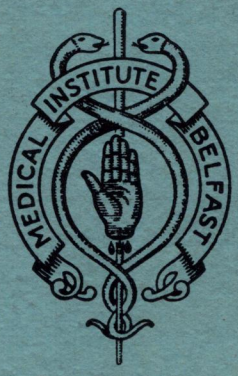
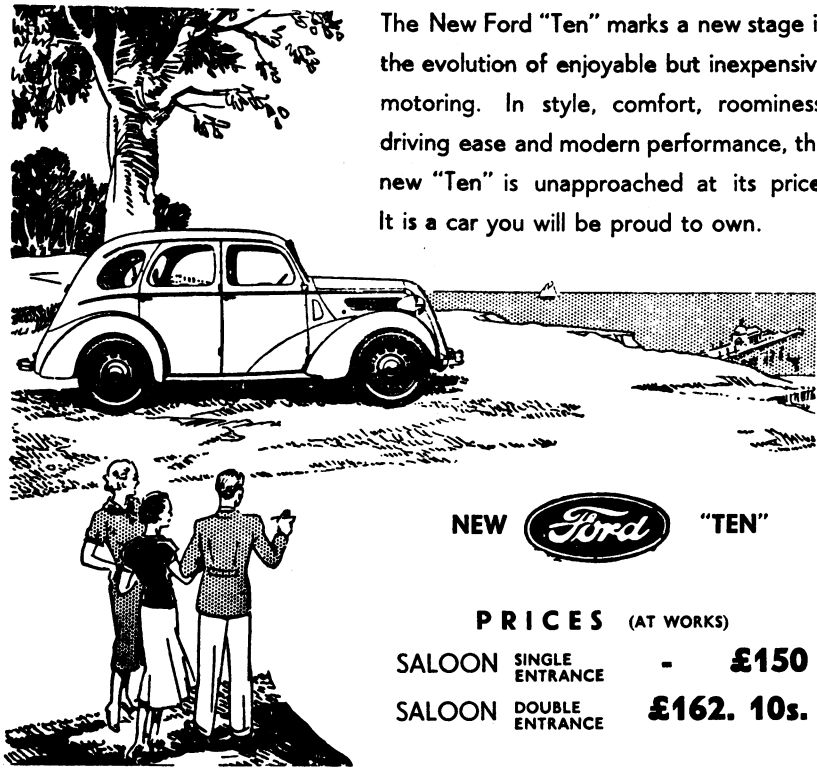
OCTOBER, 1937

# THE ULSTER MEDICAL JOURNAL




PUBLISHED BY
THE ULSTER MEDICAL SOCIETY

# A NEW **FORD "TEN"** FOR WINTER MOTORING



The New Ford "Ten" marks a new stage in the evolution of enjoyable but inexpensive motoring. In style, comfort, roominess, driving ease and modern performance, the new "Ten" is unapproached at its price. It is a car you will be proud to own.

NEW Ford "TEN"

**PRICES** (AT WORKS)

SALOON SINGLE ENTRANCE - **£150**

SALOON DOUBLE ENTRANCE **£162. 10s.**

*May we give you a trial run?*

PHONE 25208

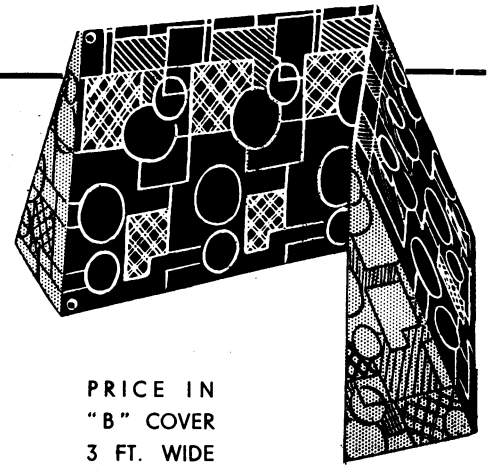
# R·E·HAMILTON & Co

MAIN DEALERS

32 LINENHALL ST · BELFAST ·

"MORE MILES PER GALLON IS GOOD—FEWER PENCE PER MILE IS BETTER"

# *A* BOLSTER *that becomes an Invalid Bed Cushion in a second!*



The Dunlopillo Adjustable Bed Cushion is invaluable to invalids and convalescents. In a second it can be converted from a bolster to a back rest, head rest, or leg rest, providing an ideal support. It avoids using extra pillows, which are not really comfortable, and enables the patient to recline or sit up in bed in luxurious comfort. The soft resilience of the Dunlopillo Bed Cushion never needs punching up, it always keeps its shape. It is porous, self-ventilating, moth proof, dustless, and completely hygienic.

PRICE IN
"B" COVER
3 FT. WIDE

**35/-**

# DUNLOPILLO

## ADJUSTABLE BED CUSHION

(U.K. Patent applied for)

★ *It can be used as a*

BOLSTER

BACK REST

HEAD REST

OR LEG REST

DUNLOP RUBBER COMPANY Ltd. (General Rubber Goods Division), Cambridge Street, Manchester, 1

MADE IN A DUNLOP PRODUCT SPECIALISED FACTORY

BELFAST:
RUBBER HOUSE, UPPER ARTHUR STREET

7DP4/73

C.F.H.

C

# 'ENTORAL'

**TRADE MARK** **BRAND**

## ORAL COLD VACCINE

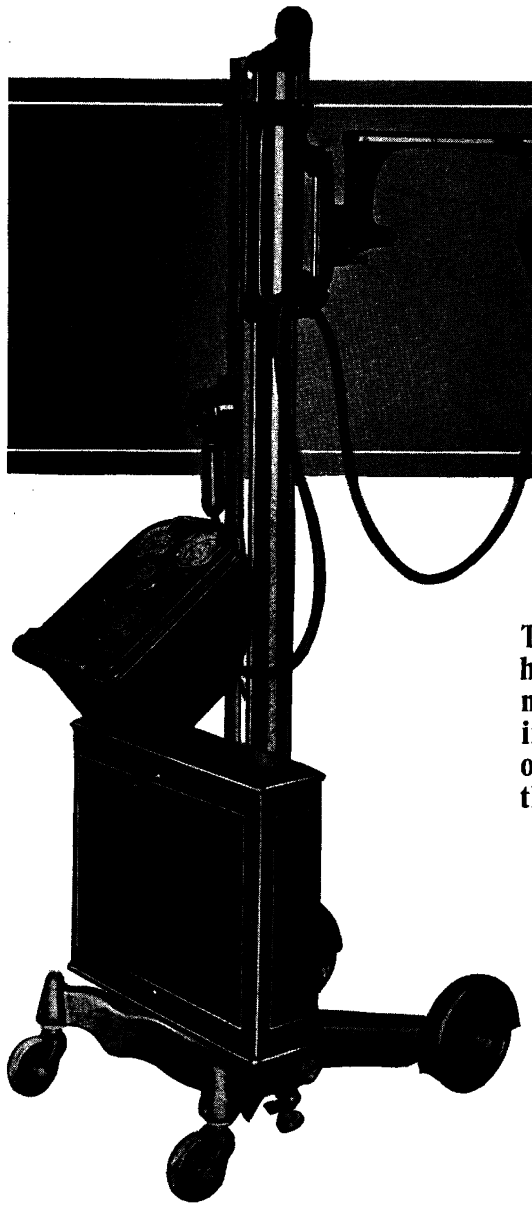
FOR PROPHYLACTIC IMMUNISATION TO RESPIRATORY INFECTIONS AND PARTICULARLY TO THE COMMON COLD. 'ENTORAL' IS AN IMMUNISING ANTIGEN, WHICH, WHEN GIVEN ORALLY, WILL PRODUCE HETEROPHILE ANTIBODY IN SUFFICIENT AMOUNTS TO INCREASE MATERIALLY THE RESISTANCE OF INDIVIDUALS TO RESPIRATORY INFECTIONS.

ISSUED IN PACKAGES OF 20 AND 60
'PULVULES' brand FILLED CAPSULES

**ELI LILLY AND COMPANY LIMITED**

**2, 3 AND 4, DEAN STREET, LONDON, W.1.**

**Distributing Agent in Britain for**
**ELI LILLY AND COMPANY, INDIANAPOLIS, U.S.A.**



To the many features which have made the Victor Model "D" mobile, shockproof (oil-immersed) x-ray unit first choice of the discerning radiologist the world over are now added

**higher radiographic power**
**greater range of control**
**wider lateral movement**
**(full 180°)**
**incorporated cassette box**
**easier running**

**YOU CANNOT BUY BETTER THAN VICTOR**

# VICTOR X-RAY CORPORATION LIMITED

12 PEARSE STREET DUBLIN

*Head Office*

15/19 CAVENDISH PLACE, LONDON, W.1

# RECOMMENDED TEXTBOOKS

**NEW BOOKS**

## GLAISTER and BRASH

**MEDICO-LEGAL ASPECTS OF THE RUXTON CASE.** Crown quarto. 312 pp., with 172 Illustrations. **21s.** net; postage 7d. (September 1937)

¶ **The medico-legal and other scientific aspects of a case without parallel in the annals of crime.**

---

**SIMSON HALL. Diseases of the Nose, Throat, and Ear.** 420 pp., 55 Illustrations and Coloured Frontispiece. **10s. 6d.** net; postage 6d. (March 1937)

---

**CURRIE. Manual of Public Health—"Laboratory Practice."** 400 pp., 169 Illustrations. **21s.** net; postage 7d. (1936)

---

**BIGGART. Pathology of the Nervous System.** 350 pp., 204 Illustrations. **15s.** net; postage 6d. (1936)

---

**MEKIE. Handbook of Surgery.** With a Foreword by Professor Sir JOHN FRASER. 712 pp., Illustrated. **12s. 6d.** net; postage 6d. (1936)

¶ The Companion Volume to Wheeler and Jack.

## THE CATECHISM SERIES

*Best for quick revision*

**64 Parts at 1s. 6d. net per Part**

**NEW EDITIONS**

## JAMIESON. Illustrations of Regional Anatomy.

*Second Edition.* Sections I—V.

| Section | Plates | Price Net | Postage |
|---|---|---|---|
| I. Central Nervous System | 48 | **7s. 0d.** | 5d. |
| II. Head and Neck | 63 | **12s. 6d.** | 6d. |
| III. Abdomen | 37 | **5s. 6d.** | 4d. |
| IV. Pelvis | 33 | **3s. 6d.** | 4d. |
| V. Thorax | 30 | **4s. 0d.** (August 1937) | 4d. |
| VI. Upper Limb | 42 | **7s. 6d.** | 5d. |
| VII. Lower Limb | 52 | **10s. 0d.** (October 1936) | 6d. |

The complete set is published at **50s.** net, but purchasers taking the seven sections at one time can obtain them at the reduced price of **47s. 6d.** net; postage 9d.

**Each Section in attractive loose-leaf case.**

---

**WHEELER and JACK. Handbook of Medicine.** *Tenth Edition.* Revised by Prof. HENDERSON. 720 pp., Illustrated. **12s. 6d.** net; postage 6d. (August 1937)

¶ "Wheeler and Jack" is as valuable now to the student and practitioner as it has ever been in its forty years of existence."—THE LANCET.

---

**STEWART and DUNLOP. Clinical Chemistry in Practical Medicine.** *Second Edition.* Revised and Enlarged. 384 pp., Illustrated. **10s. 6d.** net; postage 6d.

A new and thoroughly revised edition will be published in October.

---

**LEES. Venereal Diseases.** *Third Edition.* 624 pp., 85 Illustrations and Coloured Plates. **15s.** net; postage 6d. (January 1937)

---

**ROBERTSON. Hygiene.** *Second Edition.* 320 pp., 27 Illustrations. **8s. 6d.** net; postage 6d. (1936)

---

**HAULTAIN and KENNEDY. Midwifery and Gynæcology.** *Second Edition.* 370 pp., 44 Illustrations. **15s.** net; postage 6d. (1935)

---

**MUIR. Bacteriological Atlas.** *Second Edition.* Enlarged and Rewritten by C. E. van ROOYEN, M.D. 83 beautifully Coloured Plates illustrating 91 Organisms; 26 of these plates are entirely new. Faced by Descriptive Text. **15s.** net; postage 6d. (June 1937)

---

**BRUCE WILLIAMSON. Diseases of Children.** *Second Edition.* 340 pp., 61 Illustrations. **10s. 6d.** net; postage 6d. (Nov. 1936)

---

**KERR, YOUNG, and HENDRY. Obstetrics and Gynæcology.** *Second Edition.* 1120 pp., 500 Illustrations. **35s.** net; postage 9d. (Rept. 1936)

---

**CONYBEARE. Textbook of Medicine.** *Third Edition.* 1050 pp., Illustrated. **21s.** net; postage 7d. (October 1936)

---

**MACKIE and McCARTNEY. Practical Bacteriology.** *Fourth Edition.* 512 pp., Illustrated. **12s. 6d.** net; postage 6d.

---

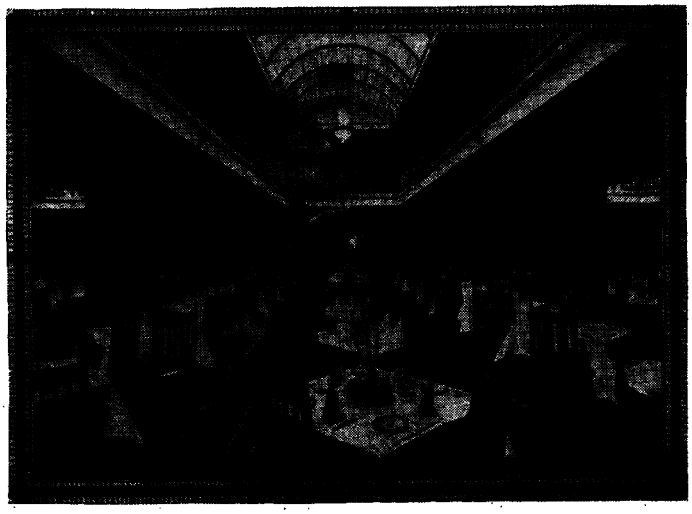
**ROSS and FAIRLIE. Anæsthetics.** *Fourth Edition.* 320 pp., Illustrated. **10s. 6d.** net; postage 6d.

*Write for our 1937 Medical Catalogue*

**E. & S. LIVINGSTONE,** 16 AND 17 TEVIOT PLACE EDINBURGH

*for*

## MORNING COFFEES, LUNCHEONS AFTERNOON TEAS, AND DINNERS



*visit Belfast's most popular rendezvous*

# THE CARLTON

## DONEGALL PLACE

*Open 8 a.m. till 8 p.m.* ✻ ✻ ✻ *Orchestral Music Daily*

PRIVATE ROOMS FOR BANQUETS, DINNERS, AND DANCING, FOR PARTIES OF TEN TO FOUR HUNDRED

*For Wedding Parties and other events requiring efficient and up-to-date catering apply to the Manager, who will only be too pleased to submit quotations and make all necessary arrangements*

Telephone 26861


# Active little bodies

**MUST have nutritious food in plenty, food so nutritious that, along with bodily growth, there should be a corresponding development of brain power**

# R.S.O. FOOD

*"A PURE WHITE PRODUCT"*

**provides the necessary nutriment; it is a natural food in easily digested form**

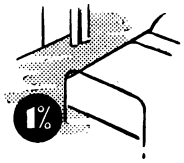
*"There's a wealth of Health in every tin"*

*Product of*

**WHITE, TOMKINS & COURAGE Ltd., BELFAST**

*Manufacturers of Pure Foods since 1841*

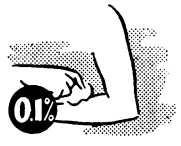

DISINFECTION
OF HANDS




SICK ROOM
DISINFECTION


PRESERVING OF
INSTRUMENTS


WOUNDS, CUTS
AND ABRASIONS




SEPTIC SKIN
DISEASES

# HAVE YOU TRIED THIS NEW ANTISEPTIC?

# 'Zephiran' TRADE MARK BRAND concentrate

## POTENT • ECONOMICAL
## CLEAR • NON-IRRITANT

**'Zephiran' Concentrate is built up on an entirely new chemical basis, contains neither phenols nor heavy metals, is non-poisonous, non-irritant and practically odourless.**

**The disinfectant value of 'Zephiran' Concentrate has been thoroughly proved by extensive laboratory tests on the continent. These tests have been repeated and their scope extended by a Bacteriologist at one of the London Teaching Hospitals.**

'Zephiran' Concentrate is issued in 6 oz. bottles (with measure cap) at 3/3 each (subject to medical discount) and in Winchester Quarts for hospital use. When used in the recommended solutions, it is exceptionally economical.


**BAYER PRODUCTS LTD., MOLESWORTH HOUSE, 1/2, SOUTH FREDERICK STREET, DUBLIN.**
(INCORPORATED IN ENGLAND)

L

# STUDENTS' BOOKS

## Diseases of the Eye

*New Work*

By EUGENE WOLFF, M.B., B.S., F.R.C.S.Eng. "Here indeed is a wonderful new book."—*Glasgow University Medical Journal.* Quarto, 234 pages. With 5 Colour Plates and 120 Text-figures. **15s.** net.

## Sick Children: Diagnosis and Treatment

*New Edition*

By DONALD PATERSON, M.D.Edin., F.R.C.P.Lond. "The best and most handy book we have yet met on the subject."—*St. George's Hospital Gazette.* **Second Edition.** Medium 8vo, 600 pages. With 15 Half-tone Plates and 76 Text Illustrations. **12s. 6d.** net.

## Manson's Tropical Diseases

Edited by PHILIP MANSON-BAHR, D.S.O., M.A., M.D., D.T.M.&H.Cantab., F.R.C.P.Lond. **Tenth Edition.** Demy 8vo. 1,004 pages With 37 Plates (22 in Colour), 381 Text-figures, 6 Maps, and 38 Charts. **31s. 6d.** net.

## Diseases of the Skin

By S. ERNEST DORE, M.D., F.R.C.P.Lond., and JOHN L. FRANKLIN, M.D., M.R.C.P. Lond. Crown 8vo, 420 pages. With 46 Half-tone Plates. **10s. 6d.** net.

## Materia Medica and Therapeutics

(Bruce and Dilling.) By Professor WALTER J. DILLING, M.B., Ch.B. **Fourteenth Edition.** Foolscap 8vo, 700 pages. **10s. 6d.** net.

## The Essentials of Medical Diagnosis

By LORD HORDER, K.C.V.O., M.D., F.R.C.P.Lond., and A. E. GOW, M.D., F.R.C.P.Lond. Crown 8vo, 702 pages. With 8 Colour and 11 Black and White Plates, and Figures and Charts in the Text. **16s.** net.

## A Manual of Physics for Medical Students

By HUGH C. H. CANDY, B.A., B.Sc., F.I.C. **Third Edition.** Foolscap 8vo, 496 pages. With Colour Frontispiece and 299 Text-figures. **7s. 6d.** net.

## An Introduction to General Therapeutics

By H. K. FRY, D.S.O., B.Sc., M.D., D.P.H. Crown 8vo, 224 pages. **6s.** net.

## Herman's Difficult Labour

**Seventh Edition.** Revised by CARLTON OLDFIELD, M.D., F.R.C.P.Lond., F.R.C.S.Eng. Crown 8vo, 574 pages. With 8 Radiographic Plates and 197 Illustrations in the Text. **16s.** net.

## Surgical Applied Anatomy

By Sir FREDERICK TREVES, Bart. Revised by C. C. CHOYCE, C.M.G., M.D., F.R.C.S. Eng. **Ninth Edition.** Foolscap 8vo, 720 pages. With 174 Illustrations, 66 in Colour. **14s.** net.

## The Student's Handbook of Surgical Operations

By Sir FREDERICK TREVES, Bart. Revised by CECIL P. G. WAKELEY, F.R.C.S.Ed., F.R.S.Ed. **Fifth Edition.** Crown 8vo, 550 pages. With 190 Figures in the Text. **10s. 6d.** net.

## Modern Operative Surgery

Edited by G. GREY TURNER, M.S., F.R.C.S.Eng., F.A.C.S.(Hon.). **Second Edition.** Two Volumes. Medium 8vo, 1,780 pages. With 11 Half-tone Plates and 860 Figures in the Text. **£3. 3s.** net the set.

## A System of Surgery

Edited by C. C. CHOYCE, C.M.G., C.B.E., B.Sc., M.D., F.R.C.S.Eng. Editor of Pathology, J. M. BEATTIE, M.A., C.M., M.D. **Third Edition.** Three Volumes. Medium 8vo, 3,330 pages. With 60 Colour and 117 Half-tone Plates, and 929 Text-figures. **£4** net the set.

*Now Printing*

*New Edition*

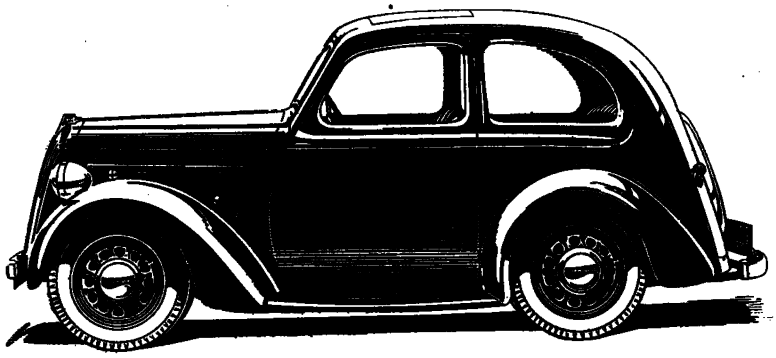
## ✱ Elements of Surgical Diagnosis

By Sir ALFRED PEARCE GOULD. Revised by ERIC PEARCE GOULD, M.D., M.Ch., F.R.C.S.Eng. **Eighth Edition.** Foolscap 8vo, 730 pages. With 22 Radiographic Plates. **10s. 6d.** net.

***Cassell and Company, Ltd., La Belle Sauvage, London, E.C.4***

# ONLY **9 H.P.** — ONLY **£152-10**

## BUT A FINE BRITISH CAR



## NEW *Flying Standard* 'NINE'

If you entered a friend's Flying Standard "Nine" for the first time and did not know its h.p. — you might well be surprised when you were told. Your comfort and ease of movement, too, would *not* suggest that this car (strictly) is in the "smaller" class. Compare it point by point—make the only sure test—you'll find it bigger, more beautiful and better equipped. As for the value you can't see, the "Nine" is built by Standard —to give a *lasting* sense of successful outlay. It's made for those of modest spending power — but it's a *fine* British car.

**GRACEFUL NEW STREAMLINE FRONT ASSEMBLY**
**4-SPEED SYNCHRO-MESH GEARS**
**12-VOLT ELECTRICAL EQUIPMENT**
**65 M.P.H. 36 M.P.G.**
**FLUSH-FITTING SLIDING ROOF**
**ACCESSIBLE JACKS**
**LUGGAGE LOCKER**
**HIGH POWER/WEIGHT RATIO**
**DOWNDRAUGHT CARBURETTOR**
**TRIPLEX GLASS ALL ROUND**

**SALOON - - - - £152. 10**
**SALOON DE LUXE - - £162. 10**

Other Models 10 h.p.—20 h.p. V-Eight £172 - 10 to £325. All prices ex works.

PART EXCHANGE — DEFERRED TERMS

# Clarence Engineering Co. Ltd.

*Jasper Johnstone*

**43-47 CHICHESTER STREET, BELFAST** **PHONE 25481**

# *Flying Standard*

«

N

# *Your Winter Clothes!*

The value of the statement, "Good clothes are a definite asset," cannot be over-estimated. . . . When we say "good clothes" we mean clothes which possess that indefinable something called "style" . . . that master touch which distinguishes them at a glance from those produced by "multiple tailors," and with which too many men appear content to-day. If you wish your new winter suit or overcoat to express your individuality, you must choose your tailor and your cloth with care and judgment; then, and then only, will your appearance bear that stamp of quality so remote from clothes made to a price. We make only "quality clothes," in which quality is apparent in the cut, the cloth, and the style.

*The new winter patterns, which comprise the very latest in style and colourings, are now available. We cordially invite your inspection with confidence.*

THE
ULSTER CLOTH HALL

*MAKERS OF GOOD CLOTHES SINCE 1875*

**REUBEN PAYNE AND IRELAND LIMITED**

MILITARY, LADIES' AND GENT.'S TAILORS AND BREECHES MAKERS

TWO CHICHESTER STREET, BELFAST

Phone 20120

# ANTI - STREPTOCOCCAL THERAPY

ORAL

## TRADE PROSEPTASINE MARK

Benzylaminobenzenesulphonam ide

(M & B 125)

The introduction of the BENZYL group into the molecule of the simple sulphonamide gives a product which is practically tasteless, well tolerated and of high anti-streptococcal activity.

*Tablets of 0.5 gramme.*

PARENTERAL

## TRADE SOLUSEPTASINE MARK

Disodium-p($\gamma$-phenyl-propyl-amino)benzene-sulphonamide-$\alpha$-$\gamma$-disulphonate

The first colourless anti-streptococcal drug to be prepared which is suitable for parenteral administration.

*Ampoules of 5 c.c. and 10 c.c.*

SOLUSEPTASINE is for use alone or in conjunction with the oral administration of PROSEPTASINE. It is intended for the treatment of severe streptococcal infections where the immediate presence of the streptococcicidal drug in the blood is necessary, and where there is difficulty in swallowing or retaining medicine given by the mouth. There is evidence to show that these drugs are also of value in *B. coli* infections.

*Samples and literature on request.*

M&B

**PHARMACEUTICAL SPECIALITIES**

**(MAY & BAKER) LTD. DAGENHAM**


# OUR MOTOR AMBULANCE

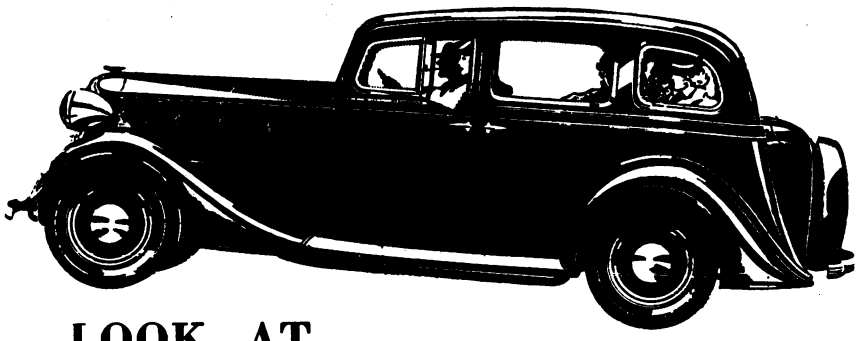
is ever ready for conveying patients to and from Hospital or Nursing Home in city or country—distance no object. Specially designed and equipped to ensure safe, comfortable travel under all conditions

MODERATE CHARGES

## MELVILLE & CO., LTD.

TOWNSEND STREET - BELFAST

In time of need ——— 'Phone ——— Belfast 26272



# LOOK AT

# THESE PERFORMANCE FIGURES!

**VAUXHALL 14 h.p.**
**PERFORMANCE FIGURES**

FROM 10 to 30 m.p.h. in 5⅘ seconds

FROM REST to 50 m.p.h. in 20 seconds

FROM 10 to 30 m.p.h. in Top Gear in 10⅕ secs.

MAXIMUM SPEED 65-70 m.p.h.

Besides its striking performance and its exceptional silence, the Vauxhall 14 h.p. has Independent Springing—proved on over 40,000 Vauxhalls. This makes "riding changed to gliding" much more than a slogan. We'd like you to discover for yourself what it means to motoring comfort,—and try out, too, No-Draught Ventilation, "Body Conformity" front seats, Super Synchro-Mesh and all the other features of this Vauxhall.

May we arrange a trial run?

**THE**

# VAUXHALL

## 14 *h.p.*

DE LUXE SALOON £225
TOURING SALOON £230

**MAY WE DEMONSTRATE?**

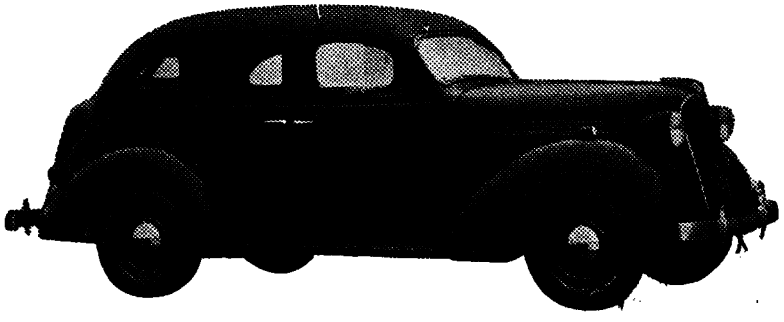
# V. H. ROBB & Co., Ltd.

*Only Address:* CHICHESTER STREET, BELFAST — *No Branches*

# CHRYSLER . . .

**THE CHRYSLER offers to the discerning motorist the finest example of quality motor car production; nothing that he could possibly wish for in a CAR is missing . . . lovely lines — great roominess — larger seats — increased head and leg room, and many important engineering improvements make the CHRYSLER stand out as a car for the motorist who is content only with the BEST.**

**PRICE**
**£355**
*ex works*
**AVAILABLE IN**
**19.8 h.p. &**
**23.4 h.p.**



*"The Kew Six"*
**CHRYSLER** *Saloon*

*For those whose needs call for a really roomy car the "Kew Six Saloon" is just the model. Call at our main showroom and inspect it, or better still, let us give you a demonstration run.*

**Sole Chrysler Distributors for N.I.:**

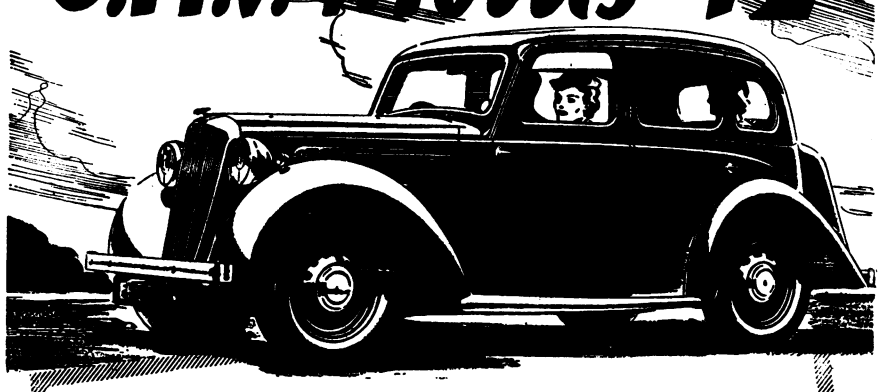
**Victor LIMITED**
DONEGALL HOUSE DONEGALL SQUARE WEST
BELFAST

**Telephone**
**21677**
**(3 lines)**

**THE CAR FOR THE MOTORIST WHO KNOWS**

*Announcing a NEW car — the NEW Series III*

# O.H.V. Morris 12



WITH

## Splendid acceleration

GIVING THE GREATEST Safety

★ **OVERHEAD VALVE ENGINE — especially accessible.**

★ **4-speed silent helical synchromesh gearbox.**

★ **Finish, springing and unique refinements in excess of anything previously achieved in a reasonably priced car.**

THIS NEW TWELVE IS OUTSTANDING FOR ITS EXCEPTIONAL PERFORMANCE

*Other features include :*

New type precision steering; New improved springing; Sound insulated body; Lockheed Hydraulic brakes; Triplex all round; Six-stud wheel fixing; 12-volt battery; Compensated voltage control; Trafficators with automatic return; Dip-and-switch headlamps with foot control; Draughtless front windows, with louvres and opening rear quarter lights; Exceptionally large capacity built-in boot with separate compartment for spare wheel and tools; folding rear arm-rest, etc., etc.

**MORRIS TWELVE-FOUR SERIES III**

Saloon (*Fixed Head*) - £205 Saloon (*Sliding Head*) - £215 Additional for Jackall System - £5

(*Prices ex works*)

*Distributors for Northern Ireland*

W.H. ALEXANDER LTD.

MAY STREET · DONEGALL STREET · HOLMES STREET

SHOWROOMS · CHICHESTER STREET · TELEPHONE NO. 22671

# British Medical Bureau

**Northern Branch**

**(The Scholastic, Clerical, and Medical Association, Limited)**

## DISTRICT OFFICE FOR NORTHERN IRELAND

Telephone Belfast 23204 — **72 High Street, Belfast** — Telegrams Vouch, Belfast

**JAMES BAIRD & CO., LOCAL SECRETARIES**

---

The business of the British Medical Bureau, which is conducted in accordance with the general policy of the British Medical Association, is divided under the following heads:—

### TRANSFER OF PRACTICES AND PARTNERSHIPS

**Medical practitioners wishing to dispose of practices, or desiring to take partners, are advised to negotiate the business through the British Medical Bureau. Vendors may depend upon receiving introductions only to eligible and *bona fide* purchasers. All information is treated in strict confidence. Full and trustworthy information regarding practices, partnerships, etc., for disposal, supplied gratis to purchasers.**

### ASSISTANTS and LOCUM TENENS

**Assistants and locum tenens can be secured at short notice. It is the foremost aim of the British Medical Bureau to ensure that only the most trustworthy and reliable locums and assistants are sent out.**

### VALUATION AND INVESTIGATION OF PRACTICES

**We undertake all work associated with the valuation and investigation of practices**

**INSURANCES EFFECTED. FINANCIAL ASSISTANCE ARRANGED FOR APPROVED APPLICANTS FOR PURCHASE OF PRACTICES AND PARTNERSHIPS**

The Bureau has long been favourably known to the medical profession throughout the United Kingdom as a thoroughly trustworthy and successful medium for the transaction of every description of medical business, and the directors have every confidence in recommending practitioners in Northern Ireland to consult the local secretaries, Messrs. James Baird & Co.

