FECHORIAS, TRAVESURAS Y CURIOSIDADES EN LA LITERATURA ANATOMO-PATOLOGICA

Juan Rosai, M.D.

Zaragoza
20 Mayo 2011

FABRICATION OF DATA

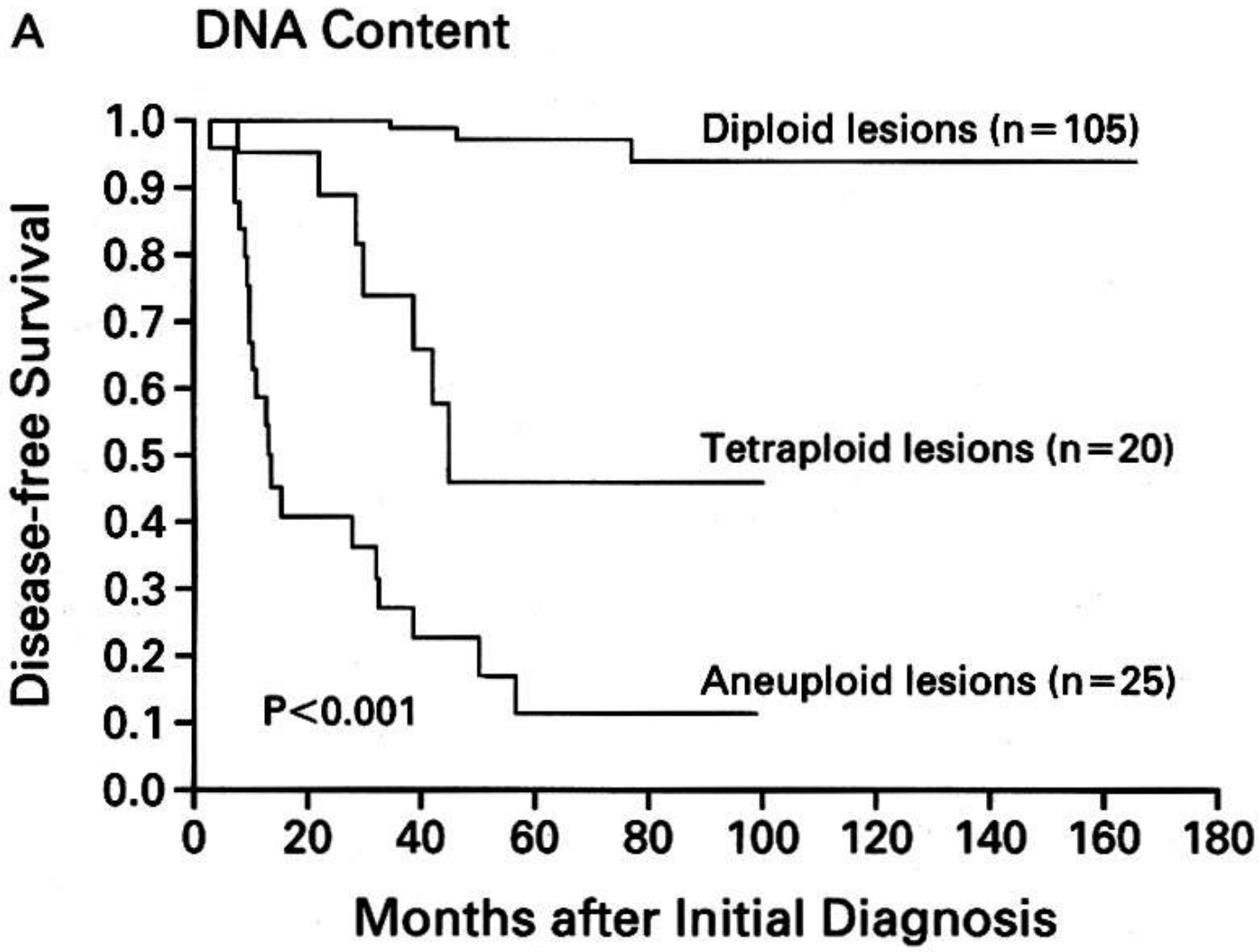
Hospital investigation reveals long-running fraud

In what could be one of the largest ever cases of medical-research fraud, a prominent anaesthesiologist has been accused of fabricating data in at least 21 papers over 13 years.

NATURE|Vol 458|19 March 2009

The New England Journal of Medicine

DNA CONTENT AS A PROGNOSTIC MARKER IN PATIENTS WITH ORAL LEUKOPLAKIA


A
DNA Content
Disease-free Survival
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
Diploid lesions (n=105)
Tetraploid lesions (n=20)
Aneuploid lesions (n=25)
P<0.001
0
20
40
60
80
100
120
140
160
180
Months after Initial Diagnosis

SCIENTIFIC MISCONDUCT

Fraud Upends Oral Cancer Field, Casting Doubt on Prevention Trial

The world of oral cancer research is reeling after one of its stars, [redacted] admitted this week through his attorney to falsifying data in three seminal papers published by top medical journals. A fourth paper is under suspicion after editors at the *New England Journal of Medicine* (*NEJM*) found that it contains a pair of duplicate images. For one of the papers, in *The Lancet*, [redacted] also appears to have claimed funding from a nonexistent grant.

The revelations have put on hold a multimillion-dollar oral cancer prevention trial, sponsored in part by the U.S. National Cancer Institute. The affair has also raised questions about whether researchers in multi-institutional collaborations should do more to

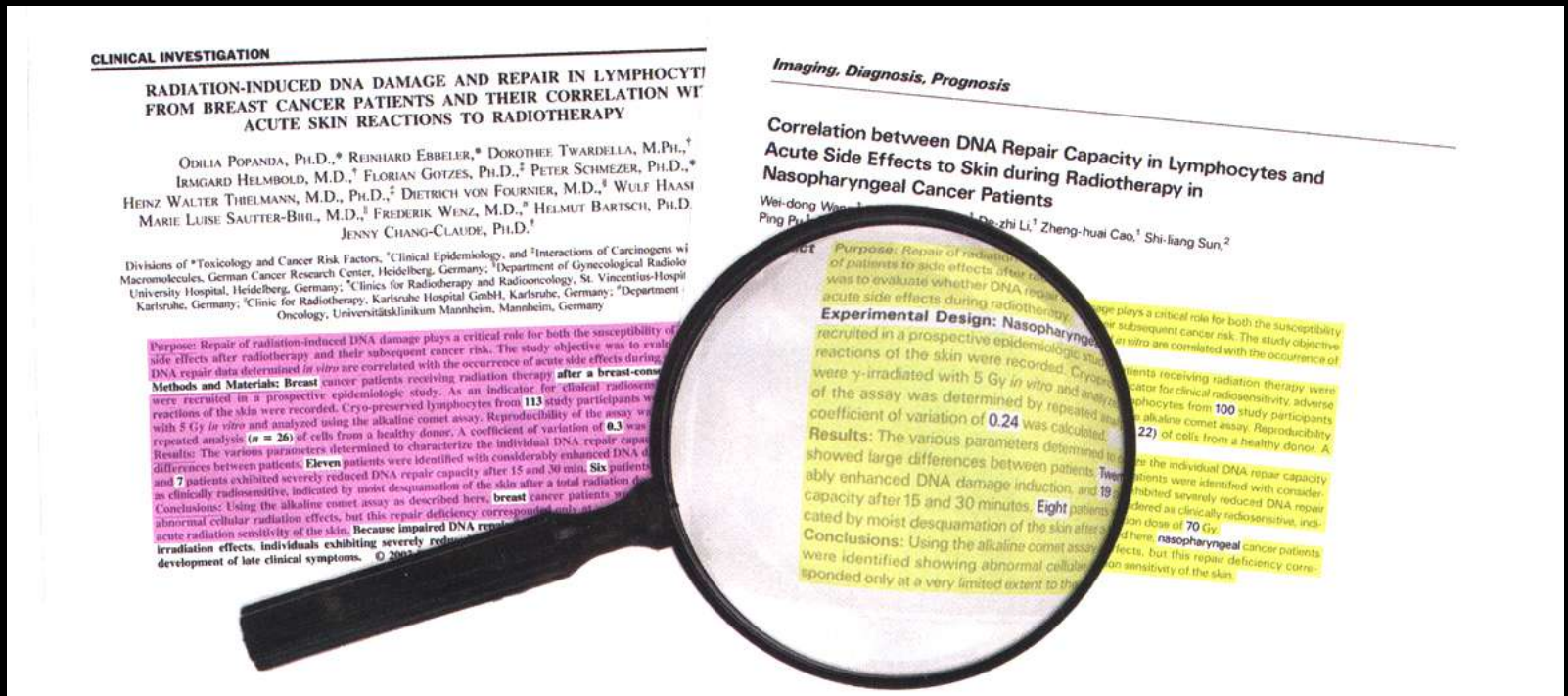
Fraud exposed. Cancer researcher [redacted] acknowledged faking data in three of these papers, and journal editors found a duplicated image in the fourth.

says, "are among the most important findings expected in a couple of months. "We don't have any suspicions that the other authors knew," says Stein Vaaler, director of strategy at the hospital, which has already found that hundreds of patient records were fabricated in the *Lancet* paper.

Some papers in question identified those at greatest risk of oral cancer, a disease often preceded by noncancerous mouth lesions. Just 20% to 30% of individuals with lesions develop oral cancer, confounding prevention efforts.

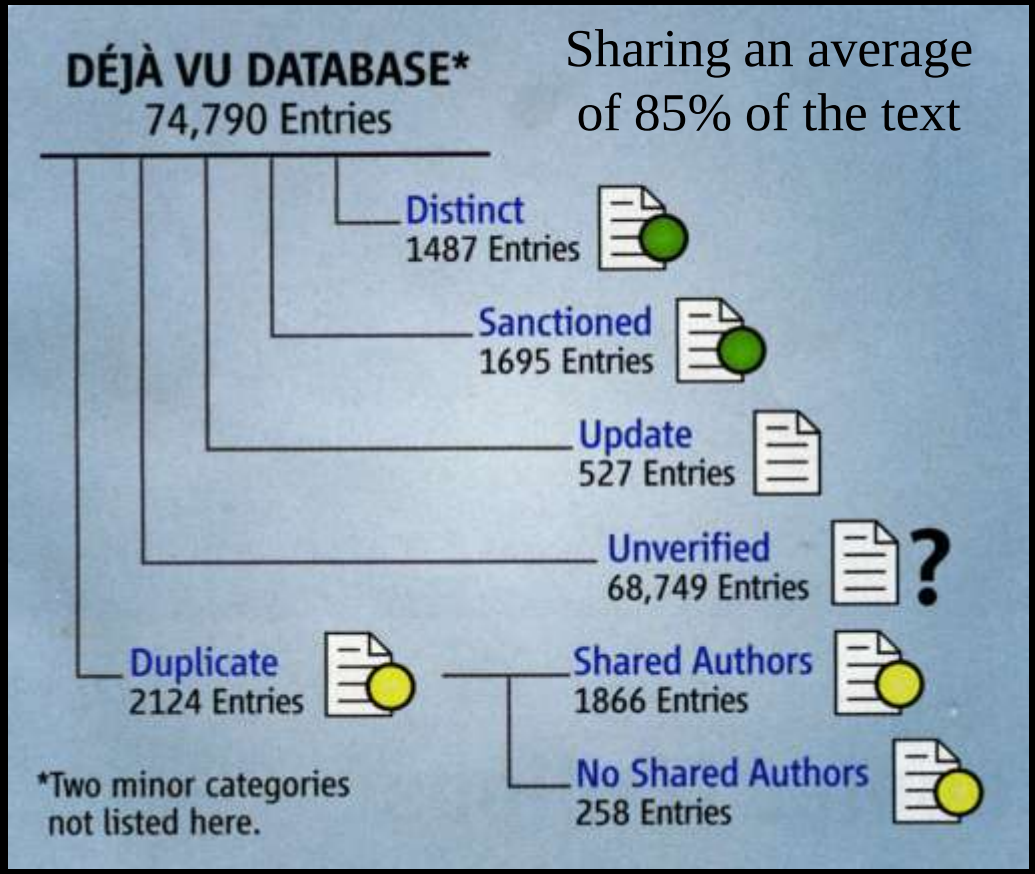
The earliest paper to contain false data, according to [redacted] attorney, Erling Lyngtveit, appeared in *NEJM* in April 2004. It reported that 26 of 27 individuals with aneuploid mouth lesions, so called because they contain abnormal numbers of chromosomes, developed aggressive oral cancer and were more likely to die of the disease than were those with other types of lesions. Lyngtveit confirmed that [redacted] did not have access to death information on which the study's conclusion was based. ([redacted] is currently on sick leave and

PLAGIARISM

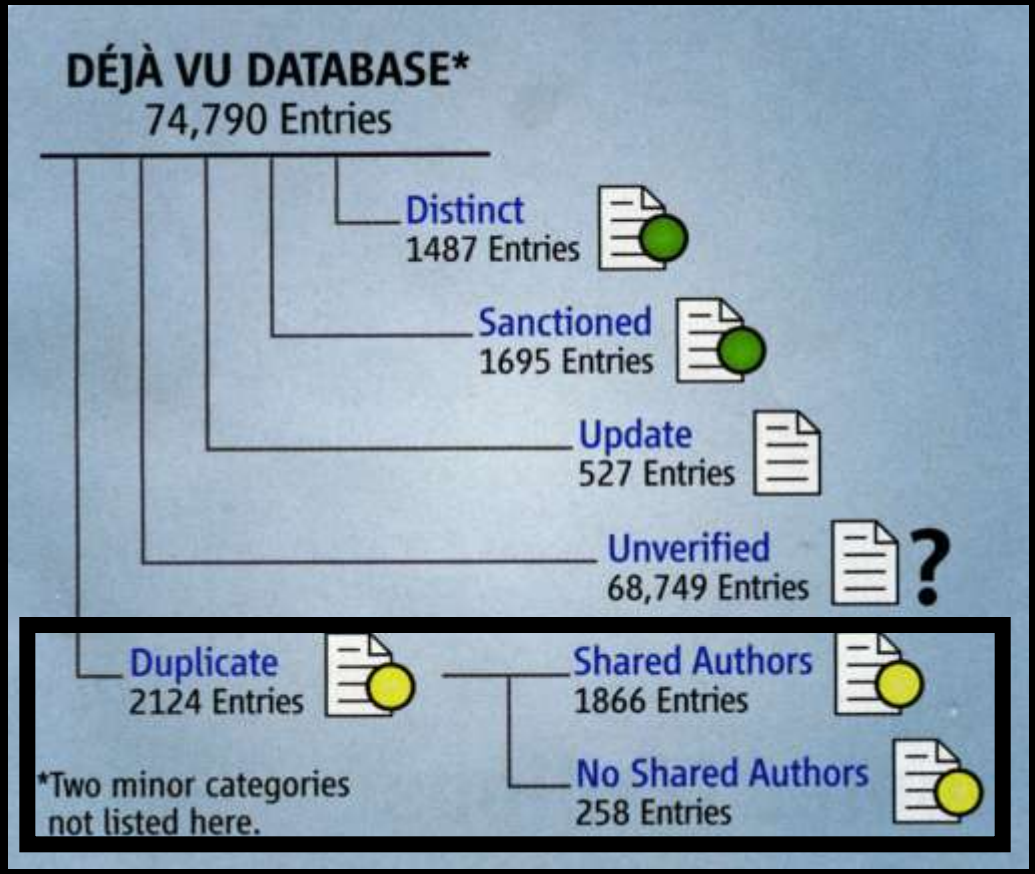


Plagiarism Sleuths

Riding high. Skip Garner, with one of his horses, runs Déjà vu and receives dozens of tips—and complaints.



Sharing an average of 85% of the text


DÉJÀ VU DATABASE*
74,790 Entries
Distinct
1487 Entries
Sanctioned
1695 Entries
Update
527 Entries
Unverified
68,749 Entries
?
Duplicate
2124 Entries
Shared Authors
1866 Entries
No Shared Authors
258 Entries
*Two minor categories
not listed here.

MUC2 Is a Molecular Marker for Pseudomyxoma Peritonei

Mod Pathol 2002;15(9):958–972

Pseudomyxoma Peritonei Is a Disease of MUC2-Expressing Goblet Cells

(Am J Pathol 2002, 161:551–564)

Myoepithelial Differentiation in High-grade Invasive Ductal Carcinomas With Large Central Acellular Zones

HUM PATHOL 30:1134-1139.

Large, Central Acellular Zones Indicating Myoepithelial Tumor Differentiation in High-Grade Invasive Ductal Carcinomas as Markers of Predisposition to Lung and Brain Metastases

The American Journal of Surgical Pathology *24(2): 197–202, 2000*

correspondence

Journals: redundant publications are bad news

Publishing the same work twice is unethical and casts doubt on the integrity of research.

