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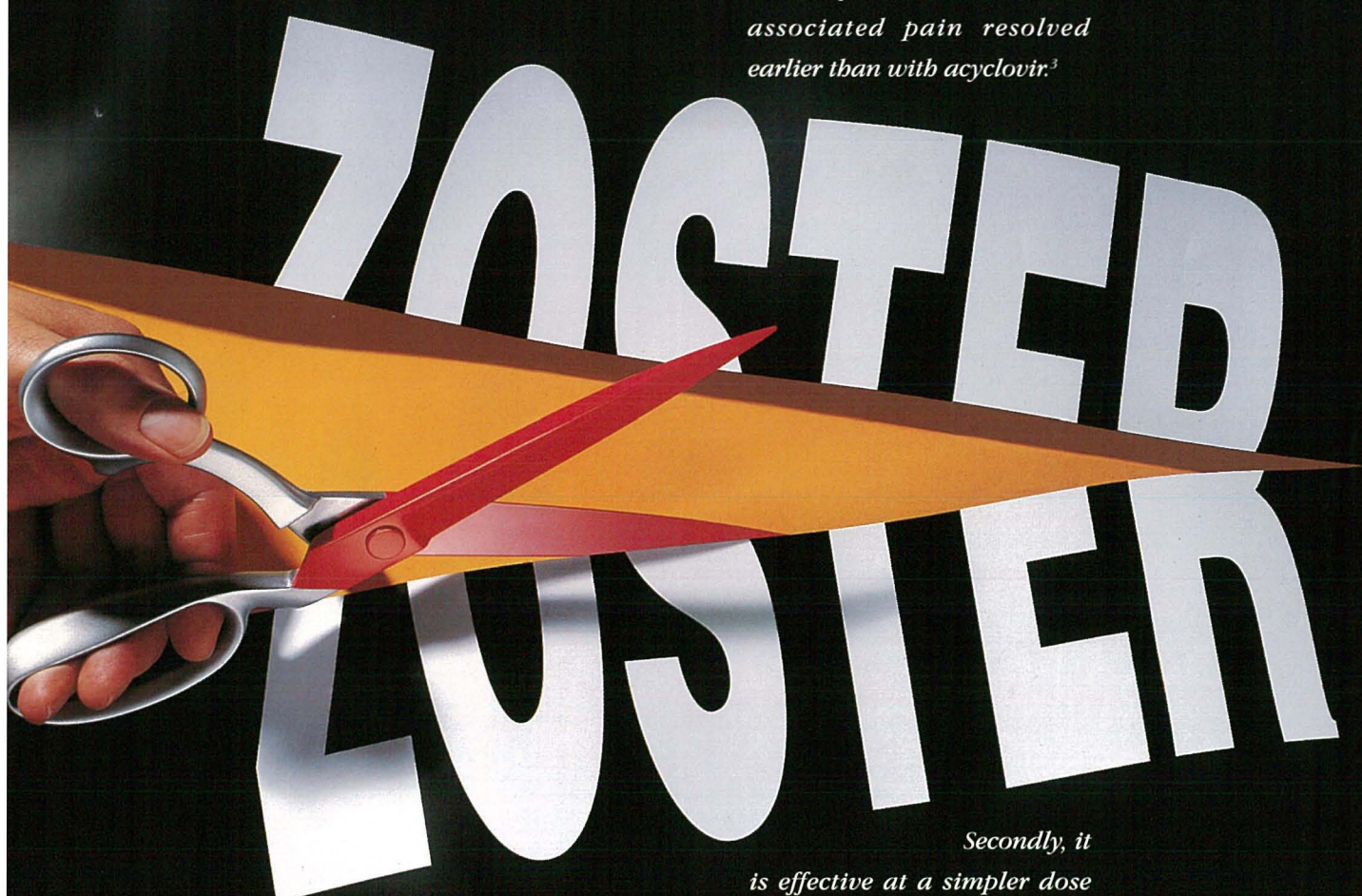
it-tabib tal-familja

an advanced antiviral

AT THE CUTTING EDGE OF ZOSTER THERAPY

Famvir (famciclovir) is a new antiviral from SmithKline Beecham. It offers the same highly-specific[1,2] action against varicella zoster virus as acyclovir – yet with two new significant advantages. Firstly, in efficacy-evaluable patients treated within 48 hours of rash onset, zoster-associated pain resolved earlier than with acyclovir.[3]



Secondly, it is effective at a simpler dose than the cumbersome 5xdaily regimen of acyclovir.

power to cut pain duration, with a simple t.i.d dose

SB
SmithKline Beecham
Pharmaceuticals

Abbreviated Prescribing Information

Presentation 'Famvir Tiltab' Tablets each containing 250 mg famciclovir. **Uses** Famvir (famciclovir) is indicated for the treatment of acute Herpes zoster (shingles) infections. **Mode of action** Famciclovir is the oral form of penciclovir. Famciclovir is rapidly converted *in vivo* into penciclovir, which has *in-vivo* and *in-vitro* activity against human herpes viruses including Varicella zoster virus. **Dosage and administration** *Adults:* One 250 mg tablet three times daily for seven days. Treatment should be initiated as early as possible in the course of the disease, promptly after diagnosis. *Elderly:* Dosage modification is not required unless renal function is impaired. *Renally impaired:* As reduced clearance of penciclovir is related to reduced function, as measured by creatinine clearance, special attention should be given to dosages in patients with impaired renal function. The following modifications in dosage are recommended.

Creatinine clearance	*Dosage*
30-59 (ml/min)	250 mg twice daily
<30 (ml/min)	250 mg once daily

No information is available in patients undergoing dialysis. *Hepatically impaired:* Dosage modification is not required. *Children:* There are currently insufficient data on the safety and efficacy of Famvir in children.

SB

Contra-indication Known hypersensi... **Precautions** Special attention should be paid to patients with impaired renal function as dosage adjustment may be necessary [See **Dosage and administration**]. No special precautions are required for hepatically impaired or elderly patients. **Drug interactions** No clinically significant pharmacokinetic interactions have been identified. Probenecid and other drugs that affect renal physiology could affect plasma levels of penciclovir. **Use in pregnancy and lactation** Although animal studies have not shown any embryotoxic or teratogenic effects with famciclovir or penciclovir, the safety of Famvir in human pregnancy has not been established. Famvir should, therefore, not be used during pregnancy or in nursing mothers unless the potential benefits of treatment outweigh any possible risk. Studies in rats show that penciclovir is excreted in the breast milk of lactating females given oral famciclovir. There is no information on excretion in human mi... **Adverse reactions** Famciclovir has been well tolerated in human studie... Headache and nausea have been reported in clinical trials. These were generally mild or moderate in nature and occurred at a similar incidence in patients receiving placebo treatment. **Overdosage** No acute overdosage with Famvir has been reported. Symptomatic and supportive therapy should be given as appropriate. No data are available on the efficacy of haemodialysis in removing penciclovir from plasma.

FAMVIR™
3 x day
famciclovir

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Editorial

Dear Readers,

It is with great pleasure that I welcome you to the first issue published in 1999.

The new year has, as always, brought with it new opportunities and new challenges to Family Medicine in Malta. At the 4th Medical School Conference this March, a session has been dedicated to Family Medicine for the first time, and we have had the pleasure to invite Prof. Henk Lamberts from the University of Amsterdam to deliver a key note speech during the meeting. Soon after this event, the College will be organising an EGPRW/WHO Research Methods Course in Primary Care in June, and presently Council is also preparing for the much larger 6th Mediterranean Medical Society meeting to be organised in Malta in September, 2000. The Collage will also soon be launching a modified version of TRANSHIS for Maltese Family Doctors. This software is a patient database based on the International Classification of Primary Care and developed in the Netherlands for Dutch GPs, but now in use in various countries all over the world.

The greatets challenge, however, must be the setting up of the Department of General Practice in the University of Malta. I quote from a recent Collage press release: "After repeated Collage proposals over the years to the University of Malta regarding the dire need of an academic unit for Family Medicine, Dr. Denis Soler announced that Prof. Mark Brincat, the Dean of the Faculty of Medicine and Surgery, had recently proposed the setting-up of a "long-overdue" new Department of Family Medicine. Such department was envisaged to organise an undergraduate programme, and also concentrate on a long-awaited postgraduate vocational training scheme. Dr. Soler had congratulated Prof. Brincat on his bold deeecision, and accepted his invitation to chair, as Collage president, an Ad-hoc Advisory Committe on Family Medicine to counsel the Dean regarding the establishment of such Department of Family Medicine".

The road ahead is long and tortuous, but I believe we have climbed over the crest of the hill and can finally see the green valley ahead.

Jean Karl Soler

Cover Photo taken by J.K. Soler:
Santo Spirito Hospital.

Santo Spirito Hospital was already in existence by 1347. It not only cared for the sick but by 1615 it also received unwanted babies who were deposites in a revolving cradle inside the hospital through a small window in the facade of the edifice. This contrivance was known as the ruota.

During the French occupation of Malta (1798-1800) SSH served as the main hospital for the sick of the Maltese living in the countryside.

Towards the end of the nineteenth century it was turned as an annexe to the government general hospital at Floriana. It was closed down in 1967. Until then it was one of the oldest functioning hospitals of Europe.

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The Effect of Environmental Hazards on The Health of The Young

Dr. A. Muscat Baron
Department of Paediatrics, St. Luke's Hospital
Dr. Y. Muscat Baron
Department of Obstetrics and Gynaecology, St. Luke's Hospital

'If you go to American city,
You may find it very pretty,
Just one thing that you must beware,
Don't drink the water and don't breathe the air'
T. Lehrer in 'Pollution'[1]

The environment refers to the medium in which we exist. Our health depends on the ongoing events taking place in our surroundings. The environment can have direct or indirect hazardous effects on our health. Several noxious effects can reach us through ingestion, inhalation, and through contact with our skin. These hazards may also affect the unborn child, evading the protective and nurturing body of its mother.

The environment is relatively more hazardous to infants and children because they are structurally and functionally different from adults. Children have a larger surface area to body weight ratio. Their higher metabolic rate requires more oxygen intake. They grow at a faster rate, especially during the first six months of their life. Some body organs are functionally immature at birth and organ maturity progresses at different rates. Children also need more energy and fluids per unit body weight compared to adults. Thus, the greater requirement of fluids, food and air makes them more vulnerable to environmental hazards.[2,3]

Children require access to clean water, clean air and protection from polluting toxic substances in order to sustain normal growth and development.[1] The environment can be instrumental in assisting the development of a child but also hazardous in many ways. Some of these noxious effects of the environment, their effects on the foetus during pregnancy and on infants and children will be described below.

Poverty is a major culprit behind many problems that will be described. Poor **nutrition** during pregnancy is harmful to the unborn child since its growth depends entirely on the maternal food supply. Lack of proper nutrition may result in intrauterine growth retardation. The proportion of low birthweight babies (usually taken as less than the 10th centile for gestational age) reflects the health and nutritional status of the mother.[2] Poverty is also the cause of widespread malnutrition affecting children across the world. Malnutrition makes children more prone to infection. Infection in turn, further exacerbates malnutrition resulting in a vicious cycle. Both malnutrition and infection are attenuated by breastfeeding. However, breast-feeding is not completely risk-free. Most fat-soluble chemicals ingested by the mother can be transferred to the child via breast-milk.

Obesity on the other hand, is the most common nutritional problem of children in many parts of the developed world, especially the United States, and is caused mainly by inappropriate environmental habits and factors. The incidence of childhood obesity in the U.S. increases especially among children of elementary and high school age. This problem is also markedly on the increase in the UK[4] and in other Western countries, where about 10-15% of preschool children are considered to be overweight.[5] Some of the factors thought to increase the risk of childhood obesity are environmental factors indirectly exerting their effect on children such as having obese parents, being an only child and leading a 'sedentary life' by spending long hours watching TV instead of engaging in physical exercise.[6]

Childhood obesity has also been noted as a serious national problem in the Maltese Islands in the mid-1990's, increasingly affecting older children (around 10 years of age), females more than males.[7] Moreover, the incidence of overweight babies in Malta, with about 12% of the newborn baby population weighing 4 kgs and over, is higher than that of many other countries.[8]

Lead poisoning can occur through ingestion of contaminated food or water and also by inhalation of lead-polluted air. Central nervous system problems such as convulsions, behavioural changes, mental retardation, irritability, lack of coordination and clumsiness may

occur in children and may persist into adulthood.[2]

Women working in lead-using trades were found to have unusually high rates of infertility, spontaneous abortion, stillbirth and neonatal death. This observation in many parts of Europe, led to the banning of such trades for women.[9] Children, especially at an early age, (< 6 years) may ingest lead through contaminated soil, dust and also by eating leaded paint chips.[10] Eating food sold by street vendors, after being exposed to road dust containing high levels of lead is yet another source.[2] 'Clean' foods such as canned fruits, vegetables and fruit juices have been found to be the biggest contributors of lead in a child's diet.[9]

Drinking water coming from lead-lined water tanks or from copper piping joined by lead solder, used in modern water systems forms another source.[2] Adding lime, or in some areas, orthophosphate, has helped in preventing lead dissolving in the water-pipes.[11]

Automobile traffic emits exhaust gases containing predominantly inorganic lead aerosol. Use of lead-free gasoline, readily available in some countries, has produced cleaner air. Removal of lead from gasoline started in USA in 1972 and was completed in 1995. This practice has resulted in almost fourfold reductions in the median blood lead level of high risk children (6 months to 5 year olds) in Chicago.[12,13] However, the opposite is happening in developing countries where increase in traffic and unrestricted use of leaded petrol occurs in urban cities.

