

THE

# ST. LUKE'S HOSPITAL

# GAZETTE

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## INTRODUCTORY

It has been said that disease as it stalks through the land cannot keep pace with the desire of people to scribble about it. The original author of this statement we have not been able to trace, but we can recall hearing it repeated, with understandable feeling, by a medical librarian. Doubtless much is written and published which does not noticeably advance scientific learning, but it is, on the whole, a good thing that experience should be recorded and so shared. This is what, it is felt, justifies the appearance of a new periodical. Malta has, on a few occasions, found itself more important than its size would normally have led one to expect and we do believe that, through its British connection, its school of medicine and its hospitals have given it a position ahead of that enjoyed by Mediterranean islands of a comparable size, say Formentera, or Korcula, the Lipari Islands or any one of the hundred in the Greek archipelago.

It is hoped this periodical will report medical activities from all over the island; in its columns we will welcome contributions by anyone who has something original or interesting to say, and in this sense it will speak for all the medical profession in Malta; it has, however, origin-

ated from St. Luke's Hospital and that, we believe, will be its mainspring, so, since this issue may have readers who do not know Malta or its main hospital, we venture to introduce ourselves.

St. Luke's Hospital was so named by Albert Bernard, a Chief Government Medical Officer who felt that this island should commemorate the beloved physician who visited it in 60 A.D. in the company of the apostle Paul. There are many St. Luke's hospitals throughout the world, but Malta had this close connection to justify, indeed to enforce the association, for it was written in the account of that visit: "Among the estates in that part were some which belonged to the leading citizen of the island, a man named Publius; it so happened that Publius' father had taken to his bed, laid up with fever and dysentery. Paul who had gone to visit him, laid his hands upon him with prayer, and healed him; whereupon all the other folk in the island who were suffering from infirmities came to him and found a cure" (Acts of the Apostles). Few medical communities can claim an inspiration older than is provided by this specific account, even though the diagnosis of the case reported almost certainly had no laboratory confirmation.

The first stone of "St. Luke's" was laid with great pomp by His Excellency Sir John du Cane, "the leading citizen" of his day, on the 5th April 1930, but the hospital came into use hurriedly and most unceremoniously in 1940 when patients from a very vulnerable fever hospital were transferred to the as yet unfinished building on the outbreak of war with Italy. "St. Luke's" was only slightly less vulnerable and was in fact hit in the very first air raid on the 11th June 1940. Damage was slight and, though it was hit again on other occasions, it survived to house first the victims of a scabies epidemic — an odd by-product of war — then in 1942 those of a classic outbreak of poliomyelitis, an illness which recurred in 1945-46. In 1944 and 1946 murine typhus made its appearance but the most dramatic illness to be treated at "St. Luke's" was bubonic

plague, of which between June 1945 and June 1946, there were 80 cases. These beginnings tend to make one agree with Parkinson, that famous but most unacknowledged legislator of mankind, who said that "During a period of exciting discovery or progress there is no time to plan the perfect headquarters. The time for that comes later, when all the important work has been done". We do feel in spite of this, that work at "St. Luke's" since then, although less hectic, has not been unimportant.

"St. Luke's" has some 650 beds, a full complement of departments representative of every speciality and extensive out-patient clinics. It is staffed almost completely by graduates of the Royal University of Malta, of which it is the teaching hospital. Almost every member of the staff has pursued studies in Great Britain, the British Colleges of medicine, surgery, obstetrics and pathology being represented by various Fellows and Members.

A number of other hospitals in the island, including the large Hospital for Mental Diseases, St. Vincent de Paul Hospital, and Victoria Hospital in Gozo, complete the medical services for a population of some 320,000 persons.

The island was medically important between 1530 and 1798 when it was the headquarters of the Knights of St. John, an order of chivalry which combined war on the Moslems with nursing of the ill and wounded; it was once again a large base hospital in the war of 1914-1918 when it nursed back to health casualties of war and disease from the armies in Gallipoli and Salonika. The medical profession played, we feel we can say, a worthy part in the Second World War, when the island as a whole was awarded the highest distinction for civilian gallantry which it was in the power of the monarch to confer. We can but hope these traditions will be maintained in the future, and that this gazette will have not only the pleasure of recording this occurrence but also perhaps, in some way, the honour of stimulating it.

## INSULIN ANTAGONISTS

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It is known that insulin is antagonised by other hormones. This is an attempt to describe the functions of all these hormones but one (thyroxin).

### **Insulin: the storage hormone**

Man eats intermittently and stores some food for periods of fasting. The digestive system limits the quantity of food that can be taken in at a time; but even before digestion of a meal is complete, renewed pangs of hunger may stimulate man to ingest more food. At times food is taken in excess, but all the food that is digested is absorbed and none of the food that is absorbed is lost.

Prevention of food loss is due to secretion of insulin in response to glucose absorption. Insulin specifically enhances glucose uptake by muscle and adipose tissue within minutes of its secretion. This specific action on cell membranes is not followed by specific action on glucose phosphorylation. Insulin does not act directly on glycogen turnover, but indirectly it favours glycogen synthesis by accelerating glucose uptake.

Excess glucose is incorporated in adipose tissue. The triglycerides of this tissue are in continuous breakdown and resynthesis (Fig. I). After breakdown, glycerol is released into the bloodstream; so that resynthesis has to make use of glycerol phosphate, and this is formed from glucose. Insulin enhances esterification of glycerides and inhibits release into the bloodstream of fatty acids that have been set free by breakdown of these glycerides. Hence, insulin favours glyceride synthesis as part of its overall function to preserve muscle and adipose tissue stores and to increment them.

## Hydrocortisone: the stress hormone

Hydrocortisone inhibits glucose uptake by cell membranes, thereby reducing phosphorylation and formation of glycerol phosphate. Reduced supply of glycerol phosphate delays resynthesis of fatty acids in adipose tissue, so that fatty acids accumulate and are released into the blood stream. There follows a high concentration of non-esterified fatty acids or "NEFA" in plasma. It would appear that high plasma NEFA concentrations inhibit glucose uptake by cell membranes, so that this action of hydrocortisone is reinforced by its own effect and plasma glucose concentration tends to rise. This rise of plasma glucose concentration is particularly useful during fasting, which is a form of stress.

It would appear that the main function of hydrocortisone is to maintain sufficient supplies of glucose to the brain during periods of stress without emergency, as during fasting. The central nervous system has a high metabolic rate whether one is awake or asleep, and glucose is its principal nutrient. The adult brain takes up about 65% of glucose that is available to the body, and of the glucose that is taken up, only 35% is used directly for oxidation. The rest is converted into aminoacids, lipids and proteins in a continuous process of breakdown and resynthesis of the brain constituents. Hence continuous supply of glucose to the brain is vitally important. Since there is no glucose or glycogen store in the brain, the blood stream supply must be uninterrupted and high, and this is taken care of by hydrocortisone.

The main action of hydrocortisone is on the liver, where it stimulates gluconeogenesis. In other terms hydrocortisone ensures a good depot of hepatic glycogen for secretion into the blood. Liver glycogen is not a store of carbohydrate in the same sense as muscle glycogen. Whereas muscle builds up its glycogen from the general plasma supply of glucose and will not give glucose back to plasma under any circumstances, the liver builds up its glycogen from several sources and supplies it

to the blood. Hence muscle glycogen is a local depot; but hepatic glycogen is a store for general use.

Sources of hepatic glycogen are several. Some of these are economical as glycerol or fatty acids derived from lipolysis, and as pyruvic or lactic acid overflowing into the blood stream from contracting muscle. But at times the source of hepatic glycogen is highly uneconomical, as amino-acids derived from tissue proteins, generally from proteins of peripheral supporting tissues. Gluconeogenesis is therefore potentially wasteful of valuable material, and may be harmful. It seems likely that hydrocortisone raises plasma NEFA concentration and plasma glucose concentration to reduce gluconeogenesis from amino-acids as much as possible. During intestinal absorption of glucose, gluconeogenesis becomes superfluous. It is therefore inhibited by insulin, which is secreted in response to glucose absorption. Insulin rebuilds the stores of carbohydrate and triglyceride that hydrocortisone had reduced; and it seems likely that it promotes amino-acid incorporation into protein to repair faults to body tissues which fasting may have caused.

### **Adrenaline: the emergency hormone**

Adrenaline is generally released under emergency conditions, and one of its functions is to supply additional glucose to the blood stream for use by essential tissues, including brain. This additional supply is obtained from the liver, where adrenaline provokes glycogenolysis by maintaining a larger proportion of phosphorylase than usual in its active form, thus increasing the effective concentration of the enzyme mainly responsible for glycogen breakdown. Adrenaline also enhances release of fatty acids by adipose tissue. In this way it provides economical material for gluconeogenesis, and by reducing material for resynthesis of triglycerides it spares the glycerol phosphate, which is derived from glucose.

Adrenaline causes also rapid muscular contraction for which rapid supply of energy is required. Yet adrenaline may

decrease the uptake of glucose by muscle, thereby impairing glucose phosphorylation. This means that muscle has to rely on its local glycogen store for contraction. Because the supply of energy has to be rapid and abundant, muscle uses anaerobic glycolysis, with accumulation of pyruvic and lactic acid. These products overflow into the blood stream and provide more material for gluconeogenesis, so that the additional supply of glucose which adrenaline brings about into the plasma from the liver, is not obtained from uneconomical sources. Adrenaline therefore provides for rapid muscular contraction without curtailment of glucose available for use in other tissues and without inducing gluconeogenesis at the expense of supporting tissues.

### **Glucagon**

The function of this hormone is difficult to understand. Like adrenaline, glucagon increases glycogenolysis and like adrenaline it exerts this effect by increasing the effective concentration of phosphorylase in the liver. Both hormones mediate their effect on glycogen metabolism by the initial formation of adenine — ribose, 3, 5 — phosphate, but glucagon affects only hepatic glycogen.

### **The growth hormone**

Like hydrocortisone, this hormone acts on cell membranes by reducing muscle uptake of glucose, as has been confirmed in man with acromegaly. There results a high plasma NEFA concentration and some rise of plasma glucose concentration. Growth hormone presumably obtains these effects to provide economical material for neoglucogenesis and spare amino-acids which are essential for growth.

### **Cushing's syndrome**

It is clear that each and everyone of the above hormones has a definite specific function to perform. They all affect carbohydrate and glyceride metabolism, and excess of hormone may provoke a metabolic state with reduced glucose tolerance,

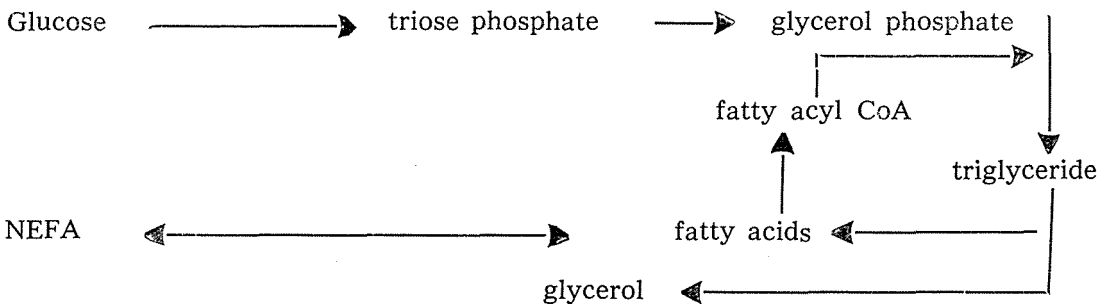
which insulin administration cannot correct well.

The most typical of these states is Cushing's syndrome. This is due to overproduction of hydrocortisone by gross overacting of the adrenal cortex or gross overstimulation by ACTH. Hydrocortisone in physiological doses does not lead to fat deposition in the subcutaneous tissues or elsewhere; indeed it mobilises fatty acids from glyceride depots and its physiological effect is characterised by increased plasma NEFA concentration and by inhibition of glucose storage. But hydrocortisone stimulates also neoglucogenesis and the hyperglycaemia which follows the oversecretion of hepatic glucose into the blood stream provokes a response by the pancreatic islets with overproduction of insulin. Hence Cushing's syndrome may give rise to severe glycosuria; but more

frequently, in the presence of an adequate reserve of insulin, it produces excessive deposition of glycerides in adipose tissue. The hyperglycaemia of Cushing's syndrome is produced by liver secretion at the expense of sources of glycogen which include amino-acids derived from body tissue proteins. Hence there is mobilisation of peripheral supporting tissue with muscle weakness and fatigueability, osteoporosis, cutaneous striae and weakening of vascular tissue with easy bruising at sites of mild trauma. In addition there are hypertension, emotional changes and androgenic changes which apparently are independent of the effects of hydrocortisone on food metabolism. It is evident from this picture that the antagonism of insulin to hydrocortisone does not correct the pathological effects.