"What an incredible advantage would be if in all branches of science there were only a few but excellent articles"

A. Schopenhauer

- PAPER SPLITTING
- SALAMI SLICING
- LEAST PUBLISHABLE UNIT (LPU)

A study of malignant lymphomas and leukemias

I. Cancer 13: 513, 1960
II. Cancer 13: 520, 1960
III. Cancer 14: 21, 1961
IV. Cancer 14: 30, 1961
V. Cancer 15: 869, 1962
VI. Cancer 15: 882, 1962
VII. Cancer 17: 277, 1964
VIII. ASC/CAP, 1963
IX. Am J Pathol 44: 35a, 1964
X. Am J Clin Pathol 42: 517, 1964

Henry Rappaport

Contrary to the conclusion of recent publications, nothing new has been discovered that had not been described previously.

Kostich & Rappaport (1965)

Cancer 13: 602, 1960

THE NATURE AND ORIGIN OF SMOOTH MUSCLE–LIKE NEOPLASTIC TISSUE IN RENAL TUMORS OF THE TUBEROUS SCLEROSIS COMPLEX

IN AN EARLY paper[7] I suggested that the elongated cells that usually constitute a prominent feature of the renal tumors of the tuberous sclerosis complex are neurilemoblastic.

In a later paper[9] I suggested that they are of neural crest origin.

Still later[10] I admitted that the same is true of some of the elongated cells found in honeycomb lungs of the tuberous sclerosis variety.

SUMMARY AND CONCLUSIONS

My former interpretation of all the smooth muscle–like tissue in these renal tumors as neurilemoblastic I now recognize to be wrong.

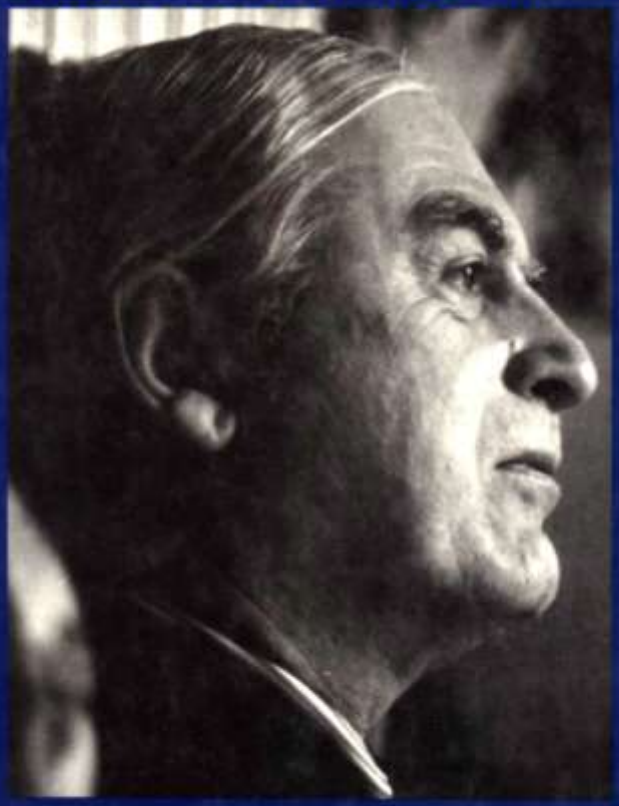
- Number of publications
- Journal of publication

"If we do not consistently take the trouble to judge authors by the content of their best papers rather than the number and location of those papers, the direction of science will come to be determined, however unintentionally, by a bunch of administrators and an editorial èlite. We shall have only ourselves to blame"

J. Davies (Nature, 2003)

PETER MEDAWAR

Memoir of a Thinking Radish

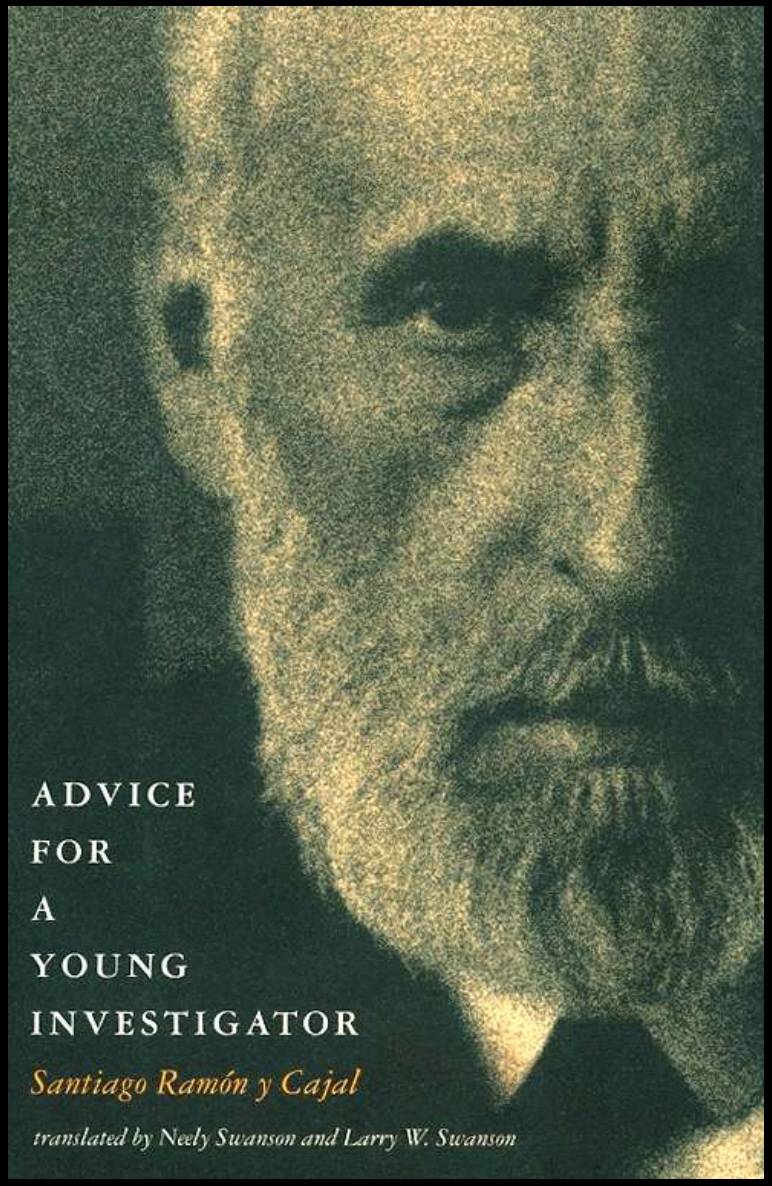


AN AUTOBIOGRAPHY

During the interview, Tizard asked me "How many papers have you written?"

"About ten to fifteen, I suppose", I replied,somewhat injudiciously.

"That's much too many, Medawar, he snapped at me, "much too many"


ADVICE
FOR
A
YOUNG
INVESTIGATOR
Santiago Ramón y Cajal
translated by Neely Swanson and Larry W. Swanson

"When we take up our pens to write a scientific article, let us bear in mind that it will probably be read by people who are too busy to waste time reading things they already know"

S. Ramón y Cajal

“rhetorical flatulence”

RULES FOR WRITING A SCIENTIFIC PAPER

(Mr Billings, Washington librarian)

(1) Have something to say

(2) Say it

(3) Stop once you said it

(4) Give the article a suitable title and order of presentation

How to Write and Publish a Scientific Paper

Robert A. Day

SECOND EDITION

- What is the problem ?
- How was it studied ?
- What where the findings ?
- What does it mean ?

RESULTS.-

The results are shown in Table 1.

"Upon awakening, the dinosaur was still there".

Augusto Monterroso

MORIBUND METAPHORS, EUPHEMISMS AND UNINTENTIONAL HUMOR

… vanishingly rare …

… it is tempting to assume …

… exquisite …

… decorating …

WHAT IS WRITTEN & WHAT IS MEANT

"It has long been known …"

I have not bothered to look up the original reference

"Three of the cases were chosen for further evaluation"

The others did not make any sense, so I threw them out

"These results will be reported at a later date"

Forget that one, baby !

"It is believed that …"

I think …

"It is generally believed that …"

A couple of other guys think so too

"It is clear that much additional work is required before achieving a complete understanding"

I sure did not understand a thing

"It is hoped that this work will stimulate further work in the field"

I hope that somebody smart picks up the subject and figures out what the hell is going on.

"235 patients were studied,

broken down by sex"

"A large mass of literature has accumulated on the cell membranes of red blood cells".

Clin Obstet Gynecol 2001 Sep;44(3):538-49

New innovations in cervical cancer screening.

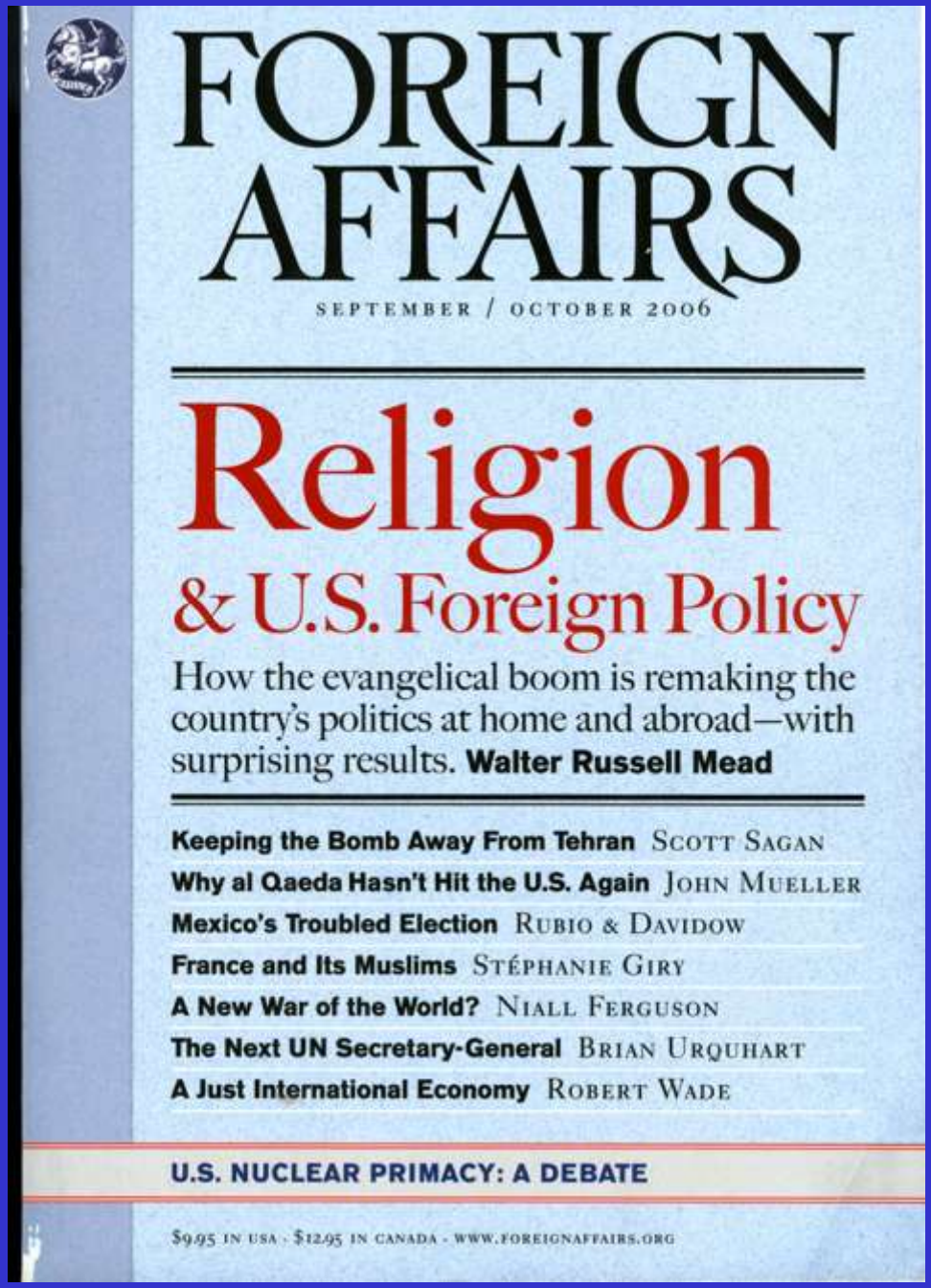
☐ 1: Best Pract Res Clin Gastroenterol 2002 Dec;16(6):933-43

The current future understanding of inflammatory bowel disease.

"The specialist who is trained but uneducated, technically skilled but culturally incompetent, is a menace"

David B. Truman
Dean, Columbia College

TYPOGRAPHICAL ERRORS ("TYPOS")


FOREIGN AFFAIRS
SEPTEMBER / OCTOBER 2006
Religion
& U.S. Foreign Policy
How the evangelical boom is remaking the country's politics at home and abroad—with surprising results. Walter Russell Mead
Keeping the Bomb Away From Tehran Scott Sagan
Why al Qaeda Hasn't Hit the U.S. Again John Mueller
Mexico's Troubled Election Rubio & Davidow
France and Its Muslims Stéphanie Giry
A New War of the World? Niall Ferguson
The Next UN Secretary-General Brian Urquhart
A Just International Economy Robert Wade
U.S. NUCLEAR PRIMACY: A DEBATE
$9.95 IN USA · $12.95 IN CANADA · WWW.FOREIGNAFFAIRS.ORG

DIFFUSE HYPERPLASIA OF GASTRIC ARGYROPHIL CELLS AND MULTIPLE CARCINOID TUMORS

An Historical and Ultrastructural Study

WILLIAM C. BLACK, MD, AND HEINZ E. HAFFNER, MD

Cancer 21: 1080, 1968

SEVENTH COMMANDMENT:

Thou shall not commit adultery

English Bible (1631)

SEVENTH COMMANDMENT:

Thou shall commit adultery

English Bible (1631)

THE ROYAL "WE"

"The use of "we" by a single author is outrageously pedantic"

R.A. Day

The American Journal of Surgical Pathology *17(6): 595–600, 1993*

Low-Grade Fibromyxoid Sarcoma

A Report of 12 Cases

Harry L. Evans, M.D.

In 1987, I reported two cases of low-grade fibromyxoid sarcoma, an indolent but metastasizing soft-tissue neoplasm with a deceptively benign histologic appearance (2). Since then, I have encountered additional examples in sufficient number to indicate that this tumor is not extremely rare. The present report adds 10 new cases of low-grade fibromyxoid sarcoma having at least 5 years follow-up to the original two.

Xeroderma pigmentosum mihi

M. Kaposi

STYLE

"The style of our work should be genuine, sober, simple and free of affectation, and it should reveal a preoccupation for order and clarity. Undue emphasis, oratory, and hyperbole should never enter into purely scientific writing"

S. Ramón y Cajal

"The stability of our civilisations, the despair of those who wish to change this world, rests upon the moulding of our ways of thought and modes of behaviour into set patterns, dependent upon the past experiences of the individual in his environment of home, school, and community life"

J D Hicks (1954)

"The stability of our civilisations, the despair of those who wish to change this world, rests upon the moulding of our ways of thought and modes of behaviour into set patterns, dependent upon the past experiences of the individual in his environment of home, school, and community life"

J D Hicks (1954)

SYNOVIAL SARCOMA OF THE TIBIA

J. DOUGLAS HICKS

Department of Morbid Anatomy,
The Royal Melbourne Hospital

J Pathol Bacteriol 67; 151, 1954

"There is no form of prose more difficult to understand and more tedious to read than the average scientific paper"

Francis Crick (1994)

"The contention that the tumours are not epithelial, but either 'synoviomas' or 'angioblastomas' is inadmissible; all of the eight examples which I have examined,like many of the reported cases, were plainly epithelial"

R. Willis (1967)

" The name 'neurogenic sarcoma' was introduced by the late James Ewing and publicized by Quick and Cutler and Stewart and Copeland of the Memorial Hospital.

It is to be deplored that such a careful observer as Shields Warren has expressed the opinion that the neurogenic sarcoma is an entity that can be recognized"

A. P. Stout (1948)

ABBREVIATIONS

PSUMP

STUMP

GIST

WHAFFT

SETTLE

CASTLE

PAPILLARY MICROCARCINOMA

PAPILLARY MICROTUMOR

PAPILLARY MICROCARCINOMA

⇩

PAPILLARY MICROTUMOR

PMT

PAPILLARY MICROCARCINOMA

⇩

PAPILLARY MICROTUMOR

PMT

PREMENSTRUAL TENSION

PAPILLARY MICROCARCINOMA

⇩

PAPILLARY MICROTUMOR

PMiT

- Electron Microscopic Features

- Electron Microscopic Features =
- EM Features

- EM Features =
- Electromagnetic Features

Anal Cell Pathol 2000;21(3-4):169-75

Experience with a dynamic inexpensive video-conferencing system for frozen section telepathology.

