tomical fractions of the pancreas of the Goose-fish are reported in Table II. All activity is concentrated in the acinar tissue and is greater, on a specific basis, than that found in rat pancreas. The mesenteric fraction seems to be more active than the capsular fraction, probably because of the larger amount of connective tissue present in the capsule. The islet tissue was tested under a variety of conditions: fresh tissue, repeatedly frozen and thawed tissue, granular and supernatant fractions obtained by light centrifuging of the raw extract, as well as granular fractions disrupted by detergent or trypsin. In all cases no proelastolytic activity could be detected.

Since one of the earlier observations(1) describing elastase activity in the endocrine portion of the islet was made by examining elastin fibers under the microscope after 24 hours incubation with the extract, we have performed an assay under similar conditions. We found that 3.5 mg of elastin had been digested, an amount corresponding to 0.2 mg of elastin digested in 20 minutes per 100 mg of tissue. This negligible amount may be due either to a slight contamination by the capsular proelastase or to bacterial contamination.

We conclude that in the Goose-fish, elastase and its precursor proelastase are produced in the exocrine pancreas, a location typical of a proteolytic enzyme.

Summary. A proelastase resembling that of the pig and the rat has been found in the pancreas of the Goose-fish. It is localized in the acinar portion of the pancreas, both in the mesenteric bands and in the capsule of the principal islet. However it is absent from the endocrine portion of this organ.

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Pasteur Reaction and Glycogen Content of Rat Brain After Noble-Collip Drum Shock.* (26271)

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Shock produced by any method results in systemic hypoxia. Several investigators have based their work on the influence of shock upon intracellular processes on this fact. They excised tissue from normal animals and subjected it to anoxic conditions, *e.g.,* Greig(1) and Furchgott et al.(2). They interpreted then the changes observed under anoxia *in vitro* as indicative of what ought to have happened in shock.

This report describes tests on the Pasteur reaction with brain tissue of rats, obtained during shock.

Methods. The shock procedure has already been described(3). Only "taped" rats were drummed. Their weights ranged from 150 to 250 g. The brains were excised and homogenized in Potter-Elvehjem homogenizers(4) with Teflon pestles under ice cooling in Krebs-Ringer or bicarbonate media(5) for aerobic and anaerobic tests, respectively. Each gram of fresh brain was made up with 10 ml of medium. The nitrogen content of the homogenates was determined with a mi-

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TABLE I. Pasteur Reaction in Rat Brain Homogenates.

	No.	Ratio of aerobic/anaerobic glycolysis	S.E.
Control	7	.17	.031
Trauma	7	.29	.037

"Student's" $t = 2.5$ $p < .05$.

Difference between control and trauma groups is statistically significant.

cro-Kjeldahl method. Non-protein nitrogen was measured after precipitation of an aliquot with 5% trichloroacetic acid (1:10) in the supernate and the difference was taken as protein nitrogen.

Aerobic glycolysis. 1.0 ml of brain suspension was pipetted into 25 ml Erlenmeyer flasks containing 1.0 ml of 10% glucose and 3.0 ml of a mixture of .85% sodium chloride and .05% potassium chloride of pH 7.4. For blank tests glucose was omitted and 4.0 ml of the salt mixture were used. The flasks were shaken in the Dubnoff shaker for one hour. Aliquots of 1.0 ml were then withdrawn and pipetted into 9.0 ml of 5% trichloroacetic acid for deproteinization. After centrifuging lactic acid was determined with the method of Barker and Summerson(6).

Anaerobic glycolysis. Homogenates were gassed for 30 minutes with a mixture of 95% nitrogen and 5% carbon dioxide in Warburg vessels. Ten per cent glucose and sodium chloride-potassium chloride mixture respectively were placed in the sidearms and tipped into the main compartment after thermal equilibrium had been reached. Analyses were done as described above.

All incubations were done at 37°C. Units of activity were micrograms of lactic acid formed from glucose/hr/mg protein nitrogen.

Glycogen. The anthrone method of Carroll *et al.* was used(7). Between 85 and 95% of glycogen (Pfanstiehl) added to brain tissue was recovered by this method. The recovery from liver and muscle was 92 to 95%.

