	Has had goitre ten years.
25	38	4 years (?).	Feb. 16, 1893.	4	4	37	33	35	Nervous and irritable four years. All symptoms of Graves's disease five weeks, following failure in business.
26	21	3 years.	Feb. 18, 1893.	6½	6·5	30	20	25	Complicated with hysteria.
27	30	6 months.	Feb. 22, 1893.	4½	....	67	67	.....	Has *imperative ideas*. March 2d, feels much
			Mar. 2, 1893.	4½	4·5	85	85	76	better.
28	29	4 years.	Feb. 24, 1893.	5	5	71	59	65	Symptoms aggravated by operation on uterus two years ago.
29	37	7 months (?).	Mar. 1, 1893.	5½	5·5	65	60	62·5	Difficult labor seven months ago, since which time is nervous and anæmic; is now two months pregnant; all symptoms of Graves's disease six weeks.
30	..	2 years.	Mar. 3, 1893.	3	3	60	57	58·5	
31	34	11 months.	Mar. 9, 1893.	4½	....	60	57	.....	Following confinement eleven months ago.
			Mar. 20, 1893.	5½	5	50	50	56·75	
32	23	2 months.	Mar. 15, 1893.	4	....	37	30	.....	
			June 13, 1893.	3½	3·75	32	30	32·25	
33	38	4 years.	May 17, 1893.	3½	3·5	45	45	45	Following a fright; sister has same disease, and is much worse.
34	38	3 years.	May 12, 1893.	5½	....	43	43	.....	Following influenza; thyreoidectomy thirteen
			May 22, 1893.	4½	5	42	45	43·25	months ago, with great improvement.
35	29	4 to 5 years.	May 12, 1893.	4½	....	57	63	.....	
			July 10, 1893.	5	4·75	55	65	60	
36	46	2 years.	Apr. 23, 1893.	4½	4·5	50	47	48·5	
37	18	2 years.	Nov. 17, 1893.	4	....	50	40	.....	Following fright.
			Jan. 18, 1894.	2	....	66	45	.....	
			Jan. 30, 1894.	3½	3·16	32	37	45	
38	26	2 years.	Dec. 5, 1893.	2	....	55	50	.....	
			Jan. 19, 1894.	2½	....	55	55	.....	
			Jan. 30, 1894.	2	2·16	40	45	50	
39	39	3 years.	Jan. 1, 1894.	3	....	50	45	.....	Epilepsy; much worse the last three years.
			Jan. 18, 1894.	3	....	40	38	.....	
			Jan. 30, 1894.	3	3	32	27	38·66	
40	29	2 years (?).	Feb. 12, 1894.	4	....	30	25	.....	After inception cured (?) by rest, etc.; recurrence one year ago.
			Feb. 24, 1894.	4	....	28	12	.....	
			Mar. 6, 1894.	4½	....	35	23	.....	
			Mar. 31, 1894.	4½	....	40	32	.....	
			Apr. 13, 1894.	4	4·2	35	25	28·5	
					4·3	..	..	43·75	
Diminution					0·5	..	..	12·61	
Diminution, per cent					10½	..	..	22⅔	

It is a matter of common remark that no two observers take the chest expansion exactly alike, so that figures obtained by different persons can not be compared with confidence. To form a safe basis for comparison, therefore, I took the chest expansion and dynamometric measurement of twenty-eight women who came to the dispensary for

various ailments, selecting those whose troubles would not, in my opinion, affect the result. These are presented in Table II.

TABLE II.—*For Comparison.*

No.	Age.	Affection.	General condition.	Chest expansion.	DYNAMOMETER.		
					R.	L.	Average.
				Cm.			
1	52	Tabes.	Fair.	4	30	25	27·5
2	24	Hysteria.	Very good.	7	80	70	75
3	31	"	Good.	5	45	45	45
4	51	Chronic rheumatism.	Fair.	4	40	25	32·5
5	41	Incipient tabes.	Good.	5½	55	55	55
6	29	Cerebro-spinal syphilis; dement. paral. (?)	Good; very muscular.	5	100	75	87·5
7	30	Epilepsy.	Good.	3½	50	50	50
8	46	Ophthalmoplegia.	"	7	47	47	47
9	23	Brach. neuralgia.	Very good.	4½	80	70	75
10	43	" "	Fair.	6	72	70	71
11	39	Chronic rheumatism.	Very fair.	5½	60	40	50
12	18	Sciatica.	Fair.	4½	55	50	52·5
13	26	Neurasthenia.	Very fair.	3¾	65	50	57·5
14	38	Hysteria and neuralgic pains.	Fair.	4½	58	42	50
15	17	Hystero-epilepsy.	Good.	5	55	47	51
16	28	Lumbago.	Very good.	6	95	95	95
17	36	Hysteria.	Fair.	4	30	30	30
18	22	"	Good.	4¾	46	46	46
19	34	Hysteria and neuralgia.	"	6	75	70	72·5
20	22	Hysteria and organic heart disease.	Fair.	4½	37	50	43·5
21	30	Headache.	Anæmic.	4	75	70	72·5
22	30	Tabes.	Very good.	5½	80	67	73·5
23	15	Hysteria.	Good; well developed.	4½	45	35	40
24	19	Valvular heart disease.	Anæmic.	4½	51	55	53
25	27	Headache.	.....	4½	50	40	45
26	41	Hysteria.	Good.	4½	61	54	57·5
27	23	Hysteria and lumbago.	Very good.	4	47	47	47
28	26	Sciatica.	Good.	6	77	75	76
				4·8	..	..	56·36