THE PAPER'S TITLE

Pathogenesis of Colloid (Pure Mucinous) Carcinoma of Exocrine Organs

Coupling of Gel-Forming Mucin (MUC2) Production With Altered Cell Polarity and Abnormal Cell–Stroma Interaction May Be the Key Factor in the Morphogenesis and Indolent Behavior of Colloid Carcinoma in the Breast and Pancreas

---

Involvement of the Chromosomal Translocation t(11;18) in Some Mucosa-Associated Lymphoid Tissue Lymphomas and Diffuse Large B-Cell Lymphomas of the Ocular AdnexaEvidence from Multiplex Reverse Transcriptase-Polymerase Chain Reaction and Fluorescence In Situ Hybridization on Using Formalin-Fixed, Paraffin-Embedded Specimens

"Did you read that paper in Nature on ribosome structure?"

"Yes, I read that paper, but I haven't finished the title yet".

- TITLE: 1,000
- ABSTRACT: 100
- WHOLE PAPER: 1

THE AUTHORS

International Journal of Gynecological Pathology
22:63–70, Lippincott Williams & Wilkins, Baltimore

Expression of E-Cadherin and Beta-Catenin in Trophoblastic Tissue in Normal and Pathological Pregnancies

H. W. Li, A. N. Y. Cheung, S. W. Tsao, A. L. M. Cheung, and W. S. O

Hum Pathol 1999 Jul;30(7):856-63

Wolffian adnexal tumor, so-called female adnexal tumor of probable Wolffian origin (FATWO): immunohistochemical evidence in support of a Wolffian origin.

Devouassoux-Shisheboran M, Silver SA, Tavassoli FA.

Please call me Dev

ARCHIVES

BRIEF REPORTS

A Sequel to Favorable and Unfavorable Histology

LOUIS P. DEHNER, MD
Lauren V. Ackerman
Laboratory of Surgical Pathology
Washington University Medical Center
St Louis, MO 63110

Veterans Administration Hospital,
St. Louis, Missouri

Veterans Administration Hospital, St. Louis, Missouri

JOHN COCHRAN

Veterans Administration Hospital,
St. Louis, Missouri

JOHN COCHRAN

John Cochran Veterans
Administration Hospital, St. Louis,
Missouri


WILLIAM OBER

WILLIAM B. OBER

- GHON BUT NOT FORGOTTEN
- FRIEDRICK ZHAN: WHAT IS MY LINE?
- EMIL ZUCKERKANDL AND HIS DELIGHTFUL LITTLE ORGAN
- BOTTOMS UP !

LEYDIG, SERTOLI, AND REINKE: THREE ANATOMISTS WHO WERE ON THE BALL

WILLIAM B. OBER AND CHE SCIAGURA

Pathol Annu 16 [Pt.1]: 1-13, 1981.

William B. Ober, M.D.
Director of Laboratories, Hackensack Hospital, Hackensack, New Jersey

Che Sciagura, D. Phil.
Professor of Tautology, Sacred Heart Institute of Theology, Siena, Italy

Tautology: Needless repetition of an idea, statement or word

GOLD MEDAL OF THE MECONIUM SOCIETY

"CHE' SCIAGURA !"

Voltaire: CANDID

Chapter 11: The history of the old woman

A GENE-EXPRESSION SIGNATURE AS A PREDICTOR OF SURVIVAL IN BREAST CANCER

MARC J. VAN DE VIJVER, M.D., PH.D., YUDONG D. HE, PH.D., LAURA J. VAN 'T VEER, PH.D., HONGYUE DAI, PH.D., AUGUSTINUS A.M. HART, M.SC., DORIEN W. VOSKUIL, PH.D., GEORGE J. SCHREIBER, M.SC., JOHANNES L. PETERSE, M.D., CHRIS ROBERTS, PH.D., MATTHEW J. MARTON, PH.D., MARK PARRISH, DOUWE ATSMA, ANKE WITTEVEEN, ANNUSKA GLAS, PH.D., LEONIE DELAHAYE, TONY VAN DER VELDE, HARRY BARTELINK, M.D., PH.D., SJOERD RODENHUIS, M.D., PH.D., EMIEL T. RUTGERS, M.D., PH.D., STEPHEN H. FRIEND, M.D., PH.D., AND RENÉ BERNARDS, PH.D.

21 authors

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Mutation of *FOXL2* in Granulosa-Cell Tumors of the Ovary

Sohrab P. Shah, Ph.D., Martin Köbel, M.D., Janine Senz, B.Sc., Ryan D. Morin, M.Sc., Blaise A. Clarke, M.B., B.Ch., Kimberly C. Wiegand, B.Sc., Gillian Leung, B.Sc., Abdalnasser Zayed, B.Sc., Erika Mehl, B.M.L.Sc., Steve E. Kalloger, B.Sc., Mark Sun, B.Sc., Ryan Giuliany, Erika Yorida, B.M.L.Sc. Steven Jones, Ph.D., Richard Varhol, M.Sc., Kenneth D. Swenerton, M.D., Dianne Miller, M.D., Philip B. Clement, M.D., Colleen Crane, B.Tech., Jason Madore, M.Sc., Diane Provencher, M.D., Peter Leung, Ph.D., Anna DeFazio, Ph.D., Jaswinder Khattra, M.Sc., Gulisa Turashvili, M.D., Ph.D., Yongjun Zhao, M.Sc., D.V.M., Thomas Zeng, M.Sc., J.N. Mark Glover, Ph.D., Barbara Vanderhyden, Ph.D., Chengquan Zhao, M.D., Christine A. Parkinson, Ph.D., M.R.C.P., Mercedes Jimenez-Linan, Ph.D., David D.L. Bowtell, Ph.D., Anne-Marie Mes-Masson, Ph.D., James D. Brenton, M.D., F.R.C.P., Samuel A. Aparicio, B.M., B.Ch., Niki Boyd, Ph.D., Martin Hirst, Ph.D., C. Blake Gilks, M.D., Marco Marra, Ph.D., and David G. Huntsman, M.D.

"No more than one author per patient in a clinical paper, …

and no more than one author per mouse in an experimental paper"

Number of authors on two 2001 papers announcing the draft sequence of the human genome.

523

Number of authors on two 2001 papers announcing the draft sequence of the human genome.

523

Number of authors on two 2001 papers announcing the draft sequence of the human genome.

Number of authors on 2010 paper of initial results from the Compact Muon Solenoid detector at the Large Hadron Collider.

523

Number of authors on two 2001 papers announcing the draft sequence of the human genome.

1,968

Number of authors on 2010 paper of initial results from the Compact Muon Solenoid detector at the Large Hadron Collider.

THE INFLATION OF RESULTS

[redacted] AS A PREDICTOR OF METASTATIC POTENTIAL IN MALIGNANT MELANOMA

A retrospective study of 188 cases with up to 12 years follow-up

CASES OF MELANOMA IN FILE 188

CASES OF MELANOMA IN FILE 188

- 34 melanomas in situ 154

CASES OF MELANOMA IN FILE 188

- 34 melanomas in situ 154
- 52 melanomas in radial growth phase 102

CASES OF MELANOMA IN FILE 188

- 34 melanomas in situ 154
- 52 melanomas in radial growth phase 102
- 7 thin melanomas in vertical growth phase 95

CASES OF MELANOMA IN FILE 188

- 34 melanomas in situ 154
- 52 melanomas in radial growth phase 102
- 7 thin melanomas in vertical growth phase 95
- 4 special melanoma types 91

CASES OF MELANOMA IN FILE 188

- 34 melanomas in situ 154
- 52 melanomas in radial growth phase 102
- 7 thin melanomas in vertical growth phase 95
- 4 special melanoma types 91
- 3 cases technically inadequate 88

CASES OF MELANOMA IN FILE 188

- 34 melanomas in situ 154
- 52 melanomas in radial growth phase 102
- 7 thin melanomas in vertical growth phase 95
- 4 special melanoma types 91
- 3 cases technically inadequate 88
- 13 cases with follow-up not available 75

CASES OF MELANOMA IN FILE 188

- 34 melanomas in situ 154
- 52 melanomas in radial growth phase 102
- 7 thin melanomas in vertical growth phase 95
- 4 special melanoma types 91
- 3 cases technically inadequate 88
- 13 cases with follow-up not available 75
- 24 cases with "incomplete" follow-up 51

[redacted] AS A PREDICTOR OF METASTATIC POTENTIAL IN MALIGNANT MELANOMA

A retrospective study of 188 cases with up to 12 years follow-up

[redacted] AS A PREDICTOR OF METASTATIC POTENTIAL IN MALIGNANT MELANOMA

A retrospective study of ~~188~~ 51 cases with up to 12 years follow-up

TABLE 1. *Immunohistochemical results*

Diagnosis	Fli-1 positive (%)
Angiosarcoma (all)	20/22 (91)
Epithelioid	11/13 (85)
Conventional	9/9 (100)
Hemangioendothelioma (all)	11/12 (92)
Epithelioid	8/9 (89)
Retiform	2/2 (100)
Dabska tumor	1/1 (100)
Hemangioma (all)	7/7 (100)
Capillary	3/3 (100)
Spindle cell	2/2 (100)
Epithelioid	2/2 (100)

TABLE 1. *Immunohistochemical results*

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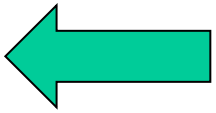
Dabska tumor	1/1 (100)
Hemangioma (all)	7/7 (100)
Capillary	3/3 (100)
Spindle cell	2/2 (100)
Epithelioid	2/2 (100)



"33.3% of the mice were cured by the test drug;

33.3% were unaffected by the drug;

the third mouse got away"

FOOTNOTES

2

Choosing an electron microscope

There is little doubt that the high cost of electron microscopes and exaggerated notions about the complexities and difficulties of operating and maintaining them have deterred many from embarking on diagnostic electron microscopy. Such fears are, perhaps, justified if one is thinking about a 'state of the art' high-performance instrument (resolution better than 0.2 nm, cost $175 000–$400 000) fitted with numerous frills and facilities rarely if ever needed by the pathologist.

Diagnostic electron microscopy and, indeed, even most research studies on pathological tissues of man and experimental animals require nothing more than the ability to look at and photograph ultrathin sectioned material at quite low or modest magnifications. For this a medium-performance instrument (resolution around 0.7 nm, cost $70 000–$100 000) is the instrument of choice and not just a poor substitute. The reasons for this are as follows*.

In ultrathin sections of plastic-embedded material the best resolution attainable is about 2 nm† (Meek, 1970). Thus, the medium-performance instrument with its 0.7 nm resolution is more than adequate for our needs. Certainly the better than 0.2 nm resolving power of the high-performance instrument can offer no additional advantage for the specimen sets the limit to the resolution‡.

The human eye can at close distance separate individual lines of a ruled grating 0.075 mm apart (Meek, 1970). However, this is a very taxing performance and requires optimum contrast and lighting conditions to attain. A more realistic figure for the resolving power of the human eye is about 0.25 mm, because this can be attained without much strain under normal situations (Meek, 1970).

As noted earlier the resolving power attainable in ultrathin sections is about 2 nm. It follows, then, that in a print (electron micrograph) at ×100 000 (where 2 nm is magnified to 0.2 mm) one should be able to, and in fact can, discern objects or distances between objects 2 nm or greater. The corollary to this is that if nothing less than 2 nm is resolved, however high the magnification, then any enlargement much above ×100 000 will be 'empty magnification' – that is to say, an increase in size without more resolution or information. Now one can comfortably obtain a print at ×100 000 magnification by using an electron optical magnification (i.e. magnification in the negative) of ×20 000–×40 000 followed by an optical enlargement (i.e. with a photographic enlarger) of ×5–×2.5, and this is well within the capability of the medium-performance electron microscope.

It is said§ that one should keep the electron optical magnification high and the optical enlarge-

*In this discussion I am omitting from consideration special studies such as virological studies carried out on negatively-stained preparations or the developing field of electron probe X-ray analysis (Ghadially, 1979), which has a great future in research and diagnostic pathology. For such studies the more sophisticated microscope is necessary.

†This figure by Meek (1970) refers to the best attainable resolution, other figures quoted in the literature are more pessimistic. For example, Ottensmeyer and Pear (1975) state: 'images of sections of biological tissue rarely show biologically significant resolution of 30 Å or better', while Carlemalm *et al.* (1982) state: 'The limit of resolution for relevant biological detail is rarely finer than 4.0 nm'.

‡It is also regrettably true that a majority of high-resolution instruments are not, in fact, operated at anywhere near their capacity or performance for reasons connected with poor maintenance and lack of operator skills.

§I have no serious quarrels with this statement. My own personal preference is to produce negatives which do not as a rule need to be enlarged more than ×5, but any 'half-decent' negative should be able to stand a ×10 enlargement.

ment low, for if the latter is more than about ×6 or at the most ×8, this might lead to a situation of 'empty magnification'*. The limiting factors here are the thickness of the photographic emulsion (the thinner the better) and grain size (depends mainly on film speed and development) in the negative. Experience, however, shows that more often the limit on enlargement is set by the fact that few negatives are perfectly focused and sharp enough† to stand much enlargement. The amazing amount of detail that is available in a perfectly focused negative taken (by Dr Newstead) at quite low magnification is demonstrated in *Figures 8* and *9*.

Figure 8 shows fragments of isolated myelin sheath in a subcellular fraction of brain photographed at the electron optical magnification of ×2500 and an optical enlargement of ×6. *Figure 9* was produced by enlarging a part of the same negative ×64. This clearly demonstrates that the trilaminar membrane structure and the intermediate line are recorded even in a negative taken at so low a magnification. While one would not wish to suggest that this kind of performance is recommended procedure, it does show that the top magnification available with a medium-performance microscope is, if anything, too much and not too little for our needs.

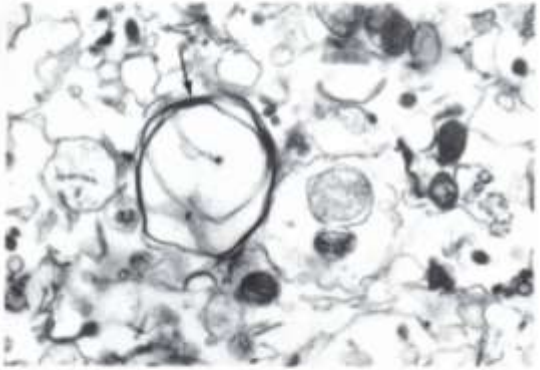


Figure 8 Isolated myelin from a synaptosome pellet. Arrow indicates portion of the myelin sheath which is shown much enlarged in *Figure 9*. Electron optical enlargement ×2500. Optical enlargement ×6. Final magnification ×15 000. (Electron micrograph supplied by Dr J. D. Newstead)

Figure 9 Isolated myelin from a synaptosome pellet. Same negative as in *Figure 8*. The area indicated by arrow in *Figure 6* is shown much enlarged here. Note the demonstration of the trilaminar membrane structure within the myelin. Electron optical magnification ×2500. Optical enlargement ×64. Final magnification ×160 000. (Electron micrograph supplied by Dr J. D. Newstead)

A glance at the magnification of electron micrographs published in books and pathology journals will convince the reader that most biological work is done at quite low magnifications and that electron micrographs published at a magnification of ×100 000 or more are quite rare. Thus, most electron microscopic negatives are taken at a magnification between ×1500 and ×15 000; only rarely does one take an electron micrograph at ×20 000 or ×30 000.

Such considerations, combined with the extra cost, effort and technical expertise needed to keep more sophisticated instruments operational, should

*There is nothing wrong or wicked about 'empty magnification' as long as we know what we are doing and do not delude ourselves, for there are times when a larger print may be deemed essential for publication purposes or for exhibition at a scientific meeting.