FIGURE I

Glucose - Fatty Acid Cycle



## THE THALASSAEMIA TRAIT IN MALTA AND GOZO

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### Introduction

An increased osmotic resistance of the erythrocytes can be tested for, and detected, by mixing a measured amount of fresh blood with a hypotonic solution of inorganic salts of an osmolarity such that the majority of erythrocytes with normal resistance will rupture in it and pro-

duce a clear solution; if the erythrocytes are more resistant than normal under the same conditions, the mixture remains an opaque suspension. A solution of sodium chloride in water which contains 0.35 grams per 100 ml has this critical osmolarity (120 milliosmoles per litre). By making a suspension of erythrocytes from fresh blood in this solution, in a haemocytometer pipette (blood 1 part, hypotonic solution 199 parts) and allowing time for lysis to occur (2 to 5 minutes), individuals with a raised osmotic resistance can clearly be distinguished from those in whom the resistance is normal.

In 1943, Silvestroni and Bianco described this simple test, making use of a

diluted Tyrode solution (osmotically equivalent to a 0.35 per cent. solution of sodium chloride) as the hypotonic medium. These workers were the first to describe, by this method of testing, a familial incidence of increased erythrocyte resistance in otherwise healthy persons ("constitutional microcythaemia" or "thalassaemia minor", this abnormality being associated with certain morphological characteristics of the erythrocytes in the majority of subjects. They also established a relationship between this anomaly and the clinical condition which had been described by other authors as "haemolytic icterus with increased osmotic resistance" (Rietti - Greppi - Micheli Syndrome), such that the anomaly was often found in healthy siblings or parents of such icteric patients. They also describe the genetic relationship between "constitutional microcythaemia" and Cooley's anaemia, such that the former represented the heterozygous state while the latter represented the homozygous state for the thalassaemia gene or genes. By this method of testing, Silvestroni and Bianco have, in a series of population surveys carried out over the last two decades, mapped the geographical pattern of incidence of this anomaly in Italy and Sicily, while numerous investigators have used the same, or a similar, test for the same purpose in Sardinia, Cyprus, Greece, India, Portugal, Portuguese East Indies, Algeria and other parts of the world.

### The Thalassaemia Trait

Great interest has developed since 1948 (when Vecchio described the presence of foetal haemoglobin in the blood of patients with Cooley's anaemia) in the study of the structure and synthesis of the haemoglobin molecule in thalassaemia. These studies have led to a realization that what had been considered to be a simple genetic and clinical entity, in reality encompasses many different genetic conditions, all of which exhibit the abnormal erythrocyte morphology which had come to be considered as characteristic of thalassaemia. According to Fessas (1965): "Recognition of thalassaemia trait

or thalassaemic conditions in general is still a haematological problem, at least at the initial stages of the diagnostic procedure. It may be necessary to emphasize this point, as it is sometimes overlooked. Accepting a deficiency in the production of a major chain (of the haemoglobin molecule), we can expect the effects of this deficiency to become manifest in the aspect of the red cells: microcytosis, hypochromia, a low M.C.H., poikilocytosis. It is realized that the alterations of erythrocyte morphology may vary in extent and, indeed, have been reported as entirely absent in a minority of cases; conversely the presence of an abnormal haemoglobin in the heterozygous form may also be responsible for mild alterations of erythrocyte morphology. Such findings do not alter the basic facts concerning the alteration of erythrocytes in all forms of thalassaemia and I am rather hesitant to accept that the morphological alterations are separable from a true "thalassaemia effect."

The criteria that have been used by different investigators for the detection of thalassaemia are many. Those which have been most frequently used are summarized in Table I. It is worth noting that the microscopical appearance of blood smears was the only criterion used in assessing the frequency of thalassaemia in a recent study in Yugoslavia (Fraser, Grunwald and Stamatoyannopoulos, 1966). Silvestroni and Bianco (1966) have this to say on the criterion of increased osmotic resistance: "This method, if properly carried out, has proved itself useful for the recognition of nearly all microcythaemics and is, even today, the most valuable and rapid single criterion for the identification of microcythaemia": translation from the original in Italian).

Since the erythrocyte morphology is the same in the various presently recognized forms of thalassaemia (alpha thalassaemias, beta thalassaemias, delta thalassaemias, etc.), a test based on this morphological abnormality should detect, without distinguishing between, these different forms. The techniques necessary for identifying the different possible haemo-

globin patterns are still too complex, time-consuming and expensive to be used for extensive population studies. It is not surprising then that a simple, rapid and inexpensive test such as that of Silvestroni and Bianco has been used by many workers for extensive population studies.

### **Thalassaemia trait in Malta and Gozo**

During March 1960, the present writer had an opportunity of visiting the "Centro per lo Studio della Microcitemia e delle Anemie Microcitemiche", at the Istituto d'Igiene of the University of Rome (directed by Professor E. Silvestroni) and of studying, at first hand, the method used by Silvestroni and Bianco in surveying large populations for the incidence of thalassaemia. He also took part in a population study in two localities near Ferrara where the frequency of thalassaemia is amongst the highest yet described. This method was then used (together with microscopical study of peripheral blood smears from those who gave positive tests) in a study carried out in Malta and Gozo during the spring months of 1960, 1961, 1962 and 1963. The results have already been described in detail (Vella, 1961; Vella and Sant Cassia, 1961; Vella, 1962; Vella, 1964).

The occurrence of thalassaemia is well known in patients of Maltese origin. It was first described in an indigenous Maltese family by Mooney (1951). The second report of its occurrence in Maltese patients came from Tunisia (Roche, Derrien, Diacono and Roques, 1953). Since then twelve instances of thalassaemia major (i.e. Cooley's anaemia) have been seen in Malta by Dr. E. Cachia (personal communication) and a set of identical twins with the disease have been seen by Dr. T. J. Agius Ferrante and their haemoglobin pattern studied by the present writer. No indication was available, however, as to the general frequency of the thalassaemia trait and its distribution in Malta and Gozo until the work that will be summarized here.

A preliminary survey was made during April 1960, amongst 2,700 healthy school children of both sexes attending

two large government primary and three private schools in Malta. This sample was considered to be representative of the general population as it was drawn from various parts of the Island. The method of Silvestroni and Bianco was used, the Tyrode solution having been made available by Professor Silvestroni. An increased osmotic resistance was found in 4.4 per cent. of this sample. Noticeable differences in the frequency of positive tests were revealed when the subjects were grouped by village or town of origin. The following percentage frequencies were found: Msida 0.0, Zabbar 1.8, Gzira 1.9, Hamrun 3.2, Qormi 3.4, Valletta 3.4, "Three Cities" 3.7, Birkirkara 4.4 and Sliema 4.6, though the sample size from each of these localities was by no means uniform. The smallest group, that from the "Three Cities", numbered only 54 while the largest, that from Birkirkara, numbered 608. Of 1000 subjects not classed as originating from these above localities, 5.6 per cent. gave positive tests.

A systematic survey was started in 1961 and continued in 1962 and 1963. Healthy pupils of both sexes attending government elementary schools in various towns and villages in Malta and in some parts of Gozo were tested. A hypotonic solution was used which contained 3.5 grams sodium chloride (Analar grade, desiccated) per litre (made up in double-distilled water at the temperature of calibration of the volumetric flask, stored well stoppered at 4°C when not in use and equilibrated to room temperature before use). In the larger schools, an attempt was made to test some 200 pupils, but when the results indicated the need, more pupils were tested. In several places, the majority of the pupils in the village school were tested. The results confirmed and extended those of the preliminary survey.

The complete investigation covered 10,400 children in 39 towns and villages in Malta and revealed an over-all frequency of positive tests of 5.6 per cent. In Gozo, 1,150 children were tested in 5 towns and villages and the over-all frequency for this island was 7.8 per cent. In 14 localities in Malta, the frequency

was above the average and the villages of Mellieha and Marsaxlokk had, by far, the highest frequencies. Only the village of Zebbug in Gozo had a frequency above the mean for the island and this was the highest found in the whole survey.

The following localities gave frequencies of positive tests of about 10 per cent and above and constituted peaks of high frequency surrounded by areas of much lower frequency: Zebbug (Malta) 9.4, Mgarr (Malta) 10, Pawla 10.5, Qrendi 11, Cospicua, 11, Marsaxlokk 15.5, Mellieha and Zebbug (Gozo) 24.5. In Table II are given the percentage frequencies for towns and villages nearest to those with a frequency of about 10 per cent. and above.

The agricultural villages of Mellieha, Mgarr and St. Paul's Bay are of recent origin. The present parish of Mellieha was established in 1844, while St. Paul's Bay and Mgarr became centres of population around 1880. All three localities were first settled by villagers who came mostly from Rabat and Mosta. It is worth noting that the frequencies in the villages nearest to Rabat and Mosta are surprisingly low (Dingli 1.2, Gargur 2.0, Naxxar 2.1, Birkirkara 4.4). It would be interesting to know if the first inhabitants of Mgarr and Mellieha were of the same family stock. None of the localities situated in the north-western half of Malta are as isolated geographically as Mellieha. This village was, until the opening of the bus service to it some thirty years ago, the most inaccessible part of Malta. Even today, most marriages solemnized there are between local residents, while some 17 per cent. of all the adults aged 21 years and over in 1959 and registered in the Electoral List, bore the same surname. From the information made available during the survey, it was possible to group the 741 pupils tested in that village, into 180 sibships each numbering between 2 and 6 children. In 87 sibships only negative results were found, in 60, one child only gave a positive test, and in 33, two or more children gave positive tests.

In the south-eastern (the most densely populated) half of Malta, the highest frequency was found in Marsaxlokk. This

secluded fishing village in the extreme south-east end of the island has a population of nearly 1000 and dates back almost one century. It is known that its population nearly doubled between 1943-1948 and that half of the children who gave positive tests had come from five sibships. This suggests that the abnormality is not as widespread as at first appears, and that it may in fact be restricted to only a few families, probably amongst the oldest in the village.

The only important localities in Malta which were not specifically covered by this survey were those of Mdina, Balzan, Lija, Attard and St. Julian's, though a number of individuals from these areas had been tested in the preliminary survey. Since the population of Mdina is similar to that of Rabat, and that of the other four villages is very similar to that of the large residential complex which comprises Birkirkara, Hamrun, Msida, Gzira, Sliema, Valletta and Floriana, in which the frequencies ranged between 1.9 and 5.1 per cent., there is no reason for suspecting that they may be regions of high frequency.

The village of Zebbug (Gozo) is similar to that of Mellieha and Marsaxlokk in its seclusion. It is one of the smallest villages in Gozo and was described by one prominent inhabitant as being "one large family". Unfortunately, it was not possible to investigate the family relationship of the children who were tested there.

The results of this survey are very similar to those found by Silvestroni and Bianco (1949, 1953) in Sicily, situated only 60 miles from Malta. There, the over-all frequency was 4.45 per cent. but ranged between 2.79 and 10.81 per cent. in the different regions studied. This survey only covered 8,000 persons and it is likely that regions may exist in Sicily with higher frequencies than these. There also, circumscribed foci were found in which the frequency of positive tests was much higher than that for the neighbouring countryside and adjacent to regions with a lower than average frequency not far away. Since the methods of testing and the criteria used for the detection of



thalassaemia in the Maltese Islands and in Sicily were to all intents identical, a comparison between the two studies is valid. The most common variety of thalassaemia found in the central Mediterranean region (Italy, Sicily, Sardinia, Algeria) is one form of beta thalassaemia. It is reasonable to infer that this is also the variety most frequently detected in Malta and Gozo in this study.

### Acknowledgements

It is a pleasure to acknowledge once more my indebtedness to the following, without whose help this study could not have been carried out: Prof. J. Galea, C.G.M.O., Malta, for permission to commence the investigation, and his successor Prof. C. Coleiro, for extending this permission and support; Prof. G. P. Xuereb, Prof. W. Ganado and the Royal University of Malta for laboratory facilities; Drs. G. Borg, A. Cremona, F. Fenech, M. Gatt, G. Hyzler, J. Jaccarini, P. Micallef and M. Sant Cassia, School Medical Officers, for their cooperation and help in testing the children under their care; Drs. A. Depares and J. Giglio, Public Health Officers, Gozo, for arrangements to carry out the study there.

TABLE I

### Criteria used by different investigators for the detection of Thalassaemia

#### Criteria based on erythrocyte morphology

1. Microscopical appearance of the erythrocytes in peripheral blood smears.
2. Increased osmotic resistance.
3. Hypochromia of erythrocytes associated with normal serum iron levels and resistant to iron therapy.
4. Presence of erythrocytes containing foetal haemoglobin as detected by an acid-elution method.

#### Criteria based on haemoglobin pattern

1. Increased amounts of foetal haemoglobin.
2. Increased amounts of haemoglobin A<sub>2</sub>.
3. Presence of an abnormal haemoglobin fraction demonstrable by electrophoretic methods (haemoglobin Barts, H, Lepcrite, Pylos, etc.).

