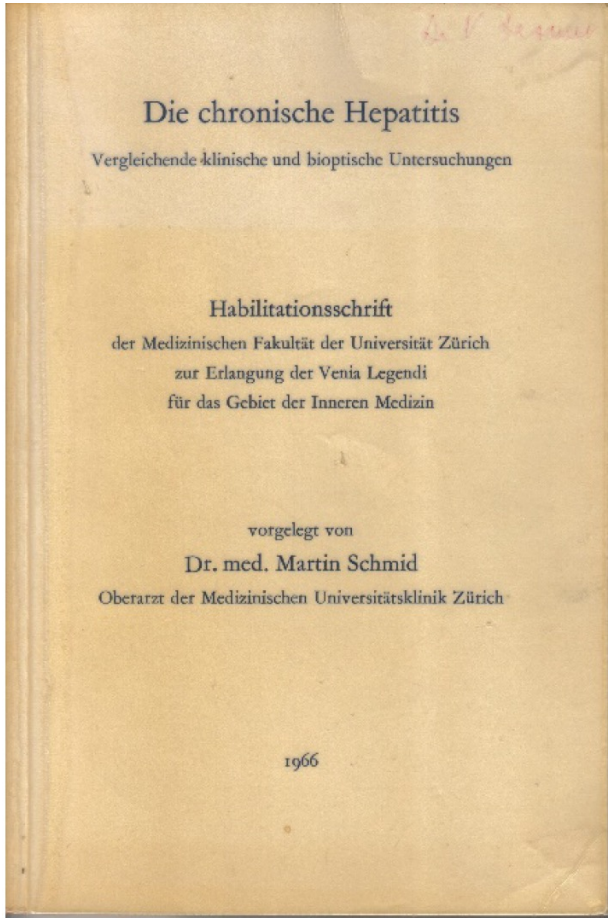
History of the Gnomes

Alastair Burt

Newcastle University and University of Adelaide

Gnomes: the ‘conception’

- 1967: meeting of European hepatologists and pathologists consider how to conceptualise chronic hepatitis at 2nd meeting of EASL; led by Jan de Groote
- By then able to see spectrum with widespread use of Menghini biopsies
- Disparate classifications: need for rationalisation
- Plans laid out for 1968 meeting in Zurich hosted by Martin Schmid
- Circulation of slides ahead of the meeting


Die chronische Hepatitis

Vergleichende klinische und bioptische Untersuchungen

Habilitationsschrift

der Medizinischen Fakultät der Universität Zürich

zur Erlangung der Venia Legendi

für das Gebiet der Inneren Medizin

vorgelegt von

Dr. med. Martin Schmid

Oberarzt der Medizinischen Universitätsklinik Zürich

1966

Gnomes: the conception

- Nine founding members
- After the 1968 meeting: presentation of findings at EASL in Prague
- President, Dame Sheila Sherlock, suggested that the authors were like the “Gnomes of Zurich” exercising undue influence on the field of liver disease!
- Although probably intended to be derogatory it was adopted by the group and still used with affection to this day
- *Gnome* in Ancient Greek means “opinion”: highly appropriate for the endeavors of the group!


Jan De Groote
Leuven 1967


Peter Gedigk
Bonn 1967


Peter J Scheuer
London 1967


Valeer Desmet
Leuven 1967


Gerhard Korb
Weiden 1967


Martin Schmid
Zürich 1967


Hemming Poulsen
Copenhagen 1997


Heribert Thaler
Vienna 1967


Wilhelm Wepler
Kassel 1967

Gnomes: the early years

- Initially none of the group was native English speaker: all were male!
- Diversification! Amelia and non-Europeans
- Sponsorship (Falk etc): meeting went from frugal to more lavish
- Embraced a broad range of aspects of liver disease


Hans Popper
New York 1969/70


Leonardo Bianchi
Basel 1969


Amelia Baptista
Lisbon 1976


Roderick NM MacSween
Glasgow 1977


Kamal Ishak
Washington D.C. 1979


James Phillips
Toronto 1986


Helmut Denk
Graz 1987


Fred Gudat
Basel 1987


Francesco Callea
Rome 1992


Bernard Portmann
London 1992

Gnomes: philosophy and modus operandi

- Circulation of challenging cases on a specified topic
- Non-combatative!
- Submission of diagnoses
- Book of diagnoses provided by host on arrival: time of reckoning!
- Presentation of cases with discussion
- Round up session
- Possible position paper/need for more deliberation/new topics