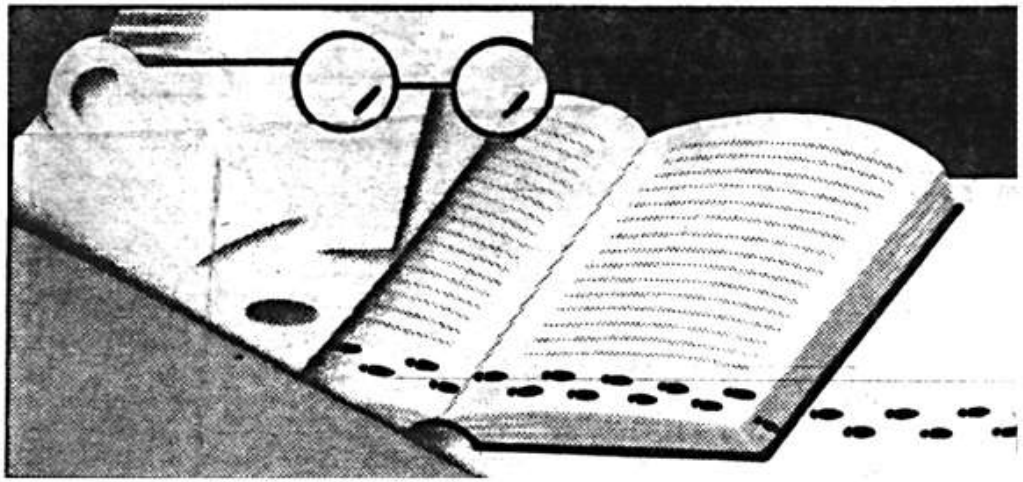
†Unsharp negatives may be the product of poor operator skills or poor microscope performance (e.g. due to contamination, stage or specimen drift, astigmatism, etc.) and resolution. The ability to resolve the trilaminar structure of the cell membrane is a good rough and ready test of these parameters. When the trilaminar structure is visualized in electron micrographs it shows that the whole system (operator, microscope and specimen) is working at an acceptable level of performance. However, it must be remembered that the specimen must also be suitable for this purpose. It takes little imagination to see that unless the cell membrane is cut at right angles to its surface and the segment of the membrane lying in the thickness of the section is straight and does not bend or fold, there will be little chance of demonstrating the characteristic trilaminar structure. The ideal specimen for this purpose is an accurately placed transverse section through intestinal microvilli, for in this instance a straight tubular segment of the microvillus lies vertically within the section thickness. Yet another factor is intensity of staining. The trilaminar structure is difficult to demonstrate in heavily stained sections. The particles of stain deposit can reach a diameter of 2–3 nm which would easily 'obliterate' the lucent lamina in the cell membrane.

Reverend John Hodgson's
HISTORY OF NORTHUMBERLAND

It contains a footnote 265 pages long

Where Have All the Footnotes Gone?

By Gertrude Himmelfarb



"Having to interrupt an enjoyable reading to go down to a footnote is like having sex on the third floor of your house and having to interrupt it to go down to see who is ringing the bell"

Joseph Epstein

REFERENCES

Cancer 48:1615–1622, 1981.

Benign Spindle Cell Breast Tumor

CYRIL TOKER, MD,* CHIK-KWUN TANG, MD,† JOHN F. WHITELY, MD,‡ SAMUEL W. BERKHEISER, MD,§ AND RAYMOND RACHMAN, MD‖

THIS REPORT will document a distinctive type of breast tumor. The lesion is histologically characteristic and is remarkable in that in three of the four cases presented, the patients were men (Table 1).

category of mesenchymal tumors capable of diverse lines of differentiation—fibroblastic, myofibroblastic, leiomyomatous, and lipomatous.

Whatever their histogenesis, recognition of these curious, frequently multicentric, tumors as benign neoplasms is of paramount importance.

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1. Enzinger FM, Harvey A. Spindle cell lipoma. *Cancer* 1975; 36:1852–1859.

MULTIPLE BENIGN SARKOID OF THE SKIN.

By Professor C. Boeck,
Christiania.

(*J Cutan Genitourin Dis* 1899;17:543-550)

"I have seen two cases in Norway, one in a female many years ago of whose case I have no notes"

"A typical case was presented at a dermatological congress in London (1896), but I do not know by whom"

ILLUSTRATIONS

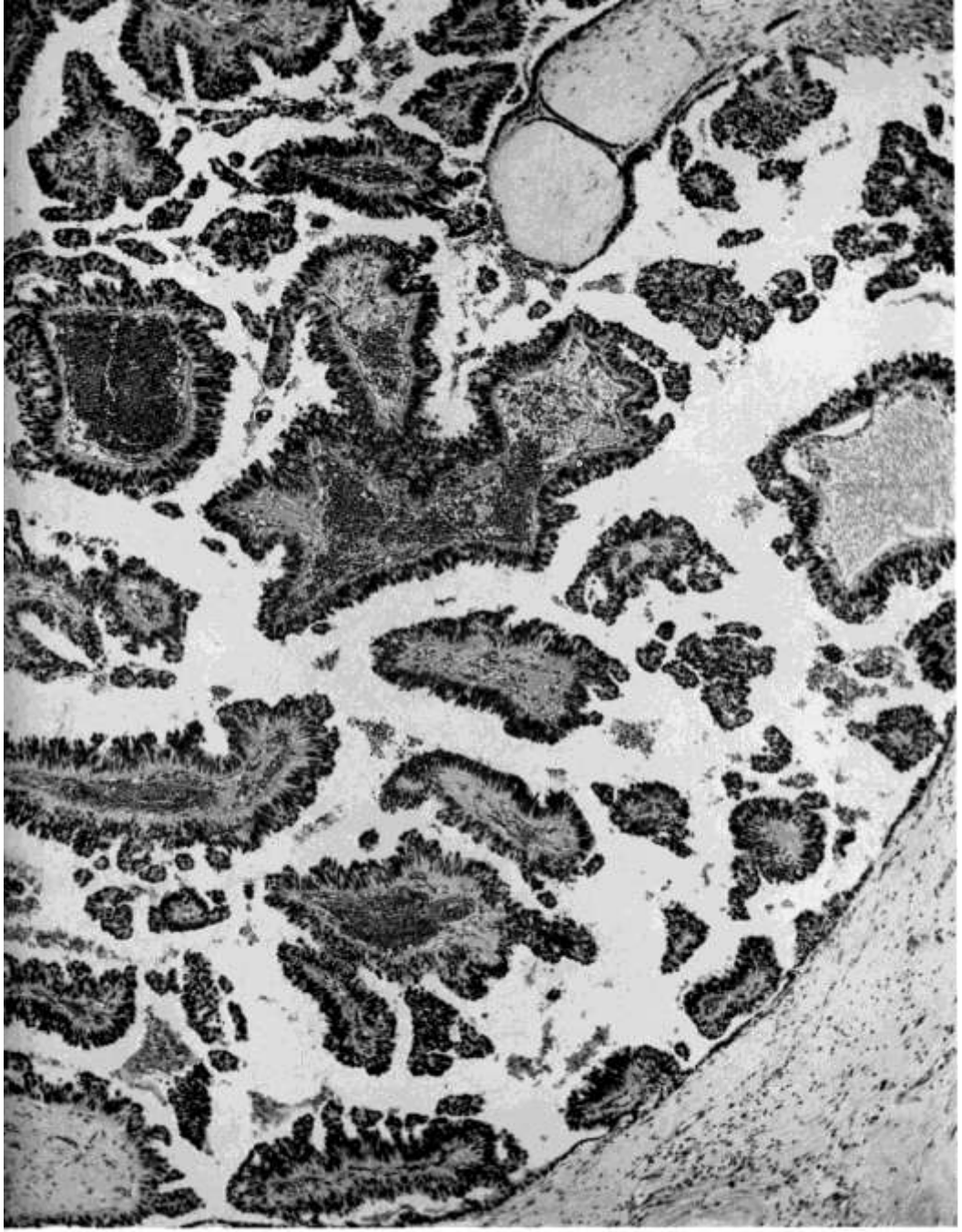


Fig. 2.—Papillary pattern of this tumor with connective tissue stalks in papillary carcinoma is well demonstrated. A.F.I.P. 55-2815 (×100) Case 2).

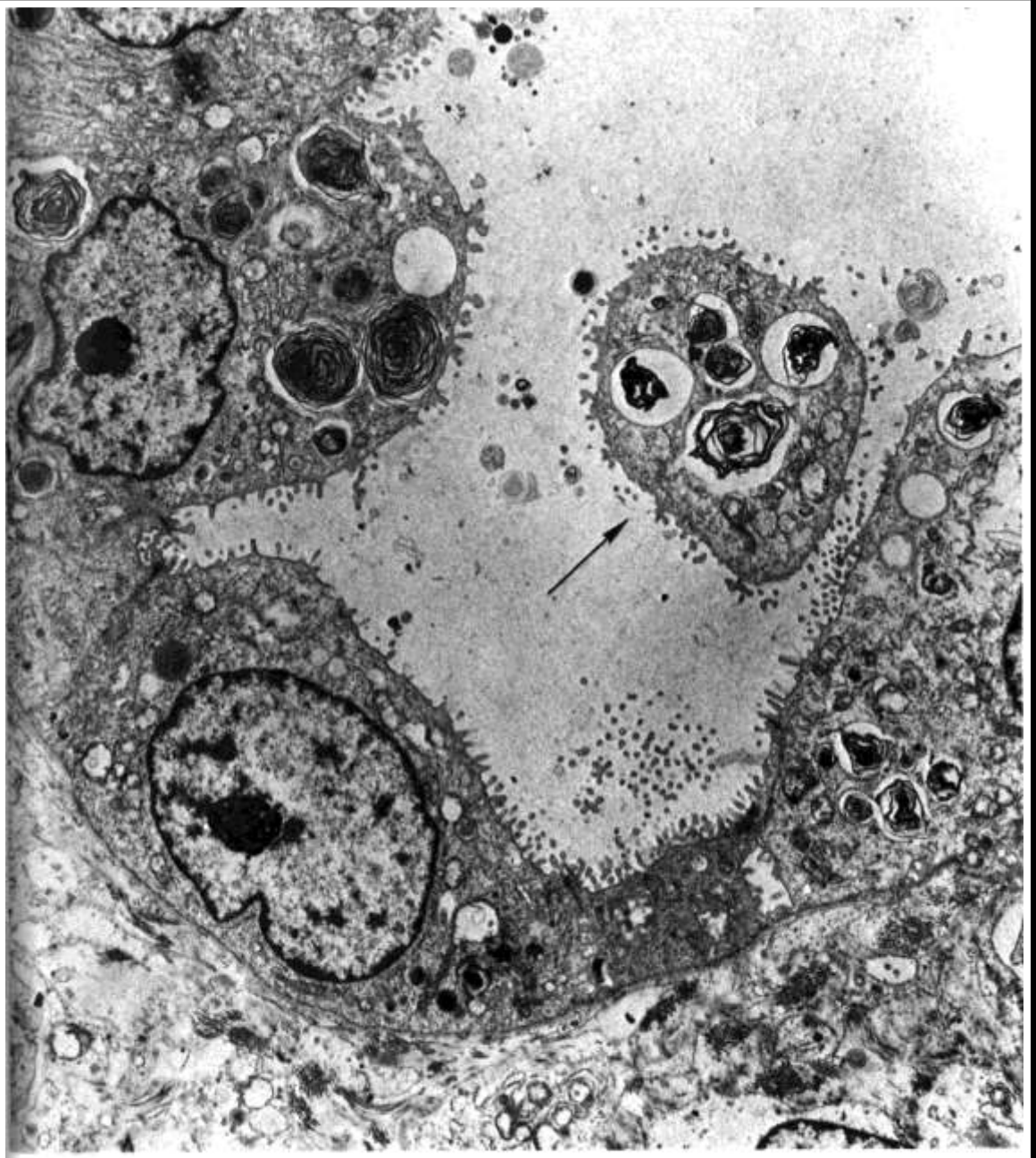
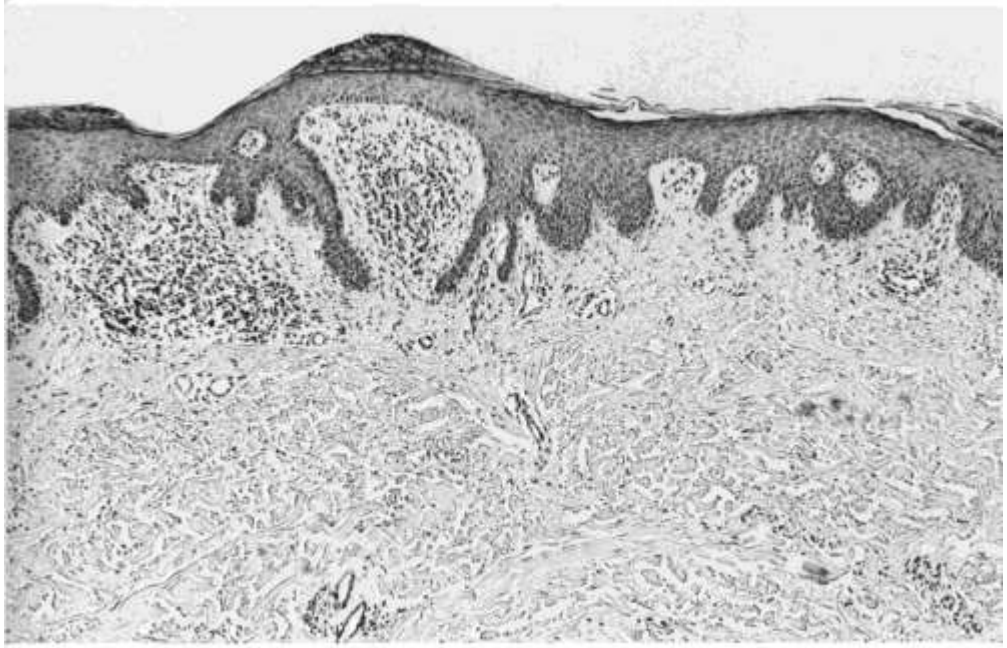
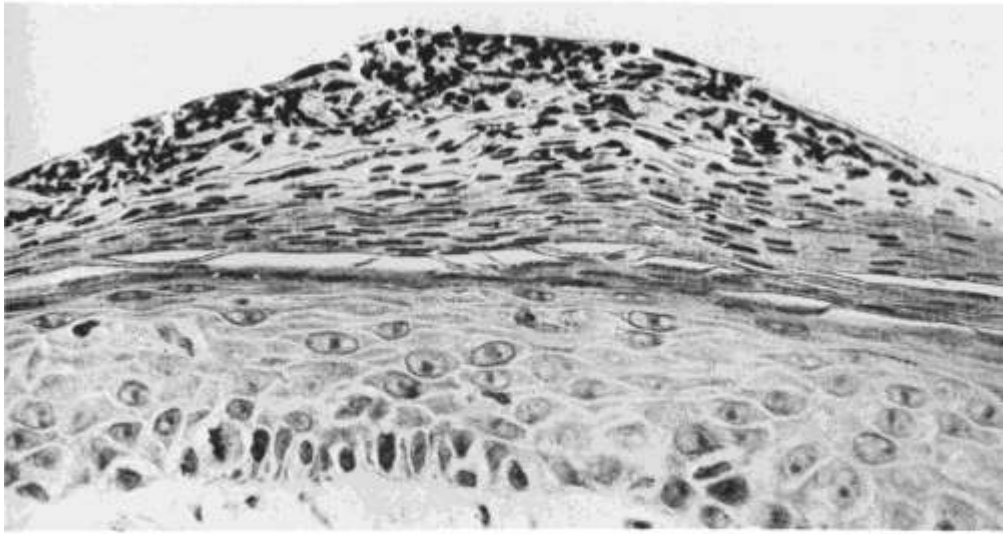


FIGURE 3
An electron micrograph of the original tumor showing a portion of an acinar structure lined by cuboidal cells and featuring the morphological characteristics of granular pneumocytes. Clearly identifiable are the well-formed microvilli on the free surfaces of the tumor cells and the intracytoplasmic inclusion ("lamellar") bodies composed of concentrically arranged lamellae. A detached granular pneumocyte lies free within the acinar lumen (*arrow*). (6,900×)



(*a*)



(*b*)

FIGURE 2
(*a*) Three well-defined foci of parakeratosis containing neutrophils may be seen in this lesion of guttate (eruptive) psoriasis. Not the edematous papillary dermis which contains tortuous capillaries and a sparse inflammatory-cell infiltrate. (*b*) Higher-powe view of the mound of parakeratosis containing neutrophils in guttate psoriasis pictured in Fig. 2*a*.

who were AWPD or DOD, all three had a recurrence after a disease-free interval ranging from 3 to 5 years, with a mean time to recurrence of 44 months (range, 36–60 months).

Clinical Details of Four Patients With Noninvasive Implants and Adverse Outcome

Patient No. 1

The first patient underwent a TAH and BSO with staging for a pelvic mass and was found to have an MPSC involving bilateral ovaries and numerous noninvasive implants. Information on the presence or absence of residual disease was not know; however, she received no postoperative therapy. This patient presented with extensive recurrent disease 5 years later, and histologic examination revealed a combination of invasive and noninvasive implants. The patient underwent debulking and chemotherapy but continued to progress and was DOD 71 months after initial diagnosis.