TABLE II

### Frequency of positive test in localities near to regions of high frequency (in per cent.)

Malta	Mellieha	22.0
	Mgarr	10.0
	Mosta	5.9
	St. Paul's Bay	5.5
	Marsaxlokk	15.5
	Gudja	8.0
	Birzebbugia	4.8
	Kirkop	4.7
	Safi	2.9
	Ghaxaq	0.6
	Zejtun	0.7
	Cospicua	11.0
	Senglea	3.9
	Vittoriosa	2.6
	Qrendi	11.0
	Zurrieq	8.2
	Mqabba	6.3
	Siggiewi	4.5
	Pawla	10.5
	Marsa	7.7
Tarxien	6.7	
Fgura	4.8	
Zabbar	1.0	
Zebbug	9.4	
Siggiewi	4.5	
Qormi	3.4	
Luqa	2.0	
Gozo	Zebbug	24.5
	Xewkija	7.0
	San Lawrenz	2.7
	Nadur	2.4
	Sannat	1.0

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## THE FIRST CHAIR OF OPHTHALMOLOGY: a Biographical Sketch of Joseph Barth

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On the 7th April 1818, after a short illness, at the age of 73 years, Professor Joseph Barth, Imperial Councillor, Oculist to His Majesty the Emperor, Professor of sublime Anatomy and Physiology and first Professor of Diseases of the Eyes in the Imperial University, died in Vienna. Well known all over Europe for his ability as teacher and surgeon and for the numerous famous pupils, to whom he was a life long inspiration, his death passed unnoticed in Malta, his native country, and remained so for many years.

Joseph Barth was born in Valletta in 1745. His parents were Nicholas Barth and Maddalena Sceberras. He became a pupil of Professor Michelangelo Grima, Master in Surgery of the Order of St. John in

Malta and member of the Academy of Apathists of Florence. Inspired by his famous teacher, Barth soon showed great interest in the study of anatomy and in its application to Surgery. In order to continue his research work, he proceeded to Rome where he was admitted into the Ospedale Santo Spirito. He never came back to his native land.

It was in Rome that his talent was noticed by Fra Francesco Carlo Von Smitmer, Commendatore of the Order of St. John and Canon of the Metropolitan church in Vienna, who decided to take him to Vienna, where Barth could find all the facilities to study Anatomy and Physiology.

Here, Barth made the acquaintance of Storch who recommended him to Baron Van Swieten from Leyden. The latter had been entrusted by the Empress Maria Theresa with the reorganization of the University and of its medical department, and with laying the foundation of the future world renowned Vienna clinic. Van Swieten, who lost no time in recognizing Barth's talents, enrolled him on the staff of the University and placed him in a good position to start a successful career.

In Vienna, Barth did not devote himself exclusively to dissections. His attention was soon attracted by the number of people suffering from blindness and eye diseases. That at that time the condition of Austrian ocular Surgery was very poor, can be shown by the fact, that in order to operate for cataract on a lady of the Court of the Empress, Baron Wenzel had to be invited to come over from Italy. Much impressed by this state of affairs, the Empress engaged Wenzel to instruct Barth in ocular Surgery.

As luck would have it, some time after, Barth was able to cure of a very painful eye condition the son of Maria Theresa, afterwards Joseph II. In the year 1773 he was asked to occupy the chair of Anatomy and Physiology along with the newly established chair of Ophthalmology. He was also given the title of Oculist to His Majesty and Imperial Councillor.

Of all the operative procedures carried out by Barth as a daily routine, the

only written record is that of Cataract extraction. His methods must have been considered revolutionary. As at that time anaesthesia was not yet introduced, as many as five people were necessary to hold down a patient even for such a short and simple operation as a needling for cataract extraction. He suggested the astonishing idea of operating alone with the patient standing, even backing him against the wall. By means of shouts and threats, he used to crush any sign of opposition or timidity on the part of the patient and reduce him to complete docility. After instructing the patient to pull down the lower lid and look down, he would pull up the upper lid. Then, after tapping the cornea with the back of the blade of the scalpel, thus ascertaining himself of all absence of resistance on the part of the patient, with one quick thrust, he would make a precise incision.

His activity must have been very considerable. In the year 1787, it is recorded that he performed 300 Cataract extractions. The rate of successful operations was 8 cases out of every 12. This is to be considered an excellent performance.

When the Allgemeine Krankenhaus of Vienna was constructed in 1784, two wards were reserved for the treatment of diseases of the eyes and they were placed under the care of Professor Barth. These places have undergone little change since that time. Barth's portrait in oils still hangs, along with those of his successors, on the wall of the office of the second Augen Klinik at present occupied by Professor Hruby.

Barth's lectures and work attracted pupils from all over Europe. Amongst these is to be mentioned Joseph Beer, who as his assistant and successor in the chair of Ophthalmology carried the speciality to unexpected heights. Other pupils were Adam Smith, Bocharke, Quadri of Naples and Magistrati of Milan.

At one time, Barth was at the centre of a controversy which troubled the serene waters of the Viennese Court and Medical world. Along with Von Storch and members of the faculty of medicine, he formed part of a commission entrusted with the

investigation of Messmer's cures in general and the case of the pianist Maria Theresa Von Paradis, in particular. The latter, an infant prodigy and blind since the age of 3, was a daughter of one of the Empress's private secretaries. Her sight was suddenly restored by Messmer. The delegation scoffed at Messmer's methods. They said that the cure was a delusion and if the patient could now see, she had never been blind. Barth further contended that the patient must still be considered blind because she did not know what the objects shown to her were called.

It seems that Barth never quite forgot his native country, for in 1789 though only 44 years old, he manifested his intention of leaving Vienna and retiring to Malta. As the Emperor Joseph II was faced with the loss of his only ophthalmic expert, he made a contract with Barth. The latter would train two doctors in ocular surgery for 1000 guldens a year. The choice fell on a German, Dr. J. A. Schmidt and on Ehrenritter. The latter was the most promising, but he died when very young. It took Schmidt four years to finish his training under Barth and pass the required examination by a special commission of Surgeons and Physicians. Six Cataract extractions had to be successful out of 12. In this way started the first government sponsored training in Ophthalmology. J. A. Schmidt lived long enough to found along with Himly the first journal devoted entirely to Ophthalmology the "*Ophthalmologische Bibliothek*".

The two weak points of Barth seem to have been money and antiques. A story is told by Ernst Fuchs of one occasion, when after treatment of the Empress for an eye condition, the latter said: "Let him take that", pointing to a purse containing his fee on an elegant valuable table. Barth took the Empress at her words, placed the purse and the table under his arm and went away.

In 1791, he was given a pension of 1000 guldens, over and above his ordinary salary. He was thus able to devote most of his time to his life long hobby, that of collecting antiques. His talent in this sector was very well appreciated even by



Portrait of Joseph Barth at the second Augen Klinik, Vienna.

such celebrities as Canova, who was one of his great friends.

It seems that the changes which followed the disappearance from Malta of the Order of St. John dissuaded Barth from returning to pass his last days in the Island where he was born. When he died, his most valuable collection of statues, paintings and cameos was taken over by the Emperor Leopold II.

It is a pity that Barth never cared to put down in writing his public lectures. He published very little. In 1797, his Monograph "*Cataract Extraction for the practising surgeon*" appeared in Vienna. His other publication "*Table of muscles*" is the fruit of his accurate dissections.

Barth's greatest and most useful achievement was the establishment of the first University Eye Clinic, where pupils from all over Europe were instructed, trained and encouraged to lay the foundations of similar eye clinics in their own countries.

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## THE CAUSES OF TOOTH LOSS

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The great majority of permanent teeth are lost because of dental caries or periodontal disease. Whilst everyone is painfully aware of the consequences of dental caries, the insidious and painless breakdown of the supporting structures of the teeth is accepted as a natural process of ageing. This fatalistic attitude is, however, wrong and recent increased interest in the prevention and treatment of periodontal disease has clearly shown that intelligent management may avoid or considerably

delay the tragedy of edentulousness.

Careful planning of preventive dental health measures must be based on accurate assessment of the factors causing tooth loss. Surveys in many parts of the world have already shown that periodontal disease is of major aetiological importance. This paper reports a pilot study into the causes of tooth loss in patients attending the Dental Department, St. Luke's Hospital, carried out in July 1964.

### Materials and methods

A note was made of 500 consecutive dental extractions, under local anaesthesia, from 297 patients (117 males, 180 females). The causes of tooth loss were classified into three main groups, viz:— (A) Caries; teeth, including roots, extracted because of complications directly attributable to dental caries. (B) Periodontal disease. (C) Sound teeth extracted for prosthetic or orthodontic reasons.

### Results

Table I shows that caries and periodontal diseases are evenly balanced as factors causing tooth loss but when the figures are broken down into those for persons under 30 years and those over 30 years of age, a different pattern emerges (Table II). The increase of periodontal disease as the factor of tooth loss from 3.2 per cent. to 43.4 per cent. is of great practical importance. Indeed, periodontal disease is responsible for the loss of approximately 60 per cent. of teeth extracted from persons over 30 years of age.

### Comment

Epidemiological studies in several parts of the world (Pelton et al., 1964; Mehta et al., 1958; Lacronique, 1964 and Jackson, 1965) have shown that periodontal disease becomes the major cause of tooth loss in adults. Valid comparisons are difficult in view of the greatly varied racial, social and economic background of the groups studied. The personnel studied must always be clearly defined. Our results are drawn from a selected group of low income bracket and of mixed rural or

urban origin. Similar studies on the middle class population of Malta would be useful but can only be carried out by the general dental practitioners. The data required for each dental extraction would be sex, age, cause of dental extraction, type of anaesthesia and tooth extracted. A consecutive series of extractions must be recorded to avoid any bias.

World public dental health authorities are realising that the only hope of reducing the present high incidence of dental disease is through preventive measures (W.H.O., 1961). Dental caries has its highest incidence during childhood and early adult life and from then on periodontal disease becomes the chief factor of tooth loss. Epidemiological surveys on the incidence of periodontal disease in school children have shown that the seeds of this disorder are already sown at that early age (James, 1963), and it is only because of the chronicity of the disease that its effects show mainly during late adult life.

It is being increasingly realised that the only hope of preventing the ravages of periodontal disease is through regular dental attention commencing at an early age. Basically this consists of a balanced diet and correct oral hygiene carried out in cooperation with the family dental practitioner, who must be made more aware that periodontal disease is just as important a factor in tooth loss as dental caries.

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**TABLE I**  
**Causes of tooth loss**

	<i>Caries</i>	<i>Periodontal</i>	<i>Prosthetic</i>	<i>Total</i>
No. of teeth	248	233	19	500
Percentage	49.6	46.6	3.8	100

**TABLE II**  
**Causes of tooth loss in persons under 30 and over 30 years**

Under 30	111 (22.2%)	16 (3.2%)	—	127 (25%)
Over 30	137 (27.4%)	217 (43.4%)	19 (3.8%)	373 (75%)

## A POSSIBLE HAZARD OF SPLENECTOMY

### Case Report: Torsion of the Wandering Spleen

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King and Shumacker (1952) were the first to draw attention to the possibility of fulminating infection occurring after splenectomy. They described the cases of

five infants under six months who underwent splenectomy for congenital haemolytic anaemia. Four of them developed fulminating meningitis or meningococcaemia in from six weeks to three years after operation and one of them died. The fifth infant developed a rapidly fatal febrile illness. Since then several papers have been published supporting the findings of King and Shumacker.

### Case Report

R.E., a four month old male infant, was admitted to St. Luke's Hospital on 11 May, 1965 for "gastroenteritis and dehydration". On the morning of admis-

sion, he had vomited bile-stained fluid three times and he had passed three loose stools over the previous twentyfour hours. The baby had been constipated for two days before then but otherwise he had been healthy since birth. He had been delivered normally at full term and his birth-weight was 9 lbs.

On examination, the infant was pale and sallow. Temp. 99.6 F., Pulse 104/m., Resp. 40/m. No cyanosis, jaundice or lymphadenopathy. Heart and lungs n.a.d. Abdomen was somewhat distended and a distinctly palpable mass could be felt occupying the right lumbar region and extending into the right iliac fossa. Investigations: Hb 72%, WBC 11200/c.mm., Neut. 57%, Eos. 1%, Lymph. 34%, Mono. 8%. A straight film of the abdomen (12 May 1965) showed small fluid levels and a soft shadow in the right iliac region. Urine analysis: traces of protein and a few granular casts. Blood urea: 56 mg./100ml.

On admission, the infant was put on intravenous fluids, the stomach was aspirated at intervals and penicillin and streptomycin were started. However, his condition continued to deteriorate and by the morning of the thirteenth May the stomach aspirate had become coffee-ground with fresh blood occurring at intervals. It was therefore decided to explore the abdomen.

