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CONTENTS

CONTENTS

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This covers one volume (two numbers) of the Journal.

CONTENTS

Ulster Med J 1981; **50**: 77-82

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VOLUME 50 1981 No. 2

FAMILIAL CARCINOMA OF THE PROSTATE IN A SIBSHIP WITH OTHER TUMOURS AND AN AGGREGATION OF PAGET'S DISEASE OF BONE

C H LEE, INGRID V ALLEN, J S LOGAN
Royal Victoria Hospital, Belfast

INTRODUCTION

IT has long been recognised that some families have a high incidence of malignancy. Where a dominant inheritance is observed, e.g., with retinoblastoma, genetic factors clearly operate. In other families though the incidence of malignancy is high in one sibship, no cases are found in the preceding or succeeding generations. In this case either the disease is due to inheritance of a recessive gene from each parent, or else to some common environmental agent. Possibly both are necessary. Further, in a cancer sibship the cancers may be of different kinds. This makes it all the more necessary to consider chance association or different environmental agents. Nevertheless, in the literature there are various reports of families with an unusually high incidence of neoplasia in which the pattern of malignancy differs from individual to individual but the overall family pattern would suggest that there might be some genetic basis. Warthin[1] [2] reported such a family with an unusually high incidence of gastro-intestinal and uterine cancer. This family has been further reviewed on two occasions.[3] [4] In 1936 there were 174 members who were 25 years of age or over. In these there were 43 cases of primary carcinomas in 41 individuals and with two exceptions all involved the gastro-intestinal tract or endometrium. By 1970 there were more than 650 blood relatives, 95 of whom had developed a malignant neoplasm (13 had more than one primary neoplasm). Gastrointestinal and endometrial carcinomas were again predominant, though leukaemia (two cases) lymphosarcoma (two cases) and plasmocytoma (one case) had appeared.

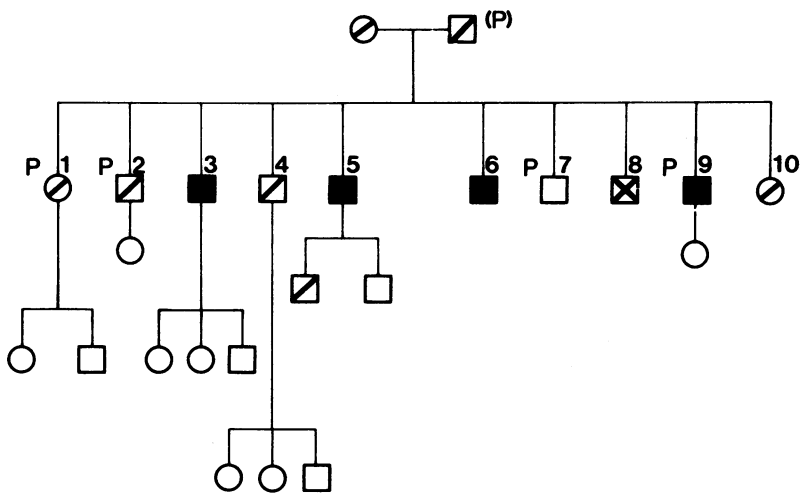
Several other "cancer families" have been reported in the United States[5] [6] [7] and Savage[8] has reported a family in the United Kingdom. The possible import-

ance of HLA type in these families is suggested by the American report[9] in which 20 of 21 members of "Cancer Family M" had HLA haplotype, HL-A2-HL-A12 (relative odds = 6.30).

Familial carcinoma of the prostate has been reported.[10] [11] [12] We have observed such a grouping in a Belfast family.

FAMILY HISTORY

The family history has been obtained partly from the hospital notes and partly from members of the family. All members of the family were born and lived in North Belfast. The figure gives the family tree and indicates those members of the family suffering from malignant tumours. The table lists the cancers, simple tumours, and other documented diseases in the sibship. The mother died in 1936 and is said to have had bone disease, probably from the children's description,



■ Prostatic Carcinoma.
⊘ Died from some other cause.
○ Alive and well. Not however surveyed for Paget's Disease.
⊠ Carcinoma of Rectum and Myelosclerosis. Alive.
P Paget's Disease of Bone.
(P) Family give History of Paget's Disease of Bone.

PEDIGREE OF FAMILY K

Paget's disease. There are no medical records in this case. The father died aged 58. Necropsy showed renal arteriosclerosis with scarring, left ventricular hypertrophy and pulmonary oedema. No malignancy was found in the prostate

TABLE

Diseases in Family K Sibship

Sex	Age at Death	Site of Malignancy	How Confirmed	Other Diseases
Female	77	—	—	Paget's disease of bone. Ischaemic heart disease. Non-functioning left kidney. Duplex right renal pelvis.
Male	67	—	—	Paget's disease of bone.
Male	53	Prostate	Death Certificate	—
Male	70	—	—	Diverticulitis of colon.
Male	50	Prostate	Histology	Multiple subcutaneous lipomata.
Male	62	Prostate	Histology	—
Male	—	—	—	Paget's disease of bone.
Male	—	Rectum. Polycythaemia vera progressing to myelosclerosis.	Histology	Rosacea. Myopathy. Secondary gout. Gallstones.
Male	—	Prostate	Histology	Paget's disease of bone. Squamous papilloma tongue.
Female	56	—	—	Multiple sclerosis.

or elsewhere. Of the four siblings with carcinoma of the prostate, three have died, the mean age at death being 55 years. The fourth affected sibling is 62 years old, is on stilboestrol therapy and is reasonably well, having had a prostatectomy. He also suffers from Paget's disease of bone. One sibling with Paget's disease is alive and otherwise well. Two other siblings with Paget's disease have died, apparently from myocardial infarction though necropsy was not carried out. The sibling with primary polycythaemia and adenocarcinoma of the rectum is still alive. The polycythaemia has progressed to myelosclerosis. He has no evidence of recurrence of the rectal tumour. In the third generation, one male died aged two years, from pneumonia. There are eleven third generation living members (aged 40-50), four of whom are males. All are apparently well.

DISCUSSION

Evidently this sibship has had familial carcinoma of the prostate. Judging by the paucity of published reports, this is rare. This rarity makes it possible that the concentration is a matter of chance, but it is somewhat against its being due to an environmental agent alone. We think it likely that this family concentration is due to these brothers inheriting a causative recessive gene from each parent. The parents were not known to be related. It may be, and it is quite likely, that a common environmental agent was also necessary to produce the cancers, in sibs genetically and immunologically vulnerable. Two of the brothers with carcinoma of the prostate were painters, one a grocer, and one a breadserver. We do not see any occupational risk in these employments. Because the parents are dead, we could not study them. The recessive gene evidently being very rare, we are not likely to see any manifestation in the next generation. Cousin marriage in any degree would be dangerous in this kinship, and the members should be warned against it. The next generation should be kept under surveillance, especially into middle age and old age.

It may be that the gene effect is on the immune system. HLA typing and other methods of studying the immune system became available only when so many of the siblings had died that no useful study was possible.

The existence of Paget's disease of bone in the family may or may not be related. Concentration of Paget's disease of bone in a family has been recorded in the past.[13] Particular care must be taken in the male members of such a family as this to distinguish the osteosclerotic secondary deposits of prostatic carcinoma from Paget's disease of bone. The presence of Paget's disease in one brother was recorded by x-ray when he was 31. This is unusually early. We have not included as Paget's disease in the figure or table two brothers in which the old clinical and x-ray reports recorded the possibility of the co-existence of both Paget's disease and prostatic metastases but were indecisive.

What is the meaning of the occurrence of carcinoma of rectum and of myelosclerosis (supervening on polycythaemia vera) in sib number 8? If either or both were caused by the recessive gene we have postulated, the expression has been very different. Perhaps different environmental agents were at work, or perhaps there were other modifying genes. Rectal examination of the prostate in this sib is impossible because of the proctectomy. A striking disability in him is a myopathy. There is severe muscle wasting and weakness. The serum creatine phosphate kinase has often been abnormally low. The serum iron has been low for a long time. The deficiency up to now has not been corrected so as to maintain a packed cell volume within normal limits.

Even more intriguing is the occurrence of multiple sclerosis in a sister. Another patient of ours, in another unrelated sibship with multiple cancers, has fibrosing alveolitis. There is evidence that the development of a disease may depend both on a genetically determined vulnerability to an environmental agent, and also on exposure to the particular environmental agent. An example is flax byssinosis.[14] It is possible that a single genetic mechanism may make the subject vulnerable to more than one environmental agent. The disease developed in any one of the

sibship would then depend on the particular agent each one was exposed to. Variation must also be due to sex differences; females could not develop prostatic carcinoma.

Since the immune constitution is not likely to be modifiable, prevention will depend on identifying the environmental agent. If, as methods improve, genetic vulnerability can be determined, epidemiologists can study transmission in the vulnerable population, the only population worth studying. Even now susceptible kinships can be kept under surveillance. In oncology units enquiry should be made about other cases of cancer in the family. In this way affected kinships can be selected for surveillance. It is noteworthy that though Thiessen[15] reported an excess of prostatic carcinoma in the male relatives (mainly fathers) of women with breast cancer, neither sister in this family had either breast or genital cancer. The daughters of the brothers with prostatic carcinoma perhaps should have special surveillance for breast carcinoma.

SUMMARY

Multiple familial malignancy is described in one sibship, all the members of which were born and lived in Belfast. Five of the eight males have had confirmed primary cancer. Four have had prostatic carcinoma. A fifth male has had a carcinoma of rectum and also myelosclerisis. One sister had multiple sclerosis. There is an aggregation of Paget's disease of bone in the family. It appears likely that a genetic influence in this family predisposes to prostatic carcinoma. The relation, if any, of this influence to the other diseases in the family remains uncertain.

ACKNOWLEDGEMENTS

We are indebted to the records staffs of the Royal Victoria Hospital, Belfast City Hospital, and the Musgrave Park Hospital for help with records, as well as to the staff of the histopathology department, Royal Victoria Hospital.

Mrs. Margaret Clarke has typed the manuscript and given us much help with records.

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Ulster Med J 1981; **50**: 83-87

SEX RATIO OF BIRTHS IN IRELAND IN 1978

A LEONARD WALBY, MB, MFCM
Director, Research and Intelligence Unit
Department of Health and Social Services, Belfast

J DESMOND MERRETT, PhD
Reader, Department of Medical Statistics
The Queen's University of Belfast

GEOFFREY DEAN, MD, FRCP
Director, Medico-Social Research Board, Dublin

PEADAR KIRKE, MB, MFCM
Child Health Section, Medico-Social Research Board, Dublin

BETWEEN 1913 and 1977 the mean sex ratio of live births registered in the six counties and the two county boroughs of Northern Ireland was 1063 males per 1,000 females. The lowest ratio was 1034 in 1913 and the highest 1096 in 1941. In 1978 the very low ratio 1007 was recorded. In 1977 the ratio was 1070, and in 1979 it was again close to normal at 1058.

DATA AND METHODS

The data for Northern Ireland for the years 1922-1978 were obtained from the Annual Reports of the Registrar General for Northern Ireland[1]. For the years 1913 to 1921 the data were obtained from the Second Annual Report of the Registrar General (Northern Ireland), 1923[2]. Provisional data for 1979[3] were provided by the General Register Office for Northern Ireland. The data for the Republic of Ireland were obtained from the Central Statistics Office, Annual Reports on Vital Statistics 1960 to 1976[4], and Quarterly Reports on Births, Deaths and Marriages and on Certain Infectious Diseases 1977-1979[5]. The data giving live births male and female and sex ratios for Northern Ireland for 1913-1978, and live births male and female and sex ratios for the Republic of Ireland for 1960-1979, and stillbirths male and female and sex ratios for Northern Ireland for 1961-1979, and on which this paper is based are available on request.*

We correlated the number of male births with the number of female births in Northern Ireland for each of the 66 years 1913-1978, and derived the linear relationship between the numbers of male and female births. We also correlated for the same period the sex ratio, males per 1,000 females, with year and derived the linear relationship. The value of the sex ratio for 1978 was tested as an outlier by the method described by Quenouille[6].

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* Dr. A L Walby, Research and Intelligence Unit, Department of Health and Social Services, Annexe 2, Castle Buildings, Stormont Estate, Upper Newtownards Road, Belfast, BT4 3UD.

RESULTS

The correlation coefficient for Northern Ireland male births with female births, $r = 0.9886$, was exceptionally high and significant at $p < 0.05$. The linear relationship between the number of female births (y) and the number of male births (x) was

$$y = 0.901084x + 598.6523$$

with a standard deviation from regression of 196.52. From this equation the predicted number of female births in 1978 for an x value of 13168 is 12464 with 95 per cent confidence limits of 12416 to 12514. These limits do not include the value 13071 (Table) observed in 1978, and so we conclude that the observed number of female births significantly exceeds that expected from the overall relationship.

TABLE

Live Births - Area, Sex, and Sex Ratio
Republic of Ireland and United Kingdom, 1978

Area	Male	Female	Total	Males per 1,000 Females
Dublin	10,460	9,932	20,392	1,053
Rest of Leinster	8,570	8,330	16,900	1,029
Cork	4,058	3,921	7,979	1,035
Rest of Munster	6,091	5,886	11,977	1,035
Connacht	4,103	3,933	8,036	1,043
Ulster [Monaghan, Cavan and Donegal]	2,286	2,274	4,560	1,005
Republic of Ireland	35,568	34,276	69,844	1,038
Northern Ireland	13,168	13,071	26,239	1,007
England and Wales	307,088	289,330	596,418	1,061
Scotland	33,059	31,236	64,295	1,058

The correlation coefficient for Northern Ireland sex ratio with year, $r = 0.2570$, was small, but was nevertheless significantly different from zero at 5 per cent level, suggesting an increasing sex ratio (y) over the period observed. The scatter diagram showed that this trend was somewhat erratic but was nonetheless positive. The relationship was

$$y = 0.206868x + 1055.13$$

where x was a code for the year, with code 1 for 1913, code 2 for 1914, etc., up to code 66 for 1978. The standard deviation from regression was 15.05. It was very obvious from the scatter diagram that the value of the sex ratio for 1978 should be treated as an outlier. The predicted ratio for 1978 is 1069 with 99.9 per cent confidence limits of 1018 to 1119. The observed ratio, 1007, is therefore significantly different from the predicted value at the 0.1 per cent level.

The sex ratio of births in the 26 Local Government Districts in Northern Ireland shows considerable variation between districts and within districts from

year to year. A sex ratio less than 1000 occurred in 3 districts in 1974, 4 in 1975, 5 in 1976, and 4 in 1977; in 1978 a ratio less than 1000 occurred in 10 districts, 8 of which, including Belfast, were in the eastern half of the country.

In parallel with other parts of the British Isles, Northern Ireland experienced a falling birth rate from a peak of 23.6 live births per 1,000 of the population in 1964 to a trough of 16.5 in 1977. A rise was expected in 1978, and the rate did rise to 17.1. In the event, however, between 1977 and 1978 the number of male births rose by only 14 while the number of female births rose by 788. It seems that the low sex ratio in 1978 may reflect a deficit of male births rather than an excess of females. The fall in the sex ratio did not begin during 1977 when the ratio in the last quarter was 1076, nor extend into 1979 when the ratio in the first quarter was 1067.

In view of reports by James[7] for England and Wales and Renkonen[8] for the United States of America that sex ratios decline regularly with increasing maternal age, we looked at the distributions of maternal age in Northern Ireland in 1977, when the ratio was high, and in 1978 when it was low. We found that the distributions were significantly different, 1978 having proportionately fewer mothers aged under 30 and more over 30. We then correlated the sex ratio with maternal age for each of the five years for which data were available, 1974 to 1978. Only one year, 1976, showed declining ratios with increasing age, the other four years having correlation coefficients opposite in sign and supporting the contrary trend. We also looked at the distributions of duration of marriage and of parity in 1977 and 1978. Although these variables differed significantly in distribution there was no evidence that either was correlated with the sex ratio of live births.

The mean sex ratio of Northern Ireland stillbirths from 1961 to 1977 was 975 males per 1,000 females. In 1978 the ratio, 1113, was the highest recorded except for 1123 in 1977. The numbers of stillbirths in 1978 were 128 males and 115 females, the lowest ever recorded, and so the high ratio accounts for only a small part of the deficit of males. No information can be obtained about the sex ratio of spontaneous abortions. Northern Ireland does not have an equivalent to the Abortion Act 1967 in Great Britain so that comprehensive data on induced abortions in Northern Ireland women are not available. A steadily increasing number of Northern Ireland women obtain abortion each year in Great Britain; in 1978 the number aborted in England and Wales was 1301[9], but the sex of the aborted foetuses is not known.