Patient No. 2

The second patient underwent a USO, omentectomy, and lymph node dissection, and was found to have an MPSC with noninvasive implants involving the omentum. The presence or absence of residual disease was not known; however, the patient received no further therapy. After a 3-year period, she developed a recurrence and underwent debulking with multiple peritoneal excisions/biopsies followed by treatment with cisplatin and Cytoxan. Histologic examination revealed invasive implants. She was well for another 7 years when she complained of abdominal pain and was found to have a large abdominal mass. Fine-needle aspiration of the mass revealed a low-grade carcinoma similar to the primary MPSC. She was treated with Taxol but her disease progressed with the development of massive ascites and intra-abdominal carcinomatosis with liver involvement. She was DOD 153 months after her initial presentation.

Patient No. 3

The third patient underwent a TAH and BSO with staging and was found to have an APST involving bilateral ovaries with two noninvasive implants. The presence or absence of residual disease was not known. After 3 years, the patient developed her first recurrence. Histologic examination of the recurrent lesions revealed a combination of invasive and noninvasive implants. In the subsequent 9 years, the patient had eight laparotomies for resection of persistent/recurrent disease and was treated with cisplatin, Taxol and ^{32}P. She subsequently had a colostomy for bowel obstruction and is alive with disease at 144 months.

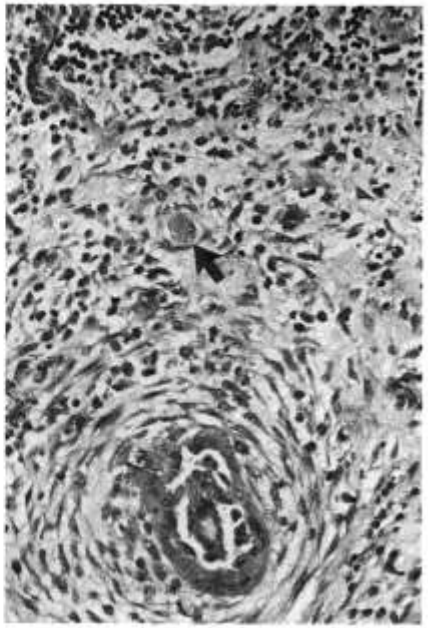


FIG. 8. Noninvasive implant with single cells in stroma. A gland-like structure lined by cells with abundant cytoplasm is present in a granulation tissue-like stroma with moderate chronic inflammation. The epithelial cells of the implant appear to merge with the surrounding stromal cells. A similar-appearing cell with abundant cytoplasm is in the stroma above the gland (arrow).

Patient No. 4

The fourth patient underwent a TAH and BSO with staging and was found to have bilateral primary MPSCs with numerous florid desmoplastic noninvasive implants. Residual disease was present at initial surgery and the patient underwent a second debulking procedure. The patient was DUC 6 months after her primary surgery.

Correlation of Primary Tumor With Implant Type

Of the 27 patients with MPSC, 18 had invasive implants, eight had noninvasive implants, and one had both invasive and noninvasive implants. Thus, 19 of the 27 patients with MPSC (70%) had invasive implants. Of the 33 patients with APST, eight had invasive implants, 21 had noninvasive implants, and four had both invasive

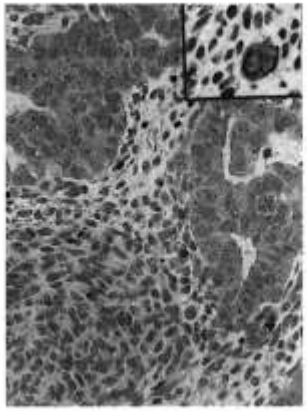


FIG. 6. Mesonephric adenocarcinoma with spindle cell differentiation. (**Inset**) Pancytokeratin immunoreactivity in spindle cell component.

plasms derived from mesonephric remnants only rarely involve the female genital tract. Nogales[16] conceptualized two distinct areas of the wolffian system: an upper zone that includes the rete ovarii and a lower zone encompassing mesonephric derivatives of the cervix and vagina. Tumors arising in these locations are generally distinct morphologically. Female adnexal tumors of probable wolffian origin (FATWO) typically arise along the upper zone of the Wolffian duct,[6,10] whereas mesonephric adenocarcinomas and the recently described malignant mesonephric mixed tumor[4] arise from the lower zone. The latter two entities have been reported mostly as isolated case reports.[1,2,7,11,13,19,20] To our knowledge, other than the current series, only one other series of eight such neoplasms has been compiled.[4] The actual incidence of these tumors is difficult to determine in part because of historical confusion in the literature, with clear cell carcinomas and yolk sac tumors once categorized as mesonephric carcinomas.[7,8,23] Mesonephric-derived neoplasms, however, may be more common than what is suggested by the number of cases reported, owing to their potential misclassification as mullerian-derived tumors or florid mesonephric hyperplasia.[4,7]

Our initial interest in cervical mesonephric adenocarcinomas derived from our recent study of FATWOs, in which the immunophenotype of this neoplasm was compared with that of mesonephric structures in the upper and lower zones of the wolffian system.[6] Whereas the immunohistochemical profile of FATWOs was found to have greatest homology with mesonephric remnants at the level of the rete ovarii, in the current study, we found the immunophenotype of mesonephric adenocarcinomas to resemble that of mesonephric remnants more closely at the level of the cervix and vagina. Specifically, the pattern of expression of EMA and CK 7 and the uniform nonreactivity for ER and PR in our study were unique to

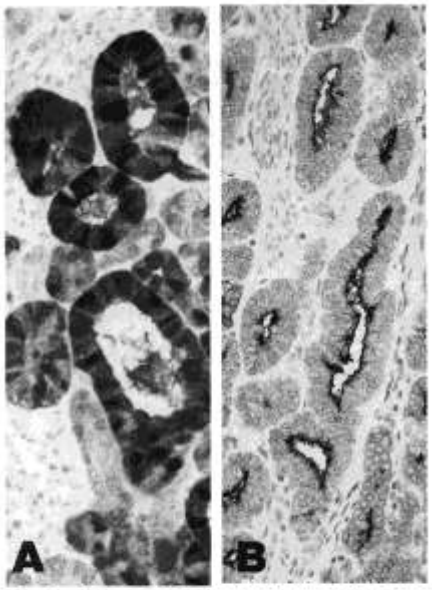


FIG. 7. Mesonephric adenocarcinoma. (**A**) Diffuse nuclear and cytoplasmic immunoreactivity for calretinin. (**B**) Epithelial membrane antigen immunostaining with uniform apical membrane staining.

mesonephric remnants and adenocarcinomas. Although no particular immunohistochemical features have been previously known to be specific for wolffian differentiation, from our current and previous[6] study, a fairly distinctive immunohistochemical profile has emerged. We

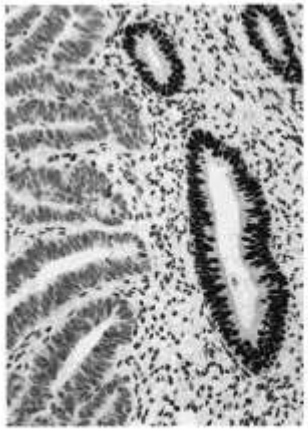


FIG. 8. Intense nuclear immunostaining for estrogen receptor in benign endocervical glands and stroma contrasts with the uniform lack of reactivity in mesonephric adenocarcinoma.

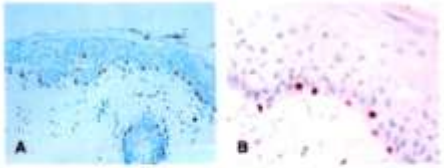


FIG. 1. Mi transcription factor protein (Mitf) antibody staining of normal skin from face. (**A**) Note the positively stained melanocytes in the basal layer of the epidermis and in the follicle. (**B**) Note the positive Mitf nuclear staining of melanocytes contrasting with the Mitf-negative nuclei of the keratinocytes.

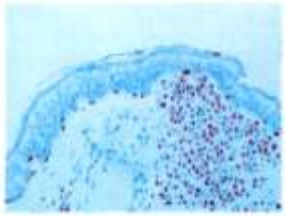


FIG. 2. Dermal nevus demonstrating Mi transcription factor protein nuclear staining. Note adjacent, positively stained basal melanocytes.

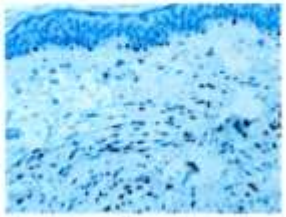


FIG. 3. Dermal spindle melanocytes of blue nevus demonstrating Mi transcription factor protein-positive staining.

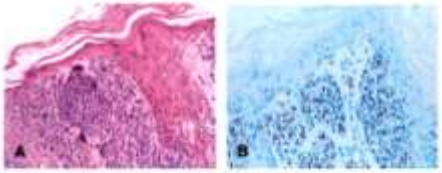


FIG. 4. (**A, B**) Mi transcription factor protein immunopositive staining in pigmented spindle cell nevus.

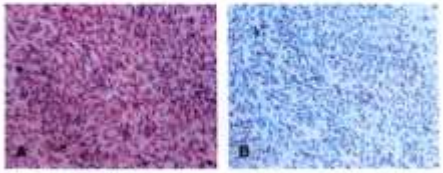


FIG. 5. (**A, B**) Cellular blue nevus demonstrating Mi transcription factor protein nuclear staining of the fascicles of spindle melanocytes.

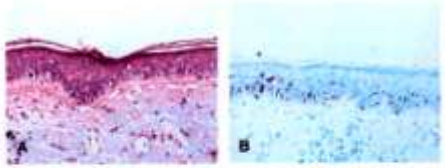


FIG. 6. (**A, B**) Malignant melanoma in situ, lentigo maligna type. Mi transcription factor protein nuclear staining highlights a lentiginous proliferation of atypical melanocytes filling the basal layer of the epidermis.

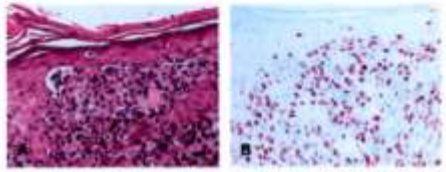


FIG. 7. (**A, B**) In situ and invasive malignant melanoma with a predominant epithelioid cell morphology demonstrating Mi transcription factor protein nuclear reactivity of melanoma cells of both the in situ and the invasive components. Pagetoid spread of melanoma cells is highlighted.

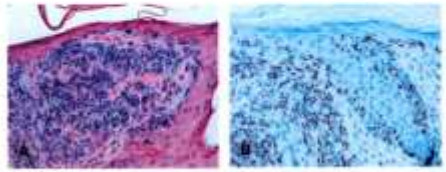


FIG. 8. (**A**) In situ and invasive malignant melanoma with a predominant spindle cell morphology demonstrating Mi transcription factor protein (Mitf) nuclear reactivity of melanoma cells of both the in situ and the invasive components. (**B**) The spindle cell morphology of the nuclei are readily apparent in the Mitf staining.

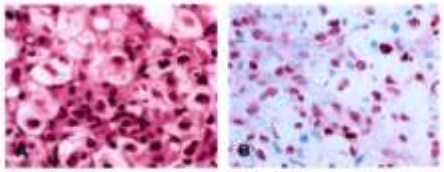


FIG. 9. (**A, B**) Mi transcription factor protein nuclear immunopositivity in malignant melanoma with balloon cell features.

strated consistent immunopositivity. In neurotized nevi, the melanocytes with prominent schwannian differentiation demonstrated a decrease in the number and intensity of positive nuclei with the non-neurotized component

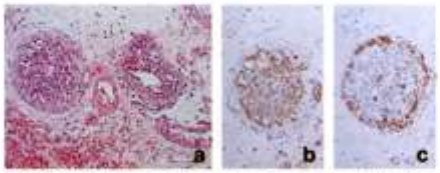


FIGURE 4. High magnification of an intravascular proliferation of endothelial cells showing glomerulus-like capillary structures in the transverse colon. a, Hematoxylin and eosin staining. b and c, Immunohistochemistry of the glomerular structure. Intravascular proliferating cells positive for CD31 (b) form capillary-like lumina, often associated with pericytic cells positive for α-smooth muscle specific actin (c).

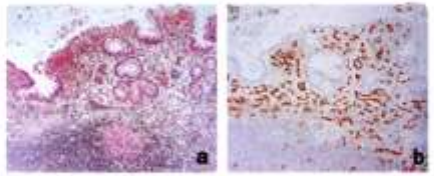


FIGURE 6. The mucosa and submucosa of the terminal ileum, demonstrating hemorrhage, edema, and proliferation of small vessels. Hyperplastic lymphoid tissue with a hyalinizing mass is also apparent. a, Hematoxylin and eosin staining. b, Immunohistochemistry for CD31. Proliferating small vessels are not associated with intravascular endothelial cell proliferation.

tures but demonstrate anastomosing capillary loops. In the Dabska tumor,[8,24] papillary endothelial tufts within newly formed vascular structures are seen, rather than within preexisting blood vessels. In malignant angioendotheliomatosis,[19,27] the intravascular proliferation is noncohesive, with large and atypical cells expressing the leukocyte common antigen and B cell antigen. In the present case, the tightly packed capillaries associated with ulcers consisted of slightly atypical cells with occasional mitosis and anti-Ki67 positivity. However, the possibility of malignant tumors, including Kaposi sarcomas and angiosarcomas, was precluded by the organized structure of single layered CD31-positive endothelial cells and the α-smooth muscle actin-positive fibrous cell lining.

Because of the presence of multiple lesions, several other specific vascular syndromes, such as the Rendu-Osler-Weber disease, blue rubber bleb nevus syndrome, Maffucci syndrome, and Klippel-Trenaunay syndrome, were also considered in differential diagnosis. In particular, several reports showed gastrointestinal bleeding due to blue rubber bleb nevi from intestinal arteriovenous malformations[2,18,22] and from diffuse cavernous hemangiomas with the Klippel-Trenaunay syndrome,[17] but intravascular lesions have not been observed in these cases.

The pathogenesis of RAE is unknown, but many cases have a setting of coexistent systemic disease. Immunologic factors are also suggested as possible factors in the pathogenesis.[16] The patient in the present case had granuloma-forming enteritis suggestive of Crohn's disease and massive lower gastrointestinal bleeding, a relatively rare complication occurring in 0.6% to 2% of Crohn's disease patients.[4,6,10,21] While the pathogenesis of Crohn's disease remains obscure, various etiologic factors, including infective agents such as enterobacteria or viruses, abnormalities of immune mechanisms, and environmental risk factors such as foods and smoking, have been postulated. There is a possibility that some of these factors or secondary infection at the floor of an open ulcer might play a role in the occurrence of RAE. However, the significance of Crohn's disease and/or massive lower gastrointestinal bleeding for the pathogenesis of RAE remains to be defined.

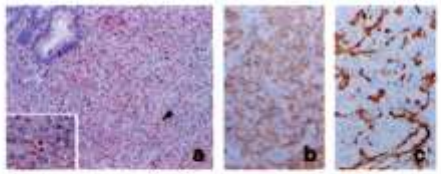


FIGURE 5. High magnification of a solid proliferation of spindle cells at the base of an open ulcer of the transverse colon. a, Note mitosis (arrowhead, insert). b and c, Immunohistochemistry of spindle cells. CD31-positive endothelial cells are densely packed (b), with pericytic cells positive for α-smooth muscle actin lining the endothelium (c).

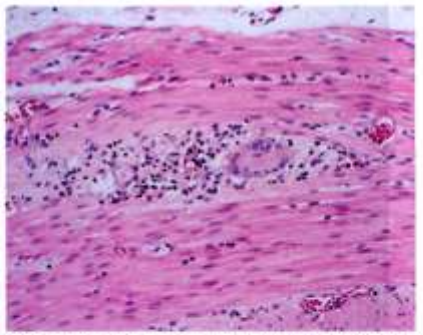
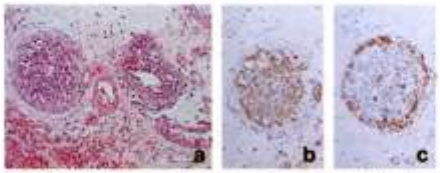


FIGURE 7. A granuloma with a multinuclear giant cell in the muscle layer of the terminal ileum.



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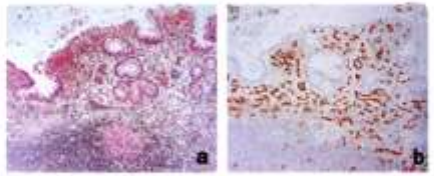

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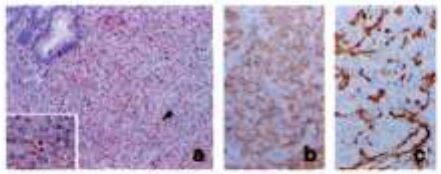

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syndrome,[17] but intravascular lesions have not been observed in these cases.

