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THE MORPHOLOGY AND ROLE OF THE PHRENIC NERVE IN THE »HEPATIC SPHINCTER«*

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I. Introduction

Arey and *Simmonds* (1920) had been the first to study the morphology of the »hepatic sphincter«. In the hepatic vein of the dog they had found strong fasciculi of smooth muscle cells which they stated to be absent in the phytophagous species. This system was described by *Eppinger* as »Lebervenensperre«. *Bergman* has published very demonstrative pictures of the constrictive system in the hepatic veins of the dog and the seal. While investigating the constrictive system of the human liver, *Conti* (1951) recorded in the walls and at the ramification of the hepatic veins fasciculi of smooth muscle-cells protruding into the lumen. Yet he attributed less importance to these in man, the blood supply to the liver and the blood content of the organ being instead regulated by the longitudinal bundles of muscle present in the branches of the hepatic artery.

Apart from morphological findings, physiologists and clinicians have confirmed the existence of the »hepatic sphincter« and investigated the nervous humoral factors giving rise to it. Among the Hungarian authors, it was *Haynal* who in his book on circulation has discussed the question in particular. Responsibility for certain fluctuations of the blood pressure has been ascribed to the hepatic veins by *Simon* and *Roca*. On the basis of their animal experiments, *Starling*, *Dale*, *Dennecke*, *Laidlaw*, *Richmond*, *Mauthner* und *Pick*, *Rich*, *Hofmeister Neubauer*, *Bang*, *Masing*, *Tenström*, *Fröhlich*, *Pollak*, *Elias* and *Sammartino* corroborate the effect exerted on the circulation by the constrictive system in the hepatic veins.

It is well known that the portal region plays a significant part in the circulation, functioning as a variable blood reservoir. *McFate* and *Lewison* have termed the liver a »Flood chamber« serving to unburden the heart.Not only is the volume of blood flowing through the liver abundant but the conveying channels are of such a great extent that the amount of blood passing within the unit of time is considerable too.

* »Hepatic sphincter« will be used to denote the phenomenon termed in German »Lebersperre«.

It was by chance that our investigations have been focussed on the problem of the »hepatic sphincter«. While examining the fibres, the origin, and the course of the phrenic nerve, we have observed several fibres piercing the diaphragm and seemingly ending in Glisson's capsule. Following their course, these fibres always run along the hepatic veins, and find their end in their walls.

II. The morphological structure of the phrenic nerve

Several authors have studied the origin of the phrenic nerve, as well as the ending and function of its fibres. *Hyrtl*, *Testut* and *Krause* have investigated