Cadmium is a heavy metal which enters the body through the same sources as lead and also through smoking (active or passive). Long-term exposure to cadmium can lead to renal tubular dysfunction and bone defects. Such medical complications arose in Japan after the ingestion of rice grown in paddies, irrigated with cadmium-contaminated river water.[2]

In cases of **methylmercury poisoning** following ingestion of seafood in Japan, and dressed seed in Iraq, many infants were born with microcephaly, irritability, cerebral palsy, or later developed epilepsy.[9]

The environment can also be hazardous through deficiencies of certain essential trace elements. **Trace element deficiency** - Severe **Zinc** deficiency in childhood is associated with dwarfism and hypogonadism and this has been found especially commonly in the Middle East. In neonates, this can cause acrodermatitis enteropathica. Infants born to mothers with **Iodine** deficiency during pregnancy have a greater risk of both motor and mental retardation. **Iron** deficiency causes anaemia, which in infants has been shown to decrease intellectual ability. Lack of **Fluoride** in children is associated with dental caries.[9]

Another source of environmental hazard is through **pesticides** such as DDT and its derivatives which pass through the placenta and affect the fetus. Reproduction is affected and birth defects and cancer have been demonstrated in animal research.[2]

Accidentally ingested **polychlorinated biphenyls (PCBs)** slow fetal growth and later impair neural development. Eating rice-oil contaminated with PCBs led to a number of Japanese women giving birth to affected children. PCBs can also be excreted in breast milk.[9]

Inhalation of hazardous compounds adds to the list of environmental dangers. High levels of **Carbon Monoxide (CO)** are found in urban cities loaded with heavy traffic. Another significant source of CO to the foetus is cigarette smoking (passive or active) by the pregnant mother.[14] This gas diffuses easily through the placental tissues, producing a concentration of 10 to 15 percent higher in the foetus than in the mother. This jeopardises foetal oxygen availability leading to retarded growth, brain damage or death.[12]

Cigarette Smoking and Environmental Tobacco Smoke (ETS): This form of indoor pollutant produces intrauterine growth retardation and increases the risk of spontaneous abortions, premature deliveries and perinatal deaths.[9,14] An interesting finding is that children born to mothers who smoked during pregnancy were on average 1 - 2 cm shorter, compared to other children, after accounting for several confounding variables.[9,14,15] The intellectual ability and behaviour of these children may be affected.[9,16,17] There has also been some speculation of increased risk of childhood cancers in children exposed to smoking during pregnancy but further studies are still required in this area.[9,14]

Cigarette smoking and ETS have been known to cause respiratory problems for a long time. Children are especially at risk of developing asthma through ETS from their parents, mostly from their mother.[14,18,19,20] Some studies [20,21,22] have shown that lung function diminishes in children exposed to ETS. A higher incidence of otitis media, rhinitis, atopy and resorting to tonsillectomy has been associated with passive smoking (ETS).[14,20] Exposure to ETS is also thought to be a risk factor for Sudden Infant Death Syndrome (SIDS).[2,14] A relationship has also been found between ETS and the development of purulent meningitis in children.[23,24] Exposure of children to ETS has shown an increased risk of leukaemia and lymphoma during adulthood.[20]

Passive smoking (ETS) also predisposes children to increased lower respiratory tract illness rates, especially in the first year of life.[20,25]

Research has also shown that the physical distance between a baby and the nursing mother who smokes and the amount of cigarettes smoked correlates with the amount of Cotinine (used as a marker) found in the baby's urine. These mothers were not exposed to ETS but were smokers themselves, smoking either away from the baby or during breastfeeding. Babies of mothers who smoked while nursing were found to have even higher levels of cotinine in the urine.[26]

Smoking among the children themselves is a growing epidemic. Regular smoking at 10 - 15 years of age is becoming as increasingly common practice (among girls more than boys), especially in places like Italy, France and Germany where more than 30% are regular smokers at this age. Moreover, smoking tends to be associated with illicit drug use among these young people.[27]

Yet another hazard emerging from the environment is **illicit drug addiction.** Cannabis is widely consumed, besides other drugs like amphetamines, barbiturates and tranquillizers.[2] Drug abuse during pregnancy gives rise to premature deliveries. Low birthweight, smaller head circumference as well as SIDS were associated with opiate addiction.[9]

In 1973, Jones et al showed that excessive consumption of **alcohol** during pregnancy can lead to a variety of congenital malformations and low birthweight - the 'foetal alcohol syndrome'.[28] The combined effect of alcohol and smoking by a pregnant mother further aggravates the situation and leads to twice as many stillbirths as when alcohol is consumed alone.[2] Children of alcoholic mothers are more likely to have behaviour problems and low IQs.[9]

Loud noise is yet another adverse effect of the environment, and which can lead to defective hearing. Pregnant mothers exposed to a lot of noise at work gave birth to children who showed an increased risk of hearing loss.[9]

Food additives and preservatives can be harmful to our health. Tartrazine (E102) is an additive commonly used in the form of an orange-yellow colouring, in both foods and drugs. E102 has been implicated in causing adverse reactions such as bronchospasm, urticaria and angioedema.[29]

Infants are more vulnerable to **waterborne chemicals and infections** than older children because of a larger water intake in relation to their body weight. Ground-water, and more so, well-water, can contain **nitrates**, especially in countries where use of nitrate fertilisers and manure in agriculture has increased. Bacteria in the GIT convert nitrates to nitrite and this induces methaemoglobinaemia, especially in infants. Another source is vegetables. WHO has recommended that infant formula milk should be prepared using low-nitrate water (at least <45mg/L). Nitrates can react with amines to form nitrosamines, which are potent carcinogens in animals. Such compounds are used in the manufacture of baby pacifiers.[2]

Lack of safe, clean **water** and sanitation services can lead to repeated attacks of infective diarrhoea in infants and children. In underdeveloped countries, this leads to malnutrition, stunting of physical and mental growth and a substantial number of deaths from dehydration.[2]

Poor drainage of stagnant waters and badly planned irrigation systems encourage mosquito infestation, many of which may carry malaria.[2] **Malaria** is endemic in 102 countries, placing over half the world's population at risk. Other **water-borne parasitic infections** such as schistosomiasis, transmitted by snails, is hazardous to the older age-group (10-14 years) who place themselves at risk by bathing and washing in infested canal water.[2]

HIV infection - AIDS poses another 'environmental' threat to the growing foetus. Babies born to infected women have 25-40 percent chance of being infected before or after birth. The infected children are almost all destined to die by the age of 5 years.[2] The few that survive are soon orphaned after their infected parents pass away. HIV infection can also be transmitted through breastmilk.[30]

Air pollution has become a major global problem affecting mostly children in more localised areas such as urban cities. Young children inhale twice as many air pollutants than do adults since more air is inhaled per unit body weight.[11] The effects of cigarette smoking and ETS on children have already been discussed above.

Another form of indoor air pollution is through **gas cooking.** Gas used for cooking, heating water or space heating is again quite hazardous to health, especially pregnant women who might be spending more time in the kitchen or in front of the fireplace. **Nitrogen dioxide** and **nitric oxide** emitted during cooking using oil stoves, gas-fired appliances and open fires leads to increased susceptibility to both bacterial and viral respiratory infections and impaired lung function.[31,32] In Moscow, the prevalence of childhood asthma was much higher in areas with high concentration of nitric ox-

ide and other pollutants.[33] Similar health effects occur with smoke and SO2 pollution from coal consumption and industrial plants in developed countries.[11] The noxious effects of gas cooking was investigated in the Middle East among children of Kuwaitis and of Europeans living there. The lung function was significantly impaired in families using gas for cooking. Moreover their children were approximately 3cm shorter than children having electric cookers at home.[34]

Wood-burning fireplaces produce several pollutants which may include cancer-causing agents such as benzo-a-pyrene and other gaseous pollutants such as CO and formaldehyde. The famous London smog of 1952 lead to over 4000 deaths affecting mostly children under the age of one. The disaster lead to the Clean Air Act (1956) and an end to coal fires in the UK.[9,11]

Ozone is a dangerous irritant to eyes, throat and lungs.[9] Studies[9,35] carried out during periods of high ozone pollution has shown a baseline shift of pulmonary function in children and an increased number of hospital visits for asthma.

Streets, playgrounds and beaches can also be hazardous to children's health. **Junk foods** sold to young children are occasionally contaminated, some contain unlicensed colouring agents and additives and others are uncooked and/or unwashed.[2]

Children may be exposed to **dog-fouling** on playgrounds. In the UK, about 100 people a year, mostly children, become partly or totally blind as a result of ingesting the eggs of a parasite (toxocara canis) found in dog faeces. Another type of toxocara causes wheezing and skin rashes. **Salmonella** bacteria has also been known to contaminate playgrounds.[11]

Toxic waste (eg. cyanide waste) dumped illegally may end up in places such as playgrounds. Beaches polluted with **sewage** contain coliform bacteria which can cause infection of the gastrointestinal tract, ear, nose and throat, eyes and skin.[11] In 1957, a 6 year old girl developed polio and subsequently died after having bathed near a sewage outflow in the Solent, UK. Such a risk is diminished nowadays with increasing immunisation coverage.

Ionising radiation can also have hazardous effects on one's health. This form of radiation mainly affects the process of cell division. Brain damage in the foetus may occur especially if the pregnant mother is exposed to radiation during the first trimester.[2] Many children born after the atomic bomb attacks in Hiroshima and Nagasaki suffered severe mental retardation. Studies[2,9] have indicated that children born to mothers irradiated during pregnancy, are more likely to die of cancers, but further research is required in this area.

The natural level of radioactivity is increased by human activity such as medicine, nuclear fallout from weapon testing and industrial and nuclear plants using radioactive by-products.[11] Radiation affects cell-division mostly of blood-forming tissues, sex-glands and skin. **Leukaemia clusters** of children living close to nuclear plants, have been cause for much concern. Such a cluster occurred in a village close to a nuclear plant in Sellafield, UK in the early 1980s, initiating much research into the matter. However, leukaemia clusters have also been found in areas where power plants have been planned but never built. This might be explained by population migration to this area leading to epidemics of common viral infections in the new towns.[9,11,36] This in turn may contribute to a leukaemia cluster (the Kinlen Hypothesis).[37]

Another type of radiation is **electromagnetic radiation** found wherever there is electric power. An increased risk of all cancers in children has been associated with the use of electric blankets. The use of electric appliances has been associated with premature labour.[9]

Children are adventurous and therefore more likely to be injured by falls, drowning, scalding, burns and accidental ingestion of dangerous liquids and drugs. Such incidents are also products of the environment. **Accidents** are more common in places where children live in poverty, poor housing and where social isolation of lone mothers exists.[38]

The environment is also a means of educating society. However, **education** is not available to all societies alike. In developing countries, around 50% of children attend primary school.[2] However, only about 20% of boys and 10% of girls attend secondary school. These percentages vary in different countries. More girls tend to be kept at home in order to do chores such as fetching wood and water in these developing countries. This later reflects itself in the lack of knowledge about hygiene and health in these girls who later bear their own children. This sex discrimination also extends further in some parts of Asia and Africa, with girls being given second rate health care and nutrition.[2] Lack of education results in lack of knowledge about the environment. This in turn, leads to inadequate use of facilities that the environment provides, with resulting malnutrition, ill health, illiteracy, poverty, crime and war.

Child labour is a preferred option to education in some countries. Poverty drives chil-

dren to go to work at a very tender age, partly to help their family income, and partly to help themselves especially if they are homeless. Some children work for long hours without proper rest and nutrition and are continuously exposed to risk especially in certain lines of work (toxic vapours, corrosive liquids, and a whole range of infections in garbage collecting). Ninety-eight percent of economically active children are in fact, found in developing countries.[2]

Poverty also drives children **homeless**; on the street with no roof over their head, begging, stealing, doing odd jobs like shoe shining and washing cars. Some of these children form gangs, or enter the drug trade or prostitution. The World Health Report in 1995 states that **extreme poverty** is 'the world's biggest killer and the greatest cause of ill-health and suffering across the globe'.[39]

Unfortunately, children are also victims of **war**. About 2 million children are estimated to have been killed in wars during the past decade. **Child soldiers** under the age of 16 years, numbered as many as 200,000 in 1988 alone. **Land-mines** provide the most lethal weapon of all, especially to children playing or working in fields. About 110 million land-mines still remain unexploded in 64 countries around the world.[40] Wars do not just kill children but many are disabled, left homelsss, orphaned and many more are psychologically traumatised, probably for life.

CONCLUSION

The state of our environment affects the health of all strata of society, especially that of infants and children. Besides the problems mentioned above, more ominous hazards with far-reaching consequences loom on the horizon. These include global warming, ozone depletion, deforestration and desertification just to name a few. Global warming and ozone depletion are expected to change disease patterns as well as perhaps decreasing our immune response to various infections.[2] Children, again, are the most vulnerable sector of our population, and tend to be affected the most by these problems.

The United Nations Convention on the Rights of the Child, Article 24 specifically deals with environment stating that 'children have the right to live in a safe, healthy and unpolluted environment with good food and clean, drinking water'. Heads of State are continuously labouring towards reaching this goal; for some countries, this will obviously take longer than others.[41]

Mankind is permanently under the reign of the environment. We, as part of society, should treat our environment with care and reverence and make sure that we do not underestimate its influence on our health. Whenever we can, we should strive to try and make it better, especially for the sake of our voiceless population, the children of the world.