The pathogenesis of RAE is unknown, but many cases have a setting of coexistent systemic disease. Immunologic factors are also suggested as possible factors in the pathogenesis.[16] The patient in the present case had granuloma-forming enteritis suggestive of Crohn's disease and massive lower gastrointestinal bleeding, a relatively rare complication occurring in 0.6% to 2% of Crohn's disease patients.[4,6,10,21] While the pathogenesis of Crohn's disease remains obscure, various etiologic factors, including infective agents such as enterobacteria or viruses, abnormalities of immune mechanisms, and environmental risk factors such as foods and smoking, have been postulated. There is a possibility that some of these factors or secondary infection at the floor of an open ulcer might play a role in the occurrence of RAE. However, the significance of Crohn's disease and/or massive lower gastrointestinal bleeding for the pathogenesis of RAE remains to be defined.

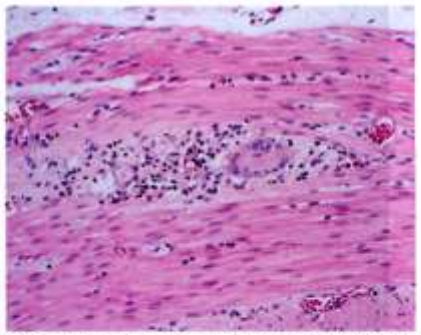


FIGURE 7. A granuloma with a multinuclear giant cell in the muscle layer of the terminal ileum.


La preuve du réchauffement de la planète
vu sur Humour.com
18th Century
1900
1950
1970
1980
1990
2006

ATLAS OF TUMOR PATHOLOGY

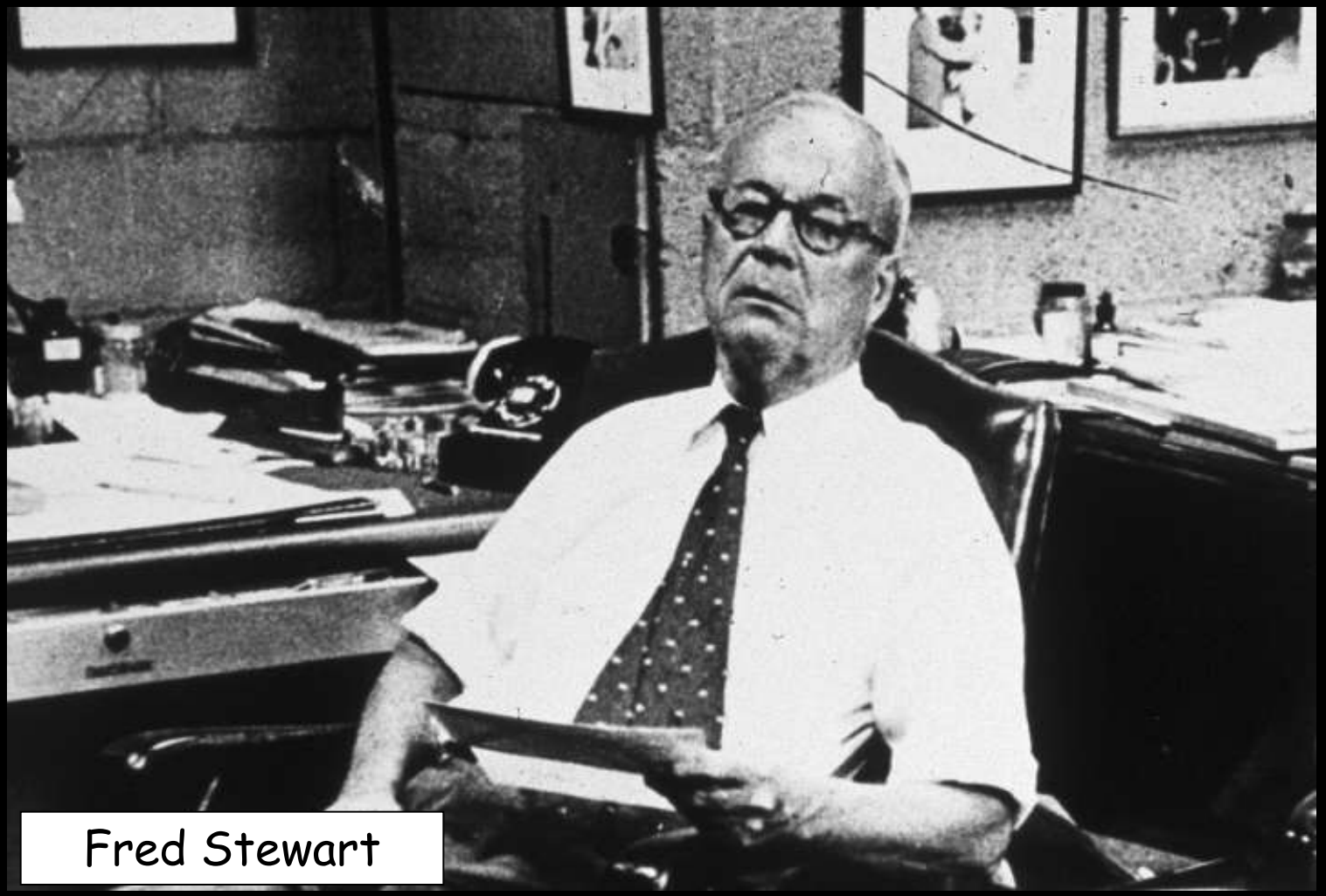
Section IX—Fascicle 34

TUMORS OF THE BREAST

by

Fred W. Stewart, M. D.

Pathologist to Memorial Hospital
Professor of Pathology
Cornell University Medical School
Attending Pathologist of New York Hospital, New York City


Fred Stewart

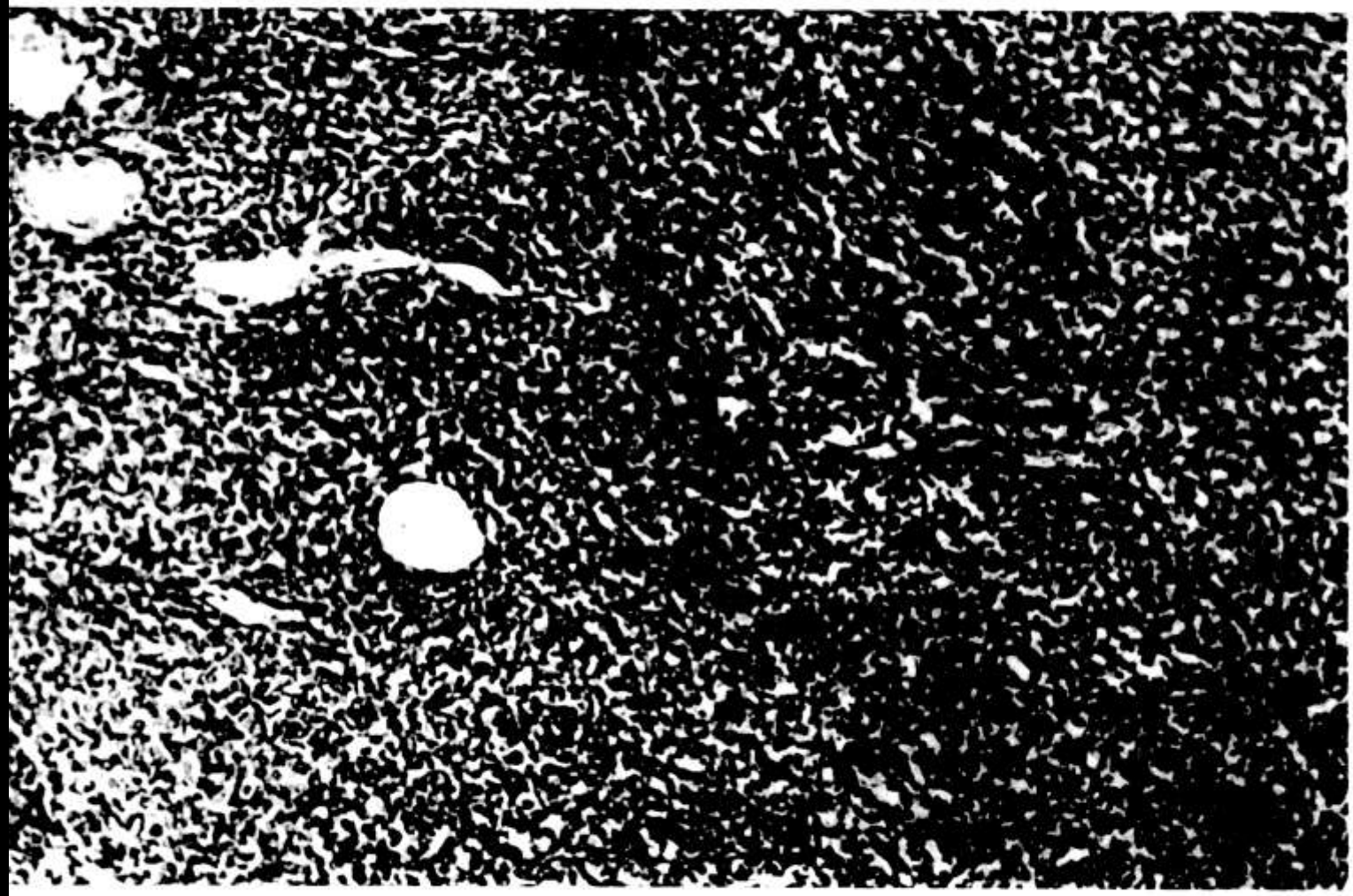


Figure 52. Reticulum cell sarcoma of the breast. A. F. I. P. Neg. Acc. No. 218758-55.

V Kneeland Frantz

ATLAS OF TUMOR PATHOLOGY

Section VII—Fascicles 27 and 28

TUMORS OF THE PANCREAS

by

Virginia Kneeland Frantz, M.D.

Professor of Surgery, College of Physicians & Surgeons, Columbia University
Attending Surgical Pathologist, Presbyterian Hospital, New York, New York

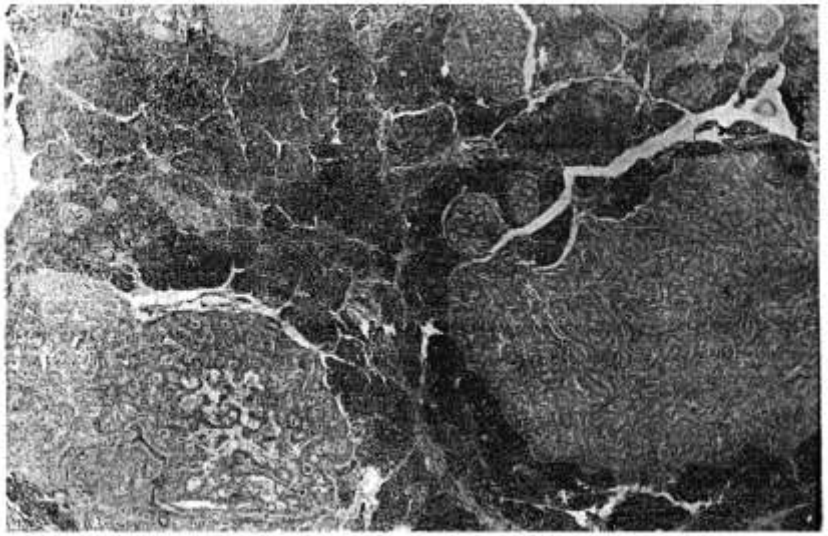


Fig. 56

ADENOMATOSIS, FUNCTIONAL, OF ISLETS OF THE PANCREAS

(Figures 47, 56–58 are from the same case)

Figure 56.* Low power photomicrograph showing topography, to be compared with figure 53. The islet diameters vary from 0.14 to 2.4 mm.

The patient, an obese 46-year-old woman, had had symptoms of hypoglycemia for two years. Blood sugar during an attack was 37 mg. percent. At exploration a hard nodule was palpable in the region of the tail of the pancreas. The tail and part of the body were excised.

Grossly the specimen consisted of a segment of pancreas measuring 5 x 4 x 3 cm., and weighing 13 gm., fresh. Attached to the splenic end there was some fat in which there were numerous calcified nodules 1 to 2 mm. in diameter. On section of the pancreas two small, circumscribed, nonencapsulated, soft red areas were found. Scattered throughout the rest of the pancreas there were tiny, translucent red areas similar to the two larger ones. The lesion, on frozen section at the time of operation, was interpreted as adenomatosis.

Microscopically, on examination of paraffin preparations, this impression was confirmed. Hypertrophy and hyperplasia of islets and ribbon-like arrangement of islet cells with finger-like extrusions into the adjacent pancreatic tissues were seen. These were typical adenomata. Multiple microscopic areas of calcification were found in the parenchyma. After considerable study, these were interpreted as calcification of multiple adenomata. The gross calcified masses adjacent to the tail (fig. 47) showed tiny remnants of pancreatic tissue and were interpreted as larger calcified tumors. One of the viable adenomas was grown in vitro.

Postoperatively the patient had no further symptoms of hypoglycemia and went through a normal pregnancy with delivery 18 months after operation. However, two years after this she entered the hospital severely depleted, with symptoms of carcinoma of the large intestine of a year's duration. At exploration a huge colloid carcinoma of the sigmoid was found, with metastases to lymph nodes and peritoneum. A palliative abdominoperineal resection was performed. She died four years after pancreatic resection, not having had, apparently, any further attacks of hyperinsulinism. No autopsy was done. S.P. 87322, 1943. A. F. I. P. Acc. No. 218895-44.

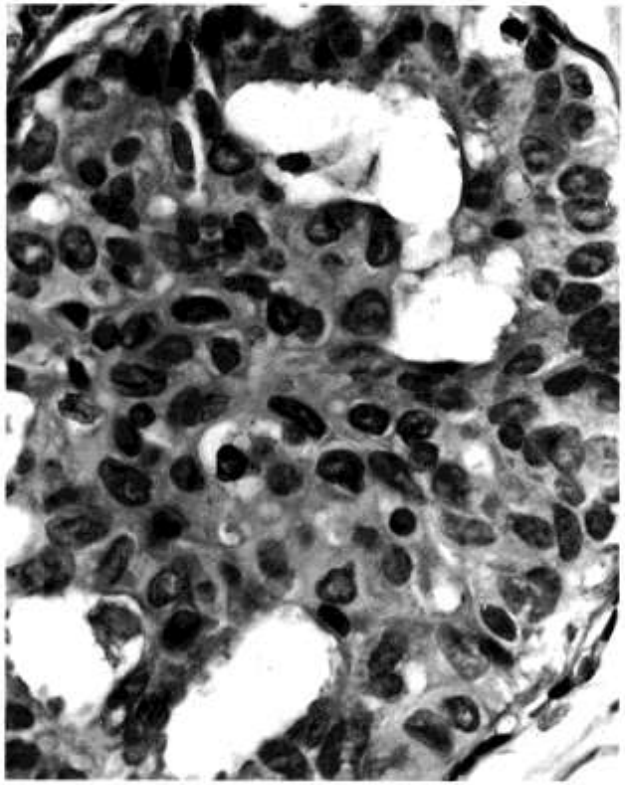


Figure 215

INTRADUCTAL HYPERPLASIA

The heterogeneity of the cellular proliferation is more apparent at higher magnification.

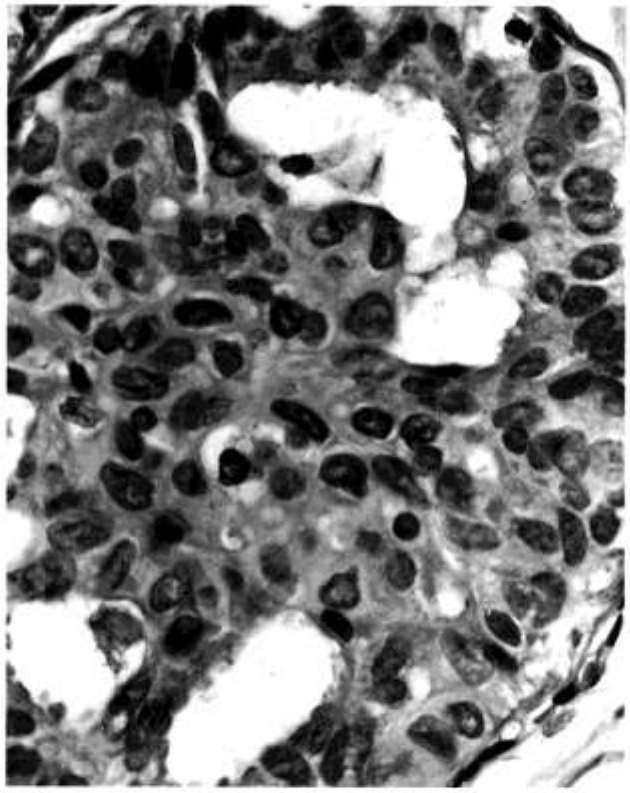


Figure 215

INTRADUCTAL HYPERPLASIA

The heterogeneity of the cellular proliferation is more apparent at higher magnification.