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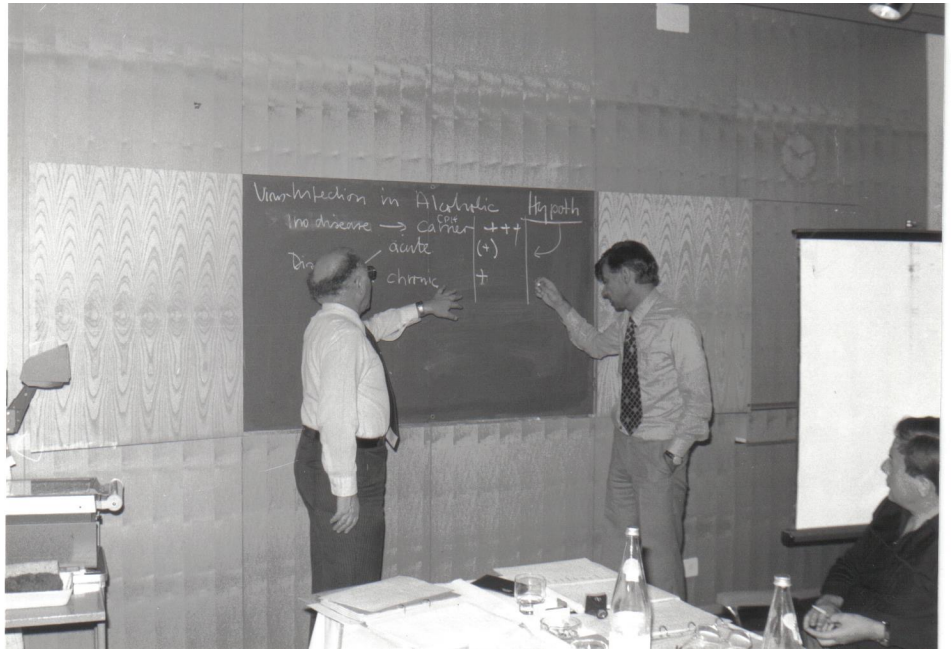

Gnomes: philosophy and modus operandi

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- Non-combative!
- Submission of diagnoses
- Book of diagnoses provided by host on arrival: time of reckoning!
- Presentation of cases with discussion
- Round up session
- Possible position paper/need for more deliberation/new topics
- Camaraderie and fun: later included social activities with ‘Gnome-mates’

Topics and outputs of early years

- Arguably greatest impact: discussions and papers on acute and chronic hepatitis
- Early treatise on DILI
- ARLD
- Biliary disease: autoimmune cholangiopathies
- Granulomas
- Epithelial tumours
- Grading and staging

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of the sex-distribution of the observed discordant sets that is difficult to reconcile with a genetic hypothesis.

The only genetic hypotheses which do seem compatible with the observations on twins are those which invoke low penetrance. For example, a single dominant gene of penetrance 0·1 would account for the observed recurrence-rate in siblings. Assuming independence of the probabilities of penetrance in genotypically concordant members of a twin set, such a model would predict a concordance-rate of only about 5% in monozygous sets and about 2·5% in dizygous sets. These rates would be undetectable in our series, either in terms of extent of loss of twin sets or of disturbance of the sex-distribution of observed surviving sets. However, penetrance-rates of this order, to be compatible with the observed sibling risk of about 5%, imply rates of recurrence of the genotype in the sibship that are associated with single major genes rather than polygenes. This raises the problems associated with the single-gene hypotheses. For example, with respect to the above model, the dominant hypothesis is not an attractive one in a condition of this lethality, in the absence of evidence for increased fertility among unaffected persons who could carry and distribute the gene.

We cannot say that any of the observations are incompatible with genetic determination of familial risk—the available genetic models are too flexible for that—but there are serious difficulties with all the genetic hypotheses so far proposed. In addition to further data on twins and on half-siblings, the most crucial additional evidence on genetic possibilities seems likely to come from work with close relatives of affected children other than siblings—particularly aunts, uncles, and cousins. Inconclusive data on this question have been reported from a study which should ultimately provide valuable evidence.[8] In the interpretation of such data, however, it is important to keep in mind the striking variation in frequency of these anomalies with ethnic origin and socioeconomic status [5 28]—characteristics which, like genes, show familial aggregation.

What, then, can be offered in the way of positive evidence as to environmental determination of the familial risk? First, there is the almost incontrovertible evidence, referred to in our introduction, that some as-yet-unidentified enviromental agent does play a role in these malformations. Apart from viral agents which confer immunity to subsequent infections, it is difficult to think of any type of environmental agent which would not be expected to show increased risk in certain families. Viruses almost certainly are not involved in the great majority of instances of these particular anomalies. That the increased risk is not uniformly spread throughout all affected families but varies from family to family—as revealed in the further increase in risk in families in which two siblings have been affected—is a pattern which might be expected if the family clustering were environmentally determined.