Through a right paramedian incision, the mass in the right lumbar region was found to be the spleen, measuring  $8.8 \times 6.6 \times 4.2$  cms. The convex "diaphragmatic" surface was lying against the anterior abdominal wall. The pedicle attached to the hilum showed three twists and was 9 cms. long, disappearing towards the left hypochondrium. Splenunculi were embedded in the pedicle, the largest of which was 0.7 cms. in diameter. All other organs were in their normal position; there was no intestinal malrotation. After transfusion and ligation of the pedicle, the spleen was removed and the abdomen was closed in layers without drainage. Microscopical examination of the spleen, which weighed 99 grams — the normal average weight in a three to six month old boy is 18.9 grams (Spector, W.S., 1956) —

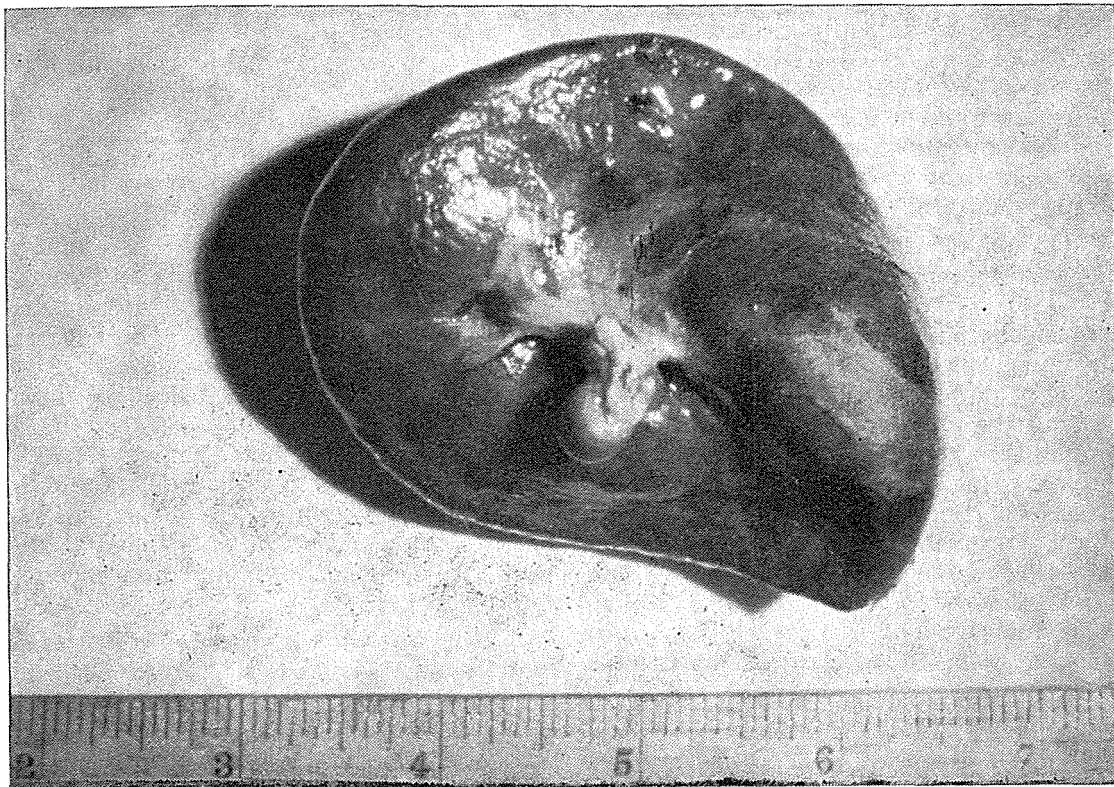
showed recent haemorrhages in the pericapsular region, as well as congestion of sinusoids in the vicinity. Malpighian corpuscles were not prominent. There was autolysis of the inner pulp.

The infant received a transfusion of 30 c.c. of blood during and after the operation from which he made a good recovery. In the postoperative period, he developed an enteritis which was treated with furoxone. No pathogenic organisms were grown from the stools. The sutures were removed on the tenth day. A mild sepsis subsided after extrusion of some catgut. The infant was discharged in a healthy state on 16 June, having steadily gained two pounds in weight over the previous month. Postoperative investigations on the infant and its parents failed to reveal any evidence of congenital haemolytic anaemia.

This was then a case of torsion of a wandering spleen. It is a rare surgical emergency and up to 1952 less than 150 cases had been described (Maingot, R., 1952). This is the only such case recorded so far at St. Luke's Hospital since it was opened in 1951. A wandering spleen, a rare condition itself, may be found in any part of the abdomen and it is particularly liable to torsion (Aird, I., 1957).

On the 25th June, 1965, the infant was seen in the paediatric outpatient department and was found to be in excellent health. Late the same afternoon, he was admitted as an emergency in a state of semi-coma in a very ill condition. His temperature was 103°F., pulse rate 170/m., respiration rate 76/m. There was a generalised purpuric rash, more marked in the extremities, and the skin was dry and dehydrated. There were no cyanosis, anaemia or jaundice. There was no obvious localising infection and a diagnosis of fulminating septicaemia was made. Penicillin, streptomycin and chloromycetin were started and an intravenous drip was set up. During the next fifteen hours, the infant vomited twice and passed loose yellow stools three times. The only investigation carried out was a blood count: Hb 88%, RBC 4580000/c.mm., WBC 28000/c.m., Neut. 83%, Eos 2%, Lymph.





**Figure: the visceral surface of the spleen distinctly shows the largest of the splenunculi found at operation as a round knob in the centre of the organ.**

12%, Mono 3%, Platelets 100700/c.m. The patient was dead at 8 a.m. on 26 June, 1965. Permission for autopsy was refused.

### **Discussion**

King and Shumacker (1952) thought that serious infection was likely to occur after splenectomy only in infants. Gofstein and Gellis (1956) concluded in their review of cases that there was some correlation between splenectomy and sepsis but did not find a relation to age at operation. Smith et al. (1956, 1957) put the incidence of severe infection after splenectomy at 28% in their series of infants and children. Huntley (1958) discussed seven patients who developed severe infection after splenectomy in a series of 43, and five of the seven were under one year. Four out of nine infants in Horan's series (1960) died. Broberger et al. (1960) calculated at 20% the risk of serious infection after

splenectomy with a 50% mortality rate if splenectomy is performed in infancy. On the other hand, Robinson and Sturgeon (1960) found the incidence of a serious infection to be 10% in their series and about 5% in other series. These last two authors introduced the phrase "life-threatening infection" to describe these cases. Lucas and Krivit (1960) saw only a slight but real increased susceptibility to infection after splenectomy. Lawdon, Walker and Walker (1962) described four cases of severe infection in 75 children who underwent splenectomy for various reasons. One of the four was under one year and two of the others died. Horan and Colebatch (1962), in a careful analysis of their own and other series, came to the conclusion that serious infection in childhood is of the order of 8% and the incidence of fatal infection is close to 5%. Lowdon, Stewart and Walker (1966), in an exhaustive study of 1167 patients



undergoing splenectomy in England and Wales in 1961, stated that "it seems clear that in about 2% of cases splenectomy does predispose to serious infection".

A number of authors have none the less denied this casual relationship. Thus, Cole, Walter and Limarzi (1949) found no cases of infection in their series; Miller and Hagedorn (1951) did not mention serious infection as occurring during the five year follow-up of some 75% of their 140 cases. Gross (1953) merely states that he found little evidence to support the view that serious infection may arise after splenectomy. Of the same opinion were Walter and Chaffin (1955) and also Laski and Macmillan (1959) whose paper though, was strongly criticised by Greenberg (1960-61). Other authors reported single cases with no resulting untoward infection.

### Characteristic pattern

A study of the literature reveals a characteristic clinical pattern in a large majority of cases of serious infection after splenectomy — a pattern that is also shown by the cases above described. A septicaemia, with or without meningitis, takes a rapid fulminating course and becomes associated with marked circulatory collapse and also sometimes with the Waterhouse-Friedrichsen syndrome. The incidence of septicaemia and meningitis in such cases is much higher than in the normal population (Horan and Colebatch, 1962). Pneumococci have been the immediate cause of serious infection in some 50% of recorded cases (Lawdon et al., 1962). Other organisms involved have been the following: Meningococcus, H. influenzae, E. coli, Staphylococcus aureus, Streptococcus haemolyticus. 8% of these infections occur within two years of splenectomy (Smith et al., 1957; Horan and Colebatch, 1962). Another feature is the recurrent nature of infection — some 40% of cases had recurrent episodes of serious infection (Lawdon et al., 1962; Horan and Colebatch, 1962). A few of the recorded infections have not been septicaemias. Thus, fatal tracheobronchitis (Gofstein and Gellis, 1956), the re-activation of tuberculosis (MacPherson, 1959) and the

dissemination of lupus erythematosus (Carpenter et al. 1959), among others, have been laid at the door of splenectomy. But not all authors (Robinson and Sturgeon, 1960) will accept the casual relationship in these cases.

### Factors influencing reaction to splenectomy

The age of the patient is an important factor for there is no doubt that serious infection is most common if splenectomy is carried out in the first year of life, though later ages are not exempt. It should be possible in most cases to defer the operation until the school period as conservative measures almost always achieve symptomatic control of the primary condition. Hence, splenectomy is rarely indicated in the first year of life; and, during childhood, it should not be recommended solely for haematological abnormality but for real disability.

Huntley (1958) noted that three of the seven patients in her series developing severe infection after splenectomy had a primary disease which made them more liable to infection. Robinson and Sturgeon (1960) presumed that in eleven of the thirteen children who developed serious infection after splenectomy in their series of 110 the infection may have been due to the primary condition like spherocytosis or portal hypertension. Horan and Colebatch (1962) concluded from a study of several series that the risk of serious infection occurring in splenectomised patients was as follows: 0% in cases of traumatic rupture; 2% in hereditary spherocytosis and idiopathic thrombocytopenic purpura; 10% in thalassemia and fully 21.4% in patients undergoing splenectomy for portal hypertension. Patients on continued treatment with corticosteroids are also more liable to serious infection after splenectomy. However, the primary disease factor does not appear to be related to the high incidence of fulminating infection in the first year when splenectomy is rarely performed for conditions that themselves predispose to infection. Smith et al. (1957) and Lawdon et al. (1966) recommended that antibiotic (penicillin cover should be given two years after splenectomy to those

infants and children in particular as well as to other patients who, in the light of what has already been stated, are considered to be specially at risk.

Though there can be no doubt that in individual cases splenectomy may predispose to infection, the reason is far from clear. The spleen is the largest single reservoir of lymphocytes in the body and is concerned with antibody production. However, in many cases extensively investigated after splenectomy (Smith et al, 1967; Krivit and Good, 1959; Broberger et al., 1960), no abnormality liable to increase susceptibility to infection has been discovered, nor was any alteration found in known immune mechanism. Saslow et al. (1959) found that 90% of splenectomised adults and older children were able to produce antibodies in amounts equal to those produced by controls. There were, though, a small number of splenectomised patients who showed a lesser antibody response than controls. Hence, Lucas and Krivit (1960) conclude that "a change in immunologic capacity after splenectomy may be an individual characteristic rather than a regular consequence".

The spleen is also concerned with the removal of micro-organisms from the bloodstream. Kerby (1950) showed that the reticulo-endothelial system of animals is less efficient in removing encapsulated bacteria from the bloodstream than in removing non-encapsulated bacteria. This may explain the 50% incidence of pneumococcal infection in these cases.

Recorded cases of congenital asplenia in children and adults coming to autopsy have been few. It is interesting to note that many of these died of fulminating illnesses associated with the Waterhouse-Friderichsen syndrome, or have suffered from life-threatening pneumococcal infections and recurrent attacks of meningitis. As this is the pattern of serious infection occurring after splenectomy, there is no doubt that absence of the spleen does predispose to serious infection (Horan and Colebatch, 1962).

### Summary

The first case of torsion of the wandering spleen in St. Luke's Hospital in a

four month old infant, is described. The infant died from fulminating septicaemia just over six weeks after splenectomy. The question of "life-threatening infection" after splenectomy is discussed. There is no doubt that it sometimes occurs, particularly in infancy and in patients suffering from a primary disease which predisposes to infection or which has required the prolonged use of corticosteroids. It is suggested that splenectomy should not, if at all possible, be performed in the pre-school child, and that antibiotic cover might well be given for two years after operation to those patients specially at risk. It would be advisable to follow up all splenectomised patients carefully and to treat, early and vigorously, any infection no matter how minor it may appear to be.

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## THE VALUE OF THE HISTORY OF MEDICINE IN MEDICAL EDUCATION

(St. Luke's Day Oration, British Medical Association, Malta Branch, 18 Oct. 1960)

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Among the several reasons that have concurred towards my choice of subject for this St. Luke's Lecture are, firstly, a personal inclination to the study of the History of Medicine of several years' standing and, secondly, a potent reinforcement of this interest by way of a very recent archaeological tour of Greece and the Levant which took me to several sites that St. Luke, in the company of St. Paul, must have visited before that eventful day when a propitious Gregale blew them to our shores — a happening which, among other effects, has resulted in this celebration of St. Luke's Day by us doctors in Malta.

Of St. Luke as a Physician we know little or nothing by way of direct evidence. Even legend has nothing to say of him in this respect, unlike its proliferation as regards his prowess as a painter, whereby hundreds of churches in Europe have made attribution to him of hundreds of their Byzantine or even more recent ikons! That he was a physician cannot be denied, since he is given that specific title by St. Paul himself, and there is much internal evidence besides this in his Gospel and in the Acts of the Apostles. Nor can it be doubted that it was both as physician and as

companion that he went with St. Paul to Cos, Rhodes, Athens, Pergamum and other places that have this summer been the route of my own historical pilgrimage.

The Asklepeia of Cos, Pergamum, Epidaurus, Athens and Corinth were medical shrines of the ancient world which naturally held great interest for me and which St. Luke must surely have visited. Asklepios, if not of divine origin, certainly became in time the Greeks' God of Medicine as Imhotep was to the Egyptians. Homer's testimony would make of him a historical, and not just a legendary personage and his sons Podalirius and Machaon figure as surgeons at the siege of Troy; and who, since Schliemann's excavation of Troy and Mycenae, would doubt the factual historical basis of most of Homer's epic? Every one of the temples of Asklepios was a marvellous combination of holy precinct and medical centre, beautifully sited, offering all the amenities and healing aids that we seek in our spas. There the sick in body and mind congregated not only to be attended by the temple priest-doctors, but also to submit at the incubation to the nocturnal visit of the god in the shape of his sacred serpents; and thence went the healed, loud in their praises, leaving behind them tokens of gratitude in the shape of the "ex voto" that now lie on the shelves of the museums.