**Recommended with every confidence to the profession by the British Medical Association as a thoroughly trustworthy medium for the transaction of all medical agency business**

PRINTED FOR THE ULSTER MEDICAL SOCIETY BY GRAHAM AND HESLIP, LIMITED, BELFAST.
ADVERTISEMENT AGENTS: GEO. A. STEWART & CO., 100 HIGH STREET, BELFAST.

# CONTENTS






*Fellows and Members of the Ulster Medical Society receive the Journal free*
*Subscription to non-members five shillings annually*

# CONTENTS






*Fellows and Members of the Ulster Medical Society receive the Journal free*
*Subscription to non-members five shillings annually*

# THE ULSTER MEDICAL JOURNAL

## NOTICE TO CONTRIBUTORS

1. Manuscript should be typewritten and fully corrected. Contributors will be responsible for the payment of any sum charged for correction of the printer's proof in excess of ten shillings per sheet (16 pages).
2. Illustrations must be in finished form ready for reproduction. They must be properly labelled in type or by hand, with reference pointers if necessary.
3. Line drawings must be sent whenever possible. Illustrations requiring half-tone blocks are costly, and unless printed on special art paper are often unsatisfactory. Authors will be charged for these half-tone blocks at cost price.
4. The legend describing an illustration must be inserted in the appropriate place in the text, and should not be placed on or appended to the drawing.
5. Orders for reprints must be given when the author returns the printer's proof. The cost of these may be obtained from the printers in advance.
6. Editorial communications should be sent direct to the ~~Acting Editor,~~ Dr. Hunter, Department of Anatomy, Queen's University, Belfast.

## ADVERTISEMENTS

First advertising forms go to press thirty days in advance of the date of issue. In forwarding copy, time must be allowed for setting up and submitting proof. All communications for space must be sent direct to the advertisement controllers : Messrs. Geo. A. Stewart & Co., Publicity House, 100 High Street, Belfast. 'Phone : Belfast 23091.

## DATES OF PUBLICATION

1st January, 1st April, 1st July, 1st October.

# THE ULSTER MEDICAL SOCIETY

THE MEDICAL INSTITUTE,
COLLEGE SQUARE NORTH,
BELFAST.

Dear Sir (or Madam),

If you are not a member of the Ulster Medical Society, we would appeal to you to give the question of joining your consideration. The Society has been in existence since 1862, and has always been active in keeping its members interested in the advances in medical science as well as in current professional affairs. The Medical Institute, situated in College Square North, belongs to the Society (through the generosity of Sir William Whitla), and is ideally adapted for meetings, committee meetings, and recreation. There is a library with current medical periodicals, and facilities for reference to medical literature are available in conjunction with the library at the Queen's University. There is also a billiards-room available to members, and lighter periodicals are also provided. An annual dinner is held each year in December, and a golf competition in June. Meetings are held at intervals of a fortnight during the winter months, and papers are contributed by members. Distinguished visitors are occasionally asked to contribute papers on subjects upon which they are specially qualified to speak. THE ULSTER MEDICAL JOURNAL, the official organ of the Society, is issued to all Fellows and Members free of charge.

The subscription to the Society is one guinea for Fellows and Members living in the country; two guineas for Fellows living in Belfast; and one guinea for Members living in Belfast who are not qualified more than seven years. The payment of a sum of twenty guineas entitles one to election to Life Membership.

May we, therefore, appeal to you to join the Ulster Medical Society, and so enable us to widen its influence and sphere of usefulness still further? Please make application to the Honorary Secretary, which will ensure your name being put forward for election to membership of the Society.

If you do not wish to become a member of the Society, will you consider entering your name as a subscriber to THE ULSTER MEDICAL JOURNAL? The subscription is five shillings per annum, payable in advance to the Honorary Treasurer.

We remain,

Yours faithfully,

P. T. CRYMBLE, *President.*
F. P. MONTGOMERY, *Hon. Secretary.*
C. A. CALVERT, *Hon. Treasurer.*

.....19.....

*To* MR. C. A. CALVERT,
8 UNIVERSITY SQUARE,
BELFAST.

*Please have my name proposed for election to the Ulster Medical Society.*

*Name*.....

*Postal Address*.....

.....

*Year of Qualification*.....

## BANKER'S ORDER

**(Ulster Medical Journal)**

.....19.....

*Name of Bank*.....

*Address of Bank or Branch*.....

*Please pay to the account of the Ulster Medical Society (Northern Bank, Shaftesbury Square, Belfast),* ULSTER MEDICAL JOURNAL *Account, the sum of five shillings, and continue to pay this amount on the 1st November each year until further notice.*

*Signature*.....

*Address* .....

2d.
STAMP

This should be sent to the Honorary Treasurer, MR. C. A. CALVERT,
8 University Square, Belfast, for registration.

THE
ULSTER MEDICAL JOURNAL

PUBLISHED QUARTERLY ON BEHALF OF THE ULSTER MEDICAL SOCIETY

VOL. VI 1st OCTOBER, 1937 No. 4

# THE 105TH ANNUAL MEETING OF THE BRITISH MEDICAL ASSOCIATION
## BELFAST, JULY, 1937

THE one hundred and fifth Annual Meeting of the British Medical Association, held in Belfast, July, 1937, has come and gone. Its success was assured from the beginning, both from the point of view of the scientific discussions and from the social aspects. Both were due to the unwearying care and arrangements made by the various sub-committees and secretaries, but mainly to the enormous amount of work and thought of Dr. F. M. B. Allen, the honorary general secretary, and Dr. R. W. M. Strain, the assistant honorary secretary. To both of these gentlemen the members of the British Medical Association in general, and the local members in particular, owe a great debt of gratitude, for the success of the Meeting depended on them almost entirely, and this success reflects on the local medical school, adding lustre to its already high reputation. To Professor R. J. Johnstone, the President of the British Medical Association for 1937-8, we offer our congratulations. He carried out his duties with the dignity which was expected of him, and his presidential address on Medical Education was, as the editor of the "British Medical Journal" wrote, "at once pointed, timely, and wise."

The main points in Professor Johnstone's address are printed in the following pages, together with the main arguments advanced by speakers in most of the more important discussions in the scientific sections. These, printed together in one volume, will make a permanent souvenir of the meeting, and a study of them will repay the time taken from the daily round of practice to read them.

# REPORTS OF SECTIONS

## BRITISH MEDICAL ASSOCIATION, BELFAST, 1937

PROFESSOR R. J. JOHNSTONE, B.A., M.B., F.R.C.S., F.C.O.G., M.P.

PRESIDENTIAL ADDRESS

## SOME THOUGHTS ON MEDICAL EDUCATION

THE subject on which I have chosen to address you is that of medical education—partly because I am much interested in it myself, as a teacher of graduate and undergraduate students of medicine ever since I got my qualifications; partly because, if one may judge by the numerous criticisms of our present system of education I have read, my interest is shared by many others; and lastly, because medical education should be a matter of vital concern to every practitioner and to every member of the B.M.A. Our profession cannot afford to recruit at a C3 standard. In this health-conscious age of ours, public authorities are constantly launching one health scheme after another. As experts, we ask for our collective advice to be taken and for our collective services to be employed. But the administrator has the last word, and while the specialist or the whole-time officer is practically immune from criticism, the incompetence or the carelessness of a few can be alleged as a reason for excluding the whole class of general practitioners from participation in a service. We are asking for our rights; but rights involve responsibilities, and if we are to enjoy the one we must be prepared to the last man to discharge the other.

I need not remind members of this Association of the many practical ways in which it shows its interest in medical education, "both post-graduate and undergraduate."

The critic of the medical curriculum assumes generally that we teachers at the end of five years should turn out a finished product, and goes on to blame us because we do not produce a finished general practitioner. No one expects us to turn out at the end of the five years a finished surgeon or anatomist or pathologist. Everyone recognizes that to form a specialist, years of post-graduate study and experience are required. Are we to take it that the path which the general practitioner must follow to reach the mastery of his art is shorter and less steep than that trodden by the specialist? I should say "No." The specialist must know almost eveything about his subject; the general practitioner must know something about almost every subject. He must learn to make his diagnoses without the skilled help which the specialist commands, and to carry out his treatment with makeshift appliances and with amateur nursing. He must acquire sufficient diplomacy to deal with unreasonable patients, with suspicious relatives, with inquisitive neighbours. He must keep in touch with the demands of public authorities and of public services. His responsibilities are, if anything, greater than those of the specialist, for he is the court of first instance, and on his decision the life of a patient may depend. His word may put a man in the dock or may even send him to the gallows.

I may very pertinently be asked how, if practice is essential to form a good practitioner, can licensing bodies be justified in giving a licence to practise to those who have not had this experience. I can only reply that after many years as an examiner and as an inspector of final examinations, I am satisfied that the dominant question in the mind of every examiner is, "Is it safe to the public to allow this candidate to practise?" and that only when that question can be answered in the affirmative is he permitted to pass.

As the practice of clinical medicine is the most important factor in forming the young practitioner, and is to be his chief pursuit in after-life, it is but natural that would-be reformers of the curriculum should demand that yet more clinical study should find a place in the education of the undergraduate. I should be the last to disagree, but do not let us be led into the fallacy of thinking that clinical training is the be-all and end-all of medical education.

A probationer nurse and a medical student have each approximately three years of clinical experience. The nurse's opportunities for clinical observation are infinitely better than those of the student. She probably sees at least as many patients, and she does not merely see them during a ward visit. She lives with them hour after hour and day after day, watches the course of their disease, notes its changing phases, and the effects of the methods of treatment that have been employed. She assists at consultations, and has the benefit of listening to clinical lectures. And she makes full use of her opportunities. Every one of us must acknowledge with gratitude the debt that we owe to the trained nurse for her help in diagnosis, in prognosis, and in treatment. But does her unrivalled clinical experience qualify her to take on the function of a medical practitioner? She would herself be the first to say "No." She lacks the background, the background of human anatomy and physiology and pathology, of knowledge of the human body in health and in disease, which the medical student has gained with much toil and sweat, and against which his clinical observations are composed and the picture interpreted. It is the main business of a medical education to provide an adequate background, and to teach how it should be used, and the more competently that work has been done the more valuable will his clinical experience be to the practitioner.

I have put human anatomy in the first place as a necessary preliminary to clinical study. Teachers of anatomy are usually the most popular of professors among medical students, so it is only poetic justice that their teaching should be a favourite point for attack by the critics. And perhaps the commonest criticism is that such an unconscionable time is wasted by the student in the dissecting-room.

Is his time really wasted? The successful clinician takes little upon trust, and so far as is possible checks all his facts by his own personal observations. But in almost all the sciences which the medical student must study he is compelled to take most of his facts on trust. Practical classes permit him to verify a few of them, but he has neither the time nor the technical skill to go further. When he takes up practical anatomy he is not merely permitted and encouraged, he is required by his own personal observation to verify the statements of his teacher

and of his textbook. This is an invaluable discipline, even if a knowledge of anatomy were of little use in practice.

If the anatomist is Public Enemy Number One of the medical student, Number Two is undoubtedly in the eyes of the critics the lecturer. We who lecture might conjugate the verb something like this, "We are dull, you are falling asleep, they think we should be painlessly exterminated." The pity of it is that the critics are to some extent in the right. There are dreary lectures and dull lecturers, and that is a pity, for after one has listened to a master of the art, one is left in no doubt as to the superiority of the spoken word above the printed page in rousing interest in a subject and in presenting it in the round.

I do not propose to take up your time with further discussion in detail of the medical curriculum and of its critics. Doubtless it could be improved, but, so far as I have been able to find out, our medical students get a training certainly not inferior to that given in any other civilized country. Of course the curriculum is overcrowded, but fortunately the brain of the undergraduate is elastic and has a well-developed protective mechanism of forgetfulness, not likely to atrophy through disuse.

However much or little the young graduate has retained of the many sciences he has become acquainted with, he has had a training in observation, in the use of his critical and his reasoning faculties, and in dealing with men, women, and examiners. He has the key to the vast storehouse of knowledge contained in medical literature, for he is familiar with its idiom. He now enters on the most important stage of his lifelong medical education. He now becomes his own teacher and taskmaster. He has had to learn to think like other people: he must now think for himself. Only thus can he transmute the ore of scientific knowledge into the gold of clinical wisdom.

In all but a few instances the neophyte begins his career under the tutelage of one of his seniors, either a member of a hospital staff or a general practitioner; and that senior has a great responsibility, for by precept and still more by example he may make or mar a promising practitioner. Both in this stage and later, when he has at last hoisted his own flag, the young physician need not want for aids in self-education. Nothing is more helpful than a circle of medical friends who are not ashamed to talk shop. Of much the same kind of value is the consultant who is willing to discuss cases at length. Lectures in a classroom may be indigestible fare for the undergraduate; delivered in a medical society they seldom fail to provide food for thought for the graduate student. His journals may be relied on to add to and to correct his store of knowledge.

We have long had the post-graduate course for the few, but the National Health Insurance Act has now made it available for the many. If I might be permitted to criticize, I think that most post-graduate courses try to administer to their medical patients what used to be called a "blunderbuss prescription," and that too much is attempted in too short a time. But these courses are still in their infancy. Some day we may see the general practitioner going back to his old school or to another school for a month, for three months, even for a sabbatical year, both to learn and

to teach. And he will come back to his patients as he does from our present courses, not merely with his skill enhanced, but, what is perhaps more important, with his interests freshened and his enthusiasm rekindled.

But I must bring to a close these disjointed remarks on medical education. I have no anxieties about our students. Their standard is as high as it ever was, and they are getting and taking an excellent training. To the seniors, of whom I am one, the two-edged compliment may be paid that it is impossible to improve us. But I do feel uneasy about the most important class in the profession, the young general practitioner in the twenties and the early thirties. Is panel practice giving him an assured income too soon? Is it taking away the incentive to study and to improve the standard of his practice? Is he sitting down at ease in Zion? I trust not, for it is upon him that we depend to maintain and to raise the prestige of our profession.

. . . . . .

CRIGHTON BRAMWELL, M.A., M.D., F.R.C.P.
*Physician to the Manchester Royal Infirmary*

## THE TREATMENT OF HEART FAILURE

HEART failure is a relative term: it signifies that the supply is unequal to the demand. The latter varies greatly according to the habits and mode of life of the patient. The reserve of the healthy heart is amply sufficient to meet all contingencies. In the trained athlete during severe physical exertion the heart is able to increase its output sixteenfold (Hill, 1927): in patients with advanced heart failure it is unable to satisfy the requirements of the body even at rest. Between these two extremes all intermediate degrees of cardiac efficiency and insufficiency may be observed in health and disease.

Diseases of the heart muscle, the heart valves, or coronary arteries entail a reduction of the cardiac reserve proportional to the severity of the structural lesion. The resulting disability is of a permanent nature. Lack of exercise, and toxic causes such as focal sepsis and recent infection, add to the disability by producing a biochemical change in the buffering mechanism in the muscles. This reduces their mechanical efficiency; they work less economically, and for the same output of energy require a greater supply of oxygen than "trained" muscles, thereby increasing the demand upon the heart. The myocardium itself suffers in a similar way. The result is twofold—an increased demand upon the circulatory mechanism and a diminished capacity of the heart to meet this demand.

In patients with heart disease the tolerance for exercise may be limited by dyspnœa or by pain. In the past it has been customary to restrict the term "heart failure" to congestive heart failure, a condition in which the venous pressure is raised and dyspnœa is the presenting symptom. I would strongly advocate that the term "heart failure" should include angina, in which the tolerance for exercise is limited not by dyspnœa but by pain. My reasons for making this suggestion are two. In the first place, the term "heart failure" signifies inability of the heart to

maintain a circulation adequate to meet the requirements of the body, and it is immaterial whether this inadequacy manifests itself by dyspnœa or by pain. The mechanism of production of these two symptoms is essentially the same. When the output of the heart is insufficient to meet the requirements of the active muscles, lactic acid accumulates in the body and the hydrogen-ion concentration of the blood rises. This stimulates the respiratory centre, and dyspnœa is produced. When, on the other hand, the coronary arteries are diseased, the effects of anoxæmia are first felt by the myocardium, and the patient's activities are limited by pain. My second reason is that the general indications for treatment in congestive heart failure and in angina of effort are identical—namely, to adapt the life of the patient to the limitations imposed by his damaged heart; in other words, to enable the heart to balance its budget.

When one considers the causes of heart failure, it is obvious that in different types of heart disease the burden falls unequally on the two ventricles. In hyperpiesia, for example, the left ventricle has to bear the strain; and not until it has given way does the right ventricle become embarrassed. Conversely, in patients with mitral stenosis or emphysema, the burden falls on the right ventricle.

Failure of the right ventricle leads to venous engorgement in the systemic circuit. The veins of the neck stand out like knotted cords, the liver enlarges, and œdema makes its appearance in the dependent parts. Failure of the left ventricle, on the other hand, leads to venous engorgement in the pulmonary circuit. This is well shown in a radiograph.

In most cases of heart failure both ventricles are involved to a greater or lesser extent; but when the left ventricle alone is at fault there may be intense congestion in the pulmonary circuit without any œdema or engorgement of the systemic veins. Left ventricular failure manifests itself clinically by certain signs, which merit special consideration. They are paroxysmal dyspnœa, presystolic gallop rhythm, and pulsus alternans.

Paroxysmal dyspnœa or, as it is sometimes called, "cardiac asthma," differs from the more common dyspnœa of effort in that it is not brought on by exertion, but occurs at rest. It often wakes the patient from sleep in the early hours of the morning. The attack itself may closely simulate bronchial asthma. A slight attack may pass off in a few minutes, but a severe one will last half an hour or more and may develop into acute pulmonary œdema. In this condition adrenaline and other asthmatic remedies fail to give relief, but morphine acts as a specific. Twenty or thirty minims of nepenthe at bedtime will often ward off attacks.

Since physical exertion makes the greatest demand upon the heart, the most important consideration in the treatment of heart failure is rest.

During the past eight years I have had occasion to make a special study of patients in whom heart disease was complicated by pregnancy. As pregnancy advances it imposes an increasing strain upon the heart: consequently the great danger to which these patients are exposed is the development of heart failure. Over and over again in the course of this investigation we have been impressed by the fact that when heart failure is recognized in its early stages, the response to

treatment is extremely satisfactory; whereas in patients who are admitted to hospital as "urgencies" with fully developed heart failure the mortality is very high. Early diagnosis is the secret of success in treatment.

In incipient heart failure, slight curtailment of the normal activities may suffice, but in advanced cases, where the patient is œdematous or is subject to anginal attacks on slight exertion, complete rest for a period of several weeks may be necessary to enable the exhausted heart to build up a reserve. Under these circumstances the patient must be confined to bed, and attended by competent nurses who will feed him, wash him, and lift him when he wishes to change his position or to use the bed-pan. He must not be allowed to make any physical effort himself, or even to attempt to assist his nurses.

When convalescence is complete, the future still remains to be considered, and precautions to prevent a recurrence must be observed. The patient should be warned against over-exertion on the one hand, and insufficient exercise on the other. Whereas the former may bring about a further breakdown, the latter imposes an additional burden on the damaged heart. The advice which I give to patients with chronic heart disease may be summarized by saying that they must lead a life of moderation in all things, taking regular exercise within the limits of their tolerance, but never making any sudden effort which entails exerting their full strength, or which gives rise to dyspnœa or precordial discomfort. For example, they must never run for a train, struggle against the wind, or attempt to start their motor-car by hand. They must stop eating before they are satisfied, and sit quietly in a chair for half an hour after meals.

When walking or participating in any form of physical exertion, the patient must set the pace. He must never attempt to keep up with others fitter than himself. The problem of suitable employment often presents insuperable difficulty. As in the case of physical exertion, the patient should, if possible, be his own master, and able to go to work or stay away at will.

Stair-climbing should be eliminated. A bungalow is the ideal residence for these patients. Failing that, the bedroom should be on the ground floor. One whole day in bed each week is often helpful. It enables the heart to build up sufficient reserve to ward off a further breakdown.

A special danger when the cardiac reserve is limited is the added strain upon the heart of intercurrent infection. An attack of bronchitis, influenza, or even a common cold necessitates immediate confinement to bed.

Obesity imposes an additional burden on the damaged heart. Moreover, it establishes a vicious circle. By diminishing the cardiac reserve it limits exercise, and lack of exercise tends to increase obesity. To break this circle, a diet low in fats and carbohydrates should be prescribed, together with massage and carefully graduated exercise.

In the treatment of all cases of heart failure, but especially in those of the ischæmic type, the nervous aspect calls for most careful consideration. The typical anginal subject is by no means a neurotic; but some patients are more highly strung than others, and the nervous pattern of the individual is largely instrumental

in determining the clinical picture, which it may modify in various ways. In the first place, the threshold of sensitivity for pain varies in different people. Other things being equal, the limitations imposed on the patient's activities are much greater in the hypersensitive than in the hyposensitive subject. Secondly, in the irritable, "nervy" type of individual, an anginal attack may be precipitated by a relatively trivial emotional stimulus, since the detonating mechanism which fires off an attack lies dangerously near the surface. Minor annoyances will, in him, suffice to produce an emotional storm adequate to provoke an attack; whereas in the calm, placid person a much more massive stimulus is required to do so. Thirdly, there is the element of anxiety, for angina is proverbially associated with sudden death.

In a nervous subject, stage-fright induced by having to make an after-dinner speech or, in an irritable business man, shortness of temper provoked by a heated discussion at a board meeting, may be warded off by a prophylactic dose of bromide. In acute congestive heart failure morphine is a most valuable drug. By allaying both physical discomfort and mental anxiety it helps to achieve that peace of body and mind which is so essential to effective treatment. When, in acute heart failure secondary to auricular fibrillation, urgency demands the administration of digitalis by the massive-dose method, I generally give a hypodermic injection of one-fourth of a grain of morphine before the first dose of digitalis. Many practitioners fail to realize that the danger of giving an opiate to cardiac patients may be far less than the danger of withholding it.

Anxiety figures prominently in the picture of heart disease. To disregard this "complication," and to make no attempt to alleviate the mental suffering, is to neglect an important aspect of treatment. To achieve success in this sphere, the physician must possess both sympathy and understanding. He must be able to put himself in the patient's shoes and to regard the case from the patient's point of view. When illness is prolonged, the possible physical damage that may result from allowing a patient to return to his work, must be carefully weighed against the mental trauma when he sees his business going to pieces and the future of his dependants at stake.

. . . . . .

A. J. CLARK, M.D., F.R.C.P., F.R.S.
*Professor of Pharmacology, University of Edinburgh*

## INDIVIDUAL VARIATION IN RESPONSE TO DRUGS

EVERY practitioner is familiar with the wide variation shown by patients in their response to drugs. Therapeutic success is indeed dependent on the adjustment of standard methods and doses to the needs and peculiarities of the individual patient. Such work makes special demands on clinical skill and experience, and here the general practitioner has a special advantage on account of his knowledge of the patient's peculiarities. It is sometimes suggested that medical practice is degenerating into an automatic process whereby diagnosis is supplied by a laboratory and the appropriate remedy by a therapeutic index. The individual variation of

patients will, however, always constitute a factor limiting such tendencies, and is a sure safeguard against the replacement of practitioners of medicine by automatic machines.

The usual method adopted in the administration of powerful drugs is to start with a small dose and to observe the effects produced on the patient before working up to a full dose. The administration of a volatile anæsthetic is an example of the manner in which it is usual to correct individual variation, since the anæsthetist concentrates his attention on each patient, and by suitable adjustment of the dosage secures a uniform response. Most well-established clinical methods of drug administration are of this type, and hence the range of individual variation is masked, and, indeed, is in danger of being forgotten. This is evidenced by the fact that from time to time therapeutic methods are recommended which involve giving patients a full dose of some potent drug without providing any opportunity for first determining their individual response to it. In most cases such a risk is unnecessary, but in some it is very desirable to produce a full therapeutic action as quickly as possible, and therefore to know the probable limits of human variation. The chief problem is to ascertain what ratio between the average lethal dose and the therapeutic dose represents the reasonable margin of safety in the use of a drug. The extent of the margin actually desirable is a matter of opinion, but it will be generally agreed that if a therapeutic measure involves the chance of producing a serious toxic effect in one case in one thousand, it must be considered dangerous. This chance will, therefore, be taken as representing the limit of justifiable risks.

The lethal dose of a drug is a primary pharmacological constant, and usually is one of the first characteristics to be determined. Until about twenty years ago the method employed was to give varying doses to a dozen or a few dozen animals, and to term the smallest dose which produced death the minimum lethal dose. Such doses were stated as mg./kg., and thus correction was made for variations in body-weight. The introduction of the biological standardization of drugs necessitated increased accuracy in such methods, and as soon as systematic investigations were made, it was found that animals showed a considerable individual variation in their responses to drugs, and that consequently the methods that had been in use for a century were inherently inaccurate.

The drugs that are shown by animal experiments to be possibly safe are obviously of chief interest, and the problem is to estimate what proportion of the mean lethal human dose is safe therapeutically.

We are at once confronted with the difficulty that the mean lethal human dose is not known for any drug. In the case of a common poison such as strychnine, textbooks of toxicology provide the following information. Several deaths have occurred when the dose taken was probably not more than half a grain. Deaths from one-fourth of a grain have been reported, but the evidence is less certain. Recovery after two grains is fairly well established, and recovery after three or four grains has been reported. Obviously it is nearly always difficult, after the occurrence of a fatal case of poisoning, to obtain reliable information as to what dose has been taken. Furthermore, in the case of recovery it is always uncertain how much of the drug

has been removed unabsorbed from the stomach. It is worth noting, however, that animal experiments with strychnine proved that there was a fourfold range of dosage between the least resistant and the most resistant five per cent. of a population, even when every endeavour had been made to exclude causes of variation. Hence there is no reason to suspect the statement that some adults have been killed with half a grain of strychnine and others have recovered from two grains.

The animal experiments are of service in that they provide formal proof of the fact that the minimum lethal dose is a quantity which cannot be measured accurately. It is simply the smallest dose that has been observed to be followed by death, and in the case of any drug the greater the population observed the lower the value is likely to be. It is therefore preferable in toxicology to describe the usually fatal dose, and to avoid the term "minimum lethal dosage."

Quantitative evidence regarding the extent of the variation of the response of patients to therapeutic doses of drugs is very scanty. In the case of the narcotics used for pre-anæsthetic medication, a certain amount of accurate evidence exists, because drugs such as nembutal, sodium amytal, and evipan are given by slow intravenous injection until a constant response is produced. Such methods provide exceptionally accurate evidence regarding the relation between dosage and the incidence of a constant response in a human population. For example, Paxson (1932) measured the amount of sodium amytal (mg./kg.) needed to produce analgesia in fifty-five women during labour. His results show that sixteen per cent. were narcotized by three-fourths of the median narcotic dose. This is a variation like that found for the lethal action of sodium amytal in cats, rabbits, and rats (Swanson and Page, 1927), and for the related compound sodium nembutal given intravenously to dogs and rats (Swanson and Shonle, 1931) and to rats alone (Gage, 1933). Variations of a similar extent were observed in these cases in respect to both the hypnotic and the lethal doses.

Similarly in the case of avertin given per rectum, the figures of Flörcken and Mues (1928) indicate that fifty per cent. of the population is narcotized by eighty per cent. of the dose needed to narcotize eighty-four per cent. The scatter as regards the proportion of a population of rats anæsthetized by graded doses of avertin (per rectum) is of similar extent (Lendle, 1928; Tunger, 1931; Gage, 1933). These facts permit the calculation of the extremes of response that are likely to be encountered in a population of one thousand.

The measurement of the concentration of alcohol in the blood seems to be a much more accurate and scientific method than the estimation of the degree of intoxication by clinical examination. It is important to note, however, that although the former method may indicate accurately the amount of alcohol that has been taken, it does not indicate the effect produced on the patient, because there is a wide individual variation in regard to alcohol concentration in the blood and intoxication. This is shown by the reports of Bogen (1927) and of Widmark (1932), which describe in 250 and 562 cases respectively both the blood concentration of alcohol and the result of clinical examination. The results agree in demonstrating that a concentration of alcohol in the blood of 1.2 mg. per 100 c.c. produces intoxication

in fifty per cent. of cases. Bogen's figures show that intoxication was not present in any case in which the alcoholic content was less than 1 mg. per 100 c.c.; whilst Widmark found none below 0.8 mg. per 100 c.c., but observed six cases of intoxication in thirty individuals in whom the blood concentration was between 0.8 and 1.0 mg. per 100 c.c. At the other end of the scale Bogen's figures show one case in thirty-five not intoxicated with 4 to 5 mg. per 100 c.c., whilst Widmark's figures give one case in twenty-nine not intoxicated by 2.5 mg. per 100 c.c. The ratio between the alcohol concentration not intoxicating the most resistant individuals, and that affecting the least resistant individuals, was about fourteen to one in Bogen's series and three to one in Widmark's series.

Examination of the figures shows that they do not follow the ordinary laws of probability, for there appears to be a lower limit of about 0.8 mg. per 100 c.c., below which intoxication never occurs; whilst a concentration of 1.2 mg. per 100 c.c. produces intoxication in about fifty per cent. of the population, but the more resistant half of the population reveals a wide scatter.

Digitalis preparations are standardized biologically on cats and on frogs, and figures for the variation in response of large populations are available. These figures show a very uniform response. The ratio $D_{16}/D_{50}$ is about 0.9, and hence a dose equal to seventy-three per cent. of the mean lethal dose would kill only one individual in one thousand. Such results suggest that massive digitalis therapy may be a relatively safe procedure. There is, however, a very wide difference between a population of healthy cats and a population of patients with heart disease of varying intensity. Animal experiments prove that this apparent uniformity in response is obtained only when acute toxic effects are observed. For example, Bauer (1933) gave cats one-half a lethal dose of digitoxin, and then measured the amount of ouabain required to produce death twenty-four or forty-eight hours later. The individual variation with this latter dose was far greater than that found for the acute lethal action of these drugs. The ratio $D_{16}/D_{50}$ was about 0.75, which indicates that one animal in one thousand would be killed by forty-two per cent. of the M.L.D. Similarly Li and van Dyke (1936) measured the individual variation in respect of cumulative poisoning in frogs. They gave doses of digitalis glucosides on alternate days, and their figures reveal a wide variation in the sum of the doses needed to produce death. The ratio $D_{16}/D_{50}$ is about 0.4, and in this case a mortality of one in one thousand would probably be produced by one-sixteenth of the M.L.D. Even with healthy animals, therefore, the uniformity of response to digitalis glucosides ceases as soon as opportunity is allowed for the action produced to be influenced by such factors as rate of excretion, etc.

The variability of the response of patients to digitalis therapy is well known; the following quantitative data give an indication of the scatter that occurs.

Murray Lyon and Gilchrist (1927) measured the changes produced in the heart-rate by massive digitalis therapy. Their results show that the reduction in heart-rate in cases of auricular fibrillation may range from twenty to one hundred beats per minute, and that the greater the initial rate the greater was the reduction. In their group of thirty-seven cases with an initial heart-rate between seventy and one

hundred per minute, the reduction varied from eighteen to forty-five per minute in fibrillating cases, and from none to forty per minute in non-fibrillating cases. Alstead (1936) noted the daily dose of powdered digitalis that produced nausea in thirty-six patients. In two, nausea was brought about by one grain daily in a week, whilst in twenty-two cases two grains daily did not produce nausea in two weeks. Figures such as these indicate a wide individual variation in the response of patients to digitalis. The scatter of the variation appears to resemble the scatter observed in cumulative experiments on animals much more closely than the relatively uniform responses met with when the acute lethal dose of digitalis for animals is measured.

The question of practical interest is the probable margin of safety in massive digitalis therapy. The existing evidence is admittedly imperfect, but it suggests that in different individuals there is likely to be at least a fourfold range in the quantity of a drug required to produce a given effect. Consequently it appears to be very improbable that any method will be found of judging *a priori* the dose needed accurately enough to ensure that the average therapeutic dose will not produce severe overdosage in the occasional patient who is abnormally sensitive.

. . . . . .

J. P. MARTIN, M.D., F.R.C.P.
*Physician to Out-patients, National Hospital for Nervous Diseases, London*

## MENTAL SYMPTOMS WITH HEAD INJURIES

In considering the mental disturbances which frequently result from injuries to the head, we recognize at once two groups or chains of possible causal circumstances. The first consists of material elements, and comprises the organic injuries of the brain and the subsequent reactive changes in the cerebral tissues. The second consists of abstract elements, and comprises the emotional disturbances connected either immediately with the accident itself or with the patient's adaptation to his normal life afterwards, and if the question of compensation arises, it adds to this group further elements of anxiety and possibly greed.

The subject as I see it contains the following groups. In many cases the initial mental symptom is unconsciousness, using the word in its popular sense, that is to say, the abolition of all signs of mental activity.

1. Cases of "simple concussion," in which the unconsciousness is usually of brief duration and there is no indication that the brain has been organically injured.—I have never seen any mental symptoms persisting in such cases, with the exception of one quite atypical instance which will be referred to in group 7.

2. More severe cases in which unconsciousness is usually more prolonged and there is conclusive or strong presumptive evidence of organic injury of the brain.—In such cases recovery is occasionally characterized by profound mental disorder lasting many weeks or months, and it is with such instances that we are most concerned to-day.

2*a*. Rare cases of this kind in which, recovery having reached a certain stage,

relapse occurs and the patient sinks into a state of dementia—cases of "late dementia."—I have seen three such cases, and the descriptions of them are fairly common in the literature.

3. Cases in which there has been actual loss or known gross local damage of cerebral tissue which may in itself be responsible for mental changes.—I have in mind cases with open wounds through which cerebral tissue has been extruded, cases of gunshot wounds and perforating injuries, and cases of depressed fracture in which the damage is local.

4. Cases of intracranial hæmorrhage, particularly (*a*) traumatic subarachnoid hæmorrhage and (*b*) subdural hæmatoma.—Here the physical factors appear relatively simple, while on the psychological side the cases differ obviously from those of the earlier groups, in that the sequence of mental changes is from normality down to coma instead of from coma upwards.