A corresponding fall in the sex ratio of births in 1978 did not occur in England and Wales[10], where the ratio was 1060, nor in Scotland[11], where the ratio was 1058, neither ratio differing greatly from that expected. In the Isle of Man[12] the ratio was 1122.

In the Republic of Ireland on the other hand the sex ratio in 1978 was 1038, the lowest recorded in the past 20 years, and the ratio in 1979 was back to normal at 1060. The ratio in 1978 in the two cities, Dublin and Cork, and in the provincial

areas of the Republic is variable, but the three counties of the province of Ulster within the Republic, Monaghan, Cavan, and Donegal, show the very low ratio of 1005 (Table).

DISCUSSION

The sex ratio of births shows considerable variability from place to place and from year to year when small areas are considered, but the main parts of the British Isles this century appear to have had very similar mean ratios, about 1060 males per 1000 females. The occurence in one region of the British Isles with over 26,000 births per annum of a ratio well below the lower 99.9 per cent confidence limit gives rise to speculation as to a possible cause. If the cause lay in some temporary change in the environment, such as an air-, water-, or food-borne contaminant or a drug having a selective effect on male spermatozoa, male conceptions or male embryos, this should in principle be discoverable and could suggest an approach to control of the sex of infants and livestock. Colleagues in agricultural and veterinary science with whom we discussed the matter had not noticed any disturbance in the sex ratio of animals, but they thought that, in the absence of records for animals comparable to human births registration, all but the grossest changes in sex ratio would have remained undetected. We have been unable to think of a likely explanation for the 1978 ratio and have found no associated conditions worth investigating. The fact that the low ratio was confined to a single calendar year does not make the matter easier to understand, but made us look for some form of artefact as a possible cause. This we did not find, and the low ratio is confirmed by the birth notification in Northern Ireland which are made quite independently of the registrations of birth. The departure from the expected sex ratio in 1978 will be noticed when this cohort of Northern Ireland children reaches school age and a number of boys sufficient to fill several schools are missing mainly from the east of the province.

We have here a phenomenon affecting the whole of Ireland, but the north more than the south, and not affecting Great Britain. The finding that the three Ulster counties outside Northern Ireland had a ratio even lower than Northern Ireland's 1007 is intrigueing and makes us reluctant to accept this as merely an extreme example of random variation.

SUMMARY

Northern Ireland has a mean sex ratio of live births of 1063 males per 1,000 females. In 1978 the very low ratio 1007 was recorded. This is significantly different from the predicted value at the 0.1 per cent level. A low ratio occurred also in the Republic of Ireland but not in Great Britain.

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A LONG TERM STUDY OF THE IRON STATUS OF PATIENTS FOLLOWING VAGOTOMY

T R J LAPPIN, T KENNEDY, A SPENCER, T H HASSARD, G A SAVAGE, E E MAYNE

Department of Surgery and Haematology, Royal Victoria Hospital, Belfast

The occurence of anemia following partial gastrectomy is well recognised: the reported incidence ranges from three to 63 per cent[1]. Iron deficiency is the commonest cause of the anaemia, deficiencies of either B12 or folic acid accounting for only a small number[1]. With the advent of vagotomy and drainage as the routine treatment for duodenal ulcer it was hoped that anaemia would no longer be a significant problem. However, Wheldon and associates[2] found that truncal vagotomy with gastrojunostomy resulted in anaemia in 43.5 per cent of males and 84 per cent of females after 15 years. There are no published data comparing the incidence of anaemia in the long term after the various types of vagotomy with or without drainage. The purpose of this study is to compare the iron status of patients after six different types of vagotomy and drainage.

PATIENTS AND METHODS

Four hundred and seventy-five patients were investigated; these comprised six prospectively studied groups of post vagotomy patients totalling 421. The three categories of vagotomy, truncal (TV), selective (SV) and proximal gastric vagotomy (PGV) were studied. The groups were sub-divided depending upon the accompanying drainage procedure; either gastrojejunostomy (GJ) or pyloroplasty (P).

Seventy patients with no known gastrointestinal disease and with comparable age and sex distributions to their counterparts were studied as controls. These patients had simple conditions such as inguinal herniae, varicose veins or benign breast lumps. Furthermore, 54 patients after partial gastrectomy (PG) were investigated as a form of quality control. If no evidence of anaemia or iron deficiency was found in this group, the study would be invalid. All patients were tested at least five years after operation. The male to female ratio of 3: 1 was similar in all groups. The details and designation of the groups are shown in Table 1.

TABLE 1

Patients studied more than 5 years after Gastric Surgery

Group		*Male*	*Female*	*Total*	*MEAN OF FOLLOW-UP (years)*
Truncal Vagotomy and Gastrojejunostomy	TVGJ	99	35	134	7.7
Truncal Vagotomy and Pyloroplasty	TVP	13	5	18	5.5
Selective Vagotomy and Gastrojejunostomy	SVGJ	27	4	31	11.9
Selective Vagotomy and Pyloroplasty	SVP	69	18	87	8.3
Proximal Gastric Vagotomy and Gastrojejunostomy	PGVGJ	76	23	99	6.5
Proximal Gastric Vagotomy	PGV	39	13	52	5.5
Partial Gastrectomy	PG	36	18	54	11.9
Controls	CONTROLS	53	17	70	—
	TOTAL	412	133	545	
		(75.6%)	(24.4%)		
		$Chi^2 = 5.62$; 7 D.F.			
		N.S.			

Serum iron and total iron binding capacity (TIBC) were measured by the method of Young and Hicks[3]. Normal ranges were 14-29 μmol/l and 45-72 μmol/l respectively. A percentage saturation of iron of less than 15 was regarded as an index of iron deficiency. Serum ferritin was determined by radioimmunoassay[4] using an antibody against human spleen ferritin raised in the rabbit. The normal mean concentration is higher in men than in women with a range between 12 and 250μg/l[5]. Ferritin shows a log-normal distribution[6] and for this reason the geometric mean values were used. In our laboratory these values are 100 μg/l for men and 60 μg/l for women; 10 μg/l is regarded as the lower limit of normal in both sexes[7]. Haemoglobin was determined by standard techniques, the normal range being 13-18 g/dl for men and 12-16 g/dl in women.

STATISTICAL METHODS

A one-way analysis of variance was used to investigate whether the mean levels of the various postoperative iron status parameters differed significantly between any of the groups. When this test confirmed the existence of significant differences, the Newman-Keuls test[8] was used to determine which groups could be considered to differ from one another. The test calculates a minimum difference value; groups whose mean levels differ by at least this value may be considered to be significantly different at the conventional $P<0.05$ level or greater (Tables 2, 3, 4). Because of the considerable variation in length of follow-up both within and between groups (Table 1) it was necessary to establish whether any of the

parameters showed a tendency to alter with time. Were such a tendency to exist, any differences between the groups could not be unambiguously ascribed to the influence of operation type. The existence of time trends was investigated by correlating the serum iron, TIBC, percentage iron saturation, ferritin and haemoglobin levels with the number of years between the initial operations and the date of the sample collection. These correlations were derived for each group in order to avoid confounding the influences of operation type and time. Variations in percentage with anaemia and percentage with low ferritin between groups were tested using the conventional Chi-squared test.

RESULTS

Of the 35 correlations of iron status parameters with time examined only one, the serum iron level in PG patients, showed a significant tendency to alter with duration of follow-up ($P<0.025$). The conventional level of statistical significance ($P<0.05$) implies that on average an apparently significant result will appear once in every twenty tests even when no true relationship exists, thus this single significant result from a total of 35 tests is not unexpected and is of little consequence. Therefore it is concluded that there is no evidence to suggest that iron status after vagotomy alters significantly during the period of investigation. This further implies that the variations in length of follow-up between the groups will not exert any influence on the levels of the parameters. Any inter-group differences can therefore be unequivocally ascribed to the influence of the types of vagotomy.

TABLE 2

Mean Values for Iron, TIBC and Percentage Saturation

Group	*Iron*	*TIBC*	*% Saturation*
1. TVGJ	18.68 (28)	79.21 (23)	26.04 (23)
2. TVP	17.97 (69)	69.97 (62)	27.26 (60)
3. SVGJ	19.43 (93)	79.44 (87)	25.24 (87)
4. SVP	20.74 (112)	72.51 (102)	29.87 (102)
5. PGVGJ	16.44 (18)	76.80 (10)	20.40 (10)
6. PGV	20.98 (52)	73.33 (48)	29.91 (47)
7. PG	15.52 (52)	77.99 (46)	21.49 (45)
8. CONTROLS	19.30 (67)	65.98 (64)	29.36 (61)
F	3.08	5.86	3.67
	7,483 D.F.	7,434 D.F.	7,427)D.F.
	$P<0.01$	$P<0.001$	$P<0.001$
Minimum difference for intergroup significance	4.37	8.72	6.90

Serum iron values are given in Table 2. The difference between PGVGJ and PGV was significant ($P<0.01$); no group differed from the controls though the levels after both PGV and SVP were significantly higher than those of PG. TIBC (Table 2) was significantly higher in all three gastrojejunostomy groups (TVGJ, SVGJ and PGVGJ) than in the controls. It was also significantly higher in the PG group ($P<0.001$) compared to controls. The percentage iron saturation in both PGVGJ and PG groups was significantly lower than the controls.

TABLE 3

Ferritin and Frequency of Low Values

Group	*Mean Values* ($\mu g/l$)	*% of Group with Low Ferritin* ($<20\mu g/l$)
TVGJ	94.50 (14)	21.4
TVP	65.43 (35)	14.3
SVGJ	53.88 (57)	29.8
SVP	61.91 (55)	26.5
PGVGJ	79.78 (9)	33.3
PGV	93.31 (39)	17.9
PG	49.85 (34)	39.7
Controls	89.11 (27)	18.5
F	2.55	$Chi^2=814$
	7,262 D.F.	7 D.F.
	$P<0.05$	N.S.

Minimum difference for intergroup significance 40.2

Mean levels of ferritin and the percentage of patients with values below $10\mu g/l$ are shown in Table 3. The ferritin levels of TVGJ and PGV were significantly higher than the PG group ($P<0.05$). However, none of the vagotomy groups differed from the controls.

Haemoglobin levels are shown in Table 4. The mean level of 13.87 g/dl after TVGJ did not differ from the controls but was significantly lower than PGV. The differences between PG on the one hand and PGV and the controls were highly significant ($P<0.001$). The percentage of individuals with anaemia is based on a lower limit of normal of 13 g/dl for men and 12 g/dl for women. According to these criteria, there was anaemia in 4.3 per cent of the control group and 31.5 per cent of PG. The Chi-squared test confirmed the existence of very highly significant differences between the groups ($Chi^2=32.242$ with 7 degrees of freedom

TABLE 4

Haemoglobin and Frequency of Anaemia

Group	*Haemoglobin Mean Values (g/dl)*	*% anaemic (men 13 g/dl, women 12 g/dl)*
TVGJ	13.87 (31)	22.6
TVP	14.23 (85)	14.1
SVGJ	14.13 (97)	14.4
SVP	14.47 (129)	7.7
PGVGJ	14.12 (17)	5.9
PGV	14.96 (50)	4.0
PG	13.23 (54)	31.5
Controls	14.67 (69)	4.3
F	6.46	$Chi^2=32.24$
	7,524 D.F.	7 D.F.
	$P<0.001$	$P<0.001$
Minimum difference for intergroup significance	0.82	

$P<0.001$). The trend was from 22.6 per cent after TVGJ through 14.4 per cent after SVGJ to 4 per cent after PGV.

There are theoretical reasons for expecting a difference between the two drainage procedures, therefore they have been compared in Table 5. The TIBC ($P<0.001$) and the percentage saturation ($P<0.01$) alone exhibit significant differences, both favouring pyloroplasty.

TABLE 5

Comparison of Gastrojejunostomy and Pyloroplasty

	GJ	*P*	
Haemoglobin (mean)	14.07 (145)	14.38 (214)	$t=1.77$, N.S.
Anaemic (%)	15.17 (145)	10.38 (214)	$Chi^2=1.49$, N.S.
Ferritin (mean)	63.90 (80)	63.28 (90)	$t=0.07$, N.S.
Iron Deficient (%)	28.75 (80)	21.11 (90)	$Chi^2=1.15$, N.S.
Fe (mean)	18.89 (139)	19.68 (181)	$t=0.88$, N.S.
TIBC (mean)	79.17 (120)	71.55 (164)	$t=4.14$, $P<0.001$
% Saturation (mean)	24.99 (120)	28.91 (162)	$t=2.63$, $P<0.01$

DISCUSSION

The frequency of anaemia in the PG group (31.5 per cent) was close to the average prevalence found by Deller & Witts. All parameters indicated a poor iron status after PG so that the "quality control" was acceptable.

The study set out to measure the patients' iron status in as many ways as practicable, including the use of a ferritin assay which theoretically is the best method. Walters[9] established that each microgram per litre of circulating ferritin represents 8 mg of storage iron, thus providing a relationship for ready estimation of the body's stores. Using this formula the mean total body iron of the PGV group and controls would be approximately 700 mg, substantially greater than the 400 mg of the PG group. Ferritin immunoassay has certain real advantages. The result may be obtained from a serum sample of 0.05 ml and it can demonstrate impending abnormality before iron stocks are exhausted. The highest mean ferritin level was found in the TVGJ group. There is no ready explanation for this anomaly but the group comprised only 1.4 patients and the results were influenced by two high ferritin levels of 260 and 300 μg/l respectively. The finding of 11.1 per cent of low ferritin levels in the controls is at first surprising but it should be remembered that iron deficiency is by no means uncommon; figures of 22 per cent for women and 6 per cent for men have been recorded[10]. Low haemoglobin values were found in 4.3 per cent of the controls. Ferritin depletion antedates iron deficiency anaemia so it is consistent that low ferritin results were found in more than 10 per cent of our controls.

Studies of the results of serum iron revealed no significant difference in any of the vagotomy groups. On the other hand, TIBC was higher in all three gastrojejunostomy groups compared to those who underwent pyloroplasty. These results are at variance with the levels obtained for serum iron and ferritin and would suggest the existence of a compensatory mechanism following gastrojejunostomy. Apart from the partial gastrectomy group, the percentage iron saturation was low only after PGVGJ, a small group of 10 patients.

On examination of haemoglobin levels there appeared to be a trend favouring PGV but none of the vagotomies differed significantly from the controls.

There are theoretical reasons for expecting poor iron absorption after vagotomy and drainage. The operations cause rapid gastric emptying and intestinal hurry. Whether vagal denervation of the duodenum, as in truncal vagotomy, influences iron absorption is not known. When the duodenum and proximal jejunum is by-passed, impairment of absorption can be expected[11]. Our data give limited support to this concept. The proportion of anaemia is much less than in either Wastell's[11] or Wheldon's[2] series.

The study indicates that iron deficiency and anaemia are not a major problem after any type of vagotomy. There is no tendency for iron status to deteriorate with time. There is limited evidence to suggest that gastrojejunostomy may cause anaemia and that vagotomy with drainage fares worse than proximal gastric vagotomy without drainage. There is no clear evidence that selective vagotomy with either type of drainage fares better than truncal vagotomy.

SUMMARY

THE iron status of 421 patients from five to 15 years after six types of vagotomy with and without drainage was measured and compared with a group of 70 controls and 54 patients after partial gastrectomy. Serum iron, total iron binding capacity, percentage saturation, haemoglobin and ferritin were measured.

The possibility of deterioration with time between five and 15 years was investigated but not found in any of the vagotomy groups.

There was no gross iron deficit after any type of vagotomy. The results of proximal gastric vagotomy were similar to the controls. The more sophisticated the surgery, i.e. the lesser the extent of denervation, the better was the iron status of the patient.