I stepped on Cos to stand in the shade of the ancient plane tree sacred to Hippocrates, Father of Medicine. He belongs to History, with records vouched for by Plato, and a fairly reliable birthdate of 460 B.C. His title he well deserves even if only because of Celsus's tribute that he "first separated medicine from philosophy", that is, made personal observations and drew inferences free of preconceptions. The famous Hippocratic Oath in the form that we know it dates only from the 3rd century A.D., but something more than pious belief must have gone to its attribution to Hippocrates. So also the great corpus of 70 to 100 treatises that form the Hippocratic collection is undoubtedly the fruit of various authors at very various dates, but much of it bears the unmistake-

able imprint of one great mind, to whom we must owe the striking Aphorisms, the descriptions of operative technique and, most remarkable of all, the vivid clinical records of actual cases.

Pergamum of the lofty Acropolis and elegant Asklepeion was the birthplace of Galen, and therefore a highlight of my pilgrimage. His was the figure that loomed giganticly in the medical field for many centuries after his practice in Rome about 180 A.D. A hundred works of his became the unimpeachable Bible of medicine, and they were the product of his remarkable activity as the first experimental physiologist and one of the first practical anatomists. He created medical science. For good and for ill, because, naturally, his writings contained fundamental errors as well as much sound truth, his enormous influence on all medical thought and teaching lasted right up to the Renaissance; and it is his blind followers rather than himself who must be blamed for perverting the authority of Galen into a stifling authoritarianism.

Nearer to us in time and place, and of particular relevance to the Maltese doctor, is Rhodes where the Knights Hospitallers of St. John have left us a Holy Infirmary, a smaller but no less interesting prototype of the great Infirmary that made Malta the envy of Europe's hospitals 300 years ago. The Infirmary at Rhodes is well-preserved and functions as a most interesting museum, in contrast to the battle-scarred and derelict state of our former proud edifice, which yet awaits reconstruction as its old clinical records yet await rediscovery and publication.

The value of the History of Medicine in medical Education is my theme, but I could just as well have chosen as a subject, and made out a good case for, the utility of the history of medicine in medical teaching. The differences entailed by my choice of words are real enough and not just terminological, and I must in the first place stress the cultural before I pass to the utilitarian aspect.

No branch of knowledge stamps a firmer or more authentic hall-mark on the cultured and educated man than does His-

tory. History, like everything else, has had its detractors and even its debunkers, and these not only among industrial tycoons; such superficial evaluators of it need not detain us. In as much as medical education rightly claims to be a liberal education, or even an education at all, it must entail a study of the history of medicine. On a reciprocal basis, the history of medicine contributes to the study of general social history in no small measure. Instances abound of how the course of history has been altered by disease. Even as but a part of the wider history of science, the history of medicine teaches general history and therefore represents essential culture.

That Medicine is a science is a self-evident truth which in our day is apt to be over-emphasised rather than the reverse. That Medicine is also an art is no less true, and to the art of medicine the history of medicine can contribute vitally. "A subject that loses its traditions is like to lose its soul", and the traditions of medicine are enshrined in its fascinating history.

Utility, pure and unashamed, also calls in the powerful aid of the history of medicine in the teaching and learning of the facts of medical science. Many a lecturer has gratefully resorted to the more dramatic and even some of the romantic highlights from medical history to re-awaken the flagging interest of a class of bored students! The anecdote which if not strictly "*vera, è ben trovata*" is a great stand-by, and to this category belongs the account of Edward Jenner's appreciation of the pretty milk-and-roses complexions of his dairy-maids and his inferences that they might have owed them to their protection by cowpox from the ravages of smallpox; and again the story of how Halsted came to introduce surgical rubber gloves to protect the hands of his favourite theatre-sister from strong disinfectants!

If medical history, like all history, tends to repeat itself, then a study of the errors that have bedevilled medical progress through the centuries should be a powerful preventive of the repetition of those old errors. It has been said that "the energies of the scientific worker are em-

ployed in correcting the mistakes of his predecessors, and in making new ones of his own". At any rate, making new mistakes is more excusable than repeating known old ones.

Hence it is that eminently practical men who have shaped the curriculum at many famous medical schools have inserted some amount of study of medical history as an obligatory subject, while at many other schools the courses in medical history are voluntary but none the less highly appreciated. At several schools the history of the various medical branches is taught separately by the various teachers, while at others there has been wise acceptance of Osler's insistence that the History of Medicine constitutes an integral entity of the highest grade, fully deserving departmental status with its own staff engaged in both teaching and research.

It is obviously outside my compass to survey medical history generally or exhaustively, or to limit my observations to but one aspect. I think I can best illustrate my view on the value of the history of medicine by some examples from the various branches of medicine that will indicate how the achievements of modern medicine can be traced back to their roots in history, or how certain basic medical principles are illuminated by reference to their historical development.

Anatomy offers itself as the prime and fundamental subject whose origins fade into the mists of time, while yet presenting to-day new facets of an ultra-modern character. The whole subject is redolent of history. Eponymous nomenclature, though now unjustly fallen into disfavour and disuse, is a continuous record through thousands of years of those "magna et minora sidera" who have shone in the anatomical firmament. To be sure, Vesalius deserves infinitely greater remembrance than is accorded to him by an inconstant cranial foramen and tarsal bone, while Poupart probably does not deserve at all that the inguinal ligament should bear his name; so also the uncouth student has been known to attribute the tendo Achillis to anatomical discovery rather than to mythology!! Yet the con-

noisseur will see in eponymy an admirable historical memorial and also a way of following the injunction in Ecclesiastes: "Let us now praise famous men".

The explosive expansion of anatomical knowledge at the Renaissance is as remarkable as the exuberant flourish shown by every other science and art at that climacteric, and is attributable to much the same reasons. Of special significance, however, was the revolutionary concept that authority, even that of Galen, had to be dethroned and replaced by the undeniable evidence of direct personal observation. Right up to the time of Sylvius, the Galenical teaching was quite literally placed on a high pedestal from which the professor expounded it, while far below him the humble and often untutored demonstrator did the actual dissection and tried to make what he found agree with what Galen had described, even when this was quite fanciful or erroneous or based on the dissection of animals. It was Vesalius who first had the temerity to do all his own dissecting almost free of Galenical preconceptions, even to cast grave doubts on the correctness of the sacred texts. The printing press and the artistic genius of Calcar, his illustrator, then gave the world the Anatomy of the new and the true dispensation.

From the history of anatomical discoveries the student can hardly fail to be impressed by the enormous amount of fine and correct detail that was determined by the old anatomists hundreds of year ago with equipment of the most primitive kind, aided only by an insatiable avidity for knowledge and brilliant powers of observation. Surely this should spur the student to a serious attempt at rediscovering for himself the wonders of structure. This is one reason why we so wisely insist on the student learning his anatomy by doing so much dissecting himself.

The fascinating history of dissection has been well documented. It ranges from the blind fumbling of the Egyptian embalmer to the meticulous analysis of the seventeenth century anatomists. Recording the difficulties that have always attended the provision of adequate material,

should serve to impress the student with a proper and grateful regard for the privilege accorded him in dissecting the human body.

The history of Physiology and that of Anatomy are as indissolubly linked as Function to Structure. In fact, History has no more valuable lesson to give us than that wild hypotheses of function divorced from correct observations of structure have been the source of most medical errors through the centuries. Erasistratus and Galen established the theory of the Pneumata, whereby the "natural spirits" were conceived as passing from the liver into the veins, the "animal spirits" from the brain along the nerves, and the "vital spirits" from the heart into the arteries. Galen did demonstrate correctly that the arteries contained blood and not air; however he fell into the fundamental error of postulating invisible perforations in the cardiac septa, to suit his physiological hypothesis even in the face of lack of anatomical proof. For centuries this prevented any approach to the true concept of a circulation of the blood until Harvey's discovery of it in 1620, based at least in part on correct observation of the structure of the valves in veins as demonstrated by Fabricius in Padova in 1603.

Much may be learned from a study of how modern Physiology has developed historically with and from progress in experimental chemistry and physics. The ancients conceived the burning of a substance as being due to its losing "phlogiston". Joseph Black showed that the exact reverse was the case, combustion involving combination with some substance in air. Boyle and Hooke showed that air is essential to most forms of life, and Mayow in 1670 showed that "some constituent of air necessary to life enters into the blood in the act of breathing". The discovery of Oxygen by Priestley came in 1774, and then the stage was set for the exact elucidation by Lavoisier of respiration as a vital function. So again the development of the physics of electricity from Galvani's experiments with frogs' legs led rapidly to significant physiological advances.

Instances abound of how random and disconnected physiological observations accumulate through the years until some genius realises their collective import and integrates them into a great discovery. Of this nature is the story of the discovery of the vitamins. The British Navy in the days of Captain Cook learned empirically that lime juice protected its sailors from scurvy. Dimly realised was the connection of malnutrition and lack of sunshine with rickets, when this condition was so common in English children that it was termed the "English disease". My audience will be specially interested in the well-authenticated observation that the starving French garrison of Valletta in 1799 found its sentries incapacitated by night-blindness. Many years later came the discovery that Chinese coolies who preferred polished rice to the whole grain for their staple diet went down with beri-beri. These four instances contain the kernels of the vital information that could have led to a precocious discovery of vitamins A, B, C and D!

Among all the medical disciplines Pathology stands out, fundamental and pre-eminent. Its history reveals how the postmortem room qualifies as the foundation of the whole medical edifice, or, to change similes, as the great Temple of Minerva Medica herself. Morgagni at Padova performed thousands of autopsies, resulting in 1761 in the publication of his monumental "On the Seats and Causes of Diseases" containing full clinical case-histories with the relevant post-mortem findings. Thus he introduced and established the anatomical basis of disease processes. Not long after him, John Hunter in England made Pathology the basis of the practice of Surgery, creating his fantastically rich museum which not only served as teaching material for countless generations of students till this very day but became a model for pathological museums throughout the civilised world.

Medicine as an art and as a science is learned and practised at the bedside as the very word "kline", Greek for bed, denotes in clinical medicine. As such it is based essentially on practical observation

of the patient. At its origins, however, it was more a matter of theorising on fanciful hypotheses. According to the Aristotelian and Hippocratic canons, there were in the world four qualities: the hot, the cold, the wet and the dry which in binary combination gave rise to the four elements of earth, air, fire and water. From the elements derived the four humours — blood, phlegm, black bile and yellow bile which manifested themselves in man as the sanguine, phlegmatic, melancholic and choleric temperaments. Disease was a matter of disturbance of temperament and medicine observed these changes and sought to correct them, within the narrow limits of these preconceived theories into which all medicine had to be fitted. It was Sydenham (1624-1689) who classified and described numerous distinct clinical syndromes and diseases on a rational basis, with a specific insistence "Go to the bedside; there alone you can learn disease". The great Dutch school particularly under Boerhaave gave an impetus to this new process of distinguishing an infinite variety of diseases. A notable advance at Leyden was that its doctors were the first to obtain their qualifying degree on their examination of actual cases and not on disputation of theses.

The story of Hygiene and preventive medicine teaches how the life and happiness not of individuals but of whole nations has lain at the mercy of disease, and still more of ignorance of disease. It is medical advance in the march of time that has given us not only a greatly increased span of life but a world fit to live it in. In basic terms civilisation can almost be equated with sanitation, and the greatest of Rome's contributions to medicine were abundant water supplies, baths and efficient drains. Moreover, the beneficent spread of Roman colonialism throughout the ancient world, like its British counterpart in our time, saw to it that the mother country fostered similar progress in its dependencies.

Immeasurable and invaluable have been the effects of Jenner's introduction in 1796 of vaccination against smallpox, the classic from which stems all modern

immunology. Epidemiology has its roots in John Snow's demonstration in 1894 that cholera is a waterborne infection and William Budd's discovery in 1873 of the contagious nature of typhoid, in the period when Edwin Chadwick created public health organisation. Malta's one luminous contribution in this field came with Zammit's discovery in 1905 that goat's milk was the agent in spreading undulant fever.

War and disease have been aptly termed the fatal partners. So often in the history of mankind have they collaborated in decimation, that the long record of the great pestilences often runs parallel with that of the great campaigns. Yet the exigencies of war, which have often advanced science, have also led perforce to medical progress. One example I would cite here is Florence Nightingale's work in the Crimea which led to her creation of modern nursing and of modern army medical services.

Bacteriology supplies the best instance of how in medicine, as in war, the critical break-through opens up vast new fields when all advance has seemed to be halted. The discovery of the bacterial cause of so many of the most important diseases must rank as an event of shattering import. As far back as 1546 Frafastoro gives indications of dim glimmerings of the truth with his "contagium vivum", but it was van Leeuwenhoek who, in 1675, through lenses he had made himself first saw bacteria. Other tentative and ineffectual gropings in the dark are made till the great sun of Pasteur's discovery shoots upon the horizon in 1860. Surely guided by Destiny he works from research on yeast to fermentation in wine and then to disease in silkworms until he can prove that putrefaction is caused by living germs. Destiny now produces the other "man of the hour" in Lister who seizes immediately on Pasteur's theory for the principle he required in controlling wound sepsis by chemical disinfection. Yet wonders will never cease and high drama operates in our own prosaic days when a mould from the murky London air contaminates a bacterial culture in the laboratory of St. Mary's Hospital, and Fleming

observes, acts, infers, records — and makes more medical history.