Second, there is the observation in our data that the risk in affected families has declined, to a degree commensurate with the decline in prevalence of the anomalies in the general population. The evidence that the secular changes in the general population are environmentally determined and not due to any of the genetic mechanisms that might be invoked has been reviewed elsewhere.[6] The additional observation reported here indicates that the relevant environmental factors are also pertinent to familial cases; thus one cannot postulate that the non-familial cases are environmentally determined but the familial cases have more important genetic components. One can of course argue that there has been a general decline in the environmental factors responsible for the penetrance of the relevant genotype—in familial as well as non-familial cases—but that the familial recurrence still results from recurrence of the genotype. This argument cannot be definitively countered, but the demonstration of the importance of environmental factors in familial cases seems to make more attractive the hypothesis that this environment may itself be a recurrent or persistent one.

We thank the administrative, medical, and record-room staffs of the Providence Lying-In Hospital, Roger Williams General Hospital, St. Joseph's Hospital, Pawtucket Memorial Hospital, and the Woonsocket General Hospital for giving permission to use their records and for providing every assistance, in particular, Miss Gertrude Cahir, Mr. Harmon P. B. Jordan, Jr., and Dr. Frederic W. Ripley, Jr. of the Providence Lying-In Hospital; Dr. Lechaim Naggan and the hospitals involved for use of material collected by him in Boston; and Dr. Olli Miettinen for his advice and comments on the manuscript. The work was aided by a research training grant (5 T01 GM7) from the National Institute of General Medical Sciences, National Institutes of Health, U. S. Department of Health, Education, and Welfare, and by a grant from the William F. Milton fund of Harvard University.

Requests for reprints should be addressed to B. MacM.

A CLASSIFICATION OF CHRONIC HEPATITIS

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THE term chronic hepatitis covers a variety of conditions of widely differing clinical significance and pathological appearances. We have tried to clarify the subject by constructing a new classification, first discussed at the second meeting of the European Association for the Study of the Liver at Göteborg in September, 1967, and later in Zürich in July, 1968.

We have omitted the special entity of cirrhosis from the scheme, although we recognise that this can also be regarded as a chronic hepatitis.

The classification is based on pathological appearances,

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914 THE LANCET, OCTOBER 29, 1977

Occasional Survey

ACUTE AND CHRONIC HEPATITIS REVISITED

Review by an International Group*

We have defined two major subdivisions of chronic hepatitis—chronic persistent hepatitis (C.P.H.) and chronic aggressive hepatitis (C.A.H.).[1] The basis of the classification was histological, and its justification was the difference in prognosis between the two groups, particularly with respect to the development of cirrhosis. When we considered the morphological features and variants of acute viral hepatitis we were especially concerned with histological patterns which suggested a possible transition to chronic liver disease.[2] Knowledge of pathogenesis in acute and chronic hepatitis remains incomplete. To improve communication we have reviewed our experience and re-examined liver-biopsy specimens in patients with hepatitis of known outcome.

Piecemeal necrosis has been regarded as important in the progression from acute to chronic aggressive hepatitis. Piecemeal necrosis was not defined by De Groote et al.[1] but is usually described as necrosis of hepatocytes at the junction between inflamed connective tissue and parenchyma. Various workers have drawn attention to other forms of hepatocellular damage. Boyer and Klatskin[3] indicated the predictive value of confluent necrosis with bridging between vascular structures. Patients with bridging necrosis were more likely to die within months of the onset of acute viral hepatitis, or to acquire cirrhosis. Peters[4] discussed the importance of regeneration as a factor in the outcome of acute hepatitis, and emphasised the role of spotty necrosis (hepatocytolysis) in C.A.H. The pathogenesis of C.A.H. and the features which allow a pathologist to predict the outcome of an acute attack are controversial. Furthermore, the nomenclature of chronic hepatitis has not been uniformly used, and pathologists and clinicians have found it difficult to communicate and to agree on the meaning of any one term or diagnosis.