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DOWN SYNDROME ASSOCIATED WITH A FAMILIAL 14/21 TRANSLOCATION

by

EJ HANNA, WP JOHNSTON, NC NEVIN

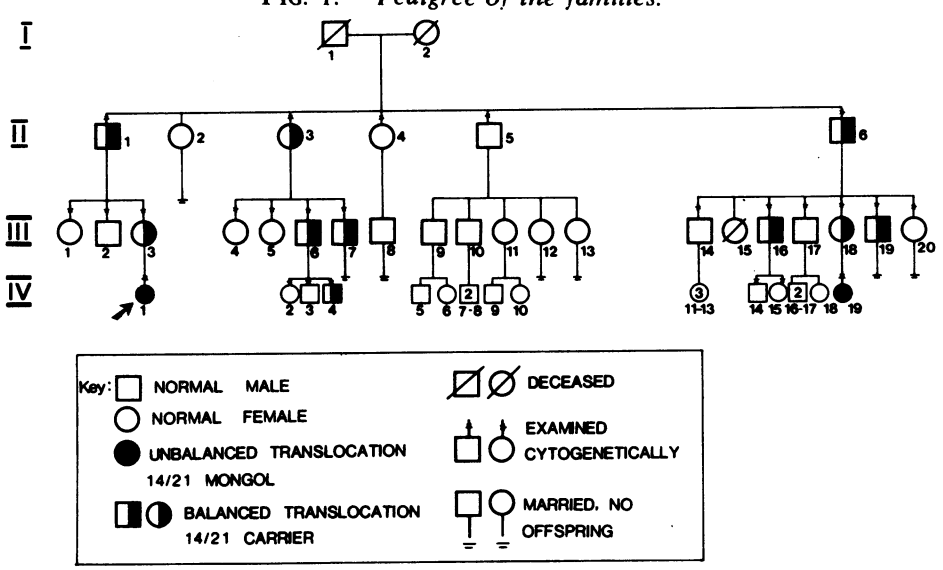
Department of Medical Genetics, The Queen's University of Belfast
and Royal Victoria Hospital

Down syndrome (mongolism) is one of the commonest chromosomal abnormalities in man with an incidence of between 1 and 2 per 1000 live births. In Northern Ireland the incidence is 1 in 630[1]. About 95 per cent have 47 instead of 46 chromosomes with an extra chromosome 21 (Trisomy 21). A small proportion, between 2 and 5 per cent are due to an unbalanced chromosomal translocation, usually involving the D(13-15) group chromosomes, particularly chromosomes 14 and 21. About half of the cases of translocation Down syndrome are inherited and thus, other family relatives may carry the chromosomal translocation and have a high risk of having affected children. The purpose of this paper is to describe our recent experience with a family which illustrates the importance of cytogenetic examination in all Down syndrome infants, of family follow-up when an unbalanced translocation is discovered, and of providing genetic counselling for those relatives who are carriers.

PATIENTS

CASE 1. The propositus (Fig. 1. IV.1), a female, born 11 April 1980, was the

FIG. 1. *Pedigree of the families.*



first born of a 27 year-old mother and a 28 year-old father. Birthweight was 3203 gms. at 38 weeks gestation. Clinical examination revealed the typical facial appearance of Down syndrome. Cytogenetic studies showed an unbalanced translocation, 46,XX,-14,t(14: 21) (Fig 2). Blood from the parents revealed that the mother was a balanced translocation (45,XX,t(14: 21) (Fig. 3).

FIG. 2. *Karyotype of the unbalanced translocation. Arrow indicates the translocation (t(14q21q)).*

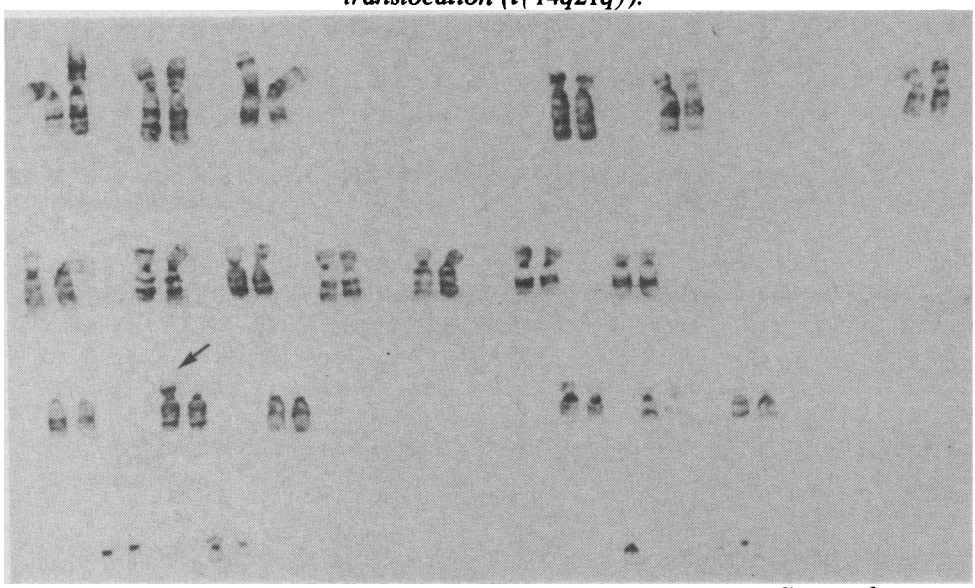
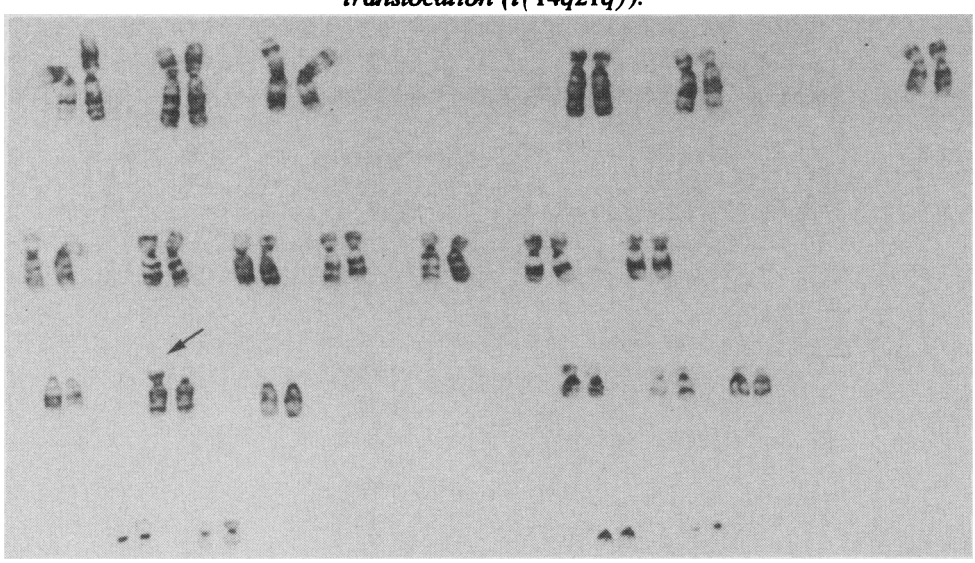


FIG. 3 *Karyotype of the balanced translocation. Arrow indicates the translocation (t(14q21q)).*



CASE 2. A female (Fig. 1. IV.19), born 7 July 1980, was the first born of a 24 year-old mother and a 28 year-old father. Birthweight was 3175 gms. at 40 weeks gestation. The baby had the typical facial appearance of Down syndrome. Cytogenetic studies showed an unbalanced translocation (46,XX-14t(14: 21). Blood from the parents demonstrated that the mother had a balanced translocation (45,XX,t(14: 21). The mothers of Case 1 and Case 2 were paternal first cousins.

Relatives of the families were visited and blood samples obtained for cytogenetic examination in a total of 21 other relatives. Apart from the above two Down syndrome infants (Case 1 and Case 2) no other relative was affected.

RESULTS

There was no difficulty in persuading the relatives to provide a blood sample for chromosome analysis once the situation had been carefully explained. Of the 21 relatives examined, 8 (7 males and 1 female) were discovered to have a balanced 14/21 translocation. Of the 10 balanced translocation carriers in the family, 7 had children. Three female balanced translocation parents produced a total of 6 children; 2 normal infants, 2 with balanced translocations, and 2 with Down syndrome. The four male balanced carrier parents produced a total of 15 children; 9 normal infants, 5 with balanced translocations, and one unknown (died in infancy).

DISCUSSION

The incidence of Down syndrome is between 1 and 2 per 1000 live births. Over 95 per cent of cases are caused by non-disjunction, the remainder resulting from translocation[2]. Down syndrome due to translocation can only be distinguished from that due to Trisomy 21 by cytogenetic examination. Our family illustrates the importance of cytogenetic examination in all Down syndrome newborn and of follow-up of the relatives when an unbalanced translocation is found. Among cases of unbalanced translocation Down syndrome about half are inherited. When inherited, the risk of maternal carriers of a 14/21 translocation producing an infant with Down syndrome is approximately 10 per cent and about 2-3 per cent when the father is the carrier[3]. Similar risks are involved when the translocation involves chromosomes 21 and 22. However, if either parent has a balanced 21/21 translocation all pregnancies will be abnormal since the only alternatives are the unbalanced translocation Down syndrome and the lethal monosomy 21. Our family also emphasises that the risk of Down syndrome offspring is greater when the mother carries the translocation. Both Down syndrome infants had been born to females with balanced 14/21 translocations. The four males with balanced 14/21 translocations had a total of 15 children, of whom 9 had normal chromosome constitutions, 5 had balanced 14/21 translocations, and one was unknown (died in infancy). When individuals in this family had been identified as having balanced translocations, they were advised of the risk of having an infant with the Down syndrome and of the availability of prenatal diagnosis by cytogenetic examination of cultured amniotic fluid cells in any future pregnancy.

SUMMARY

The finding of an unbalanced translocation t(14q21q) in two Down syndrome infants, born within three months of each other to mothers who were paternal first cousins, led to a clinical and cytogenetic investigation of the families. Chromosome examination in 25 individuals revealed 10 balanced translocation carriers, 3 females and 7 males. Both Down syndrome babies had been born to balanced female carriers. The study emphasises the importance of chromosome examination in all Down syndrome infants, and in relatives when an unbalanced chromosome constitution is discovered.

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INTENSIVE CARE IN THE BELFAST CITY HOSPITAL

The First Two Years — an Audit

by

J P ALEXANDER, FRCPI, FFARCS, J A S GAMBLE, MD, FFARCS
T H GAWLEY, MD, FFARCS, K A GEORGE, MS, FFARCS
The Intensive Care Unit, Belfast City Hospital

THE Intensive Care Unit (ICU) in the Belfast City Hospital was opened on 1st September, 1978. The object of this paper is to present an audit of the first two years work. The ICU is a four-bedded unit which was intended to provide support for the needs of the acute services of the South Belfast Group. The main hospitals are the Belfast City Hospital (1116 beds) and the Musgrave Park Hospital (700 beds). A breakdown of the bed complements is shown in Table 1. It can be seen that there are a total of 600 beds providing acute medical, surgical and gynaecological care. Patients requiring specialised investigations or surgical intervention for cardio-thoracic or neurosurgical problems are referred directly or indirectly to the Respiratory Intensive Care Unit at the Royal Victoria Hospital.

TABLE 1

Bed complement of the Belfast City and Musgrave Hospitals.

Belfast City Hospital		*Musgrave Park Hospital*	
Total Beds	1116	Total Beds	700
General Medicine	180	General Medicine	82
Surgery	142	Surgery	80
Acute Orthopaedics	48	Gynaecology	32
Gynaecology	34	Orthopaedic	300
Obstetrics	79	Other	206
Other	633		

THE UNIT

The building is a converted staff canteen adjacent to the main operating theatres. It had previously been used as a postoperative recovery room. Two beds are in separate cubicles, while two more beds are situated in the large open area. The lay-out of the building does not lend itself to easy expansion beyond four beds.

The function of an ICU will not be defined. The unit was set up because anaesthetists and surgeons were dissatisfied with the quality of care that patients were received after major surgical interventions. In addition, the new Central

Hospital is designed to include an eight-bedded ICU, and it was considered essential that experience in this type of work be gained before the new hospital opens. The unit opened with sufficient nursing staff to care for two critically ill patients. This was patently inadequate to serve the needs of 600 acute beds, and led to immediate problems when excessive demands were made on the unit. At the end of the first year staff shortages due to resignations and sickness were so acute that for a time only one patient could be cared for. The second year has been fraught with fewer difficulties of this kind with the recognition of the demands made on the unit, and the need to maintain a high nurse to patient ratio.

Medical cover has been shared by four consultant anaesthetists, with one junior anaesthetist assigned on a monthly rotation, and additional night cover using a three or four tier on-call rota.

ADMISSIONS

The admission policy has been unrestricted. Providing there is a reasonable prospect of restoring the patient to an acceptable level of health all who require facilities for mechanical support of breathing, close monitoring, or intensive therapy are accepted. The number of patients admitted in the first year was 227, giving a bed occupancy of 2.1 (53 per cent of maximum). In the second year there were 162 admissions, and the bed occupancy averaged 2.6 (65 per cent) and ranged from 35 to 95 per cent on a monthly average. Admission categories are shown in Table 2, and it is clear that the majority were postoperative; these patients required either respiratory support, controlled oxygen therapy, electrocardiographic monitoring, or close observation of vital functions.

TABLE 2

Primary Reason for Admission to Intensive Care Unit.

Reason for Admission	*Number of Patients*	*Per cent of Admissions*
Postoperative	269	69.2
Respiratory failure	47	12.1
Neurological (head injury, epilepsy)	37	9.5
Overdose	24	6.2
Abdominal (pancreatitis, etc)	8	2.1
Cardiac	2	0.5
Renal failure	2	0.5
Total	389	

The average age of the patients was 54 years (range 10-90), and 50 per cent were aged 60 or more. Sixty per cent were male. The length of stay in the ICU varied from a few hours to 104 days, the average being 4.1 days. Twenty-one per

cent of patients were ventilated with a mechanical respirator (average time of ventilation was six days), while a further 5 per cent were intubated and received controlled oxygen therapy via a 'T-piece' without artificial ventilation.

DEATHS

Eleven per cent of the patients admitted during the first year died in the unit. The mortality during the second year was 14.8 per cent, giving an overall mortality for the two year period of 12.6 per cent. The average age of the patients who died was 62 years. There was a "late" mortality (after discharge from the unit but before discharge from hospital) of 3.6 per cent and the average age of these patients was 56 years.

It is not always possible to give a precise cause for death. Of the 49 deaths which occurred in the unit, 75 per cent were considered to be due to a combination of respiratory failure combined with a surgical complication (sepsis, renal failure or shock). Some patients who survived for several days in the unit developed "multi-organ" or multiple systems failure"[1] with almost simultaneous failure of a number of major organs including the heart, liver, kidneys and brain. The remaining deaths were nearly equally divided between primary cardiac or neurological causes, with only two deaths being due to overdose and one to primary renal failure.

PSYCHOLOGICAL PROBLEMS

Intensive care units are a potent source of psychological problems[2,3]. Anxiety, exhaustion and communication difficulties have been identified as particular problems. Tomlin[4] described reactive apathetic depression occurring when the patient had got over the worst of his illness and was apparently getting better. Marked intolerance to physical disturbance by nurses and physiotherapists was a feature and these patients may inflict physical harm on their attendants. Some patients are most reluctant to allow the ventilator to be removed from the bedside when weaning after respiratory failure has been prolonged or difficult. About one quarter of all patients had no memory of their stay in the ICU, although, unlike patients in coronary care units, most were pleased to return to their own ward since this was a land-mark in their recovery[5].

Psychological pressures are just as severe for the nursing staff.[6] The close contact with death, the inability to relax, the technical equipment which has to be mastered and the general air of anxiety is too much for some to cope with. The attitudes of medical staff and senior nursing administrators can make or break the morale of a unit. A degree of over-staffing may be necessary to cope with unexpected absences and illness. Background music is helpful to both staff and patients. Short periods of relative inactivity should be welcomed as an opportunity for teaching and to allow the staff to recharge mental batteries.

CONTROL OF INFECTION

Intensive care units present an obvious hazard to the patients being treated in them. The accumulation of a number of compromised hosts in a small area provides a fertile medium for the development and spread of infection. On two occasions *Pseudomonas aeruginosa* has colonised all the patients in the unit. These episodes presumably reflect transfer of organisms from patient to patient by the staff. Such problems have largely been eliminated by common-sense restriction of equipment to one patient only and allocation of one nurse to look after one patient during her tour of duty. On one occasion, *Klebsiella aerogenes* was probably passed from one patient to another by a breakdown in ventilator sterilization procedures. Patients with potentially infective lesions or those who have an increased susceptibility to infection are, whenever possible, nursed in a cubicle.

COST

The exact cost of patient care under the National Health Service is remarkably difficult to determine. We have attempted to calculate the annual cost of running the unit and the cost of care per patient/day. The basic salaries of the nursing staff and auxillaries are known. Medical staff salaries are assumed to be the equivalent of a full-time consultant and two senior house officers. Overtime payments are not included. Central sterile and pharmaceutical supplies were costed over three months and averaged over a year with allowance for bed occupancy. These may well be underestimated since both these departments are reassessing their methods of estimating costs. Bills for routine and emergency servicing of equipment are included. However, costs of heating, light, power, X-rays, laboratory and other special investigations are not included. All prices are as of January, 1980 (Table 3). The average daily cost per patient was £148 and the true cost probably nearer £170. This was approximately 3.5 times the average per patient/day for the hospital as a whole.[7]

TABLE 3

Estimated annual costs of running the ICU for the two years September, 1978 to August, 1980. Prices as of January, 1980. Capital equipment totalled approximately £60,000.

