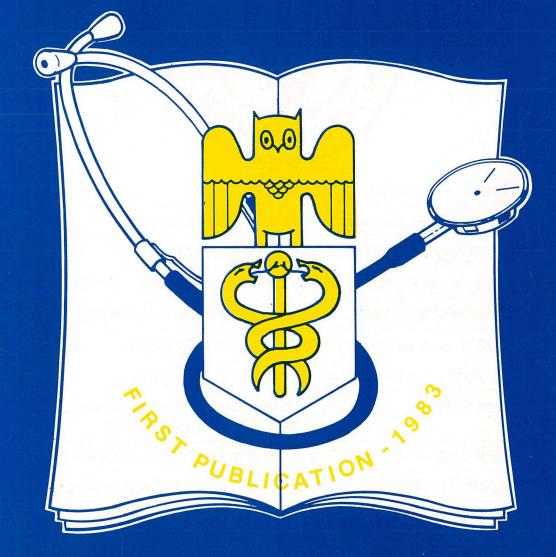


**ISSUE No. 10** 

**March 1987** 



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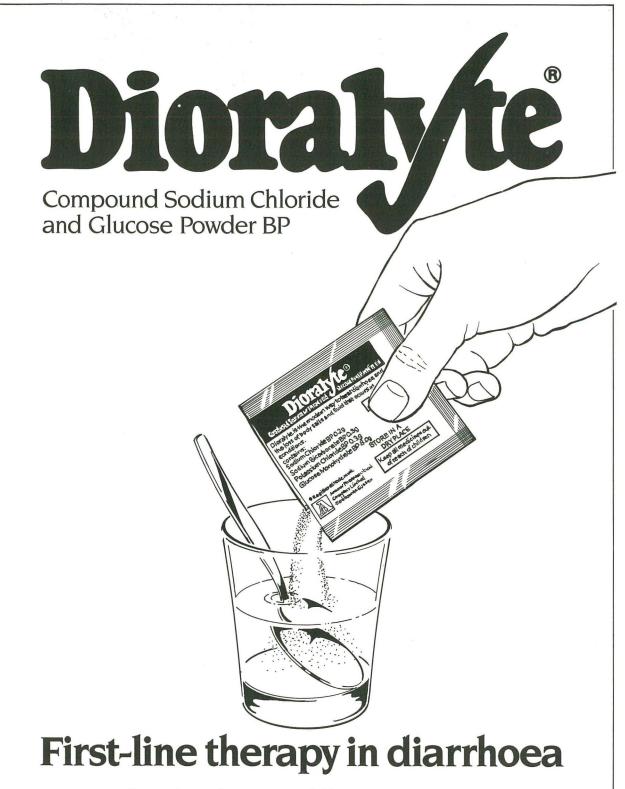
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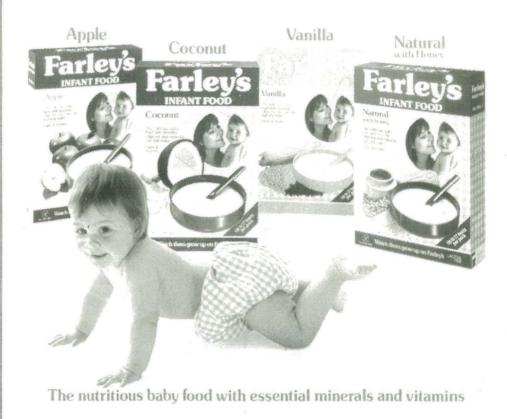
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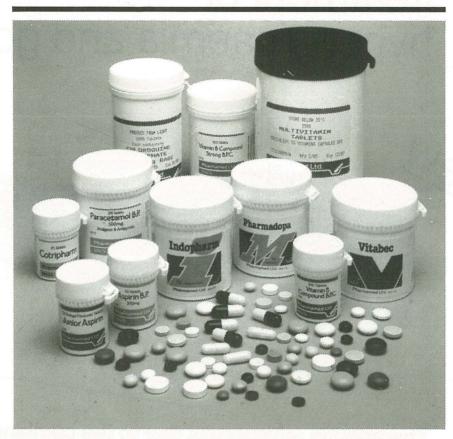


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Medi-Scope Issue No. 10 March, 1987

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Editor's Letter

he last few decades of this century are experiencing the havock created by a new disease known to all and saundry as AIDS - the Acquired Immune Deficiency Syndrome.