I am well aware also of the varying results given by the dynamometer, and I do not allege absolute accuracy for my figures or wish to make too sweeping deductions from them, but all measurements were taken by myself in as nearly a uniform manner as possible, and sources of error excluded as well as might be.

It will be seen that the average chest expansion in the forty cases of exophthalmic goitre is 4·3 centimetres and the hand grasp 43·75 kilogrammes; in the twenty eight other cases, 4·8 centimetres and 56·36 kilogrammes respectively—that is, the expansion in Graves's disease is diminished half a centimetre, or ten and a half per cent., the hand grasp 12·61 kilogrammes, or twenty-two and two fifths per cent. In other words, the grasp is diminished more than twice as much as the expansion. It may be contended that the average expansion of the twenty eight women used for comparison is not up to the normal, and I think this may possibly be true, but I also think they make a far better basis for comparison than statistics of measurements taken by some one else whose methods would doubtless differ in some degree from mine, and consequently whose results, other things being equal, would differ from mine. Further, these measurements were made in the same dispensary which furnished the majority of the cases of Graves's disease,* and the subjects are fairly representative of the class of patients furnishing the material for this report, and, as before mentioned, the pathological conditions in these women were not such as to materially affect chest expansion or hand grasp. But even supposing that the expansion of the twenty-eight might be materially below the normal, we could not suppose in all these various cases a condition which would affect chest expansion to the exclusion of other muscular action, including hand grasp, and as we find that the hand grasp in Graves's disease is diminished twenty-two and two fifths per cent. and the expansion only ten and a half per cent., the natural conclusion would be that if one of these two is pathognomonic of this malady it would be the weakened grasp and not the diminished chest expansion, an assumption I would not for a moment entertain. Again, we find of the forty cases twenty-six with an expansion below the average of the twenty-eight, while thirty show a diminished grasp.

Could we take accurate dynamometric measurements of the lower extremities and of the pelvic muscles, I have no doubt we should find an equal falling off. Many of these patients complain of a sudden "giving way of the legs," and placed in the recumbent posture show their muscular weakness in the manner of rising.

A woman now under observation, not included in this report, is a case in point. It is not what could be called a very severe case; she does her own housework, gets about very well, hand grasp and chest expansion only slightly diminished, and yet she rises from the supine position much like a patient with idiopathic muscular atrophy; that is, she first raises the trunk by means of the arms till she rests on her elbows; then, again by help of the arms and with a peculiar wriggle or writhing movement to bring accessory muscles into play, she brings the trunk to the perpendicular; the erect position is then attained, with manifest effort, by turning round and pushing herself up with her hands first on the floor and then on her thighs or adjacent objects.

I think the diminished power of convergence often observed, as well as the occasional affection of the laryngeal muscles, is quite analogous to these other findings, and is simply a part of a general myasthenia, sometimes affecting one set of muscles more, sometimes another.

I find, further, that many of these cases show a rapid and marked falling-off in both chest expansion and hand grasp on repeated effort. Thus either may diminish one half after three or four trials in rapid succession, and in a general way the decrease affects both and is proportional to the debility.

The idea that the diminished chest expansion is simply part of a diminished vitality or energy is borne out by the history of individual cases. I have generally found that as the patient improves in general tone the expansion increases (and *vice versa*), although not necessarily in the same ratio, and that the grasp goes with it hand in hand. But as the diminished hand grasp shows the larger percentage in the table, so here it is apt to show the larger fluctuations, which would seem again to indicate that it is the more delicate index of the two. These facts are well illustrated by Case III. The rule, however, is not absolute, and

* Professor Mendel's *Poliklinik*, Berlin.

Case XXXVII is a striking exception in the first two examinations.

The only one of my patients (Case III) who ever showed an expansion as low as half an inch (1·25 ctm.), indicating, according to Dr. Bryson, an absolutely fatal termination, had, four months later, gained over twenty pounds in weight, and was better than at any time during the six months she was under observation.