Item	*Cost (£)*
Nursing and auxillary	55,000
Medical	27,000
Central Sterile Supplies	23,000
Pharmaceutical Supplies	21,000
Routine maintenance of equipment and emergency repair	2,500
Total	128,500

Comparing costs of different units is probably irrelevent since the work undertaken can be so varied. However, some observations on our figures may be of interest. Salaries and wages are a major item of expenditure and since these units are so demanding of manpower, economics cannot be made here. Suction catheters made up from one third to one half of the cost of sterile supplies in any month. We have tried re-sterilising these, but the results were highly unsatisfactory. Of the pharmaceutical items, antibiotics made up 24 per cent, intravenous and parenteral feeding solutions 18 per cent, and muscle relaxants (for patients being artifically ventilated) 12 per cent, the annual cost of the latter item being nearly £3000.

An alternative method of costing patient care was suggested by Cullen.[7] He used the Therapeutic Intervention Scoring System which attempts to classify the severity of illness by quantitating the therapeutic interventions, these being scored on a 1 to 4 basis, according to the time and effort required for nursing care. For example, routine ECG monitoring would score 1 point, a central venous pressure (CV_1') line 2 points, an arterial line 3 points and a pulmonary artery catheter 4 points. This approach overlooks the prophylactic value of intensive care, where a simple manoeuvre such as chest physiotherapy or ECG monitoring and arrhythmia detection (which score one point each) may prevent a major crisis requiring bronchoscopy or cardio-pulmonary resuscitation which score 4 points.[1]

DISCUSSION

Three hundred and eighty-nine patients were admitted to the unit in the period under review. One hundred and forty-one were critically ill, 49 died in the unit and a further 12 died later, so that perhaps 80 patients (21 per cent of admissions) might have died without intensive care. If the unit existed purely to save life, then each life "saved" cost an average of £3200. Tomlin[1] considered that 80 per cent of the patients admitted to his ICU would not have survived without intensive care facilities, but this figure seems unduly optimistic.

Our experience in the treatment of septic shock confirmed the value of aggressive therapy and early institution of intermittent positive-pressure ventilation.[8] An aggressive approach to postoperative surgical problems was also found to be beneficial in terms of patient survival. On the other hand, severely ill elderly patients who continue to require a high level of therapeutic intervention after days of intensive care were unlikely to survive, and put a considerable strain on manpower and resources. A reliable means of predicting an unsatisfactory outcome would be of great value.

Is intensive care justified? Some would argue that intensive care units remove the most skilled nurses from the wards, others that the ability of ward personnel to deal with sick patients lessens as difficult cases become sequestered in the ICU. On the other hand, no one will deny the value of close observation of ill patients. During periods when the ICU was full, several postoperative deaths occurred in the wards. These deaths were probable preventable. Tarhan et al[9] showed that

patients who had suffered a myocardial infarction 6 months or more before surgery had a 5 per cent incidence of re-infarction postoperatively, that half the infarcts were fatal, and that a significantly higher number of infarctions occurred on the third day after surgery.

Apart from considerations of mortality, there are obvious advantages in concentrating highly skilled nurses and sophisticated, expensive equipment in one area. An unknown number of patients may have benefited from careful monitoring and supervision in the early postoperative period. Pain relief with local anaesthetics or small doses of morphine injected through an epidural catheter has virtually abolished postoperative distress in selected patients. The experience gained in the unit can be applied to less ill patients in both the operating theatres and in the wards. Procedures which are now standard in the ICU are being used with increasing frequency in other areas of the hospital, hopefully leading to a corresponding reduction in morbidity and hospital stay.

SUMMARY

The Intensive Care Unit in the Belfast City Hospital opened on 1st September, 1978. In the first two years, 389 patients were admitted. Of 141 patients considered to be critically ill on admission, 80 survived who would probably have died without intensive care. This form of care is expensive, the daily cost being about three and a half times the average for a hospitalised patient.

We are indebted to all the nursing, medical and ancillary staff who have worked in the unit, without whose enthusiasm and devotion this paper could not have been written.

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INVASIVE LOBULAR CARCINOMA OF THE BREAST

by

DOROTHY HAYES and LINDA M CAUGHLEY,
The Laboratories, Belfast City Hospital BT9 7AD

The classical pattern of invasive lobular carcinoma (ILC) has become well established since the tumour was first designated in the 1940's. It is a distinct histological type of breast cancer, yet the incidence rates accumulated from the literature are widely divergent and vary from 0.7-20 per cent,[1] suggesting the criteria for diagnosis are not well defined. Martinez and Azzopardi[1] in the only series reported from the United Kingdom studied in detail the histological criteria for diagnosis and discussed and illustrated the recently described rarer variants. This study, and the problems of management posed by lobular carcinoma, encouraged us to review a censecutive series of invasive breast carcinomas from our records presenting during 1979. The incidence of ILC, with and without the in situ lesion, was assessed, the rarer variants of the tumour were sought and some of the problems presented by this diagnosis considered.

MATERIALS AND METHODS

The material studied comprised 298 consecutive invasive carcinomas of the female breast in a Caucasian population presenting during the year 1979. Five carcinomas diagnosed on needle biopsy and without a subsequent mastectomy specimen were excluded on the basis of inadequate tissue for classification. This left 293 invasive carcinomas in the series diagnosed on either a local excision (lumpectomy) or a mastectomy specimen. Lumpectomy specimens were adequately sampled for diagnosis (average 5.2 blocks per case). They were retained in the series, as their exclusion would have removed patients in the eighth and ninth decades of life, often preferentially treated by local exision of the tumour.

In the carcinomas reviewed the classical pattern of ILC was easily recognised, and separated from invasive duct carcinoma (IDC). Its incidence along with the co-existing in situ lesion was assessed, and the variants summarised and illustrated by Martinez and Azzopardi[1] were sought.

RESULTS

The review of 293 invasive breast carcinomas identified 34 lobular carcinomas (11.6 per cent), 254 duct carcinomas (86.7 per cent) and five carcinomas which could not be classified (1.7 per cent). The duct carcinomas were not further separated.

In four of the lobular carcinomas, variant pattern dominated; two tumours had a solid pattern with sheets of confluent uniform cells and sparse hyalinised stroma and two were tubulolobular in pattern with small open and solid tubules

in a targetoid arrangement. Variant patterns more limited in extent were not unusual in the 30 invasive lobular carcinomas with classical histology. They included two tumours with small foci of closed and open tubules, two tumours with an alveolar arrangement, one tumour with signet-ring cell carcinoma and one tumour with a solid area containing ill-defined clumps of cells. These variations in pattern did not create any diagnostic difficulties, but their recognition is relatively recent.

The in situ lesion of lobular carcinoma involving either lobules or ducts or both was associated with 33 of all carcinomas examined (11.4 per cent). Lobular carcinoma in situ (LCIS) occurred in 28 of the 34 lobular carcinomas (79.1 per cent) and in six of the 254 duct carcinomas (2.4 per cent). In the four lobular carcinomas dominated by variant patterns, in situ lesions were present in one of the solid and two of the tubulolobular carcinomas.

Lymph nodes were available from 17 cases of ILC. In eight cases the lymph nodes contained metastases which maintained a small dissociated cell pattern without sclerosis or structure formation. A tubulolobular carcinoma with lymph node metastases had the same histology as classical ILC.

DISCUSSION

Classical ILC has distinctive morphology with a small cell infiltrating pattern in a targetoid arrangement around uninvolved ducts. Associated LCIS involving lobules or ducts or both may occur with all types of invasive breast carcinoma, but its predominant association with ILC is a useful indication of type.

In this series 11.6 per cent of the invasive breast carcinomas were infiltrating lobular in type, and LCIS co-existed in 79.1 per cent of the tumours. The in situ lesion was not confined to lobular carcinoma and 2.4 per cent of the infiltrating duct carcinomas had concurrent LCIS. While the incidence of ILC is lower than the 14.7 per cent found in the other comparable United Kingdom series,[1] the concurrent incidence of LCIS is almost identical and supports the validity of the diagnosis.[2]

The divergent incident rates of ILC reported from comparable recent series mainly originating in the USA[1] are unlikely to be due to racial or geographic differences, and the lack of established criteria for diagnosis and failure to recognise the recently described variants are more important factors. With the now better definition of the histological spectrum of ILC incident rates should become more uniform.

Four variant forms of ILC occurred, two dominated by the solid pattern of Fechner[3] and two by the tubulolobular carcinoma described by Fisher and associates.[4] Co-existing LCIS in the tubulolobular carcinomas and in one of the solid tumours was a useful aid in diagnosis. It is unlikely these variants would have been recognised and included in series of infiltrating lobular carcinomas prior to these reports. While ILC dominated by solid and tubulolobular patterns is rare it is not unusual for some minor variation in pattern to occur in association

with the classical histology. If as has recently been suggested [3], [4] variant patterns represent differentiation in ILC and possibily a better prognosis their recognition may be useful.

Cases of ILC present over a wide age range, but an unusually high predominance of patients was found in the later decades (Table) and also a high mean age of 61 years. This is almost ten years older than the mean age 52.6 years of several

Age	<40	40-49	50-59	60-69	70-79	>80
Number of patients	0	6	9	10	7	2

Table — Age range of 34 patients with invasive lobular carcinoma of the breast.

series taken together,[2] but the tumours in the older age range continue to have concomitant LCIS. The two patients in the ninth decade both had LCIS accompanying the invasive lobular carcinoma and extension of the malignancy into this decade has previously been reported.[5]

The identification of an invasive breast carcinoma as lobular in type has some practical importance in predicting the response of metastatic disease to endocrine manipulation. A high proportion (85-90 per cent) [6], [7] of infiltrating lobular carcinomas are oestrogen receptor positive, and in the absence of tissue assay the histological type of the tumour provides strong presumptive evidence of a positive status which relates to regression with endocrine therapy.

The bilaterality of ILC and its preceding lesion, LCIS, is well recognised, and 26-30 per cent of infiltrating lobular carcinomas are associated with subsequent invasive carcinoma, usually lobular in type, in the contralateral breast. [8] [9] More recently, a report describes 30 cases of ILC with a 16.6 per cent incidence of subsequent invasive carcinoma in the opposite breast in a minimum follow-up of four years.[1] Since 11.6 per cent of invasive carcinomas in this series are lobular in type, the diagnosis poses a considerable problem if supervision is to detect a possible contralateral carcinoma at its earliest stage and before metastases have occurred and thus ensure the lowest possible mortality. This problem of clinical management is greatly magnified if the whole spectrum of lobular carcinoma is considered. The in situ lesion which does not by itself produce a tumour nodule[10] is being increasingly recognised on histological examination as an incidental finding in breast tissue usually excited for fibrocystic disease (diffuse fibroadenosis). It identifies a further group of patients having a future risk of invasive breast carcinoma seven to 12 times greater than a normal population of women.[11] Since the risk is known to be equal in both breasts ipsilateral mastectomy, the usual treatment prior to 1975, is no longer acceptable and a conservative policy of management is replacing surgery.

The diagnosis, therefore, of ILC and/or its preceding in situ lesion indicates a potential bilateral breast malignancy extending over a wide age span, which creates a formidable problem of follow-up both for the clinician in charge and the patient, to detect subsequent invasive cancer at its earliest possible stage.

SUMMARY

Thirty-four invasive lobular carcinomas, including four tumours with variant patterns, were found in a review of 293 consecutive invasive breast carcinomas, an incidence of 11.6 per cent. The in situ lesion was associated with 79 per cent of the tumours. The recognition of this type of breast carcinoma, with its long-term bilateral risk, poses considerable problems in follow-up, both for the clinician and the patient.

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TECHNOLOGY AND THE DISABLED

The Development of the Northern Ireland Prosthetics/Orthotics/Aids Service

W. V. JAMES, F.R.C.S.
Musgrave Park Hospital, Belfast

HERODITUS, in 484 BC made one of the first of many references to the use of devices for the disabled, but it was not until the 1800's that the inventiveness of the Victorian era was to apply technology to the problem of the disabled on a large scale. The impression is that since the Victorians, we have not advanced all that much, although we are on the brink of another era of advance.

In 1800, James Potts of London produced an artificial leg with an articulated knee and ankle, and the ankle even had a 'toe lift' during the swing phase. The leg acquired fame by being provided for the Marquis of Anglesea, who had an above-knee amputation at the battle of Waterloo in 1815. The limb bears a remarkable resemblance to those produced today. Verduin, in 1696 produced a below-knee limb that was reintroduced by Serre in 1826, and this also is remarkable like the 'Number 8' limb of today. Ernst, who was an orthopaedic technician, published 'Orphopaedic Apparatus' in 1861, which contains an illustration of 'Dr. Little's walking Apparatus', which is almost identical to the long-leg weight-relieving caliper of today. It would seem that the disabled of today are coasting along on the inventiveness of the Victorian.

There are signs of an upsurge in the application of technology to assist the disabled. In the 1960's, engineers working with orthopaedic surgeons in the United States started to scientifically investigate the basic problems of the disabled. Rehabilitation engineering centres were created, based on university orthopaedic hospitals. Rehabilitation engineering is that aspect of bio-engineering that is applied to the long-term disabled, and amongst other things, includes artificial limbs, appliances, aids, mobility and environmental control. Further evidence of change lies in the use of the word 'prosthetics' to refer to the use of artificial limbs, and the word 'orthotics to refer to the use of appliances. The basic research resulted in devices th'at had to be developed and evaluated, and then introduced to the disabled. This is necessarily a long slow process, but the results of the work are now starting to come through.

The 1970's saw the introduction of several bio-engineering courses in the United Kingdom, and even the medical profession became aware of the term. Centres at the University of Strathclyde and the Paddington Technical College also started three year courses for prosthetists and orthotists, to replace the old apprenticeship system. At several universities and other centres, units were set up where doctors and bio-engineers collaberated in the application of technology to the disabled.

On the international plane, the International Society for Prosthetics and

Orthotics was formed, which also took in the rehabilitation engineering field, and provided a world-wide flow of information between centres, and promoted collaberation.

NORTHERN IRELAND EVENTS

In the early 1970's, doctors, the administration and the voluntary organisations became aware of the events taking place in the care of the disabled, and the need to upgrade services. The Northern Ireland Hospitals Authority, set up a working party in 1973, under the chairmanship of Mr. Roy Whitlock, to report on the prosthetic and orthotic services in Northern Ireland. At the same time a consultant visited the United States to see the advances that were taking place. Because of the reorganisation of the Health Service, the report was presented to the Eastern Board in 1975. The main recommendations included the building of an orthotic workshop at Musgrave Park Hospital, to be leased to commercial orthotic manufacturers. This was a similar arrangement to the already existing commercial prosthetic workshop at the Limb Fitting Centre at Musgrave Park Hospital. It was also recommended that the Limb Fitting Service should be better integrated into the hospital and community services. Finally, it was recommended that a rehabilitation engineering centre should be built at Musgrave Park Hospital, similar to those seen in the United States, and this would provide specialised services in that field. The recommendations were accepted by the Eastern Board, and with commendable speed, the Rehabilitation Engineering Centre and the contractors commercial workshop were completed in 1979. At the same time the Eastern Board made provision for six trainees to be sent to the three-year course at the University of Strathclyde to become qualified prosthetists/orthotists.

After the opening of the Rehabilitation Engineering Centre, it was found difficult to integrate the Limb Fitting Service with the hospital and community service until it was integrated with the other services for the disabled. It was also apparent that there had been no provision for improving the supply of aids for the disabled. The decision was taken to combine the Limb Fitting Service and the Wheelchair Service with the Rehabilitation Engineering Centre, and to open an Aids Demonstration Centre. The latter was carried out in the area vacated by the invalid cars, which were being phased out. The combined service came into being in 1980, and was called the Prosthetics/Orthotics/ Aids Service.

There had always been co-operation in Northern Ireland between the Health Service and scientific departments of academic institutions in the province. With the opening of the Rehabilitation Engineering Centre, the Department of Technology of the Ulster Polytechnic and Queen's University, started a fruitful co-operation to apply technology to the disabled.

At the same time, the Department of Health continued to be aware of the need to improve the lot of the disabled. A grant was made to the Rehabilitation Engineering Centre to study gait. The OUTSET survey was also funded, to provide a survey of what the disabled felt that they needed, and this included technological needs. Finally, the Department undertook a reappraisal of the rehabilitation services by means of working parties, although implementation of findings will be hampered by the financial situation.

The organisation of the provision of devices to assist the disabled in the United Kingdom have long been a subject of examination. The BMA Planning Report of 1968 on 'Aids for the Disabled' spoke of glaring defects, and of administrative and educational deficiencies. It condemned the administration of the limb fitting separate from the Health Service, and suggested changes in almost every aspect of the service. The Denny Report of 1970 recommended the integration of the Scottish Limb Fitting Service with the Health Service and this has been implemented. Northern Ireland has gone further, and has provided an integrated service for the provision of artificial limbs, appliances and aids, and has also integrated this with education and research in these subjects. This is a unique situation in the United Kingdom, and indeed, there are few countries that have had the opportunity to provide such an overall coverage of the problems of the disabled.