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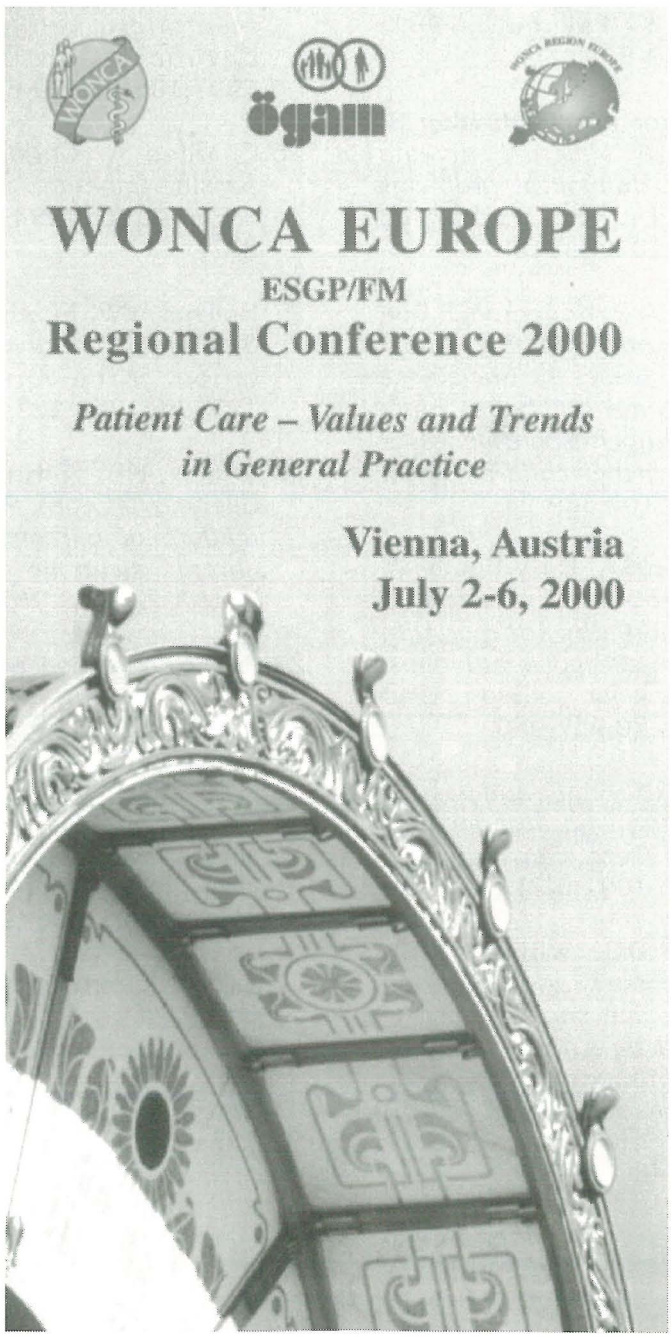
WONCA EUROPE

Regional Conference 2000
Vienna, Austria 2-6 July, 2000

World-Wide Exchange of Information on General Practice

The Society for General Practice (OGAM) is the Organiser of the 6 th European Congress on General Practice to be held from 2 to 6 July 2000, at the Vienna Hofburg. This Congress on the theme of "Patient Care - Values and Trends" is intended as a forum for a world-wide exchange of information on general practice as well as teaching and research in this area. The modfern conference centre at the Vienna Hofburg and the atmosphere of Vienna with its varied offer of cultural activities provide an ideal framework for this major event.



Aim of the Congress

The congress will draw attention to general practice and, above all, provoide a forum for information and communication among general practitioners.

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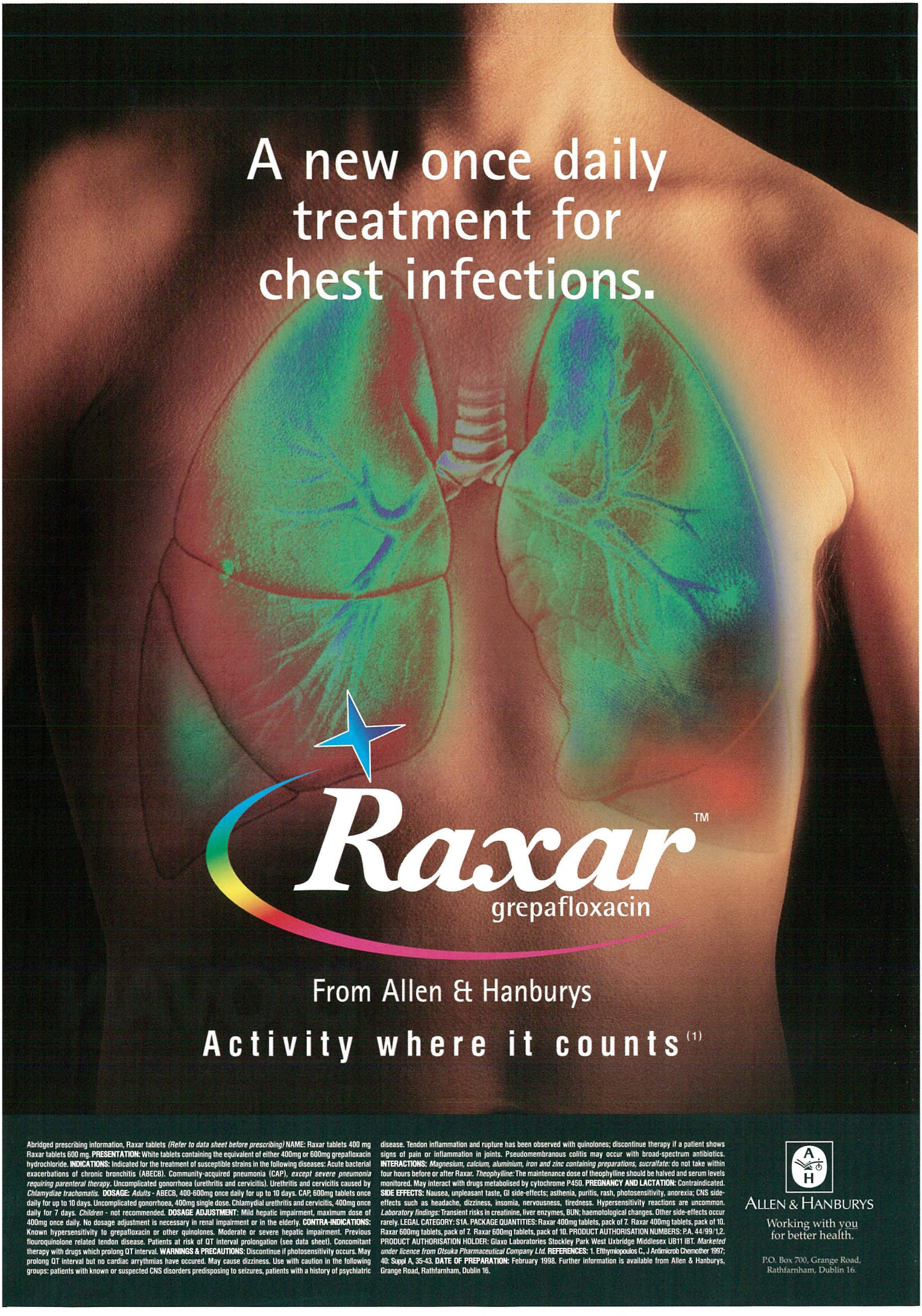

Abridged prescribing information. Raxar tablets *(Refer to data sheet before prescribing)* **NAME:** Raxar tablets 400 mg Raxar tablets 600 mg. **PRESENTATION:** White tablets containing the equivalent of either 400mg or 600mg grepafloxacin hydrochloride. **INDICATIONS:** Indicated for the treatment of susceptible strains in the following diseases: Acute bacterial exacerbations of chronic bronchitis (ABECB). Community-acquired pneumonia (CAP), *except severe pneumonia requiring parenteral therapy*. Uncomplicated gonorrhoea (urethritis and cervicitis). Urethritis and cervicitis caused by *Chlamydiae trachomatis*. **DOSAGE:** *Adults* - ABECB, 400-600mg once daily for up to 10 days. CAP, 600mg tablets once daily for up to 10 days. Uncomplicated gonorrhoea, 400mg single dose. Chlamydial urethritis and cervicitis, 400mg once daily for 7 days. *Children* - not recommended. **DOSAGE ADJUSTMENT:** Mild hepatic impairment, maximum dose of 400mg once daily. No dosage adjustment is necessary in renal impairment or in the elderly. **CONTRA-INDICATIONS:** Known hypersensitivity to grepafloxacin or other quinolones. Moderate or severe hepatic impairment. Previous flouroquinolone related tendon disease. Patients at risk of QT interval prolongation (see data sheet). Concomitant therapy with drugs which prolong QT interval. **WARNINGS & PRECAUTIONS:** Discontinue if photosensitivity occurs. May prolong QT interval but no cardiac arrythmias have occured. May cause dizziness. Use with caution in the following groups: patients with known or suspected CNS disorders predisposing to seizures, patients with a history of psychiatric disease. Tendon inflammation and rupture has been observed with quinolones; discontinue therapy if a patient shows signs of pain or inflammation in joints. Pseudomembranous colitis may occur with broad-spectrum antibiotics. **INTERACTIONS:** *Magnesium, calcium, aluminium, iron and zinc containing preparations, sucralfate:* do not take within four hours before or after Raxar. *Theophylline:* The maintenance dose of theophylline should be halved and serum levels monitored. May interact with drugs metabolised by cytochrome P450. **PREGNANCY AND LACTATION:** Contraindicated. **SIDE EFFECTS:** Nausea, unpleasant taste, GI side-effects; asthenia, puritis, rash, photosensitivity, anorexia; CNS side-effects such as headache, dizziness, insomia, nervousness, tiredness. Hypersensitivity reactions are uncommon. *Laboratory findings:* Transient risks in creatinine, liver enzymes, BUN; haemotological changes. Other side-effects occur rarely. **LEGAL CATEGORY:** S1A. **PACKAGE QUANTITIES:** Raxar 400mg tablets, pack of 7. Raxar 400mg tablets, pack of 10. Raxar 600mg tablets, pack of 7. Raxar 600mg tablets, pack of 10. **PRODUCT AUTHORISATION NUMBERS:** P.A. 44/99/1,2. **PRODUCT AUTHORISATION HOLDER:** Glaxo Laboratories Stockley Park West Uxbridge Middlesex UB11 IBT. *Marketed under licence from Otsuka Pharmaceutical Company Ltd.* **REFERENCES:** 1. Efthymiopoulos C., J Antimicrob Chemother 1997; 40: Suppl A, 35-43. **DATE OF PREPARATION:** February 1998. Further information is available from Allen & Hanburys, Grange Road, Rathfarnham, Dublin 16.

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Updated Recommendations for Endocarditis Antibiotic Prophylaxis Summarised from Recommendations by the American Heart Association

Dr. V. Grech
Paediatric Senior Registrar, St. Luke's Hospital
Prof. A. Fenech
Consultant Cardiologist, Department of Medicine, St. Luke's Hospital

INTRODUCTION

Bacteraemia may occur spontaneously or may complicate a focal infection or surgical/dental procedures. Blood-borne bacteria may lodge on abnormal heart valves or near structural defects or on normal endocardium causing endocarditis.

Although relatively uncommon, endocarditis is associated with substantial morbidity and mortality despite improvements in antimicrobial therapy and enhanced ability for early diagnosis. Hence, primary prevention of endocarditis is extremely important.

The following is a summary of the new recommendations for antibiotic prophylaxis by the American Heart Association (1,2). These recommendations reflect analyses of the literature regarding procedure-related endocarditis, and are an update of those drawn up in 1990(3), incorporating new data which has become available since that time. Changes from the previous recommendations are also detailed.

Changes in the updated recommendations include:

1. Cardiac conditions are stratified into high-(4,5), moderate-(4,5,6), and negligible-risk categories (no need for prophylaxis) based on potential outcome if endocarditis develops.
2. Procedures that may cause bacteraemia and for which prophylaxis is recommended are clearly specified.
3. Conversely, procedures for which prophylaxis is not recommended are also clearly specified.
4. For oral or dental procedures the initial antibiotic dose is reduced and a follow-up antibiotic dose is no longer recommended. If a series of dental procedures is required, it is prudent to observe an interval of time between procedures to both reduce the potential for the emergence of resistant organisms, and allow repopulation of the mouth with antibiotic susceptible flora. Various studies have suggested an interval of 9 (7) to 14 (8) days. Alternatively, if possible, a combination of procedures should be planned within the same period of prophylaxis.
5. Regimens for gastrointestinal or genitourinary procedures have been simplified.
6. Prophylaxis for mitral valve prolapse has been reviewed. The risk of endocarditis is not increased above that of the normal in prolapse without clinical or echo detectable regurgitation (4).On the other hand, patients with prolapse and regurgitation are at a higher risk of developing endocarditis and should receive prophylaxis (9). The risk is also increased in mitral valve prolapse associated with myxomatous degeneration, and in these patients, the mitral valve leaflets appear thickened on echocardiography (10), even in the absence of regurgitation on echo.
7. For penicillin-sensitive individuals, clindamycin is preferred over erythromycin due to the latter's higher incidence of gastrointestinal upset and the complicated pharmacokinetics of the various formulations (11).

Doctors should exercise their own judgement in determining the choice of antibiotics and number of doses that are to be administered in special circumstances. It should also be remembered that endocarditis may occur in spite of appropriate prophylaxis. Unusual clinical symptoms or signs following dental or other surgical procedures in patients who are at risk for developing bacterial endocarditis should be regarded with suspicion. Furthermore, most episodes of endocarditis occur in previously normal hearts.

New antibiotic prophylaxis cards.