The dramatic irruption of Surgery into the dynamic, vital, and progressive art and science of the last hundred years from the crude status of mere manual dexterity and craftsmanship of all the centuries before, we owe to the discovery and development of the essential ancillaries of anaesthesia and antiseptics. Before this, surgery was shackled and handicapped. Traumatic and emergency conditions supplied practically all the indications, or rather the excuses, for surgery; hardly ever was it elective or deliberate. Surgery was all too often the counsel of despair, the last resort, "desperate remedy" indeed. It is not altogether surprising that to this very day residual fears and prejudices linger in the folk-memory of the uneducated. Horrors of excruciating pain accompanied the surgery of the bad old days, and the final disappointment of death from infection all too often followed the patient's submission to the torture. Until about a hundred years ago surgery was, in more than one sense, at a dead end.

Anaesthesia was the first step forward. In the early 1800's Humphrey Davy and Faraday suggested "laughing gas" and ether respectively as possible pain killers during surgical operations. It was "from the New World" that the practical application came, in 1842 with Long's use of ether and in 1846 with Horace Wells' use of gas, initially for tooth extraction, followed in the same year by Morton's use of ether for excision of a tumour in the neck at Massachusetts General Hospital. On this historic occasion, the surgeon paid his pithy but eloquent tribute with the words "Gentlemen, this is no humbug!" Within the year, Dr. Oliver Wendell Holmes gave the world the term "anaesthetic" and in London Robert Liston having amputated a leg under ether turned to his admiring audience saying "This Yankee dodge beats mesmerism hollow!" So fast did the glad tidings spread that within 3 months, in March 1847, Spencer Wells was using ether at Bighi Hospital, Malta.

For as long as the world shall last,

the history of Surgery will be divided into two eras, "Before Lister" and "after Lister". Anaesthesia made surgery endurable; antiseptics made its results durable, by saving the lives of those who had formerly run risks from infection no less serious than those of the operation itself. Before Lister, disastrous infection could follow the simplest surgery, so that a king who was gratefully surprised at the happy outcome of the excision of his sebaceous cyst rewarded his surgeon with a knighthood. The surgeon operating in an old frock-coat encrusted with the blood and filth from hundreds of previous operations, pulling his ligatures out of the buttonhole where he had threaded them at the start of the day, picking his scalpel from the dirty floor, could see no connection between these conditions and the "hospital fever" which carried away his patients days after the operation. He welcomed the suppuration of his operation wound with "pus bonum and laudabile", knowing only vaguely by tradition and experience that this indicated a localised and non-invasive infection. Contamination and infection were indeed quite meaningless terms until Pasteur revealed the essential role of bacteria in these processes. The genius of Lister lay in his appreciation of the significance of Pasteur's discovery, and still more in his practical application of chemical methods of destroying bacteria i.e. antiseptics, soon to be followed by the sounder principle of asepsis. Thus did he earn in 1865 the incontestable title of Father of Modern Surgery. Thus did Surgery at last attain the therapeutic ideal of "cito, tuto et jucunde". The advance thus made possible has resulted in the surgeon being deterred by no pathological condition in any site of the body, so that surgery is now in the proud position of being confidently sought as the preferred and sure means rather than the desperate remedy, as the safe and certain cure where all other therapy may be impotent.

If these and so many other medical specialities have such fascinating aspects to their millennial history, no less does the basic discipline of General Practice through the ages present notable features



of value in medical education. The family doctor is as old as medicine itself, and a study of the varying standards of his work, and of the conditions in which he has carried it out, has much to teach us. The very status of the doctor has changed with the centuries. When the physician was more than somewhat of a magician, he could be regarded as semi-divine. The practical Romans of the time of Julius Caesar, according to Suetonius, gave all their physicians the great benefits of Roman citizenship. Almost within living memory, the doctor's frock-coat and goldheaded cane were symbols not merely of affluence but of the respect in which he was held in the repute of his fellowmen. Ups and downs are man's natural lot, but it is a chastening thought that the recent trend of change in the status of the medical profession may have been gradually and subtly for the worse! It is supremely ironical that today, when the doctor can be something more than a pompous and ignorant humbug, when he has at his command an impressive diagnostic and therapeutic armamentarium, respect for the doctor may have lessened. Why should this be? Is it just a matter of familiarity breeding contempt by way of a certain loss of mystique, resulting from the modern diffusion of medical knowledge among laymen? If it were so we would count it no very great loss. But can we be sure that there is not some much more serious reason, such as a decay and decline in the personal human relationship between the doctor and his patients? And will not this decline reach its nadir when the doctor can no longer say "Everyman, I will go with thee and be thy guide, in thy most need to go by thy side", because he has become just another State functionary with an allegiance other than that of the interests of his patients as his supreme consideration?

It is in the mutual Love of Doctor and Patient that the medical relationship can find and keep its soul. In the Epistle to the Colossians, St. Paul crowned St. Luke with the beautiful title of THE BELOVED PHYSICIAN. Would that we could all attain to its deserving.

## A CASE OF BERI-BERI HEART DISEASE

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A thirty year old male was referred for progressive heart failure of unknown etiology. His personal history was as follows: At 15 he had joined the Royal Navy, playing Rugby football and taking part in athletics. There was not, therefore, at that time anything to suggest either congenital or rheumatic cardiac lesions. Nine years ago, he passed his "medical", and joined the Fleet Air Arm. Three years later he began to complain of palpitations which were attributed to emotional problems, because exhaustive investigations in a naval hospital had failed to show any abnormality. Since one year he has found himself progressively getting cut of breath on exertion — a symptom which he attributed to heavy smoking (80 cigarettes per day). Since two months his exertional dyspnoea had become worse and he noticed swelling of the ankles. No history of chest pain could be obtained. After close questioning he admitted that for the last eight years he had been a heavy drinker of whisky (up to one bottle a day) and of beer (up to 20 bottles daily).

Physical examination showed a thin man of medium height, who got easily out of breath while undressing. The usual signs and symptoms of congestive heart failure were present, that is, exertional dyspnoea, mild cyanosis, anorexia, raised jugular venous pressure, basal rales, hepatomegaly. There was pitting oedema of the lower limbs. Palpation revealed a biventricular thrust. There was a regular tachycardia of 130/m and grade 2 systolic (ejection type) murmurs over the apex and base of the heart. The blood pressure was 140/0. Urinalysis showed a trace of albumen but no sugar. The E.C.G. showed low-voltage of the QRS complex and flat or slightly inverted T-waves. The X-rays confirmed the clinical findings of an enlarged heart.

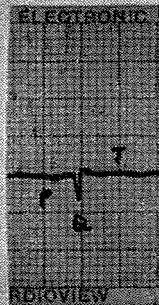
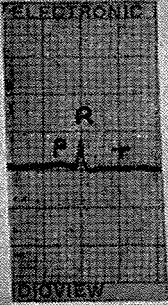
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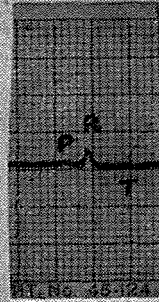
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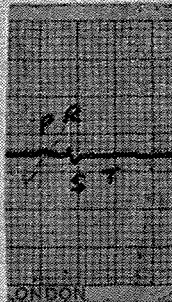
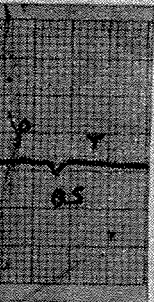
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Lead 3

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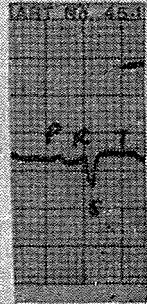
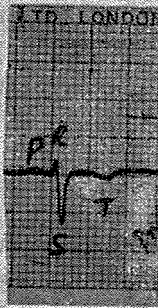
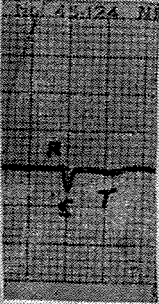


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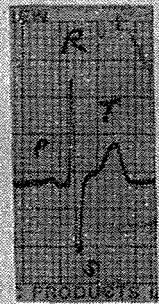
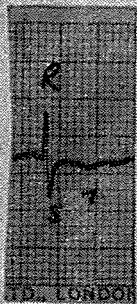
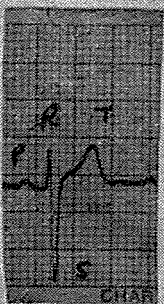
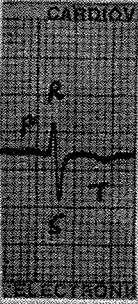
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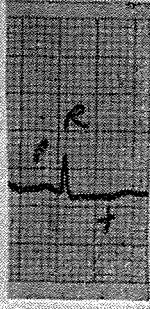
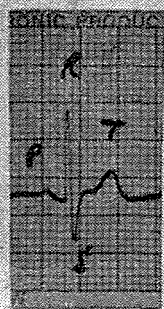
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case of congestive heart failure with a blood pressure of 140/0 and an abnormal electrocardiogram showing no specific pattern. Since aortic incompetence was not present the diagnosis was one of high-output failure with a collapsing pulse which was not due to an aortic lesion. Clinical and laboratory findings ruled out hyperthyroidism and anaemia (radio-iodine uptake and metabolic rate were normal, there were 4,800,000 r.b.cs. per ml and 92% Hgb) and we were therefore left with the following possibilities of high-output failure:

1. A—V fistula; 2. Severe liver failure; 3. Beri-beri heart disease. At this stage the following investigations were carried out: skeletal X-ray survey, radio-iodine uptake, metabolic rate estimation, liver function tests, electrophoretic pattern study, serum cholesterol, serum electrolytes, serum calcium and phosphorous estimation, alkaline phosphatase, transaminase and haemochromatosis tests all being normal. Wassermann test was negative. Pyruvic acid estimation showed an abnormal rise after glucose: 25 mgm, normal being 1 mgm. Blood urea was 10 mgm. per 100 ml.

A diagnosis of beri-beri heart disease was made and the patient was treated accordingly. He was put on vitamin B<sub>1</sub> 100 mgm by intramuscular injection three times daily for one week, followed by 150 mgm. orally daily. He was also given Digoxin 0.25 mgm. twice daily and 100 mgm. hydrochlorticoside weekly.

After one month's treatment the patient felt much better, was able to return to work, did not get out of breath, the murmurs disappeared and his blood pressure was 130/85. His blood urea had risen to 30 mgm. per 100 ml. The electrocardiogram now showed a normal T-wave and higher voltage of the QRS complex.

Mechanism of heart failure in Vit. B<sub>1</sub> deficiency: Since B<sub>1</sub> is the co-enzyme of carboxylase and is required for normal carbohydrate metabolism and utilisation, its absence renders the heart muscle unable to utilise lactate pyruvate normally. This leads to diminution of myocardial O<sub>2</sub> extraction, insufficient energy production

and functional failure. In addition there is an accumulation of the vasodilating intermediate catabolites of glucose (pyruvic, lactic and other keto-acids) which cause widespread peripheral arteriolar dilatation. This acts like a large arterio-venous fistula augmenting the venous return to the right ventricle and making demands upon the left ventricle for increased output, thus putting a burden on a heart already working at a disadvantage (Konstam G. & Sinclair H. M. 1940). Cardiac catheterisation in a patient with beri-beri heart shows an average cardiac output of 16 litres per minute (normal 4—7 litres) and an O<sub>2</sub> consumption of 355 ml/m (normal average 760 ml/m). Alcoholics who take more of their calories in the form of alcohol (which requires B<sub>1</sub> for its metabolism) and do not eat enough B<sub>1</sub> containing foods, eventually develop heart failure through the mechanism described above.

I would like to thank Dr. R. I. S. Bayliss M.D., F.R.C.P., Physician in the Westminster Hospital, London, for the biochemical tests carried out in the investigation of this case.

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## STAPEDECTOMY WITH A SPRING PROSTHESIS

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In deafness due to otosclerosis the footplate of the stapes becomes ankylosed to the niche of the oval window in such a way that the stapedovestibular joint progressively disappears.

The treatment for this form of deafness is surgical. The stapes is removed and the gap between the incus and the oval window is bridged by means of a prosthesis. The operation is called a stapedectomy and the prosthesis may be either a polythelene tube or Teflon. The oval window is usually closed by means of a vein graft, but Prof. H. Schuknecht uses as his prosthesis a plug of fat taken from the lobe of the ear (which occludes the oval window) and stainless steel wire which is attached to the incus. Another



method that is nowadays used is the Teflon rod or the McGee piston, both of which are attached to the incus and go down into the labyrinth through a hole bored in the stapes footplate.

Results from these various techniques are usually good, but they all carry a risk of sensory-neural changes occurring in the labyrinth which come on suddenly days, weeks or months after a successful operation. The patient hears a rushing noise in the ear and his hearing deteriorates rapidly until a "dead" labyrinth develops. Vertigo may or may not be complained of.