THE PROSTHETICS/ORTHOTICS/AIDS SERVICE

There are obvious advantages in having all the services providing devices for the disabled on one site, and combining them. The staff providing the service are common to all, and consequently there is considerable co-operation. It encourages the 'total' approach to the disabled patients' problems, rather than a 'demand and supply' approach. To labour the point, the staff have an interest in 'the patient', rather than in the obvious primary disability.

There is also, because of the wider spectrum of responsibilities, an awareness of the need to improve services as a whole, in the hospital service and in the community. To this end there has been an active attempt to give information to all sections of the hospital and community services by means of courses, lectures, symposia and demonstrations. Many of the courses have specially prepared manuals provided from the Rehabilitation Engineering Centre, for suitable literature is scarce. This educational programme has proved popular, and at present demand outstrips supply.

Because of the volume of work, and the encouragement of referral of patients with difficult rehabilitation problems, It is inevitable that clinical research has to be carried out to solve individual problems. Sometimes, as the result of success with an individual, it is seen that a similar solution can be applied to a wider group. The capacity of the Rehabilitation Engineering Centre to undertake the design and manufacture of devices, and the regional connections of the service alows a widely available and specialised service in a neglected area of need. The device created to lift weak arms has been extensively applied to children with muscular dystrophy, and the 'Musgrave Park drop-foot polypropylene splint' is now in wide use. A Gait Analysis Laboratory has been set up with a research grant from the Department of Health, to investigate the effect of devices on gait. This has benefited another project to depict discrete pressure areas under the foot, which is financed from private funds.

THE REHABILITATION ENGINEERING CENTRE

Rehabilitation engineering is that branch of bio-engineering that deals with the long-term disabled. The Centre is based on an American concept, and provides specialised advice and devices over the whole spectrum of locomotor disability.

Until it came into being, there was no source of specialised advice about the application of technology to the disabled, nor was there the facility to provide individual devices of a specialised nature. The Centre operates clinics for rehabilitation problems, with an orthopaedic surgeon and bio-engineer, and they can call on specialised advice from the Limb Fitting Centre, the Wheelchair Service and the Aids Demonstration Centre.

There is a workshop with a capacity to make orthoses, prostheses, aids, mechanical, electrical and electronic devices. There is a Gait Analysis Laboratory, and computer facilities. The workshop is staffed by qualified prosthetists/orthotists, appliance makers and a fitter. They produce the devices that are not normally available from commercial sources.

Patients with problems are referred. If the solution is routine, then the patient is referred to a commercial source on the site. If the solution is complex, then a device can be created and made within the Centre. The Centre also has an interest in a wide variety of bio-engineering problems, and assists and promotes projects, ranging from orthopaedic instruments and implant devices, to projects for schools and universities.

THE LIMB FITTING CENTRE

The Service provides an assessment for all amputees, and the prescription of artificial limbs. These limbs are produced by commercial firms who have workshops within the Centre. Consultation with the Centre before amputation is encouraged, and also early referral after operation.

THE WHEELCHAIR SERVICE

Some 12,000 wheelchairs are on loan throughout the Province, and the chairs are supplied and maintained through the service. There are technical officers who visit the patients in their homes and in institutions, and there are clinics for patients with particular problems. There is also collaberation with the Rehabilitation Engineering Centre and the Aids Demonstration Centre to adapt existing wheelchairs and to provide suitable seating.

THE AIDS DEMONSTRATION CENTRE

The Centre at Musgrave Park Hospital has a wide variety of aids on display, which can be sampled by patients referred there. An occupational therapist provides assessment and advice, and closely collaberates with District Occupational Therapists. If assessment finds that no suitable device is available then there is collaberation with the Rehabilitation Engineering Centre to adapt an existing device, or to design and make a suitable device.

CONCLUSION

The disabled make use of a wide variety of devices to diminish disability. The 1980's will see an increasing interest in the provision of technology for the disabled. Northern Ireland, through the Prosthetics/Orthotics/Aids Service is in the unique position of having a Regional service that can undertake this type of work and can deliver the services to the disabled.

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THE TREATMENT OF FEMORAL SHAFT FRACTURES USING A CAST BRACE

by

PATRICK C. PYPER and TREVOR C. TAYLOR
Mater Infirmorum Hospital, Belfast

The current trend in the non-operative treatment of femoral shaft fractures is away from prolonged traction and towards early ambulation. The cast brace treatment involves external support to the limb in such a way that maximal use can occur during healing. Early ambulation, while a fracture is healing, provides an environment for complete bone healing as well as allowing joint movement so that the function of the limb is as near normal as possible at the completion of treatment. It is widely accepted that early ambulation of tibial fractures in plaster casts leads to union without significant complications [1 2 3]

The conventional method of treatment of femoral shaft fractures involves the use of skeletal traction in a Thomas's splint for a prolonged period. In the majority of cases this method is very satisfactory but it has certain inherent disadvantages, the major one being the long period of immobilisation required and therefore the greater difficulty in subsequent recovery of full function of the limb. The pressure of long term bed occupancy often places great strain on a hospital's resources particularly if the patent is treated in a general surgical unit.

Küntscher[4] in 1958 felt that external fixation of femoral shaft fractures was not satisfactory since complete immobilisation could not be achieved. His intramedullary nail technique has been widely accepted but has the disadvantage of introducing potential infection into a closed fracture. It is also inappropriate in distal or comminuted fractures.

Adair[5] has used a long leg quadrilateral plaster with good effect. However, in this method the knee is encased in plaster and so knee flexion returns slowly. Mooney[6] reported much better knee flexion using a cast brace instead of a traditional plaster of Paris spica and described the use of the cast brace in the treatment of distal femoral shaft fractures in 150 cases. He produced good healing in all cases with no cases of non-union or refracture in a mean healing time of 14.5 weeks. They applied the cast brace after seven weeks in traction. Connolly and King [7 8] found that the cast brace could be applied earlier at three to four weeks with no deterioration in results. Their incidence of non-union of one per cent is less than that occurring with the traditional method of Thomas's splint and traction. More recently it has been shown that the third week after injury is the best time to apply the cast brace in order to minimise ultimate shortening[9]. Brown and Preston[1] found satisfactory results in 68 out of 76 cases of fractures mainly in the middle and distal shaft. They felt that these results were sufficiently good to encourage the use of this treatment. Wardlaw[10] published a series in which 29 out of 31 (94 per cent) had a satisfactory (or

better) result compared to 30 out of 38 (79 per cent) treated by traction alone. He concluded that the cast brace method is a great advance in conservative treatment.

MATERIALS AND METHODS

Twelve cases of femoral shaft fractures have been treated with a cast brace in the Mater Hospital, Belfast between September 1978 and November 1979. During this time six other patients were treated for fractures in similar sites, one in traction until the fracture was united and five by internal fixation either as early definitive treatment or for delayed union. The details of the patients are given in

TABLE 1

Details of Patients and Treatment

	Age	*Sex*	*Fracture*	*Traction Time (wks)*	*Cast Brace Time (wks)*	*Hospital Time (wks)*	*Complication*
RA	16	F	L mid 1/3	7	8	8	—
EA	73	F	R mid 1/3	7	8	10	—
PB	16	M	R mid 1/3	5	10	7	—
WC	57	M	L low 1/3	9	6	13	f–L humerus f–L metatarsals
TC	20	M	L mid 1/3	8	8	9	DVT — PE
AG	26	M	L mid 1/3	6	12	12	Delayed union
FG	25	M	R mid 1/3*	8	15	9	Delayed union
DM	20	M	R mid 1/3	7+7	3 days	16	Refracture
AMcN	12	F	L mid 1/3	7	10	8	—
WS	42	M	L mid 1/3*	6	9	8	DVT
ER	24	F	L mid 1/3	6	10	7	—
GT	14	M	R mid 1/3	5	9	6	—

* Compound. DVT — Deep venous thrombosis. PE — Pulmonary embolus.

Table 1 and it can be seen that most ofthe fractures were closed injuries involving the mid shaft (Table 2). The anatomical definition of site was classified according to Dencker [11]. Two of the cases were compound, one as a result of a fall and one due to a gunshot injury.

TABLE 2

Anatomical Site of Fractures

Upper third	=	0
Middle third	=	9
Lower third	=	3

All patients were reviewed finally at times ranging from four to 29 months after the initial injury. Attempts were made to recall patients; the one last reviewed four months after injury had emigrated. At this time, hip, knee and ankle movements and stability were assessed; any shortening of the affected leg measured and wasting of quadriceps and calf muscles determined. The final position of the fracture was determined clinically by measuring intercondylar and intermalleolar distances and previous X-rays were reviewed.

CAST BRACE TECHNIQUE

The cast brace should be applied between three and eight weeks after the fracture has occurred. During this period conventional treatment by traction (skin or skeletal) with a Thomas's splint is applied. This delay allows for 'stickiness' to occur at the fracture site and so increases the stability of the limb when ambulation is started. It can be successfully used in femoral shaft fractures in the middle and lower thirds. It is particularly useful in comminuted fractures unsuitable for intramedullary nailing. Connolly, Dehne[8] felt that transverse fractures of the middle and upper thirds were better treated by internal fixation. The cast brace itself entails a quadrilateral plaster around the thigh hinged at knee joint level to a short leg plaster.

Application

Prior to application mild sedation is required, usually Diazepam 10 mg orally suffices. The traction apparatus is dismantled and the leg is gently held by an assistant. A special knitted cast sock is then placed from foot to groin and a stockinette bandage around the knee.

The next stage is the application of the thigh brace[10]. This part is not expected to provide ischial weight bearing though proximal total contact support of the thigh is essential. The plaster is applied and then shaped using an appropriate sized quadrilateral box. The box is open-ended and shaped at the brim and should be applied as high up the thigh as possible. The quadrilateral shape is essential to control rotation. This shape is extremely effective and it has been shown[7] that there is less rotation in the cast brace than in conventional traction apparatus. A below knee cast is then applied with the foot plantigrade..

Finally the hinge joints are positioned at the knee. These are dicentric hinges placed in parallel at the knee axis which is located two centimetres posterior to the mid line of the limb in the sagittal plane and at the level of the adductor tubercle. An alignment jig is used to hold the hinges parallel and jubilee clips are used to secure the arms of the hinges to the thigh and calf cylinders while the range of movement is tested. The arms of the hinges are malleable and so can easily be adjusted to suit any case. It is important that the hinges clear the femoral condyles by at least one centimetre. When the hinges are in the correct position they are fixed to the rest of the apparatus by plaster.

The patient is returned to the ward and the position of the fracture checked by X-ray. He is rested in bed for two days to allow the plaster to dry and then

gets up to walk. A boot is supplied for the foot and the patient is usually able to leave hospital one week after application of the cast brace.

RESULTS

Traction Time

It can be seen (Table 3) that traction time is reduced using this technique, a mean time of 8.5 weeks for all cases. Only one patient (DM), as a result of a refracture, had a traction time of greater than nine weeks.

Hospital Time

This was reduced to an average of 9.5 weeks. Three patients had a longer than average stay due to complications. One suffered a refracture, one had delayed union and a third had associated fractures of the humerus and metatarsals which hindered his mobilisation.

TABLE 3

Treatment Times

Average traction time	(weeks)	8.5 (range 5-14)
Average hospital time	(weeks)	9.5 (range 6-16)
Average treatment time	(weeks)	17.5

Fracture Healing

All patients were treated in the same way, that is, appropriate traction followed by application of the cast brace at a suitable time following injury. Three of the patients encountered problems in fracture healing.

(i) DM suffered a mid shaft fracture as a result of a road traffic accident. He seemed to have a healing fracture and after seven weeks a cast brace was applied. Three days later after vigorous straight leg-raising he complained of pain and X-ray revealed a refracture. After a further seven weeks in traction his fracture healed soundly.

(ii) AG suffered a comminuted mid shaft fracture of his left femur as a result of a road traffic accident. A cast brace was applied after six weeks. He had a varus angulation in the brace and after its removal complained of pain. A polypropylene brace was applied and the fracture healed.

(iii) FG suffered a compound mid shaft fracture of his right femur following a fall from a roof. He had a cast brace applied after eight weeks. On removal he complained of pain but no movement of the fracture was noted. His pain resolved after a further four weeks in the cast brace.

TABLE 4

Final Results

Time since accident	*(mths)*	*Shortening (cms)*	*Quadriceps wasting (cm)*	*Knee Movement Flex.*	*Ext.*	*Fracture Position*
RA	17	0	1.0	Full	Full	Good
EA	29	1.0	0	65°	Full	Valgus less than 10°
PB	4	1.0	0	120°	Full	Good
WC	19	0	1.0	110°	Full	Good
AG	24	3.0	1.0	120°	Full	Varus 18°
FG	21	1.0	0	Full	Full	Varus less than 10°
DM	23	0	0	125°	Full	Good
AMcN	7	1.0	0	Full	Full	Good
WS	10	0	1.0	Full	Full	Good
ER	7	<1.0	0	Full	Full	Good
GT	7	<1.0	0	Full	Full	Good
TC	21	0	0	120°	Full	Good

Hip and Knee Movement

All patients had a full range of hip movement at final review. Knee movements were also recorded at this time (Table 4). All patients had full extension at the knee and all but one had flexion greater than 110°. The exception (EA) was an elderly lady with a supracondylar fracture. Flexion was said to be full when it equalled that of the other normal knee. One patient (WC) demonstrated minimal colleteral ligament laxity.

Shortening

Only one patient suffered shortening greater than one centimetre. AG had three centimetres shortening as a result of his varus deformity.

Quadriceps Wasting

Four patients demonstrated some degree of wasting but none greater than one centimetre. Only one patient had calf wasting (0.5 cm) though one (TC) did have an increase of 3 centimetres as a result of his deep venous thrombosis.

Overall Results

The late results were grouped using the Dencker classification[11] which has four categories — excellent or good; satisfactory; poor; very poor. Ten of the patients

were in the good or excellent group (83 per cent) and two were in the satisfactory group (17 per cent). No patients were in the poor or very poor categories. The patients in the satisfactory group were EA with reduced knee flexion and AG with a significant varus deformity. No patients complained of pain in the affected leg at final review.

DISCUSSION

The proponents of fracture bracing cite three main advantages of this form of treatment, quicker fracture healing, earlier recovery of knee flexion and earlier discharge from hospital. Patients in the Mater Hospital are nursed in general surgical wards and therefore carry a higher risk of infection after surgery than those in a fracture unit. Mainly for this reason primary internal fixation of femoral shaft fractures in the middle and distal thirds is only occasionally undertaken. Therefore, to ease the considerable pressure on beds the cast brace technique was introduced. It is in fact possible to make a plastic brace but this requires specialised workshops and an extra delay of one week for construction and is very much more expensive. Using plaster of Paris the surgeon can apply the brace in 45 minutes and have the patient walking in two days. In none of our cases was there any structural problem with the brace and the patients were extremely happy with the treatment.

Whether or not the fractures in this series healed more quickly than they would have done with prolonged traction is difficult to know. One is reluctant to test the fracture too soon when the patient attends for follow-up, so usually the cast braces are not removed before 14 weeks after the injury. If the fractures healed any earlier we would not know.

Ultimate recovery of knee bending is easier to assess and was good in all but one of our patients. Eleven (92 per cent) had greater than 90° of knee flexion, and all had full extension. This compares favourably with Dencker's series where 12 per cent of patients with closed fractures of the femoral shaft had less than 90° of knee flexion, and Nichols' series in which 33 per cent had less than 90° of knee flexion in all types of closed femoral shaft fractures. An interesting feature of our patients was the fact that most of them were young and this probably aided the recovery of function.

Traction time and overall hospital time were both significantly reduced. The average traction time with our patients was 8.5 weeks but it is felt by others[7 8 9] that the cast brace can be safely applied much earlier than this. The shorter hospital stay (9.5 weeks) is a marked reduction as Wardlaw[10] found that conventional treatment needed an average hospital time of 15 weeks. This reduction is an obvious economic benefit and relieves the pressure on bed occupancy.

SUMMARY

In just over one year 12 patients with femoral shaft fractures were treated with a cast brace. Only one failure occurred requiring substitution of alternative

treatment and leading to a good result. We feel that fractures in the mid and distal shaft can be satisfactorily treated by plaster of Paris cast brace and would recommend this method with its advantages of shorter hospital stay and earlier ambulation.