Caused by a retrovirus (the Human Immunodeficiency Virus-HIV formerly known as Lymphadenopathy-Associated Virus-LAV or Human T-Lymphotropic Virus type III-HTLV III), this disease is effecting an alarmingly increasing number of people world-wide. Its fatal outcome within a few years of infection has instilled great fears in the entire populations particularly as it is being recognised that susceptible people include those outside the riskgroups-homosexual/bisexual males, IV drug abusers and recipients of blood and its products e.g. haemophiliacs.

AIDS is basically a sexually transmitted disease although it has been transmitted through the use of infected syringes and needles, infected blood/blood products and sex-toys. Vaginal intercourse is just as risky as anal intercourse. There is no evidence to suggest, however, its transmission through saliva (e.g. kissing or sharing of drinking cups), toilet seats, social and classroom contacts. Infants born to HIV positive mothers acquire the virus during intra-uterine life, parturition or subsequent breast-feeding.

The disease appears to have originated in Central Africa with eventual spread to America and Europe. Until January 1987 there have been nearly 30,000 cases\* reported in the USA and over 630 cases\* in the UK. In Malta 5 lives\* have been claimed until December 1986 and at least 27 are known to be HIV positive (\*weekly Epidemiological Record No. 7, 13 February, 1987).

Albeit the fact that the disease is still incurable much can be done by medical personnel in the hope of dampening the ever increasing number of infected persons. Educating people about the nature of the disease and the adoption of preventive measures should contribute significantly towards this aim. Although fear itself may help in keeping this 20th century plague in check, it is a doctor's duty to manage individual cases not only medically but also by offering, tactfully, continuous psychological and moral support both to the patient and his immediate relations. "When frightened by the problem of AIDS we should not simply cross to the other side of the street. We have a responsibility and a duty towards the victims and we must not treat them as outcasts. The country must face this challenge as a united nation, preserving our humanity." (Mr. Norman Fowler, Secretary of State for Social Services, U.K.).

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### **Smoking and Health**

### A Statement of Concern from the Department of Medicine, University of Malta Medical School

There can be no reasonable doubt that smoking is now a major public health problem in Malta. The trends of smoking patterns among the Maltese are alarming. Much to the detriment of the health of the Maltese, there has been a steadily rising trend in smoking habits. This is especially evident among school-children, young adults and women in general. It can safely be said that among these groups, smoking has now reached epidemic proportions.

A recent survey among the Maltese population revealed that in the age-group 25 to 29 years, 60% of males and 45.7% of females smoked regularly. This trend is even more worrying when one considers Maltese school-children. In the age-group 14 to 16 years, 29.3% of boys and 14.9% of girls were selfdeclared smokers. In the majority of these, the age of starting smoking was 13 years, while boys tended to start smoking even earlier. This rapidly increasing addiction to tobacco in Malta is also mirrored by the rate of consumption of cigarettes. Over the years 1980-1982, there was a 15.5% rise in sales of cigarettes and in fact consumption in 1982 stood at a staggering 4.02kg per head. Moreover, unless curbed, these trends will continue to rise as tobacco is more insidiously addictive than heroin. At a population level, the consequences and the burdens that the Maltese health system will have to shoulder in the future will be massive and disastrous.

Addiction to tobacco is a most important and preventable cause of ill-health, severe disability and premature death among the Maltese. The harmful effects of smoking are many and they are not only restricted to smokers themselves but also to passive or 'second-hand' smokers.

Extensive and accurate scientific information supports the following main conclusions:

Smoking causes severe disability and shortens life: In both sexes and irrespective of the age of death, early deaths occur from coronary heart disease, cancer of the lung and chronic bronchitis.

Smoking affects the health of the unborn and the **newborn**: Smoking retards growth of the fetus and both the birth weight and size are reduced. Maternal smoking also increases the risk of miscarriages, still-births and early infant deaths.

Smoking by parents directly affects the health of their children: In the first year of life, chest infections are twice as common in infants whose parents smoke. Later on in childhood, both physical and intellectual development are slower in those whose parents smoke. Smoking directly affects the health of adult nonsmokers: In involuntary or 'second-hand' smokers, pre-existing disease is aggravated, particularly asthma and coronary heart disease. Furthermore, the spouses of persons who smoke have a higher risk of death from cancer of the lung.

