A Complex Homeopathic Preparation for Hepatic Health

H. Vill, M.D.

Reprinted from *Biologische Medizin*, 14; 1, 1985:339-46.

In recent years the diagnosis of liver ailments has undergone refinement resulting in a plethora of groups, subgroups, and special forms of liver disease. However, there have been no great advances in therapy. In most cases, treatment of the various liver ailments remains palliative and limited to eliminating dangerous noxae. Only chronic aggressive hepatitis responds to glucocorticoids and immunosuppressive therapy, and here too no cure is possible.

This is a difficult situation for a practicing physician who is entrusted with the care of patients. Thus, a number of different remedies have been identified for daily usage, although their effectiveness at preventing hepatic disease has not been conclusively proven by clinical statistics. These remedies are:

1. The B vitamins in various high dosages, especially for alcoholics
2. Phospholipids and orotic acid in various concentrations, for stimulating liver regeneration
3. Extracts from the milk thistle plant, Carduus marianus; the pure substance silymarin is used primarily for liver detoxification. Twenty years ago, I reported on the positive effects of Carduus marianus in a published article.[1]
4. Practitioners of unconventional medicine are familiar with a series of other phytotherapeutic remedies that demonstrate very positive effects if administered early enough, when the body still has its endogenous defenses.
5. Cell extracts from embryonic livers for regeneration at very variable dosages

This author has also observed and reported good results with the Heel suis organ preparations, both in the potency chord of 10X/30X/200X and as single potencies for targeted indications.

Based on his knowledge of individual homeopathic remedies, Dr. Reckeweg developed Hepar® compositum, a combination preparation aimed specifically at liver function. In addition to organ extracts, this product includes Vitamin B12, silymarin, and a large number of other plant and mineral substances known to affect the liver. These ingredients are potentized and seem to work synergistically. Here is a full listing of the ingredients in Hepar® compositum:

Hepar 8X
Duodenum 10X
Thymus 10X
Colon 10X
Vesica fellea 10X
Pancreas 10X
China 4X
Lycopodium 4X
Chelidonium 4X
Carduus marianus 3X
Histamine 10X
Sulfur 13X
Avena sativa 6X
Fel tauri 8X
Natrum oxalaceticum 10X
Acidum α-ketoglutaricum 10X
Acidum DL-malicum 10X
Acidum fumaricum 10X
α-lipoic acid 8X
Orotic acid 6X
Cholesterol 10X
Calcium carbonicum Hahnemanni 28X
Taraxacum 4X
Cynara scolymus 6X
Veratrum 4X
Vitamin B12 4X

I began using this product, at first only in a limited number of cases, and then more frequently as I became convinced of the beneficial effects it had on my patients. In addition, it reduced costs since it contained such a large number of ingredients. The purpose of this paper is to present empirical data on 100 patients who received this preparation for indications commonly seen in daily practice. Hepar® compositum was used primarily in cases of hepatotoxicity, chronic persistent hepatitis, parenchymatous hepatopathies, fatty liver, and various forms of cirrhosis of the liver.

A primary clinical symptom of both chronic hepatitis and hepatotoxicity is sensitivity to pressure in the area of the left hepatic lobe in which the costal margin is palpated. If the patient then takes a deep breath, the pain upon palpitation of the left hepatic lobe is readily apparent.

Hepatotoxicities show up on serum tests primarily in elevated levels of γ-globulins, which are paralleled by elevated results of the thymol turbidity test.[2,3] The prothrombin test is also often positive.

In addition, disorders ranging from chronic hepatitis to cirrhosis of the liver are evident by changes in:

GOT (glutamic-oxaloacetic transaminase)
GPT (glutamic-pyruvic transaminase)
LAP (leucine aminopeptidase)
LDH (lactate dehydrogenase)
γ-GT (γ-glutamyl transpeptidase)
GLDH (glutamate dehydrogenase)
cholinesterase

Serum testing was used to check many different parameters, but only those indicating pathological changes appear in the statistics.

