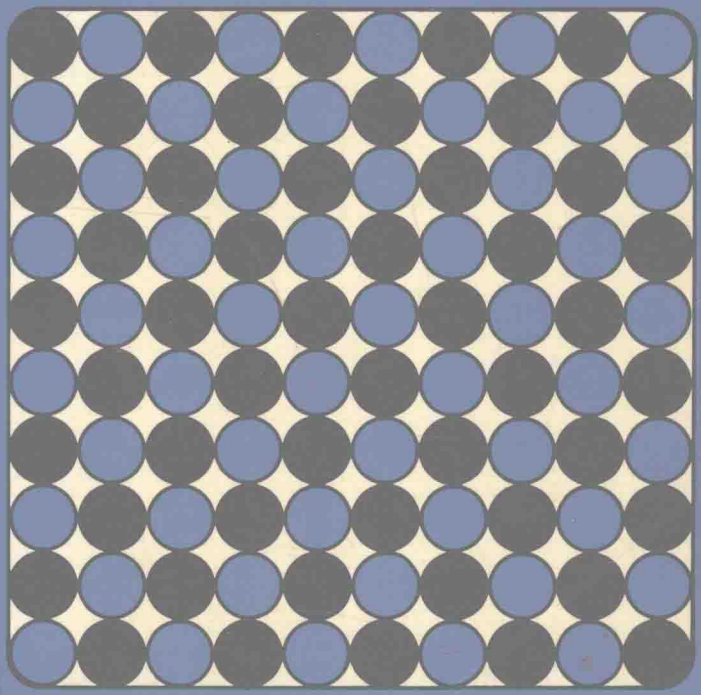
100 DATA INTERPRETATION QUESTIONS FOR THE MRCP

RICHARD ASHFORD
AND PATRICK VENABLES



Churchill Livingstone

100 Data Interpretation Questions for the MRCP

by

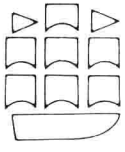
Richard Ashford

MA, MB, B.Chir., MRCP(UK)
Senior Medical Registrar,
St Stephen's Hospital,
London

and

Patrick Venables

MA, MB, B.Chir., MRCP(UK)
Research Fellow,
Charing Cross Hospital,
London



CHURCHILL LIVINGSTONE
EDINBURGH LONDON AND NEW YORK 1979

CHURCHILL LIVINGSTONE
Medical Division of Longman Group Limited

Distributed in the United States of America by
Longman Inc., 19 West 44th Street, New York,
N.Y. 10036, and by associated companies,
branches and representatives throughout
the world.

First Edition 1979

ISBN 0 443 01810 3

British Library Cataloguing in Publication Data

100 data interpretation questions for the MRCP.
1. Diagnosis – Problems, exercises, etc.
I. Ashford, Richard II. Venables, Patrick
III. Hundred data interpretation questions for the
MRCP
616.07'5'076 RC71.3 78-40711

Printed in Singapore by
Kua Co., Book-Manufacturers Pte Ltd

100 Data Interpretation Questions for the MRCP

Introduction

The aim of this book is to enable the reader to assess his or her ability to make deductions from laboratory data. We would not claim that data interpretation should be the sole means of assessing patients, but we do feel that, as the laboratory is acquiring increasing importance in diagnosis and management, a book devoted to investigations and their interpretation is justified.

The layout of the book is similar to that of the written section of the MRCP. The hundred questions are divided into ten 'papers' each consisting of 2 ECG's and eight other questions which include chemical pathology, haematology, lung function tests, blood gases and cardiac catheter data. Most of the questions and all of the ECG's are based on patients seen by us or our colleagues, though obviously some of the information has had to be altered slightly to enable fair questions to be asked. As a result some of the examples may seem rather artificial or perhaps too 'typical' to be real. Nevertheless, we feel that such an adjustment is necessary to ensure that none of the information is irrelevant or misleading.