*The following members of the group participated in this study: Prof. L. Bianchi (University of Basle, Switzerland), Prof. J. De Groote and Prof. V. J. Desmet (Academisch Ziekenhuis St. Rafaël, Leuven, Belgium), Prof. P. Gedigk (University of Bonn, West Germany), Prof. G. Korb (Pathologisches Institut, Stadtkrankenhaus, Weiden/Opf, West Germany), Prof. H. Popper (Mount Sinai School of Medicine, New York, U.S.A.), Prof. H. Poulsen (Hvidovre Hospital, Denmark), Prof. P. J. Scheuer (Royal Free Hospital, London), Prof. M. Schmid (Stadtspital Waid, Zürich, Switzerland), Prof. H. Thaler (Wilhelminenspital, Vienna, Austria), and Prof. W. Wepler (Kassel, West Germany). The manuscript was prepared for publication by Professor Scheuer and Professor Thaler.

NOMENCLATURE OF CHRONIC HEPATITIS

The classification of chronic hepatitis continues to be based on morphological criteria (table I), although clinical information is often essential for diagnosis. Difficulties may arise when the time of onset of the disease is uncertain.

The idea that the term chronic aggressive hepatitis should be used to describe a morphological lesion and the term chronic active hepatitis should be restricted to a clinical syndrome[1] has not been universally accepted. Some physicians use chronic aggressive hepatitis as a final diagnosis, based both on morphological and clinical findings, while others have objected to the word "aggressive" because it may upset patients and relatives. The handbook[5] of the International Association for the Study of the Liver and the John E. Fogarty Center in Bethesda divide chronic hepatitis into chronic persistent hepatitis and chronic active hepatitis, but sanction chronic aggressive hepatitis as a synonym (table I). Although the term chronic persistent hepatitis is not used consistently, we believe it should be retained. Its definition and diagnosis are discussed later.

In 1971 Popper and Schaffner[6] proposed a new category, chronic lobular hepatitis (C.L.H.), to describe the liver lesion in those patients with prolonged hepatocellular dysfunction in whom the pathological changes were mainly confined to the lobules. These changes comprised spotty necrosis and inflammation as found in acute viral hepatitis. We do not regard piecemeal necrosis and bridging necrosis as part of the C.L.H. lesion. The term C.L.H. was not sanctioned by the Fogarty group, who regarded C.L.H. as an example of prolonged, unresolved, or persisting acute hepatitis, continuing for more than six months. In cases where portal inflammation was also a prominent feature, chronic persistent hepatitis was thought to be the appropriate diagnosis. The terms persistent viral hepatitis and unresolved viral hepatitis denote similar lesions.

The term carrier is most often used to describe symp-

TABLE I—FORMS OF CHRONIC HEPATITIS

Morphological categories	Terms recommended by international group, 1976[5]	Clinical terms in current use	Salient morphological features	Likelihood of direct progression to cirrhosis
C.P.H.	C.P.H.	C.P.H.	Portal inflammation	0
C.A.H.	C.A.H.	C.P.H., chronic active liver disease with subacute hepatic necrosis (when bridging present)	Portal and periportal inflammation, periportal necrosis	+ to ++
			Severe periportal lesion, or chronic hepatitis with bridging necrosis	++ to +++
C.L.H.	(Acute hepatitis lasting more than 6 mo)	Prolonged, persistent unresolved hepatitis, &c.	Predominantly intralobular inflammation and necrosis	0

C.P.H.=chronic persistent hepatitis.
C.A.H.=chronic aggressive or active hepatitis.
C.L.H.=chronic lobular hepatitis.

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Occasional Survey

ALCOHOLIC LIVER DISEASE: MORPHOLOGICAL MANIFESTATIONS

REVIEW BY AN INTERNATIONAL GROUP*

IN previous reports our group has reviewed the morphological features of acute hepatitis (viral and drug-induced) and chronic hepatitis.[1-4] We now review the histological patterns of alcohol-induced liver disease, defining those which are of value in establishing the diagnosis and outlining those which may be of prognostic significance. We pay particular attention to alcoholic hepatitis, a lesion whose morphological designation dates from the early 1960s[5,6] and whose characteristic cytological and histological features require precise definition. We also draw attention to other conditions in which the histological changes in liver-biopsy specimens may simulate alcohol-induced liver damage.

Biopsy material from patients with proven or suspected alcoholic liver disease was circulated among the members of the group, and this paper is based on the results of examination of this material, the presentation and discussion of these cases by us in joint conferences, and exchange of views arising from members' experiences of large numbers of cases.

HISTOLOGICAL DIAGNOSES IN ALCOHOLIC LIVER DISEASE

The most important alcohol-induced lesions which can develop in the liver are as follows:

1. Fatty liver.
Reversible; may be associated with lipogranulomas; clinical complications may include cholestatic episodes, acute liver failure, and Zieve's syndrome.