5. Cases in which fits occur and are accompanied by intellectual deterioration.—The fits are likely to begin long after the injury, and the factors responsible for the mental symptoms are evidently different from those operating in the preceding groups.

6. Cases of so-called "traumatic neurasthenia."—The physical evidences of injury, if there were any, have disappeared (or have been allowed for), but mental symptoms of a nature that we know well have become apparent. This is a difficult and complex group, which we must try to unravel.

7. Cases in which the mental symptoms, persisting after the accident, are those of one of the well-known types of psychosis, and probably depend essentially on the patients' antecedent mental constitution.—Cases of manic-depressive psychosis, schizophrenia, and paranoia have all been described following head injuries. I have myself had one case in which a dementia with schizophrenic features followed, in a boy of 16, an injury which caused unconsciousness for about half an hour and no outward signs of organic damage to the brain. It is difficult to know for how much the physical factors count in such instances.

The earlier groups in my list are characterized by loss of consciousness as popularly understood—that is to say, by the abolition of all signs of mental activity. As the patient gradually regains his mental faculties, at what stage are we to say that he is "conscious"? How are we to know when he is aware of himself and his environment? It is evident that an important stage in awareness has been reached when he begins again to remember, and is consequently able to realize changes in his environment. I therefore accept the suggstion that the patient should be considered to be "unconscious" as long as he has no conscious memory, but it must be realized that in many cases the return of memory is not by any means a clearly demarcated step.

In group 2 the physical concomitants usually leave little doubt that the brain has been organically damaged. There are probably signs of fracture of the skull, and possibly cranial nerve disturbances, reflex changes, signs of local weakness of limbs, or blood in the cerebro-spinal fluid. The initial stupor may last a number of hours or several days, and the return of mental activities is gradual. In the vast

majority of cases the patient passes as "normal" mentally within a few days, and if I describe cases in which recovery takes weeks or months, it is because of their great intrinsic interest, and because of the light they throw on other cases. The course of events is somewhat as follows :—

The patient ceases to be entirely passive when moved or touched, and makes grimaces or movements—and soon utters words—expressive of resentment. Within a very short time he may become abusive when attended to or even when spoken to. The phase varies in duration from a few hours to many weeks. It is sometimes accompanied by periods of restlessness and noisiness, and I have had several cases in which the patients for weeks were almost continuously noisy and unruly throughout their waking periods. As the patient's abusiveness subsides, and it becomes possible to carry on a conversation with him, two features become apparent—confusion and perseveration of words.

There may follow a more subdued state in which the patient answers rationally but is disorientated in time and place. This again may last many weeks.

One of my patients, a consulting engineer, spent a considerable part of his recovery period (in imagination) in India, inspecting a line and locomotives with which he had been concerned several years before. Dr. Symonds in a recent paper has given an interesting description and analysis of this disorientation in time. My patient, when questioned, was ready to confabulate about his daily visits to India. Another, confined for months to hospital, used to describe visits to his own home and the very questionable goings-on of his grown-up sons and daughters. These two patients thus showed all the elements of Korsakoff's psychosis. One of them had a good deal of subarachnoid hæmorrhage, and I think that, apart from this, confabulation is uncommon; in my experience it does not occur during the more rapid recoveries.

During this time memory begins to return. The answer to the question, "Have you ever seen me before?" instead of being "No" or "I don't know," becomes "Yes, I think so," and finally "Yes" with confidence, and eventually he can tell exactly when, and describe what conversation took place. Memory at first—like developing memory—is patchy.

When the talkative phase has subsided, or if it has never been present, the patient's general mental activity, as evidenced by his behaviour, is in most cases low. He lies idly in bed or sits about, seldom speaks spontaneously, or if he does, he is muttering something to himself about years long past; he takes little interest in what is going on around him and shows no desire to read or to engage in other forms of mental or physical activity. At first he carries out single actions in response to an immediate external stimulus. As he improves, the effect of an external stimulus is more prolonged, it produces more and more "affective drive," it carries him farther. Part of this improvement may be due to conditioned reactions, but it is apparent where conditioning can hardly be a factor, and the patient performs new activities with less and less urging. At a later stage he takes in stupidly something of the content of the picture on the front page of a newspaper that is placed

on his bed. As the days pass he looks at the pictures with more understanding, but it may be a long time before he reads the headings of the paper with intelligence.

When memory returns and he becomes normally orientated, he begins again to talk spontaneously. At first his observations are about his immediate requirements, but gradually what is said to him arouses more and more response, and the reverberations of a previous conversation give rise to spontaneous statements. Simultaneously, spontaneous physical activity has been developing. It may, however, be years before the patient regains the degree of spontaneous mental and physical activity that he had before his accident, or he may never regain them. We shall return to this point in considering " traumatic neurasthenia."

### The Emotional State

The patient's emotional state throughout this period of intellectual recovery may go through different phases. At first he is irritable, resentful, abusive. At a subsequent stage he is often elated and childishly happy. This has already been described, and I shall refer here only to some of the possible consequences of it. The patient is so pleased with himself, and so anxious to be nice to everyone, that he would be easily wheedled into making a bad settlement of whatever claim he might have for damages. One of my patients told me how a "terribly nice gentleman" had visited him at his home in Sussex, and explained to him that the accident (in which he had been knocked down by a lorry) was entirely his own fault, and that he would have no claim for damages at all. I do not suggest that any advantage was taken in this instance of the patient's pleasure and simplicity, but the danger is obvious.

You will have gathered from the trend of my remarks that even when mental symptoms are prolonged, their course is towards recovery, and in the majority of cases recovery is eventually complete. It is seldom more than six months before the patient behaves as a normal individual, though he may still have some residual symptoms. But you will have noticed, too, that I made a special sub-group of certain cases in which, after partial recovery, the patients lapsed into a state of dementia which persisted. I cannot stay to discuss this group further, but it is noteworthy that the youngest of my three patients was fifty-five years of age.

In all the cases we have been discussing, the symptoms have begun with stupor and gradually or quickly receded. It is possible in two small groups of cases to watch the progress of the symptoms in the opposite direction, from normality down to stupor. I refer to the cases of sub-arachnoid and subdural hæmorrhage resulting from injury. In the former sub-group, stupor may take from a few minutes to an hour or two to develop, according to the severity and perhaps the site of the hæmorrhage. There is little opportunity for first-hand observation, and we are dependent on the account of the symptoms given by the patient's friends. The symptoms are excitability, restlessness, irritability, unreasonableness, lack of reserve, an activity which is rational but not fully comprehended, and which is accompanied by a hypomaniac "damn the consequences" emotional state, disorientation, confusion, then diminishing activity, paraphasia, drowsiness, stupor. In the

cases which recover, some degree of irritability often persists throughout the stupor. One of my patients who, after a relatively gentle fall from a horse, was forced by his friends, because of his unreasonable behaviour, to go to bed, got up almost as soon as they had left the room, pulled on some clothes, rushed to the garage and got out his car, and drove forty miles before he was found comatose over the steering-wheel. His cerebro-spinal fluid, when I took it twenty-four hours later, was indistinguishable in appearance from blood. Memory eventually returned for the early events and the first thirty miles or so of the drive.

What is the physical pathological basis of the cases with pronounced and long-lasting mental disturbances?

One of my patients died sixteen months after the injury. She had recovered to a great extent by the end of eight months—sufficiently to be removed to her own home from the private mental home where she had been cared for—and for another three or four months she had maintained her improvement. But she had a physical disability as well, and following a severe fall down a few steps in her own house her mental state suffered a considerable relapse. She was a vigorous old lady of 76, but after that she gradually deteriorated mentally and physically. The post-mortem examination revealed a well-healed depressed fracture in the right frontal region, and a corresponding shallow but wide depression on the antero-lateral aspect of the right frontal lobe. Behind and below this there was an area of brown staining on the cortex, evidently corresponding to old sub-arachnoid or sub-pial hæmorrhage, but there was no sign of laceration of the cortex. Over the parietal and occipital areas on the same side the dura was covered, on its inner side, with a delicate brown membrane indicating old subdural hæmorrhage. Thorough examination revealed no lesion within the brain, and there were no signs of a contrapolar lesion.

The details of this case do not, I think, matter greatly. I wish only to draw attention to the widespread nature of the abnormalities. I referred to the cases of meningeal hæmorrhage, because in them we had similar symptoms associated with anatomical conditions which must involve depression of activity over a large area of cortex. That the changes in the cortical cells need not be profound is, I think, shown by the quick recovery of the patient's alertness and intelligence after removal of a subdural hæmatoma (provided the patient has not reached the stage of coma).

Whatever the disturbance is which effects the cortex, it is one that in certain cases takes months to recover. Oedema, hæmorrhage, the effect of degenerating blood elements on the cortical cells, damage to association tracts, and the reactive changes of the interstitial elements in the brain, may provide an adequate explanation of disturbance and recovery over such a period. There is also another pathological feature which may be of great importance in this connection. Dr. Greenfield has recently found massive demyelination with persistence of axis cylinders in portions of injured brains that had evidently experienced severe œdema. We know from other diseases that although the axis cylinders in an area of demyelination survive, their function is evidently impaired for a considerable period, and when

we recall the severe dementia in cases of demyelinating encephalitis, and the complete recovery from it which occurs in the course of several months, we realize that this finding of Dr. Greenfield's offers an explanation of the course of the traumatic cases.

. . . . . .

J. W. McNEE, D.S.O., M.D., D.Sc., F.R.C.P.
*Regius Professor of Practice in Medicine, University of Glasgow*

## CHOLECYSTITIS

THE combined results of many observations, by physicians, surgeons, and pathologists, seem to show without any doubt that disease of the gall-bladder is now the commonest of all organic troubles of the digestive tract. Statistics in different countries and in different clinics must vary, but Piersol (1936) has recently published an analysis of the admissions to the gastro-intestinal clinic of the University of Pennsylvania Hospital, Philadelphia, and shown that 16.3 per cent. of all the patients were diagnosed as suffering from gall-bladder disease. Duodenal ulcer, cancer of the stomach, gastric ulcer, and chronic appendicitis followed, in this clinic, in order of frequency, but these are merely the results in one American hospital. I am unable to present the statistics of any large series of gastro-intestinal cases in Britain, but it may safely be accepted that cholecystitis is now established here as one of the most common and important intra-abdominal diseases.

Biliary stasis is the only important predisposing cause on which almost everyone will agree. It would not be justifiable here to discuss disturbances in the physiology of the biliary tract as a cause of biliary symptoms, since so little is known with absolute certainty. Problems concerning the sphincter of Oddi, contraction of the gall-bladder, and spasm of the main bile ducts are all involved here; and frankly, in my view, the time has not yet come to bring questions of disturbances in the physiology of the biliary tract into the realms of ordinary practical medicine. We have enough to deal with at present in gall-bladder pathology, due to microbic infection of the gall-bladder and biliary tract, the essential cause of the subject we are discussing—namely, cholecystitis. Here, too, we are faced with the difficulty of reconciling the divergent views of physicians and surgeons for the ultimate benefit of our patients.

The points of importance are :—

1. What are the common microbes which infect the gall-bladder, and what is their relative frequency?
2. By what route is the gall-bladder infected—by passage along the ducts from the intestine or by the blood-stream? Do the microbes in fact exist mostly or partly free in the bile, or is the infection essentially deep-seated in the wall of the gall-bladder?

The importance of focal sepsis in teeth, tonsils, appendix, or elsewhere in the etiology of cholecystitis is an essential part of the problem, which I only mention to give a lead to other speakers.

Moynihan, in the days of the rapid development of gall-bladder surgery, described the "inaugural symptoms of gall-stones." These, we know now, were the symptoms of the preceding cholecystitis, but at a later stage than we must now identify them. At that time it is fair to say that the early stages, except of the acute and fulminating variety constituting a surgical emergency, were seldom, if ever, sought for or recognized. It is the diagnostic problem involved in the common catarrhal cholecystitis that I wish to emphasize, and a correct diagnosis will often be missed and treatment too long delayed if the doctor fails to investigate further all cases of dyspepsia which do not respond to simple treatment. He may, and generally does, consider the possibility of gastric or duodenal ulcer, and is disappointed when the X-ray examination by barium meal which he advises yields a negative result. He may think of chronic appendicitis; but up to the present he too seldom thinks of the great frequency of gall-bladder disease as a cause of dyspepsia. The association of flatulence with the dyspepsia is suggestive, but by no means conclusive.

It is best at the very outset, when considering how the diagnosis of gall-bladder infection may be proved, to admit the difficulties involved. The diagnosis by simple clinical methods is easiest when gall-stones are present. Biliary colic occurs in roughly forty per cent. of such cases, while recurrent acute attacks of cholecystitis lead to obvious signs in the right upper quadrant. Further, it is in these cases that cholecystography is of such great diagnostic value. Even when gall-stones are absent, the local signs of tenderness or pain, or both, below the right costal margin may, along with the presence of flatulent dyspepsia, strongly suggest the diagnosis. The real difficulty arises in the cases, and they are many, in which a catarrhal cholecystitis is associated with dyspepsia only, without local pain or tenderness over the gall-bladder at all. These are the early cases in which medical treatment may be of avail. How are such cases to be completely and positively diagnosed so that proper treatment may be carried out? Ordinary simple clinical methods fail us here—what help can we obtain by other methods?

I must first refer to cholecystography. I will only say that in my experience the use of the dye followed by X-rays generally does not yield information of positive diagnostic importance in the early stages of catarrhal cholecystitis in which physicians are so interested. I am too often confronted with an X-ray diagnosis of "pathological gall-bladder," or "poor filling," or "faintness of shadow," which are taken to indicate disease, when I know full well the differences in technique and the differences in absorption of the dye, even on different days, in the same person. On the whole, I am still very chary of accepting a diagnosis of early cholecystitis on X-ray evidence, whatever improvements in technique may later make possible.

We now come to the method of duodenal intubation, and here I feel on quite different and much more secure ground. I would go so far as to say that in early cholecystitis it is at present the only diagnostic method on which we can positively rely. It is far too little used in Britain, but is a routine method in America and elsewhere.

I must refer to the form of treatment now commonly known as "non-surgical

drainage of the biliary tract." This treatment began with the use of the duodenal tube, repeated emptying or rather partial emptying of the gall-bladder being induced by the direct injection through the tube into the duodenum of either a concentrated solution of magnesium sulphate or olive oil. Later it was found in practice that the passage of the duodenal tube was unnecessary, the same drainage of the biliary tract being obtained by giving the patient a small dose of concentrated magnesium sulphate solution on an empty stomach. I am convinced of the great value of this form of treatment when persisted in daily for months.

As regards diet in the treatment of early cholecystitis and the prevention of gall-stones, I am far less strict (except when obesity forms part of the problem) than are many physicians. I do not see the need for restriction in cholesterol-containing foods, since the body can obtain a supply from so many foods. The only essential restriction is cooked fat, and especially "fried foods," which aggravate the common flatulent dyspepsia. I do not exclude butter or milk in moderate amount, and olive oil (a food) has great value in preventing flatulent dyspepsia and in emptying the biliary tract, though it does *not* dissolve gall-stones.

. . . . . .

JOHN RITCHIE, M.B., M.R.C.P.Ed., D.P.H.
*Medical Officer of Health, Dumfriesshire*

## ENTERIC FEVER

If enteric in its endemic form is no longer a constant menace, it is still capable of manifesting itself on occasion in formidable epidemics. The greatest water-borne outbreak on record—that which occurred at Barcelona and caused 1,847 deaths—was no farther back than 1914. The outbreaks at Pforzheim in 1919, at Hanover and Rostoff-on-Don in 1926, and at Lyons in 1929, were all epidemics of the first magnitude, affecting thousands of people, while so recently as 1927 Montreal experienced a milk-borne epidemic, wherein 5,002 persons were infected and a tenth of them died. We have fortunately escaped tragedies of such dimensions, but sufficient outbreaks have occurred in recent years to remind us that enteric is still to be reckoned with as a serious problem in public health.

The diminution of mortality, so far as Great Britain is concerned, came in three stages. From the early seventies it fell fairly steadily till 1885. From that date till about 1900 there was little improvement. At the beginning of the century the fall recommenced, and has continued till the present day.

Further advance had to wait on the more accurate knowledge of pathology which accumulated during the last years of the century. The bacterial origin of the disease was recognized by the profession, the introduction of Widal's test put diagnosis on a sound basis, and the conviction that the enteric patient was the real source of enteric infection gained ground. Finally, the discovery of the potentialities of the carrier provided a credible explanation of the apparent *de novo* origin of epidemics.

Meantime, sanitary administrative machinery was being improved. More attention was given to the protection of food against gross contamination, and, in particular, the practice of removing enteric patients to hospital was increasing. This, I suggest, was one of the most important causes of the disappearance of endemic enteric, especially in rural areas, where "sanitary" improvement was often non-existent. I know many parishes where the disease, common during the 'nineties, has been practically unknown throughout the present century. There are still no sewers, the water supplies are still obviously open to pollution, flies still pass unchecked from privy to larder, but the enteric patient is no longer allowed to remain in a country cottage throughout the course of his illness, a constant danger to his immediate associates, a potential menace to the whole district.

Recently the carrier problem has been exhaustively studied, much information on preventive inoculation has been collected, and important advances in bacteriological technique have made it possible in many cases to obtain direct evidence on what in former days could only be inferred. But nothing has happened to modify the orthodox belief that the prevention of epidemic enteric is essentially a matter of protecting drinking-water and food, especially milk, from specific infection.

Now, it is obvious that if the incidence of an infectious disease falls, not from natural causes, but as a result of human effort, that effort must be maintained if recrudescence is to be avoided, and we must consider from time to time whether accumulation of knowledge justifies, or altered conditions demand, any change of our methods.

An indication that defensive arrangements are not yet perfect is the fact that water-borne and milk-borne epidemics still occur from time to time. These differ in their genesis. Recent investigations have shown that the life of B. typhosus in sewage may be considerably longer than was generally supposed, but the actual occurrence of water-borne epidemics seems to depend on recent and gross contamination, which disappears fairly rapidly—from an epidemiological if not from a bacteriological point of view—unless pollution be continuous. In any case, whether a sewage-polluted stream, a clean source accidentally fouled by excreta, or a well or leaking main contaminated by a faulty sewer be in question, infection is generally massive, and comprises a vast bacterial flora in addition to B. typhosus. This appears to be the explanation of the occurrence of the widespread outbreaks of gastritis and enteritis which frequently precede typhoid epidemics.

Infection by shellfish may perhaps be considered as a special form of water-borne disease resulting from gross and continuous pollution. It is interesting to note that in many instances shellfish infection has been associated with preliminary enteritis resembling that occurring before ordinary water-borne epidemics. The enteric-infected oyster still constitutes a serious problem in some places. That its importance for us has diminished is due in no small degree to research on the subject in Belfast.

Infection of milk is another affair. Though dairy hygiene still falls short of perfection, *gross* excremental pollution from human sources is unlikely. The amount

of pathogenic material, whether derived from infected water or from a carrier's hands, is presumably small, but it is introduced into what may be an excellent medium for the growth of B. typhosus.

As water and milk become infected in different ways, they must be protected by different methods. Safeguarding of water is largely a question of finance, and there should be little need for discussion so far as large towns are concerned. Sources protected against pollution, adequate storage, efficient filtration, and, when necessary, chemical treatment, constitute a succession of lines of defence sufficient to secure immunity. If one of those desiderata be lacking, special emphasis must be laid on the others. Large communities, moreover, can provide expert supervision and sufficient staff to maintain efficiency. The experience of London in particular shows that by taking proper precautions the risk of water-borne infection may be reduced to negligible proportions.

The position of small towns, villages, and rural communities is different. Their water supplies are still frequently open to serious criticism, but the confidence resulting from years of immunity, and the relatively heavy financial burden entailed by improvements, discourage sanitary reform.

An adequately protected water supply is of importance in the prevention of milk-borne epidemics. It is of course possible that an outbreak originally due to polluted water may be continued by milk through some one connected with the dairy trade having been a victim of the water-borne infection. This was well exemplified at Santa Ana (Ca.) in 1924, when an epidemic due to sewage-polluted water, and affecting 370 persons, was followed by a milk-borne outbreak of 200 cases in a neighbouring town, which had a separate water supply but derived much of its milk from a dairy at Santa Ana.

Again, milk may be infected through polluted water being used to wash utensils. Theoretically this should never happen, but until all dairies are fully equipped in accordance with modern ideas this risk cannot be ignored. There are cases in which milk has been infected during the cooling process, either by a leaking cooler allowing water to gain access to the milk, or, in the absence of a sufficient water supply, by the milk-cans having been set to cool in a stream which proved to be sewage-polluted. This appears to have caused the St. Catherine (Ontario) epidemic of paratyphoid fever in 1931. In this case 475 cases were traced to milk from two farms where milk-cans were cooled in a small creek that was contaminated by drainage from a district in which paratyphoid fever had been prevalent in the previous year.

The main cause of milk-borne outbreaks is, of course, infection by a carrier, missed case, or contact who handles milk. Unfortunately, his existence is generally unsuspected until the outbreak occurs, and even then it may be difficult to confirm. Proof of the carrier state entails detection of the causative bacillus in urine or fæces, and the intermittency of bacillary excretion by carriers may prevent this proof being obtained. Moreover, as I have learned by experience, samples submitted by a suspected carrier may have been borrowed for the occasion ! Even after the carrier has been recognized, the period during which he can be certified and

put under restrictions is too short, and recertification may present difficulties. It should be recognized that after a year the carrier condition is permanent, and recertification should not be required.

. . . . . .

C. H. ANDREWS, M.D., F.R.C.P.

## INFLUENZA: FOUR YEARS' PROGRESS

In the absence of any exact method of clinical or pathological diagnosis, the only attempts to define the disease have been on an epidemiological basis; and this is not very satisfactory for ordinary mortals, whatever it may be to epidemiologists. I propose to-day to try to convince you that it has recently become possible to separate off from the influenza scrap-heap a definite disease, one, moreover, which is probably responsible for the more important outbreaks labelled "influenza." The basis of separation is in the first place etiological: one can recover from garglings in the early stages a virus which will infect ferrets and mice.

To recover virus from a patient, unfiltered garglings are dropped from a pipette on to a ferret's nose. When virus is present in the inoculated material, the ferret shows fever and nasal symptoms after about forty-eight hours. It goes off its food, lies about apathetically, and develops nasal discharge, sneezing, and, later, pronounced nasal obstruction. The temperature chart often shows two spikes forty-eight hours apart. Influenza in the ferret is infectious by contact, and to guard against cross-infections we isolate each animal in a separate cubicle.

When we appear to have infected a ferret with material from a patient, we usually kill the animal at the height of its disease on the third or fourth day. We then find that the mucous membrane over its turbinate bones is engorged and that the nasal cavities are full of muco-pus. We remove the turbinates and grind them up with sand; an emulsion is obtained which can be filtered and studied in other ways to prove that it really is influenza virus which has produced the symptoms.

In mice the virus produces lesions of the lungs only; the nasal passages are not affected. Perhaps in relation to this fact influenza in mice is entirely non-contagious; normal mice kept in the same cage as infected ones neither contract the disease nor become immune. This makes work with mice much easier, but, on the other hand, we lose the opportunity of what would be a really exciting study in experimental epidemiology. When first adapted to mice, influenza is a relatively non-fatal disease; but after many passages it becomes more lethal, and infections can be produced with one ten-millionth of a cubic centimetre of filtrate. Filtrates are readily neutralized by immune sera, and a neutralization test that has yielded much interesting knowledge is thus made available.

I will now summarize the evidence, which convinces us that the virus we are studying is the primary cause of epidemics of influenza in man.

1. First, we have recovered the virus from garglings of fifty-three patients with the symptoms of influenza, obtained at times of rather widespread prevalence of the disease. During the recent epidemic, virus was isolated from thirty-one out of

forty-one uncomplicated cases tested. Virus has only been obtained during the first few days of infection, and not during convalescence. We have failed to recover the virus from normal people, and we have usually failed also with garglings from sporadic cases, or patients in localized outbreaks diagnosed as influenza but occurring in the absence of a widespread epidemic.

2. The virus has been recovered as described above from influenza garglings taken during outbreaks not only in London (Smith, Andrewes, and Laidlaw, 1933), but from all over the world—in the West Indies, United States, and Alaska (Francis, 1934), in Australia (Burnet, 1935), in Russia (Smorodintseff, *et al.*, 1936a), in Holland, and, during the recent outbreak, in Manchester, France, Germany, Hungary, and again in the United States.

3. The serum of patients taken during the acute stages of infection is found to have little or no power to neutralize the influenza virus. But as early as the eighth day of the disease it will have acquired very definite neutralizing powers. No such neutralizing powers appear in the sera of patients suffering from respiratory diseases other than influenza. We now have some evidence that sera from the population at large, including people who have not recently suffered from influenza, contain better neutralizing antibodies just after an epidemic than just before one.

4. In 1933 we made two attempts to infect volunteers with virus which had been passed serially through ferrets; these were unsuccessful, but at the time we failed to obtain any volunteers who had not good antibodies in their sera, and such antibodies may have protected the two victims of the experiment. Last year an accident completed our chain of evidence for us. A ferret infected with a virus which had been passed in series through 196 ferrets, sneezed upon Dr. Stuart-Harris and produced in him a typical attack of the disease. There was no influenza prevalent at the time, and the virus recovered was distinguishable by its biological effects from ordinary strains of human origin. There is therefore almost no room for doubt that Stuart-Harris's infection actually came from the ferret (Smith and Stuart-Harris, 1936). Smorodintseff and his co-workers (1936a) have recently recorded their success in infecting five human volunteers with the virus.

5. The irregularity with which Pfeiffer's bacillus has been recovered from influenza epidemics is notorious, and cannot be explained away by blaming differences in technique. Smorodintseff *et al.* (1936b) have lately carried out extensive attempts to infect volunteers with cultures of Pfeiffer's bacilli, but produced nothing comparable with influenza. Experimental "swine 'flu" has been demonstrated by Shope (1931) to be due to the combined action of a virus and a bacillus related to Pfeiffer's bacillus. On the other hand, experimental influenza in ferrets and mice is a pure virus disease in which bacteria ordinarily play no part. The evidence at present suggests that ordinary influenza in man resembles the disease in ferrets and mice in being purely a virus infection; further, it seems likely by analogy that the virus alone may cause pulmonary complications; but it is all too familiar to us that streptococci, pneumococci, staphylococci, and Pfeiffer's bacilli may on occasion play a very serious part as secondary invaders of the lung.

Ferrets who have recovered from an attack of influenza develop potent neutralizing antibodies in their sera, and are completely resistant to reinfection for about three months. But after six months their immunity has definitely waned; antibodies, though still recognizable in their sera, are less than before, and the animals will develop some fever and nasal symptoms when a heavy dose of virus is given intranasally.

As already mentioned, influenza virus given subcutaneously, or by routes other than into the respiratory tract, does not infect—or only very exceptionally. It will, however, produce some immunity, especially when several doses are given. The immunity produced by vaccination is shown by :—(i) the production of antibodies; (ii) the milder nature of the fever and symptoms; (iii) complete protection against the development of lung lesions; (iv) protection against the less severe test of exposure to contact with an infected ferret; (v) restoration of waned immunity to the complete state. By this is meant that ferrets whose immunity has fallen off six months after an infection are rendered once more completely immune by one dose of subcutaneous vaccine (Smith, Andrewes, and Laidlaw, 1935).

In mice, influenza is a disease of the lungs; and just as vaccination is very effective in protecting ferrets against lung lesions, so it is in mice. Two subcutaneous or intraperitoneal injections give a substantial immunity; in many experiments all the mice of a vaccinated group have survived a dose of virus which has killed all, or almost all, controls. Further, when the vaccinated mice have been killed at the close of the experiment, the lungs of most have appeared normal. After two doses of vaccine, the immunity of mice appears to last for ten, but not for sixteen, weeks; this is as long as the immunity of mice which have actually recovered from an infection. Protection experiments in mice have lent themselves to the quantitative study of a number of problems, particularly those designed to find a suitable vaccine for use in man.

Though we have not yet succeeded in eliminating the mouse protein from the vaccine available without spoiling it, we have made preliminary attempts at vaccinating human beings. Francis and Magill (1937) have made similar attempts, using living virus grown, as many viruses can be grown, on tissue cultures of chick embryo. They have succeeded with such a vaccine in inducing a rise in antibodies in the vaccinated subjects. Stokes *et al.* (1937) have also used a living vaccine made from mouse tissues, and claim that there was a lower incidence of influenza among the vaccinated members of the community they studied than in the control group; there is, however, no certainty that the respiratory disease which afflicted that community really was epidemic influenza. We have chosen to err on the side of caution by using only formalin-inactivated virus, and we too have followed the neutralizing antibodies in the people we have vaccinated. One dose of 2 c.c. of vaccine subcutaneously has produced only a trifling local tenderness, and has engendered a very satisfactory rise in antibodies. Among thirty volunteers, all soldiers at Woolwich, we obtained an average increase in antibodies of twenty-five-fold when we compared bleedings taken a fortnight after vaccination with those obtained beforehand. A few results of ours confirm those of Francis and Magill,

that not much rise occurs until after a week from the time of injection. A satisfactory feature was that one dose of vaccine produced as good an antibody rise as did two spaced a fortnight apart.

. . . . . .

L. J. WITTS, M.D., F.R.C.P.
*Professor of Medicine, University of London*
*Director of the Medical Unit, St. Bartholomew's Hospital*

## THE HAEMORRHAGIC STATES

I AM increasingly impressed by the frequency and diversity of hereditary hæmorrhagic diseases and by their resistance to treatment. The study of hereditary diseases in man has been vitiated by premature dogmatism and by the expectation of a constancy in the mode of inheritance which the student of genetics in plants and animals would from the first have thought improbable. Hereditary hæmorrhagic states in man are not subject to immutable laws in pattern and pedigree, and it is wiser to think in terms of affected family groups than of specific diseases and laws of heredity. The hereditary hæmorrhagic diseases provide some of the most pathetic cases we see, and it is natural that we should strive to do everything possible to relieve them. Nevertheless, their suffering is not lightened by the publication of methods of treatment which time proves to have no curative value. The local application of coagulant snake venoms is the only new treatment whose value has been confirmed. The venom of Russell's viper (1 in 10,000 solution), or of the Australian tiger-snake (1 in 5,000 solution), has been chiefly used (Macfarlane and Barnett, 1934; Rosenfeld and Lenke, 1935). Loose clot is washed away from the bleeding-point, and tampons or other appropriate dressings soaked in the venom solution are then applied. With the use of snake venom in hæmophilia, it is now possible to repair wounds or to carry out small operations such as dental extraction which would previously have been fatal.

Other new treatments have already passed into the limbo of discarded things, or show signs of passing there, as they have not lived up to the claims made for them. Any curative action of liver or œstrin in hæmorrhagic states has by now been abundantly disproved.

Injections of moccasin snake venom have been recommended for all forms of chronic and recurrent bleeding, especially those of a purpuric type (Peck *et al.*, 1935, 1936). It is difficult to test a remedy which admittedly makes no difference to the platelets, the coagulation time, or the bleeding time. In three cases of hereditary thrombasthenia I have had no success with treatment with moccasin snake venom, and Davidson (1937) records a similar lack of success with this treatment in idiopathic thrombocytopenic purpura. Moccasin snake venom is reported to be more successful in hereditary telangiectasia, and in one case of this disease where I advised its employment, there was a temporary remission of symptoms, but it was soon followed by relapse in spite of continuing treatment. Eugenists may derive a melancholy satisfaction from this catalogue of failures in the treatment of the

hereditary hæmorrhagic states, and there is no doubt that prevention will always be better than cure.

Idiopathic thrombocytopenic purpura stands out more clearly now that we have been able to detach from it the hereditary purpuras, the allergic thrombocytopenic purpuras due to sensitivity to food and drugs, and the symptomatic thrombocytopenic purpuras due to destruction of the foci of platelet production in the marrow by organic and inorganic poisons. There are no gross changes in the spleen in idiopathic thrombocytopenic purpura, apart from simple hyperplasia, and the only abnormalities in the marrow are some increase in number and decrease in maturity of the megakaryocytes. We still do not know whether the disease is due to a diminished production or an increased destruction of the platelets. The dramatic results of splenectomy are somewhat discounted by the discovery that within an interval of months or years about half the patients relapse, though as a rule symptoms are much less troublesome than before the operation (Pemberton, 1934; Williamson, 1934). This discovery suggests that splenectomy is not a cure for idiopathic thrombocytopenic purpura in the same sense as it is a cure for acholuric jaundice. Remissions of thrombocytopenic purpura after splenectomy, like remissions of pernicious anæmia following this operation, are apparently due to "the lifting of a mysterious restraint which the spleen exercises upon the delivery of immature blood cells by the marrow" (Cornell, 1927), and do not signify any fundamental change in the underlying disease process.

The mortality of splenectomy in acute attacks of idiopathic thrombocytopenic purpura has been greatly reduced by the practice of repeated or massive transfusions, but in view of the frequency of spontaneous recovery, the difficulty of diagnosis from infectious and malignant purpuras, and the inability to promise complete security from relapse, I should like to ask how long we should persevere with medical treatment before asking for the surgeon's help. My present opinion is that medical treatment should be tried for seven to ten days. In chronic cases the operative mortality is small, but I should advise restricting splenectomy to patients in whom the disease is really disabling or is subject to acute exacerbations.

The outstanding event of the last decade in the hæmorrhagic diseases has been the isolation and synthesis of vitamin C or ascorbic acid. It can now so easily be administered in large doses by mouth or by injection, that it has been tried in many diseases, and there has been a spate of papers, comparable with those which followed the introduction of insulin and liver extract, suggesting that ascorbic acid is a panacea for all kinds of hæmorrhagic states, and extending its range of usefulness even to dropsy and leukæmia. It is fortunate that at the same time methods have been devised for the estimation of vitamin C in the body fluids, and the test of the resistance of the capillaries has been put on a quantitative basis. It has been shown that while overt scurvy is rare in civilized countries, subclinical scurvy is not uncommon, especially in patients who are febrile or who take special diets. Vitamin C cures scurvy rapidly and dramatically, but it has no action on hæmophilia, thrombocytopenic purpura, Henoch-Schönlein purpura, leukæmia, or other non-scorbutic hæmorrhagic states.