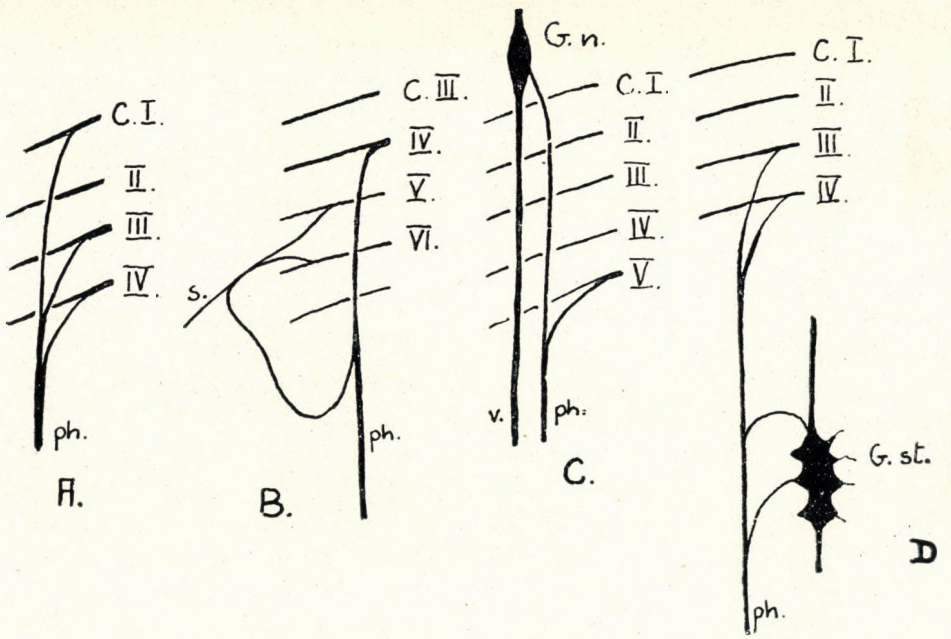

Fig. 1. Variations of the origin of the phrenic nerve. ph. : phrenic nerve; s : subclavian nerve ; v : vagus nerve ; Gn : nodous ganglion ; G. st. : stellate ganglion

the cervical section, *Luschke* its connection with the coeliac ganglion. *W. Felix Timofeyev*, *Henle*, *Schwalbe*, *Bertelli*, *Ramström*, *Eisler* and *Fuchs* have endeavoured to clarify its role in the innervation of the diaphragm. Important are the examinations of *F. Kiss* and *Ballon*, according to whom the vegetative fibres in the phrenic nerve are mainly of sympathetic origin and exert a vasomotor activity. They, moreover, emphasize that the number of vegetative fibres in the phrenic nerve does not exceed that encountered in other peripheral nerves. They regard the anastomoses between the phrenic nerve and the dia-

phragmatic plexus as a source for supplementing the vasomotor and secretory fibres.

Our own investigations have followed two lines.

a) By means of macroscopic and microscopic preparations we have studied the origin, anastomoses, and ending of the phrenic nerve, in 10 human (8 of them were fetuses) and 20 animal (10 dog and 10 cat) cases.

b) On the basis of the morphological findings we have endeavoured to determine the influence of nervous factors on the mechanism of the »hepatic sphincter«.

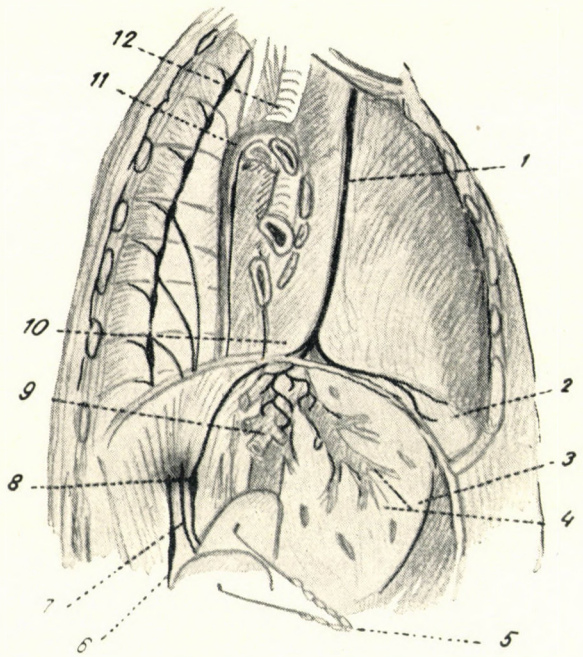

Fig. 2. 1. phrenic nerve ; 2. diaphragm ; 3. liver ; 4. hepatic vein ; 5. kidney and suprarenal gland ; 6. sympathetic trunk ; 7. splanchnic nerves ; 8. phrenic ganglion ; 9. hepatic vein ; 10. inferior vena cava ; 11. azygos vein ; 12. trachea

The results of our morphological investigations may be summarized as follows :

(i) The constant fibres of the phrenic nerve originate from the segments C. IV. and C. V.

(ii) In 50 per cent of the cases the nerve receives supplementary fibres from C. III. : in 40 per cent from C. IV. and also from the superior trunk of the branchial plexus (Fig. 1. A).

(iii) In 10 per cent fibres arise also from C. I. (Fig. 1. A).

(iv) In rare cases, an accessory phrenic nerve was encountered. (Fig. 1. B).

