We believe this is explicable on the basis that the virus, which has been detected in the nasopharynx by ourselves and others, did not reach the central nervous system after excretion onto the nasal mucosa, because of the break in the olfactory pathway.

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Hyperthyroidism and Brain Oxidations.

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Many workers have shown that general tissue metabolism is increased in hyperthyroid animals; few have attempted an analysis of the specific cell enzymes involved. Dye and his coworkers[1] reported such studies on hypothyroid dogs, and McEachern[2] on thyroxinized rats (liver, kidney and muscle). The present experiments on hyperthyroid rat brain, directed to this end, were begun before the appearance of McEachern's paper.

Male white rats (160 to 190 gm.) were used. Experimental animals were fed the same stock diet as controls, plus 0.6 gm. desiccated thyroid daily for 16-19 days. A brei of finely minced whole brain was suspended in phosphate Ringer and its oxygen consumption determined at 37°C. in Warburg manometers.

Typical substrates were used alone and with dyes and/or inhibitors. Because of great variations in the early readings (probably determined by the amount of substrate initially present) the tissue was allowed to respire for 2 hours, at which time remarkably constant readings were obtained. The substrates were then tipped in, and the oxygen consumption for the next 90 minutes determined. With few exceptions each figure given is based on 6-10 experiments.

Results. (1) The Q_{O_2} of hyperthyroid brain (H) is initially 20% greater than that of normal brain (N), becomes equal to it in 2 hours, and is 10% lower in 4½ hours (due to substrate exhaustion).

(2) The Q_{O_2} of H is increased approximately 4 times as much

[1] Dye, J. A., *Am. J. Physiol.*, 1933, **105**, 518; Dye, J. A., and Waggener, R. A., *Ibid.*, 1928, **85**, 1; Dye, J. A., and Maughan, G. H., PROC. SOC. EXP. BIOL. AND MED., 1929, **26**, 439.

[2] McEachern, D., *Bull. Johns Hopkins Hosp.*, 1935, **56**, 145.

TABLE I.

Qo_2 of brei plus inhibitor and substrate is expressed in: I as per cent of brei + substrate without inhibitor; II as per cent of brei + inhibitor without substrate.

Added		M/1000 NaCN			M/1000 NaCN + .02% Cresyl blue			M/100 Iodoacetate			M/100 Malonate			0.1% Barbital			0.1% Urethane		
Substrate		N	H	H/N	N	H	H/N	N	H	H/N	N	H	H/N	N	H	H/N	N	H	H/N
None	I	20	18	0.9	69	63	0.9	13	13	1.0	96	96	1.0	94	93	1.0	97	101	1.0
	II				223	255	1.1												
Glucose	I	23	28	1.2	28	28	1.0	34	34	1.0				103	102	1.0	100	100	1.0
	II	183	275	1.5	224	275	1.2	365	430	1.2				122	183	1.5	121	181	1.5
Lactate	I							12	22	1.8				93	89	0.9	152	152	1.0
	II							154	297	1.9				164	179	1.1	130	238	1.8
Succinate	I	12	11	0.9	83	75	0.9	21	22	1.0	31	25	0.8	80	80	1.0	100	100	1.0
	II	187	293	1.6	1240	1900	1.5	423	755	1.8	134	139	1.0	125	348	2.8	194	485	2.5
P-phenylene-diamine	I	22	22	1.0	27	28	1.1	90	96	1.1	106	98	0.9	99	103	1.0	100	100	1.0
	II	490	560	1.2	505	700	1.4	2230	3150	1.4	360	424	1.2	347	440	1.3	350	435	1.4

as that of N on adding substrates (S) which are probably involved in carbohydrate oxidation and glycolysis, even though the absolute increases for individual substrates varied widely. Percent increase $= \frac{N+S}{N} \times 100$ and $\frac{H+S}{H} \times 100$ for each substrate: glycogen = 3, 12; glucose = 18, 81; fructose = 72, 190; glycerophosphate = 22, 74; lactate = 19, 86; succinate = 92, 385. For p-phenylenediamine the increase in H is 35% greater than N (250, 337); while the increase for other substrates is essentially the same for N and H; (pyruvate = 68, 72; galactose = 32, 33; glycine = 11, 10; methyl glyoxal = 18, 16).

(3) Methylene blue (0.01%) and cresyl blue (0.02%) affect H and N alike, leading to an early increase (30 and 25% respectively in 15 minutes) and a later decrease of Qo_2 (equal at 30 minutes, 20 and 30% in 90 minutes).

(4) See Table I. Inhibitors decrease the respiration of brei and substrate by the same percentage in both H and N. On the other hand, the percentage increase of Qo_2 produced by adding substrates to inhibited brei is always distinctly higher in H than in N. These are consequences of the fact that with inhibitors alone H and N are alike in absolute values, while with substrates alone H is more than N.

Conclusions. These data suggest that the increased Qo_2 of hyperthyroid brain is especially associated with carbohydrate oxidations. The results with inhibitors further indicate that this greater respiration depends on proportional increases of both dehydrases and oxidases in the normal cell; but stronger concentrations of narcotics must be used to clinch this point. These results do not exclude Haffner's,[3] or allied hypotheses, which regard an increased glycolysis as the essential metabolic effect of hyperthyroidism and the increased Qo_2 as a secondary consequence.

[3] Haffner, F., *Klin. Wochnschr.*, 1927, **6**, 1932.