2. Alcoholic hepatitis.
Transition lesion in development of cirrhosis;† without cirrhosis probably reversible, but with varying degrees of residual fibrosis; essential lesion is liver-cell necrosis with related polymorphonuclear-leucocyte infiltrate; may be focal, diffuse, or massive in distribution; may cause acute liver failure and portal hypertension without cirrhosis.

3. Cirrhosis.
Irreversible; nearly always micronodular in the early stages.

4. Hepatocellular carcinoma.

The above histological diagnoses and the various features which comprise them show considerable individual variation and may be found in different combinations and to a variable extent in biopsy and necropsy material. Thus, alcoholic hepatitis is often accompanied by fat accumulation, and these two lesions may coexist in an established cirrhosis. Primary hepatocellular carcinoma probably arises only when cirrhosis has already developed,[7,8] and there is little evidence at present that alcohol is a direct carcinogen in either man or animals.[9]

ALCOHOLIC FATTY LIVER (STEATOSIS)

Accumulation of fat in the liver is a regular finding in association with any degree of continued alcohol intake and is thus an almost invariable finding in the early stages of alcohol abuse. There is often a striking lack of clinical symptoms, however, even in patients with massive hepatomegaly due to fat accumulation. Occasionally, simple massive steatosis may be accompanied by cholestasis or hepatic failure (see below). The presence of fat is not a *sine qua non* for a diagnosis of alcoholic liver disease. Its absence seems to be mainly determined by alcohol withdrawal before biopsy or necropsy; the fat which accumulates in the alcoholic's liver may, in contrast to some of the other types of steatosis, be quickly mobilised because of reduced[10] or altered dietary intake or because of other complicating illness. The fat accumulates preferentially in zones 3 and 2 of Rappaport's acinus (i.e., centrilobular and midzonal). In the most severely affected liver the steatosis is diffusely distributed. However, there can be considerable variation in the zonal distribution of fat, and so the distribution pattern is of no great value in distinguishing the fatty liver of the alcoholic from the fatty liver seen in other disease states.

The fat may be present in hepatocytes in two morphological forms: (i) macrovesicular (large-droplet) steatosis, in which the fat forms a single intracellular globule producing a variable degree of compression, with displacement of the cytoplasm and nucleus to the periphery of the cell; and (ii) microvesicular (small-droplet) steatosis, in which the fat forms multiple droplets, sometimes disposed around the nucleus, sometimes disposed at one cell pole, pushing the nucleus aside. The former is much commoner.

Lipogranulomas represent a focal inflammatory component in fatty liver.[11] They vary from irregular aggregates of lymphocytes and macrophages, occasional plasma cells and eosinophils and, rarely, neutrophils surrounding an extracellular fat droplet (fig. 1), through larger focal aggregates of similar inflammatory cells in which ceroid-containing macrophages and lipophages are more numerous, to true epi-

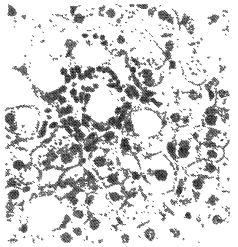

Fig. 1—Lipogranuloma.

A number of fat-containing hepatocytes are illustrated, and among these an aggregate of chronic inflammatory cells surrounds a fat space. (Haematoxylin and eosin, reduced by half from × 300.)

*MEMBERS OF THE GROUP:
Dr Amelia Baptista (Faculdade de Medicina de Lisboa, Portugal); Prof. L. Bianchi (University of Basel, Switzerland); Prof. J. de Groote, Prof. V. J. Desmet (Academisch Ziekenhuis St. Rafael, Leuven, Belgium); Prof. P. Gedigk (Pathologisches Institut der Universität, Bonn, W. Germany); Prof. G. Korb (Pathologisches Institut am Stadtkrankenhaus, Weiden, W. Germany); Prof. R. N. M. MacSween (University of Glasgow, Scotland); Prof. H. Popper (Mount Sinai School of Medicine, New York, U.S.A.); Prof. H. Poulsen (Patologisk afdeling, Hvidovre Hospital, University of Copenhagen, Denmark); Prof. P. J. Scheuer (Royal Free Hospital School of Medicine, London, U.K.); Prof. M. Schmid (Stadtspital Waid, Zürich, Switzerland); Prof. H. Thaler (Wilhelminenspital, Vienna, Austria); Prof. W. Wepler (Stadtkrankenhaus, Kassel, W. Germany).

†The development of fibrous in the absence of alcoholic hepatitis with a possible progression to cirrhosis is still controversial and not well established.