The following points and regimens will be incorporated in a new antibiotic prophylaxis card. This will have a pale green background as does the current card, in order to avoid confusion between patients and carers.

Antibiotic prophylaxis is indicated in:

- Congenital heart disease except as above
- Acquired valvar dysfunction (e.g., rheumatic heart disease)
- Hypertrophic cardiomyopathy
- Mitral valve prolapse with valvar regurgitation and/or thickened leaflet

High-risk patients:

- Previous bacterial endocarditis
- Prosthetic cardiac valves, including bioprosthetic and homograft valves
- Complex cyanotic congenital heart disease including transposition of the great arteries, tetralogy of Fallot and conditions repaired using surgically constructed systemic- pulmonary shunts or conduits

Patients who normally require antibiotic prophylaxis do not need prophylaxis when undergoing the following procedures:

Respiratory tract	Endotracheal intubation Flexible bronchoscopy ± biopsy† Tympanostomy tube insertion
Gastrointestinal tract	Transesophageal echocardiography† Endoscopy ± gastrointestinal biopsy Dilatation of oesophageal stricture† Biliary tract surgery/procedure involving intestinal mucosa†
Genitourinary tract	Vaginal hysterectomy† Vaginal delivery† Caesarean section Circumcision
In uninfected tissue	Urethral catheterisation Uterine dilatation and curettage Therapeutic abortion Sterilisation procedures Insertion or removal of intrauterine devices
Other	Cardiac catheterisation, including balloon angioplasty Incision or biopsy of surgically scrubbed skin Implanted cardiac pacemakers, defibrillators, and coronary stents

†Prophylaxis is optional for high-risk patients (see above)

There is no need for antibiotic prophylaxis in the following conditions as risk of endocarditis is not greater than the general population:

- Isolated secundum atrial septal defect
- Surgically repaired atrial septal defect, ventricular septal defect and patent ductus arteriosus with no residual defects and 6 months after intervention
- Previous coronary artery bypass graft surgery
- Mitral valve prolapse with no valvar regurgitation
- Physiologic, functional or innocent heart murmurs

Regimens for antibiotic prophylaxis:

Dental, oral, respiratory tract, or oesophageal procedures		
Amoxycillin	PO	
Amoxycillin/ampicillin	IM/IV	if unable to take PO
Macrolide	PO	if allergic to penicillins
Non-oesophageal gastrointestinal procedures and genitourinary procedures		
Ampicillin/amoxycillin	IV	
Vancomycin	IV	if allergic to penicillins
High-risk patients (see above)		
Ampicillin/amoxycillin + gentamicin	IV	repeat the penicillin 6 hours later at ½ standard dose
Vancomycin + gentamicin	IV	if allergic to penicillins
Intervention on infected non-oral soft tissues or bone/joint infections		
Flucloxacillin/1st generation cephalosporin	PO	
Macrolide	PO	if allergic to penicillins
Vancomycin	IV	if unable to take PO or known/suspected MRSA
Routes and administration		
	PO	1 hour before procedure
	IM/IV	complete within ½ hour of starting procedure, including vancomycin infusion
Doses		
Ampicillin/Amoxycillin	PO/IM/IV	50 mg/kg/dose up to 2000 mg
Macrolide: Clindamycin/Erythromycin	PO	20 mg/kg/dose up to 600 mg
Gentamicin	IM/IV	1.5 mg/kg/dose up to 120 mg
Cephalexin/Cefadroxil (or other 1st generation)	PO	50 mg/kg/dose up to 2000 mg
Flucloxacillin	PO	50 mg/kg/dose up to 2000 mg
Vancomycin	IV	20 mg/kg/dose up to 1000 mg IVI over 1-2 hours

- Previous Kawasaki disease/ rheumatic fever with no valvar dysfunction
- Cardiac pacemakers (intravascular and epicardial) and implanted defibrillators
- If on penicillins already, wait until 14 days after finishing penicillins or use clindamycin instead.
- If procedure involves infected tissue, it may be necessary to provide additional doses of antibiotics for treatment of the established infection.
- IM route is contraindicated in patients who receive heparin or warfarin. IV or PO regimens should be used whenever possible.

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MMR Immunization - an Appeal

Dr. P. Vassallo Agius
Consultant Paediatrician

Immunization has long been established as an efficient and effective means of preventing mortality and morbidity of potentially serious infections. Indeed it has been instrumental for the complete elimination of a once-deadly scourge, viz, smallpox. It is envisaged that poliomyelitis will be the next serious infection to be literally eradicated from the face of the earth. And after that, measles will be next on the list, no doubt to be followed by others. Even now, most of the young doctors are unlikely to have to care for a case of measles throughout their career.

The steady conquest of infectious diseases by immunization has sometimes been hampered by often-unfounded scare mongering, often fuelled by the media. The older doctors will remember the vociferous campaign by the media in the UK in the seventies against (whole cell) whooping cough vaccination, because it allegedly caused "brain damage". Vaccine uptake plummeted, with a consequent increase in the number of reported cases of whooping cough, with the inevitable morbidity and mortality. The saga went on for over ten years. It can be confidently stated that not even a single case of "brain damage" has been unequivocally proved to have been due to vaccination by whole cell pertussis vaccine. It has been amply shown that the brain damage can be temporally related to pertussis vaccination, but this is not proof that there is a causal relationship between the brain damage and pertussis vaccination. The myth has, hopefully, finally been laid to rest. In Malta, vaccination against whooping cough has caught up with other countries, and recent figures show consistent uptake of vaccine at over 90 per cent.

Unfortunately, the same cannot be said of vaccination against mumps, measles and German measles (rubella) - MMR vaccine. Recent figures have shown uptake of only 51% at two years, which is well below what is required to ensure herd immunity. This is not acceptable. MMR vaccination was initially dented some years ago when the mumps component was withdrawn in the UK because of alleged implication of the particular strain used in the causation of meningeal irritation. Even though this particular scare has apparently subsided, the MMR vaccine uptake has remained low. It may be due to incomplete reporting. But the real reason may well be the more recent scares linking measles in utero to Crohn's disease(1,2), measles vaccination to inflammatory bowel disease (3), and MMR to infantile autism in another report (4). In both instances, the association was a tenuous one which did not stand up to the rigours of scientific proof. An expert group met recently at the Royal Free Hospital and, after critically examining the controversial evidence claiming that MMR vaccination was linked with inflammatory bowel disease and autism, has backed the continuation of the combined vaccine, and dismissed calls to administer these vaccines separately(5).

At a recent meeting of the Advisory Committee on Immunization Policy (ACIP) these issues were discussed. There was no reason to change present policy of administering MMR vaccination at 15 months of age with a booster dose at 10 - 11 years of age. It is thought that the low uptake of MMR vaccine in Malta may well be due to under-reporting and doctors administering the MMR vaccine are solicited to send the appropriate report card.

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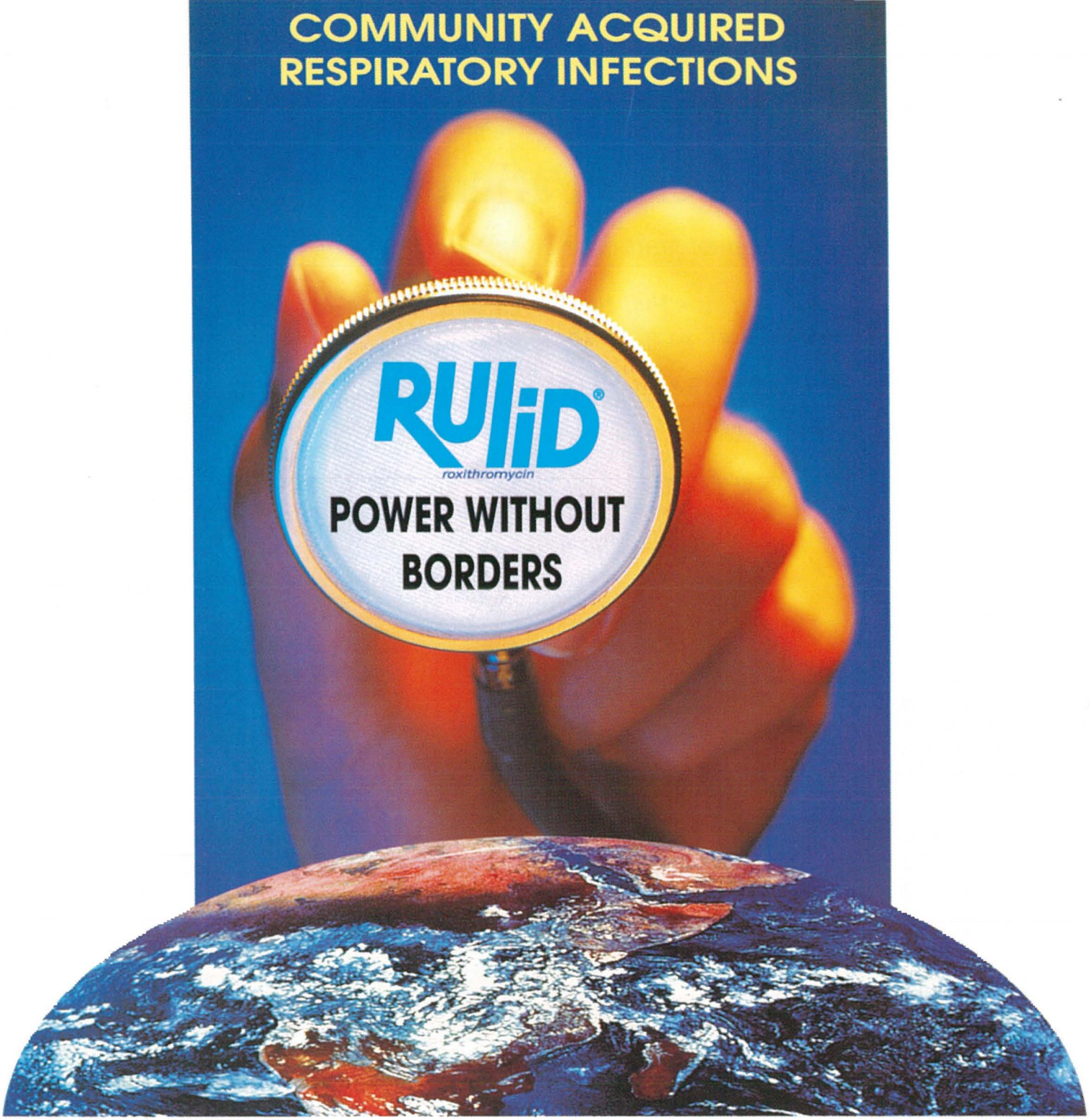

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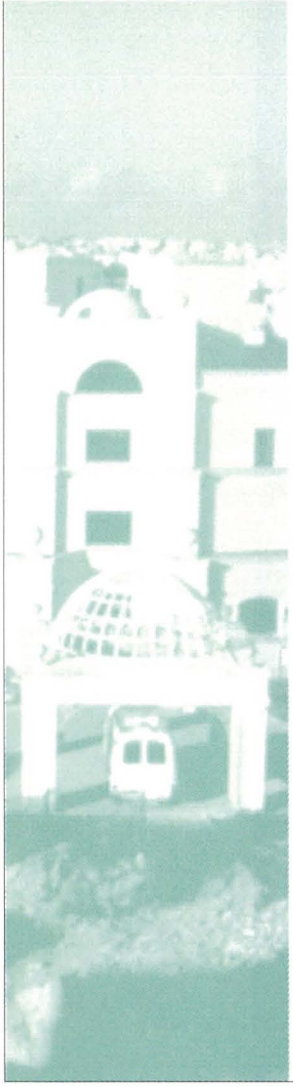

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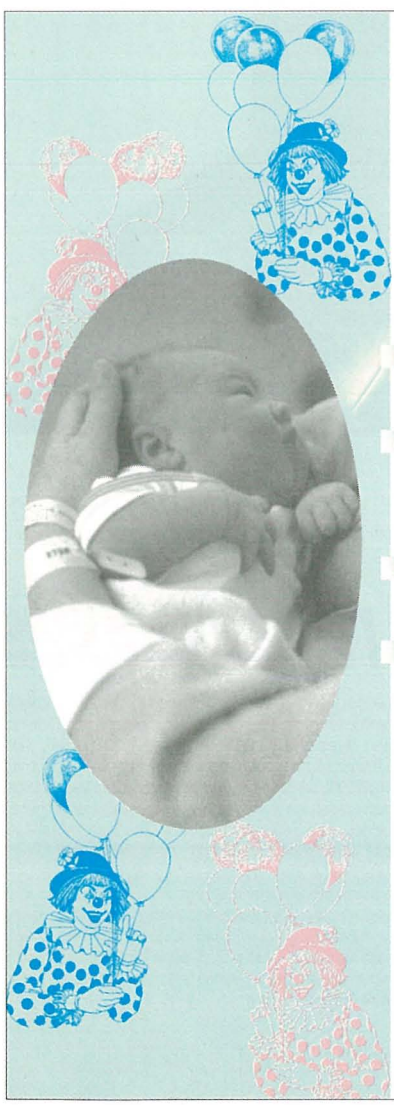