I think, then, we may conclude that the Bryson symptom, although present in many cases of exophthalmic goitre, is in no wise pathognomonic of this affection, or even an important sign; that it has no special significance in relation to the prognosis, pathology, seat of the lesion, or treatment, and should be relegated back to the comparative obscurity of an individual in a large community of manifestations which all depend alike upon the general state; a state which makes the French designation of the disease, exophthalmic cachexia, quite as appropriate as any other.*

Venetian Building.

THE VALUE OF GUDE'S PEPTO-MANGAN IN THE TREATMENT OF ANÆMIA.

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The year 1893, with the publication of the results of very careful chemical investigations of the conditions of the blood in various diseases, especially those of the blood itself by Professor von Jaksch (1), the well-known author of the *Handbook on Clinical Diagnosis*, marks a new era in our understanding of the various anæmic processes. Our former vague knowledge of these conditions was molded into definite shape and form chiefly by his successful effort to elucidate all the characteristic features common to the various forms of anæmia. He was thereby enabled as the first one to give a definition of this, up to this time, so pliable and undoubtedly much-abused term, anæmia.

Anæmia, in the broadest sense of the word, includes all those processes characterized by a decrease in the amount of albumin and by an increase of the liquid part of the blood; in other words, hypalbuminæmia and hydræmia are conditions present in all forms of anæmia, and this holds good not only in cases of primary anæmia, like leucæmia and chlorosis, but also in all so-called secondary anæmia.

This discovery enables us to understand the hitherto empirical fact that the treatment of anæmia requires not only or exclusively the administration of iron, but that all the metabolic processes, especially the introduction and assimilation of albuminous substances, must be increased, should the treatment be followed by success. But just this part of the treatment is exceedingly difficult, since one of the most constant symptoms which we meet with in the various forms of anæmia is a more or less high degree of anorexia. This anorexia completes the "circulus vitiosus" so frequently observed in clinical pathology—a circulus vitiosus which must be understood in each individual case in order to be amenable to successful treatment. For it is evident that a continuous anorexia will lead to insufficient nutrition, to subnutrition, thereby constantly increasing the condition of hypalbuminæmia.

The anorexia is, however, a natural sequelæ of this abnormal condition of the blood, in consequence of which, at least in the greater number of cases, the secretion of hydrochloric acid is decidedly diminished (2).

In the treatment of these cases, therefore, we must constantly bear in mind the condition—hypalbuminæmia. In order to facilitate the increase of albumin in the blood, notwithstanding the anorexia already existent, its administration in the form of easily assimilated peptones would be most rational.

Prompted by this thought, I began in the spring of 1893 to make use of Dr. Gude's preparation, known as pepto-mangan, in most all cases of anæmia that came under my observation, with the exception of those accompanying or following chronic infectious diseases, such as tuberculosis, or of malignant tumors, such as cancer, etc. I collected from my clinical record thirty-four cases. The greater number of these were closely observed, not only as to the influence of the remedy upon the subjective symptoms, but also as to its effects upon the blood by careful examinations which I carried out with the aid of Gärtner's hæmatokrit (3).

This excellent instrument, which requires the use of Professor Gärtner's *Kreisel* (spinning top) centrifugal machine, enables, in a very accurate manner, a determination of the volume percentage of the red blood-cells within about ten minutes.

I prefer this method of determining the efficacy of a remedy against anæmia to the old method of counting the red blood-corpuscles.

Although, generally speaking, the number of the red blood-corpuscles bear a certain proportion to the volume percentage, yet it would be wrong to identify both. In blood diseases especially, the knowledge of the volume percentage is undoubtedly of great importance.

During the above-mentioned period I observed neither cases of leucæmia nor of pernicious progressive anæmia. The thirty-four cases I treated with pepto-mangan were partly cases of chlorosis and partly secondary anæmia, occurring chiefly after subacute malaria and typhoid fevers. Of these I select six as paradigmata, as it were.

Two of these were cases of chlorosis and four cases were secondary anæmia:

Case I.—Miss A. S., aged eighteen years; chlorosis rubra; oligocythæmia and oligochromæmia; palpitation of the heart; frequent pulse; coated tongue; fœtor *ex ore*; constipation; irregularity in menstruation; easily fatigued; muscular weakness.

At the beginning of treatment, thirty-per-cent. volume; eight days later, thirty-eight per cent.; at the end of the fourth

* I wish to express my great gratitude to Professor Mendel, Professor Jolly, Professor Oppenheim, Professor Bernhardt, Professor Eulenburg, and Dr. Goldscheider of Berlin, Professor Brissaud of Paris, and Dr. J. Hughlings Jackson of London, for permission to examine cases entering into this report.