In each case answers are provided and where relevant the differential diagnosis briefly discussed. The book is not intended to be a textbook and we hope that any information that is provided in the answers is used more as an aid in the technique of interpretation than as a source of factual knowledge. The veracity of every answer has been checked carefully and in this context we are particularly grateful to our colleagues for their helpful criticisms. In particular we would like to mention Dr J Silva who has reviewed the chemical pathology; Dr D Samson, the haematology and Dr R Sutton, the ECG's and catheter data. Others who have given either data or advice include Dr K Venables, Dr M Brenner, Dr P Butler and Dr A Scott-Keat. Our thanks go to Karen King, Angela Millen and Mary Wheeler for typing the manuscript.

We should also draw attention to the MRCP candidates who attended the course at St. Stephen's Hospital, London, where the contents of this book have been used as teaching material for the last 18 months. Their comments (not always lacking in emotion!) during our many sessions have helped enormously in the evolution of what we believe to be a fair and moderately testing collection of questions.

Table of normal ranges

Plasma or serum	SI Units	Conventional Units
Alanine transaminase ALT (SGPT)	0.20 U/l	0-20 U/l
Albumin	35-45 g/l	3.5-4.5 g/100 ml
Aldosterone	100-330 mmol/l	2.5-12 ng/100 ml
Alkaline phosphatase	20-100 U/l	3-12 King-Armstrong U/100 ml
Anti-Diuretic Hormone (ADH)		4-8 ng/l
Aspartate transaminase AST (SGOT)	0-25 U/l	0-25 U/l
Bicarbonate	22-28 mmol/l	22-28 mEq/l
Bilirubin	2-17 μmol/l	0.1-1 mg/100 ml
Calcium	2.25-2.62 mmol/l	9-10.6 mg/100 ml
Chloride	93-108 mmol/l	93-108 mEq/l
Cholesterol	3.6-7.2 mmol/l	145-280 mg/100 ml
Cortisol:		
9 am	170-720 nmol/l	6-26 μg/100 ml
midnight	170-220 nmol/l	6-8 μg/100 ml
Creatinine phosphokinase (CPK)	$<$100 U/l	
Creatinine	$<$80 μmol/l	1.0mg/100 ml
DNA Binding	$<$25 U	
Growth hormone		$<$10 ng/ml
Glucose (fasting)	3.6-6.6 mmol/l	65-120 mg/100 ml
Immunoglobulins		
IgA	1.25-4.25 g/l	125-425 mg/100 ml
IgG	5-16 g/l	500-1600 mg/100 ml
IgM	0.5-1.7 g/l	50-170 mg/100 ml

I-131 uptake	11-33% of dose at 4 hours	
Iron:		
males	16-30 μmol/l	90-170 g/100 ml
females	11-27 μmol/l	60-150 g/100 ml
Iron binding capacity (TIBC)	45-72 μmol/l	250-400 g/100 ml
Magnesium	0.65-1 mmol/l	1.3-2.0 mEq/l
Osmolality (plasma)	285-295 mmol/l	285-295 (mosmols/l)
pCO_2	4.7-6.0 kPa	35-45 mmHg
pO_2	12-13.3 kPa	90-100 mmHg
pH		7.36-7.45
Phosphate	0.8-1.4 mmol/l	2.5-4.3 mg/100 ml
Potassium	3.5-5.0 mmol/l	3.5-5.0 mEq/l
Protein (total)	58-72 g/l	5.8-7.2 g/100 ml
Protein (CSF)	0.15-0.4 g/l	15-50 mg/100 ml
Serum hydroxy-butyric-dehydrogenase (SHBD)	50-170 U/l	
Sodium	133-145 mmol/l	133-145 mEq/l
Thyroxine (T4)	70-160 nmol/l	5.5-12.5 μg/100 ml
T3 Resin uptake	88-110%	
Thyroid stimulating hormone (TSH)	0.8-3.6 mU/l	
Urea	3.3-7.0 mmol/l	20-42 mg/100 ml
Urate:		
male	0.24-0.44 mmol/l	4.0-7.5 mg/100 ml
female	0.21-0.37 mmol/l	3.5-6.2 mg/100 ml
Faecal fat	0-17 mmol/24 hr	0-59/24 hr
Urine		
Coproporphyrins		<0.1 mg/24 hr
Hydroxy-methoxy-malonic acid	5-35 μmol/24 hr	1.7 mg/24 hr