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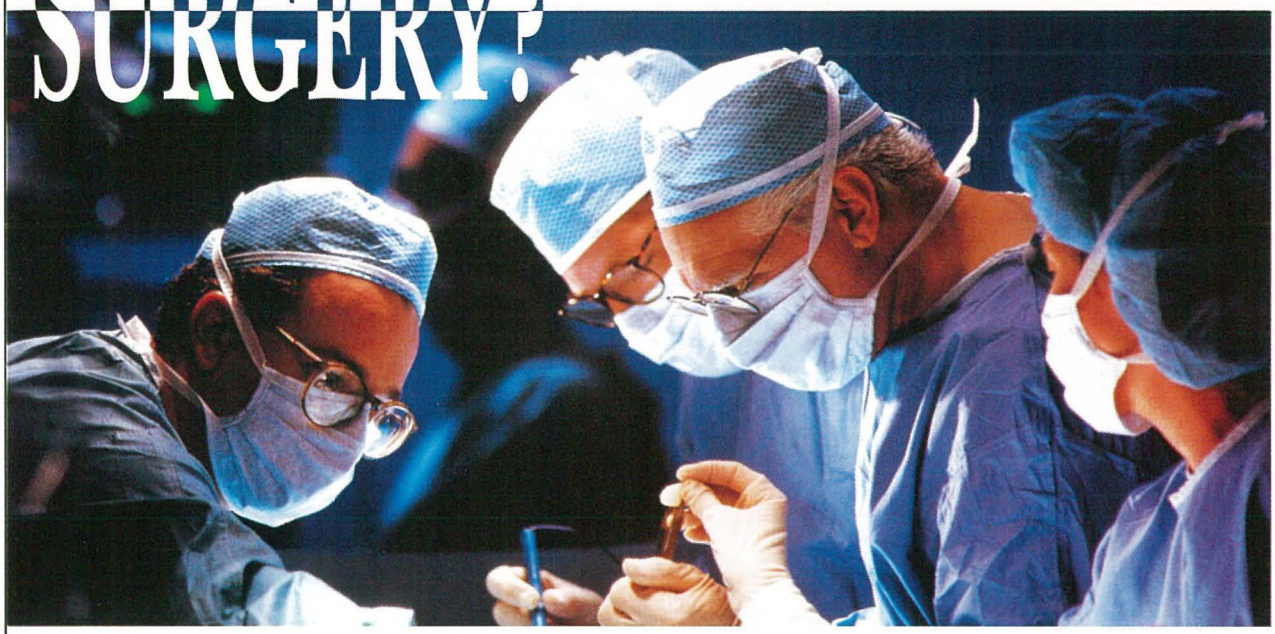

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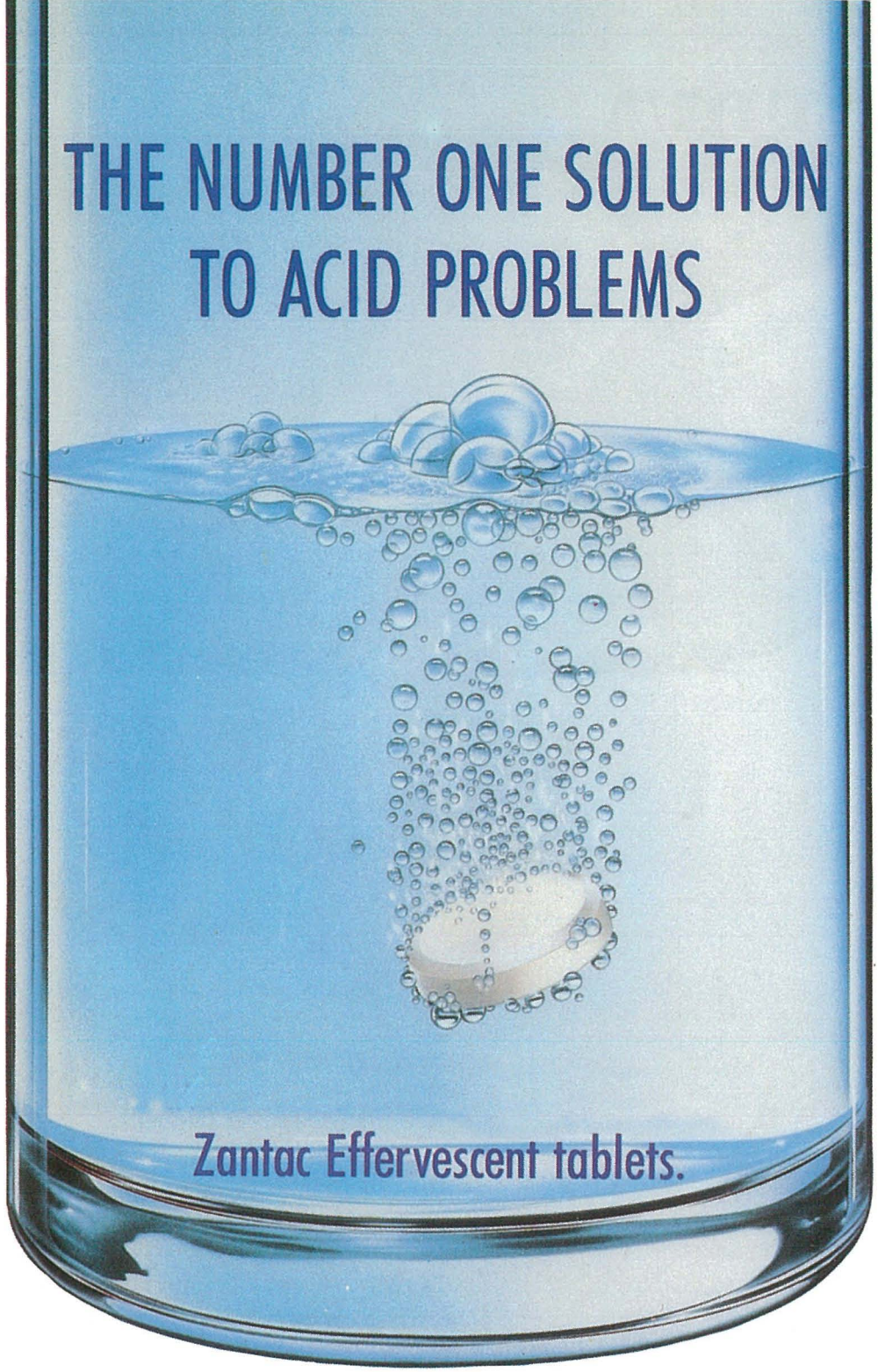

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Two Epidemics in French Occupied Valletta in 1798-1800

Dr. Paul Cassar
Medical Historian

INTRODUCTION

General Bonaparte captured the Maltese Islands on the 10 June 1798 and drove away the Knights of St John. He took over their Holy Infirmary and turned it into an ***Hopital Militaire*** *for his sick troops. Dr Claude Etienne Robert, was appointed* ***Medecin-en-Chef*** *in charge of medical cases and Jean Pierre Fauverge* ***Chirurgien Major*** *was made responsible for surgical patients.*

During the following two years Robert treated no less than three thousand medically sick troops; and Fauverge treated ninety-two surgical cases. There were besides a number of venereal patients, forty of whom died within three months (1).

Two outstanding events marked the tenure of office of these two French practitioners - an epidemic of scurvy and an outbreak of infestation by intestinal worms.

SCURVY

Modern medicine has shown that this disease is caused by a deficiency in the diet of vitamin C which is found naturally in fresh vegetables and fruits; but in 1798-1800 the cause of scurvy was still debatable. Some suggested that it was due to excessive moisture in the air but others were nearer the truth when they ascribed it to dietary deficiencies such as fresh vegetables and fruits (2).

Following the uprising of the Maltese on the 2nd September 1798 and the consequent blockade of the French inside Valletta, food rations for the troops were limited in quantity and quality to make them last as long as possible but finally they were reduced to salted meat, beans and rice. Vegetables and fruits were unobtainable. The soldiers were encouraged to cultivate vegetables on the ramparts but these efforts had to be given up to save the water in the cisterns of the city as the hospital aqueduct had been cut off by the insurgents.

Scurvy made its appearance gradually towards the end of November but it made such rapid progress that soon there were about six hundred soldiers in hospital suffering from this disease. Fauverge has left us a classical picture of the clinical signs and symptoms as he saw them at the *Hopital Militaire* :- "The gums were swollen and bled, the teeth became loose and the mouth foul-smelling. The sick complained of acute pains in the chest and a feeling of heaviness in the precordial region, loss of appetite and general weakness. Almost the entire surface of the body was covered with livid areas; the skin of the lower limbs was dry and taut; the lips parched and livid; the face swollen and pale. Sometimes there occurred bleedings from the nose and mouth. Swelling of the lower abdomen and respiratory failure heralded the approach of death"(3).

Both Robert and Fauverge were aware that the only treatment that could have benefitted the scorbutics was a vegetarian fare and fresh meat, but these articles of food were not available. Consideration was given by the military command to the idea of making an incursion on Comino and a landing at Mellieha Bay to raid the countryside for fresh vegetables; but this idea was not followed up. (4)

NIGHTBLINDNESS

Another disorder due to prolonged dietary deprivation was nightblindness. This is due to lack of Vitamin A which occurs naturally in green leafy vegetables. This is a condition of impaired vision occurring during the feeble light of night-time when small objects cannot be seen from a distance, and there is also the inability to distinguish colours. This condition was not alarming in itself in so far as the general health of the garrison was concerned but it had one serious consequence from the military view point for those soldiers suffering from it could not be assigned guard duties on the ramparts at night as they were unable to detect any movements on the part of the insurgents prowling outside the walls of the fortifications. In the absence of the only effective remedy - the consumption of green vegetables - Robert tried to treat the patients by fumigations of the eyes with "aromatic plants" and "the livers of animals"; but he found that these remedies gave only transient relief.

FEVERS

The scurvy was followed by a wave of "intermittent" fevers which responded well to the administration of laxatives and the application of compresses soaked in iced water and vinegar and placed over the forehead and head; so much so that it was not always necessary to prescribe quinine. In other patients, however, the fevers assumed a "malignant" form with prostration, dry tongue and lips and stupor, but they responded well to quinine in high doses.

Other cases that had to be dealt with were those suffering

from lung tuberculosis with emaciation and dyspnoea which proved to be fatal. Postmortem examination of these men by Robert showed tubercles and purulent ulcers in the lungs with involvement of the mesentery.

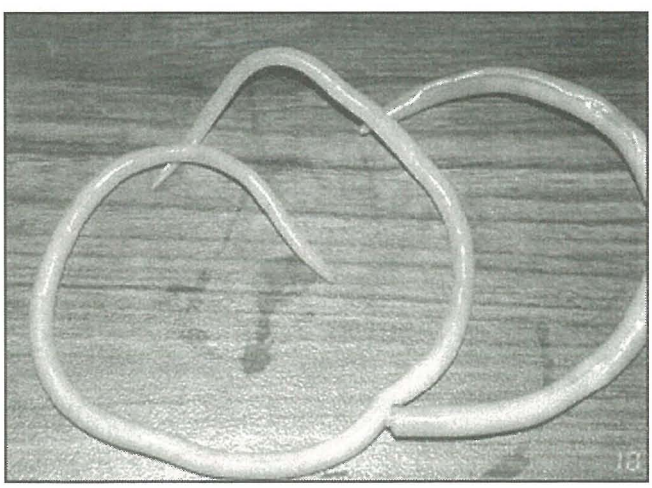
INTESTINAL WORMS INFESTATION

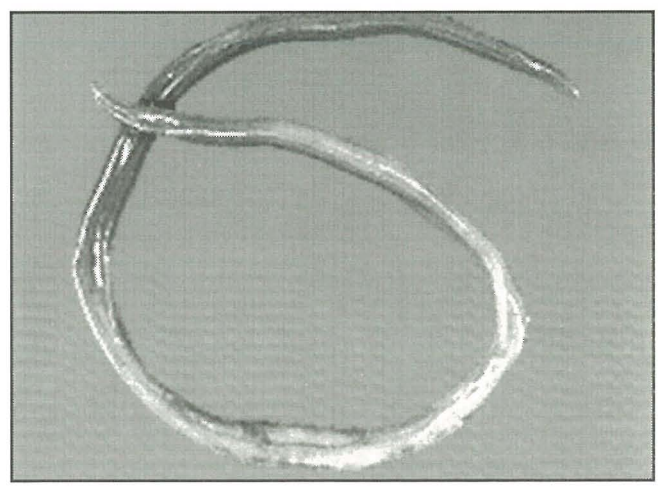
Concurrently with the outbreak of"fevers", Robert had to deal with an invasion of intestinal worms from which no one in the garrison escaped. These worms ***Ascaris lumbricoides*** were of an "extraordinary" length, volume and numbers. There were instances where the host expelled two hundred of them in one stool. Some patients extruded the worms through their mouth during sleep with the risk of suffocation. Others complained of abdominal pain and tension. Fauverge remarked that even domestic animals - such as fowls and rabbits - were infected (5).

This roundworm infestation caused great dismay in the garrison with the result that "everyone" began having vermifuges - even those who showed no evidence of being affected. "I myself followed their example", confessed Robert when he himself extruded "a dozen worms of astonishing size and volume". Robert treated the hosts with vermifuge powders as recommended in French hospitals formularies with very good results. However there were a few deaths and Robert took the opportunity to perform autopsies. He found that the presence of worms was confined to the intestines, the stomach, oesophagus and throat.

Since Robert's time, research workers have shown that the ***Ascaris lumricoides*** could be as long as 35 cm with a diameter "of a lead pencil". It has been estimated that twenty adult round worms will consume 2.8 grams of carbohydrate each day from the host's small intestine thus interfering with the absorption of food with consequent loss of nutrition of the host. In Robert's days, therefore, this infestation was an added cause of dietary depletion apart from the starvation diet to which the host was already subjected due to lack of food supplies.

In Robert's time it was not known that round worm infestation was conveyed to man by the ingestion of the worm's eggs from contaminated soil and water and from the improper disposal of human faeces (6).