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Special Article

Histological grading and staging of chronic hepatitis

Kamal Ishak[1], Amelia Baptista[2], Leonardo Bianchi[3], Francesco Callea[4], Jan De Groote[5], Fred Gudat[6], Helmut Denk[7], Valeer Desmet[8], Gerhard Korb[9], Roderick N. M. MacSween[10], M. James Phillips[11], Bernard G. Portmann[12], Hemming Poulsen[13], Peter J. Scheuer[14], Martin Schmid[15] and Heribert Thaler[16]

[1]*Armed Forces Institute of Pathology, Washington, USA,* [2]*University of Lisbon, Lisbon, Portugal,* [3]*Hofstetten, Switzerland,* [4]*Servizio di Anatomia e Istologia Patologica, Spedali Civili, Brescia, Italy,* [5]*Department of Medicine, University of Leuven, Leuven, Belgium,* [6]*Institute for Pathology, University of Basel, Basel, Switzerland,* [7]*Department of Pathology, University of Graz, Graz, Austria,* [8]*Department of Pathology, University of Leuven, Leuven, Belgium,* [9]*Weiden, Germany,* [10]*Department of Pathology, Western Infirmary, University of Glasgow, Glasgow, UK,* [11]*Department of Pathology, Hospital for Sick Children, University of Toronto, Toronto, Canada,* [12]*Institute of Liver Studies, King's College Hospital, London, UK,* [13]*Frederiksberg, Denmark,* [14]*Watt, Switzerland,* [15]*Vienna, Austria*

THERE has been much recent discussion of the nomenclature and classification of chronic hepatitis. Since the proposal for a simple histological grading system by this group in 1968 (1), substantial information has been gained on the viral and other causes of chronic hepatitis, necessitating a review of the way in which liver biopsies in patients with chronic hepatitis are evaluated and reported. We do not wish to comment in detail on the most appropriate nomenclature for the 1990s, but recognise that several authors have recommended that the simple subdivision of chronic hepatitis into chronic persistent (CPH), chronic active (CAH) and chronic lobular forms (CLH) should no longer form the principal basis for classification (2–4). We agree with the view that, from the standpoint of the clinician, the most important single factor in diagnosis and evaluation of therapy is the cause of the hepatitis. Once the aetiology is established, however, histological data continue to provide the clinician with information essential for management of the patient.

The purpose of the present paper is to outline the morphological features which are of importance in undertaking grading and staging in chronic hepatitis and to make suggestions with respect to the semi-quantification of these features so as to produce a numerical index of histological activity. In addition, we suggest ways in which these data can be incorporated into histological reports so that the significance of the morphological lesions is meaningfully conveyed to the clinician. The ways in which this information can best be organised are also discussed in the following paragraphs.

Received 2 January 1995

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Grading and Staging

The concepts of grading and staging have traditionally been applied to neoplasms. Grading describes the degree of differentiation of a neoplasm, while staging denotes the extent of its spread. The same principles can be applied, however, with some modifications, to non-neoplastic conditions such as chronic hepatitis (3). Grading may be used to describe the intensity of necro-inflammatory activity in chronic hepatitis. Staging, on the other hand, is a measure of fibrosis and architectural alteration, i.e. structural progression of the disease; these features are currently believed to be the consequence of the necroinflammatory process.

The purpose of staging and grading is to record those histological features which are thought to indicate the severity and the progression of chronic hepatitis, and which might also be of prognostic significance. In addition, numerical scores can be attributed to both staging and grading, thus providing a semi-quantitative assessment of the observed histological features. The extent to which grading and staging can be accurately performed in a single biopsy is limited by potential sampling error, so that grading and staging must be regarded as approximations, depending to some extent on the size and quality of the sample. Where many biopsies are evaluated, however, as for instance in a clinical trial of an antiviral therapy, a greater degree of accuracy can be achieved. We do not

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- Stefan and I invited to Loch Lomond meeting 1995
- Importantly: invited back!
- Since then: meetings in 12 countries across 4 continents
- Inducted new members and farewelled others
- Broad spectrum of topics covered: vascular disease; liver in the immunocompromised; ductular reaction; unusual fatty liver diseases, tumour-like lesions; pathology of the sinusoids; acute liver injury; DILI

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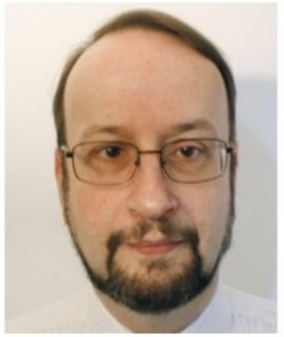
Tania Roskams
Leuven 1998

Luigi Terracciano
Basel 1998

Hans-Peter Dienes
Cologne 2002



Jean-Yves Scoazec
Lyon 2002

Zachary Goodman
Washington D.C. 2003

Pierre Bedossa
Paris 2003

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Gnomes: the ‘Brummie’ experience

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- As always: “the best meeting ever”
- Second Gnomes meeting in Birmingham in 2015: Acute hepatitis
- Great hospitality with a fabulous exposure to the culture of the Black Country and a very proud host exposing us to the home of the Baggies!