I. Examples of toxic hepatopathies with the following primary diagnoses:

a) with follow-up testing after 3 months

H. Margrit	Test Results		Diagnosis
	5/80	8/80	Rheumatism
γ-GT	18	15	
GLDH	4.4	4.4	
γ-globulins	20	17	

Sch. Beate	Test Results		Diagnosis
	7/77	10/77	Hyperuricemia, gout nephropathy
γ-GT	18	21	
GOT	16	5	
GPT	21	5	
LDH	237	148	
γ-globulins	24	20	

b) with follow-up testing after 6 months

W. Helmut	Test Results		Diagnosis
	12/79	6/80	Chronic osteomyelitis of the jaw with partial amputation
γ-GT	75	26	
prothrombin	35	55	
turbidity	2.0	1.2	
γ-globulins	23	28	

c) with follow-up testing after 9 months

K. Elfriede	Test Results		Diagnosis
	12/77	9/78	Chronic kidney insufficiency
GOT	8	5	
GPT	12	10	
γ-GT	14	13	
GLDH	4.4	3.2	
γ-globulins	23	21	

d) with follow-up testing after 1 year

K. Elfriede	Test Results		Diagnosis
	7/75	6/76	Chronic recurrent fungal infections with episodes of endometritis
GOT	32	6	
GPT	27	13	
γ-GT	37	23	
prothrombin	90	100	
LDH	638	169	
turbidity	9.2	1.5	

B. Ilse	Test Results		Diagnosis
	2/78	4/79	Chronic cardiac insufficiency with hepatopathy
GPT	13	11	
γ-GT	40	25	
GLDH	6.1	3.7	
prothrombin	85	100	
turbidity	1.3	1.2	
γ-globulins	19.6	20	

e) with follow-up testing after 15 months

H. Kurt	Test Results		Diagnosis
	4/79	7/80	Suffered 2 heart attacks, coronary insufficiency
GLDH	5.4	2.2	
prothrombin	70	90	
turbidity	1.5	1.3	

II. Examples of chronic persitent hepatitis

a) with follow-up testing after 3 months

H. Joachim	Test Results		Diagnosis
	7/80	10/80	Chronic persistent hepatitis
GOT	16	11	
GPT	27	23	
γ-GT	30	28	
GLDH	5.1	4.7	
prothrombin	100	90	
LDH	164	206	
turbidity	1.2	1.4	
γ-globulins	22	17	

Sch. Beate	Test Results		Diagnosis
	11/78	1/79	Chronic persistent hepatitis
GPT	19	16	
γ-GT	17	9	
GLDH	3.7	3.7	
turbidity	3.3	4.4	
γ-globulins	26	24	

b) after 3 months, followed by a 3 month interruption of therapy with faulty diet, then repeated follow-up testing 3 months later

Dr. K. Frid	Test Results		Diagnosis
	12/79	4/80	Chronic persistent hepatitis
GOT	128	10	
GPT	41	16	
γ-GT	56	25	
GLDH	2.9	1.9	
prothrombin	90	80	
γ-globulins	20	19	
	6/80	9/80	
GOT	27	17	
GPT	58	28	
γ-GT	42	35	
GLDH	5.1	3.3	
prothrombin	100	100	
γ-globulins	21	19	

III. Example of parenchymatous hepatopathy

W. Hannelore	Test Results		Diagnosis
	12/79	3/80	Aplastic anemia
γ-GT	13	12	
GLDH	5.1	3.7	
prothrombin	35	100	
turbidity	1.1	1.8	
γ-globulins	16	22	

IV. Example of fatty liver

Rev. K. Alois	Test Results		Diagnosis
	11/79	9/80	Hypertension, hyperuricemia, chronic enteritis
γ-GT	30	23	
GLDH	14.6	5.8	
turbidity	2.7	3.1	
γ-globulins	21	17	

V. Example of cirrhosis of the liver with follow-up testing after 9 months and again 4 months later

Prof. Pf. Hermann	Test Results			Diagnosis
	1/78	11/78	3/79	Liver cirrhosis with microscopic nodules, hypertension, cardiac arrhythmia
GOT	42	41	13	
GPT	103	58	27	
γ-GT	151	58	77	
GLDH	5.4	5.6	7.3	
prothrombin	70	100	40	
LDH	201	164	201	
turbidity	8.2	5.7	3.5	
γ-globulins	27	24	24	

The examples show that there is a general trend toward normalization of pathological and borderline lab test values, although isolated individual findings worsened. The main thrust is toward improvement.