Haematological

Haemoglobin (Hb):		
males	13.5-18.0 g/dl	13.5-18.0 g/100 ml
females	11.5-16.5 g/dl	11.5-16.5 g/100 ml
Red blood cell count:		
males	4500-6.500 x 10^9/l	4.5-6.5million/mm^3
females	3900-5600 x 10^9/l	3.9-5.6million/mm^3
Packed cell volume (PCV):		
males	0.4-0.54	40-54 per cent
females	0.35-0.47	35-47 per cent
Mean corpuscular haemoglobin (MCH)	27-32 pg	27-32 μg
Mean corpuscular haemoglobin concentration (MCHC)	32-36 g/dl	32-36 per cent
Mean corpuscular volume (MCV)	76-98 fl	76-98 μm^3
Reticulocyte count		0.2-2 per cent
White blood count (WBC)	X10^9/l	/mm^3
total	4.0-11.0	4000-11000
neutrophils	2.5-7.5	2500-7500
lymphocytes	1.5-3.5	1500-3500
cosinophile	0.04-0.44	40-440
basophile	0-0.1	0-100
monocytes	0.2-0.8	200-800
Platelets	X10^9/l 150-400	/mm^3 150,000 - 400,000
Vitamin B_{12}	200-800 ng/l	200-800 μg/ml
Leucocyte Alkaline Phosphatase (LAP)	20-70/100 neutrophils	
ESR (Westergren) all ages, both sexes	$<$25mm/hr	
Prothrombin Time (PT)	14 sec	
PT Ratio (Test/control)	$<$1.2	

Activated Partial Thromboplastin Time (PTT)	15 sec	
Kaolin Cephalin Clotting Time (KCCT)	40 sec	

Arterial Blood

pCO_2	4.7-6.0 kPa	35-45 mmHg
pO_2	12-13.3 kPa	90-100 mmHg
pH	7.36-7.45	7.36-7.45

Cerebral Spinal Fluid

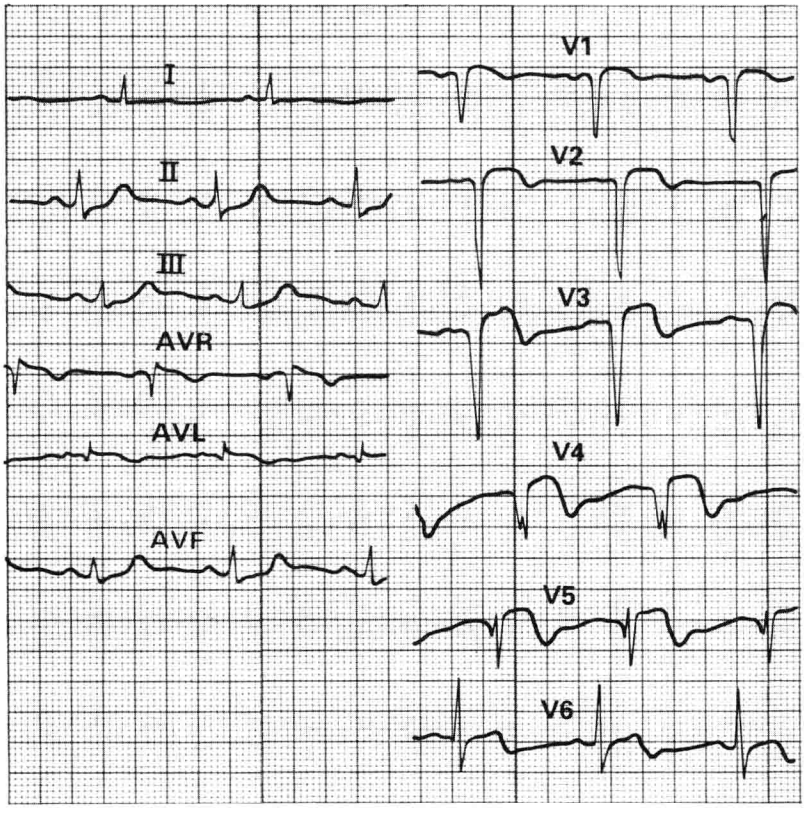
Glucose	3.3-5.0 mmol/l	60-90 mg/100 ml
Protein	0.15-.04 g/l	15-40 mg/100 ml