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THE EFFECT OF ALCOHOL ON GASTRO-ENTERO-PANCREATIC HORMONES

R W HENRY, T LAVERY and K D BUCHANAN

Department of Medicine, The Queen's University of Belfast
Department of Biochemistry, Royal Victoria Hospital, Belfast

OWING to the association between alcohol and chronic pancreatitis in man[1] there has been considerable interest in the effect of alcohol upon gastro-entero pancreatic (GEP) hormones. Previous estimations of GEP hormones following oral alcohol ingestion have shown raised serum gastrin concentrations in response to 18 per cent and 15 per cent ethanol in man (Becker, Reeder and Thompson[2]), and Straus, Urbach and Yalow[3] showed an early secretin response to 60 ml of vodka (86° proof) whereas Llanos et al[4] showed a late secretin response at 90 minutes. Serum alcohol was not measured in these studies. We have re-studied the effect of oral alcohol upon GEP hormone release.

METHODS AND MATERIALS

The response of gastro-entero pancreatic hormones to 60 ml of 80° proof (40 per cent alcohol) Smirnoff Vodka were measured. There were 6 subjects (5 male and one female) aged from 22 to 30 years. All were within 90 to 110 per cent of ideal body weight. None had a family history of diabetes mellitus.

After an overnight fast (12 hours) a butterfly needle (19 gauge) was inserted in a vein in the ante-cubital fossa. Thirty minutes later a basal blood sample (time 'O') was taken and the sample assayed for serum alcohol (Calbiochem US Patent 2,926, 736 and foreign patents), blood sugar (using an auto-analyser) and plasma insulin, plasma gastrin, plasma glucagon (two antibodies were used — one reacting with the N-terminal (N-GLI) and one reacting with the C-terminal (C-GLI) regions of the glucagon molecule), plasma secretin [5], [6] and plasma vasoactive intestinal polypeptide (VIP) (J.E.S. Ardill, personal communication).

Each subject then drank 60 ml of vodka in less than 3 minutes. Following oral administration of alcohol serial blood samples were taken at 5, 10, 15, 30, 45, 60 and 90 minutes for measurements of hormones, serum alcohol and blood sugar.

RESULTS

Alcohol had been absorbed five minutes after ingestion and serum levels were highest 60 minutes after ingestion (Table). Blood sugar did not change throughout

	TIME (minutes)							
	0	5	10	15	30	45	60	90
GLUCOSE (mmol/l)	5.3±0.4	5.2±0.3	5.1±0.3	5.2±0.2	4.8±0.2	4.9±0.2	4.7±0.2	4.3±0.6
INSULIN (mU/l)	4.3±0.7	6.0±0.8*	4.5±0.8	5.8±1.3	5.3±0.7	4.3±0.8	4.3±0.6	4.3±0.6
GASTRIN (ng/l)	27±8	29±7	25±10)	24±8	26±8	24±6	26±8	19±6
N-GLI (ng/l)	125±13	113±14	118±12	122±13	111±10	106±10	111±12	118±9
C-GLI (ng/l)	102±11	104±12	102±13	92±12	105±12	84±10	95±14	96±12
VIP (ng/l)	97±9	74±4	82±4	71±4	63±9	72±6	68±11	84±8
SECRETIN (ng/l)	69±7	67±6	54±5	67±9	62±8	61±6	63±7	70±11
ALCOHOL (mmol/l)	0	2.2±0.7	3.7±0.7	5.9±0.7	11.1±1.3	11.6±1.5	8.7±1.3	6.7±0.9

Table Gastro-entero-pancreatic hormones (±S.E.M.) before and after 60 mls of 80° proof alcohol given orally.

* Significantly different ($p = < 0.05$) From basal

the study. Insulin was stimulated and had risen significantly at five minutes but had fallen to control values at ten minutes. None of the other hormones was affected by the alcohol.

DISCUSSION

This study demonstrates that 60 ml of vodka on an empty stomach gives peak serum levels of alcohol at about 60 minutes in healthy volunteers. Insulin release is stimulated but hypoglycaemia does not occur. This dose of alcohol, administered in this fashion, does not affect plasma gastrin or plasma secretin concentrations in the 90 minute period of the study. The lack of response of secretin is at variance with results obtained by Llanos et al[4] and Yalow and Berson[7] using similar doses of alcohol. We were also unable to show any response of plasma gastrin following oral alcohol administration; the concentration of alcohol used in our study was similar to (40 per cent compared to 50 per cent) the higher concentration used by Thomson, Becker and Reeder[2]. Insulin was stimulated but the rise was slight and we were unable to record any change in blood sugar or glucagon secondary to this change in insulin. Thus oral 80 per cent proof alcohol alone does not result in the release of gastro-entero pancreatic hormones, with the exception of insulin, in a period of 90 minutes after ingestion and in some other studies it may have been substances ingested with the alcohol that caused GEP hormone release.

SUMMARY

In response to oral alcohol plasma insulin rose transiently in a group of 6 human volunteers. Blood sugar did not change, nor did serum gastrin, serum alcohol, plasma glucagon or VIP over a period of one hour following alcohol ingestion.

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SOLITARY CAECAL DIVERTICULUM

CHRISTINE DEARDEN, FRCS, (Surgical Registrar)

D O TODD, MB, BCh, DRCOG, (Surgical Registrar)

W G HUMPHREYS, MD, FRCS, (Consultant Surgeon)

Surgical Unit, Waveney Hospital, Ballymena.

QUITE commonly a clinical diagnosis of acute appendicitis is not confirmed by the operative findings. On finding a normal appendix at laparotomy other pathology should be sought and on occasions unusual lesions may be detected as illustrated by the following cases, all of which presented in a District General Hospital during the last year.

CASE REPORTS:

Case 1. A 61 year old male was admitted with a two day history of anorexia, vomiting and abdominal pain which was initially central and crampy, becoming continuous in the right iliac fossa.

On examination he was dehydrated, pyrexial and had tenderness, guarding and rebound in the right iliac fossa, where it was thought that a mass was palpable; rectal examination revealed right sided tenderness. A diagnosis of appendicitis was made and at laparotomy through a right paramedian incision, the appendix was found to be normal, but a 3 cm. diameter mass was identified in the ileo-caecal angle which was considered to be a small carcinoma. A right hemicolectomy was performed with an uneventful recovery.

Case 2. A 54 year old male was referred with a 48 hour history of central abdominal pain, later referred to the right iliac fossa. The patient had been nauseated, but had not vomited and had no other symptoms.

Examination showed a fit man, pyrexial (37.8°C) with tenderness, guarding and minimal rebound in the right iliac fossa; there were no palpable masses and rectal examination revealed no abnormality. Acute appendicitis was diagnosed but at laparotomy, through a grid iron incision, the appendix was normal. There was a 1.5 cm. mass palpable in the posterior wall of the caecum. This was thought to be either a diverticular abscess or a neoplasm, and as the latter could not be excluded the incision was extended and a right hemicolectomy was carried out followed by an uneventful recovery.

Case 3. A 45 year old male presented with a one week history of abdominal pain; at first central and crampy and later continuous in the right iliac fossa. There were no other symptoms.

On examination he was in good health, apyrexial and the only positive abdominal finding was tenderness on deep palpation in the right lower abdomen. Rectal examination demonstrated right sided tenderness. He was thought to have

acute appendicitis and the abdomen was explored through a grid iron incision. A suspected tumour 1.5 cm. in diameter was palpable in the caecum. The grid iron incision was closed and a right hemicolectomy performed through a right paramedian incision. The patient made an uncomplicated recovery.

The pathology reports in each of these cases identified the lesion histopathologically as an inflammed solitary caecal diverticulum.

DISCUSSION:

Solitary caecal diverticula are rare; approximately 400 have been described in the world literature[1].

Although diverticular disease of the left colon may sometimes spread to the ascending colon and caecum, the solitary caecal diverticulum is considered to be an entirely separate entity[2], tending to present in a younger age group, usually around the fourth decade. Anatomically all the muscle layers found in the bowel wall are present in at least part of the diverticulum. Although the aetiology is unknown, some authors implicate an embryological remnant which appears at the sixth week and fails to atrophy; others, that they occur at a congenital weak spot in the wall of the caecum[3], [4].

Solitary caecal diverticulae rarely give rise to symptoms unless they become inflamed. Inflammation is usually precipitated by a faecolith becoming lodged in the cavity of the diverticulum. It may progress to perforation, or to an inflammatory mass or abscess in the wall of the caecum simulating a carcinoma[5]. Acute inflammation of the diverticulum usually presents with symptoms and signs indistinguishable from acute appendicitis as demonstrated in these cases.

At operation a high index of suspicion is important in making a correct diagnosis. If the appendix is normal and a caecal mass is encountered this may be benign inflammation, but is more likely to be a carcinoma. It is vital to differentiate between these conditions to obviate the need for extensive bowel resection. The mortality from emergency right hemicolectomy for carcinoma of the caecum is approximately five times greater than that for any local form of operation[6].

On encountering such a mass at laparotomy the caecum should be palpated through an unaffected part of the opposing bowel wall. Carcinoma always involves the mucosa giving rise to the characteristic malignant ulcer or to cauliflower or polypoid growths. In caecal diverticulitis the mucosa is not affected, and the ostium of the diverticulum may be identified or it may be obvious that the process is restricted to the bowel wall and surrounding fibro-fatty tissue. If doubt persists caecotomy may be justified to inspect the mucosa as this procedure with limited resection carries a much lower mortality than right hemicolectomy. The treatment of solitary caecal diverticulum depends on the findings at laparotomy in each individual case. It should, however, consist of the simplest operative procedure compatible with eradication of the condition[7].

When the condition is recognised as acute diverticulitis and the inflammation

is limited to the diverticulum, diverticulectomy or merely drainage with antibiotic cover is sufficient. If the diverticulum is perforated with local peritonitis or abscess formation, closure of the perforation, drainage and appropriate antibiotic therapy is an acceptable form of management.

If the perforation is too large to be closed or cannot be differentiated from carcinoma a Mickulicz procedure with excision of the mass and secondary ileocolostomy may be performed but a right hemicolectomy may be carried out with the inherent risks of this emergency procedure.

SUMMARY:

Solitary caecal diverticulum is a rare condition, usually mistaken for acute appendicitis pre-operatively and for carcinoma of the caecum at laparotomy. Although most masses in the caecum are neoplastic, the differentiation of inflammatory from neoplastic lesions may be aided by careful palpation and inspection of the bowel wall and mucosa, leading to less aggressive surgery. Treatment should consist of the simplest operation compatible with eradication of the condition.

We would like to thank Mr W S Hanna and Mr J G Kinley for permission to study their cases and to Mrs BMcAllister for preparing the manuscript.

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THREE CASES OF BROMIDE POISONING

KENNETH W MOLES, MRCP, Wds 10/18, Belfast City Hospital

MARY HENRY MSc, Pharmacist, Belfast City Hospital

Kenneth W Moles, Wards 8/14, Belfast City Hospital, Lisburn Road, Belfast

BROMIDE toxicity, though once fairly common, is now rarely encountered or considered. Though overall numbers are small, reports continue to appear from time to time[1-3]. A review from the John Hopkins Hospital described only 22 cases between 1952 and 1975[4]. Perkins in Boston reported 27 cases in 17 years[5]. Over the past ten years there have been only three cases diagnosed in the Belfast City Hospital. If considered, the characteristics biochemical picture makes diagnosis and its confirmation relatively easy. Treatment is then simple. We wish to report three patients with bromism, including two sisters in whom the common presenting features and the rapid response to treatment are well illustrated.

CASE REPORTS

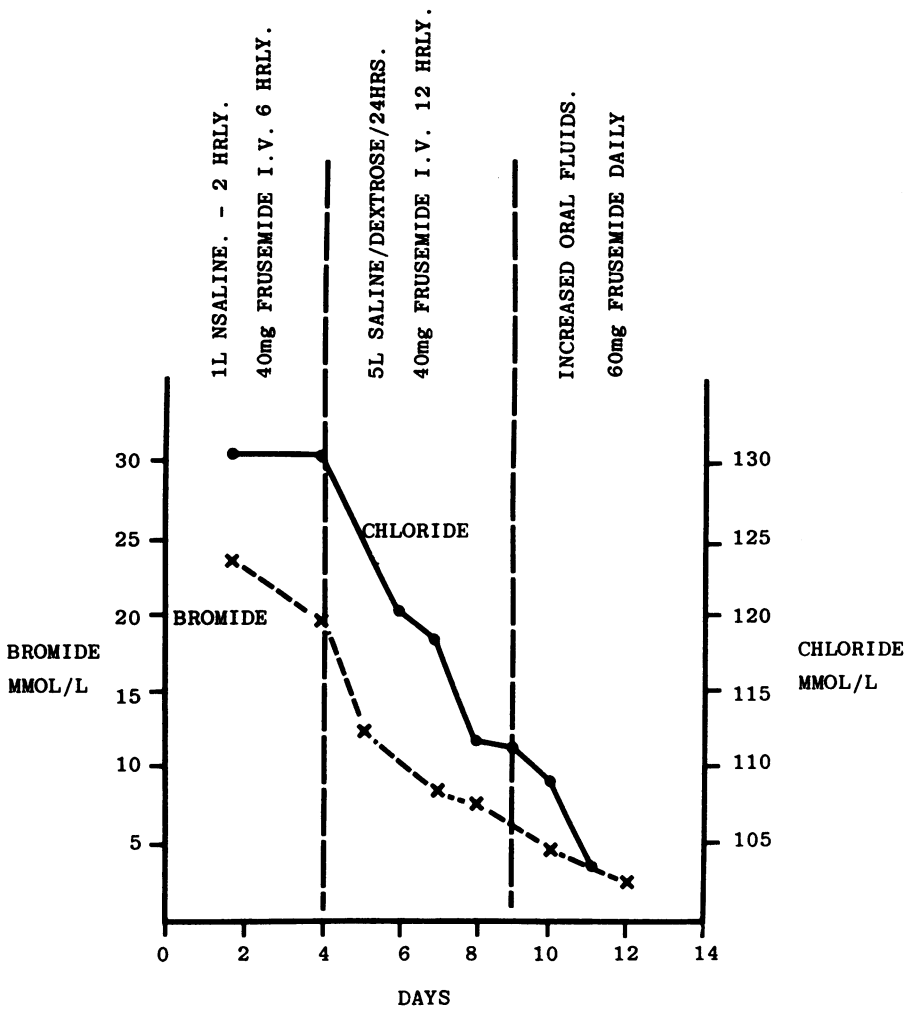
A 44 year old woman with a five week history of general weakness, aches and pains, lethargy, loss of appetite and unsteadiness was admitted after being bed-ridden at home for three weeks. On admission it was difficult to obtain any history for although conscious and fairly alert, she was apathetic and her co-operation was poor.

On examination, there was no obvious head injury or neck stiffness though there was slight photophobia. Memory was poor and speech was slurred. Examination of the nervous system revealed no abnormality apart from poor co-ordination without any definite localisation and generally impaired sensation to pain and touch. Her temperature was 38.4°C. There were no abnormalities noted in the cardiorespiratory system or in the abdomen and there were no skin lesions.

Over the next few hours her condition deteriorated with increasing drowsiness, restlessness and disorientation. Examination of the cerebrospinal fluid, electro-cardiogram, chest x-ray and full blood picture revealed no cause. Electrolytes were normal except for a very high chloride level and this suggested bromide poisoning. This was confirmed by a bromide level of 23 mmol/L.

Treatment with frusemide and normal saline supplemented with potassium produced a rapid fall in chloride and bromide levels (Figure) with a remarkable improvement in mental state, speech, co-ordination and general well-being.

Further enquiry revealed that the patient and her sister had been consuming approximately four litres of Mist. brom et chloral weekly for some years. The sister, who was unwilling to be admitted, had a chloride level of 130 mmol/L and bromide level of 30.9 mmol/L. She too exhibited vague abnormalities of behaviour with poor memory and lack of concentration. The third case, an 84 year old lady



was admitted with a history of confusion and drowsiness progressing to coma. Once again there were no specific findings on examination and the bromide level in this case was 10.5 mmol/L with a chloride level of 93 mmol/L. Her condition improved as the bromide level fell with simple intravenous rehydration.

COMMENT

In bromism, the commonest symptoms are those involving the central nervous system. Although there is no characteristic pattern, features include general lethargy and weakness associated with mental detorioration, drowsiness and confusion; dysarthria, ataxia and tremor also occur. Sometimes there are non-

specific changes in tendon and pupillary reflexes. Various skin lesions including acneform, erythematous or nodular rashes may occur but were not seen in our three cases.

Bromide levels regarded as toxic vary between 6 mmol/L and 12 mmol/L depending on the laboratory, but the level at which toxicity occurs is very much reduced if the chloride level is low as illustrated by our third case.

Treatment of bromide poisoning is simple consisting of fusemide and intravenous saline supplemented with potassium. The speed and amount of the infusion depends on the cardiovascular and renal status. These three cases illustrate clearly that bromism must still be considered in any patient exhibiting vague, non specific neurological findings.