This problem has worried otologists since stapedectomy came into general use and no specific reason can be adduced to explain it.

However two important factors have to be taken into consideration because they may be responsible for this tragedy.

1. Before removing the stapes, the stapedius muscle, which is attached by its tendon to the neck of the stapes, is cut. It is known that one of the roles of the muscles of the middle ear is to protect the internal ear from excessive stimulation. Whenever the ear is subjected to a loud noise, the tympanic muscles contract and the strength of the stimulus reaching the labyrinth is reduced.

In stapedectomy with prosthesis, this damping effect of the muscle is lost and the artificial stapes is free to "pump" the labyrinth strongly or weakly according to the strength of the stimulus. Excessive "pumping" action by a prosthesis may be one of the factors which produce sensory-neural changes in the labyrinth.

2. Another consideration is a leak of perilymph which occurs after all forms of stapedectomy techniques. This has been proved by injecting radio-opaque substances immediately after a stapedectomy into the spinal column and taking serial X-rays of the middle ear. The radio-opaque substance was almost invariably shown in the middle ear. This was found to be most abundant when a Teflon rod or a McGee piston was used, and least when the oval window was closed by means of a vein graft (J. Shea, personal communication).

In an endeavour to minimise the

above factors, I developed in March 1965 a stapedectomy technique which has so far proved very satisfactory.

After removing the stapes, the oval window is closed by means of a vein graft taken from the dorsum of the foot. The gap between incus and vein is bridged by means of a spring made of stainless steel wire which could, in theory, attenuate a loud stimulus.

This spring prosthesis has now been used in over 40 cases in Malta and, except for one case which showed no improvement, all the others show marked improvement, normal or above-normal hearing being the rule. No "dead" labyrinths have so far occurred.

The spring prosthesis is also being used in the United Kingdom and in the United States of America, and, in fact, it is being produced commercially by a well known firm of surgical manufacturers.

Reports from various centres show that the spring prosthesis does all that is claimed for it, and what is more important, is that to date no sensory-neural changes in the labyrinth have been observed following its use.

### Summary

The causes of sensory-neural changes in the labyrinth following stapedectomy operations are discussed. A new technique is described and its advantages outlined.

## OCULAR TOXOPLASMOSIS: A report on a case discovered in Malta

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Before the discovery of toxoplasmic retino-choroiditis, all cases of fundus diseases in infants and young children were diagnosed as foetal chorioretinal infection of unknown origin, or congenital developmental anomaly of the retina and choroid

or chorioretinal birth injury.

Thanks to clinical and laboratory examinations, a good number of these cases can now be diagnosed as due to infection by toxoplasma. No case of ocular toxoplasmosis has ever been reported in Malta. The aim of this paper is to report such a one.

Toxoplasma is a protozoan parasite possessing a cytoplasm and a distinct nuclear chromatin. Its size may vary from 4 to 7 microns in length and 2 to 4 microns in width. These organisms may be present either as free parasites in the tissues or within fixed tissue cells, mononuclear phagocytes, eosinophiles and polymorphonuclear leucocytes. It is an intracellular parasite, reproducing itself in all tissue cells. Multiplication is rapid within cells, finally resulting in rupture of the cell wall. The infection may remain latent for a long period. It is poorly resistant to drying and freezing.

Toxoplasma is pathogenic in a large number of mammals and birds. Propagation can happen by the transfer of infected tissues and fluids by a variety of routes: by mouth or by conjunctival, intramuscular or intracerebral inoculation.

It is not easy to explain the mode of infection of ocular congenital Toxoplasmosis. It is thought that the protozoon reaches the ocular globe not only through the blood, but rather through neurotropism and direct propagation from the brain towards the eye via the cerebro-spinal fluid and the meningeal coverings of the optic nerve. A milk-borne infection cannot be excluded.

No evident sign of toxoplasmic infection is present in the pregnant mother because of a natural or acquired resistance to the infection.

### **Clinical manifestations**

Infection may occur at any period of life and may or may not give rise to symptoms. The clinical findings depend upon the age at which infection happens and upon the dosage of toxoplasma received.

When the infection occurs in foetal or early infantile life, there is always the

presence of signs and symptoms and the results are widespread. In the congenital form, the infection acquired from the mother is usually at an advanced stage when the child is born. On the other hand, the infant may be born with the early signs of Toxoplasmosis when infection happens in late uterine life.

The most important ocular signs are chorioretinitis, papilloedema and optic atrophy as a result of papilloedema and chorioretinitis. Other ocular signs are microphthalmia, iridocyclitis and cataract. Among the general symptoms are to be mentioned: convulsions, hydrocephalus, cerebral calcification and microcephalus. Calcifications are the result of the precipitation of calcium in the periventricular region and in other parts of the brain, following upon encephalitis with destruction of brain tissue. Hydrocephalus is caused by the inflammation followed by obstruction of the foramen of Monroe and the region around the, aqueduct of Sylvius.

Toxoplasma has an elective tropism for the nervous tissue, brain and retina which no doubt offer favourable conditions for its proliferation. Poor diffusion of serum antibody into the brain substance and retina have also been suggested as an explanation. It has a particular virulence for embryonic tissues, as shown by relative frequency and particular seriousness of the infection in the foetus and in the infant.

The retina is the tissue initially attacked. The infection extends secondarily to the choroid. The choroid may be the first tissue to be attacked. In the majority of cases, the macular area is the site of election of the choroidoretinitis. It is usually bilateral and symmetrical. In one third of the cases, there is only one focus. It is present on one side only in 1 out of every 5 cases. It is generally agreed that choroidoretinitis in toxoplasmosis is typical and characteristic. In healed choroidoretinitis, the focus is usually large and irregular. There is a marked central atrophic area, with a pigmented border. Clumps of pigment may cover the atrophic central area.

## THE GRANDE MULTIPARA

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If the infection occurs at an early stage of foetal life, pronounced changes occur in the eyes and microphthalmos may result.

A presumptive diagnosis of congenital toxoplasmosis can be made if the characteristic findings of cerebral calcifications, chorioretinitis and hydrocephalus are found. The diagnosis is confirmed, if the serological findings are sufficiently positive.

### Case report

On the 21st January 1966, a girl aged 11 years was referred to the ophthalmic out-patients department for defective vision.

The patient had a history of squint when very young. Vision R.E. unaided 6/18. Vision L.E. unaided 6/60. Examination of the fundi after pupillary dilatation by homatropine showed no abnormal changes in the right eye.

The left fundus showed a square shaped cicatricial chorioretinal focus, situated on the temporal part of the posterior pole about 4 disc diameters from the optic papilla and measuring  $4 \times 4$  D.D. The surface of the focus was covered by small clumps of pigment, surrounding a central atrophic area. Some sinuous choroidal vessels were visible at the bottom of the focus. The retinal vessels were normal. The temporal half of the optic disc showed a marked pallor.

Vision in the right eye could be improved to 6/9 with glasses. The vision of the left eye could not be improved.

Radiological examination of the cranium did not reveal any calcifications.

Physical examination did not show any abnormal changes. Two tests for cytoplasm modifying antibodies (the Sabin and Feldman test) carried out a fifteen days' interval were positive at 1/16 which suggested a past infection or a chronic infection of long duration.

Our thanks are due to Dr. Alastair Dudgeon of the Hospital for Sick Children, London, for carrying out the C.M.A. tests for us.

The problem of the grande multipara has naturally always been with us. It wasn't, however, until 1934 that our attention was first focussed on it when Bethel Solomons termed the patient who had borne five or more children as "the dangerous multipara". Solomons had found that the maternal mortality rate increased progressively from the fifth to the tenth parity. A para 10, for example, had five times the likelihood of a para 5 of her pregnancy ending fatally. Eastman (1940) and Greenhill (1951) agreed with this view and brought up evidence of their own to show that there was an increased maternal mortality attached to great multiparity. From a study of his cases, Eastman deduced that para 9 had three times the chances of a fatal outcome than the woman who was para 5 or less. The result was that the multipara was considered such a bad risk that often sterilisation was resorted to prophylactically on the basis of multiparity alone.

Since those days, the picture has changed considerably. Firstly, the average family of to-day is unlikely to reach the size that was commonplace previously. The reason for this is mainly economical rather than medical, though, paradoxically, it seems to be that women in the lower income groups are the ones who still beget the larger families. Secondly, great multiparity no longer offers the same grave possibilities from the obstetrical standpoint that obtained in the past. Eastman (1955) was one of the first to recognise this. More recent reports reflect the same opinion. Scharman and Silverstein (1962) write that "the old-fashioned designation, the dangerous multipara, should unalterably be stopped"; while Israel and Blazar (1965), after an exhaustive study conclude that "the present evidence is clear-cut that she (the grande multipara) is nowadays cared for with no greater

risk of life than that of any other pregnant woman."

### Material and Findings

In this survey, a study was made of all the para 7 or over delivered at St. Luke's Hospital, Malta, during the two-year period 1963-1964. A comparison was also made between the results obtained in all para 5 patients and those who were para 10 or over during the same period. The figure for the para 7 or over was 638 out of a total of 4052 admissions, constituting 15.7 per cent. This high figure indicates the class of patients admitted, mostly from the lower social strata. A large proportion of these patients converge to the one hospital from the villages. Though they all had their delivery in hospital, antenatal attendances had often been erratic.

**TABLE I**  
Distribution according to parity of 638 grande multipara

Parity	%
7	22.6
8	20.5
9	15.4
10 or over	41.5

The highest parity recorded in this group was 22. The patient was aged 43 and had a normal pregnancy and delivery.

**TABLE II**  
Distribution according to age

Age	%
— - 29	4.3
30 - 34	20.7
39 - 39	47.8
40 - 44	26.0
45 or over	1.2

The youngest patient was 24 years old and was para 7. There were three patients aged 46, the oldest in the series.

Toxaemia. Under this heading are included all patients whose blood pressure was found to be above 140/90 on more than one occasion during the pregnancy. There were 142 cases, forming 22.9 per cent. This is a high rate and is possibly related to the frequent occurrence of gross

obesity in the series, which together with glycosuria forms almost a picture of endemic disease in this class of patient in Malta. Another factor for the high figure has been the difficulty in following up and adequately treating hypertensive patients who refuse in-patient treatment.

There were 13 cases of abruptio placentae i.e. 2.03 per cent. and 10 cases of placenta praevia i.e. 1.56 per cent. There were 6 cases of retained placenta or 0.95 per cent.

Operative intervention. Caesarean section was carried out in 43 cases or 6.7 per cent. of the series. Hysterectomy was performed in 5 cases, three times for the three cases of rupture of the uterus in the series and twice for intractable bleeding during Caesarean section on friable uteri. The three cases of uterine rupture occurred in women who were para 7, 8 and 9 respectively.

There was one maternal death or 0.15 per cent. This was para 11, aged 38, who had a Caesarean section for fulminating toxæmia and died five days later from pulmonary oedema and heart failure. (Table III).

Prematurity — here taken as being 5 lbs. 8 ozs. or less at birth — accounted for 5.8 per cent. or 37 cases. There were 11 sets of twins or 1.8 per cent. The rate for still-births was 5.0 per cent. or 32 cases — an understandably high figure when considering the incidence of toxæmia.

Malpresentations. There were 16 cases involving a transverse presentation or prolapsed cord or both, an incidence of 2.5 per cent. 12 of these required a Caesarean section, 1 had a hysterectomy for rupture of the uterus and three had a normal delivery. The rate of breech delivery was 6.2 per cent. i.e. 40 cases. There were 2 cases of face presentation, one of which, a persistent mento-posterior, necessitated a Caesarean section.

A comparison is made below between the obstetrical behaviour of the para 5 group and that of the patients who were para 10 or over. There was no appreciable difference in the incidence of placenta praevia and premature births. Toxaemia



TABLE III

Showing incidence of obstetrical complications in women who were para 7 or over according to various authors.

	<i>Toxæmia</i>	<i>Abruptio placente</i>	<i>Placenta prævia</i>	<i>Retained placenta</i>	<i>Prematurity</i>	<i>Still-births</i>	<i>Maternal mortality</i>
Miller (1954) 563 cases	4.1	3.1	1.0	1.6	8.8	1.9	0.17
Schram (1954) 502 cases	11.1	2.39	1.6	2.9	5.9	3.1	0.4
Scharfman et al. (1962) 403 cases	9.6	0.74	1.5	0.99	9.6	1.0	0.2
Present series 638 cases	22.2	2.03	1.56	0.95	5.8	5.0	0.15

was more frequent in the higher parity group. Abruptio placentae and Caesarean section were approximately four times as common, while malpresentation and still-birth rates were twice as high.

**Discussion**

The grande multipara is or should be at no greater risk to her life than the one who has borne less children. The improved results are directly related to improved ante-natal care with early recognition and treatment of abnormalities, better nutrition and hygiene, the introduction of blood replacement therapy and antibiotics and the increased safety and more frequent use of Caesarean section. The result is that the atmosphere is now rightly one of optimism.

The findings of the present series are in consonance with the above statement.