ENVOY

Robert and Fauverge left the *Hopital Militaire* and Malta and returned to France as a result of the French capitulation of 5 September 1800. They continued to pursue a successful professional career in the French army and found time to write about their medical experiences in Malta. In 1802 Dr. C.E. Robert published his ***Memoire sur la topographie physique et medicate de Malte;*** and in 1803 Surgeon J.P. Fauverge gave to the press his ***Des Maladies qui ont regne a Matte pendent Le Blocus de l'an Vll et Vlll.***

These two works form an outstanding chapter in the medical annals of Maltese history.

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Dyslexia - A Medical Overview

Dr. Christopher Sciberras
Registrar - Department of Paediatrics, St. Luke's Hospital

"It is a lonely existence to be a child with a disability which no - one can see or understand, you exasperate your teachers, you disappoint your parents, and worst of all you know that you are not just stupid. "

Susan Hampshire, President - The Dyslexia Institute, U.K.

SUMMARY

Dyslexia and other related learning disabilities are serious problems. Early diagnosis and educational remediation is of paramount importance. There is no known eye or visual cause for dyslexia and learning disabilities, and no effective visual treatment. Multidisciplinary evaluation and management must be based on proven procedures demonstrated by valid research.

INTRODUCTION

Nearly a century has passed since the first published observation that some otherwise normally - intelligent children have specific difficulty in learning to read. The intervening years have seen both an intense search for the mechanism of this disorder and an ongoing debate about whether it exists.[1]

The disorder was first termed "congenital word blindness" by British ophthalmologists, and for several decades it was assumed to be primarily visual, until the 1930's. Then the American neurologist Samuel Orton called attention to the frequent association between reading disability in the primary grades and disorders of spoken language during the preschool years, hence pointing to an underlying disturbance of language.[2] The further association with left - handedness or ambidexterity suggested a disorder of cerebral hemispheric specialisation. This concept of "mixed dominance" was extended to account for the reversals of single letters (b and d) or letter order ("was" for "saw") that were frequently observed in dyslexic readers - a phenomenon Orton referred to as "strephosymbolia."

The 1950's and early 1960's saw a wave of enthusiasm for the interpretation of specific learning disabilities as disorders of perceptual faculties. It was during this period that the distinction between "auditory" and "visual" learners became fashionable, and the popularity of the "language disorder" concept waned. Also during these years, the first studies appeared suggesting that dyslexia may have more than one mechanism.[3] The resulting controversy between "lumpers" who see dyslexia as a unitary disorder, and "splitters", who believe there are multiple causes, persists to the present.

More recently, a series of studies begun by Avan Liberman and co-workers at the Haskins Laboratories[4] have focused on how the brain (specifically the major temporal lobe) uses the "speech code" to make language out of acoustic signals. The resulting formulation of dyslexia as a disorder of phonemic awareness has been supported by a wide variety of anatomical, psychophysiologic, and neuro-psychological findings. The great responsibility placed on teachers and schools in the early recognition of the child with special educational needs with particular reference to dyslexia, emphasises the important role of doctors and other healthcare professionals in this field. This role includes early recognition, medical assessment of the whole child holistically, advising the Local Education Authority and liaising with all the relevant members of the teaching, psychological and health professions as well as the parents.

It is particularly important that the valuable knowledge of the child's early development and strengths and weaknesses should be shared with the teachers. This should enable them better to understand the child and his teaching needs.

The child with dyslexia, who is not recognised early, suffers severe stress and anxiety and frustration as he falls further and further behind his peers. The loss of self - image results in secondary emotional problems and can also severely affect his behaviour.

Definition of Dyslexia:

('Dys' = difficulty, 'lexicon' = words or symbols).

"Dyslexia is a specific learning difficulty that hinders the learning of literacy skills. This problem with managing verbal codes in memory is neurologically based and tends to run in families. Other symbolic systems, such as mathematics and musical notation, can also be affected.

Dyslexia can occur at any level of intellectual ability. It can accompany, but is not a result of, lack of motivation, emotional disturbance, sensory impairment or meagre opportunities.

The effects of dyslexia can be alleviated by skilled specialist teaching and committed learning. Moreover many dyslexic people have visual and spatial abilities that enable them to be successful in a wide range of careers. "

The Dyslexia Institute, February 1996.

Dyslexia is a neurologically based disorder in which there is an unexpected failure to read.

As defined by the **World Federation of Neurology, the disorder is "manifested by difficulty in learning to read despite conventional instruction, adequate intelligence, and sociocultural opportunity and is dependent upon fundamental cognitive disabilities which are frequently of constitutional origin."**[5]

Dyslexia is a learning disability that alters the way the brain processes written material. The effects of the disorder vary from person to person. In fact, the only common trait among people with dyslexia is that they read at levels significantly lower than typical for people of their age and intelligence.[6] Dyslexia is also referred to as "specific reading disability" or "specific reading retardation." It is generally assumed that the failure to learn to read represents a specific syndrome that is distinct from the normal distribution of poor readers. Rather than representing the lower end of a continuum of reading disability and reading ability, dyslexia (or specific reading disability) is viewed as a biologically coherent disorder that is distinct from other, less specific reading problems. Support for this point of view comes from the work of Rutter and Yule,[7] who found that "children with dyslexia form a 'hump' at the bottom of the normal curve."[8] They used these findings to argue that reading ability is bimodally distributed, with specific reading disability appearing as the extreme lower tail. This notion of reading disability as a specific, discrete entity serves as the basis both for investigations into the neurobiology of dyslexia and for the diagnosis of dyslexia and the provision of services to persons with the disorder.

Rather than following the bimodal - distribution posited by Rutter and Yule, another model continues to dominate thinking in the field. Shaywitz et al.,[9] who investigated both the distribution and the temporal stability of reading disability by analysing data from the Connecticut Longitudinal Study, hypothesised that dyslexia occurs along a continuum and is best conceptualized as the tail of a normal distribution of reading ability. Dyslexia is therefore a specific aptitude deficit, leading to underachievement in reading by children of otherwise normal intelligence. These findings therefore provide support for a fundamental revision in the concept of dyslexia; rather than existing as a discrete entity, dyslexia, like hypertension and obesity, occurs along a continuum and varies in severity.

Prevalence: Dyslexia affects I pupil in 25, affecting about 350,000 pupils in the U.K. Dyslexia is believed to affect 4 - 5 % of the population, or some 12 million in America.

Sex prevalence: Boys are affected with greater severity than girls.

Classification: Learning disabilities, including dyslexia, are divided into two types: **primary** (inherited) and **secondary** (caused by a physical factor that interferes with learning).

Aetiological Factors:

Reading is a complex function that involves integrating multiple factors related to an individual's experience, ability, and constitution. Although it is obvious some children do not read well because they have visual problems, research has shown that the majority of children and adults with reading difficulties experience a variety of language defects that stem from complex, altered brain morphology and function, and that the reading difficulty is not due to altered visual function per se. Furthermore, no scientific evidence supports claims that the academic abilities of dyslexic or learning - disabled children can be improved with treatment based on visual training, neurological organizational training, or tinted or coloured lenses.

The exact cause of learning disabilities is not yet known. Basic scientific research into the role that brain structure and function play in learning disabilities has demonstrated that the basis of dyslexia and other specific learning disabilities is within the central nervous system and is multifactorial and complex.

There is now substantial evidence to suggest that dyslexia is a disorder of neurobiological origin. In addition to the well - known deficit in phonological processing[10], dyslexic individuals have altered lateral cerebral symmetry[11], impaired visual[12] and auditory processing[13], disordered magnocells[14], and altered patterns of cerebral activation on verbal, visual, and auditory tasks[15]. The area of the brain most frequently implicated is the temporo - parietal cortex and, more recently, the cerebellum[16].

Factors associated with the origin of dyslexia:

(i) A family history of difficulty with written language or speech is present in the majority of cases.

(ii) A history of placental dysfunction, resulting in a small - for - dates baby, can be significant.

(iii) Acquired dyslexia - secondary to a difficult birth with anoxia, can manifest as dyslexia.

(iv) Abnormal migration of the grey cells in the cortical layer, towards the end of the second trimester of pregnancy.

(v) Abnormal sequence of function of the magna and parvo cellular systems in the visual and / or auditory, and / or kinaesthetic pathways.

(vi) Possible effect of allergies and genetic factors on

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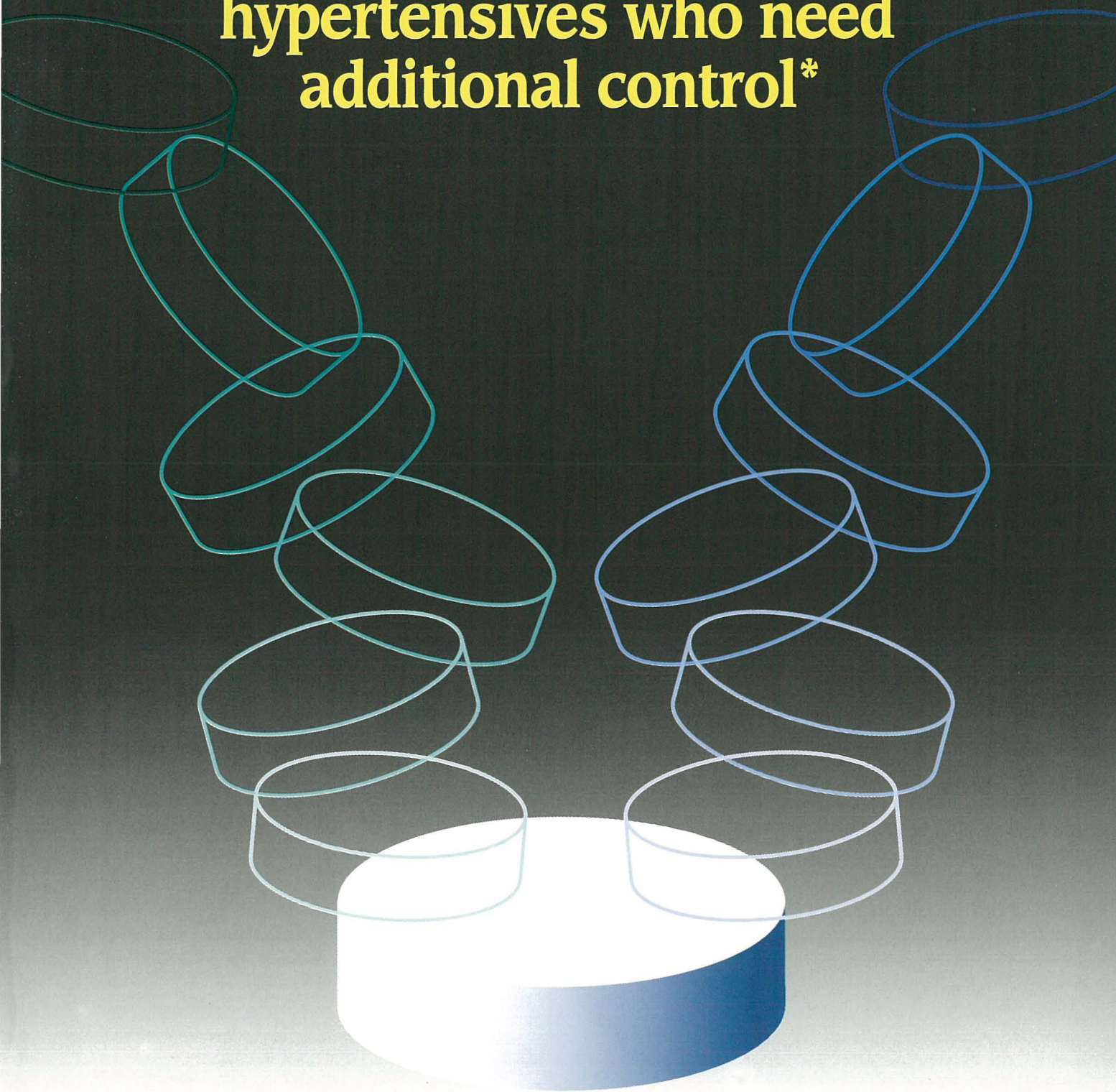