Even more important than the isolation of ascorbic acid is the impact of biochemistry on the hæmorrhagic states, the ability to reproduce them in animals, and the exchange of fertilizing ideas between ward and laboratory. The bearing of nutrition on the hæmorrhagic states is not limited to scurvy and vitamin C. Szent-Györgyi (1936) and his colleagues were at first disappointed to find the action of pure ascorbic acid limited to scurvy, as they had successfully treated cases of non-thrombocytopenic purpura with impure preparations of vitamin C. They have now isolated from these sources a substance, flavon, which they have called vitamin P, as they believe it influences the permeability of the vessels. Nothing is more difficult than to establish criteria for capillary permeability and its variation in hæmorrhagic states, nor is it easy to prepare large quantities of flavon. For these reasons little further progress has been made on these lines.

Fanconi (1928) in his work on intestinal infantilism noted not only scurvy, but a condition resembling hæmophilia, due to a defect in the coagulating power of the plasma. Other pædiatricians have observed the same phenomenon in under-nourished children, and have been able to relieve it by a diet rich in protein (Kugelmass, 1932). Prothrombin is present in abnormally small amounts in the blood of the newborn infant, and it is probable that the hæmorrhagic disease of the newly born, melæna neonatorum, is due to depression of prothrombin below a certain critical level (Brinkhous *et al.*, 1937). A disorder characterized by a lack of prothrombin in the plasma has been produced in birds by a defective diet (Dam *et al.*, 1937). Hæmorrhagic chick disease is due to deprivation of a fat-soluble vitamin, the Koagulations-Vitamin or vitamin K, which is present in mammalian liver, egg-yolk, and certain vegetable substances such as alfalfa. It is doubtful whether hæmorrhagic chick disease can be exactly duplicated in mammals. A very similar disorder, the sweet clover disease of the rabbit, is likewise due to lowering of the prothrombin in the plasma, but though it is a dietetic disease, it does not appear to be identical with hæmorrhagic chick disease, and while it can be cured by extracts of alfalfa, the curative fraction is said to have properties different from those of vitamin K (Quick, 1937). At present it is not clear what bearing these discoveries will have on human therapeutics. Liver and vitamin K have no action on hereditary hæmophilia, but there remains a wide field for study in the treatment of melæna neonatorum and the hæmorrhagic manifestations of malnutrition and disease of the liver.

. . . . . .

ROBERT HUTCHISON, M.D., LL.D., F.R.C.P.
*Consulting Physician to the Hospital for Sick Children*
*Great Ormond Street, London*

## ENURESIS

FOR the purposes of this discussion it will be well to define enuresis as "bed-wetting," although it is true that diurnal incontinence is often present as well. On certain points we shall probably all be agreed: that enuresis should not be

regarded as abnormal until the child has reached the age of 3; that it is commoner in boys than in girls; that most cases date from birth, although occasionally bladder control is lost after it has been once acquired; that it is worse in cold weather.

As to the mechanism of the disorder there has been much speculation, but, broadly speaking, it may be said that the underlying defect is some failure in the neuro-muscular mechanism of the bladder, so that the reflex micturition of infancy persists. Further than this very general statement I do not think one can safely go. It has been suggested that the failure is brought about by the cessation during sleep of the control exercised by the higher centres over the lower; but that surely is the case in every child, whether an enuretic or not.

Various contributory conditions or exciting causes have been put forward, some of which are of doubtful validity. I do not believe, for instance, that high acidity of the urine is a cause, and I am very doubtful about phimosis. Threadworms have been specially blamed by some, and their importance as a factor is worth discussing. On the other hand, I am quite sure that adenoids and enlarged tonsils, especially if causing obstruction, may sometimes lead to enuresis—explain it as we may.

There is one interesting group of cases, however, which deserves special mention, a group small in proportion to the total number of enuretics, but perhaps larger than we think—namely, those in which the enuresis seems to depend upon a nocturnal polyuria, or, to put it in another way, upon a reversal of the normal relative amounts of day and night secretion. Such cases are not generally recognized, but they certainly exist. They may be detected either by actual measurement of the day and night urine, or by finding that the specific gravity of the day secretion is unduly high, that of the night unwontedly low. To explain these cases is difficult. One is tempted to suppose that there must be a delay in excretion or some vasomotor factor at work, but these are matters we might well discuss.

The question of treatment must chiefly interest us, and we must all, unfortunately, be convinced that there is no one plan which is sure of success; the very number of the remedies proposed is proof of this. All admit the importance of a firm mattress, adequate bed coverings, and abstinence from the ingestion of much fluid in the later hours of the day. "Accidents" may be rendered less disagreeable by the wearing of a thick pad of sphagnum moss, and I have sometimes, in the case of adolescents, recommended the use of a rubber urinal such as is worn by patients suffering from incontinence the result of spinal paralysis.

The "taking up" of the child when the mother goes to bed is really only a way (I would suggest) of dodging unpleasantness. It cannot be really curative, for the normal child does not need to be so "taken up." None the less, it is a proper part of treatment.

Remote causes should be dealt with. By all means get rid of threadworms, although I cannot agree with some enthusiasts that they are the main cause of enuresis. I doubt if phimosis is worth dealing with, except on its own merits (or

demerits), but I am convinced that I have seen cures result from the removal of tonsils and adenoids.

Among drugs, belladonna has remained the most popular, but its mode of action is ill understood. I usually give a dose about five o'clock in the afternoon and another at bedtime, gradually increasing the quantity up to a point just short of toxicity. It should be continued for some weeks, and then gradually left off. Ergot also has a considerable reputation, although it is less used than formerly. It is difficult to see pharmacologically how it can do good, but I have had at least one case of the nocturnal polyuria type in which it seemed quite definitely to control the enuresis. Ephedrine has lately been recommended in a half-grain dose at bedtime, increased by another half-grain every week. It is said to act by increasing the tone of the sphincter whilst relaxing that of the bladder musculature. It will be interesting to learn the experience of anyone here who has tried it extensively. The endocrines, I think, will not help us much. A good deal has been claimed for thyroid in the case of the phlegmatic child with enuresis, but I consider the claims exaggerated. Pituitrin, from its antidiuretic action, ought theoretically to be useful, especially in the nocturnal polyuria cases, but it is an inconvenient substance to administer, and I have had no experience of it. I gather that those who have tried it have been disappointed with the results.

The more heroic and semi-surgical procedures such as epidural injections, the passage of sounds, and so forth, will, I imagine, be condemned by most of us, so we are left only with psychotherapeutic methods. As to the value of these, there is a considerable difference of opinion, although all are at one about the importance of creating the right psychological atmosphere for treatment by seeing that the disability is regarded in a common-sense and unemotional way, and that the patient is not allowed to be either frightened or fussed. But can we do more than this? Suggestion has been used in an endeavour to teach the child "to control the bladder during sleep." But can any child—or adult for that matter—be expected to exercise such control? Hypnotism I have never practised myself, and I have not been impressed by the results it has yielded in the hands of others. There is a form of suggestion for which success has been claimed in America, and which consists in the weekly subcutaneous injection of 1 c.c. of sterile salt solution. It is impressed upon the child at each injection that the treatment will certainly abolish the enuresis, but that if this should persist further injections will be required. For my own part, I regard such treatment as a disguised form of punishment hardly to be distinguished from the use of the nurse's palm, and, besides, we should not lie to the young.

. . . . . .

GEOFFREY MARSHALL, M.D., F.R.C.P.
*Physician, Guy's Hospital; Assistant Physician, Brompton Hospital*

## EARLY DIAGNOSIS OF PULMONARY TUBERCULOSIS

MOST of the symptoms in the earlier stages of the disease are those of toxæmia, and many of them are not specific. They are fatigue, loss of morning appetite, loss of

weight, and psychological disturbances which are often described as neurasthenia or as a nervous breakdown. In young women another common toxæmic symptom is amenorrhœa. This symptom in a young girl may arise from a variety of causes; two causes which must always be considered are tuberculosis and pregnancy. The more specific symptoms are cough, expectoration, night sweats, and hæmoptysis. Any one of these symptoms in an adolescent or a young adult should arouse suspicion, and several of them in combination would go far towards clinching the diagnosis. There is one symptom which in the absence of all other evidence is in itself diagnostic, and that is a free hæmoptysis "out of the blue." By this is meant a copious hæmorrhage, a cupful or more of bright liquid blood welling up into the mouth in a young subject who has no other symptom or sign of disease.

In such a case, announce the diagnosis and order your patient to bed for two months on the spot. Do not wait to investigate, for such hæmorrhages often occur at such an early stage that all investigations prove negative. In the meantime you may lose control of the treatment, and the patient may not come back to you until months later, when the disease has become firmly established and perhaps incurable.

We are sometimes enabled to diagnose the disease at an early stage because of the occurrence of complications that are apt to develop before the focus in the lungs has attained a size which renders it liable to detection. The more common of these complications is a sterile pleural effusion, and it is not necessary for me to insist that such an effusion, in the absence of evidence of malignant disease, is diagnostic. The less common and less universally recognized of these early complications is perianal fistula. This lesion is so frequently followed by manifest tuberculosis of the lungs, that I believe its diagnostic significance is little less than that of the pleural effusion.

Many cases are submitted to us in which the diagnosis cannot be established by the history, symptoms, signs, or complications; but we dare not in any case give a negative diagnosis without investigation. What are the methods of investigation we should employ?

First in importance comes examination of the sputum, if there be any. If there be none, then the patient should be given potassium iodide or ammonium carbonate in ten-grain doses until expectoration is provoked. The sample so obtained may be poor, thin stuff, devoid of pus; and the pathologist may regard it with contempt and doubt whether it be worth the trouble of staining. Give him courteous encouragement, for tubercle bacilli have been found in many such unpromising specimens. One negative result is of no significance, but indicates the need for two further samples to be submitted. In many cases three negative results must be regarded as inconclusive, and then recourse is had to a more searching test than that of microscopical inspection of a stained smear. Until recently the test favoured has been injection into a guinea-pig, followed six weeks later by killing and necropsy of the animal for evidence of tuberculous inflammation of the lymph-glands draining the site of injection. Objections to this method are the long period involved before the test can be completed, and the proneness of captive guinea-pigs to sudden and

premature demise, which may remove them from the scene before the test is complete, but when several weeks have been wasted in fruitless waiting. The guinea-pig is therefore being supplanted by cultivation of the sputum for tubercle bacilli on special media such as that of Loewenstein. If bacilli are present, colonies usually develop within fourteen days; and the proportion of positive results is little if at all inferior to that obtained by animal inoculation.

After examination of the sputum for tubercle bacilli, the investigation next in importance is the taking of an X-ray photograph of the chest. The improved definition obtained in recent years by exposures of a fraction of a second has greatly increased the diagnostic value of radiography of the lungs.

The third form of investigation which we commonly employ is a diurnal record of the temperature. Satisfactory readings can be obtained with a calibrated "half-minute" thermometer kept in the closed mouth for three minutes. In a case of early tuberculosis, when the patient is up and about, we shall generally find an abnormal range, the morning readings being unusually low and the evening readings above 98.4° F. It must be remembered that in young women the temperature commonly rises to 99° or over during the week preceding the menstrual flow, even in perfect health.

These three methods of investigation—examination of the sputum, radiography of the chest, and recording of the temperature — are a necessary part of our technique in diagnosing pulmonary tuberculosis in its early stages; but there are other investigations which some of you will have in mind. First there are the various tuberculin tests—cutaneous, subcutaneous, or conjunctival. These tests are still held in great respect by our veterinary colleagues and by those of our legislators who supervise the production of the milk supply. The result of a tuberculin test decides the question of life or death in the herd of an attested dairy farm. It is fortunate that the human species is not subject to the same dubious regulation; for it would involve the slaughter of some eighty per cent. of the adult population of these islands. There is overwhelming evidence that a positive tuberculin test indicates sensitivity to tuberculin, and that it signifies that the individual has probably at some previous period been infected by and acquired some immunity to tubercle bacilli; but it is no evidence at all that the individual is suffering from tuberculous disease, except in the case of infants.

. . . . . .

JAMES CROCKETT, M.D., D.P.H., F.R.C.P.Ed.
*Glasgow*

## ARTIFICIAL PNEUMOTHORAX

COLLAPSE therapy "won its spurs" in the treatment of advanced pulmonary tuberculosis. Forlanini, in working out the technique and establishing the reputation of artificial pneumothorax, limited himself to advanced cases of the disease with bilateral lesions. For many years it was looked upon as a last resort—this was due

to the fact that in the past it was not without its dangers. It being a principle in the profession that no remedy must be more dangerous than the disease which it is intended to cure, the early, even the acute, case was not considered to be bad enough to subject it to the risk involved. Nowadays, with the technique perfected and the treatment practically safe, should artificial pneumothorax not be looked upon as the method to adopt whenever an early or acute case of tuberculosis presents itself? What may happen to such a case? One of three things :—

1. Great improvement.
2. The lung disease may remain more or less stationary.
3. The lung disease may become worse.

Lawrason Brown years ago predicted that "artificial pneumothorax would be used in all early cases in the future." He is a sound and experienced phthisiologist who would not make such a statement without considering it carefully. It is also a conclusion to which many reliable workers have come. The early stage is certainly the ideal one for effective therapeutic collapse. Adhesions are few, collapse is easily effected, and the sputum is rapidly rendered negative. The bad results are met with when pneumothorax treatment is resorted to in advanced and toxic cases, although even then it may produce marvellous results. It is my considered opinion that whenever a patient presents himself with a unilateral lesion of the exudative or the bronchopneumonic type, the induction of an artificial pneumothorax should be attempted without delay. It is the best, the safest, and the shortest method of securing arrest. The "wait and see" policy is disastrous.

In 1928 I was asked to see an Italian with lung disease, and diagnosed pulmonary tuberculosis. The lesion was largely unilateral, and I advised him to go into a sanatorium with a view to having an artificial pneumothorax induced. He refused to do so. As he did not improve after a few months, he returned to his native land. There he continued to go downhill. Nearly a year after the diagnosis was made he went to a hospital in Naples to consult Dr. Castillani. He was X-rayed, and was told there was but one cure—artificial pneumothorax. The following day Dr. Castillani attempted the induction in the out-patient department, and was successful, although it caused a good deal of pain. The patient was kept resting for two hours, and was then allowed to walk back to his hotel. Refills were given on the second, fourth, and seventh days, and repeated thereafter weekly for two months. At the end of that period he felt fairly well, so he returned to this country to resume work. He was referred to me for treatment, and I gave him fortnightly refills at my rooms. This patient has continued to do well, and has built up a prosperous business.

The facts of this case made me think. I could quite well have given him the same treatment at my rooms or at the dispensary. I determined to follow the procedure in other cases. It had much to commend it, especially in view of the usual difficulty in obtaining early admission to a sanatorium.

Following upon this, during these past nine years I have carried out ambulatory pneumothorax on a large number of patients with most encouraging results. In fact, when I now meet with a new case of tuberculosis, provided (*a*) that the lesion

is unilateral, (*b*) that the general condition is not undermined by toxins, (*c*) that loyal co-operation can be secured, (*d*) that the patient's home conditions are satisfactory, I make it a rule to advise domiciliary measures and ambulatory artificial pneumothorax. It has undoubtedly proved a most valuable procedure. So far, having carefully selected the cases, they have all done well.

There is not the slightest doubt that ambulatory artificial pneumothorax treatment could be more extensively carried out than it is at present. If it were practised, in the first place, many hospital and sanatoria beds would be freed for acute cases or patients whose home circumstances demand institutional care; secondly, the stigma of being an ex-sanatorium patient, and the handicap it involves, would be nullified; thirdly, the cost to the State for treating tuberculosis would be lessened, although the facilities for out-patient treatment might require to be increased; and, finally, the morale of so many patients would be conserved, and not undermined, as so often happens as the result of prolonged hospitalization. Ambulatory artificial pneumothorax is not a heroic measure, provided it is not used in acute, advanced, or toxic cases.

It has always been recognized that cases of pulmonary tuberculosis with bilateral involvement have a definitely bad prognosis. What can artificial pneumothorax do for such? The fact that bilateral pneumothorax is compatible with life has been demonstrated by the bilateral spontaneous cases which have been reported by various workers.

The honour of introducing the treatment for this purpose must be given to Parry Morgan of Cardiff and Ascoli of Palermo. Parry Morgan administered it to an apparently hopeless case in 1912. Four years later the patient was doing well. Ascoli in the same year, at a conference in Rome, asserted that the high pressures advocated by Forlanini in pneumothorax treatment were not necessary. A low pressure sufficient to cause simple relaxation of the lung often did more good. Further, he asserted that if one used low pressures, artificial pneumothorax could be given to both sides simultaneously, and instanced a very serious case with bilateral mischief which he successfully treated in this way.

In simultaneous artificial pneumothorax the treatment is given on both sides simultaneously or alternately. Although a quarter of a century has elapsed since this method was first advocated, simultaneous pneumothorax has not been widely adopted. The reason for this is not far to seek. By most workers it has been looked upon as a heroic measure, and only resorted to when a patient was very ill. Consequently the end-results as published in various quarters are as a rule not encouraging: usually not more than ten per cent. respond favourably to the treatment, although a few American workers have reported good results in over fifty per cent. of their cases. In my own experience, simultaneous artificial pneumothorax treatment, judged by the end-results over a period of years, was in the first series of cases a record of failure. I carried it out on several cases each year—round about 1920—without the ultimate survival of any: the longest duration of life after treatment was begun was two years. I recognized that I made a mistake in using it only for the serious advanced type of case. In later years I resorted to it

with greater confidence in less advanced and less toxic cases, with more encouraging results. Approximately one-third were definitely helped, and had the sputum rendered negative. One-third improved symptomatically, and one-third went downhill in spite of the treatment. About half the cases were treated for a flare-up in the contra-lateral lung, the other half were cases with bilateral mischief from the time of diagnosis. Later experience, therefore, demonstrated that fairly good results could be expected if the collapse were effected before widespread adhesions formed, before the production of thick-walled cavities, and before the toxæmia resulted in cardiovascular debility.

In carrying out the treatment, the principle of low-pressure technique must be observed, and the patient be left always with a definite negative pressure. If, therefore, it be determined on during unilateral pneumothorax treatment for contralateral activity, the pressure on the side already treated must be brought well below the zero level to enable any healthy lung tissue present to function. In cases which have not previously had pneumothorax treatment, it must be instituted with great care and consideration. One lung—the one more affected—should be dealt with first, and the patient should be able to go for a week without refills before the other lung is treated. Of necessity the refills have to be small, and therefore have to be given fairly frequently. The two pleural cavities may receive refills simultaneously, but that is rarely advisable, at least until the treatment has been well established.

It is important to aim at having the pressure on both sides equal, or nearly so. If they differ much, and the mediastinum is mobile, symptoms suggestive of a spontaneous pneumothorax may be produced.

Some American workers have instituted a bilateral collapse on ambulatory patients: I have not attempted this so far—the risks appear too great. There are two things that always cause not a little anxiety while carrying out the treatment, bilateral treatment more so than in unilateral treatment. These are, first, the risk of puncturing the lung and causing a traumatic pneumothorax or an air embolism; secondly, the production of a fluid effusion which may cause excessive intrapleural pressure and dyspnœa.

. . . . . .

J. R. LEARMONTH, Ch.M., F.R.C.S.Ed.
*Regius Professor of Surgery in the University of Aberdeen*

## ELONGATION AND DILATATION OF THE COLON

"MEGACOLON can be defined as dilatation and hypertrophy of a part of the colon or of the whole colon, occurring in the absence of a gross obstructive lesion" (Adamson and Aird, 1932). It is a rare disease. [Four in 131,000 cases in a general hospital (Walton, 1930); one in 2,620 cases in a children's hospital (Hurst, 1934).]

The present view is that the enormous hypertrophy and dilatation so characteristic of the disease are the result of neuro-muscular dysfunction, and are congenital. The nature of this dysfunction is still problematical. The local changes in Auerbach's plexus described by Cameron may well, as Adamson and Aird (1932) point out, be

secondary to other pathological changes in the wall of the bowel. The occasional association of dilatation of the bladder and/or ureters with megacolon focuses attention on their common nerve supply—pelvic splanchnic sympathetic nerves and sacral parasympathetic nerves; it must be realized that over-activity of the one or under-activity of the other might produce the same result; and, indeed, Adamson and Aird have produced the condition in animals by dividing the parasympathetic nerves to the distal colon. Of neurogenic hypotheses mention may be made of spasm of the internal sphincter (Fenwick), achalasia of the sphincter ani (Hurst), and incoordination of the musculature of the colon, so that it does not relax before peristaltic wave (Hawkins).

My own view is that the colon does not behave as if a terminal obstruction were present, for in such cases surgeons are accustomed to depend on early distension of the cæcum as a constant diagnostic sign, and this is inconstant in megacolon. Adamson and Aird (1932) put the incidence of the disease as pelvic colon alone thirty-seven per cent., with dilatation of the upper part or the whole of the rectum in thirty-four per cent. of cases. In only twenty-five per cent. does the dilatation extend to the ileo-colic valve, and in the remainder the descending, transverse, and ascending colon are involved with decreasing frequency. Radiological observations on the barium-filled colon show that it is active, although its activity is without result and may even give the impression of being purposeless.

My opinion at the moment is that the whole picture points to the presence of local segmental neuro-muscular incoordination when the disease does not involve the rectum, and that when the rectum is dilated there is added achalasia of the sphincter ani internus.

*Treatment*.—It has been shown by Morton and Scott (1930) that after spinal anæsthesia the colon may be able to evacuate its contents. As this procedure paralyses parasympathetic as well as sympathetic nerves, one must conclude that the intrinsic neuro-muscular mechanism is adequate in such cases. In less severe types, preliminary enemata, training of the bowel, and the administration of liquid paraffin are usually adequate if the patient (and the parents) are intelligent and co-operative; when the rectum is involved, graduated dilatation of the internal sphincter, as suggested by Hurst, may also be usefully employed. When such simple measures are not effective, the operation of sympathectomy may be considered, with the purpose of depriving the distal colon of its inhibitory nerves. Of the many technical procedures suggested, by far the most eclectic is that devised by Telford and Stopford (1934), and I consider that it should be employed exclusively. It consists in the resection of the mesially directed branches of the lumbar ganglia from the second to the fourth inclusive, and avoids the paralysis of the vaso-constrictor nerves to the legs which follows the (equally efficacious) removal of the lumbar cords, and the sterility which follows resection of the presacral nerve in male patients.

The results of sympathectomy are distinctly good; in the series collected by Ross (1935), twenty-one out of twenty-nine patients were benefited, and many other surgeons have reported successes. My own experience is similar. The mechanism

of the improvement is obscure. Any explanation must take into account the often dramatic immediate improvement in function, which disposes of valves, kinks, or elongations as the essential cause of the failure of evacuation of the colon. Indeed (and this emphasizes the point made by Hurst), there is often little change in the appearance of the colon after a barium enema, though its function is satisfactory. I do not think one can go further than to say that the distal colon, under the unbalanced dominance of its extrinsic motor nerves, regains its physiological adequacy. It is interesting to note that when the urinary tract is dilated it shares in the improvement following sympathetic neurectomy (Adamson and Aird, personal experience).

. . . . . .

G. PERCIVAL MILLS, F.R.C.S.
*Surgeon, General Hospital, Birmingham*

## TREATMENT OF "TENNIS ELBOW"

"TENNIS elbow" occurs mainly in tennis players, but occasionally in golfers, and more rarely in metal-workers and amateur gardeners who use respectively the hammer and the trowel. There is practically never a history of injury—the pain comes on gradually during or after the favourite pastime. The most prominent symptom is pain on the outer side of the elbow when gripping, and this pain is usually much worse on gripping with the forearm pronated; indeed, the grip in the supine position is often painless. The pain may come on while grasping some trifling object like a teacup, which may even be dropped, but most characteristically it occurs only during indulgence in the favourite game. It may be merely a nuisance and noticed only on mishitting a backhand shot, but in some cases the patient cannot even hold a racket. On examination there is nothing to see, but there is always a tender point, either exactly over the radio-humeral joint, on the epicondyle just above, or in the extensor group of forearm muscles. It may be difficult to locate the exact spot, but the patient can nearly always find it, and pressure on it will make him wince.

Of fifty-eight cases of which I have full notes, the tender point was over the joint—front, back, or side—in thirty-two, the epicondyle in twelve, and somewhere in the muscles in fourteen; but the respective points are so close together that certainty as to the exact position is not always possible.

The only other physical sign is a slight limitation of extension at the elbow-joint, more evident and often only evident with the forearm pronated; this occurred in approximately two-thirds of my cases, and is, I believe, the essential indication for manipulation. It may be very slight and no more than a springy resistance to complete extension, which is not felt in the opposite limb. It may be so slight, in fact, that it can be overlooked. On two occasions I have noted no limitation of extension, but against my general rule have for some reason tried manipulation. In each case I obtained further extension with a definite click, and in each case there was a quick cure. Any gross limitation of extension, such as could be

measured with an arthrometer, would suggest a much more serious lesion than the condition we are considering. In cases which present this typical limitation of movement, the tender point is usually over the radio-humeral joint (seventy-four per cent.), less frequently over the epicondyle (twenty-six per cent.), and never in the muscles themselves.

Cases with muscular tenderness are of an entirely different character. My practice has been to advise manipulation only in cases in which I have found limitation of movement. The technique, which I described some years ago, is very simple. Under nitrous oxide anæsthesia, the elbow is forced into full extension with the wrist and fingers flexed and the forearm pronated. At the same time, in epicondylar cases, firm pressure is made over the tender point with the thumb of the hand which is controlling the elbow. When this movement is carried out, one of five things is observed.

1. The elbow comes straight with a click, which gives one the impression of something slipping in the joint (thirteen cases).

2. It comes straight gradually, with a feeling as if adhesions were giving way (seven cases).

3. It gradually comes straight with continued effort, but neither click nor adhesions are felt (seven cases).

4. As soon as the patient is under the anæsthetic, the elbow comes straight without the slightest resistance (three cases).

5. The elbow cannot be got straight (one case). This, of course, was a failure, and will be referred to later.

The routine after-treatment is simply two weeks' rest from tennis or golf, during which time the patient is instructed to maintain by daily exercises the extension that has been secured.

I believe that the common type of case with joint tenderness and limitation of extension is due to a fraying of the thin edge of the coronary ligament and the nipping of a semi-detached portion between the bones. My reasons are as follows :—

1. The condition is frequently cured dramatically by a manipulation which produces a click.

2. It may relapse suddenly and be cured by a further manipulation. This happened to a medical man, referred to above.

3. In a case treated without anæsthesia the patient was certain that something had slipped back into place. He came to me in the middle of an important tournament and said he could not even hold a racket. A few hours after manipulation I watched him play a fine game.

4. On one occasion, I opened the radio-humeral joint and found the thin edge of the coronary ligament frayed out as I have suggested.

5. The similarity to a displaced internal semilunar cartilage is very striking. Curiously enough, my manipulation for tennis elbow is exactly analogous to Sir Robert Jones's manipulation for reducing a knee cartilage.

ERNEST W. HEY GROVES, M.S., F.R.C.S.
*Emeritus Professor of Surgery, Bristol University*

# MODERN TREATMENT OF FRACTURES OF THE NECK OF THE FEMUR

THE Smith-Petersen nail was introduced at a time when the whole trend of surgical opinion was set strongly against "plating" of any kind which involves burying large foreign bodies in the tissues. I well remember when Smith-Petersen introduced his device before a joint meeting of the British and American Orthopædic Associations in 1929, and how shocked many of us felt at the deliberate reversion to the steel-plate method of fixing fractures. And yet now, less than ten years after, it may be fairly said that the Smith-Petersen nail has completely dominated the situation, not only in America and England, but also in Germany, France, Belgium, Austria, and Italy. The rapid adoption of this method has been due to the very quick immediate restoration of function which it affords. The patient who we were accustomed to think of as languishing in bed for months is now able to get up within a week.

At present I do not think we have enough accurate information as to the ultimate results of the Smith-Petersen nail treatment. I have seen cases in which the nail has been perfectly applied, and the patients have been able to walk out of hospital, and yet in less than twelve months they have extruded the nail and suffered disunion, being left with an ununited fracture. I should very much like to know in what percentage of cases this disappointing result occurs. That it must occur in a certain proportion is obvious. Nailing the fracture will sometimes imprison a fold of capsule between the fragments, and no bony union can cross this fascial barrier. In other cases, even without interposition of capsule, new bone will be very slow in growing from the neck into the head, and will be so feeble that it is quite unequal to bearing any strain. For both these reasons it is certain that failure must sometimes occur, and before long we ought to be able to state the probability of this mischance with some accuracy.

So far I have merely referred to the insertion of the Smith-Petersen nail without distinguishing between the different methods of its application. These may be grouped under three headings, namely—

1. The open operation.
2. Blind insertion over wire guides.
3. Blind insertion with the help of a mechanical director.

*The Open Operation.*—Smith-Petersen's original operation in this country has been modified by Watson Jones. The method was well described and illustrated in the July number of the "British Journal of Surgery" last year. In the case of comparatively young and robust patients this is the method of choice. It gives a

good view of the fracture, and enables the operator to draw aside the flap of capsule which so often is folded over the fractured neck of the femur. And, further, it allows an accuracy of reduction under the guidance of direct vision which is denied to any method guided only by the X-rays. The insertion of a stout drill right through the neck and head, and on into the acetabulum, will hold the fragments in position whilst the nail is being driven into place. But, on the other hand, it is foolish to pretend that the open operation is either easy or free from danger. It must take nearly an hour in performance; there must be some loss of blood, and there must be a wide exposure of the soft parts and a free opening of the cavity of the hip-joint. For all these reasons I hold that it is unjustifiable in old and feeble patients. And it is just these feeble old patients who constitute the real problem in the wards of many hospitals, and particularly in the municipal institutions.

*The Blind Insertion over Wire Guides.*—This method, which is associated with the names of Sven Johansson, Jerusalem, and King, has had a great vogue. Frankly, I feel quite unable to do justice to the method, of which I have had so little experience. It involves two great drawbacks. First, it is long and tedious, and involves keeping the patient for one or two hours under an anæsthetic. Secondly, it is dependent on taking and interpreting many X-ray pictures, on which reliance must be placed for guiding the nail into position. These drawbacks, to my mind, make it unsuitable for the feeble old patient whom, I have already said, is the real problem we have to treat.

*Blind Insertion with Mechanical Director.*—Instead of being solely dependent on the X-rays to guide the Smith-Petersen nail into position, various mechanical devices have been invented to secure accuracy of insertion. There are a host of these, and I will only speak of one which I have designed, and which has the great advantage of simplicity. The fracture having been reduced, a two-pronged guide is placed over the line of the bones so that one prong touches the middle of the head of the femur and the other the base of the femoral neck. This enables a drill to be passed accurately into the bone in the true axis of the neck of the femur. Only a short incision is required; the operation is bloodless; it can be done under gas-oxygen anæsthesia; and it occupies only about ten to fifteen minutes. It is therefore suitable for the feeble old patient, who will suffer neither loss of blood nor shock.

The tendency nowadays is to give very little after-treatment, but to consider that the properly placed nail has cured the patient. Eversion of the leg is prevented by a bar fixed to the sole of the shoe, or by slinging the flexed knee to an overhead cradle. The patient is usually allowed up within one week, and walks with sticks or crutches, bearing no weight on the injured leg for about six weeks. I do not know why the tried and trusty walking calliper has been abandoned, but unfortunately it has. It seems to me that it would be much wiser to adhere to the use of this appliance as being a safeguard against weight-bearing until bony union is assured. The nail should be removed about three months after its insertion. It can do no further good, and its continued presence tends to produce bone atrophy.

JOHN MORLEY, Ch.M., F.R.C.S.
*Professor of Surgery, Manchester University*

# VISCERAL PAIN

THIS problem is a peculiar one, differing from that of pain in the genito-urinary system, since the gastro-intestinal tract is quite insensitive to the stimuli—cutting, burning, etc.—which cause pain when applied to the somatic nerves, and yet is supplied with afferent nerve-fibres capable of causing direct visceral pain in response to an adequate stimulus.

The one stimulus adequate to produce direct visceral pain in the gastro-intestinal tract appears to be tension on the afferent nerves in its muscular coats, and this tension is usually caused by spasmodic and exaggerated peristaltic contraction.

The afferent nerves supplying the insensitive intraperitoneal viscera show no anatomical features that distinguish them from the somatic sensory nerves, but their physiological limitations are so striking that we are justified in regarding them as a separate system, to which we may perhaps give the name of viscero-sensory nerves. It must not be forgotten that the mesentery and mesocolon are supplied by somatic sensory nerves to within a short distance of their attachment to the bowel, though the great omentum appears to be devoid of any sensory nerves subserving pain. The sensitive mesentery is often involved in such lesions as strangulation and volvulus, and this causes a combination of visceral and somatic pain that is not easy to unravel.

We can study the features of visceral pain in the intestine in its purest form in strangulation of a small knuckle of the lower small intestine in a femoral hernia. I insist that the knuckle must be a small one, because then the sensitive portion of the mesentery is not involved. Another equally good lesion for our purpose is a stenosing cancer of the transverse or pelvic colon, causing a high grade of obstruction. In both conditions the pain occurs in intermittent spasms lasting half a minute or so, and repeated at regularly recurring intervals.

If we interrogate and examine a patient suffering from such a visceral pain, we find that it is felt as a central pain and not on the surface of the abdomen. We also find that it is quite vaguely localized, though in the case of the small-intestine strangulation it is localized at or above the level of the umbilicus, whereas in cancer of the colon, whether in the transverse or pelvic portion, the patient points to the hypogastrium as the seat of the pain. Physical examination reveals a complete and remarkable absence of objective somatic manifestations. We find no superficial hyperalgesia, no deep tenderness, and no muscular rigidity, and movement of the abdominal wall does not accentuate the pain, so that the patient often writhes about in a vain attempt to obtain relief.