Miscellaneous

Faecal Fat	0.17 mmol/24 h	0-5 g/24 h
Xylose	23% of oral dose in 5 h more than half within the first 2 h	

Paper 1

1.1

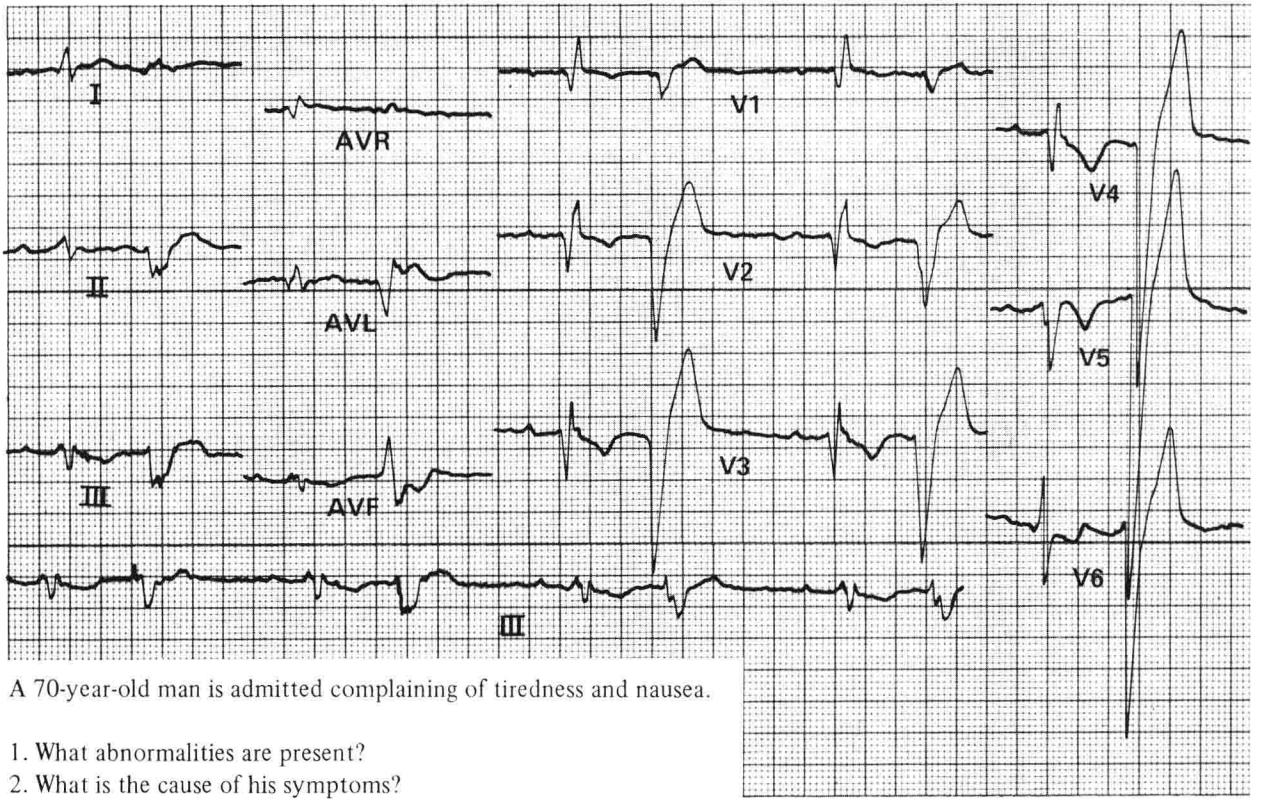


What is the diagnosis?

Recent Anterior Myocardial Infarction

Deep Q waves, elevated ST segments and partial T wave inversion are seen in V1 - V5. These changes are pathognomonic of recent anterior infarction. The early T wave inversion suggests the infarct is between 3 and 7 days old.

The notching of the QRS complex in V4 and V5 is due to an associated intraventricular conduction defect; **not** a bundle branch block.



A 70-year-old man is admitted complaining of tiredness and nausea.