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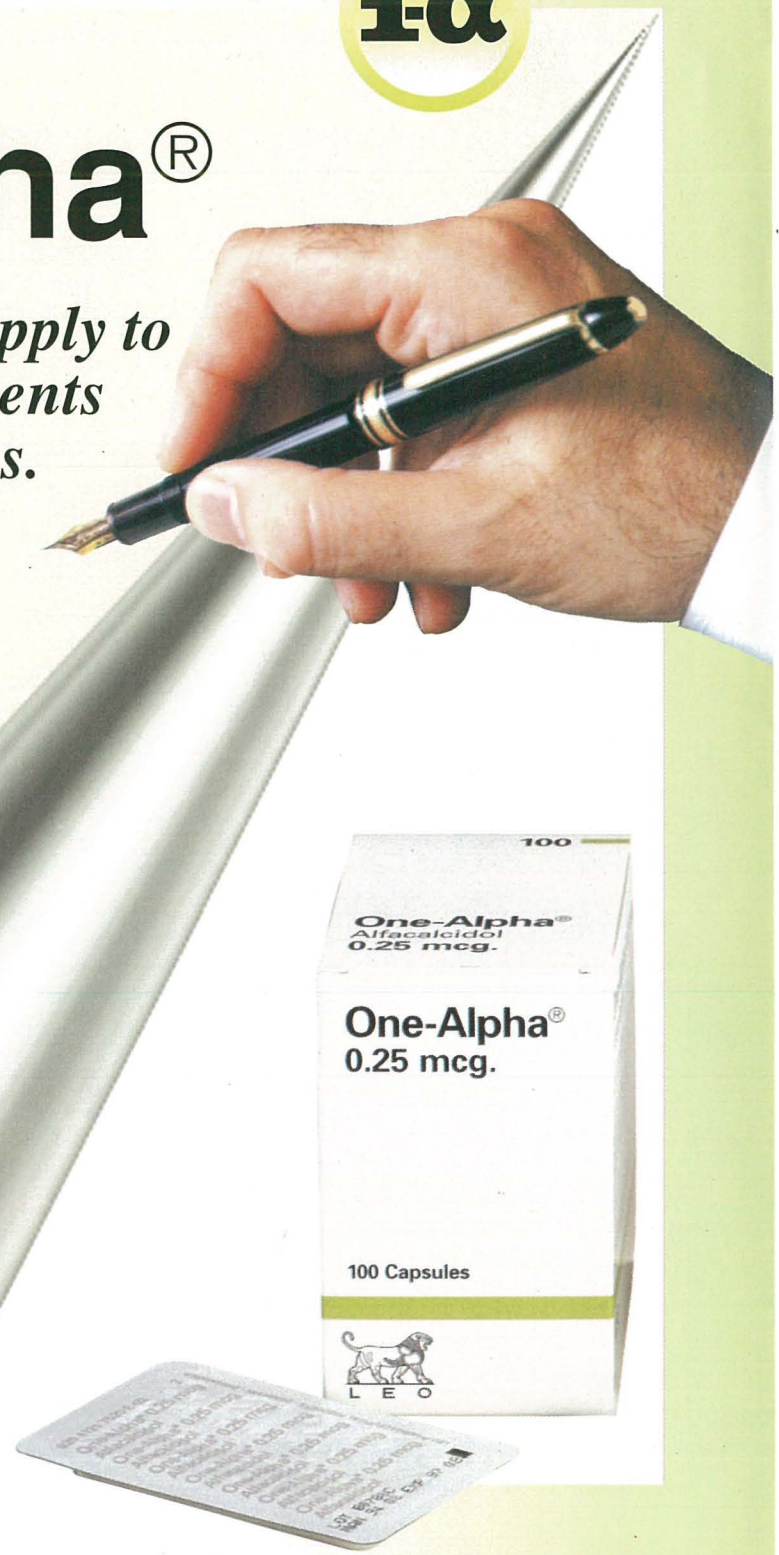

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neurotransmitters - research topic. (Genes which may be responsible for dyslexia have been identified).

(vii) Variation in the size of the right temporal area and corpus callosum detected on recent anatomical studies on post mortem specimens and on MRI / CAT images.

(viii) Research led by Dr.Albert Galaburda and Glenn Rosen of Harvard and Beth Israel Hospital in Boston, and written in the Proceedings of the National Academy of Sciences,[17] present evidence that may pinpoint a spot in the cortex where dyslexia originates. It is an area of tissue called the medial geniculate nucleus (MGN), which affects hearing by acting as a relay station for auditory signals. It was found that the size of the neurons in the MGN of dyslexics is smaller in the left hemisphere than it is in the right hemisphere, by a size differential of 10 - 15%. This may therefore be enough to throw off the brain's timing and disrupt its crucial word - processing skills.

(ix) Biochemical assymetry of the cerebellum indicates altered development of the organ and direct evidence of its involvement in dyslexic dysfunction.[18]

Clinical Picture:

- Dyslexia is a specific learning difficulty which results in a significant and persistent difficulty with reading, spelling, written prose and sometimes arithmetic.
- The child shows a marked discrepancy between his literacy skills and his achievements in other spheres.
- It occurs in spite of adequate teaching and is independent of socio - cultural background.
- Boys seem to be affected more severely than girls.
- In addition to difficulties with written language, the individual may also have difficulties with orientation, time, short term memory (auditory or visual), sequencing, auditory or visual perception and motor skills. Each individual will present a different pattern of difficulties, according to which areas are chiefly affected.
- Behavioural manifestations:
  Shy and withdrawn.
  Overactivity, with poor attention span.
  Severe attention deficit, with or without hyperactivity.
  Signs of emotional stress in persistently undiagnosed cases, which may take the form of disruptive behaviour or psychosomatic symptoms, loss of self - confidence and very low self - image.
  Development of school phobia.
- Motor manifestations:

  (a) *Gross motor:* 'clumsiness' - present with signs of an awkward gait or difficulty in kicking a ball, skipping or riding a bicycle.

  (b) *Fine motor:* poor fine motor control with poor handwriting (dysgraphia), and difficulty with buttons and shoelaces.
- Visual manifestations: poor visual perception and poor visual sequential memory will lead to difficulty with copying letters and words, reversal and inversion of letters and numbers and putting them in the wrong order, resulting in bizarre spelling.
- Problems with memory: poor sequential memory with difficulty in remembering the days of the week, months, alphabet and tables.
- Orientation difficulties: Difficulties with time, left / right orientation and numbers (dyscalculia).

Diagnostic pointers towards the recognition of dyslexia:

Early diagnosis is crucial to the treatment of dyslexia and other learning disabilities. It is difficult to recognise with certainty a learning disability before the age of 6 or 7 years.

All ages:
- Bright in some ways with a 'block' in others;
- Family history of similar difficulties;
- Difficulty in carrying out three instructions in sequence;
- Late in learning to talk, or speaking clearly.

Children aged 7 - 11 years:
- Continued mistakes in reading, and / or lack of reading comprehension;
- Strange spelling, perhaps with letters missed out or in the wrong order;
- Poor concentration span for reading and writing;
- Unusually clumsy and disorganised at home and at school;
- Difficulty in copying accurately from blackboard or textbook;
- Difficulty in remembering and processing oral instructions;
- Difficulty in understanding time and tense;

- Growing lack of self - confidence and increasing frustration;
- Trouble with sounds in words, e.g. poor sense of rhyme.

Children aged 12 and over:
- Tendency to read inaccurately, or without adequate comprehension;
- Inconsistent spelling;
- Difficulty with planning and writing essays;
- Tendency to confuse verbal instructions and telephone numbers;
- Severe difficulty in learning a foreign language;
- Low self- esteem;
- Difficulty with perception of language, e.g. following instructions, listening comprehension.

N.B. Not all dyslexic children will display all these characteristics.

Treatment:

The issue of learning disorders, including dyslexia, has become a matter of increasing personal and public concern. Inability to read and comprehend is a major obstacle to learning and may have far - reaching social and economic implications. Concern for the welfare of children with dyslexia and learning disabilities has led to a proliferation of diagnostic and remedial treatment procedures, many of which are controversial. This policy statement addresses these issues, which are of importance to affected individuals, their families, teachers, physicians, allied health personnel, and society.

A broad - based consensus of educators, psychologists, and medical specialists recommend that individuals with dyslexia or related learning disabilities should receive:

(1) early comprehensive educational, psychological, and medical assessment; and
(2) educational remediation combined with appropriate psychological and medical treatment.

Approximately 4% of school children are considered to suffer from dyslexia to a degree severe enough to warrant individual help. The main focus of treatment should be on the specific learning problems of affected individuals. The usual course is to modify teaching methods and the educational environment to meet the specific needs of the individual with dyslexia. Without appropriate help and teaching a dyslexic child will fail to reach his / her intellectual potential, while the early recognition of the child's difficulty is vital to prevent complicated consequences such as in cases of secondary emotional problems, lack of confidence and disordered self-esteem.

Structured 'multisensory' (or 'intersensory') teaching is usually recommended. Teaching through all the combinations of sensory and motor channels facilitates integration along the relevant neurological pathways. This encourages and reinforces efficient processing and integration of visual and auditory symbols, resulting in improved learning and linguistic skills.

The Medical Role in the management of Dyslexia:

1. Liaison, with appropriate referral at each stage: An important and useful role for the doctor or health visitor is to act as co - ordinator between the parents and the various people concerned.

2. Early recognition of the **Pre - School Child** 'at risk' of developing Specific Learning Difficulties:

Doctors and health visitors doing regular developmental screening are in the unique position of seeing all pre - school children. They should be aware that there are certain early pointers in the development of the child who may be 'at risk' of experiencing subsequent specific learning difficulties. The screening would normally cover development in four major areas - gross motor, hearing speech and language, vision and fine motor, and social behaviour and play.

(a) The child with Speech and Language delay: The child who presents with delayed development of speech and language will require a multi - disciplinary assessment to exclude causes such as sharing loss, global retardation and social and emotional factors. The child diagnosed with specific language delay is seen by the speech therapist who not only helps the child with expressive speech, but also includes work on concentration and listening skills, sound - symbol relationship and sequencing, etc. This should help to alleviate some of the difficulties experienced during the early school years.

It has been found that many children with specific language delay, who speak normally by the time they go to school, subsequently have difficulty in learning to read and write.

Information regarding the pre - school language difficulty should be passed on to the school by the speech therapist or doctor. The teacher should be alerted to the possibility that the child might need early individualized teaching, if the child does not seem to be benefiting from the general classroom teaching.

(b) The child with Poor Hand / Eye Co - ordination and Visual Perceptual Problems:

Such a child seems to be developing normally in most areas but has marked difficulty in copying shapes with a pencil or brick patterns, etc. Such problems with eye / hand co - ordination and visual perception suspected by the doctor / health visitor, should be discussed with the educational psychologist, who in turn may suggest activities and games that the parents can play with their child in order to help to strengthen the areas of weakness before school entry. The child's subsequent progress can then be followed up, and further assessment conducted if necessary.

The importance of early recognition of the child 'at risk' is not to make the parents anxious, but rather that the speech therapist or teacher, health visitor or psychologist can recommend ways in which the parents can reduce the areas of developmental delay, before the child goes to school.

3. Early Recognition of the Child 'At Risk' at **School Entry (5 1/2 years):** The developmental screening test done as part of the routine medical school entry examination includes tests of fine and gross motor development, speech and language (including auditory discrimination and auditory sequential memory), behaviour and emotional development. This examination should pick out both the slow - learning child missed by the pre - school screening, and the seemingly bright child who shows signs of possible specific learning difficulty.

It is helpful for the doctor to discuss the child's weakness with the teacher, who in turn will give a little extra time and thought to such a child. If the child fails to make the expected progress, early referral and discussion between the appropriate professionals should be encouraged.

4. The **Older Child** Failing at School: The presence of psychosomatic symptoms, resulting from anxieties about school, together with unexpected failing in school work, should arouse the suspicion of the school doctor with regard to dyslexia, and the child should therefore be referred to the educational psychologist for assessment and diagnosis.

The Medical Assessment:

A proper history should be taken in which special attention is given to:

- any family history of dyslexia,
- a history suggestive of hypoxia or low apgar score at birth;
- a post mature low birth weight baby,
- the developmental history (noting especially delay in speech or hand / eye development),
- emotional / behavioural problems (noting whether these started before or after beginning to fail at school).

The medical examination should reveal any treatable conditions such as glue ear, allergies etc. Auditory discrimination and verbal hearing tests should be included as well as tests for poor gross and fine motor coordination.

Referrals:

With the increasing recognition of dyslexia as a genuine handicap, more of these children are being referred for assessment by psychologists, teachers and parents. Earlier referral and provision are now strongly recommended and a multidisciplinary approach to diagnosis and treatment involving educators, psychologists, and physicians, coordinated by paediatricians, is required.

All these children should be referred to an optician or optometrist for detection of refractive errors. Those between the ages of 7 and 12 years should also be referred to the ophthalmologist for the attention of the orthoptist who in turn will include reference eye and ocular fusion.

An audiological or ENT consultation would be appropriate in cases with a suspicion of recurrent hearing loss.

Gross Motor Development problems presenting with 'clumsiness' are recommended to the paediatric physiotherapist.

Fine Motor Co - ordination difficulties are seen by the Occupational Therapist or physiotherapist.

Speech and language development problems are dealt with the speech therapist.

A child with a history of extreme distractibility and over - activity possibly due to food allergies or sensitivities can be referred to a dietician, as it is often the food or drink which the child craves that causes the problem.

Cases of A.D.D. with or without hyperactivity should be referred to the paediatrician or psychiatrist with a special interest in this field.

The information from all the specialists should be collated and shared with the psychologists and all the teachers concerned with the child. A plan of action can then be drawn up.

Counselling:

It is very important that once the child is diagnosed as dyslexic, a proper and simple explanation should be give to both parents and child. They should be told that the child is of normal intelligence and that he has a genuine difficulty. They should also know that if the child is of good intelligence, has motivation and drive, has been diagnosed early, and has had support from school and at home, then, provided he can obtain appropriate specialised teaching, he should make good progress over a period of time.

The parents can be advised to:

- help the child to keep up his self- image;
- encourage hobbies and activities in which the child can succeed;
- keep up his interest in books and read him books of his own choosing;
- remember that the child will have to work twice as hard at school to achieve half as much as his peers, so he will be tired after school; there appropriate help with home work should be given;
- keep up regular contact and good relationship with his teachers;
- help the child select interesting and educational TV programmeds;
- check that the child is receiving appropriate teaching and technology.

The doctor can recommend the national voluntary association which the parents can contact if they wish, ie: the Malta Dyslexia Association.

Dyslexia - Hopes for the future:

The present awareness of dyslexia as a major disability in the process of learning in an increasing number of children, creates an ever difficult task in that the number of teachers available locally with appropriate training is still below the number required to make provision for the special educational needs of these children. It is time however to realise that these children should be taught by dyslexia - trained teachers.. Private tuition by qualified specialist teachers may also be available. On the other hand, units for children with dyslexia which are attached to ordinary schools, enable a child to receive appropriate help more sensibly, in the sense that the child can spend most of the day in the classroom with his peers and is only withdrawn for part of the day for specialised teaching in the unit. The unit teacher is able to advise the class and subject - teachers in the school, while the children benefit from meeting other children with the same difficulties, which helps relieve their frustration and feeling of isolation.