On the other hand, where an abdominal pain is associated with reflex muscular rigidity and with deep tenderness on palpation, the strict localization of this rigidity and tenderness suggests a somatic origin for the reflexes. We see this most convincingly in acute appendicitis, where the site of the second pain in the right iliac fossa, with its corresponding area of tenderness and rigidity, varies from case to

case with the position in which the appendix happens to be lying; so that in almost every case before we operate we can predict where the inflamed appendix will be found.

I have argued before that these clinical facts drive us to the inevitable conclusion that the nerves of the sensitive parietal peritoneum must receive the stimulus that gives rise to the pain and tenderness in the superficial parts of the abdominal wall, and to the associated reflex muscular rigidity, and I have described the process as peritoneo-cutaneous radiation and a peritoneo-muscular reflex. An appreciation of this peritoneo-cutaneous radiation is essential for any true understanding of abdominal pain, and the mechanism is best illustrated by stimulation of the phrenic nerve terminals below the diaphragm. When the under-surface of the central portion of the diaphragm receives a painful stimulus, immediate pain is felt in the region of the descending branches of the third and fourth cervical nerves supplying the skin of the supra-acromial region.

Infiltration with novocain of the area of skin supplied by the third, fourth, and fifth cervical nerves either abolishes or greatly modifies the referred pain caused by stimulation of the diaphragm (Morley, 1931). The experiment has been indirectly criticized (Woollard, Roberts, and Carmichael, 1932), but it has been several times repeated, and the fact must be accepted as established. The mechanism of this shoulder-tip pain is a matter of extreme interest. It is beyond question purely somatic, since the third, fourth, and fifth cervical segments of the cord have no neural connections with the abdominal viscera.

Recent experimental work on action currents, combined with division of the posterior nerve roots, would seem to provide us with a simple explanation of this peritoneo-cutaneous radiation of pain. Barron and Matthews (1934-5) have shown that some forty per cent. of the posterior root fibres of the spinal nerves can carry centrifugal impulses. Of these a large proportion start in a peripheral sense-organ and run to the cord. There one collateral (or more) passes a long or a short distance up or down the cord, and leaves it again by a different rootlet from that containing the entrant fibre, though it may be in the same or a different posterior root. It then passes to the periphery, often to a point far distant from the original sense-organ. Barron and Matthews found that of fibres entering the cord by the posterior roots, more than half send collaterals out to the periphery via a posterior rootlet different from that by which they enter.

They have found, further, that when a single afferent fibre in a posterior spinal rootlet is stimulated, the impulse emerges through its efferent collateral or collaterals in other rootlets with no more delay than can be ascribed to conduction time. This observation gives confirmatory evidence that the efferent fibres referred to are collaterals of the posterior root ganglion cells, and that no synapse is interposed in their course within the central nervous system. Yet, despite this absence of a synapse, inhibition or modification of impulses within the central nervous system was proved, since discharges enter the cord as a continuous series of impulses and leave it as an intermittent series.

It is plain that the old conception of the posterior root fibres as purely afferent in

function is no longer tenable. This recent discovery of efferent collateral fibres in continuity with the cells of the posterior root ganglia, appears to give us a probable key to the mystery of peritoneo-cutaneous radiation of pain, without any violation of the law of forward conduction associated with the names of Bell and Magendie. Is it not probable that, on stimulation of the phrenic nerve, efferent fibres in the posterior root run down to the skin supplied by the third, fourth, and fifth cervical segments, and there liberate a chemical metabolite which stimulates the endings of adjacent pain fibres in the skin (see diagram)? The work of Sir Thomas Lewis has made us familiar with the conception of pain as due to the action of a chemical substance on the sensory nerve endings. But it is plain that the nocifensor system of nerves postulated by Lewis (1937) cannot be held responsible, since the hyperalgesia produced in his experiments took many minutes to develop and persisted many hours, whereas shoulder-tip pain and its associated hyperalgesia develop instantly when the phrenic nerve is stimulated, and when the stimulus is removed they almost as instantaneously disappear. In the pain that we are considering, the reaction that occurs in the skin is both rapid and reversible.

A study of phrenic pain leads us inevitably to the conclusion that a precisely similar peritoneo-cutaneous radiation occurs in the superficial nerves of the anterior abdominal wall when the underlying parietal peritoneum receives a painful stimulus from an inflamed viscus. Infiltration experiments with novocain again confirm this conclusion. It is easily demonstrated, for instance, that the localizing pain in acute appendicitis or acute cholecystitis is greatly modified or completely abolished by inducing local anæsthesia by subcutaneous infiltration of the area in which the pain is felt.

Holding, as I do, that true visceral pain in the intraperitoneal organs is in no sense projected to the surface of the body, and that Mackenzie's theory of a viscero-sensory and viscero-muscular reflex is unsound as far as these organs are concerned, I suggest that a more precise and just definition of referred pain is required : that we limit the term "referred pain" to pain resulting from stimulation of a somatic sensory nerve, and referred to a remote part of the distribution of that nerve or of the segmental sensory distribution with which it is connected. This definition covers such well-known examples of referred pain as that felt in the knee in disease of the hip-joint, or as the striking experiment of Sir Thomas Lewis, in which stimulation of nerve endings inside his maxillary antrum caused referred pain and hyperalgesia in the whole distribution of the second division of the trigeminal nerve.

. . . . . .

JOHN MORLEY, Ch.M., F.R.C.S.
*Professor of Surgery, Manchester University*

## TREATMENT OF NON-STENOSING PEPTIC ULCER

In the treatment of non-stenosing ulcers there has been a movement during the last decade, in practice rather than opinion, towards conservative measures. The

best surgical opinion has always upheld a sane conservatism. It was a favourite *obiter dictum* of Lord Moynihan's that he never operated on a case of duodenal ulcer until it had been cured nine times by a physician. But there is no doubt that some years ago there were numerous surgeons who regarded all chronic peptic ulcers as suitable grist for their mill, though, being what are known as "practical surgeons," they were not given to airing their views in the medical literature. The swing of medical opinion that has effectually prevented this wholesale and indiscriminate operating upon non-stenosing ulcers is, it will be agreed, a good thing.

The change in practice to which I have alluded has been brought about partly by the unfortunate results of indiscriminate surgery, but chiefly by improved medical technique. The almost universal use of fractional gastric analysis has provided a much better guide to treatment than to diagnosis. Far more attention has been devoted to diet, and it has been widely realized that diet is more important than alkalis in the continuous neutralization of hyperchlorhydria which is the aim of medical treatment. There is also a praiseworthy tendency in many quarters to test the results of medical treatment by repeated radiological studies, though, as we shall see later, there are dangers in placing too much reliance on the picture of an ulcer crater.

The proper treatment of non-stenosing duodenal ulcer is medical. Few, I imagine, will dispute this statement, or deny that if the medical treatment is thorough, including an adequate period of rest in bed at the start, and the most punctilious regulation of diet and manner of life in the later stages, the results will usually be good. But cases are not uncommon in which medical treatment fails because the patient is unable or unwilling to carry out instructions as to diet, or for various other reasons.

Another indication for surgery in duodenal ulcer is the occurrence of severe hæmorrhage, in one or more attacks. I do not now advocate operation during or immediately after a grave hæmorrhage, for I believe the risks are too great. But a serious hæmorrhage is very prone to recur, and our aim should be to prevent the chance of a recurrence if we can do it with reasonable safety. The grave emergency can generally be tided over by blood transfusion, and then, when the pulse-rate and volume and hæmoglobin percentage show that we can operate with safety, we should not delay longer if the patient is on general grounds a good surgical risk, and if we are sure that he has a chronic ulcer.

We have not solved the problem, however, when we have laid down the indications for operation. An equally important question is, What operation should we perform? Where the main indication for operation is the prevention of recurrent hæmorrhage, I am sure that the right answer is this, that the first essential is to excise the ulcer, and with it the vessel that has been bleeding. Gastro-enterostomy alone is a very ineffective operation for a bleeding duodenal ulcer, and gastro-enterostomy combined with excision of the ulcer is both difficult and dangerous. The most effective method of excising the ulcer is to include the first part of the duodenum in some form of gastrectomy. This both controls the source of the bleeding and averts the danger of a recurrence. But where the ulcer is a penetrating

one on the posterior wall of the duodenum, with great surrounding fibrosis, its excision may involve too much danger. In such cases it may be more prudent to leave the ulcer alone, divide the pre-pyloric portion of the stomach some two inches above the pyloric sphincter, and do a high Polya gastrectomy. The ulcer will be left in a cul-de-sac of duodenum removed from contact with acid gastric juice, and may be relied upon to heal without delay.

We must now consider the choice of operation for simple non-stenosing duodenal ulcers with no history of hæmorrhage. It is comparatively rarely that we are called upon to operate on such cases, but in recent years opinion has been more divided as to the best operation to perform in this condition than upon any question in gastric surgery. We may, I think, exclude from our consideration such operations as gastro-duodenostomy in its various forms, which have never achieved great popularity among surgeons in these islands, and other "fancy operations," and limit our attention to the question of posterior gastro-enterostomy *versus* gastrectomy. Posterior gastro-enterostomy is an operation with a very low mortality—not more than two per cent. in competent hands—and the vicious-circle vomiting so often met with twenty years ago has been eliminated by modern technique; but over it hangs the dread of gastro-jejunal ulcer, and gastro-jejunal ulcer is a far worse calamity than the duodenal ulcer to which it may succeed. What is the risk of this calamity after a posterior gastro-enterostomy? The most careful and comprehensive inquiry into this problem was the collective investigation carried out for the Fellows of the Association of Surgeons of Great Britain and Ireland by Garnett Wright, and published in 1935. In a series of 1,730 cases of duodenal ulcer treated by posterior gastro-enterostomy he found an incidence of gastro-jejunal ulcer amounting to 8.49 per cent. But there are certain difficulties in accepting this figure as the whole truth.

This is a grave indictment against the operation of posterior gastro-enterostomy for the non-stenosing duodenal ulcer, and this is the reason why many surgeons in this country are abandoning gastro-enterostomy except where stenosis is present. There is good evidence for the belief that gastrectomy, by permanently lowering the secretion of hydrochloric acid, involves a much lower risk of anastomotic ulcer later, but it is a more difficult operation, with a higher mortality, and is certainly no operation for the occasional surgeon.

What is the operative mortality of gastrectomy for duodenal ulcer? It is higher, I believe, than that of the same operation when performed for gastric ulcer, because of the greater technical difficulties, and it may be estimated as somewhere in the region of six per cent. But it is essentially capable of reduction in competent and experienced hands, though it is too much to hope that gastrectomy for duodenal ulcer will ever carry as low an operative mortality as gastro-enterostomy.

The conclusion I submit is the following. Where the gastric analysis shows a high hydrochloric acid curve, as it commonly does in young men with duodenal ulcer, the risk of gastro-jejunal ulcer later is so grave that we should do a high gastrectomy, preferably of the Polya type. In duodenal ulcer in women the risk of recurrent ulcer is much less, and here, if the test-meal shows a low acid curve,

gastro-enterostomy is a very satisfactory operation. In elderly patients who are poor risks by reason of cardiac or pulmonary degeneration, there is also less danger of recurrent ulcer, and where some operative measure is inevitable the less severe operation of gastro-enterostomy should be performed.

Although gastric ulcers share with duodenal ulcers a common, and largely uncertain, etiology, their surgical treatment presents very different problems. As we have seen, our treatment of a duodenal ulcer is conditioned by the fear of a more or less remote gastro-jejunal ulcer. Operations for gastric ulcers, however, because of the much lower gastric acidity, are very rarely followed by anastomotic ulcers. But gastric ulcers carry their own special nightmare—the dread of carcinoma. Carcinoma in the first part of the duodenum is so rare that it may almost be said not to occur there, nor does a cancer of the pyloric end of the stomach ever spread into the duodenum. But the stomach is the commonest site of cancer in the body.

The danger of cancer supervening on a chronic gastric ulcer has been greatly exaggerated in many quarters. It is a long time since Wilson and MacCarty (1909) of the Mayo Clinic startled the medical world by the statement that sixty-eight per cent. of all gastric ulcers removed at operation showed microscopical evidence of malignancy, and that seventy-one per cent. of all gastric carcinoma showed pathological evidence of a preceding simple ulcer. It has been proved by many succeeding investigators, approaching the problem from both the pathological and the clinical aspects, that the incidence of the so-called ulcer-cancer is far smaller than Wilson and MacCarty claimed.

It is not the frequency with which they become malignant that should influence our attitude to the treatment of gastric ulcers, so much as the difficulty in many cases of arriving at an accurate diagnosis between simple and malignant ulcers. There is a type of slow-growing cancer of the stomach, by no means uncommon, which very closely simulates the clinical picture of gastric ulcer. In these cases the pain is at first periodic in character, and is relieved both by food and by alkalis. Appetite is often retained, and the gastric analysis in the early stages shows a high or normal acidity. The growth may be regarded as a simple ulcer for a year or two, and yet is a carcinoma from the beginning. The radiological appearance may be either doubtful or suggestive of simple ulcer.

In view of the rarity of simple ulcers and the frequency of carcinomata in the pyloric region, lesions in that position that appear to be simple ulcers should always be regarded with suspicion. It is a sound rule that where a pre-pyloric ulcer has been treated medically for three weeks, and there is still occult blood in the stools, gastrectomy should be performed. Whenever a patient of cancer age presents a considerable ulcer crater in any part of the stomach, with a short history of gastric pain, a suspicion of malignancy should be entertained, no matter how innocent the radiological appearance may be.

Quite apart from the urge that the fear of cancer gives us to radical measures for gastric ulcers, the results of surgical treatment of ulcers in this field are more favourable than in the non-stenosing duodenal ulcers. This is mainly attributable to the fact that in gastric ulcers we find a much lower average acidity than occurs

with duodenal ulcers, and consequently there is comparatively little danger of recurrent or anastomotic ulceration. Medical treatment, on the other hand, especially in the larger ulcers, is often followed by relapse after relapse.

. . . . . .

E. R. FLINT, F.R.C.S.
*Professor of Surgery, University of Leeds*

## OBSTRUCTIONS OF THE COMMON BILE DUCT

THE causes of obstruction are numerous, and many of them rare. I do not propose to enumerate them all. Those with which we have commonly to deal are: (1) stone and its complications, whether pathological or the result of surgical treatment; (2) pressure on the duct by pancreatic lesions, malignant or simple. I am prevented by lack of space from doing more than mention a presumed and possibly not uncommon cause due to spasm of the sphincter of Oddi. It is an interesting problem, and one on which a good deal of research is being carried out.

The problem is usually one of determining the cause of jaundice of the obstructive type. In the main the difficulty is not great, the chief characteristics being :—

| STONE | CARCINOMA OF PANCREAS | CHRONIC PANCREATITIS |
|---|---|---|
| Painful | Painless | Usually some pain |
| Intermittent and varying | Persistent and progressive | Variable |
| Short duration | Long duration | Long duration |
| Not deep | Deep | Fairly deep |
| Often infection (intermittent hepatic fever) | Aseptic | May be infection |
| Gall-bladder not palpable | Gall-bladder usually palpable | Gall-bladder often palpable |
| Wasting variable (may be rapid) | Rapid wasting | Some wasting |

With these features, the history, and a positive prompt van den Bergh reaction there is usually no difficulty in deciding whether a stone is the obstructing agent or not; but it is often impossible even at operation to differentiate between malignant and simple lesions of the pancreas.

Unusual cases occur not infrequently, and the difficulties may then be considerable. For example, in calculous obstruction there may be complete absence of pain, and jaundice, which as a rule is of short duration and not intense, may be deep and lasting. Carcinoma of the ampulla of Vater may ulcerate and permit bile to flow again after the customary deep jaundice and be associated with infection. Again, in the calculous cases, sepsis varies, sometimes dominating the picture and at other times being altogether absent. Indeed, where there is infection without jaundice, there is a risk of overlooking the biliary system as a possible source of the trouble.

Moreover, the difficulties in diagnosis arise not entirely in sorting out one cause of obstructive jaundice from another, but also in determining whether the jaundice is of intrahepatic or extrahepatic origin. When obstruction is due to pressure on the duct from without, it has a bluish colour, and as a rule contains dark inspissated bile, because the gall-bladder is usually normal and carries on with its concentrating function. Where stone is the cause, infection in the duct is common, and the gall-

bladder is more or less functionless; the bile is thin and yellow, or muddy with possibly a fæcal odour; and the wall of the duct is thick and has a pinkish-yellow hue.

In about five per cent. of cases of common-duct obstruction, the duct is filled with "white bile"; experimentally white bile occurs only in those cases where the gall-bladder has been disconnected from the obstructed duct; clinically it is most often seen when obstruction is due to stone and where the gall-bladder has been more or less destroyed by inflammation. The ducts are filled with a mucoid secretion derived from their walls. There is still some doubt about the explanation of this state of affairs, but it indicates very serious hepatic injury, and is therefore a signal to the surgeon for the least operative intervention compatible with efficiency at the time.

Of great practical importance from the point of view of the patient's safety are the hepatic changes resulting from obstruction; some degree of injury is always present, varying from mild central necrosis to acute atrophy, and slight periportal fibrosis to definite cirrhosis. It is not known why one patient should present minimal changes and another gross damage, nor have we the means of accurate determination as we have in the case of the kidney. The best guide, I think, is the bilirubin content of the serum. In many respects bilirubinæmia and high blood-urea have a similar meaning as regards the respective organs. Both result from back pressure and functional insufficiency; neither in itself is harmful in the circulation, and in both cases the variations in the blood are detectable by reasonably easy and accurate tests.

Bilirubin accumulates in the plasma even if the obstruction persists for months; bile salts, on the other hand, soon disappear from both the blood and the urine; the cholesterol content of the plasma rises by retention; the blood-urea mounts, and may be followed by casts and albuminuria and vomiting of altered blood. These indicate renal injury, and it is a practical point of the highest importance to keep in mind that the convoluted tubules of the kidney seem to be particularly vulnerable to something appearing in the blood as a result of hepatic injury.

Ascites and splenic enlargement may complicate common-duct obstruction if hepatic fibrosis is extensive, and a stone in the ampulla may cause dilatation of the pancreatic ducts, with atrophy of the secretory cells from interlobular fibrosis: interacinar fibrosis is rare and thus true diabetes uncommon, though glycosuria is not infrequently seen with stone obstructing the common duct.

The longer the disturbances initiated by common-duct obstruction last, the more they pass from physiological dysfunction to structural and irreparable damage. Delay in treatment is therefore to be avoided, and as the obstruction is mechanical it can only be dealt with by surgery. Waiting a week or two is permissible; first, because if the obstruction is due to stone, spontaneous relief will generally be observed in this time, and the need for immediate surgery passes; and, secondly, because whatever the cause of the obstruction, this period gives opportunity for pre-operative preparation, which I regard as imperative. It is in my opinion as essential a part of the treatment as is iodine, etc., in cases of toxic goitre.

E. C. DODDS, M.V.O., D.Sc., Ph.D., M.D., F.R.C.P.
*Professor of Biochemistry in the University of London, Middlesex Hospital*

# THE SEX GLANDS

It is a well-known fact that the injection of extracts from the anterior lobe of the pituitary into immature animals will produce the precocious appearance of puberty associated with ovulation and the formation of corpora lutea in the case of the female; and in the case of the male, descent of the testis and spermatogenesis will occur. Conversely, in the hypophysectomized adult animal all sexual activity ceases; and this, again, can be restored by the injection of a suitable extract. Up to the present, however, there is no such extract of the anterior lobe available for clinical trial.

It was discovered that in certain species the urine of pregnancy contains a substance capable of stimulating the ovaries of the immature animal. The test for the presence of this gonadotropic substance forms the basis for the well-known Aschheim-Zondek reaction; the substance was given the name of prolan by the original German investigators. The problem then arose as to whether this was a single substance or whether, according to the German view, it was composed of two parts—prolan A stimulating the ripening of the follicle, and prolan B being responsible for the luteinization of the follicle. The material which is at present on the market is agreed by nearly all workers to be almost entirely the luteinizing fraction. Therefore, when we speak in subsequent discussions of the urinary gonadotropic hormone, it must be borne in mind that we are dealing almost entirely with the luteinizing fraction.

It has recently been shown that the serum of the pregnant mare during certain stages of gestation contains a very active ovary-stimulating factor. This has been separated, and is available for clinical trial under the name of "antex." Most workers have agreed that antex is mainly follicle-stimulating, as opposed to the luteinizing fraction.

With these two substances—namely, urinary prolan and antex—it would appear that the clinician is provided with all the factors necessary for the control of ovarian secretion.

Hormones of the ovary consist of three substances: œstrone, œstradiol, and progesterone. Both œstrone and œstradiol, when injected into ovariectomized animals, will produce all the changes associated with the sexual cycle in the rodent. They will also cause the onset of premature puberty, and they only differ one from the other in their relative potency; œstradiol is by far the more potent of the two substances. Both œstrone and œstradiol are available clinically in the form of benzoate. These esters are used because their absorption is slower, and therefore with a single injection it is possible to get an effect very nearly approaching that of the normal function.

If either of these substances be administered to the monkey, it is found that the menstrual cycle cannot be reproduced. Experiments on ovariectomized rabbit have shown that a second factor is necessary for the complete reproduction of the cycle,

and that this may be obtained from the corpus luteum. This material, called progesterone, has been isolated from alcoholic extracts of the corpus luteum, and has been characterized chemically. It is produced commercially in two ways—from the corpora lutea and also by the complex degradation of stigmasterol. If œstrone or œstradiol be injected into an ovariectomized woman for a certain length of time, and this be followed by injections of progesterone, a complete menstrual cycle can be instituted. It can thus be stated that the clinician is supplied with the necessary agents for the production of the menstrual cycle.

When the pure crystalline hormones were first isolated, one of the difficulties at once appreciated was the question of absorption from subcutaneous injection. Earlier experiments had shown the fundamental difference of behaviour between the injection of hormones dissolved in oil and those administered in the form of an alcoholaqueous solution. In general, it may be stated that the ideal medium for injection is one which will allow a slow and steady absorption, thus imitating as nearly as possible the natural conditions. In the case of the sex hormones, this is accomplished by their administration in the form of oily solutions. In order to slow up the absorption still more, various esters have been prepared. Thus œstrone and œstradiol are available in the form of oily solutions of their benzoates. The benzoate is slowly hydrolyzed in the body, thus giving a steady supply of the œstrogenic agent to the subject.

In the case of testosterone, a very interesting observation was made when it was shown that the presence of certain fatty substances very much enhanced the activity. Out of this has developed the ester testosterone propionate, in which form the hormone is available to-day. This ester is many times more potent than the free testosterone, and the whole question of potency of this group of substances is very largely bound up with the type of ester which is administered.

. . . . . .

SIR ROBERT McCARRISON, LL.D., M.D., F.R.C.P.

## NUTRITIONAL NEEDS IN PREGNANCY

It is customary to speak of nutrition in pregnancy as though it differed in some directions—in the supply of certain food essentials, for instance—from nutrition at other periods of life. Indeed, the very title of our subject suggests such a distinction. It is customary also to speak of pregnancy as though it involved an additional burden on the maternal organism, as, unfortunately, it often does. But I suggest to you that the nutritional needs in pregnancy are those necessary to the continued efficiency of the function of nutrition throughout life. For in this efficiency, the efficiency of that other fundamental function of the body — reproduction — is inherent. As well might we regard the taking of a steep hill as a feat additional to the normal function of a motor-car, as regard the making of a seven-pound baby as a feat additional to the normal function of woman. The properly constructed car, the properly adjusted, oiled, greased, fuelled, and tended car takes the hill in its stride; the properly constructed, adjusted, fuelled, and tended woman takes

pregnancy in hers. It is this construction, this adjusting, this fuelling, this tending, that, in the perfection of its operation, the function of nutrition effects. But the case is different with the ill-constructed, the ill-adjusted, the ill-tended woman, in whom the function of nutrition from infancy onwards has been faulty. Then, indeed, pregnancy becomes a feat to which some succumb, and from which many emerge with some weakness exposed, some permanent damage incurred.

What are the things needful to the efficiency of the function of nutrition? They are :—

*First,* the adequate provision of the materials with which this function is effected: oxygen, water, properly constituted food, and a substance or substances produced in the skin by the action of sunlight.

*Second,* the efficient performance of each one of the acts or processes involved in nutrition, these acts or processes being themselves dependent on the proper nourishment of the organs and tissues performing them.

*Third,* the practice of measures and habits of life favourable to the efficiency of the function of nutrition : appropriate exercise in the open air, proper breathing, agreeable mental occupation, promotion of the function of the skin by bathing, suitable clothing, and exposure of parts of the body to such sunlight as is available, promotion of the action of the kidneys and bowels by proper food and the free consumption of water.

*Fourth,* the avoidance of all influences that adversely affect the function of nutrition : such, for instance, as insufficient rest and want of sleep, bad ventilation, insanitary conditions generally, worry, emotional excitement, constipation, alcohol, and infection.

These are the nutritional needs in pregnancy as they are the nutritional needs of the body throughout life. Their satisfaction makes of pregnancy one of the happiest, the most healthful, periods of a woman's life.

The dietetic requirements in pregnancy I shall not attempt to discuss in detail, but will content myself with an enumeration of the foodstuffs which, if properly produced, properly treated, and properly combined, provide all the food essentials the prospective mother needs. These foodstuffs are, in order of precedence :— (1) Milk and the products of milk (butter, cheese, skimmed milk, buttermilk). (2) Whole or lightly milled cereal grains; in particular a good wholemeal bread or standard bread, and oatmeal. (3) Green and leafy vegetables. (4) Root vegetables, particularly potatoes, carrots, and onions. (5) Fruit, including the tomato. (6) Pulses. (7) Egg. (8) Meat, including fish, fowl, and glandular organs. To these there must in this country be added cod-liver oil, not in the large doses commonly prescribed, but in the sufficient dose of a teaspoonful daily. And as an additional assurance of functional efficiency of blood, muscle, and nerve, a portion of yeast extract is a wise precaution.

Those of you who have studied the revised report of the Technical Commission appointed by the League of Nations to define the nutritional needs of the human being in the course of development from conception to adult age, will be aware that it is from these foodstuffs that the Commission prepared their dietary schedules

for all periods of life up to and including pregnancy. They had in mind, no doubt, the preparation of the female of the human species for motherhood.

. . . . . .

DAME LOUISE McILROY, D.B.E., LL.D., M.D., D.Sc., F.R.C.P., F.C.O.G.

## NUTRITION IN PREGNANCY

THE reduction of maternal mortality has now become a national responsibility, and if further progress is to be made, more attention must be paid to nutritional defects in the diet of the expectant mother. The more one studies the diseases of pregnancy, and even the complications of childbirth, the more is one convinced that these owe their origin in many cases to dietetic defects and that they should be classified as deficiency diseases. In spite of all the valuable research that has been undertaken with regard to nutrition both in man and animals, it is only during the past few years that these results have begun to be applied to obstetrical practice.

The maternal organism has not only to maintain its normal condition of nutrition, but it has to act as a storehouse for the needs of the fœtus. If these reserves become depleted at any time, the fœtus may become a parasite upon the maternal host, as it is imperious in its demands for growth and maintenance. We know that, for example, the liver stores up glycogen, calcium, iron, and other substances for the needs of the fœtus. If these reserves become inadequate, the result is shown by the onset of anæmia, toxæmia, eclampsia, or vomiting of pregnancy. If the supply of vitamins and mineral salts is deficient, abortions and stillbirths may take place. Such knowledge has been of inestimable value clinically in the treatment of the complications of pregnancy by substances which after all reinforce, one might say, the diet of the expectant mother.

In the ordinary course of work in an ante-natal clinic, we find that many patients complain of dyspeptic disturbances, muscular cramps, sleeplessness, backache, and other symptoms during pregnancy. These can be treated with good results, not by sedatives as formerly, but by the administration of mineral salts such as iron and calcium, and by careful advice as to diet, together with the observation of routine weighing and control of open-air exercise. If we could prevent the occurrence of such symptoms by dieting, the results would obviously be much more satisfactory.

How much time does the average medical officer of a clinic spend upon each patient in giving advice as to her diet? What are the possibilities of carrying out this advice when given? The average woman cares little about the mathematical exactitude of the caloric value of foodstuffs or their vitamin contents. Her concern is the best possible return for her financial outlay, so that the hunger of her family may be satisfied. Her attention is directed towards appetizing foodstuffs which give little trouble in preparation and allow of the smallest amount of waste. Too often do we find that in the poorer districts the patient sacrifices her own needs to those of her family, and it is for this reason that the supply of food for those in attendance in clinics has been somewhat of a failure. The problem of expense is the greatest

difficulty, when we know that the chief source of vitamin supply is to be obtained from fresh dairy produce and green vegetables. Not only is the price prohibitive, but there is the difficulty of obtaining pure milk and fresh food. Even in pastoral counties I have known of instances where it is impossible to obtain an adequate supply of milk for expectant mothers. Dairy produce is purchased in quantities for the supply of cities, and the local requirements are neglected. Green vegetables are as a rule prohibitive in price, especially in the winter months.

What is the diet of the average working-class mother of to-day? We have only to look at the shops in the country villages as well as in towns. Windows are heaped with bread made from milled flour, which is now the substitute for the home baking of our ancestors. It is cheap and satisfying, and is ready prepared. There are piled up tins of every conceivable form of foodstuff, meat, vegetables, and fruits, which are not an addition to the diet, but a substitute in many cases. These tinned foods are appetizing, are ready prepared, and have little waste, but in spite of assurances to the contrary they can never replace fresh foods. Supplies come from abroad from long distances, and we find our working-classes consume chilled meat, imperfectly thawed and then cooked out of all recognition of its original nutritional substances.

I know all this may seem below the standard of a scientific discussion, but in my opinion little progress will be made until we tackle the problem from this point of view. It is a national concern which involves the support of our own agricultural producers, the transport of foodstuffs, the wise buying, education in cooking, and the provision of houses where facilities for storage are made available.

. . . . . .

STUART J. COWELL, M.B., F.R.C.P.
*Professor of Dietetics, University of London*

## THE PHYSIOLOGICAL BASES OF NUTRITION

I PROPOSE to limit my observations to certain practical aspects of the problem of human nutrition. The main questions I wish to discuss are: What can we hope to accomplish by any dietary recommendations that we may feel justified in making? and, What diets are most likely to secure the desired effect?

Only tentative answers can be given to these questions: we cannot always wait for full knowledge before planning a course of action. The primary aim in making any dietary recommendations is, except possibly in rare instances of medical emergencies, to secure the best possible state of nutrition which a suitable combination of foods can bring about. The term "best possible" is used rather than "optimal," for the reason that hereditary defects, previous disease, or deleterious environmental factors may prevent a perfectly good diet from exercising its full nutritive effect, as we shall no doubt learn from the contributions of Dr. Brock and Dr. Spence. The fact that so many private individuals and official committees have of late considered it advisable to make dietary recommendations, implies that in their opinion the state of nutrition of a considerable proportion of our existing popula-

tions is not as satisfactory as it might have been under the influence of an ideal dietary. And there is much evidence to support this opinion. There is, for example, no doubt that the physical development of very large numbers of our children could be improved by better feeding during the whole period of growth. The milk-feeding tests which have been carried out in many different countries have shown that the mean rate of growth of any large group of children can be accelerated by regular supplements of this valuable food. Sufficient time has not yet elapsed for information to be available regarding the ultimate effect of such accelerated growth in the final state of development. The experience of most of those who have been engaged in controlled feeding work with laboratory animals, has been that diets which promote the most rapid growth in early life maintain the animals in the most vigorous health in adult life. It is, however, interesting to note that Dr. Hayward of Cornell University found he could prolong the life of rats by stunting them in early life, a result apparently in direct contradiction with the experience of Professor Sherman, whose rats survived to old age better when they were given diets which promoted the most rapid rate of growth.

The skeletal development of children could without doubt be improved by better feeding. While the propaganda of the last fifteen years has resulted in a considerable diminution in the incidence of the grosser manifestations of rickets, there are indications that minor skeletal defects due to suboptimal bone calcification are still common. Of equal or even greater importance is the fact that the development of the teeth is undoubtedly handicapped in the majority of our children by suboptimal nutrition in early life. The relation between diet and the development and maintenance of sound teeth is of paramount importance, and I shall refer to it again in a few moments.

Before leaving the question of diet in relation to development, I would draw your attention to certain less gross defects of faulty nutrition in the matter of physical development. Under this category might be included the failure of the hæmoglobin-producing mechanism caused by iron deficiency in childhood. As far as is known, this nutritional fault is completely reversible, but there is evidence that other faults may not be completely reversible—for example, the abnormal development of the gingival epithelium in young dogs, described by M. Mellanby as arising from deficiency of vitamin A. This made them susceptible to pyorrhœa in adult life, even if adequate supplies of vitamin A were given for long periods after growth had ceased.

The main lesson we have to learn from the fundamental research work of the present century, is that optimal nutrition depends on the liberal supply in the diet of every food component which cannot be manufactured in the body from some other constituent of food, as well as on an adequate allowance of calories and utilizable nitrogen. The term "liberal" which I have just used in reference to the supply of essential food constituents was deliberate. In some quarters there is a tendency to assume that an adequate supply of these constituents is one which prevents the appearance of gross signs of deficiency diseases, whereas there is plenty of experimental evidence to show that improved states of nutrition can be secured by

increasing the intake of such constituents above the level at which mere protection from recognizable deficiency symptoms occurs.