Results. Both aerobic and anaerobic glycolysis were increased in shock, 65% and 3% respectively (Table I). The ratios of aerobic and anaerobic glycolysis calculated for control and for shock provided a measure of comparison. These ratios were 1:4.96 for control and 1:3.10 for shock with a statistical significance of $P < .05$. This is, then, an increase of lactic acid formation from glucose in presence of oxygen or a repression of the Pasteur reaction in shock. The Pasteur reaction was 1.6 times less in shock than in the control. The change of glycogen content in shock is shown in Table II. Glycogen content of liver and muscle was likewise found to be decreased in shock.

Discussion. The homogenates from shocked rats continued to use the glycolytic pathway under aerobic conditions with a correspondingly decreased yield of ATP. Our earlier experiments(8) had shown that oxidative phosphorylation was uncoupled in mitochondria from shocked rats' hearts and that the phosphorylation of ADP was inhibited in brain preparations from shocked animals. It thus appears that the repression of the Pasteur effect and the uncoupling of oxidative phosphorylation went hand in hand in shock. Seits and Engelhardt(9) have demonstrated *in vitro* that the Pasteur reaction was decreased under the influence of poisons which caused uncoupling of oxidative phosphorylation. There are observations on 2 rat brains made by LePage(10) which show a sizeable decrease of glycogen content within 68 and 230 minutes respectively following 13 minutes of tumbling, when respiration had just ceased. These data bespeak the same trend observed by us and which we have

TABLE II. Glycogen Content of Rat Tissues, Control and Trauma, mg %.

	Brain			Liver			Muscle		
	No.	Avg contrast	S.E.	No.	Avg contrast	S.E.	No.	Avg contrast	S.E.
Control	22	48.56	2.28	5	3869.0	483.6	2	464.2	13.45
Trauma	22	42.95		5	1193.2		2	313.1	
	"Student's" $t = 2.46$ $P = .011$			"Student's" $t = 5.53$ $P < .01$			"Student's" $t = 13.45$ $P > .05$		

shown to be significant. We observed a decrease of 20% only, whereas in the tests reported by LePage almost no glycogen was present. This difference may be explained by the fact that our samples were analyzed at a predetermined time when all rats were surviving and were not moribund.

The decrease of brain glycogen is in agreement with the impairment of the Pasteur reaction. Decreased phosphorylating processes in brain may contribute to the glycogen deficiency as was also shown for muscle and liver after shock. Low oxygen tension has been reported by Gurdijan *et al.*(11) to have decreased the glycogen content of the brain of one morphinized dog in severe hypoxia and cyanide poisoning causes also a highly significant decrease of glycogen in rat brain as seen by Albaum *et al.*(12). Burdette(13), as well as Kovach *et al.*(14) and Takacs *et al.*(15) have also found that in muscle there occurs depletion of glycogen in shock.

Summary. Increased aerobic and anaerobic glycolysis occurred in brains of tumbled (fettered) rats in shock. The Pasteur reaction was significantly decreased. This indicates an increased anaerobiosis in the brain tissue as a consequence of shock with concomitantly decreased yield of high energy phosphate. Glycogen content of brain is also significantly decreased in shock.

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Immunologic Differences Between Murphy-Sturm Lymphosarcoma and Normal Rat Lymph Nodes.* (26272)

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The Murphy-Sturm lymphosarcoma of rats is derived from bloodborne leukemic cells(1), and is thus related to lymph tissue. This relationship was corroborated by the use of fluorescein label technics. We showed previously that rabbit antisera prepared against the ascites form of the lymphosarcoma strongly cross-react with rat lymph node and spleen(2).

We have now been able to show differences in the antigenic patterns of the lymphosarcoma and lymph node (taken as a normal counterpart) by the use of radio-iodine labeled antibody technics. I^{131} labeled globulins from anti-lymphosarcoma and anti-lymph node sera were specifically fractionated by successive adsorption and elution from tumor and lymph node components. It was thus possible to separate the antibodies into fractions of different specificities, indicating the presence of characteristic anti-

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