SUMMARY

Though bromide poisoning with its vague and nonspecific neurological findings is relatively uncommon, diagnosis can be simple if the unusually high chloride levels found are remembered. Treatment with diuretics and intravenous saline rapidly brings a good response.

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A CASE OF EMPYEMA OF THE GALL BLADDER

JAMES ELLIOTT, FRCS
Waveney Hospital, Ballymena, Co Antrim

Fistulae originating in the biliary tree are now rare[1,2,3,4,5]. This has only occured since cholecystectomy, with or without exploration of the common bile duct, came to replace non-intervention or simple cholecystostomy. Even rarer are spontaneous external biliary fistulae [1,2]. These arise in the biliary tract and, by devious routes, emerge at various sites on the body surface. Before they rupture through the epidermis they are referred to as empyema necessitatis of the gall bladder. About half present in the right hypochondrium as would be expected, with some appearing through previous cholecystostomy wounds. The remainder recorded in the English language literature have appeared at the left costal margin, the umbilicus (via the falciform ligament), the right flank, the right iliac fossa, the right groin and the right thigh[1,2,3,4,5].

The present case adds another site to this list.

CASE REPORT

A 33 year-old married woman was referred with a 12-month history of a small, painful, soft abdominal swelling. She had no history of dyspepsia. She had gross enlargement of both kidneys due to congenital polycystic disease, and had had a vaginal hysterectomy and left ovarian cystectomy two years previously. The swelling had already been opened once, as it was thought to be a haematoma related to her previous surgery. A cavity containing mucous fluid and grey calculi had been found. Unfortunately the calculi were mislaid, and so were not sent for analysis. The wound was closed and healed satisfactorily, however the swelling recurred about one month later, and it was at this stage that she presented to the Waveney Hospital.

On examination, there was a 4cm. diameter, tender swelling, situated in the mid-line, equidistant from the umbilicus and the symphysis pubis. Due to the site of the swelling, what was known of its previous contents, and the fact that it had recurred, it was thought to be a urachal cyst. Consequently, the patient was admitted for formal excision of the "cyst".

On admission the haemoglobin was 10.9g/dl, the white cell count $6.0X10^9$/L and the ESR was 50mm/hour.

At operation the "cyst" was found to be the termination of a fistula which had passed along the deep surface of the anterior abdominal wall from the right hypochondrium. The fistula was covered by the parietal peritoneum and was found to originate in the gall bladder, which contained a single large calculus in Hartmann's pouch. The fistula was excised and cholecystectomy performed. The common bile duct was not judged to contain any calculi, and so was not explored.

The patient made an uneventful recovery, and was fit for discharge 12 days post-operatively. At review 10 months later she was well.

DISCUSSION

This condition has been referred to variously as spontaneous external biliary fistula, cutaneous biliary fistula and empyema necessitatis of the gall bladder [1,2,3,4]. Empyema necessitatis is probably the most appropriate term here since the fistula had not actually penetrated the epidermis at the time of presentation

The first case to be described was in 1670 by Thilesus [1,2,3,5]. In the last century individual series contained 100-200 cases each. Due to the routine surgical treatment of gall bladder and biliary calculous disease, the incidence had fallen dramatically. In fact, the case here described is only the thirty-second to appear in the English language literature since 1900 [1,2,5]. A little over twice this number have appeared in the total world literature in this time.

As with gall bladder disease in general, this condition tends to occur in females, the female: male ratio being approximately 3: 1[4]. It is rare before the age of fifty[1,5]. In this case the age at diagnosis was 33.

Various authors [1,2,3,4,5] have suggested that the commonest mechanism of formation is as follows. The inflammed and distended gall bladder becomes adherent, usually via its fundus, to the parietal peritoneum of the anterior abdominal wall. Increasing distension gives rise to impairment of the blood supply to the gall bladder wall, which then perforates. Due to the occurrence of previous episodes of inflammation it is already surrounded by dense adhesions, and these prevent a free intra-peritoneal perforation. Consequently, rupture occurs through the point of adherence to the parietal peritoneum and into the anterior abdominal wall. The fistula has now been started and, over a period of time, burrows its way along tissue planes and through the layers of the abdominal wall, to emerge at one of the previously mentioned sites. It may remain quiescent for a while, but will eventually rupture, discharging mucus, bile and pus. This settles to a continuous or intermittent discharge of mucus and bile in various proportions, depending on whether any biliary tract obstruction exists, and if so, its site.

Consensus opinion suggests that in the acute phase treatment should consist of antibiotics and supportive measures as for simple acute cholecystitis[1]. Often the patient is elderly, and occasionally is very ill. In this case, treatment should remain conservative, unless the discharge consists of large amounts of bile. In any patient where this is so, surgical correction becomes urgent, to prevent the loss of large amounts of fluid and electrolytes.

In the majority of cases, once the acute phase has settled, a fistulogram should be obtained, followed by excision of the fistula and cholecystectomy[1,4,5]. An intra-operative cholangiogram should be performed if any doubt exists as to the patency of the common hepatic and bile ducts.

These comments with regard to treatment are only appropriate if the diagnosis is appreciated pre-operatively. To increase the likelihood of this all fluid and calculi obtained from swellings of uncertain origin should be sent for analysis. Even so, it is likely that a number of cases of empyema necessitatis of the gall bladder will present at unusual sites before rupturing, and so without presenting pre-operative evidence as to their identity.

SUMMARY

A case of the rare condition of empyema necessitatis of the gall bladder is presented and discussed.

The site of the empyema in this case was unusual, in that it did not appear in the right hypochondrium or at any of the other less common sites previously recorded in the English language literature, but at a point in the mid-line, equidistant from the umbilicus and the symphysis pubis. The condition rarely occurs before the age of 50, but this patient was 33 years old.

I would like to thank Mr W S Hanna, FRCS, formerly senior Consultant Surgeon, Waveney Hospital, Ballymena, for his consent to present this case.

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DON'T FORGET SYPHILIS

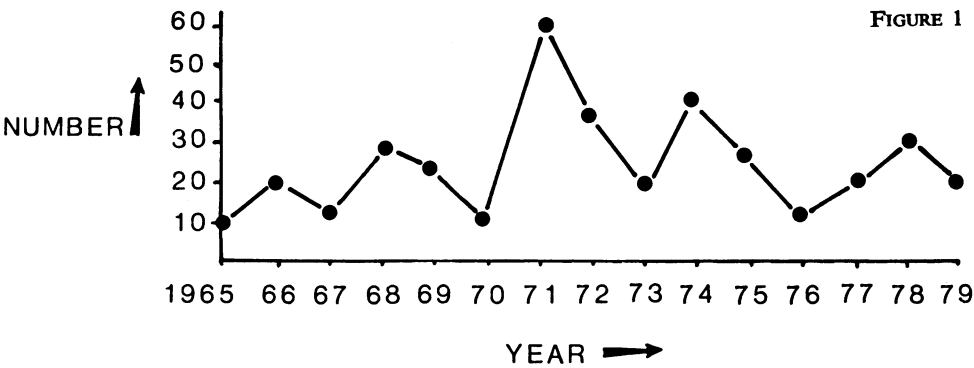
R D MAW and T HORNER

From the Department of Genito-Urinary Medicine
Royal Victoria Hospital, Belfast

INTRODUCTION

THE incidence of primary and secondary syphilis has shown a steady increase in Great Britain in the last decade, from a figure of 1,162 in 1970 to 1,543 in 1979 for England and Wales. The incidence in Northern Ireland has been somewhat more sporadic (Figure) but we have found, in common with our colleagues in the United Kingdom,[1] a tendency to forget these conditions in arriving at a differential diagnosis. The following cases were collected at the Genito-Urinary Medicine Department of the Royal Victoria Hospital, Belfast between 1978-80 and illustrate some situations where misdiagnosis can easily be made.

FIGURE 1



Number of cases of primary and secondary syphilis cases seen in period of 1965-1979 *in N. Ireland.*

CASE HISTORIES

Case 1

Mr. B presented voluntarily at the Genito-Urinary Medicine Department, Royal Victoria Hospital, Belfast. He gave a history of a lump on his foreskin for eight to nine days. There was no history of pain or swelling in the groin. Examination revealed a 1 cm diameter indurated, non-tender ulcer at the corona. Darkground examination was positive for Treponema pallidum and subsequent serology showed a positive Venereal Disease Research Laboratory (VDRL) Wassermann (WR), *Treponema pallidum* haemagglutination (TpHA) and fluorescent treponemal antibody (FTA).

Sexual history revealed ten different female contacts in the previous three months. There was no homosexual contact. Of his 10 contacts three were definitely identified and attended our clinic in response to his request. One patient

defaulted after an initial negative assessment. One developed a primary chancre on the vulva on follow up and the third was found to have an asymptomatic primary chancre on the cervix on speculum examination. This left seven unidentified contacts.

Cases 2, 3 and 4 all socialized at the same club as Mr. B and were thought to be at least secondary sexual contacts of his.

Case 2

Mr. K presented to a casualty department in Belfast complaining of soreness in the right groin. Examination at that time revealed swollen tender glands in the right inguinal region. No site of infection was found in the right leg or foot. There was no record of a genital or perianal examination having been made. Magnapen, one capsule three times daily was prescribed for one week and review arranged for one month later.

At follow up the patient said his inguinal pain and swelling had resolved. On examination painless right and left inguinal adenopathy persisted and, as well, adenopathy was found in the right axilla. No liver or spleen was palpable. A provisional diagnosis was made of reticulosis[2] and the patient admitted for lymph node biopsy. On the eve of biopsy the patient was noted to have developed a generalised macular, symmetrical, non irritable rash. Syphilitic serology was taken and reported positive and the patient referred to our department.

On examination at this time when the prepuce was retracted a still active primary sore of the penis was found and he had a typical macular rash of secondary syphilis. Mr K had not noticed this lesion or complained of it at any stage. Although at first an inaccurate sexual history was obtained, eventually three contacts were established. No history of homosexual contact was given.

Case 3

Miss L presented to her general practitioner complaining of "blisters" on the vagina. An external genital examination was performed and clotrimazole cream and pessaries prescribed. The blisters persisted despite this therapy and further local applications were prescribed two weeks later. At this stage she informed the doctor that her boyfriend had a sore on his penis and was advised he should use Savlon baths. No examination was recommended.

The patient returned to her doctor one week later complaining of a sore throat, headache and generalised rash. A diagnosis of measles was made. A further examination was requested one week later from a different doctor who carried out luetic serology which was strongly positive and she was referred to us. Examination at that time revealed a generalised macular rash on the body and bilateral inguinal adenopathy. Genital examination revealed thickening of the right labium majus where the ulceration had been. Cervical erosion was noted and proctoscopy revealed papular lesions on the rectal mucosa. Darkground examination from cervical and rectal lesions both demonstrated *Treponema pallidum*.

Sexual history revealed Miss L had been out with Mr K but she denied sexual intercourse with him, although he later confirmed such contact. Her regular partner was found to have two active primary lesions on examination and a further sexual contact of his subsequently was found to have an asymptomatic cervical primary lesion.[3]

Case 4

Mrs N a 26 year old woman attended our clinic giving a history of having had pains in the head and joints two months ago which resolved spontaneously after two weeks. This was followed two weeks later by a generalised copper rash involving the whole body. The rash was said to be clearing spontaneously. The reason for attending us was that she had read in a womans' magazine that syphilis could cause such rashes. Examination revealed a fading generalised rash and lymphadenopathy of cervical and inguinal areas. No spirochætes were noted but her luetic serology was reported as TpHA positive 1280, WR positive 512, VDRL positive and FTA positive.

On questioning Mrs. N said her husband had developed a penile sore six months previously followed by the onset of a generalised rash which looked just like her own. At that time he was referred by his general practitioner to a dermatological clinic where a diagnosis of exacerbation of psoriasis was made.[4] On perusing his notes, no mention was made by the patient of the genital sore nor was any such question recorded. Mr N was subsequently confirmed to have syphilis and his contact was traced and she was also positive, but none of her multiple contacts subsequently appeared for investigation.

Case 5

An approximately seven month gestation infant was born at home to Miss D. There had been no antenatal supervision of the pregnancy. Shortly after birth the child was noted to be in respiratory distress and transferred to a special care paediatric unit. The child was noted to have a peculiar rash, especially marked on its hands and feet and had a five finger breadth liver and spleen. Chest x-ray showed a patchy infiltration of the lungs. The first examining doctor who was from Africa, diagnosed congenital syphilis. Unfortunately the serological specimen was mislaid and the child subsequently treated for pneumonia on which treatment the generalised bodily rash resolved and the liver and spleen size resolved. It was only after belated serological tests confirmed the diagnosis that we saw this case. The mother was tested and confirmed as suffering from syphilis. History taken from her confirmed sexual contact with the above group, although she denied having had any symptoms or signs that might have suggested primary or secondary syphilis.

Case 6

Mr R, a 31 year old homosexual, who was not associated with the above contacts, was being treated as an inpatient for depressive illness when he began to complain of rectal pain and bleeding, worse on defaecation. Rectal examination at that time revealed an anal lesion to which local antiseptic applications were

made. Subsequently he developed inguinal adenopathy and had a swinging temperature. At this stage he was referred to a surgical department for investigation with a presumptive diagnosis of rectal abscess or infected haemorrhoids.[5] An anal dilatation was carried out and antiseptic applied to the perianal lesions seen. The next day a generalised macular rash was noted and the patient referred to our department where spirochætes were isolated from multiple scrotal ulcers but none from the perianal lesions, presumably due to the antiseptic applications. The patient had a typical generalised secondary luetic rash.

DISCUSSION

These cases illustrate many of the difficulties encountered by the practising doctor and venereologist in the handling of cases of sexually transmitted disease and of syphilis in particular. Syphilis was traditionally described as the great mimic. In the Western World this has passed to the collagen diseases because of the comparative rarity of infectious syphilis in the last three decades. Consequently the diagnosis which was once included in many differentials is now understandably enough forgotten. From the figures given the incidence of infectious syphilis is seen again to be rising in our community and hence should once again be considered in many situations.

Case 1 with his seven missing contacts emphasises the need for a contact tracer for an efficient Sexually Transmitted Disease Service. The situation in Northern Ireland has been that because of the civil disturbance, contact tracing was withdrawn and only now have we been in a situation to re-advertise this position.

Case 2 emphasises the need for adequate physical examination which must include a genital history and examination if indicated. Too many doctors baulk at this due to their own embarrassment. In Case 3 at least one girl could probably have been prevented from contracting syphilis if the undiagnosed genital ulceration had been referred to the local Genito-Urinary Medicine Clinic.

Case 4 underlines the fact that in a generalised body rash syphilis still needs to be excluded. Case 5 would undoubtedly have been avoided if the mother had attended for antenatal care but once again could have been avoided earlier with effective contact tracing. It was interesting that the African doctor made the clinical diagnosis. Congenital syphilis is a very common disorder in the African continent.

Case 6 in particular illustrates a common situation today in Great Britain where the ratio of male to female primary and secondary syphilis is 8 to 1 and the majority of these males are homosexuals.[6] We feel that it is essential that any young male presenting with rectal pain and bleeding and anal lesions should be adequately questioned and investigated as to a homosexually transmitted infection which, of course, may include gonococcal, herpetic or Chlamydial proctitis, as well as syphilis, to mention the commoner infections.

The purpose of this paper is to draw attention of practitioners to this problem which will undoubtedly become commoner and to stress the importance of an adequate sexual history and examination in patients to avoid the obvious dangers of neglected sexually transmitted disease.[7]

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BOOK REVIEWS

EMERGENCY PSYCHIATRY FOR THE HOUSE OFFICER. By William R Dubin and Robert Stolberg. (Pp 166, £8.55). Lancaster: MTP Press, 1981.

THIS is a good useful book which is easy to read and is well tabulated.

The early part focuses on differentiating organic brain disease from psychoses, the importance of reversable brain disease in the elderly and the part the emergency physician has in making this diagnosis. The authors must be commended for the amount of space given to these topics in such a short book. Other points stressed in this part of the book are, not to label patients with behavioural problems as psychic before a determined effort has been made to rule out organic brain disease, or to admit a patient with organic brain disease who presents with behavioural problems to a psychiatric unit before every effort has been made to treat these problems with neuroleptic drugs.

The chapter on psychoses gives clear. simplified and useful advice on diagnosis and stresses the importance and value of early treatment of these conditions in casualty.

On management of grief they advocate that the relative be allowed to view the body. They stress the importance of allowing the patient to grieve and that this process should not be prevented by sedatives or tranquillisers.

Chapter six gives good clear practical guidelines on the management of the violent patient and the same applies to chapter seven and the suicidal patient. It points to the fact that every suicide is tinged with ambivalence and that it is the task of the physician in casualty to initiate positive thinking in these patients.