This brings us to practical recommendations for securing optimal nutrition. We shall lay stress on the provision of a liberal supply of foods rich in vitamins and in mineral elements, especially calcium and iron in an assimilable form—that is, foods which have lately received the name "protective." This is the essential feature of the diets recommended by the League of Nations Commission. Its corollary is a corresponding decrease in the amount of inert if not actively harmful foods such as refined sugar and cereals. Adequate provision of calories and protein is by no means to be neglected, but in our own country this is not such a pressing problem. The protective elements of our diet can most easily be secured by increasing the consumption of milk, especially safe liquid milk, and other dairy products, all kinds of vegetables, especially green vegetables including salads, eggs, fat fish, and liver. In a diet planned on these lines the proportion of cereals will be reduced. The question of the relative merits of refined and whole-grain cereals was not treated at all exhaustively by the Commission, who supported the proposal that part of the cereal eaten should be lightly milled. It seems possible that the best practice would be so to increase the proportion of starch eaten in the form of vegetables, especially potatoes, that a comparatively small amount of cereal would be eaten, of which the bulk should be lightly milled, so as to increase in the total diet the quantity of B vitamins and assimilable iron.

The Commission rightly stresses the extreme importance of providing the best possible diets throughout the whole period of growth. This is, of course, one of the reasons why the diet it recommends for pregnant and lactating women contains such a large proportion of protective foods. A few words must be said regarding the proposed diets for children. Their special feature is the high proportion of the total energy value contributed by protective foods, which is largely due to the inclusion of a liberal allowance of milk throughout the whole period of growth. The diets for children between the ages of 1 and 3 contain rather more than three-quarters of their total calories in the form of protective foods; those for children between 3 and 7 contain rather more than two-thirds, and this proportion is diminished in the pre-adolescent age to rather less than one-half. This insistence on a high proportion of protective foods is really the essence of the scheme for securing good nutrition. It runs directly counter to the attitude not infrequently adopted in some quarters that as long as a diet contains enough of the essential food constituents to prevent symptoms of deficiency diseases, there is no point in worrying about its make-up.

. . . . . .

G. F. GIBBERD, M.S., F.R.C.S., M.C.O.G.
*Obstetric Surgeon to In-patients, Queen Charlotte's Maternity Hospital, London*

## PRONTOSIL AND SIMILAR COMPOUNDS

At the isolation block of Queen Charlotte's Hospital, prontosil was used in the first part of 1936, and streptocide (a preparation of sulphanilamide) has been

employed since; as it is my purpose to study the clinical value of this group of drugs as a whole, I have drawn no distinction between the earlier cases treated with prontosil and the later cases with streptocide. I have referred to these drugs collectively as the "new aniline derivatives," and to their use as the "new chemotherapy." Prontosil has been given by intramuscular injection and by mouth in daily doses to 20 to 60 c.c. (0.5 to 1.5 grammes) intramuscularly, and six to twelve tablets (two to four grammes) by mouth. Streptocide has been administered by mouth in daily doses of eight to twenty tablets (two to five grammes). The maximum doses have been given to patients who appeared to be severely ill, and these doses have been continued until clinical improvement has occurred. After this the dose has been decreased, but smaller amounts have usually been used for as long as a week after the temperature has fallen to normal.

Some patients have developed marked cyanosis due to met- or sulph-hæmoglobinæmia, but in no case has this complication given rise to anxiety, and it has always disappeared on withdrawal of the drug. In a few cases of severe infection the use of the drug has been continued in spite of the cyanosis, without any untoward effect. There does not seem to be any constant relation between the dose administered and the tendency to develop cyanosis. Apart from cyanosis, no serious ill-effects have followed the giving of these drugs.

Since the introduction of the new chemotherapy there has been a very great improvement in the results obtained at the isolation block of Queen Charlotte's Hospital. The improved results are demonstrated: (1) by a considerable fall in the total mortality-rate; (2) by a reduction in the proportion of cases in which the infection spread beyond the limits of the birth canal, and by the relative infrequency with which an inflammatory mass developed after treatment had been instituted; (3) by a significant fall in the mortality-rate in cases of proved septicæmia, associated with a relative decrease in the proportion of severe cases of septicæmia, and by a fall in the incidence of septicæmia developing after the treatment had been instituted; and (4) by the relative infrequency with which generalized peritonitis has been found post mortem. There is no doubt about these facts. There is some doubt about their meaning.

Although they can be explained by assuming that the new aniline derivatives have supplied a highly effective remedy for hæmolytic streptococcal infections, most of them can also be explained by assuming that the virulence of the hæmolytic streptococcus has diminished spontaneously in the period during which the new drugs have been employed. There are few features revealed by the results so far discussed that cannot be accounted for on either hypothesis. These good results are mainly an expression of diminished invasion of tissues by the organisms. This may be the result of increased resistance on the part of the tissues (that is, it may be due to the chemotherapy), or it may be due to diminished virulence of the organism itself. It is a matter of personal fancy which of these alternative explanations will appeal to any of us as individuals, but the accumulated experience of the past shows that a fortuitous change in the virulence of a prevalent organism is no more improbable than the discovery of an effective chemotherapeutic substance.

# Multiple Pregnancy

*By* H. I. McCLURE, M.B., B.SC., F.R.C.S., M.C.O.G.

Assistant Surgeon and Registrar, Royal Maternity Hospital, Belfast.

THIS paper is based on a review of all cases of multiple pregnancy occurring in the Belfast Maternity Hospital, Townsend Street, and the Royal Maternity Hospital, Belfast, during the last ten years 1927-36. During this period, 164 mothers were delivered of twins, and one mother was delivered of triplets, i.e., 165 mothers gave birth to 331 babies. The number of twin pregnancies over this period represents a frequency of 1 in 50 births. The frequency of twin pregnancy is given by Curtis[1] to vary from 1 in 80 to 1 in 101 births, but it is possible that the higher incidence of this series is due to the fact that abnormalities gravitate to hospital, and the figures for this series, therefore, can hardly be regarded as a true index. At the same time, it has long been known that multiple pregnancy occurs more frequently in this country and in Russia. The case of triplets herewith recorded represents a frequency of 1 in 8,243, but no reliance can be placed on one instance. We have not had the pleasure of delivering any more than three children at one confinement in these hospitals during the last ten years. The most famous case of multiple pregnancy recorded in the literature is that of the Dionne quintuplets, and the story of their birth by Dafoe[2] is most interesting reading.

Twins may develop from the fertilization of one ovum, or of two separate ova. In the former case they are known as uni-ovular, monozygotic, identical, or single-ovum twins, and in the latter as binovular, dizygotic, fraternal, or double-ovum twins. Binovular twins, being the result of fertilization of two ova, are really not twins biologically; they are examples of simultaneous development of two ova, the result of an inherited tendency to twinning. Uni-ovular twins, on the other hand, are true biological twins, being the splitting of a single ovum into two separate and complete components, i.e., twoing, twaining, or twinning. In the case of binovular twins, the ova may come from one ovary, or one from each ovary. They are the more frequent, and may be of the same or different sex. There is a placenta for each ovum, which also possesses its own amnion and chorion. The four layers of the membranes can be shown in the septum uniting the two sacs, and the placentæ may or may not be fused. Fusion depends on the proximity of the placentæ to each other, and although the fusion may appear to be complete, it is possible to separate the placentæ from each other without disturbing the circulation, as no communication between the two circulations exists. In the case of uni-ovular twins there is one placenta with a free communication between the two circulations. In rare instances, instead of two umbilical cords, only one has developed, but as it approaches the fœtûs it bifurcates. As a rule, two amniotic cavities exist, but many textbooks describe cases where only one amniotic cavity was present, and it is presumed that the septum between the two original sacs has disappeared. Uni-ovular twins are always of the same sex, resemble each other to a remarkable degree, not only

physically, but mentally and temperamentally, and they have also been known to exhibit the same developmental physical abnormalities.

In the case of binovular twins, the two ova are probably, as a rule, fertilized at the same time, but it may be that in some cases binovular twins result from fertilization at different dates, i.e., superfecundation and superfœtation. Superfecundation is the fertilization of two ova, which have matured at the same period of ovulation, by spermatozoa from separate and distinct coital acts, i.e., fertilization has taken place at different intervals during the one intermenstrual interval. Superfœtation is the fertilization of two ova maturing at different ovulation periods, so that nidation of the second ovum occurs in the uterine cavity when the same process has already taken place in the case of the first ovum. Superfœtation cannot occur, of course, after the uterine cavity has become completely obliterated by the fusion of the decidua reflexa and decidua vera, which takes place at the twelfth week of maturity. A powerful argument against the occurrence of superfœtation is the fact that usually ovulation does not occur during pregnancy.

In twin pregnancy, the children are smaller and weigh less than the single child; for example, the average weight of each twin born in the Royal Maternity Hospital last year was five pounds two ounces. The combined weight, however, is usually greater than that of a single child, and in the same series the average combined weight of both children was ten pounds four ounces. It is not unnatural that twin children should be somewhat smaller, but this may be explained by the fact that, in many cases of twin pregnancy, labour sets in prematurely on account of the over-distension of the uterus. The weights of the children are said to be much the same, but the average difference between the weights of the twin children born in the Royal Maternity Hospital last year was 8.4 ounces. In one case, the difference was as much as one pound nine ounces, although delivery occurred at term.

### Diagnosis of Multiple Pregnancy.

During the later months the diagnosis of multiple pregnancy can usually be made with a fair degre of certainty by ordinary clinical examination. The main points are (1) excessive enlargement of the abdomen or uterus, (2) frequent changing of presentation and position, (3) multiplicity of fœtal parts, (4) early fixation of the presenting part, especially in a multipara, (5) the palpation of three fœtal poles, (6) the presence of a fœtal head which appears small for the size of the uterus or period of gestation, (7) the auscultation of two fœtal hearts. It is frequently very difficult to make out the presence of two fœtal hearts, and to make a diagnosis of twins the fœtal heart-sounds should be heard at some distance apart and to differ in rate. They should be counted over the same unit of time by two observers. In this connection Stander[3] states that "a positive diagnosis should not be made unless there is a difference of at least ten beats per minute in the rate of the two hearts, the sounds being counted for at least a minute in each location." In order to make the difference in rates more marked, De Lee[4] irritates one fœtus by gentle uterine massage in order to accelerate its heartbeats. (8) X-ray examination makes the

diagnosis absolute. It should be carried out in all cases where multiple pregnancy is suspected to demonstrate the presence or absence of fœtal abnormalities.

Difficulty arises in the early months of pregnancy when the only abnormal sign may be undue enlargement of the uterus. Here the important differential diagnosis is hydatidiform mole, and a negative X-ray examination up till twelve or fourteen weeks maturity is of no value in distinguishing multiple pregnancy from this condition. It is claimed that definite evidence of the existence of fœtal parts may be made out on radiographic examination at twelve weeks maturity, but as yet I have not seen the case where diagnosis at this stage by this means was definite. However, the diagnosis of vesicular mole may be made by means of the quantitative Aschheim-Zondek test. If this test is not suggestive of mole, the alternative is to leave the pregnancy to continue till the diagnosis becomes definite. In this connection, the conditions laid down by underwriters regarding insurance against plural births are as follows :—(1) The policy must be effected at least six months before the expected date of confinement. In some instances it may be deferred for a further month. (2) The premium is usually five pounds per cent. (3) In the event of more than one child being born, at least two must live for a longer period than twenty-four hours. (4) No claim is payable should delivery take place more than six weeks before the expected date.

PREGNANCY.

The evidence regarding the frequency of multiple pregnancy in primigravidæ and multiparæ is conflicting. De Lee[4] is of the opinion that the latter are more likely to bear twins, while the opposite view is expressed by Das.[5] In this series multiple pregnancy occurred as follows :—

| | | | |
|---|---|---|---|
| Primigravidæ | 55 cases. | Seven-gravidæ | 12 cases. |
| Two-gravidæ | 20 cases. | Eight-gravidæ | 7 cases. |
| Three-gravidæ | 20 cases. | Nine-gravidæ | 4 cases. |
| Four-gravidæ | 15 cases. | Twelve-gravidæ | 2 cases. |
| Five-gravidæ | 17 cases. | Sixteen-gravidæ | 1 case. |
| Six-gravidæ | 11 cases. | Eighteen-gravidæ | 1 case. |

Primigravidæ accounted for one-third of the total number of cases. It does not necessarily follow that the same frequency holds good for all cases of multiple pregnancy, as there are two fallacies in this statement—(1) more women are primigravidæ than are multigravidæ, and (2) more primigravidæ than multigravidæ are admitted to the hospitals concerned.

One of the commonest complications of multiple pregnancy is the onset of premature labour, possibly due to over-distension of the uterus in most cases, but also to induction of premature labour for pregnancy toxæmia. The periods at which pregnancy terminated in this series are as follows :—

| | | | |
|---|---|---|---|
| At 28 weeks maturity | - | 7 cases. | At 35 weeks maturity - 3 cases. |
| ,, 30 ,, ,, | - | 10 cases. | ,, 36 ,, ,, - 31 cases. |
| ,, 31 ,, ,, | - | 2 cases. | ,, 37 ,, ,, - 3 cases. |
| ,, 32 ,, ,, | - | 9 cases. | ,, 38 ,, ,, - 30 cases. |
| ,, 33 ,, ,, | - | 2 cases. | ,, 39 ,, ,, - 3 cases. |
| ,, 34 ,, ,, | - | 8 cases. | ,, 40 ,, ,, - 57 cases. |

It will be seen that of the total 165 cases, pregnancy terminated before the end of the thirty-eighth week in 105, i.e., 63.6 per cent. In this connection, Titus[6] records an interesting case where induction of therapeutic abortion was performed. One fœtus and its sac were recovered, and it was assumed that the uterus was empty. The patient went on to term with a twin which was born alive. He does not state the most interesting part of the story, namely, the indication for the therapeutic abortion.

Pre-eclamptic toxæmia is a common complication of multiple pregnancy, occurring in this series in sixty-two cases, i.e., 37.5 per cent. Induction of premature labour was performed in twelve of these sixty-two cases., i.e., 7.2 per cent. of the total number of cases of multiple pregnancy. In addition, there were four cases of eclampsia gravidarum, so that sixty-six of the total series, i.e., forty per cent., showed evidence of pregnancy toxæmia. The reasons for the high incidence of toxæmia in association with multiple pregnancy are unknown, but McIlroy[7] attributes it to the larger placental surface, the greater drain on maternal stores and nutrition, the added excretory function of the fœtûs, and, to a certain extent, pressure effects, due to bulk and to the hydramnios which so frequently accompanies twin pregnancy. In a series of fifty cases of twin pregnancy which she reviewed, she found evidence of toxæmia in thirty-five cases, i.e., seventy per cent.

Hydramnios is said to be a common complication of multiple pregnancy. In McIlroy's series the frequency was twenty-four per cent., but in mine the condition was noted in only six cases, i.e., 3.6 per cent. As a rule, only one sac becomes hydramniotic. The disturbing feature of hydramnios is that it tends to increase discomfort by aggravating such symptoms as dyspnœa and œdema.

## Labour.

As shown above, in many cases labour sets in prematurely. Not infrequently there is some degree of uterine inertia, due to over-distension of the uterus; pains occur at long intervals, and are deficient in intensity, so that labour is prolonged. Another factor in the prolongation of labour is early rupture of the membranes, and it may also be that much of the force of the contractions is expended uselessly in compressing the second sac. The average duration of labour in twin pregnancy recorded in the Royal Maternity Hospital, Belfast, was 12.6 hours for primiparæ, and 14.52 for multiparæ. The reason for the longer time taken by the latter is that probably the multiparous uterus possesses less tone, so that it cannot accommodate its contractile powers to the over-distension as well as the uterus of the primigravida.

As regards presentation of the fœtus, the frequency in the present series was as follows :—

| | | |
|---|---|---|
| First fœtus cephalic, second fœtus cephalic | - | 65 cases (40% approx.). |
| First fœtus cephalic, second fœtus pelvic | - | 42 cases (25% approx.). |
| First fœtus pelvic, second fœtus pelvic | - | 25 cases (15% approx.). |
| First fœtus pelvic, second fœtus cephalic | - | 24 cases (15% approx.). |
| First fœtus cephalic, second fœtus shoulder | - | 8 cases ( 5% approx.). |

From these figures it will be seen that in ninety-five per cent. of cases the lie of the fœtus was longitudinal. It follows, therefore, that in the actual conduct of labour, the "policy of non-intervention" should be adopted. In this series, the following operations or manipulations were carried out :—

Cæsarean section (six cases).—In two cases for cardiac disease, in two cases for pre-eclamptic toxæmia, in one case for contracted pelvis, and in the remaining case for "locked twins."

Forceps delivery of the first twin (three cases).—All for P.O.P.

Forceps delivery of the second twin (two cases).—In one case for P.O.P., and in the other for uterine inertia.

Forceps delivery of both twins (one case).—For eclampsia.

Internal version was performed on fifteen occasions, as follows :
For transverse lie and prolapse of the arm of the second twin (one case).
For transverse lie of the second twin (seven cases).
For prolapse of the cord of the first twin (one case).
For prolapse of the cord of the second twin (two cases).
For marginal placenta prævia on both fœtûs (one case).
For inertia, due to hydramnios, on both fœtûs (one case).

Labour should be left to proceed normally till the first twin is born. Munro Kerr,[8] however, is of the opinion that rupture of the membranes of the first sac is often of advantage, as it diminishes over-distension. When the maternal end of the cord has been effectively ligatured, the presentation and position of the second child should be determined by both abdominal and vaginal examination. Vaginal examination is essential to diagnose and treat presentation of the cord or of a limb. If the presentation is cephalic or pelvic, there is no indication for interference for at least twenty minutes. However, if pains with descent of the fœtus do not set in by the end of that time, the membranes should be ruptured. If uterine contractions do not recur forthwith, three minims of pituitary extract should be injected intramuscularly. If the second child is not born within a further twenty minutes, extraction should be performed immediately, either as a breech or with the aid of forceps. An alternative method dealing with the second fœtus is as follows :— If, on vaginal examination, the head is not in the pelvic cavity, internal version should be performed, and the fœtus extracted as a breech gently and without haste. This procedure is also carried out when the second fœtus is found to be lying transversely. I have practised this method of version and extraction as a method of

dealing with the second twin in many cases, and have not yet seen any difficulty arise as a result. It is an important measure in avoiding the risk of losing the child.

In this series, prolapse of the cord occurred in four cases, twice in the case of the first twin and twice in the case of the second. The most serious complication of labour is post-partum hæmorrhage, and this results from two factors—the over-distension of the uterus and uterine inertia during the third stage. The management of this stage of labour demands the greatest watchfulness, and immediately the patient should show signs of shock, the placentæ must be expelled, or manually removed if not yet separated. The pulse-rate should be continually noted, so that the placentæ may be dealt with at the first sign of rise, which is invariably the earliest sign of impending shock. Even in those cases when the third stage has passed off uneventfully, an injection of ergometrine should be given when it is completed, on account of the very real risk of relaxation of the uterus afterwards. Post-partum hæmorrhage of a severe degree occurred in this series, in 7.8 per cent. of the cases, and was responsible for the death of one patient.

Locked twins is another complication of labour which occurred once in this series. The head of the first fœtus was in the pelvic cavity, but was prevented from advancing by the head of the second child being wedged between the chin and chest of the first, so that the first head could not descend. The case was treated by Cæsarean section performed through the lower segment. This condition, although very fully described in all textbooks, is really a great rarity; it has been estimated to have occurred once in ninety thousand cases in two Vienna clinics (V. Braun, quoted by Munro Kerr[8]). Fairbairn[9] offers the following comment: "Locked twins are extremely rare, and the attention paid to them in textbooks seems to have arisen rather from the exciting pictures supplied by imaginative artists calling for adequate explanatory matter to justify their insertion, than the experience of authors in dealing with such cases." Although a great rarity, it will always remain the commonest and most important complication of twin labour in the mind of the medical student. Quite recently, however, two cases have been reported. Dawson[10] gives details of a case where the locking was due to the head of the second fœtus being in collision with the head and shoulder of the first. He treated the condition by lower segment Cæsarean section, both fœtûs being born alive. The second case is described by Preston,[11] who treated his case by pushing up the first child after failing to extract it with the cranioclast, and extracting the second child as a breech. He then performed internal version on the first child, which was now the second child, and delivered it also as a breech. This child was still-born.

### Maternal Mortality.

There were three maternal deaths in this series, i.e., 1.8 per cent., or 25.5 per thousand live births. The figures for all cases in the Belfast and Royal Maternity Hospitals (McClure[12]), over the last ten years, are:—(*a*) Maternal mortality, per cent.: 0.34 for ante-natal cases, and 0.92 for ante-natal and emergency cases; and (*b*) maternal mortality, per thousand live births: 4.47 for ante-natal cases, and

12.92 for ante-natal and emergency cases. The maternal mortality per thousand live births for 1936 for the County Borough of Belfast[13] was 5.7, and for Northern Ireland[14] was 7.3. It will be seen, therefore, that multiple pregnancy adds greatly to the ordinary risks of pregnancy. The first maternal death occurred in a patient, pregnant for the third time, who was admitted to hospital at thirty-seven weeks maturity, suffering from a severe degree of pre-eclamptic toxæmia. Labour was induced, and was completed in forty-eight hours. Following delivery, the patient developed signs and symptoms of acute yellow atrophy of the liver and died in three days. The second maternal death was that of a primigravida who was delivered easily after a spontaneous labour at thirty-eight weeks maturity. Post-partum hæmorrhage followed the delivery of the placentæ after a normal third stage, and the patient died from the resulting shock. The third fatal case was that of a primigravida who was suffering from pre-eclamptic toxæmia. Spontaneous labour at thirty-six weeks maturity lasted fifty-six hours, and although there was no post-partum hæmorrhage, severe obstetric shock supervened, from which the patient died in one hour after delivery.

### The Fœtus.

The total number of fœtûs born was 331, i.e., 164 cases of twin pregnancy and one case of triplets. The sex of the fœtûs, and the order in which they were delivered, were as follows :—

First male, second male—fifty-five cases, 110 babies.

First male, second female—forty cases, eighty babies.

First female, second female—thirty-five cases, seventy babies.

First female, second male—thirty-four cases, sixty-eight babies.

The triplets were two males and one female, in this order, so that in all the males numbered 186 and the females 145. In ninety-six of the total number of 165 cases, the first child was a male, while the first child was a female in sixty-nine cases.

Thirty-seven of the 331 fœtûs were born dead, twenty-four being still-born and thirteen macerated. The gross fœtal mortality is, therefore, 11.1 per cent., or 111 per thousand. Of the dead-born children, thirteen were macerated, one was an anencephalic monster, and in one case pregnancy was complicated by a marginal placenta prævia. In order to obtain the corrected fœtal mortality-rate, i.e., the mortality-rate for which multiple pregnancy is responsible, these sixteen cases, where no other factor was in whole or in part responsible, must be subtracted from the total number of dead-born fœtûs and from the total number of babies born. We therefore obtain the rate for which the multiple pregnancy was directly responsible, i.e., twenty-one dead-born fœtûs out of 315 babies born, 6.6 per cent., or sixty-six per thousand. These figures compare unfavourably with the general rate of forty-one per thousand (quoted by F. J. Browne), and with the gross fœtal mortality-rate of seventy-four per thousand, and the corrected fœtal mortality-rate of thirty-three per thousand in the Royal Maternity Hospital, Belfast, for 1936. In addition, they

compare unfavourably with the corrected fœtal mortality-rate of sixty-one per thousand[15] for breech delivery, which is probably one of the worst methods of delivery as regards fœtal mortality. It follows, therefore, that the risk to the child in multiple pregnancy is much greater than in single pregnancy.

The effect of maturity on fœtal mortality in this series is shown in the following table:—

| Maturity (weeks) | Babies born | Macerated | Still-born | Total dead-born babies | Gross mortality-rate (per cent.) |
|---|---|---|---|---|---|
| 28 | 14 | 1 | 7 | 8 | 57.1 |
| 30 | 20 | 2 | 1 | 3 | 15.0 |
| 31 | 4 | — | 1 | 1 | 25.0 |
| 32 | 18 | — | — | — | — |
| 33 | 4 | — | — | — | — |
| 34 | 16 | 2 | 2 | 4 | 25.0 |
| 35 | 6 | — | — | — | — |
| 36 | 62 | 2 | 5 | 7 | 11.2 |
| 37 | 7 | — | — | — | — |
| 38 | 60 | 4 | 1 | 5 | 8.3 |
| 39 | 6 | — | — | — | — |
| 40 | 114 | 2 | 7 | 9 | 7.8 |
| | 331 | 13 | 24 | 37 | 11.1 |

Both fœtûs were dead-born in eight cases; the first twin was dead-born in fourteen cases; and the second twin was dead-born in seven cases—total thirty-seven cases.

Twenty-three of the total number of male children were dead-born, i.e., 12.3 per cent., or 123 per thousand; and fourteen of the female children—9.6 per cent., or ninety-six per thousand, were dead-born. The difference in these figures may be explained by the fact that the first child was a boy in ninety-six of the 165 cases, i.e., 58.1 per cent.; being the first child it was subjected to more stress, and therefore bore more of the burden and heat of the day than the second, with a resultant increase in mortality-rate. Twenty-three of the dead-born fœtûs were delivered by the vertex, and fourteen by the breech.

### Fœtal Abnormalities.

One fœtus was an anencephalic monster; its twin was macerated. There was one case of double monster or conjoined twins. Double monsters, or symmetrical or asymmetrical disomata, are uni-ovular twins which have resulted from incomplete division of the common blastoderm. The case met with in this series was one of the three main varieties of symmetrical disomata, namely, thoracopagus, where the two trunks of the fœtûs are united, but the heads and the limbs are distinct. The patient was admitted to the hospital as a case of "failed forceps"; the head

was protruding from the vulva, but traction failed to deliver the trunk. Careful examination under anæsthesia revealed the presence of a second fœtus, joined by the entire length of the trunk in the middle line to the first. Division of the trunks was effected, the first child was extracted easily, and the second delivered as a breech after internal version had been performed.

The only other fœtal abnormality in the series was that known as *fœtus compressus* or *fœtus papyraceus*. In this condition one fœtus dies at an early stage of pregnancy, and is retained in the uterus while pregnancy continues. The dead fœtus becomes flattened out between the uterine wall and the membranes of the surviving child. In this instance, the *fœtus compressus* was palpated in the lower uterine segment, and was at first thought to be a placenta prævia. However, careful examination revealed that the mass was not a placenta, and it was not until after extraction of the other fœtus that its true nature was determined. The surviving fœtus was discharged from hospital in good condition.

## Summary.


1. A series of consecutive cases of multiple pregnancy is reviewed.
2. Maternal complications during pregnancy and labour are discussed.
3. Fœtal complications are reviewed.



I have to thank Professor C. G. Lowry and Mr. H. L. Hardy Greer for permission to use the data from which this article has been prepared.


## References.


1. Curtis, A. H., "Obstetrics and Gynæcology," 1934.
2. Dafoe, A. R., "Dionne Quintuplets," *Journal American Medical Association*, 1934, 103, 673.
3. Stander, H. J., "Williams' Obstetrics," Seventh Edition, 1936.
4. De Lee, J. B., "Principles and Practice of Obstetrics," Fifth Edition, 1929.
5. Das, K., *Journal of Obstetrics and Gynæcology of British Empire*, 1930, 37, 841.
6. Titus, P., "The Management of Obstetric Difficulties," 1937.
7. McIlroy, Dame Louise, "The Toxæmias of Pregnancy," 1936.
8. Kerr, J. M. Munro, "Operative Obstetrics," 1937.
9. Fairbairn, J. S., "Gynæcology with Obstetrics," 1924.
10. Dawson, J. B., *Journal of Obstetrics and Gynæcology of British Empire*, 1936, 43, 507.
11. Preston, P. G., *Journal of Obstetrics and Gynæcology of British Empire*, 1937, 44, 723.
12. McClure, H. I., "Maternal Mortality in Hospital Practice," 1937.
13. Report on the Health of the County Borough of Belfast, 1936.
14. The Registrar-General's Annual Report, Government of Northern Ireland, 1936. H.M. Stationery Office.
15. Macafee, C. H. G., and McClure, H. I., "A Critical Survey of 349 Cases of Breech Delivery," *B.M.J.*, 14th August, 1937.

# Obstetric Shock

*By* Doris B. Brown, B.Sc., M.B., F.R.C.S.
Obstetric Tutor, Queen's University, Belfast

Obstetric shock is one of the most serious of the accidents associated with childbirth. Puerperal sepsis is the only condition responsible for a larger proportion of maternal deaths. Out of 141 consecutive deaths occurring in the Belfast Maternity Hospital, and the Royal Maternity Hospital, Belfast, during the ten years, 1927-36, twenty-three, i.e., 16.3 per cent., were due to obstetric shock. More distressing still is the fact that twelve out of the twenty-three women who died had attended the hospital ante-natal clinic. As there were forty deaths in ante-natal cases, this means that thirty per cent. of these deaths were due to obstetric shock.

Table I.

COMPARISON OF DEATH-RATE IN ANTE-NATAL AND EMERGENCY CASES.

| | Ante-natal cases | Emergency cases | Total |
|---|---|---|---|
| Obstetric shock - - | 8.5% | 7.8% | 16.3% |
| Puerperal sepsis - - | 4.2% | 19.1% | 23.3% |
| Eclampsia - - | 0.7% | 10.0% | 10.7% |
| Heart disease - - | 2.2% | 8.5% | 10.7% |

From Table I it appears that obstetric shock is the only common cause of death that is not benefited by ante-natal supervision.

Apart from the fatal cases of obstetric shock, there is a much larger number whose lives hang in the balance for several hours.

Obstetric shock appears to be the same condition as surgical or traumatic shock, and is usually divided into two types—(*a*) primary shock, (*b*) secondary shock.[1]

Primary shock is usually attributed to afferent nervous impulses causing reflex vaso-dilatation and a profound fall in blood-pressure. This is due chiefly to dilatation of the capillaries of the skeletal muscles of the limbs. The patient may recover from this completely or temporarily, or pass directly into a state of secondary shock.

Secondary shock is little understood, and it is very uncertain what part afferent nervous impulses play, and to what extent vaso-dilator substances liberated from injured tissues can be held responsible.

Cannon and Bayliss were of the opinion that secondary shock was produced by some vaso-depressor chemical agent derived from injured tissues. They produced traumatic shock in anæsthetized animals by crushing the muscles and bones of the hind limb, and stated that the shock could be prevented if the venous return from the damaged area was occluded; also that division of the nerve-supply to the limb

did not prevent the development of shock. The vaso-depressor substance was thought to be histamine, because traumatic shock resembled the condition produced by poisonous doses of histamine, and also because histamine can be liberated at any rate from the skin by injury.

Doubt has recently been placed on these experiments and conclusions. Some workers say that very little shock may follow trauma if the afferent nerves of the limb have been severed and the blood-supply left intact. It has been suggested that in the experiments of Cannon and Bayliss the afferent limb-nerves were unable to transmit impulses, as they were deprived of their blood-supply. Also, it has not been possible to isolate histamine or a depressor substance in the blood leaving the traumatized limb. In addition, the appearance of the viscera in experimental shock is quite different from that in histamine poisoning. In the former the organs are pale and anæmic, and in the latter intensely congested. It seems, therefore, that in some way nocuous afferent impulses in injury do depress the circulation.

In experimental shock, loss of fluid from the circulation is found to be an important factor. This is caused by hæmorrhage from the cut vessels, and also by escape of fluid from vessels that have become more permeable but are not themselves ruptured.

This point is interesting in relation to obstetric shock, for in the twenty-three cases of shock mentioned above there was post-partum hæmorrhage in eighteen cases; and as two cases died before delivery, this means that ninety per cent. of the cases of obstetric shock were associated with excessive blood loss.

Three possible factors, therefore, are present in shock—

1. Reduction of blood volume.
2. Depressor afferent impulses.
3. Undetermined chemical depressor agents.

### Clinical Evidence of Shock.

A profound degree of shock is easily recognized. In the early stages it may easily be missed unless the blood-pressure is taken. In a well-marked case the patient lies still and takes no notice of her surroundings. Her limbs are flaccid. The skin is cold and pale, often with a slightly grey or bluish tinge, and is frequently covered with drops of sweat. The respirations are shallow. The pulse is running and of poor volume, and is usually rapid. The blood-pressure is low—it may even be too low to be recorded. This varies as the condition improves or deteriorates, and is the most important single point in the prognosis and treatment.

Shock is often associated with hæmorrhage. Loss of blood alone usually causes restlessness, and the patient is mentally alert and often anxious. The late stages of shock and hæmorrhage are the same. This is because shock is the result of paralysis

of the medullary centres due to over-stimulation, and excessive loss of blood leads to anæmia, and thus to paralysis of their centres.

The following fatal case of obstetric shock is instructive. The patient was a primigravida, aged 41, who was admitted with pre-eclamptic toxæmia when she was thirty-four weeks pregnant. She was treated by starvation, diaphoresis, and moderate purgation. Induction was advised soon after admission, but was refused until the thirty-seventh week. At this time the membranes were artificially ruptured. Labour commenced thirteen hours later, but the pains were poor. During the whole of labour the patient showed evidence of extreme fear, and insisted that she was going to die. She was also under the impression that people were talking about her and saying she was insane. This was counteracted to some extent by scopolamine amnesia. The first stage lasted sixty-seven hours, and forceps were applied one hour later. The head was high, but the delivery was not difficult. The placenta failed to separate, and was manually removed forty minutes later and the uterus curetted. This was easy, and the blood loss was, if anything, less than usual. The patient was by this time extremely shocked, with a systolic blood-pressure of 65 mm. Hg., but after warmth, morphia, coramine, and intravenous gum saline solution, her condition was greatly improved, and the systolic blood-pressure had risen to 114 mm. Hg. The blood-pressure remained at this level for about one hour, but then fell rapidly. More gum saline was given, and the blood was cross-typed with that of her husband. While the saline was being given the uterus relaxed and bled freely, and did not respond to stimulation by pituitrin, ergometrine, and massage. The patient died within five to ten minutes.