(v) In the cervical area, connection with the middle and superior cervical ganglions could not be traced in every instance, while connection with the stellate ganglion was demonstrable in each case (Fig. 1. D).

There is an ascending branch anastomosing with the vagal nodose ganglion (Fig. 1. C). The latter can be found only on the right side. Our investigations have revealed a point of which no mention has been made in the literature, viz. the essential difference between the right and the left phrenic nerves. Only the right phrenic nerve shows anastomoses with the vagus nerve. The right nerve is stronger than the left one and contains a much greater number of vegetative fibres without a myelin sheath. The fibres of the left phrenic nerve

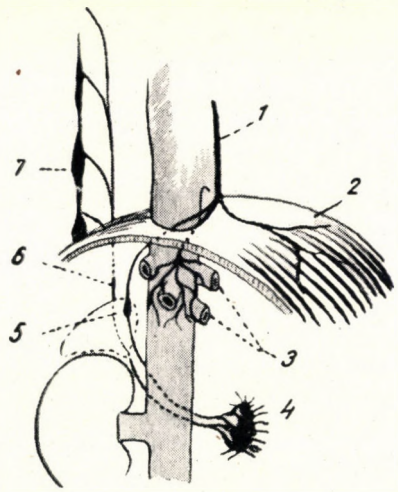

Fig. 3. Schematic description of the ending and connections of the phrenic nerve. 1. phrenic nerve ; 2. diaphragm ; 3. hepatic vein ; 4. coeliac ganglion 5. phrenic ganglion ; 6 splanchnic nerve ; 7. sympathetic trunk

end in the diaphragm, whereas the right nerve passes through the diaphragmatic aperture of the vena cava inferior and, dividing into three branches, forms a rich plexus around the point where the hepatic veins drain into the vena cava inferior (Fig. 2). The posterior branch passes the abdominal surface of the diaphragm and proceeding along the lateral crus, traverses the phrenic ganglion Fig. 3. The latter is usually a solitary structure but in about 15 per cent of the cases it consists of 2 to 3 members connected partly with the posterior branch of the right phrenic nerve and partly with the diaphragmatic plexus. In some cases, the left phrenic nerve too pierced the diaphragm and, making its way behind the oesophagus, anastomosed with the opposite phrenic nerve. These anastomoses play their part in the radiation of pain to the left shoulder in cases of lithiasis of the biliary vesicle *(Vorobyov)*.

Among the results of our morphological investigations, the findings that drew our attention to the connection between the phrenic nerve and the liver were the following :

(i) The presence of a strong bundle of nerves at the inflow of the hepatic veins,

(ii) The fact that this bundle is formed by the right phrenic nerve which receives a plentiful supply of vegetative fibres from the vagus and the sympathetic nerves,

(iii) Phrenic ganglia can be found on the right side only.

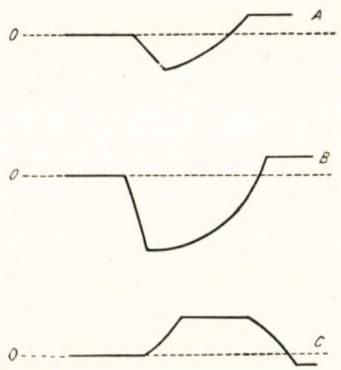

Fig. 4. Diagram of the pressures recorded in the inferior vena cava. A : Effect of the stimulation of the phrenic nerve ; B : Effect of the Witte pepton ; C : Effect of the stimulation of the splanchnic nerve or of 0,1 mg Adrenaline

III. Methods

First of all we endeavoured to determine whether the fibres observed have any connection with the vegetative mechanism of the hepatic veins. Three regulating mechanisms had to be taken into account.

(i) The portal vein, its roots (including the spleen) together with the splanchnic arterioles.

(ii) The hepatic arteries (compare the longitudinal muscle fasciculi described by *Conti)*.

(iii) The »hepatic sphincter« recorded by *Mauthner* and *Pick*, which is, according to present knowledge, provoked by the effect of shock producing agents (H-substances, peptone).