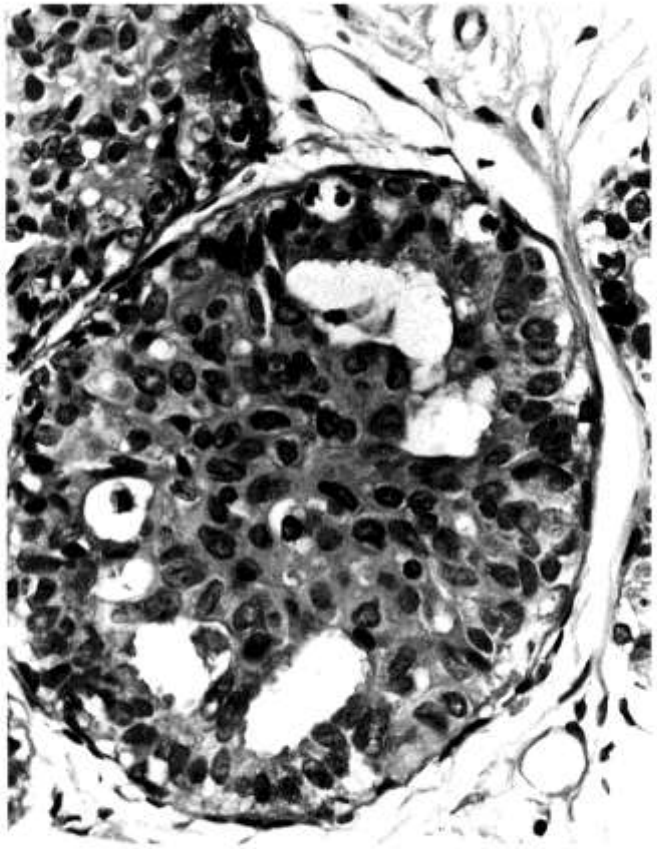


Figure 222

INTRADUCTAL HYPERPLASIA WITH ATYPISM

Secondary lumens are rounded and the cell population appears to be heterogeneous.

The
American Board
of
Pathology

BOOKLET OF INFORMATION 1998

The American Board of Pathology
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FAX (813) 289-5279
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AMERICAN BOARD OF PATHOLOGY
INCORPORATED IN MICHIGAN
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LOGO

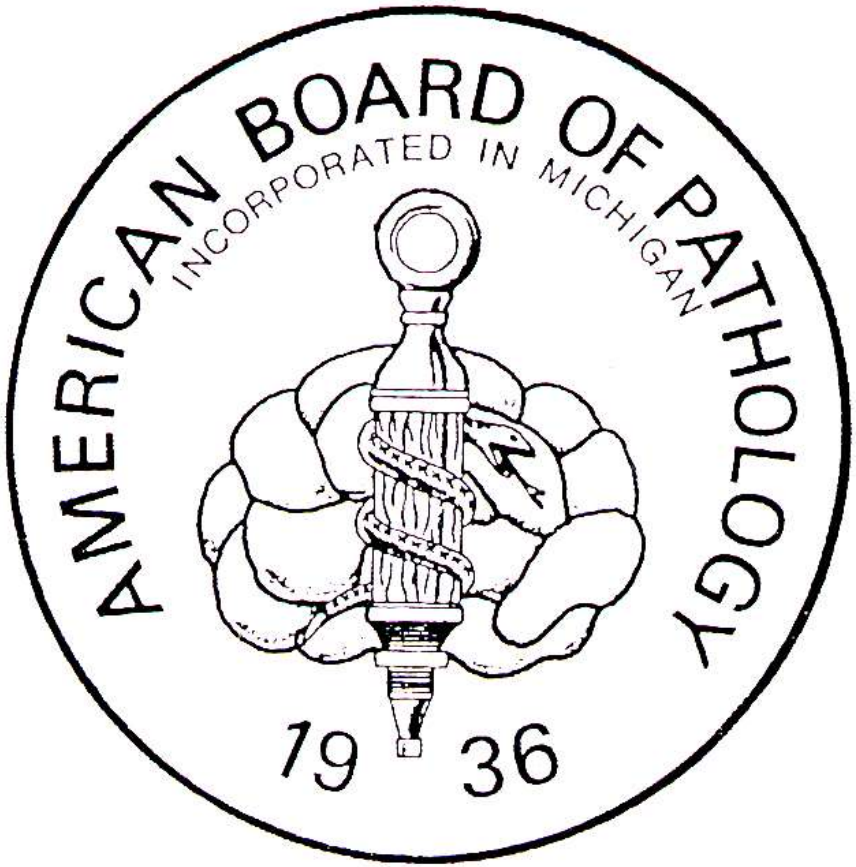
The American Board of Pathology (ABP) logo is based on a bronze plaque designed in 1954 by John R. Schenken, M.D., Trustee of the ABP from 1951 to 1963 The background is a reproduction of the Mycenaean coils of intestine, thought to be the first representation of visceral pathology This sculptured votive offering, made approximately 600 B.C., was excavated by Schliemann in 1876 from the ruins of the extinct city of Mycenae, Greece.

The superimposed syringe-like cylinder represents Hooke's microscope of 1666 A.D., the first compound microscope to be accurately illustrated. The snake coiled about the barrel of the microscope depicts the sacred Aesculapian serpent which, by shedding its skin, is a sign of renewal—the symbol of medicine. Collectively, this logo represents medicine resting firmly upon the foundation of pathology.

The superimposed syringe-like cylinder represents Hooke's microscope of 1666 A.D., the first compound microscope to be accurately illustrated. The snake coiled about the barrel of the microscope depicts the sacred Aesculapian serpent which, by shedding its skin, is a sign of renewal—the symbol of medicine. Collectively, this logo represents medicine resting firmly upon the foundation of pathology.

(fig. 14)

Robert Hooke microscope.
Micrographia. *(Robert Hooke, 1665).*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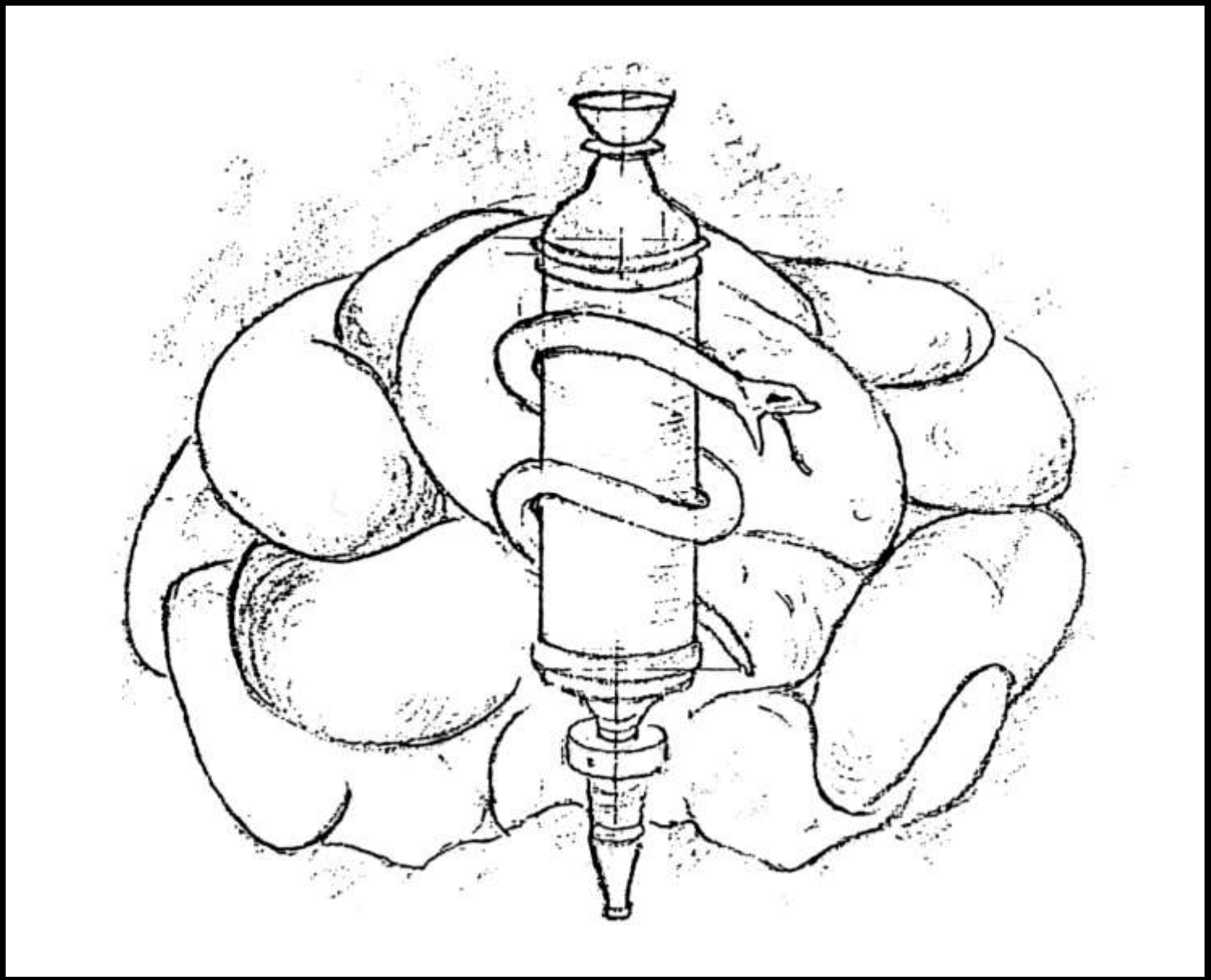