### Predisposing Factors in Shock.

Why one woman becomes shocked and another does not is unknown, but there are several well-marked predisposing factors. These are :—

1. *Hæmorrhage.*—This is the most important single factor, for although marked and fatal shock does occur without loss of blood, in most cases there is associated hæmorrhage. This may be ante-, intra-, or post-partum.

2. *Fear.*—This is a very potent factor in the production of shock. Many women have a profound fear of labour which they are usually unwilling to admit even to themselves. One result of this is that uterine contractions are weak and irregular, and the cervix may take many hours or even days to dilate. This results in excessive fatigue, with its associated evils. As a result, forceps are usually required, and post-partum hæmorrhage in these cases is not uncommon. Fear and anxiety alone can apparently cause death. The classical example is that recorded in the Bible : "And his daughter-in-law, Phinehas's wife, was with child, near to be delivered: and when she heard the tidings that the ark of God was taken, and that her father-in-law and her husband were dead, she bowed herself and travailed; for her pains came upon her. And about the time of her death the women that stood by her said unto her, Fear not; for thou hast born a son. But she answered not, neither did she regard it"—(I Samuel iv, 19, 20).

3. *Obstetric Interference.*—Profound shock is nearly always present in such conditions as inversion of the uterus, concealed accidental hæmorrhage, ruptured uterus, etc. Manual removal of the placenta is commonly associated with obstetric shock, although hæmorrhage is not always a factor. Out of our twenty-three fatal cases, manual removal was necessary in twelve, and excluding two cases who died undelivered and two who died with the placenta *in utero,* this means that in sixty-three per cent. of the fatal cases of obstetric shock the placenta had been removed manually.

Phillips[2] thinks that lacerations of the pelvic floor are associated with shock possibly due to the liberation of histamine from the damaged muscles. In most cases of shock, extensive lacerations do not seem to be present, but there is no doubt that difficult deliveries, e.g., high forceps, craniotomy, etc., are more liable to be followed by shock than cases which are easily delivered. Packing the vagina is highly productive of shock, and even suture of the perineum may make all the difference between life and death.

4. *Fatigue.*—There is no doubt that fatigue plays an important part in the production of shock.

5. *Starvation.*—It was frequently found during the Great War that deprivation of food and water strongly predisposed to shock. Great difficulty arises in this connection in the treatment of the next condition of—

6. *Toxæmia.*—Cases of pre-eclamptic toxæmia are not infrequently associated with shock. The toxæmia is not alone responsible; probably the starvation, purgation, and diaphoresis to which patients suffering from this complaint are subjected during treatment plays no small part in the production of shock. The capillary vessels are damaged in toxæmia, so if any stagnation occurs the fluid will leak out of the vessels into the tissues more readily than normal.

7. *Pain.*—Pain is no small factor in the production of shock. That pain can cause profound shock can be noted in nearly every case after a vigorous Crede's expression of the placenta, although some other factor is also concerned, as some degree of shock occurs even when the patient is anæsthetized. Shock is marked in severe cases of concealed accidental hæmorrhage. This is at least partially due to the increased intrauterine tension which produces pain, and the general condition of the patient immediately improves when the pressure is relieved, either by rupture of membranes or by Cæsarean section.

8. *Anæsthesia.*—Although in many cases the intelligent use of anæsthesia and analgesia diminishes shock, it may have exactly the opposite effect. In some cases, especially in hospital, analgesics such as scopolamine and the barbiturates cause extreme restlessness. As a result, the patient becomes fatigued and frequently chilled. Again, if the analgesia is commenced too soon, labour may be unduly prolonged. Hæmorrhage is also slightly more severe after analgesia.

Prolonged anæsthesia leads to shock. Chloroform is the most dangerous in this respect. Ether, during the actual administration, is safer, as it stimulates the blood-

pressure, but the fall afterwards may be great. Dale[3] has shown that chloroform and ether, but not nitrous oxide, sensitize the capillaries to the action of histamine, so that in unanæsthetized animals about ten times the dose of this drug is needed to produce an equivalent depression of blood-pressure. Nitrous oxide given skilfully does not produce shock, but this may occur if there is inexpert administration producing restlessness and cyanosis. There is much less danger from an anæsthetic which is too deep than from one which is too light; the latter is specially to be guarded against.

9. *Exposure.*—This causes chilling of the patient, and is very productive of shock. On the other hand, heating the patient until she sweats is almost worse, as it causes loss of fluid and also lowers the temperature.

10. *Poor general condition from any cause.*

TREATMENT OF SHOCK.

(*a*) *Prophylaxis.*—Prevention of shock is better than cure. Many cases of shock should be prevented by ante-natal supervision. The general condition can be improved by adequate food, exercise, and sleep. Toxæmia should be treated, but it is well to remember that the treatment of toxæmia by starvation and elimination may be more productive of shock than the toxæmia itself. If the toxæmia is of such a degree that a very restricted diet is considered necessary, the patient should be induced within a few days.

The chief value of ante-natal care in relation to the prevention of shock is the opportunity it provides for gaining the confidence of the patient, and so being able to reassure her that all is likely to be well. This is comparatively easy in private practice, but is apt to be neglected in the rush of a busy ante-natal clinic. If fear is eliminated, labour is likely to proceed rapidly, provided there is no gross abnormality. Emphasis should be laid on the fact that labour is a physiological process, and that most cases are perfectly normal. The modern tendency is to regard pregnancy and labour as some dangerous and terrifying experience that should be undertaken only once or twice in a lifetime. Delivery at home is probably much less alarming to a woman than in the strange surroundings of a hospital labour ward. It is difficult for a patient to approach delivery calmly after she has heard the cries of a woman in an adjacent bed.

Primigravidæ should be warned against discussing their condition with friends and relatives. Many multiparæ delight in relating to those pregnant for the first time the pain and horror of labour. The Church lays stress on the "great pain and peril of childbirth." The amount of discussion in the lay press about maternal mortality also has a profound effect on the attitude of a woman facing her first labour. The following is taken from a resolution by the British Medical Association, 1935: "Maternal mortality is a scientific and administrative problem which deserves careful and scientific study, but, in the experience of practising doctors, the publi-

city which it is receiving to-day is tending to terrify the child-bearing woman, and is, in itself, a cause of increased mortality."

Morphia should be given before ill-tidings of any description are conveyed to a recently delivered woman. It has been said that shock is more common after a lost battle than after a victory. She should not be told immediately that the baby is dead or deformed, nor should she be shown a child delivered by the face or one that is much moulded or marked by forceps. On the other hand, when a woman is delivered of a healthy child she should be shown it at the earliest opportunity—"A woman when she is in travail hath sorrow, because her hour is come: but as soon as she is delivered of the child, she remembereth no more the anguish, for joy that a man is born into the world"—(John xvi, 21).

Proper management of the delivery, especially the third stage, is more important than ante-natal care. If the first stage is prolonged, sedatives should be given, and the patient should not be left any longer than necessary in the second stage. If labour is prolonged, large amounts of glucose should be given.

About ninety per cent. of cases of shock are associated with excess hæmorrhage, either before or after the delivery of the placenta. Hæmorrhage cannot be prevented in all cases, but frequently it is associated with bad management in the third stage. The person in charge becomes impatient before the placenta is separated, and attempts at expression of the partially separated placenta are a potent cause of hæmorrhage. Even if the placenta is expressed completely, pieces of chorion are frequently left behind. There is some difference of opinion with regard to the management of the third stage. Some authorities advise control of the fundus, while others leave the uterus completely alone. If the outline of the uterus can be seen through the abdominal wall, there is no need to touch the uterus at all during the third stage; if not, the height of the fundus should be felt from time to time with the hand, but on no account should it be squeezed, grasped, or massaged. Only when the attendant is satisfied that the placenta is completely separated should it be expressed. No attempt to determine this should be made for at least twenty minutes after the birth of the child. Patience is the keynote of the safe conduct of the third stage. If general anæsthesia is necessary for delivery, the injection of from three to five units of pitocin counteracts the depressing effect of the anæsthetic on the uterine contractions and hastens the separation of the placenta.

If bleeding occurs during the third stage, this must be controlled. Hæmorrhage with the placenta *in utero* is always difficult to treat. Expression is a potent cause of shock, and if this is necessary because of hæmorrhage, the patient should first be anæsthetized. Each case must be treated on its own merits, and although manual removal of the placenta is usually fatal if the shock is severe, there is no use in attempting to treat the shock while the patient is bleeding badly. The wisest thing is to remove the placenta, either by expression after cord injection or manually before the shock is severe. Manual removal should be preceded by the administration of morphia. Any operative interference is highly dangerous if the systolic

blood-pressure is below 100 mm. Hg., and should be undertaken under gas and oxygen anæsthesia.

The vagina and perineum should not be sutured if any degree of shock is present.

(*b*) *Treatment of Established Shock.*—Shock is a temporary condition, and the aim in treatment is to keep the blood-pressure raised until the patient recovers.

Rest and warmth are the chief essentials. Rest is obtained by adequate dosage of morphia. The patient must be warm, but care should be taken that she is not overheated—this is at least as serious as not warming the patient sufficiently. It is often forgotten that blankets should be placed under as well as over the patient. It is not rare to see a woman covered with many layers of blanket, but who is lying on a cold mackintosh sheet possibly in a wet nightgown. If hot-water bottles are used, they should be covered and great care taken to prevent burns. A shocked patient burns very easily. Warmth and morphia alone are sufficient for a mild case of shock.

Movement of a shocked patient should be avoided as much as possible. Women with retention of the placenta should be treated in their own homes, and not transported to hospital from their warm beds, possibly in a cold, draughty, jolty ambulance. In many cases the transport of the patient removes any chance of recovery she may originally have had. If she has to be moved at all, this must be done as gently as possible. It has been shown that even turning a patient over lowers the blood-pressure 10 mm. Hg.[3]

The blood-volume must be restored. In mild cases hot drinks or rectal salines may be sufficient. In the more severe cases the volume should be restored by intravenous fluid. If blood is not immediately available, gum saline is probably the most efficacious. The fluid should be given at a temperature of 105 to 110° F., and should be run in very slowly, especially where shock is not associated with hæmorrhage. The deeper and more lasting the shock, the more slowly must the fluid be injected, or there will inevitably be a subsequent drop in pressure. This fact is very difficult to remember. One pint of fluid is quite sufficient at one time, but this may need to be repeated. Too large amounts of fluid probably do more harm than good by diluting the peripheral blood. The only safe guide to transfusion is repeated blood-pressure readings. Intravenous transfusions should be given early, as it was found during the war that in the late stages of shock the capillaries are so damaged that they will not even retain gum saline. Blood-transfusion is of very great value, especially when blood has been lost.

It is doubtful if raising the foot of the bed is of very great value. It is only if the blood is in the large veins that this can be of use, and although this may occur to some extent in primary shock, it does not in secondary shock. This position is also very uncomfortable for the patient. With the same object in view a tight abdominal binder after delivery is useful, but if the patient is bleeding it causes difficulty in controlling the fundus. Binding the lower limbs very tightly over cotton-wool is much more useful. The person in charge must watch the condition

of the toes carefully, and the bandages must be removed in a few hours and immediately there is any evidence of defective circulation in the toes. Gangrene of the feet has occurred from the abuse of this very valuable therapeutic measure.

Drugs, other than morphia, are of less value in the treatment of shock. Strychnine mainly raises the blood-pressure by stimulating the vasomotor centres in the medulla, and these in a condition of shock are inhibited or exhausted by over-stimulation. Strychnine in mild degrees of shock will raise the blood-pressure, but the effect is temporary, and is followed by greater depression than before owing to the further exhaustion of the centres. The use of strychnine in shock is, therefore, definitely harmful. The same may be said for caffeine, ether, etc. Coramine is probably of more value, as it is a direct respiratory stimulant.

Adrenalin has often been used, as it causes vaso-constriction in the shocked animal, and its action is apparent even in an animal whose brain has been destroyed by pithing. The effect, however, is transitory, and ephedrine, which has a slower but similar action, would seem to be more useful. Phillips[2] says that adrenalin, although causing vaso-constriction in the skin-vessels, also causes an increase in the volume of blood in the voluntary muscles and in the intestine. If this is so, the use of adrenalin in shock is definitely contra-indicated. Pituitrin is useful, as it causes a marked vaso-constriction by peripheral action. It is of added value if uterine hæmorrhage is also present, and should be given in combination with ergometrine in post-partum hæmorrhage. "Post-pituitary shock" is described. This is a condition of shock which may follow immediately upon a dose of pituitrin. It is particularly liable to occur if the dose is repeated within one hour. The shock is thought to be due to intense spasm of the coronary arteries possibly due to some impurity. Pitocin, being free from any pressor principle, is said not to cause shock. The treatment is to give adrenalin.

The patient who is shocked or has bled after delivery must not be left for several hours. Far more patients die of shock two to six hours after delivery than before this period. Miles Phillips has emphasized the fact that the only true guide to the condition of the patient is the systolic blood-pressure. This should be taken at least every half-hour for several hours, and any fall should be met by restorative measures.

I have to thank Professor C. G. Lowry and Mr. H. L. Hardy Greer for access to the case records of their patients, and Mr. H. I. McClure for supplying the figures from which Table I was compiled and for his advice and criticism.

## References.

1. Wright, Samson, "Applied Physiology." Oxford Medical Publications, 1936.
2. Phillips, Miles H., *British Medical Journal,* 16th May, 1931.
3. Cannon, W. B., "Traumatic Shock." Appleton.

# The Dispensary Doctor

*By* W. LYLE, M.B.

"SOLDIER, sailor, tinker, tailor"—the Irish dispensary doctor has to be a veritable "jack of all trades." Situated, as he often is, remote from any professional assistance, he must needs be somewhat of a universal specialist—surgeon, physician, obstetrician, gynæcologist, anæsthetist, and expert in sanitary science, for he is medical officer of health for his district, all in one person, not to speak of his other activities, for he is his own dispenser, and also, in most cases, registrar of births, deaths, and marriages.

He is on duty for twenty-four hours a day, for seven days a week, during from forty-eight to fifty weeks a year, and is at the beck and call of every Tom, Dick, and Harry who can get a ticket to commandeer his services; and it is not difficult to obtain these, seeing that they are on issue by wardens and guardians who are, in many cases, grocers or publicans, and who dispense them as bonuses with quarter pounds of tea or botles of stout, irrespective of the medical needs or financial circumstances of the applicant.

He is confined, more or less as a prisoner, within the boundaries of his district, and woe betide him if he happens to have strayed outside those boundaries when his services are called upon for some patient who is, not infrequently, in as good physical health as the doctor himself.

He sees with envy his more fortunate neighbour, the bank official, the civil servant, or the teacher in the public elementary school, occupy his ample leisure with rod or gun, tennis racket or golf clubs, whilst any time which he may have to spare from his official duties is necessarily taken up with the attempt to earn a living for himself and his family by private practice, his official salary leaving but little margin after his necessary professional expenses are defrayed.

This salary, though greater in amount than when the service was initiated in 1851, is less in actual purchasing power, and his work has greatly increased since then in spite of a large fall in population. This is due to several causes, the chief of which are the gradual elimination of the small farmer, who employed no labour outside of his own family, and who was a private patient paying moderate fees for attendance on himself and his household, and his replacement by the larger farmer employing several labourers, all of whose dependants are poor law patients; the absence of any definition of a "poor person" in the Act of Parliament, which entails upon the dispensary doctor the duty of attending free of charge all "poor persons," and the steadily increasing tendency of that section of the population somewhat above the poverty line to seek free medical attendance, as well as the equally steadily increasing tendency of those members of the proletariat who are, or think they are, ill, to send for the doctor instead of going to his dispensary to consult him.

For many years after the dispensary system was introduced, the proportion of

tickets issued requiring the dispensary doctor's attendance at the homes of the patients, to those requiring the patients to go to the dispensaries for advice and medicine, was as one to two or three, whereas to-day the proportion is reversed, and at least twice as many of his poor law patients send for the doctor to visit them as come to him at his dispensary, and the majority of these send for him late in the day or after night, necessitating many entirely unnecessary journeys. It is no unusual thing for a dispensary doctor in a rural area to travel along the same road three or four times in one day to visit patients, for all of whom the messages could quite reasonably have been delivered to him before he started on his morning round, and who in many cases were as able to go to the doctor as he to them.

For the registration of births, deaths, and marriages, the dispensary doctor to-day receives exactly the same remuneration as when registration started, about seventy-five years ago, in spite of the greatly decreased value of money and greatly increased cost of living. Neither has the meagre salary he receives as medical officer of health ever been increasd, and for a pittance of ten or fifteen pounds a year he is compelled to act as such in a district of thirty, forty, or more square miles, and if he faithfully performs his duty in the prevention of disease, as he in most cases does, by insisting upon owners keeping their properties in efficient sanitary condition, he loses much more in fees from private patients than he receives as salary, owing to the offence he so often gives to property owners who are his patients—often, indeed his masters, as the worst offenders are frequently members of the local board of guardians, whom he thus not only loses as patients, but antagonizes to such an extent that they become his bitter enemies and take every opportunity to vent their spite when occasion arises.

There has been no attempt on the part of the Ministry of Home Affairs, which controls the administration by the Boards of Guardians of the Poor Law, to improve the conditions under which the dispensary doctor works, since our present Government started its career in 1920, previous to which the Local Government Board was making a genuine effort to do so, and had compelled the majority of the boards of guardians in Northern Ireland—in extreme cases by the issue of sealed orders—to pay largely increased salaries, and was using its authority over these irresponsible bodies to minimize the abuse by guardians and wardens of the issue of tickets for poor law medical relief to people in a position to pay the very moderate fees charged to people of this class.

The Local Government Board, after many years of neglect or indifference, was showing its sympathy with the dispensary doctor and its consideration for the trying and difficult conditions under which he was obliged to work.

On the other hand, the Ministry of Home Affairs in the Northern Ireland Government has consistently displayed an utter want of sympathy and lack of consideration, and has materially increased his burdens and diminished his fees. It has never made the slightest attempt to complete the work, upon which the Local Government Board was engaged, of compelling the few recalcitrant unions to pay the salaries standardized throughout the province.

The dispensary system has been in operation for nearly ninety years without any material change or modification. Started to cope with abnormal conditions due to famine and pestilence, it is still applied to a country in which such conditions are now merely an unhappy memory, and in which the material prosperity of all classes has progressively increased and is still increasing.

The only change in the system of any importance has been a change for the worse—the substitution of the board of guardians for a local committee in each dispensary district composed of residents in that district to appoint the doctor and supervise his work.

The local committee worked well and was in close touch with the doctor and his work; and it was in the interests of its members to choose the best qualified man available, as in many districts they were dependent on the doctor appointed by them for medical attendance to themselves and their families. The board of guardians, on the contrary, has no such incentive to appoint the best man, and is influenced by political and religious motives rather than by any desire for efficiency.

The dispensary system is an anachronism, and should be discontinued, with adequate compensation to the doctors for loss of office, and there should be substituted for it a system under which dependants of the insured and other poor persons should have free choice of doctor, with the doctor paid by capitation as in the National Health Insurance scheme, whilst the duties of the dispensary doctor as medical officer of health for his district should be performed by a whole-time assistant to a county medical officer of health.

Thus would we "keep step with England."

In conclusion, I would quote *in extenso* the generous appreciation of the dispensary doctor by the editor of the "Irish News," in its issue of 20th July, as follows :

"The dispensary medical officer may not have the glamour of the surgeon or the renown of the specialist. He may be one of the forgotten men of medicine, moving obscurely about a country district on an unremitting round of duty, night and day, in fine weather and in foul, his name achieving the accolade of print but once a year, when he applies for his annual holidays to the local board, but ultimately he is the guardian of the health of the people.

"Spectacular operations, unique treatments, lightning diagnosis, may catch the public eye. We may talk in an awed hush of the wonders of modern medicine, but we are so intent on the breath-taking exceptions that we are apt to forget the man with the little bag on his prosaic round.

"These men often lead an arduous life, harassed by circumstances, red tape, ignorance, but on the whole they put a lot more into their work than they ever get out of it, do a lot more than they are ever thanked for, perform wonders in adversity that are forgotten as soon as they are done.

"They are the servants of the poor, outposts against disease, indispensable units of our social organization."

# CASE REPORT

## A CASE OF SEPARATION OF THE LOWER FEMORAL EPIPHYSIS, PRODUCED BY AN OBSTETRICAL INJURY AND RESULTING IN GANGRENE OF THE TOES.

*By* R. J. W. WITHERS, M.D., M.CH., F.R.C.S.ED.
*Crumpsall Hospital, Manchester*

ON 12th February, 1937, in the maternity department of this hospital, a primigravida, aged 30, having been in labour for two days, gave birth to a female child.

The child was born by the breech, and the delivery was extremely difficult. I cannot ascertain just how the baby was delivered, but it is undoubted that extreme force was required to extricate the infant from its unasked-for position.

After birth it was discovered that the infant's right clavicle was fractured in its middle part, and a peculiar deformity of the right knee-joint was noticed.

Three days after birth I was asked to see the case. The toes of the right foot were found to be blue, and colder than the opposite side, and ulceration on the dorsum of the foot had commenced. On examination of the knee it became evident that the lower epiphysis of the femur had separated and was pressing backwards in the popliteal fossa. Fine, silky crepitus was obtained on attempting to move the knee, and the child resented any such attempt in the usual way. It was also noticed that the child did not move the right leg freely. Pulsation in the popliteal artery was good, but none could be felt round the ankle-joint.

An X-ray examination of the right knee-joint showed a displacement of the lower epiphysis of the femur backwards and slightly to the lateral side. The bony nucleus of the epiphysis was well developed.

On the day of examination, i.e., three days after the injury, the epiphysis was replaced and the leg suspended from the end of the bed by strapping. X-ray examination showed perfect reduction of the separation.

Unfortunately, the gangrene of the toes, which was showing its early presence on the first examination, progressed, and some ten days afterwards the toes sloughed off at the metatarso-phalangeal joints. Within two weeks of this occurrence a healthy granulating surface presented, and this was skin-grafted, using the inner lining of the prepuce from three circumcised male infants.

The child is now six months of age, and is healthy in every other way. The foot has completely healed up, movements of the knee and ankle-joint are normal, and the foot is half an inch to three-quarters of an inch shorter than its fellow. The fractured clavicle has united without deformity.

Further X-ray examination shows a fair amount of subperiosteal new bone formation at the lower end of the femur, extending upwards from the epiphyseal line to above the middle of the shaft (see X-ray plates). The epiphysis itself appears normal.

The mother's Wassermann reaction is negative.

This, then, is a case of displacement of the lower femoral epiphysis, presumably as a result of an obstetrical trauma.

Poland, in his book on "Traumatic Separation of the Epiphyses," has analyzed seventy-one cases of separated lower femoral epiphyses at all ages, and not one is described as occurring at birth, although he has been able to discover a case in a fœtus of six months.

Massart in 1921, before the Anatomical Society of Paris, described a case in which the lower femoral epiphysis on both sides had become separated as the result of an obstetrical injury, the child being born by the breech. The X-ray examination of his case gave a somewhat similar appearance to this one, in that, in both cases, there was a large amount of subperiosteal bone formation at the lower half of the femur. In Massart's case this was so marked as to produce a firm bony swelling which could be easily felt by the examining hand.

Whilst in the cases occurring during adolescence, both direct and indirect traumata have been evoked as causative, as long ago as 1868 Colignon at Paris showed that violent torsion was the essential cause in separations occurring during the process of birth.

The displacement of the epiphysis in this case is quite opposite to what one usually sees in the adolescent, since here forward displacement of the epiphysis is the most usual occurrence.

I can find no case in the literature of displaced epiphysis in the new-born which developed gangrene of the toes.

Detachment of the periosteum with new bone formation seems to be a common, if not almost constant, occurrence. Injury to the popliteal vessels would appear to be a most likely complication to occur, but it is surprising how often these are left uninjured.

Little and Tapret note absence of pulsation in the posterior tibial artery without actual gangrene of the foot occurring. It does, indeed, appear definite that gangrene of the foot may occur without rupture of the popliteal vessels taking place.

Union appears to occur very quickly, as in this case, and is usual in simple separations. In unreduced separations, ankylosis of the knee-joint has been described. With the displacement of the epiphysis, even if careful reposition is carried out, it is likely that some degree of varus or valgus deformity of the knee-joint will be inevitable through injury to the epiphyseal cartilage, either during the production of the separation or on its replacement. Arrest of growth, producing a short thigh, is, as one might expect, a possible complication.

## Summary.

The points in this case are :—

1. A separation of the lower femoral epiphysis in a new-born child, the injury being called obstetrical for want of a better name.

2. The rapid onset of gangrene of the toes without any obvious interference with the popliteal vessels themselves, and also although early reposition of the epiphysis was carried out.

3. It would appear likely, from looking back on this case, that the possible

explanation of the gangrene was a peripheral vasospasm produced by two factors :

(*a*) The irritation of the sympathetic nerve plexuses on the adventitia of the popliteal artery; and

(*b*) The pressure on the posterior tibial nerve stimulating its contained sympathetic fibres which supply the posterior tibial and plantar vessels peripherally.


REFERENCES.

POLAND, "Traumatic Separation of the Epiphyses."

MASSART, R., *"Décollement épiphysaire de l'extrémité inférieure des deux fémurs consécutif à un traumatisme obstétrical," Bull. et Mêm. Soc. Anat. de Paris,* 1921.


# TWO CASES OF GRANULOSA CELL TUMOUR OF THE OVARY


*By* C. H. G. MACAFEE, M.B., F.R.C.S., F.C.O.G.


THESE tumours, while not so rare as previously thought, are sufficiently uncommon to warrant particular attention. They belong to a group of ovarian tumours which are of particular interest on account of their biological properties.

The two cases described below present the typical biological and pathological changes associated with these tumours. It is of interest to state that in the second case, Professor Young in his report suggested what the patient's clinical history should be as the result of his histological examination. His suggested clinical history corresponded almost exactly with that given by the patient.

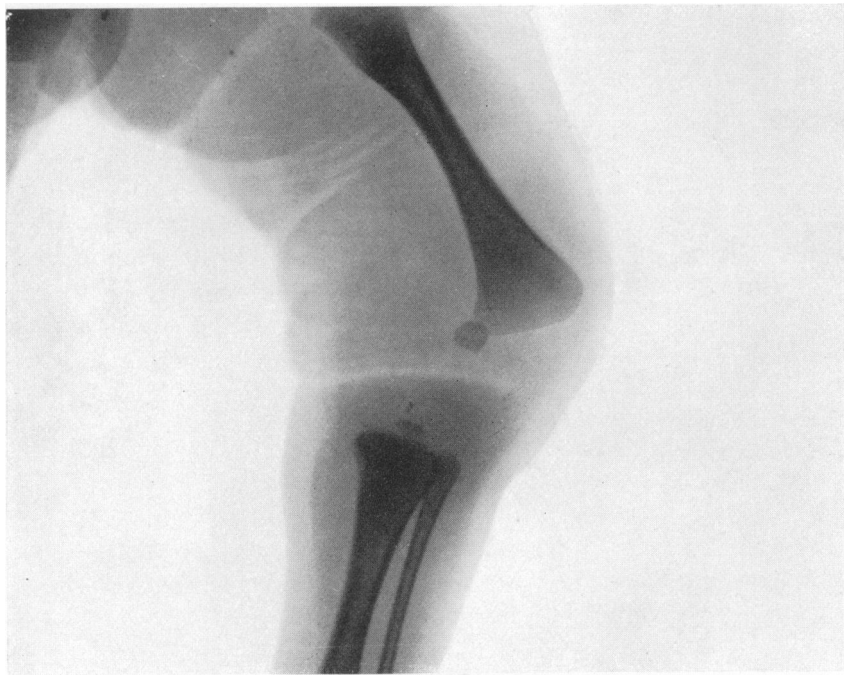
CASE 1. Aged 58. Three children; youngest 23; one miscarriage (the first pregnancy).

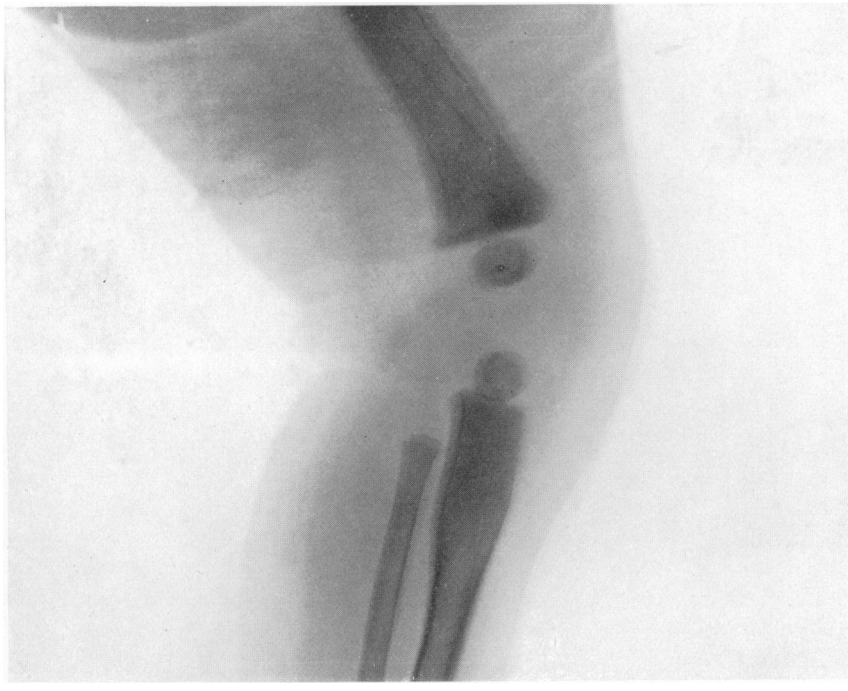
*Clinical History.*—14th June, 1934—For the past four years the menstrual periods had been irregular. Previously 3/31; moderate loss, no dysmenorrhœa. Since March, 1934, had been complaining of constant bleeding. On examination there was a profuse uterine loss; the uterus was in good position, definitely enlarged, and a hard swelling was felt in the left fornix. The palpable swelling was thought to be a fibroid.

4th January, 1935—Admitted to the Royal Victoria Hospital. General condition good. No notable anæmia. Vaginal examination as above.

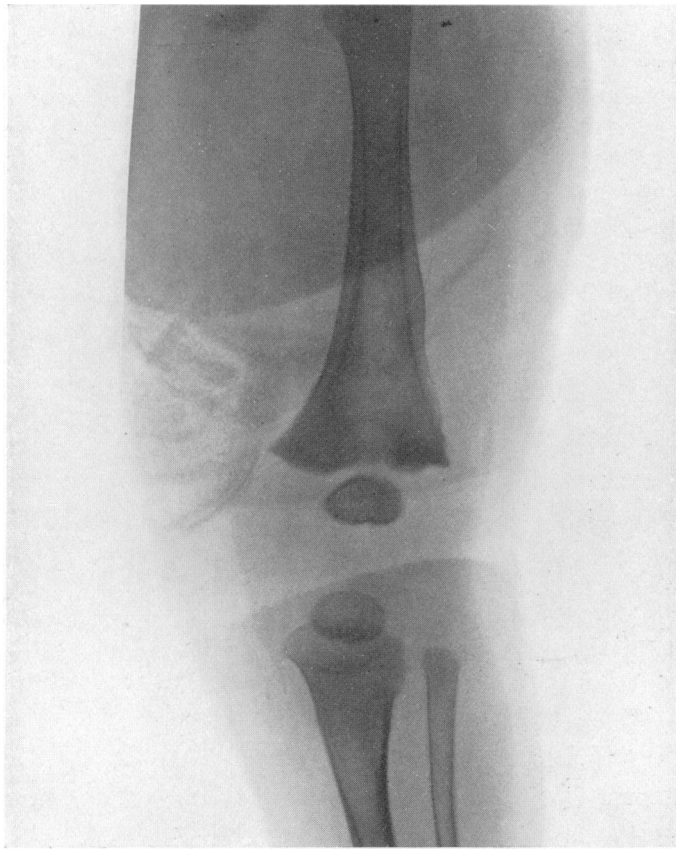
9th January, 1935—Operation, abdominal section. There was a small fibroid in the anterior wall of the uterus, which was enlarged. Both ovaries were enlarged to about the size of a large Brazil nut, firmer than normal, of a pale yellow colour and quite smooth. The tumour palpated in the left fornix proved to be the left ovary. Total hysterectomy, removing both ovaries, was performed.

*Description of Specimen.*—The left ovary is increased in size, and is largely replaced by a cellular tumour of yellowish appearance, which contains an irregular





## Dr. R. J. W. Withers' Paper



explanation of the gangrene was a peripheral vasospasm produced by two factors :

(*a*) The irritation of the sympathetic nerve plexuses on the adventitia of the popliteal artery; and

(*b*) The pressure on the posterior tibial nerve stimulating its contained sympathetic fibres which supply the posterior tibial and plantar vessels peripherally.


REFERENCES.

POLAND, "Traumatic Separation of the Epiphyses."

MASSART, R., *"Décollement épiphysaire de l'extrémité inférieure des deux fémurs consécutif à un traumatisme obstétrical," Bull. et Mêm. Soc. Anat. de Paris,* 1921.


# TWO CASES OF GRANULOSA CELL TUMOUR OF THE OVARY


*By* C. H. G. MACAFEE, M.B., F.R.C.S., F.C.O.G.


THESE tumours, while not so rare as previously thought, are sufficiently uncommon to warrant particular attention. They belong to a group of ovarian tumours which are of particular interest on account of their biological properties.

The two cases described below present the typical biological and pathological changes associated with these tumours. It is of interest to state that in the second case, Professor Young in his report suggested what the patient's clinical history should be as the result of his histological examination. His suggested clinical history corresponded almost exactly with that given by the patient.

CASE 1. Aged 58. Three children; youngest 23; one miscarriage (the first pregnancy).

*Clinical History.*—14th June, 1934—For the past four years the menstrual periods had been irregular. Previously 3/31; moderate loss, no dysmenorrhœa. Since March, 1934, had been complaining of constant bleeding. On examination there was a profuse uterine loss; the uterus was in good position, definitely enlarged, and a hard swelling was felt in the left fornix. The palpable swelling was thought to be a fibroid.