Excitement of the vagus nerve also promotes the constriction of the hepatic veins. Their relaxation is induced by excitement of the splanchnic nerves, of the sympathetic nervous system in general or by a small dose of adrenalin (0,1 mg). Larger doses of adrenalin exert a constrictive influence.

There were two courses open for a close investigation of the question, experimenting on the surviving liver as *Mauthner* and *Pick* had done, and the surgical solution in vivo. We decided in favour of the latter, all the more so since the study involved the investigation of the nervous system and thus we considered it necessary to maintain physiological conditions as far as possible. Dogs and cats were used for the experiments. Operations were performed under Evipan anaesthesia. The thoracic section of the vena cava inferior was exposed by resecting 4 to 6 ribs from a longitudinal incision. To exclude eventual contraction of the diaphragm and the resulting mechanic venous congestion, the vein was isolated from the diaphragm by a circular incision 1 to 1,5 cm from the foramen venae cavae. Then a water manometer was introduced into the vena cava inferior and the venous pressure measured. Subsequently the cervical section of the phrenic nerve was stimulated for 10 seconds with a current of 4,5 V, both faradic and galvanic, then 2 ml of a saturated solution of Witte's peptone in physiological saline administered directly through the cannula. Then the splanchnic nerve was excited in the same way as the phrenic nerve. Finally 0,1 mg of adrenalin was injected into the vena cava inferior, similarly to the procedure followed with peptone.

For registering the effects of protracted excitation, comparative liver function tests were carried out on dogs and cats. A total of 24 animals, of approximately the same weight and fed the same diet were observed in 6 groups, each containing one control and three operated. Tests of hepatic function were performed in blood and urine and then, under anaesthesia with Evipan, the cervical section of the phrenic nerve was exposed and crushed. In some cases the nerve was ligated to the surrounding area in order to give rise to a protracted stimulus. 3, 5, 7 or 10 days after the operation the animals were sacrificed and the tests of hepatic function repeated. In the last two groups tests were performed also during the period between operation and sacrificing.

As tests of hepatic function, serum bilirubin and serum cholesterin ester were determined, and Gross' reaction, in the modification of *Takata* and *Dénes*, and the complement fixation test were performed. For qualitative determinations a *Hellige—Autenrieth* colorimeter was used and some tests were carried out in the laboratory of the Ist Dept. of Medicine. Urobilinogen and bilirubin in the urine were determined qualitatively. In part of the cases special examinations were also undertaken, such as the bilirubin tolerance test recommended by *Bergmann* and *Eilbot*, and the bromsulphalein clearance. In animals giving markedly positive results the filling of the gall bladder was examined with X rays after intravenous administration of 20 mg of Jodtetragnost (Merck).

The livers of the animals were fixed in Carnoy's solution and, after combined embedding, stained with haematoxylin-eosin, and subjected to histological examination.

IV. Observations

The following conclusions could be drawn from the manometer readings and the pressure kymograms.

(i) On stimulation of the phrenic nerve for 10 seconds, pressure in the vena cava inferior falls by 3 to 4 cm H_2O. The effect manifests itself as soon as stimulation has begun, the pressure decreases gradually, parallel with the duration of the excitation and reaches the lowest value in the eighth to twelfth second. The pressure remains unchanged at the low level for 3 to 5 minutes