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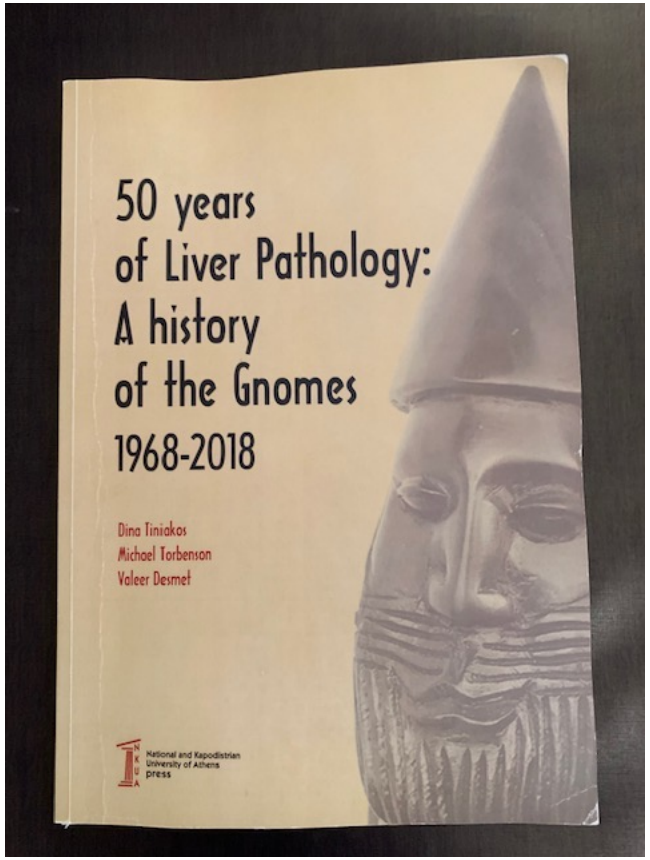
Gnomes: celebrating 50 years

- Anniversaries marked every 10 years since inception in 1968
- 50 years celebrated in Athens, hosted by Dina Tiniakos
- Book published to commemorate; in addition to history contains very philosophical musings from Valeer Desmet
- Subsequently summarized in paper by Mike Torbinson et al. on the value and success of small professional bodies in medicine



Gnomes: personal reflections – standout memories

- “Valeer Desmet’s final presentation in Paris 2010 on the eternal romance of the bile ducts”
- “Believing that we had a consensus on what we meant by ballooning”
- “Francesco’s meetings: personalized tour of the Sistine chapel and a recital by the Pope’s organist and the post-meeting tour in Tanzania of the Serengeti”
- “Lunch at Bel Canto in Paris: meal served by singing operatic students”
- “Too, too many!”

Gnomes: personal reflections – what epitomizes the group?

- “Respect, trust, camaraderie”
- “Commitment, civility, friendship”
- “Sticking to the ethos of Hans Popper: All you need is a good H&E and a microscope connected to a good brain”
- “Knowledge, ethics, logical reasoning”
- “Scientific competence, loyalty and mutual respect”
- “The ability to talk frankly and freely about any aspect of liver pathology (and more besides) and to have differences of opinion without taking (or giving) offence!”

Gnomes' "magic"

This is a story about Gnomes, a successful group of pathologists who have lasted so long because they all have an Eros and Love for liver disease

Athens, May 2018

Could you imagine their passion?
Can you share their satisfaction?
For science, knowledge, correct diagnosis
In cases unique?

I can see them inside the liver
Digging the labyrinth of ducts
Ideas producing question each other
And searching for truth...
like Socrates would do....!