This is not to claim that the grande multipara is the equal physically of the woman of lower parity. The title "grande" is often synonymous with "elderly" and thus any medical disease that might be present, such as hypertension and diabetes, is bound to be more advanced. Repeated pregnancies take their toll in the form of obesity, lordosis, varicose veins and the fatigue associated with caring for a large family. Moreover, she is often in economic straits and her nutrition is correspondingly poor.

The delivery of the grande multipara should on no account be undertaken in the home. The most favourable domiciliary conditions can never provide sufficient safeguard against the complications that are more likely to arise in this particular type of patient. The increased incidence of malpresentations and abruptio placentae and the possibility of uterine rupture

TABLE IV

Showing incidence of complications according to parity.

	<i>Toxæmia</i>	<i>Abruptio placente</i>	<i>Placenta prævia</i>	<i>Retained placenta</i>	<i>Prematurity</i>	<i>Still-births</i>	<i>Breech</i>	<i>Uter. rupture</i>	<i>Cæsarean sect.</i>	<i>Maternal mortality</i>
Para 5 259 cases	16.9	0.39	0.39	0.39	5.79	2.7	3.47	0	1.54	0
Para 10 or over 265 cases	23.4	1.5	0.37	1.1	6.04	5.66	6.8	0	6.41	1 case.

make it imperative for all facilities for operative intervention and blood transfusion to be near at hand. Only in this way can the maternal risk be held in check.

### Summary

A series of 638 grande multiparae is studied. The findings, and those of other authors, show a higher rate than average for certain obstetrical abnormalities such as toxæmia, abruptio placentae, still-birth and malpresentations. The maternal mortality is not appreciably affected.

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### PUBLICATIONS LIST

The following is an incomplete list of scientific publications since 1961 by graduates of our medical and dental schools; we would be glad to add to it in our next issue.

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## MEDICAL NEWS

We congratulate Dr. Herbert Lenicker and Dr. Carol Jaccarini for obtaining the D.C.H. of the Conjoint Board, and Dr. Alexander Warrington for his F.R.C.S. Also Lt. Col. (D) Anthony Cremona on his recent promotion from Major.

On the initiative of the local agent, that well known benefactor of medical education, Mr. Charles Degiorgio, Vitamins (Export) Ltd. have established a scholarship of £50 per annum, which can be renewed up to a maximum period of six years. It is open to candidates who intend to register for the preliminary Course of Science with a view to joining the

Course of Medicine and Surgery at the Royal University of Malta.

Dr. Paul Cassar, who is president of the B.M.A. (Malta Branch) for 1966, on the 10th March read a paper on "The Maltese Sanitary Organisation in 1743" to B.M.A. members and their guests.

Professor Andrew Semple of Liverpool University, a British Council Specialist Visitor, lectured here on the 12th April, on "Rehabilitation after serious illness", on April 15 on "Food and Tourism" and on April 18th on "Health and Welfare problems of emigration".

Mr. Henry Shaw of the Royal Marsden and Royal National E.N.T. Hospital, London, Lectured, recently, on "Modern Treatment of Cancer of the head and neck".

Dr. E. M. M. Bestermann of St. Mary's Hospital, London, lectured on 1st April on "The present status of cardiac surgery and the selection of patients".

Mr. Stuart Southwood, under the auspices of the Dental Association of Malta, lectured on the 13th April on "Fundamentals and complexities of Crown and Bridgework".

Professor W. Blackwood of the Institute of Neurology, National Hospital, Queen's Square, University of London, lectured on the 23rd May on "Vascular Diseases" and on the 24th May on "Cerebral Tumours".

Sir Clement Price Thomas, President of the British Medical Association, gave a lecture, under the auspices of the Malta Branch, on "Cancer of the Lung" on the 7th June.

## OUR COLLEAGUES ABROAD

In an optimistic moment we thought it would be a good idea to list where our colleagues abroad have got to and what they are doing, but so many have gone so far that we can only present a random selection to which we will be glad to add in our next issue, especially if information is provided by friends and relatives.

June Naudi, of the 1960 vintage, is now the wife of Lt. Commander D. Gunn R.N. and she turns up, surprising as ever, in Moscow where her husband is Naval

Attacheè at the British Embassy. Amongst the seniors, Tom Anastasi Pace, O.B.E. ('37) now a Colonel is A.D.M.S. Singapore; Tom Warrington ('37), also a Colonel, is Assistant Director of Pathology with the British Army of the Rhine, at Munster in Germany. Of the two sons of Professor J. E. Debono, Edward ('60) is at present in the Bacteriology and Immunology Department at Harvard Medical School, being on loan from the Department of Investigative Medicine at Cambridge, England; his brother Anthony ('60) is assistant lecturer on surgery at the postgraduate medical school at Hammersmith, where John Azzopardi ('49) is one of the shining lights of the pathology department. Vincent Briffa ('49) is also there, an E.N.T. registrar in the department of surgery, whilst his brother Joseph, who married Dr. Elizabeth Innes and has 5 children, is a consultant anaesthetist at the Kent and Sussex Hospital and lives, lucky man, in Tunbridge Wells.

We congratulate Joseph Attard ('55) who obtained the Edinburgh F.R.C.S. last January, and, after being a few years in Coventry (strictly in the literal sense of the words) has now moved with his family to Worcester where he is a Senior Registrar at the Royal Infirmary. John Casaletto ('59) is now a Surgeon at Whipps Cross Hospital at Leytonstone in Essex. Donald Sultana, a medical truant who has long forsaken medicine for literature, is now lecturing the Scots on English at Edinburgh University. This could only be beaten, if we get a Scotsman to lecture us on Maltese; although unlikely, this is not beyond the bounds of possibility. The present lecturer in Arabic at our University (and he seems excellent at his work) is an Englishman.

Canada has had an understandable attraction for our colleagues. One of the senior inhabitants is Ivan Dedomenico ('52), who married an Irish-Canadian lady called Doreen Tambling and has two children. He has the Canadian fellowship of the College of Surgeons in Urology and is also a fellow of the American Academy of Pediatrics. He works, *inter alia*, in the General Children's Hospital and at the

Cambrini Hospital at Montreal. Also in Montreal at the world-famous Neurological Institute is Albert Pace Florida ('58), a Fellow of the Canadian College of Physicians, who has recently acquired the Diploma in Anaesthesia of McGill University. He has married a Hungarian lady, who left her country after the 1956 rising; they have one son. Frank Vella ('52) last year acquired not only the important Fellowship of the Royal Institute of Chemistry and the membership of the College of Pathology, but also a Knighthood in the Order of St. Sylvester. Since this last was whilst he was in Mohamedan Sudan, it probably means much more than such things usually do. After Singapore and Khartoum, Frank is now happily settled with his family in Saskatchewan as an associate professor of Biochemistry. As can be seen elsewhere in this issue, he is a prodigious worker. Alexander Saliba ('52), now a fellow of the Canadian College of Physicians, married with 4 children, is physician superintendent at the Hazelwood Sanatorium, Louisville, Kentucky, U.S.A.

Lino German ('55) is a gynaecologist with the R.A.F. in Singapore (I can almost visualise Wilde's Lady Bracknell wondering whatever the R.A.F. could possibly have to do with a gynaecologist). Also with the R.A.F., but in Yorkshire, is Victor Debono ('59). Paul Vassallo Agius, who got the D.C.H. in 1965, is to be congratulated for acquiring the M.R.C.P. last January. He is at the Queen Elizabeth Hospital for Children at Paddington in London. Carol Jaccarini ('61) is at the Jessop Hospital for Women in Sheffield. Ramon Consiglio ('55) is Consultant Anaesthetist at Preston Royal Infirmary in Lancashire. Roger Parnis, M.B.E. ('46) is Senior Surgical Specialist at the Ade-Oyo Hospital, Ibadan, Nigeria, and part-time lecturer in surgery at the University.

So the list could go on, almost endlessly. "Far and far our homes are set, round the Seven Seas". If the old Alma Mater rubs her eyes and looks afar, she will definitely find something to surprise her in the achievements of her medical faculty boys and girls.



## NOTICE

This gazette will be published biannually in June and in December. Contributions for our next issue should reach the editor at the Bacteriological Laboratory, St. Luke's Hospital, by the 15th November. Reprints of individual articles will be available to contributors by arrangement.

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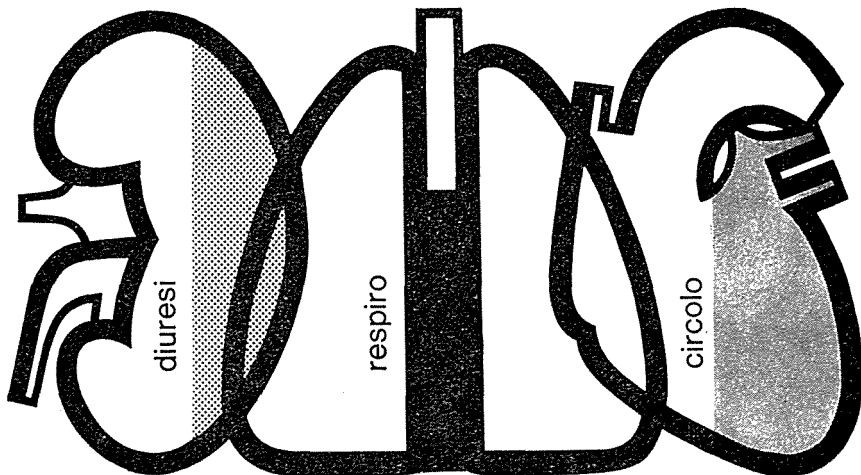
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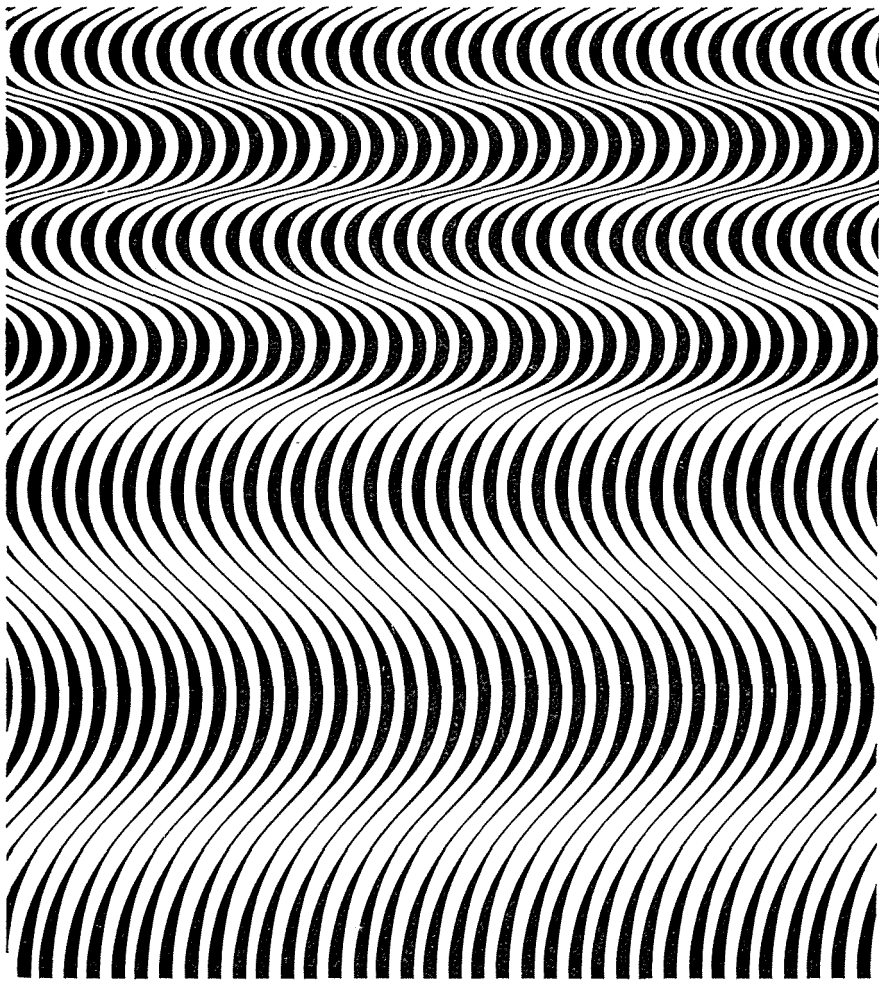
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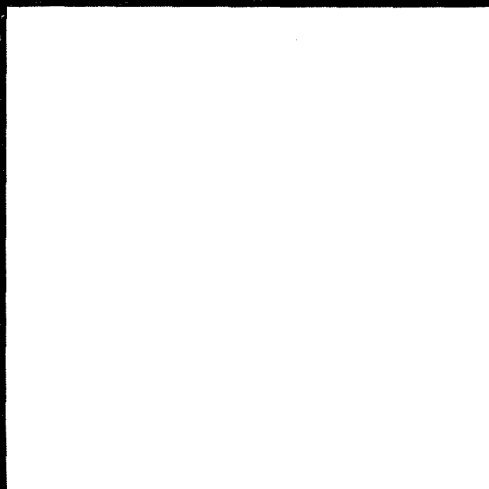
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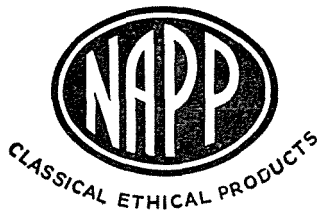
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