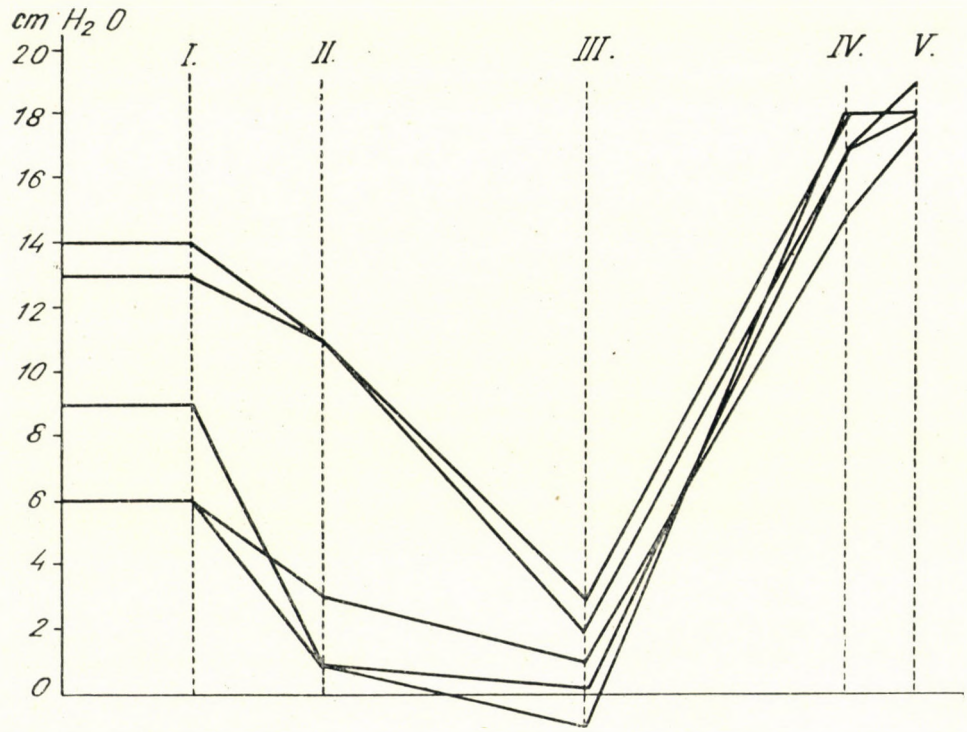

Fig. 5. Diagram of the pressures recorded in the inferior vena cava. I : Normal ; II : Stimulation of the phrenic nerve ; III : Effect of the Witte pepton ; IV : Stimulation of the splanchnic nerve ; V : Effect of the Adrenaline

and then begins to rise slowly Fig.4. (A.) Concurrently with the fall in pressure, enlargement and hardening of the liver can be observed. The pressure regains the initial value in 10 minutes but in part of the cases it remains somewhat below it.

(ii) Under the influence of 2 ml of a saturated solution of Witte's peptone administered directly into the vena cava inferior, the pressure decreases in almost every case below 2 cm H_2O. The effect is immediate, the fall precipitious.

The increase in size and hardening of the liver becomes still more pronounced (Fig. 4. B).

(iii) Upon excitation of the splanchnic nerve, the pressure rises to from 2 to 8 cm H_2O above the initial value. The pace of the elevation is slow, starting only after 10 seconds' stimulation; the maximum value is reached 30 to 40 seconds after stimulation has been discontinued (Fig. 4. C).

(iv) 0,1 mg of adrenalin injected directly into the vena cava inferior through a cannula also elicits a sudden rise amounting to from 2 to 8 cm H_2O in the pressure, as suddenly as pepton provokes the opposite (Fig. 4. C).

The conclusion appears justified that electric stimulation of the phrenic nerve is similar in effect to intravenous administration of Witte's peptone.

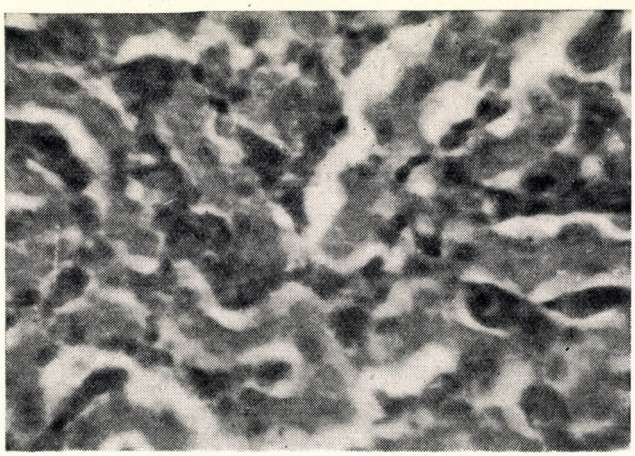

Fig. 6. Cat's liver 5 days after operation. Wide, stretched Disse-spaces

The constrictive system of the hepatic veins contracts. The constriction can be relieved by adrenalin or stimulation of the splanchnic nerve. These findings lead us to infer that while the shock poisons may be regarded as a humoral factor of the »hepatic sphincter« mechanism, the phrenic nerve, acting through the vegetative fibres it contains, would represent a nervous factor (Fig. 5).