Gnomes is respect trust and loyalty,
Passion, thoughtfulness and collegiality,
Commitment, civility and friendship
With no antipathy at all to keep

Gnomes is a heart behind the microscope
A high level of competence
A critical mind respecting the founders
And respecting each other

This is a song for "Gnomes' magic" celebrating the 50th Anniversary Gnomes Meeting

D. Dougenis, D. Tiniakos

Selected papers on the history of the Gnomes

- Scheuer PJ (1997) What's in a Gnome? BMJ 315: 1668
- Scheuer PJ (1999) The Gnomes: an adventure in hepatopathology. Ann Diagn Pathol 3: 134-9
- Reuben A (2002) Where are the Gnomes of yesteryear? Hepatology 35: 1554-7
- Torbenson M et al (2021) Fifty years of impact on liver pathology: a history of the Gnomes. Virchows Archiv 478: 191-200

Gnomes: Stefan's unique contributions

- Thoughtful and inciteful
- Always wonderfully prepared
- Logical, clear and inspiring presentations
- A great eye for detail
- Humble, incredibly polite and always very supportive
- Finished each talk with an annual progress report on the success (?) of the Baggies

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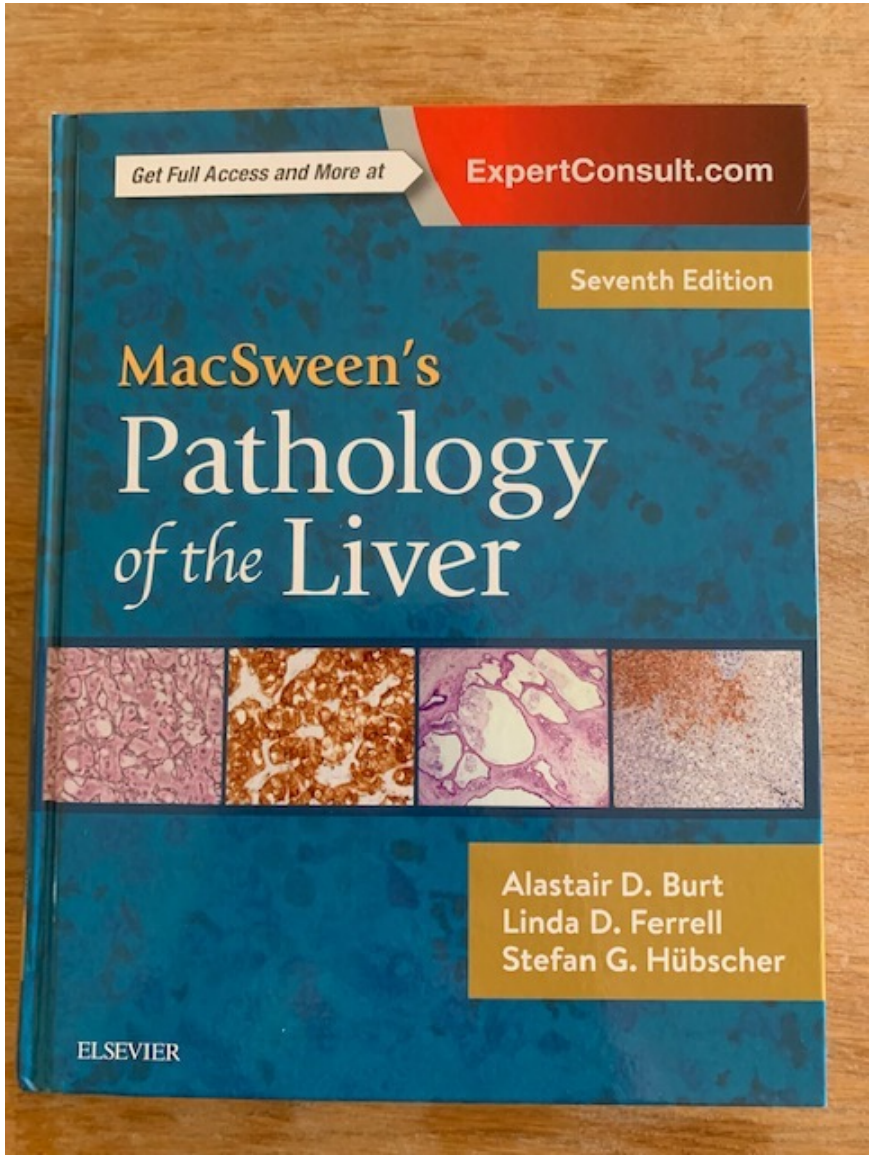
Stefan and POL

- Initial POL authored by Roddy, Peter Scheuer and Peter Antony
- Proof reading of First edition!
- “If it’s not in MacSween it’s not in the liver”
- Now into POL8: out late 2022
- Stefan co-editor of POL7 and 8



Farewell from Gnomes 2022

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