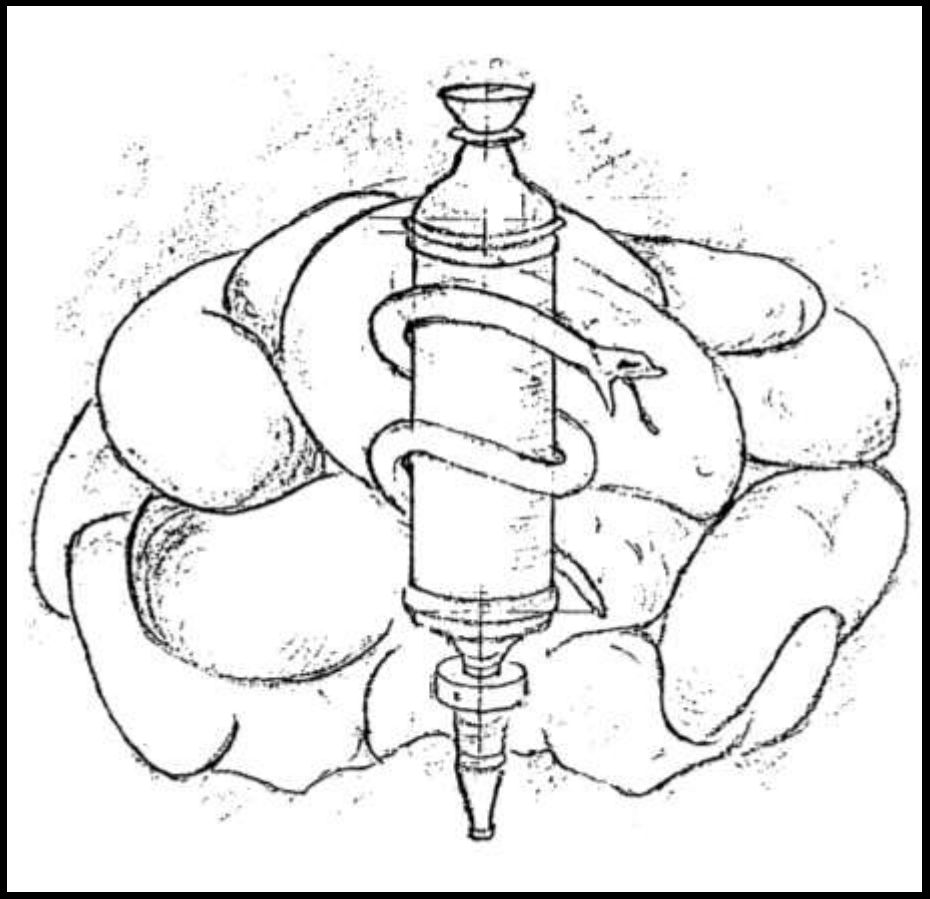


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ATLAS OF TUMOR PATHOLOGY

Third Series
Fascicle 24

TUMORS OF THE PERIPHERAL NERVOUS SYSTEM

Pioneers in Peripheral Nerve Pathology

Theodor Schwann, 1810–1882

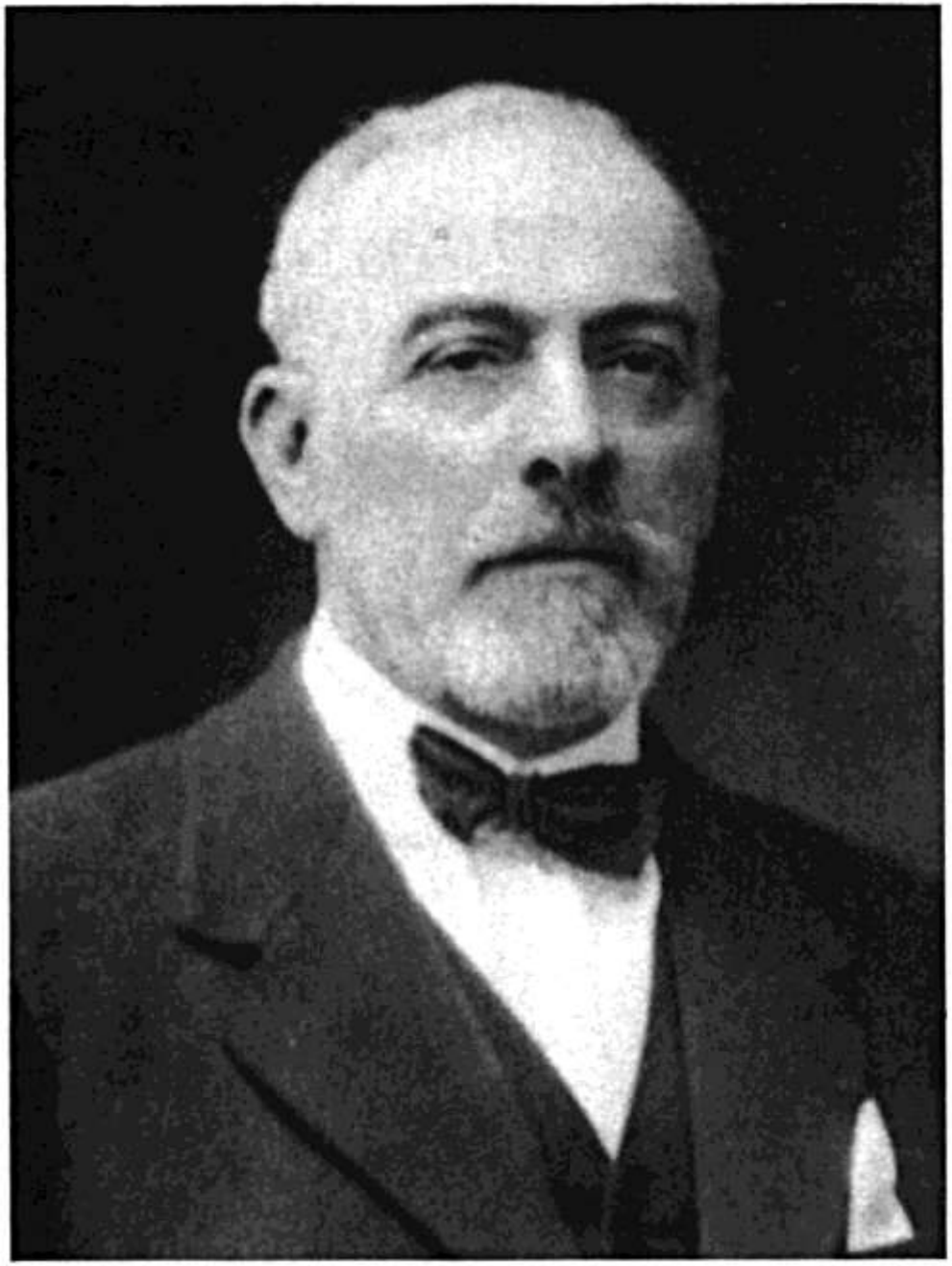
Rudolph Virchow, 1821–1902

Friedrich von Recklinghausen, 1833–1910

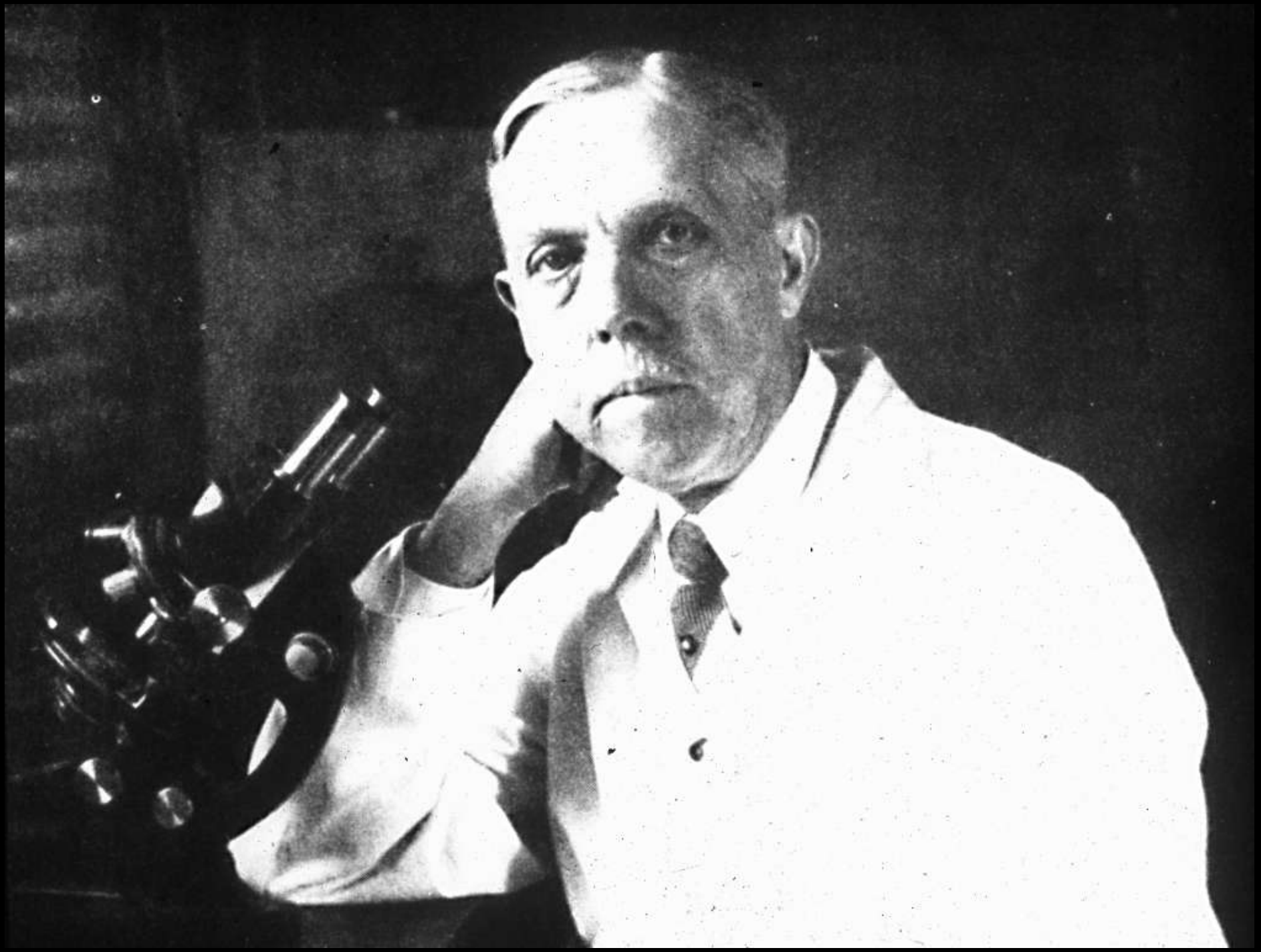
José Verocay, 1876–1927

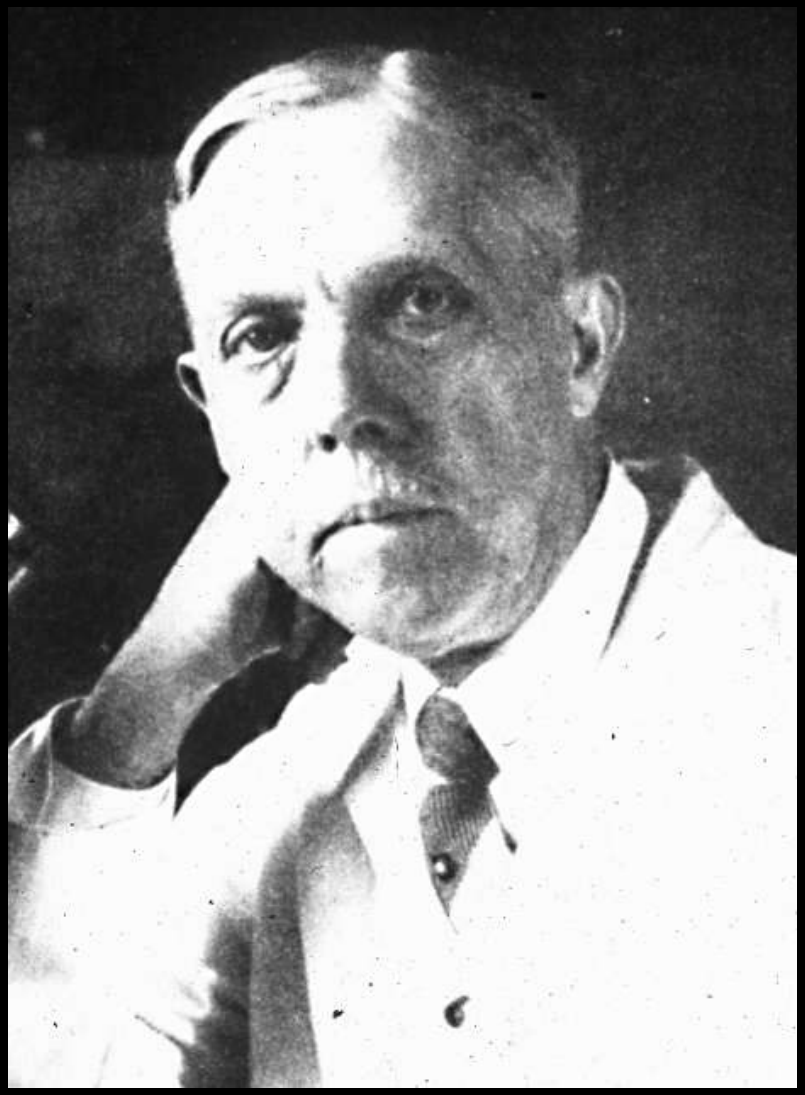
Pierre Masson, 1880–1959

Arthur Purdy Stout, 1885–1967



Pierre Masson, 1880–1959





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"We decided against acknowledgments. When one mentions various pathologists who made the wrong diagnoses and clinicians who did not treat the patients correctly, one is not doing them a favor by acknowledging their participation in the case"

R.E. Scully (1983)

Are Metaplasias in Colorectal Adenomas Truly Metaplasias?

(Am J Pathol 1984, 115: 253–265)

MANJULA BANSAL, MD, CECILIA M. FENOGLIO, MD,
STANLEY J. ROBBOY, MD, and DONALD WEST KING

Five thousand seven hundred seventy-eight adenomas or adenomas containing carcinoma from 3215 patients were examined by routine histologic methods for the presence of epithelial metaplasias.

Acknowledgments

The authors wish to acknowledge their heartfelt gratitude to the superb secretarial and technical staff of the Division of Surgical Pathology of the Presbyterian Hospital, New York, who pulled the slides and reports on the thousands of cases discussed in this manuscript and who then managed to refile them in their proper place. Most particularly, we wish to acknowledge the dedicated services of Mrs. Barbara Fegeley, Mr. Andy Arroyo, Ms. Carole Miller, Mr. Pete Alvarado, Mrs. Julia Zervos, Mrs. Edie O'Brien, Mrs. Anna Romagnoli, and Ms. Lee Stecher.

ACKNOWLEDGMENTS:

Thanks are due to J. Jones for assistance with the experiments and to R. Smith for valuable discussion.

ACKNOWLEDGMENTS:

Thanks are due to J. Jones for assistance with the experiments and to R. Smith for valuable discussion.

Thanks are due to J. Jones for doing the work and to R. Smith for explaining it to me

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F. M. ENZINGER, MD

Volume 26

Cancer

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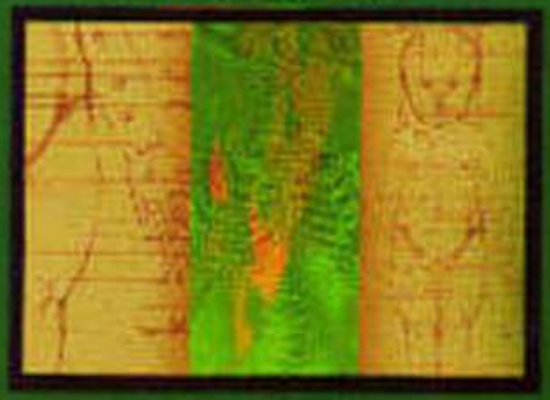
17th edition



Behrman

Kliegman

Jenson

NELSON'S TEXTBOOK OF PEDIATRICS

7th Edition (1959)

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Birds, for the, 1-1413

BOOK REVIEWS &

LETTERS TO THE EDITOR

Book Reviews

This atlas would be an attractive addition to the library of any pathologist or pathologist-in-training.

This text belongs in the library of most busy pathology services.

I recommend that each and every hospital and medical school pathology department keep a copy on its shelf as a quick reference

this book would be a valuable addition to the library of any working surgical pathologist.

certainly warrant a place on any surgical pathologist's bookshelf, even for those with the second edition.

"The covers of this book are too far apart"

Ambrose Bierce

Letters to the Editor

To the Editor:

We read with interest the article by Evans[1] describing multinucleated giant cells in plantar fibromatosis.

To the Editor:

We read with interest the recent article by Chan and Epstein[1] detailing their experience with in situ adenocarcinoma of the bladder.

To the Editor:

We have read with interest the paper entitled "Eccrine Porocarcinoma (Malignant Eccrine Poroma). A Clinicopathologic Study of 69 Cases."

Letters to the Editor

To the Editor:
We read with great interest the article of Ochoa et al.,[4] which demonstrates the possibility of clonal evolution of B cells in Küttner's tumor into a mucosa-associated lymphoid tissue (MALT) lymphoma.

To the Editor:
We read with great interest the recent article by Requena et al.[4]

To the Editor:
We read with great interest the recent article by McKenney et al.,[1] which discussed the expression of CD31 in intratumoral macrophages.

To the Editor:
We read with great interest the article by Fulford et al.[3]

To the Editor:
We read with great interest the article on five cases of pulmonary inflammatory pseudotumor with human herpesvirus-8 (HHV-8) gene expression.[3]

Dedifferentiation in Salivary Gland Carcinomas

To the Editor:

It was with the utmost interest that we read the article by Cheuk et al.[1] describing tumor dedifferentiation in three cases of adenoid cystic carcinoma of the salivary glands.

Letter to the Editor

To the Editor:

I was fascinated by the article by Lehman and Hart[1] on simple and complex hyperplastic papillary proliferations of the endometrium.

"American science has been vitiated by too much politeness…

Conciliatory smoothness is the life blood of diplomacy; it is the death of science".

Editorial, Am J Physiol (1983)

Translocation-Based Molecular Diagnosis of Sarcomas

To the Editor:

I read with some interest the recent article by Hill et al.[5]

Author's Reply:

I have read the letter sent by Drs. Wolber and Chercover responding to my comments in letter form (Am J Surg Pathol. 2002;26:1235–6). In fact, I read it more than once to be sure I understood what they were saying, and it is clear that they completely missed the point.

AUTHOR
VERSUS
EDITOR & PUBLISHER

"… the tumor cells burro deeply into the dermis …"

A "burro" is an ass;

A "burrow" is a hole in the ground;

One really should know the difference

"This article is both good and original".

"Unfortunately the part that is good is not original, and the part that is original is not good".

Dear Contributor: We are returning your manuscript.
It does not suit our present needs.

P.S.: We note that you sent your paper by first class mail. Please be aware that junk mail may be sent third class.

"Thank you for submitting your paper to your journal. We regret that it does not suit our present needs".

To save time, we are enclosing two rejection slips.

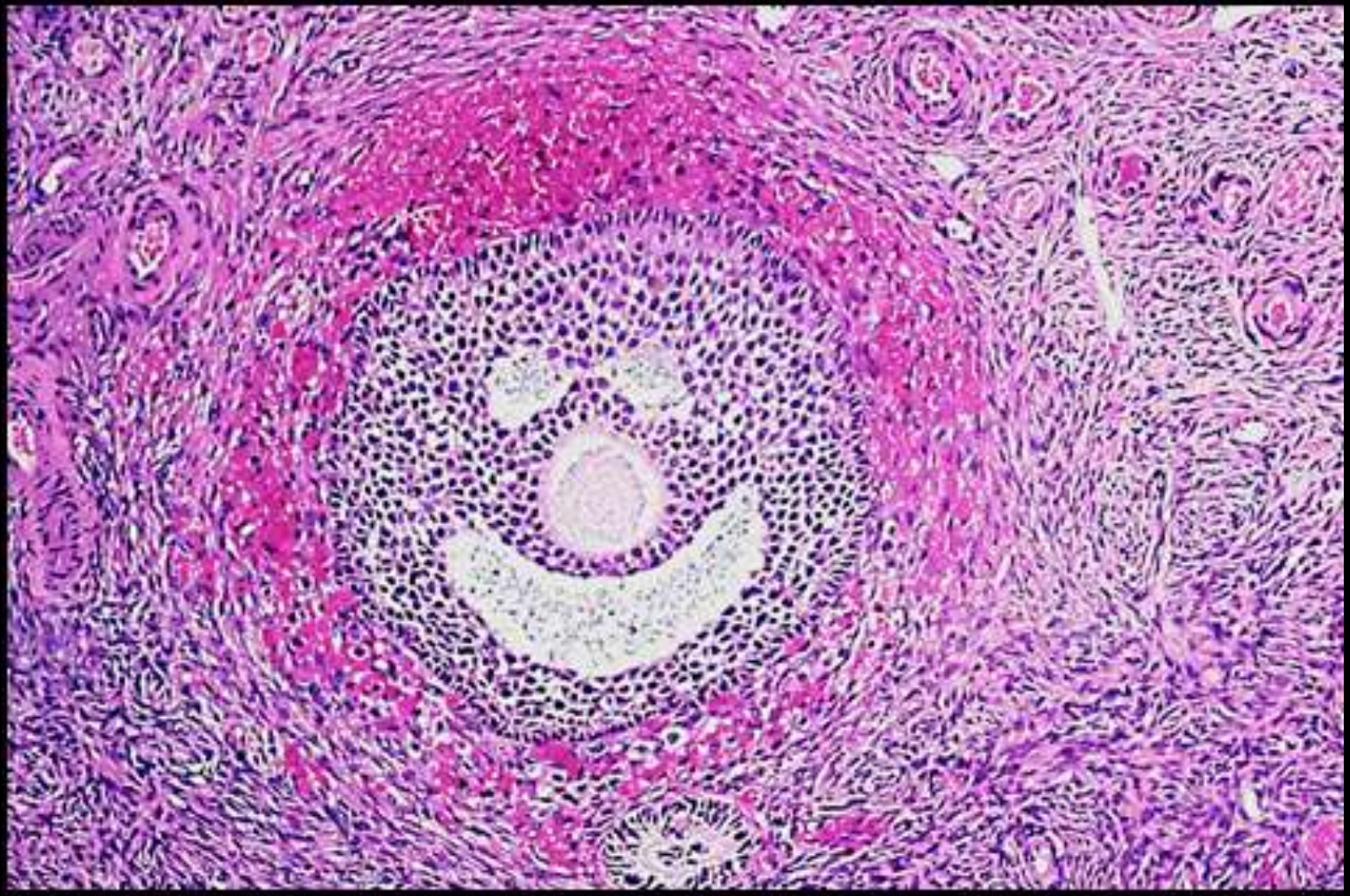
One is for this article, and one for the next one you will be sending us".

"We are happy to accept your superbly written paper for publication in the journal.

But tell me, who wrote it for you?"

" I am so happy that you found my paper acceptable, but tell me: who read it to you?"

"I am reading your article in the smaller room of my house. Right now the article is in front of me. In a few minutes it will be behind me".



Muchas gracias por vuestra atenciòn