In the animals sacrificed 3, 5, and 7 days, respectively, after crushing of the phrenic nerve, marked hepatic congestion occurred between the third and fifth days, which was relieved at about the seventh to the ninth day; after ten days, hepatic function again approached or returned to normal conditions. (Table I—II.)

(i) — *Serum bilirubin* showed an increase between the third and fifth days and then gradual decrease. As a rule, an indirect reaction was obtained; three cases yielded a prolonged direct reaction.

(ii) — *Serum cholesterin ester* showed a pronounced fall and did not attain the initial value even after eight days.

(iii) — *The complement fixation test* was in almost every instance positive ; in a few cases only did it become negative by the time the animal was sacrificed.

(iv) — *Gross' reaction* yielded a positive result on the addition of 0,5 to 1 ml of Hayem's solution in most part of the cases.

(v) — *Takata-Ara test* was found positive in three cases only (maximum^{++}).

(vi) — *Urobilinogen in the urine* gave a markedly positive reaction in each case and as a rule remained positive even after the eighth day. Bilirubin was demonstrable in the urine in two cases.

(vii) — *The bromsulphalein-clearance* was determined in six cases, after an intravenous dose of 0,005 mg per gram of body weight. After 30 minutes serum values of 8 to 10 per cent were found (for details see Table). *Dean* and

TABLE I

Variation in pressure of the Vena Cava Inferior (cm. water.)[1]

No.	Prior to excitation	Phrenic nerve	Witte's peptone	Splanchnic nerve	Adrenalin (0,1 mg)
1. (dog)	13	11	2	16	16
2. «	8	1	0,5	—	12
3. «	9	3	1	—	17
4. «	14	5	3	19	19
5. «	6	3	1	15	17
6. «	9	4	3	11	15
7. «	11	9	2	10	13
8. «	14	3	1	19	19
9. «	10	4	3	18	18
10. «	13	6	1	12	17
11. «	11	5	2	—	19
12. «	8	4	0,8	15	19
13. «	14	6	3	14	16
14. «	12,5	7	2	16	16
15. «	11	3	1	12	12
16. «	9	3	2	11	10,3
17. (cat)	7	4	3	7	7
18. «	8	6	4	9	9
19. «	7	5	2	—	8
20. «	7,6	5	1	—	16
21. «	14	6,6	3	17	16
22. «	10,4	4,2	2	18	15

[1] These are not absolute values. We measured the pressure in a capillary tube.

Macdonald's modified 45 minute procedure showed a concentration of 12 to 18 per cent which implies a retention of a minor degree.

(viii) — *Filling of the gall bladder.* Each animal was administered 10 mg of Jodtetragnost (Merck) dissolved in physiological saline or (in two cases) in 20 per cent dextrose. 2, 2,5 and 4 hours after the injection the animals were anaesthetized with Evipan and examined with X rays. The control animals received the same amount of the dye. In general, the operated animals showed a decreased filling in comparison with the control animals.

TABLE II

Tests of Hepatic Function

Group No. 1 (cats)

3 operated, 1 control; tests performed 3 days after operation.