4th January, 1935—Admitted to the Royal Victoria Hospital. General condition good. No notable anæmia. Vaginal examination as above.

9th January, 1935—Operation, abdominal section. There was a small fibroid in the anterior wall of the uterus, which was enlarged. Both ovaries were enlarged to about the size of a large Brazil nut, firmer than normal, of a pale yellow colour and quite smooth. The tumour palpated in the left fornix proved to be the left ovary. Total hysterectomy, removing both ovaries, was performed.

*Description of Specimen.*—The left ovary is increased in size, and is largely replaced by a cellular tumour of yellowish appearance, which contains an irregular

cavity. The right ovary is also enlarged to a less extent, and presents a similar cellular appearance. Two small polypi can be seen in the cervical canal.

*Histology.*—The tumour of the ovary is highly cellular. Its structure is variable, comprising spindle and spheroidal cells for the most part, but also ill-formed tubular structures lined by cubical epithelium. In addition, there are numerous spaces more or less completely filled with small spheroidal cells bearing a very notable resemblance to lutein cells (see micro-photographs, case 1).

The appearances conform to those of a benign granulosa cell tumour. The cervix is the seat of a chronic endocervicitis, and exhibits a very marked hyperplasia of an irregular type affecting its glandular epithelium. There is also a small cervical polypus, which shows extremely irregular hyperplasia of its glandular epithelium. There is, however, no evidence of invasive growth.

CASE 2. Aged 54.

*Clinical History.*—Married thirty-three years. Seven children and two abortions. Last pregnancy in 1927, terminating in abortion at the third month. General health fair; troubled with bronchitis. Menopause three years ago, i.e., at 51.

4th January, 1937—Complained that "the periods have come back at intervals for the past six months with slight staining." Also complained of a swelling in the abdomen, backache, and general weakness for a few months. The abdominal swelling had been noticed only two weeks previously.

On examination there was a large rounded swelling about the size of a melon rising out of the pelvis which had the clinical characteristics of an ovarian cyst. On pelvic examination the uterus was generally enlarged, and the cervix was bulky, containing Nabothian follicles.

7th January, 1937—Operation, abdominal section. A large tumour (see description below) arising from the left ovary was present. The right ovary seemed normal. The uterus was generally enlarged. There was no free fluid in the peritoneal cavity. A total hysterectomy was performed, removing the tumour and the right ovary.

*Description of Ovarian Tumour.*—The tumour weighed five pounds seven ounces. Its outer surface is quite smooth. On section, it exhibits a rather cellular structure, but is studded with innumerable cystic spaces of variable size. Widespread hæmorrhage has occurred in the central part of the growth.

*Histology.*—The structure of the growth is variable, sometimes alveolar, sometimes papillary, and occasionally more glandular. The cells are spheroidal, rather small in size, with more or less vesicular nuclei, but they are remarkably uniform in their shape, size, and nuclear content. They bear a notable resemblance to granulosa cells. The tumour is so cellular that there must be some doubt as to whether it is completely benign, but the prognosis should probably be favourable.

UTERUS.—The uterus has been bisected in the sagittal plane (see photograph). It is moderately enlarged. The endometrium is thickened but not notably congested. Cystic dilatation of the endometrial glands has occurred in the distal half. Cystic dilatation is also evident in the cervical glands. The right ovary, which has been cut across, is strophic.

*Histology*.—The glandular elements of the endometrium are very actively hyperplastic, and the stroma is congested.

I am indebted to Professor J. S. Young for the description of the above specimens, and for the use of the photographs which accompany this communication.

In both cases the common feature in the clinical history was uterine hæmorrhage. In the first case the menopause had not occurred, but was late, as the patient was fifty-eight years old. In the second case "the periods had returned" after the menopause.

In both cases, but particularly marked in the second, the endometrium was actively hyperplastic at a stage of life when it should have been atrophic. This hyperplasia of the endometrium is a common, but not invariable, characteristic in association with granulosa cell tumours, and is due to production of excessive amounts of œstrin in the tumour.

According to some authorities, these tumours occur most frequently in elderly women, but Novak and Brawner,[1] who have described a series of thirty-six cases, found that only six patients were past the menopause, although ten were in the fifth decade. In the series of seven cases described by Lepper, Baker, and Vaux,[2] the age of the patients varied from 23 to 60 years.

As mentioned above, the interest of these tumours lies in the biological effects produced by them. When the tumours are present before puberty, or after the menopause, these effects are more manifest. For example, the presence of the tumour before puberty is associated with the precocious onset of menstruation, appearance of secondary sex characteristics, and marked breast changes, all of which disappear on removal of the tumour. These changes are also due to overproduction of œstrin by the tumour.

When the tumour occurs after the menopause, it is associated with uterine bleeding, which varies in amount but may be rhythmical in character, and the patient usually states that "the periods have returned."

The common cause of post-menopausal uterine hæmorrhage is an adenocarcinoma, and a diagnostic curettage is necessary to make the diagnosis. Novak and Gray[3] state : "If, in a case of uterine bleeding occurring after the menopause, the diagnostic curettage yields a typical endometrial hyperplasia, one may strongly suspect the presence of a granulosa cell tumour of the ovary, even if palpation is negative, as it may be if the tumour is very small or the patient very stout. If, on the other hand, a tumour can actually be felt in such a patient, there is little doubt of its granulosal nature."

In Case 1, the tumour which was found on examination was thought to be a fibroid, while in Case 2 the nature of the tumour might have been suspected from the findings on examination and the clinical history.

When the tumour occurs during the reproductive period of life, it may not produce marked symptoms apart from those associated with any ovarian tumour, but many patients show excessive menstrual loss or excessive bleeding following a period of amenorrhœa.

*Malignancy*.—The degree of malignancy of these tumours varies, but it has

been stated that five to ten per cent. of them are actively malignant. Novak and Brawner[1] in their series found that twenty-eight per cent. were malignant.

In both cases described the patients are well, one after two years and the other after five months.


REFERENCES.

1. NOVAK, EMIL, AND BRAWNER, JAMES N., *Amer. Journ. Obstet. and Gynæ.*, 1934, Vol. 28, p. 637.
2. LEPPER, E. H., BAKER, A. H., AND VAUX, D. M., *Proc. Roy. Soc. Med.*, June, 1932, Vol. 25 (Sect. Obstet. and Gynæ., p. 45).
3. NOVAK, EMIL, AND GRAY, LAMAN A., *Amer. Journ. Obstet. and Gynæ.*, 1936, Vol. 31, p. 213.


# ROYAL MATERNITY HOSPITAL: REGISTRAR'S REPORT FOR THE YEAR 1936.

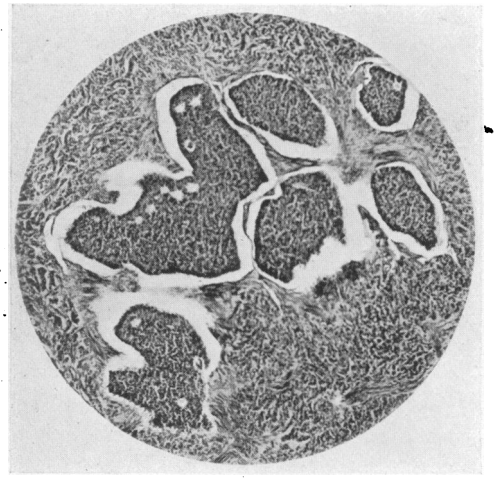
RETURNING to its annual form, this report contains much of interest. Statistics, like experience itself, may be fallacious, and it is well to try and grasp something of what lies behind them. So that while the reader will find more to interest him in the section "Details of Cases," the reviewer must give some attention to the figures with which the report begins.

The Rea Unit, admitting established cases already septic, continues to pervade and complicate the whole report. As it is in fact segregated from the hospital, so we would counsel the registrar to segregate it finally into a separate section of his report. Much simplification would result immediately.

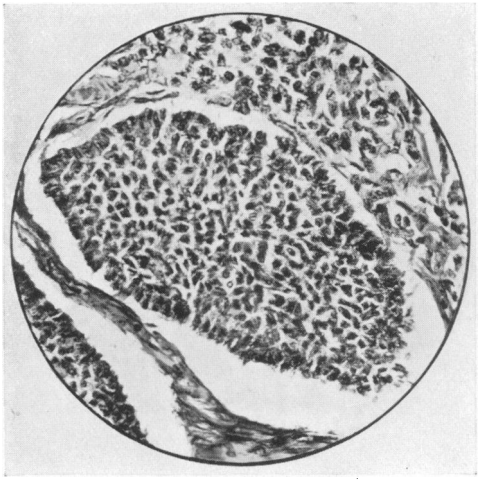
Table I (of general statistics) shows, in round figures, that 1,700 in-patients were admitted. This figure includes four houndred emergency cases, and, of this four hundred, two hundred were frankly septic on admission to the Rea Unit. From the hospital's own Ante-natal Department comes 1,300 cases; but we prefer the terms "booked" and "unbooked" as being unequivocal. Among the total of 1,193 babies born, we find the "ante-natal live births" phenomena hard to visualize.

Table II deals with maternal mortality. The figure of 0.31 per cent. for 1,300 booked cases is excellent. The percentage mortality for all admissions is four times, and for unbooked admissions is fourteen times, as great. (The corresponding figures of last year's report we found to be in the proportion of one, three, and seven.) Even more interesting, however, is the departure, new to this report, of calculating the maternal mortalities per thousand live births. There is, of course, a strong prima facie case for calculating maternal mortality according to the method of the Registrar-General's report, but the reader must beware of assuming that the figures emanating from a hospital such as this are comparable to those of the community.

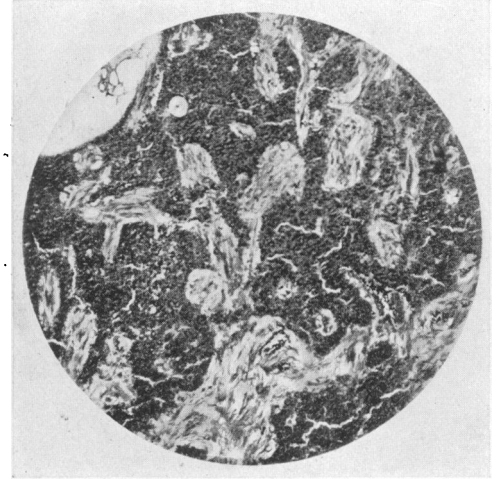
That this is not the case is suggested by what was, to our mind, the most striking or conspicuous figure in this whole table. The figure for maternal mortality



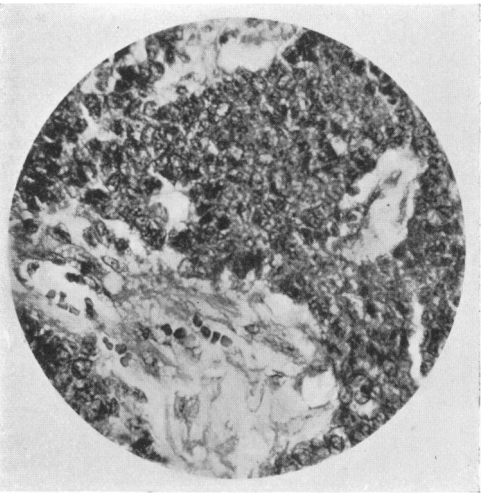
CASE 1—Ovarian tumour.
( × 65)



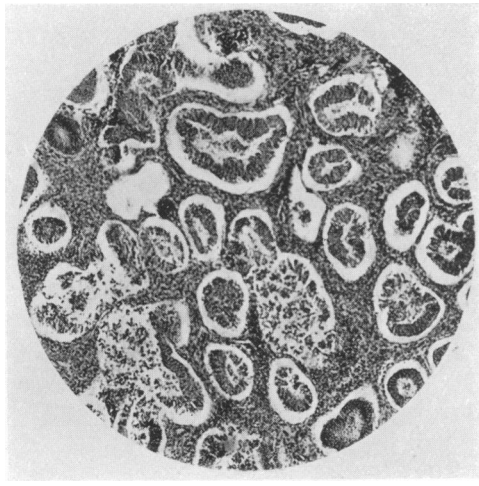
CASE 1—Ovarian tumour.
( × 300)



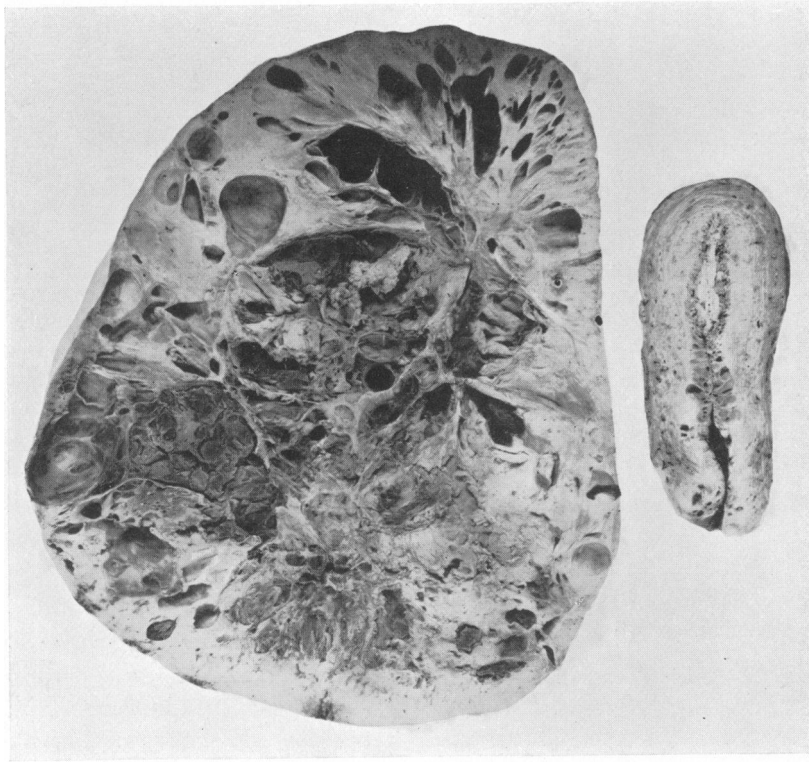
CASE 2—Ovarian tumour.
( × 65)



CASE 2—Ovarian tumour.
( × 300)



CASE 2—Hyperplasia of endometrium.
( × 65)



CASE 2
Granulosa of cell-tumour of ovary. Uterus in sagittal section.
( × 3/7)

been stated that five to ten per cent. of them are actively malignant. Novak and Brawner[1] in their series found that twenty-eight per cent. were malignant.

In both cases described the patients are well, one after two years and the other after five months.


REFERENCES.

1. NOVAK, EMIL, AND BRAWNER, JAMES N., *Amer. Journ. Obstet. and Gynæ.*, 1934, Vol. 28, p. 637.
2. LEPPER, E. H., BAKER, A. H., AND VAUX, D. M., *Proc. Roy. Soc. Med.*, June, 1932, Vol. 25 (Sect. Obstet. and Gynæ., p. 45).
3. NOVAK, EMIL, AND GRAY, LAMAN A., *Amer. Journ. Obstet. and Gynæ.*, 1936, Vol. 31, p. 213.


# ROYAL MATERNITY HOSPITAL: REGISTRAR'S REPORT FOR THE YEAR 1936.

RETURNING to its annual form, this report contains much of interest. Statistics, like experience itself, may be fallacious, and it is well to try and grasp something of what lies behind them. So that while the reader will find more to interest him in the section "Details of Cases," the reviewer must give some attention to the figures with which the report begins.

The Rea Unit, admitting established cases already septic, continues to pervade and complicate the whole report. As it is in fact segregated from the hospital, so we would counsel the registrar to segregate it finally into a separate section of his report. Much simplification would result immediately.

Table I (of general statistics) shows, in round figures, that 1,700 in-patients were admitted. This figure includes four houndred emergency cases, and, of this four hundred, two hundred were frankly septic on admission to the Rea Unit. From the hospital's own Ante-natal Department comes 1,300 cases; but we prefer the terms "booked" and "unbooked" as being unequivocal. Among the total of 1,193 babies born, we find the "ante-natal live births" phenomena hard to visualize.

Table II deals with maternal mortality. The figure of 0.31 per cent. for 1,300 booked cases is excellent. The percentage mortality for all admissions is four times, and for unbooked admissions is fourteen times, as great. (The corresponding figures of last year's report we found to be in the proportion of one, three, and seven.) Even more interesting, however, is the departure, new to this report, of calculating the maternal mortalities per thousand live births. There is, of course, a strong prima facie case for calculating maternal mortality according to the method of the Registrar-General's report, but the reader must beware of assuming that the figures emanating from a hospital such as this are comparable to those of the community.

That this is not the case is suggested by what was, to our mind, the most striking or conspicuous figure in this whole table. The figure for maternal mortality

per thousand live births in booked cases is 4.2. The Registrar-General's figure for England and Wales in 1935 is 3.94. If these figures do not admit of further qualification, the case for ante-natal care (and ante-natal care *was* care in this hospital when the present registrar taught the reviewer) collapses completely.

The explanation, as we see it, depends on two important factors. In the first place, the in-patient admissions, so far from being a cross-section of the community, will of necessity consist of cases suspected of some abnormality. Were this not so, these patients would have been confined in their homes on the district. It is in the latter, or rather in the combined cases, that we should look for evidence of the value of ante-natal care. We were about to calculate the maternal mortality per thousand live births from this point of view when we realized with a shock that the District Report seems to have been omitted, for the first time, we think, for many years.

Secondly, why calculate per thousand *live* births? The figures for England and Wales have not been so calculated for some years, but are based on total (live and still-) births. A maternity hospital can reasonably be expected to produce relatively fewer live births per confinement than does the community. The effect is to raise the maternal mortality per thousand live births. Thus one suspects that the extravagant maternal mortality figure of 114 per thousand live births for unbooked emergency cases owes much of its magnitude to the fact that as the emergency (central placenta prævia; impacted shoulder) increases, so does the proportion of live births per confinement fall. Halving the latter total doubles, of course, the maternal mortality, although no more mothers have died. So much for statistics.

The registrar's notes on this table are commendably well expressed, although we thought that the word 'column' had one generally accepted meaning, and horizontal ones took a little getting used to. One would be happier still if he removed the Rea Unit to a separate report. Like the old man of the sea, it bulks large, and precludes us from comparing readily this hospital's figures with those of others.

The details of cases are given more fully than in the last report. They tell the story of those cases which could not be saved, and the romance of the battles won is characteristically, if inevitably, withheld. For the reviewer to criticize would be impertinent, and only three comments, of the most general application, are here offered.

Two cases failed to continue their attendances at the Ante-natal Clinic, and were subsequently admitted to die. As an interesting point of administration, one wonders whether a local authority, undertaking to provide a maternity service directly, would have to follow such cases up on their ceasing to attend. The question, though perhaps scarcely relevant, has several interesting facets.

Three cases confirm our own latent and unconfessed distaste for blood transfusion. A chance question to us by a layman a few days ago started a train of thought which reached a startling terminus—in many cases we have privately blamed blood transfusions for hastening death: in none can we recall a transfusion which, in an emergency, saved life. Let others with wider experience produce their figures to correct these impressions.

# REVIEWS

## MUIR'S BACTERIOLOGICAL ATLAS. Second enlarged edition. Rewritten by C. E. van Rooyen, M.D. Edinburgh: E. & S. Livingstone, 1937. pp. 90; coloured plates 83. Price 15s. net.

This work is intended for use in conjunction with a textbook on Bacteriology and Parasitology. The present edition includes twenty-six new coloured plates by Dr. van Rooyen, in addition to the illustrations contained in the first edition.

Perhaps the most admirable feature of the atlas is that it is comprehensive and yet compact. The excellent illustrations should be helpful and stimulating to the undergraduate student; and their wide scope offers a book of reference to the post-graduate bacteriologist the usefulness of which is not likely to be exhausted by the first years of his experience. In this regard, special reference should be made to the sections dealing with the Spirochætes, Rickettsia and Virus Diseases, Tropical Diseases, and the Pathogenic Fungi.

A surprising amount of relevant information is compressed into the descriptions attached to each illustration.

The book is well printed on good paper, and is handsomely bound.

## DISEASES OF THE EYE. By Eugene Wolff, M.B., F.R.C.S. London: Cassells & Co., 1937. pp. 234; figs. 120; coloured plates 5. Price 15s. net.

This book is intended primarily for students and those in general practice, rather than for the specialists, and is a kind of illustrated guide to ophthalmology rather than a textbook in the usual sense. It does not, therefore, make any claims to include every branch of this department of medicine. But what branches are included present clearly recognized teaching, which is at the same time modern in its outlook. The book is beautifully illustrated in a manner which serves admirably the purposes of instruction, although one might carp at the predominance of anatomical drawings in a students' textbook. Another point on which a reviewer might find room to criticize is a lack of balance between the commoner and the rarer diseases. For example, retitinis pigmentosa is deal with in almost the same number of words as angioid streaks, and fewer than the description given the somewhat rare condition of keratoconus. This lack of balance is rather disturbing to a student beginning the study of ophthalmology for the first time, and it is hoped that in the next edition, which will certainly be called for, that critical revision of this point will be given.

## MINOR MALADIES AND THEIR TREATMENT. By Leonard Williams, M.D. Seventh Edition. London: Baillière, Tindall & Cox, 1937. pp. 440. Price 10s. 6d.

The newly qualified doctor doing his first locum is often surprised to find that his waiting-room is filled, not with cases of disseminated sclerosis, aortic regurgitations, or cancer of the rectum, but with people suffering from coughs, colds, rheumatism, and vague gastric disorders which he can neither classify nor treat. To him Dr. Williams' valuable book will supply the wisdom and experience of one who has spent many years both in general and consultant practice, and has profited more than most by his opportunities. The book is a gold mine of practical information and wise advice. This latest edition includes a useful section of dietetics. Of course, where so much is a matter of opinion and experience rather than scientific fact, there are bound to be openings for criticism. Thus, though the chapter on constipation contains much that is true and even more that is amusing, it is disappointing to find that treatment is almost confined to purgatives, and that more rational methods of diet, remedial exercises, etc., are scarcely touched on. But these are minor criticisms of a book which has been described as being worth its weight in gold. It should be in the hands of every general practitioner.

# REVIEWS

## MUIR'S BACTERIOLOGICAL ATLAS. Second enlarged edition. Rewritten by C. E. van Rooyen, M.D. Edinburgh: E. & S. Livingstone, 1937. pp. 90; coloured plates 83. Price 15s. net.

This work is intended for use in conjunction with a textbook on Bacteriology and Parasitology. The present edition includes twenty-six new coloured plates by Dr. van Rooyen, in addition to the illustrations contained in the first edition.

Perhaps the most admirable feature of the atlas is that it is comprehensive and yet compact. The excellent illustrations should be helpful and stimulating to the undergraduate student; and their wide scope offers a book of reference to the post-graduate bacteriologist the usefulness of which is not likely to be exhausted by the first years of his experience. In this regard, special reference should be made to the sections dealing with the Spirochætes, Rickettsia and Virus Diseases, Tropical Diseases, and the Pathogenic Fungi.

A surprising amount of relevant information is compressed into the descriptions attached to each illustration.

The book is well printed on good paper, and is handsomely bound.

## DISEASES OF THE EYE. By Eugene Wolff, M.B., F.R.C.S. London: Cassells & Co., 1937. pp. 234; figs. 120; coloured plates 5. Price 15s. net.

This book is intended primarily for students and those in general practice, rather than for the specialists, and is a kind of illustrated guide to ophthalmology rather than a textbook in the usual sense. It does not, therefore, make any claims to include every branch of this department of medicine. But what branches are included present clearly recognized teaching, which is at the same time modern in its outlook. The book is beautifully illustrated in a manner which serves admirably the purposes of instruction, although one might carp at the predominance of anatomical drawings in a students' textbook. Another point on which a reviewer might find room to criticize is a lack of balance between the commoner and the rarer diseases. For example, retitinis pigmentosa is deal with in almost the same number of words as angioid streaks, and fewer than the description given the somewhat rare condition of keratoconus. This lack of balance is rather disturbing to a student beginning the study of ophthalmology for the first time, and it is hoped that in the next edition, which will certainly be called for, that critical revision of this point will be given.

## MINOR MALADIES AND THEIR TREATMENT. By Leonard Williams, M.D. Seventh Edition. London: Baillière, Tindall & Cox, 1937. pp. 440. Price 10s. 6d.

The newly qualified doctor doing his first locum is often surprised to find that his waiting-room is filled, not with cases of disseminated sclerosis, aortic regurgitations, or cancer of the rectum, but with people suffering from coughs, colds, rheumatism, and vague gastric disorders which he can neither classify nor treat. To him Dr. Williams' valuable book will supply the wisdom and experience of one who has spent many years both in general and consultant practice, and has profited more than most by his opportunities. The book is a gold mine of practical information and wise advice. This latest edition includes a useful section of dietetics. Of course, where so much is a matter of opinion and experience rather than scientific fact, there are bound to be openings for criticism. Thus, though the chapter on constipation contains much that is true and even more that is amusing, it is disappointing to find that treatment is almost confined to purgatives, and that more rational methods of diet, remedial exercises, etc., are scarcely touched on. But these are minor criticisms of a book which has been described as being worth its weight in gold. It should be in the hands of every general practitioner.

# REVIEWS

## MUIR'S BACTERIOLOGICAL ATLAS. Second enlarged edition. Rewritten by C. E. van Rooyen, M.D. Edinburgh: E. & S. Livingstone, 1937. pp. 90; coloured plates 83. Price 15s. net.

This work is intended for use in conjunction with a textbook on Bacteriology and Parasitology. The present edition includes twenty-six new coloured plates by Dr. van Rooyen, in addition to the illustrations contained in the first edition.

Perhaps the most admirable feature of the atlas is that it is comprehensive and yet compact. The excellent illustrations should be helpful and stimulating to the undergraduate student; and their wide scope offers a book of reference to the post-graduate bacteriologist the usefulness of which is not likely to be exhausted by the first years of his experience. In this regard, special reference should be made to the sections dealing with the Spirochætes, Rickettsia and Virus Diseases, Tropical Diseases, and the Pathogenic Fungi.

A surprising amount of relevant information is compressed into the descriptions attached to each illustration.

The book is well printed on good paper, and is handsomely bound.

## DISEASES OF THE EYE. By Eugene Wolff, M.B., F.R.C.S. London: Cassells & Co., 1937. pp. 234; figs. 120; coloured plates 5. Price 15s. net.

This book is intended primarily for students and those in general practice, rather than for the specialists, and is a kind of illustrated guide to ophthalmology rather than a textbook in the usual sense. It does not, therefore, make any claims to include every branch of this department of medicine. But what branches are included present clearly recognized teaching, which is at the same time modern in its outlook. The book is beautifully illustrated in a manner which serves admirably the purposes of instruction, although one might carp at the predominance of anatomical drawings in a students' textbook. Another point on which a reviewer might find room to criticize is a lack of balance between the commoner and the rarer diseases. For example, retitinis pigmentosa is deal with in almost the same number of words as angioid streaks, and fewer than the description given the somewhat rare condition of keratoconus. This lack of balance is rather disturbing to a student beginning the study of ophthalmology for the first time, and it is hoped that in the next edition, which will certainly be called for, that critical revision of this point will be given.

## MINOR MALADIES AND THEIR TREATMENT. By Leonard Williams, M.D. Seventh Edition. London: Baillière, Tindall & Cox, 1937. pp. 440. Price 10s. 6d.

The newly qualified doctor doing his first locum is often surprised to find that his waiting-room is filled, not with cases of disseminated sclerosis, aortic regurgitations, or cancer of the rectum, but with people suffering from coughs, colds, rheumatism, and vague gastric disorders which he can neither classify nor treat. To him Dr. Williams' valuable book will supply the wisdom and experience of one who has spent many years both in general and consultant practice, and has profited more than most by his opportunities. The book is a gold mine of practical information and wise advice. This latest edition includes a useful section of dietetics. Of course, where so much is a matter of opinion and experience rather than scientific fact, there are bound to be openings for criticism. Thus, though the chapter on constipation contains much that is true and even more that is amusing, it is disappointing to find that treatment is almost confined to purgatives, and that more rational methods of diet, remedial exercises, etc., are scarcely touched on. But these are minor criticisms of a book which has been described as being worth its weight in gold. It should be in the hands of every general practitioner.

## ILLUSTRATIONS OF REGIONAL ANATOMY. By E. B. Jamieson, M.D. Second Edition. Edinburgh: E. & S. Livingstone, 1937. 305 plates. Price (seven sections) 47s. 6d.

Since its first publication in 1934, this series of plates made under the supervision of Dr. E. B. Jamieson to illustrate the structure of the human body, has more than proved its usefulness to the student of anatomy, and indeed as a work of reference for those who have passed on to the study of the nature and treatment of the morbid conditions to which the body is liable, and of which no understanding knowledge can be gained without the necessary basis on which to build—a sound acquaintance with the normal structure.

Under present-day conditions, where the scarcity of material for dissection unfortunately makes the study of anatomy more and more one to be pursued in the textbook rather than in the dissecting-room, it is to just such a work as this that the student may turn as a supplement to and a check on his textbook when his dissection is no longer at hand for reference. The high standard of accuracy reached in this work, and the clarity of the majority of its plates, make it especially useful in this respect.

For this edition Dr. Jamieson has completely revised his work; several plates have been enlarged to full-page format and the majority of the illustrations are now coloured. The addition of an index to each section fills a gap that was noticeable in the first edition. These several improvements bring this edition of the five sections (Central Nervous System, Head and Neck, Abdomen, Pelvis, Thorax) into uniformity with the two sections on the Upper and Lower Limbs that were first published last autumn, so that the whole work now consists of over three hundred plates, bound for greater ease of reference on the loose-leaf system.

## WHEELER AND JACK'S HANDBOOK OF MEDICINE. Revised by John Henderson, M.D., F.R.F.P.S. Tenth Edition. Edinburgh: E. & S. Livingstone, 1937. pp. 703. Price 12s. 6d.

An attempt to write a "handbook" of medicine which will be at once comprehensive and concise might seem an ambitious project, but Wheeler and Jack's volume has long been esteemed for these very qualities. The reviser has made a success of the latest edition, and has succeeded in packing a surprising amount of information into the small space at his disposal. A good example is the chapter on diseases of the kidneys. Here we are given a section on clinical examination of the urine, a good classification of the different types of nephritis, with the essentials of pathology, symptomatology, and treatment in each case, and finally a useful tabular summary.

It is, we suppose, impossible in such a work to keep an even balance of emphasis, but it is rather surprising to note the inadequate treatment given to some important conditions. Thus empyema is dealt with in a rather perfunctory manner. And surely it is as important to know the technique and indication for exploratory aspiration of the chest as it is to know the details of the phenyl-hydrazine test for urinary sugars? Again, while some recent advances have been mentioned, others just as important have been omitted, e.g., the chemotherapy of streptococcal infections.

Faults of this kind are, however, surprisingly few in view of the nature and difficulty of the task. Professor Henderson is to be congratulated on a book which should continue in its popularity both with students and practitioners.

## ILLUSTRATIONS OF REGIONAL ANATOMY. By E. B. Jamieson, M.D. Second Edition. Edinburgh: E. & S. Livingstone, 1937. 305 plates. Price (seven sections) 47s. 6d.

Since its first publication in 1934, this series of plates made under the supervision of Dr. E. B. Jamieson to illustrate the structure of the human body, has more than proved its usefulness to the student of anatomy, and indeed as a work of reference for those who have passed on to the study of the nature and treatment of the morbid conditions to which the body is liable, and of which no understanding knowledge can be gained without the necessary basis on which to build—a sound acquaintance with the normal structure.

Under present-day conditions, where the scarcity of material for dissection unfortunately makes the study of anatomy more and more one to be pursued in the textbook rather than in the dissecting-room, it is to just such a work as this that the student may turn as a supplement to and a check on his textbook when his dissection is no longer at hand for reference. The high standard of accuracy reached in this work, and the clarity of the majority of its plates, make it especially useful in this respect.

For this edition Dr. Jamieson has completely revised his work; several plates have been enlarged to full-page format and the majority of the illustrations are now coloured. The addition of an index to each section fills a gap that was noticeable in the first edition. These several improvements bring this edition of the five sections (Central Nervous System, Head and Neck, Abdomen, Pelvis, Thorax) into uniformity with the two sections on the Upper and Lower Limbs that were first published last autumn, so that the whole work now consists of over three hundred plates, bound for greater ease of reference on the loose-leaf system.

## WHEELER AND JACK'S HANDBOOK OF MEDICINE. Revised by John Henderson, M.D., F.R.F.P.S. Tenth Edition. Edinburgh: E. & S. Livingstone, 1937. pp. 703. Price 12s. 6d.

An attempt to write a "handbook" of medicine which will be at once comprehensive and concise might seem an ambitious project, but Wheeler and Jack's volume has long been esteemed for these very qualities. The reviser has made a success of the latest edition, and has succeeded in packing a surprising amount of information into the small space at his disposal. A good example is the chapter on diseases of the kidneys. Here we are given a section on clinical examination of the urine, a good classification of the different types of nephritis, with the essentials of pathology, symptomatology, and treatment in each case, and finally a useful tabular summary.

It is, we suppose, impossible in such a work to keep an even balance of emphasis, but it is rather surprising to note the inadequate treatment given to some important conditions. Thus empyema is dealt with in a rather perfunctory manner. And surely it is as important to know the technique and indication for exploratory aspiration of the chest as it is to know the details of the phenyl-hydrazine test for urinary sugars? Again, while some recent advances have been mentioned, others just as important have been omitted, e.g., the chemotherapy of streptococcal infections.

Faults of this kind are, however, surprisingly few in view of the nature and difficulty of the task. Professor Henderson is to be congratulated on a book which should continue in its popularity both with students and practitioners.