Table 1 shows the average age (48 years) of the 100 tested patients. Equal numbers of men and women were treated.

Tab. 1	
100 Patients	∅ Age = 48.3 Years
50 Males	∅ Age = 50.5 Years
50 Females	∅ Age = 46.1 Years

In looking at the diagnoses (Table 2), it becomes apparent that three quarters of all cases involved hepatotoxicities. Chronic liver disease accounted for only one quarter, with chronic persistent hepatitis accounting for over half of these.

Tab. 2: Diagnosis

	Tox. Hepatopathy	Chron. per Hepatitis	Parenchym. Hepatopathy	Fatty Liver	Cirrhosis of the Liver
100 Patients	78	12	5	4	1
50 Males	34	9	4	2	1
50 Females	44	3	1	2	

Blood test results improved in 83% of the patients in the course of treatment. Pain sensitivity to pressure decreased in 38 out of 55 cases.

Tab. 3: Blood Test Results

	Improved	Same	Worse	n/a
100 Patients	83	8	9	
50 Males	39	4	7	
50 Females	44	4	2	
Sensitivity to Pressure				
53 Patients	38	10	5	47
20 Males	11	5	4	30
33 Females	27	5	1	17

The more specific tables (Tables 4, 5, and 6) give details of the results and show how quickly the patients responded to this therapy: 45% responded within 4 months, an additional 24% within 8 months, and only 14% required therapy for 12 months or longer.

Tab. 4: Duration of Treatment, 100 Patients

	Diagnosis	Blood Test Results			Sensitivity to Pressure			
		improved	worse	same	improved	worse	same	n/a
up to 4 months 56 Patients	4x Fatty Liver	2	1	1	1			3
	4x Parenchym. Hepatopathy	3	1		1			3
	6x Chron. Per. Hepatitis	6			1			5
	42x Toxic Hepatopathy	34	5	3	19	4	3	16
	56 Patients	45	7	4	22	4	3	27
up to 8 months 27 Patients	1x Parenchym. Hepatopathy	1			1			
	4x Chron. Per. Hepatitis	3		1	2			2
	22x Toxic Hepatopathy	20	2		9		1	12
	27 Patients	24	2	1	12		1	14
up to 12 months 11 Patients	1x Cirrhosis of the Liver	1						1
	2x Chron. Per. Hepatitis	7			1		1	
	8x Toxic Hepatopathy	7		1	1	1	3	3
	11 Patients	10		1	2	1	4	4
more than 12 months 6 Patients	6x Toxic Hepatopathy	4		2	2		2	2
	6 Patients	4		2	2		2	2
100 Patients (Total)		83	9	8	38	5	10	47

Tab. 5: Duration of Treatment, 50 Female Patients

	Diagnosis	Blood Test Results			Sensitivity to Pressure			
		improved	worse	same	improved	worse	same	n/a
up to 4 months 31 Patients	2x Fatty Liver	2			1			1
	3x Parenchym. Hepatopathy	3			1			2
	6x Chron. Per. Hepatitis	6			1			5
	19x Toxic Hepatopathy	15	2	1	6	3	1	9
	31 Patients	25	2	1	8	3	1	19
up to 8 months 10 Patients	1x Chron. Per. Hepatitis	1			1			
	9x Toxic Hepatopathy	7	2		2			7
	10 Patients	7	2	1	2			8
up to 12 months 7 Patients	1x Cirrhosis of the Liver	1						1
	2x Chron. Per. Hepatitis	2			1		1	
	8x Toxic Hepatopathy	3		1		1	2	1
	7 Patients	6		1	1	1	3	2
more than 12 months 2 Patients	2x Toxic Hepatopathy	1		1			1	1
	2 Patients	1		1			1	1
50 Patients (Total)		39	7	4	11	4	5	30