No.	Serum cholest. ester mg. per 100 ml	Serum bilirubin mg per 100 ml	Complement fixation	Gross ml	Bromsulphalein		Takata	Jodtetra gnost	Urine	
					30′	45′			Ubg	bili-rubin
1.	140	1,2	—	1,5	15	—	—	—	—	—
	120	1,6	+	1,0	—	8,4	—		+	+
2.	105	0,4	—	1,3	—	—	—		—	—
3.	110	1,4	—	1,3	16,4	—	—		—	—
	106	1,8	+	0,6	—	9,2	—		+	+
4.	120	0,8	—	1,4	—	—	—		—	—
	130	0,8	—	1,3	—	—	—		—	—

Group No. 2 (cats).

3 operated, 1 control; tests performed 5 days, after operation.

5.	150	1,1	—	1,1	16,3	—	—	no filling	—	—
	50	2,0	+++	0,80	—	9,4	+	no filling	+++	+
6.	140	—	—	1,2	17,4	—	—	no filling	—	—
	70	—	++	0,06	—	10,0	++	no filling	+++	+
7.	125	0,8	—	1,3	—	—	—	partial filling	—	—
	100	2,0	+	0,2	—	—	+	partial filling	++	+
8.	130	0,7	—	1,3	—	—	—	partial filling	—	—
	125	0,6	—	1,5	—	—	—	partial filling	—	—

Bilirubin tolerance test.	after 4 hours	after 8 hours
(animal No. 5)	8 mg per 100 ml.	5,4 mg per 100 ml.
(animal No. 7)	7,8 mg per 100 ml.	5,0 mg per 100 ml.

(ix) — *A bilirubin tolerance test* was performed in 5 animals, by injecting intravenously 20 mg of bilirubin dissolved in 10 ml of warm 5 per cent sodium bicarbonate. 4 hours later the serum bilirubin level was still very high and did not return to normal even after 8 hours. Histological findings revealed hepatic congestion in each case. The *Disse—Kiernan* spaces were distended and filled with lymph and blood. In most sections the veins were greatly congested ; the capillary walls were detached from the hepatic trabeculae. (Fig. 6).

Group No. 3 (cats).
3 operated, 1 control ; tests performed 7 days after operation.

9.	138	0,6	—	1,2	—	—	—	medium	—	—
	130	—	—	0,8	—	—	—	filling	+	—
10.	150	0,6	—	2,3	—	—	—	slight filling	—	—
	126	0,8	+	0,7	—	—	+	slight filling	+	—
11.	140	0,8	—	1,5	—	—	—	slight filling	—	—
	115	1,7	+	1,1	—	—	—	slight filling	+	—
12.	136	0,9	—	1,3	—	—	—	well filled	—	—
	128	0,8	—	1,3	—	—	—	well filled	—	—

Group No. 4 (cats).
3 operated, 1 control ; tests performed 3 and 8 days after operation.

No.	Serum bilirubin mg per 100 ml	Serum cholest. ester mg per 100 ml	Complement fixation	Gross ml	Bromsulphalein		Takata	Jodtetra gnost.	Urine	
					30′	45′			ubg	bili-rubin
13.	1,0	138	—	1,1	—	—	—	slight filling	—	—
	1,2	125	+	0,5	—	—	—	slight filling	+	—
	1,0	130	+	0,8	.	.	.	slight filling	—	—
14.	0,6	150	—	1,3	—	—	—		—	—
	0,9	115	+	0,6	—	—	—		+	—
	0,8	125	+	1,0	—	—	—	slight filling	+	—
15.	1,2	145	—	1,2	8,5	—	—		—	—
	1,4	70	+	0,4	—	13,2	+	slight filling	++	—
	0,7	110	+	0,8	—	—	—		+	—
16.	0,7	110	—	1,5	—	—	—		—	—
	0,6	120	—	1,4	—	—	—	well filled	—	—
	0,6	126	—	1.5	—	—	—		—	—

Bilirubin tolerance test	after 4 hours	after 5 hours
(animal No. 15. 3 days after operation).	4,2 mg per 100 ml	2,0 mg per 100 ml

Group No. 5 (dogs)
3 operated, 1 control; tests performed 3 and 9 dyas after operation.