1. What abnormalities are present?
2. What is the cause of his symptoms?

1. a. **First Degree Heart Block**
   The PR interval varies between 0.22 and 0.24 secs. This is prolonged. The upper limit of normal is generally taken to be 0.22.
   b. Coupled Ventricular Ectopics (bigemini)
   c. Old Anterior Myocardial Infarction
   In the sinus beats there is poor R wave progression in the chest leads.
   d. Right Bundle Branch Block
   The QRS complex is prolonged (0.12 secs) with an RSR pattern in V1 - V4.
   e. Digoxin Effect
   The ST segments slope downwards leading into an inverted T wave; the 'reversed tick' sign. This is seen in patients on cardiac glycosides but does not necessarily indicate toxicity. The change is best shown in the infero-lateral leads.

2. **Digoxin Toxicity**
   Tiredness and nausea are both symptoms of digoxin toxicity. Other symptoms that may occur include anorexia and disturbances of colour vision of which xanthopsia is said to be the most characteristic. Bradycardia, first degree heart block and coupled ventricular ectopics are common features of digoxin toxicity though almost any arrhythmia may be seen.

1.3

A 60 year old man is admitted unconscious.
Blood glucose 72 mmol/1
Sodium 154 mmol/1
Potassium 4.9 mmol/1
Bicarbonate 22 mmol/l
Total Protein 82 g/1
Osmolality 405 mmol/kg (NR: 285-295)

1. What is the diagnosis?
2. What three therapeutic measures would you institute?

1. **Hyperosmolar hyperglycaemic non-ketotic coma**
   There is gross hyperglycaemia, and dehydration shown by a high total protein. The low normal bicarbonate is evidence against ketoacidosis. The hyperosmolality is due to hyperglycaemia and is exacerbated by the resultant osmotic diuresis.
   Plasma osmolality may be calculated as follows:
   $(2 \times Na^+) + (2 \times K^+)$ + blood glucose + blood urea ≙ plasma osmolality.

2. **a. i.v. ½ normal saline**
   Under CVP monitoring to correct the fluid deficit. Normal saline can be used if the sodium is less than 145 mmol/1.
   b. i.v. Insulin Infusion 2 – 6 U/Hour or 5U hourly i.m.
   c. i.v. or s.c. Heparin in the absence of any contraindication because there is a high incidence of thrombotic complications in these patients.
   After the above have been instituted rigorous monitoring of blood sugar and electrolytes is necessary and the cause of the coma must be sought.

Types of Coma in Diabetes:
Diabetic ketoacidosis
Non-ketotic hyperglycaemic coma
Lactic acidosis (biguanide therapy)
Hypoglycaemia
Uraemic coma

1.4

A woman who had a mastectomy 10 years ago complains of backache, bruising and tiredness: She has received no treatment:
Haemoglobin 10.7g/dl
Platelets 50 x 10^9/1 (50 000/mm^3)
Prothrombin time 26 secs (control 12 secs)
Kaolin cephalin clotting time 55 secs (control 38 secs)

1. What is the haematological diagnosis?
2. Name one test to confirm your diagnosis.

1. Disseminated Intravascular Coagulation (DIC)

Probably due to carcinomatosis. Prolongation of prothrombin time and kaolin cephalin clotting time indicates deficiency of more than one clotting factor. This in the presence of thrombocytopenia suggests DIC. The formation of thrombi in small blood vessels consumes platelets and clotting factors. The fall in haemoglobin is due to haemolysis. The process may be initiated by:

a. Release of thromboplastic factors into the blood stream from damaged tissues.

b. Extensive endothelial damage.

Causes of DIC

Acute:

Obstetric accidents:
- abruptio placentae
- ammiotic fluid embolism

Heart and lung surgery
Haemolytic transfusion reaction
Septicaemia-especially meningococcal
Pulmonary Embolism
Snake Bites
Anaphylaxis
Diabetic Ketoacidosis

Chronic:

Disseminated carcinoma (particularly pancreas, stomach, breast)
Acute leukaemia
Intrauterine foetal death

2. Fibrin Degradation Products (FDP's)

Following fibrinolysis Fibrin Degradation Products (FDP's) circulate. Small amounts can be detected in normal people but increased amounts circulate in DIC.

Other helpful tests include: Fibrinogen level – reduced in DIC;
Blood film – for evidence of fragmented red cells.