The unit acts as a teaching and resource centre for teachers from other schools. The unit also acts as a multi - disciplinary assessment centre where teachers, psychologists, doctors and other professionals in the Child Health Services can assess and share information on the children. Any subsequent therapies agreed upon can take place within the unit and be fitted into the child' s timetable, without his/her having to make visits outside school, thus avoiding time-loss or non - attendance. The unit should have a 'nominated' school doctor to whom and the teacher in charge can have access discuss any concerns about the child's health and also to simplify any medical referrals.

It is also hoped that the Health Authorities increase the recognition of the very important part played in the early recognition, assessment and management of these children by health care personnel.

It is also suggested that the future sees the development of '**Middle Tier Clinics**' where pre - school children showing only mild developmental or specific delays are assessed, after being referred by a G.P., health visitor, or speech therapist. These are children in the 'grey area', who would not seem to warrant a full multi - disciplinary assessment at a Child Development Assessment Unit (C.D.A.U.).

A Middle Tier Clinic would be run by an experienced clinical medical officer, who would carry out a full assessment, and make immediate referrals to a speech therapist, audiologist, psychologist, paediatrician, dietician, home teaching team etc., as appropriate. The aim of this is to strengthen any specific weakness even before the child starts attending school. The child will therefore be seen and referred quickly and the waiting list for the C.D.A.U. would be shortened. These clinics would hopefully identify children who may be 'at risk' of specific learning difficulties but who may well have otherwise been missed. Such an arrangement should enable a close liaison with the receiving nursery school and ensure a close follow up of the child's progress.

The child whose problem is recognised early and who receives appropriate teaching and support, both at home and at school, has every chance of overcoming his disability and should grow to adulthood without additional emotional traumas. Many of these children have considerable skills and talents in other spheres. These should be encouraged to the full. In addition, many develop qualities of determination and persistence which will serve them well throughout their lives.

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Enuresis: The Silent Epidemic

Mr. Chris Fearne
Paediatric Surgeon, St. Luke's Hospital

INTRODUCTION

It is estimated that between 10% and 15% of all children over 5 years of age wet themselves accidentally[1]. Whilst the vast majority of these children will cure themselves with time, about 2% have an anatomical anomaly which needs correcting. Even in the funcitonal cases treatment modalities exist to relieve symptoms and help keep the child dry.

Definitions:

Enuresis is defined as accidental wetting in children over 5 years of age. Primary enuresis occurs in children who have never been dry for a significant period of time. Secondary enuresis on the other hand occurs when the child starts wetting after a period of dryness of 6 months or more. Nocturnal - or night-time – enuresis is the commonest form and is 3 times commoner in boys. Daytime enuresis can occur alone, or together with nocturnal enuresis.

Causes:

The vast majority of children with enuresis have a functional anomaly. The commonest form of this is detrusor instability. The bladder wall in these cases has a tendency for spontaneous contraction resulting in urgency and wetting if the child does not go to the toilet at once.

Recent findings[2] have shown that many children with nocturnal enuresis have a relative lack of vasopressin secretion at night. Vasopressin, which decreases the volume of urine by allowing increased tubular reabsorption, is normally secreted in larger quantities at night. In some children with nocturnal enuresis, this night-time increase in the production of vasopression is absent.

It has also been postulated that there may be a genetic basis for enuresis[3]. In fact in many cases of primary enuresis there is a strong family history. The risk of a child having enuresis is over 70% if both parents were enuretic in childhood, 40% if one parent had enuresis and only 10% if neither parent was wet as a child.

In about 2% of enuretics there is an underlying anatomical basis for the wetting. This could vary from spinal problems leading to a neuropathic bladder, to posterior urethral valves, to an ectopic ureter. In other cases urological anomalies lead to urinary tract infections which in turn cause the enuresis.

Management:

An important part of the management of enuresis is to exclude an underlying anatomical problem. It is also wise to exclude less common condiitons such as childhood diabetes and diabetes insipidus. This usually involves testing the urine for infection, as well as for glucose and sodium. An ultrasound scan of the renal tract is indicated if there is suspicion of an ectopic ureter (this is often part of a duplex system) or if there is a proven urinary tract infection. An ectopic ureter is diagnosed as the source of enuresis by performing the Methylene Blue Test. This invovles filling the bladder with the dye and placing a pad in the child's underpants. If the resultant wetness is colourless then the urine must be coming from outside the bladder, i.e. from an ectopic ureter.

In cases of long standing enuresis or suspected spinal problems, urodynamic studies are essential to look for signs of a neuropathic bladder.

Treatment:

In most cases treatment is expectant once an anatomical abnormality has been excluded. Indeed spontaneous resolution occurs at the rate of 15% per annum.

However for those children, or families, to whom wetting is becoming distressing and stressful, one should advise pharmacological treatment to relieve symptoms until the child cures himself with time. Thus for daytime enuresis Oxybutanin is often effective in decreasing the frequency of accidents. For nocturnal enuretics, intranasal Desmopressin will help in about 70% of cases.

Bed wetting alarms and bladder training programmes have been advocated as being successful by many[4] but are often impractical and time consuming.

Of course if an anatomical problem is discovered this must be treated in its own right.

Conclusion:

Enuresis is a common condition in childhood. Because many parents of enuretic children were wetters themselves, many families tend to take a philosophical attitude towards the problem and suffer in silence. The general practitioner has a role in advising these families that treatment for keeping these children dry exists and is readily available.

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DR.THOMAS HODGKIN'S PERSONAL LINK WITH MALTA

The Editor,

Dr. C. Savona Ventura's note on the incidence and mortality of Hodgkin's Disease in Malta in the late 19th century (It-Tabib Tal-Familja June 1998 p24) is of interest to the epidemiologist but has no bearing on Thomas Hodgkin's personal connection with Malta during the 19th century (P. Cassar, Dr. Thomas Hodgkin; The Malta Connection, It-Tabib Tal-Familja December 1997 p.22). My paper was only concerned to highlight Hodgkin's historical link with Malta and not with the epicemiology of the disease, named after him, in our island. The fact that I alluded to the description in 1949 of the clinical, pathological and therapeutic features of the disease as being the earliest publicationsabout the disease in no way means that Maltese medical practitioners were not familiar with the occurance of the disease in our island prior to 1949. Unfortunately the medical practitioners (whose identities are unknown) who reported their cases to the Health Department for statistical purposes (1896-1910) did not publish any papers on the clinical and other features of the disease as observed by them. Had they done so they would have established their priority regarding the earliest published accounts of the disease in our island. We are therefore thankful to the two contemporary colleagues quoted by me for recording the existence of the disease in our time (1949).

Similarly Dr. C. Savona Ventura's epidemiological note is a welcome addition to our knowledge of the history of Hodgkin's Disease in our midst. May he continue to add to our incomplete annals of maltese medical history.

Paul Cassar

it-tabib tal-familja
Malta College of Family Doctors,
St. Philip's Hospital
Santa Venera,
Malta.

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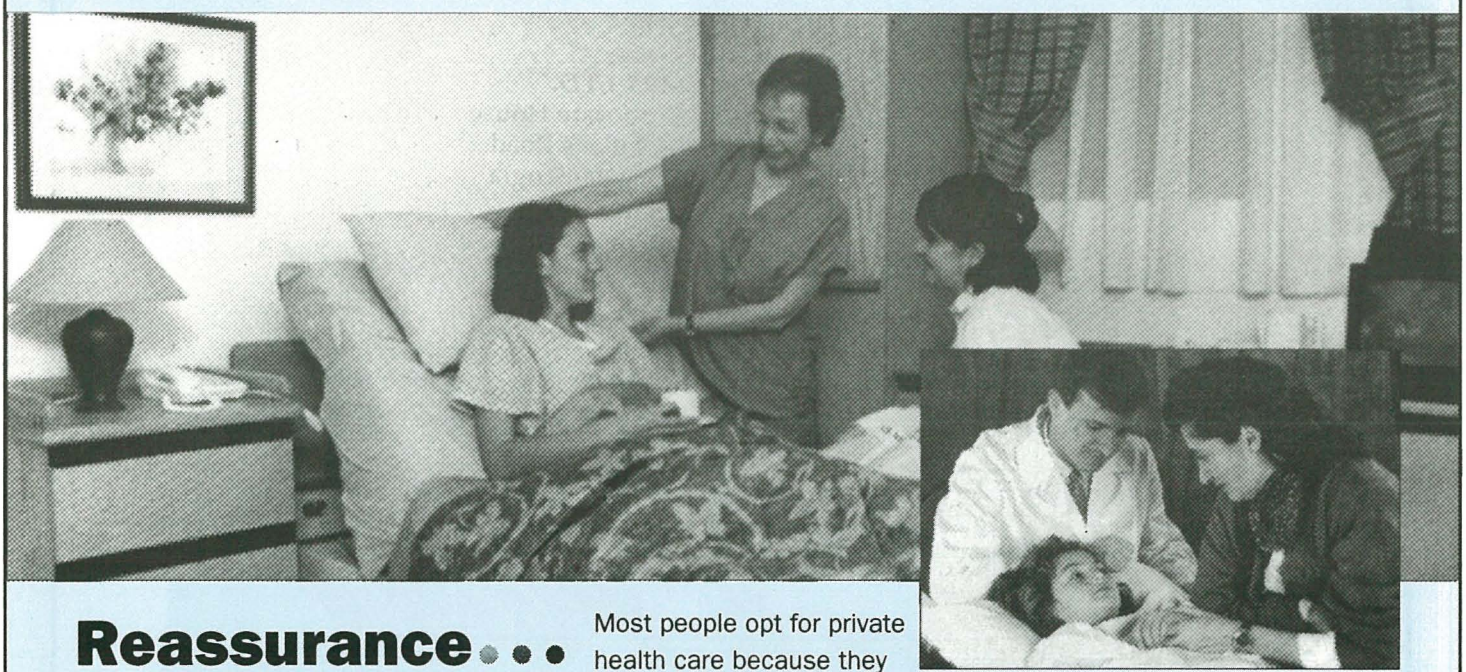

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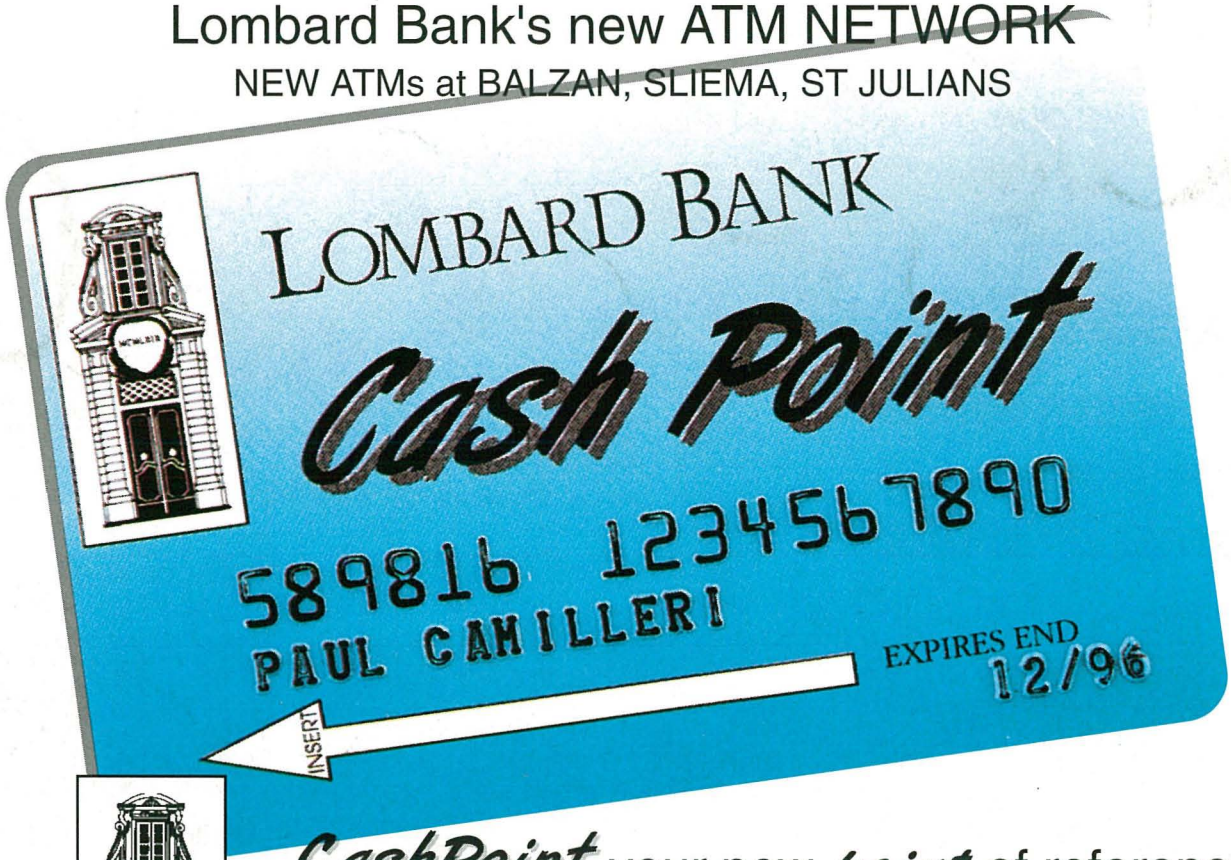

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