17.	—	150	—	1,2	—	—	—		—	—
	—	115	+	0,8	—	—	—	slight filling	++	—
	—	125	—	0,9	—	—	—		+	—
18.	0,6	130	—	1,3	—	—	—		—	—
	0,9	115	+	0,6	12,2	—	—	slight filling	+	—
	0,6	130	—	0,8	—	8,4	—		+	—
19.	0,6	145	—	1,0	—	—	—		—	—
	1,0	125	+	0,2	—	—	—		+	—
	0,9	117	—	1,1	—	—	—		+	—
20.	0,7	120	—	1,5	—	—	—		—	—
	1,8	120	—	1,4	—	—	—		—	—
	1,7	122	—	1,4	—	—	—		—	—

Group No. 6 (dogs)
3 operated, 1 control; tests performed 8 days after operation.

21.	0,6	142	—	1,3	—	—	—		—	—
	1,0	130	+	1,1	—	—	—		+	—
22.	0,4	130	—	1,3	—	—	—		—	—
	0,3	120	—	1,4	—	—	—		+	—
23.	0,48	136	—	1,2	—	—	—		—	—
	0,5	119	—	1,3	—	—	—		+	—
24.	0,42	125	—	1,1	—	—	—		—	—
	0,36	125	—	1,1	—	—	—		—	—

Summary

It was found on human and animal material that the right phrenic nerve supplies fibres to the site of inflow of the hepatic veins. Animal experiments proved the connection between these fibres and the constrictive system of the hepatic veins. These findings are supported by the results of physiological and clinical studies. On stimulation of the phrenic nerve, pressure in the vena cava inferior decreases similarly as in consequence of shock poison. Simultaneously enlargement and hardening of the liver are observable. On stimulation of the splanchnic nerve or after an intravenous injection of 0,1 mg of adrenalin, the pressure increases and the constriction becomes relieved. It therefore appears probable that the vegetative fibres running within the phrenic nerve constitute one of the nerve components of the »hepatic sphincter« mechanism.

Partial crushing of the cervical portion of the phrenic nerve results in prolonged stimulation of the nerve. The above-mentioned fibres of the nerve then bring about a reversible hepatic congestion that, depending on the susceptibility of the experimental animal, gives rise to more or less grave transient hepatic lesion. The lesion is most marked from the third to fifth days following the intervention and gradually subsides by the tenth day. The histological picture of the liver at the height of congestion, between the third and fifth days presents the characteristic appearance of hepatic stasis.

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МОРФОЛОГИЯ ДИАФРАГМАЛЬНОГО НЕРВА И ЗАПОР ПЕЧЕНИ

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Резюме

При вскрытии животных и человеческих трупов оказалось, что диафрагмальный нерв. (n. phrenicus) на правой стороне дает волокна к месту впадения печеночных вен. Экспериментальные исследования доказывают, что вышеупомянутые волокна стоят в связи с запирательным аппаратом печеночных вен.

Результаты физиологических и клинических исследований подтверждают морфологические данные. При раздражении диафрагмального нерва в нижней полой вене замечается падение давления, в такомже направлении как и при падении вследствие вызывающих шок ядов. В то же время печень становится более плотной и увеличивается при раздражении внутренностного нерва или же при внутривенной подаче 0,1 мг. адре-

налина, даление повышается, запор печени прекрашается. На основе этих данных авторы считают вероятным, что вегетативные нервные волокна, идущие в диафрагмальном нерве представляют часть нервного компонента запора печени.

При частичном разрушении шейного участка диафрагмального нерва, нерв переходит в состояние постоянного раздражения. Вышеупомянутые волокна этого нерва таким образом вызывают временный застой печени, ведущий — в зависимости от степени чувствительности данного подопытного животного — к более или менее выраженным, переходящим изменениям печени. Повреждение печени наиболее выражено на 3—5-ом дне после оперативного вмешательства, а затем до 10-го дня изменения постепенно исчезают. Гистологическая картина печени, исследованная во время максимального застоя (т. е. на 3—5 дне после вмешательства) соответствует характерной патогистологической картине застойной печени.