The high mortality rate associated with delirium tremors is one of the most important points made in the chapter on alcoholism. Chapter ten which is concerned with drug abuse looses much of its impact because of the American terminology used. This also applies to the sixty pages devoted to listing the psychiatric side effects of chemotherapy, although the idea itself is a very useful one.

The dangers in this book are in its simplicity and brevity. It cannot be considered a text book and the user should have a good knowledge of psychiatry and organic brain disease.

It will probably be used, as intended, in the casualty room in the U.S.A. more than in Great Britain. It may also prove a useful revision aid to the medical student and others involved in the care of the mentally ill.

ECO'G

A TEXTBOOK OF CLINICAL PHARMACOLOGY. By HJ Rogers, RG Spector and JR Tronce. (Pp vii + 853, Illustrated. £12.95). London: Hodder & Stoughton, 1981.

CLINICAL pharmacology has evolved during the past 30 years as a distinct discipline. This has occurred for a number of reasons including the great increase in the number of drugs, the more widespread use of drugs. the availability of methods for more detailed evaluation of their effects in man and concern about the unwanted effects of drugs.

The development of clinical pharmacology has brought with it an increasing number of textbooks. the most outstanding of which has been that by DR Laurence. which has been widely used by many students. This new book may rival Laurence's in popularity amongst students for whom it is primarily intended. While it may lack the individuality and the anecdotes of Laurence, it is an excellent book, with a slightly more didactic approach. In

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this new book the authors have been successful in obtaining an acceptable mixture of pharmacology, clinical pharmacology and therapeutics so that the book can be used as a single textbook for students during courses in all three subjects.

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The case for more clinical trials is well argued and only in this way will progress be made. Unfortunately one is still left with a feeling of uncertainty as to which trials one should enter. Nonetheless the book is a useful document outlining the main points in favour of the clinical application of cell kinetics and is a useful background reference for a number of clinical trials currently underway.

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In the absence of real patients the examiners attach particular importance to the viva voce. The Log Diary and Problem Solving Orals are discussed authentically and consideration of the sample questions and answers provides many helpful hints, as to how the candidate should behave face to face with the examiners. I envisage the book having a wide circulation amongst doctors who intend to sit the M.R.C.G.P. It would be of the utmost value to young doctors. who are undergoing vocational training for general practice, in order to enhance their performance in the real test on completion of training.

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The reference lists are invaluable and Dr. Noble's ability to express a balanced opinion in areas of knowledge which are still the subject of dispute leaves the reviewer full of admiration. At today's prices this book is a "best buy" and is a must for microbiologists and especially those who need to update their lectures.

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This book is the report of a conference held in the Royal College of Physicians of Edinburgh in which the care of the elderly was discussed. The conference started with a survey of the problem. The large amount of bed days occupied by a very small number of elderly people who stay in hospital a long time is highlighted: less than 4 per cent of male discharges are over 65 and stay 30 days or more but they account for 20 per cent of the beds used; for females the figures are 6 per cent of discharges but 35 per cent of beds used. Other authors discuss the spectrum of care for the elderly, misplacement in hospital and the problem of the demented patient. It is interesting to see data indicating that misplacement of patients is a problem which is even more important in geriatric medical units than in general medical units. In Glasgow with 1,100 medical beds and 2,100 geriatric beds, approximately 600 are lost to their proper use because they are occupied by patients who would be elsewhere if facilities for their accommodation were available.

The second half of the report looks at some possible clinical solutions to the problem. It is notable that two solutions which are sometimes suggested do not appear to have beneficial results. The provision of support beds for general medical wards merely allowed a very high proportion of these to be occupied by misplaced patients. In Dundee a policy of admitting all emergency medical problems to a special admitting ward did not limit the number of elderly admissions to the general medical ward. Rather it has the reverse effect by encouraging a predominance of elderly patients in general medical wards and a reduction in acute medical conditions nursed on these wards. More effective solutions have come from schemes in Hull and Edinburgh, details of which have already been published. In Hull an age related policy for geriatric admissions, where all medical patients over 75 years old are admitted to the geriatric unit, has resulted in a high turnover, no waiting list and apparently no misplaced patients in general medical wards. An alternative procedure employed in Edinburgh is for the geriatric team to be attached to the general medical ward, without clinical responsibility for patients, but acting in an advisory capacity particularly with respect

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to the discharge of elderly patients. This has increased the turnover and shortened the length of stay without an increase in transfer of patients to the geriatric unit.

This book is a useful summary of an important problem and will be of interest to physicians in both general and geriatric medicine.

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THE first edition of this book was published in 1959 and reprinted on ten occasions from then until 1979, thus illustrating its popularity and usefulness. This second edition should be no less in popularity as, with one or two exceptions, it is merely a duplication of the original. In fifteen chapters, the author covers many of the simpler statistical concepts and their uses in biological and medical research, e.g. variability. estimation, significance tests, correlation and regression, and experimental design. Non-parametric and distribution-free tests are increasingly being used in medicine today and the author has recognised this fact by including a new chapter on these. However, as only a few of the simpler non-parametric techniques are discussed the reader should have been directed to a more comprehensive text book on this subject, e.g. Sigel (1956).

In general, the non-mathematically qualified reader should have little difficulty in reading this book. Mathematical symbolism is minimal, no trigonometry, goemetry, or calculus is used. All that is required is an elementary knowledge of algebra. The practical examples are well set out and simple to follow. For these reasons I would recommend this book as a useful introduction to the uses of statistical methods in medical research.

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The volume is to be welcomed and is very easily read; a few criticisms include the inaccuracy of Koch's postulates (which still have relevance today) and is it necessary (p.16) to tell the clinician that *Neisseria gonorrhoeae* is the causative agent of gonorrhoea ?

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A MINORITY of healthy individuals given the antimalarial drug primaquine will develop acute haemolytic anaemia. The demonstration, some 20 years ago, that this was a consequence of their having low levels of glucose-6-dehydrogenase in their red cells. was the starting point for very intensive studies of red cell metabolism in health and disease and this publication summarises the present state of knowledge in this field.

The first chapters are concerned with the investigation of a patient suspected of suffering from such a disorder and deal in considerable detail with the abnormalities in enzyme systems associated with red cell carbohydrate metabolism.

In the section on methaemoglobin, the contribution made by QH Gibson whilst working in the Queen's University Department of Biochemistry with Professor Harrison is acknowledged. Gibson was the first to define the normal pathway of methaemoglobin reduction, the pathway utilised when methylene blue is added and correctly identified the site of the enzymatic defect in hereditary methaemoglobinaemia.

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JMB

OPHTHALMIC ELECTRODIAGNOSIS. By NR Galloway, MD, FRCS. Second Edition. (Pp X + 180, Illustrated. £14.00). London: Lloyd-Luke Medical Books, 1981.

THIS book is intended to be a basic guide for those working in the field of ocular electrodiagnosis. It is divided into two sections, the first dealing with the theory and method of electrodiagnostic techniques as applied to the eye, and the second encompassing the clinical applications of ocular electrophysiology.

The initial chapters provide sound guidelines for obtaining electroretinographic, electrooculographic and visually evoked responses under different stimulus conditions and states of ocular adaptation. The origin, nature and physiological variations of the various responses are analysed in some detail and problems of artefacts and quantification carefully discussed. Less commonly used ocular electrodiagnostic tests such as electronystagmography and electromyography are also discussed but in much less detail. Practical advice is provided for those about to establish an ophthalmic electrodiagnostic laboratory, including an evaluation and costing of currently available equipment. The application of electrodiagnostic techniques to the diagnosis and elucidation of inherited and acquired degenerative disease of the retina is discussed in detail in the second section. The effects of medial opacities on the various electrophysiological responses are described and a small section devoted to electrodiagnostic tests in toxic and deficiency states and ocular injuries.

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DBA

TOPICS IN RENAL DISEASE. Lancaster: MTP Press.

THERE are five books in this series each dealing with a single topic or related group of topics. They seem a little expensive for their size at £5.95 each but they have hard covers and are pleasantly produced on good quality paper. Taken together they cover renal disease in sufficient detail for anyone except the specialist nephrologist. They certainly would be adequate for preparation for the membership examination and would be very helpful for a registrar coming to work in a renal unit. They are, however, somewhat uneven in quality.

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FOR many years there have been several excellent reference books on Thoracic Medicine, such as Crofton and Douglas and Frazer and Paré. Until very recently there have been very few good concise textbooks available.

The early chapters, not surprisingly, deal with pulmonary physiology. This very complex subject has been treated in a simple and readable manner. So many authors manage to make this relatively straightforward topic totally incomprehensible even to those who have a fair knowledge of the subject.

In the chapter on pneumonia, it was pleasing to see the importance of a Gram's stain being emphasised in the diagnosis of the offending organism. As might be expected the chapters on chronic bronchitis and emphysema are very good. The management of asthma reflects the Edinburgh style and is all the better for it. The author states that "treatment is best considered as that of the acute attack of asthma, followed by preventive medicine which aims to inhibit the development of further attacks of asthma" and, later on, in a reference to the number of asthmatic deaths in the United Kingdom, he states "the suspicion remains that efficient delivery of our present methods of treatment should reduce this figure, this idea lying behind the emergency of asthma self-admission schemes available for known severe asthmatics". Much more should be made of this. The greatest advance in asthma will occur, not as a result of new discoveries under the electron microscope, but when doctors are able to understand and use correctly the drugs presently available.

The chapter on Adult Respiratory Distress Syndrome and, later on, on Non-cardiogenic Pulmonary Oedema, should be mandatory reading for all doctors. should they be surgeons, obstetricians or general practitioners.

I was surprised and disappointed that byssinosis did not even receive a mention in the chapter on Occupational Lung Diseases.

When I need to check some aspect of pulmonary medicine I always start with a simple textbook before proceeding to reference works and journals. I recommend this policy to all my students and shall send them and colleagues to this excellent book written in the very readable style of a first class lecturer.

RCL

CONTROVERSIES IN CLINICAL CARE. Edited by Victor M Rosenoer, MD, and Marcus Rothschild, MD. (Pp 234. £17.75). Lancaster: MTP Press, 1981.

CONTROVERSY in medicine is not new. Unfortunately modern sophisticated methods of studying the effectiveness of new therapies have not reduced the controversy. In the opening chapters of this book it is shown how faulty methods of analysis and incomplete reporting of the results of clinical trials can contribute to controversy.

The remaining chapters deal with 10 aspects of clinical care where controversy is rife. These range from the treatment of alcoholism to the management of borderline hypertension, and include two papers on the quality of control in diabetes. These are relatively concise and useful summaries of recent publications on each subject.

Sometimes the authors conclude that recent work has largely settled the controversial question. For example the chapter on mild hypertension ends with the statement "an agressive approach to therapy, including pharmacotherapy, is now justified in many individuals with mild systemic hypertension". In other chapters it is frankly admitted that the question

RESPIRATORY MEDICINE CMT (Concise Medical Textbook). By DC Flenley. (Pp 263, Figs 88, Table 1. Paperback £6.50). London: Bailliere Tindall, 1981.

FOR many years there have been several excellent reference books on Thoracic Medicine, such as Crofton and Douglas and Frazer and Paré. Until very recently there have been very few good concise textbooks available.

The early chapters, not surprisingly, deal with pulmonary physiology. This very complex subject has been treated in a simple and readable manner. So many authors manage to make this relatively straightforward topic totally incomprehensible even to those who have a fair knowledge of the subject.

In the chapter on pneumonia, it was pleasing to see the importance of a Gram's stain being emphasised in the diagnosis of the offending organism. As might be expected the chapters on chronic bronchitis and emphysema are very good. The management of asthma reflects the Edinburgh style and is all the better for it. The author states that "treatment is best considered as that of the acute attack of asthma, followed by preventive medicine which aims to inhibit the development of further attacks of asthma" and, later on, in a reference to the number of asthmatic deaths in the United Kingdom, he states "the suspicion remains that efficient delivery of our present methods of treatment should reduce this figure, this idea lying behind the emergency of asthma self-admission schemes available for known severe asthmatics". Much more should be made of this. The greatest advance in asthma will occur, not as a result of new discoveries under the electron microscope, but when doctors are able to understand and use correctly the drugs presently available.

The chapter on Adult Respiratory Distress Syndrome and, later on, on Non-cardiogenic Pulmonary Oedema, should be mandatory reading for all doctors. should they be surgeons, obstetricians or general practitioners.

I was surprised and disappointed that byssinosis did not even receive a mention in the chapter on Occupational Lung Diseases.

When I need to check some aspect of pulmonary medicine I always start with a simple textbook before proceeding to reference works and journals. I recommend this policy to all my students and shall send them and colleagues to this excellent book written in the very readable style of a first class lecturer.

RCL

CONTROVERSIES IN CLINICAL CARE. Edited by Victor M Rosenoer, MD, and Marcus Rothschild, MD. (Pp 234. £17.75). Lancaster: MTP Press, 1981.

CONTROVERSY in medicine is not new. Unfortunately modern sophisticated methods of studying the effectiveness of new therapies have not reduced the controversy. In the opening chapters of this book it is shown how faulty methods of analysis and incomplete reporting of the results of clinical trials can contribute to controversy.

The remaining chapters deal with 10 aspects of clinical care where controversy is rife. These range from the treatment of alcoholism to the management of borderline hypertension, and include two papers on the quality of control in diabetes. These are relatively concise and useful summaries of recent publications on each subject.

Sometimes the authors conclude that recent work has largely settled the controversial question. For example the chapter on mild hypertension ends with the statement "an agressive approach to therapy, including pharmacotherapy, is now justified in many individuals with mild systemic hypertension". In other chapters it is frankly admitted that the question

remains as controversial as ever; for example in the matter of adjuvant therapy for breast cancer. The range of subjects covered is so wide that no one practicing physician is likely to read all the chapters.

Provided the reader does not expect to find absolute answers to all the subjects reviewed, he will find this a convenient way of "catching up" on recent publications on several controversial aspects of patient care.

MES

GYNAECOLOGICAL THERAPEUTICS. Edited by DF Hawkins. (Pp 287, Figs 32, Tables 16. £14.50). London: Bailliere Tindall, 1981.

THE editor in his preface rightly states that gynaecologists are at a crossroads, for developments in the basic medical sciences have led to a greater understanding of the disorders which bring women to our out-patient clinics and suggest therapeutic regimens which are less surgically orientated.

Advances are rapidly being made in the fields of gynaecological endocrinology and its related subspeciality infertility, the non-surgical treatment of infection and malignant disease. The demands of modern society are causing an increasing awareness of the effects of emotional problems presenting or reflected in the pelvis. The topics selected by Professor Hawkins for presentation are those where some of the most significant and worthwhile advances have been made in gynaecological therapeutic practice.

The first contribution by Sir John Dewhurst is a succinct, well presented and readable description of genetic and congenital sexual disorders in the female. The author's experience in dealing with these difficult problems is universally recognised and his approach to their investigation and management, including counselling of the patients. is logically and humanely discussed. A succinct chapter by Masson and Klopper follows on the menstrual cycle and its disorders together with a consideration of the multitude of endocrine preparations that are now available for their treatment. Their introduction is a concise summary of the basic physiology followed by a rational and comprehensive discussion of the treatments of such conditions as dysmenorrhoea, premenstrual tension, endometriosis, steroid contraception and the menopause. This is followed by a chapter of exceptional clarity by Gordon on non-ovulatory infertility which is a detailed but rational discussion of the investigation and management of a problem which has many causes, but whose successful treatment can bring much satisfaction both to the patient and her doctor.

Psychosomatic aspects of gynaecology are increasingly coming to the fore in many aspects of our practice and experts in sexual and marital problems are appearing who have no basic training in gynaecology. Goldie, in his chapter on this subject, discusses many of the problems that may present in the clinical situation. In what was for the reviewer the most difficult chapter to read and understand because of the non-clinical language used, the most useful topics discussed were counselling in practice and referral to a psychotherapist. The editor teams up with Gaya to contribute an exhaustive chapter on pelvic infection and every problem from simple vaginitis to the detailed management of the patient with life-threatening bacteriogenic shock is presented in detail. Major advances have been made in the radiotherapy and chemotherapy of gynaecological cancer in recent years and these are suitably detailed and reviewed by Walter and Tattersall.

The editor is to be congratulated on providing a new book which achieves what it set out to do. providing an excellent and up to date well referenced presentation of many of the key areas in gynaecology. A small but significant omission is a chapter on the medical management of the incontinent female. It is hoped that in the next edition a place will be found for its consideration.

The consultant who practices general gynaecology rather than concentrating on a narrow subspeciality. the senior registrar and candidates for higher examinations in gynaecology will all derive benefit from reading this new and welcome book.

GAM

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