Tab. 6: Duration of Treatment, 50 Male Patients

	Diagnosis	Blood Test Results			Sensitivity to Pressure			
		improved	worse	same	improved	worse	same	n/a
up to 4 months 25 Patients	2x Fatty Liver	1		1	1			1
	23x Toxic Hepatopathy	19	2	2	13	1	2	7
	25 Patients	20	2	3	14	1	2	8
up to 8 months 17 Patients	1x Parenchym. Hepatopathy				1			
	3x Chron. Per. Hepatitis	3			2			1
	13x Toxic Hepatopathy	13			7	1		5
	17 Patients	17			10	1		6
up to 12 months 4 Patients	4x Toxic Hepatopathy	4			1		1	2
	4 Patients	4			1		1	2
more than 12 months 4 Patients	4x Toxic Hepatopathy	3		1	2		1	1
	4 Patients	3		1	2		1	1
50 Patients (Total)		44	2	4	27	1	5	17

The breakdown of results in Table 7 yields the following improvements in individual serum factors:

GOT	improvement in 5 patients
GPT	improvement in 16 patients
GLDH	improvement in 27 patients
γ-GT	improvement in 21 patients
LAP	improvement in 1 patient
LDH	improvement in 7 patients
prothrombin	improvement in 33 patients
γ-globulins	improvement in 40 patients
thymol turbidity	improvement in 44 patients

Tab. 7: Blood Levels

	Normalized	Improved	Stable within normal range	Pathological	Worse	n/a
GOT	4	1	47	2		46
GPT	10	6	42	2	3	37
GLDH	20	7	30	4	7	32
γ-GT	8	13	63	1	6	9
LAP		1	26		1	72
LDH	5	2	26	2	4	61
prothrombin	20	13	19	3	21	24
γ-globulins	17	23	26	3	16	15
turbidity	11	33	9	3	26	15

This underscores the favorable test results in that good responses were noted not only in hepatotoxicity, which changed mainly in the areas of γ-globulins, prothrombin, and thymol turbidity, but also in a relatively high percentages of the GLDH, GPT, and γ-GT results, which are more characteristic of chronic pathological processes.

That these therapeutic results are not merely coincidental is confirmed by the statistics from a different study, reported elsewhere in February 1983. In that study, 100 patients were treated exclusively with organ remedies. Twenty patients suffering primarily from chronic liver damage received Hepar 3X; 27 received Hepar 4X. Seventeen cases of hepatotoxicity received Hepar 30X and 36 received Hepar suis. Results of the 1983 study were similar to those of the current study, as demonstrated by Table 8:

Tab. 8: Blood Levels

	Improved	Unchanged	Worse
GOT	5	1	2
GPT	14	0	2
GLDH	38	2	10
γ-GT	26	1	7
LAP	0	1	1
LDH	6	0	2
prothrombin	37	6	19
γ-globulins	51	5	34
turbidity	40	4	38

Once again, this demonstrates that both hepatotoxicity and chronic liver diseases can respond favorably to therapy with complex homeopathic preparations.

These investigations show that it is not reasonable to simply observe liver ailments and passively wait for them to run their course. Therapy with 'biological pharmaceuticals' is indicated. The combination preparation Hepar® compositum has proved to be the most potent of these, since it is capable of favorably influencing both acute and chronic liver disorders. An advantage of this medication is that it does not require complicated monitoring examinations, since side effects have not been reported to date and are not expected, even in the most wide-ranging applications.

References

(1) Vill H. On the diagnosis and treatment of acute hepatogenic irritation syndrome. *Arzneimittel Praxis* XV, (49) 1963.

(2) Okuda K. [Clinical significance of thymol turbidity test.] In: *Nippon Rinsho* (1989 Dec) 48 Suppl:190-92.

(3) Monna T, Okuda K. [Thymol turbidity test (TTT).] In: *Nippon Rinsho* (1995 Feb) 53 Suppl, Pt 1: 163-66.

Address of the author:

H. Vill, M.D.
(Internal medicine)
Memelstrasse 22
D-8520 Erlangen
Germany