### Recommendations

This department recommends that Government should accept the responsibility of carrying out more effective smoking control action and of stimulating non-governmental organisations to take action also. Such action should include the promotion of legislation for effective smoking control, the dissemination of information and the institution and support of activities to help people stop smoking. The general objectives should be to reduce the social acceptability of smoking and to ensure a smoke-free environment for non-smokers. The methods through which these objectives may be reached will have to be two-fold: education and legislation.

### Education

Anti-smoking health education should be regarded as part of general health education and the favourable aspects of non-smoking should be emphasised more than the unfavourable effects of smoking.

The health education of children starts early at home, in kindergartens and at primary schools. It should be re-inforced at different stages throughout the whole educational period.

Public information programmes should also emphasise the rights of non-smokers. In particular, children and pregnant women must be protected from involuntary exposure to tobacco smoke.

### Legislation

This may be seen as an index of Government concern as well as cutting out blatant encouragement to smoke. Legislation should be aimed to prohibit **all** forms of advertising and sales promotion of tobacco, including sponsorships of sports competitions, sportsmen, and raffled cars for good causes.

Packets of cigarettes should carry an effective health warning that smoking is dangerous to health. The form of this information should be varied periodically to ensure that it does not become stale.

Every tobacco packet should carry a product description to convey information about the tar, nicotine and carbon monoxide emission products.

### An Unusual Cause of Liver Abscess -A Case Report

PROF. A.J. PSAILA MD DCH(London) MRCP (UK) FRCP(E) PROFESSOR OF MEDICINE, A/DIRECTOR DEPARTMENT OF MEDICINE

> DR. TANCRED AGIUS MD HOUSE PHYSICIAN-SURGEON

A 65-year old male presented with a three week history of weakness, dyspnoea, dry cough and right sided lower chest pain. He had spikes of temperature of 100-102<sup>3</sup> F. The patient had been prescribed various antibiotics during this period but their effect was short - lived. His past medical history was unremarkable except that hypertension was diagnosed 8 years before and was well controlled on propranolol.

The patient had emigrated to Australia in his youth and had now returned for a holiday. His former occupation was as a pump-fitter. The patient smoked 20 cigarettes daily for the last 40 years and drank a bottle of wine every day.

### Physical Examination

On examination the patient's general condition appeared satisfactory. He was afebrile. The blood pressure was 110/70 and there were no signs of heart failure. Further examination revealed an area of dullness over the base of the right lung with decreased air entry, respiratory crepitations and expiratory rhonchi. In the abdomen a smooth, firm, non-tender 'liver edge was felt 4 finger breaths below the costal margin. There were no splenomegaly, ascites or lymphadenopathy; signs of hepatocellular failure were absent.

### **Basic Investigations**

A chest X-Ray showed increased lung markings over the right base. The ESR was 110 mm/1st hour

It must be ensured that tobacco products marketed in Malta do not contain higher levels of toxic substances than those marketed under the same brand designation in the country of origin.

It will be necessary to regularly revise levels of taxation on tobacco in order to discourage further cigarette consumption.

The rights of the non-smoker should be emphasised by adopting regulations to protect nonsmokers from exposure without their consent. Smoke-free areas must be provided or extended in public places and special attention must be given to protect infants, children and pregnant women from contact with persons who are smoking. and the white cell count  $14\times10^{9}/1$  predominantly neutrophils. Liver function tests showed an alkaline phosphatase and  $\mathcal{F}$ -GT at more than double the normal values. Serum bilirubin and ALT were normal. The haemoglobin, urea, creatinine and electrolytes were within the normal limits.

A provisional diagnosis of right sided chest infection was made and the patient given parenteral Ampicillin to which he responded well. He remained afebrile, the chest signs cleared and the chest X-ray features showed signs of improvement. Above all the patient felt well and was allowed to go home for the weekends while investigations regarding his hepatomegaly were being performed.

### **Further Investigations**

An ultrasound study of the upper abdomen showed a well encapsulated mass (9.4×7.8 cm) in the right lobe of the liver suggestive of hydatid disease. A Casoni test revealed a strongly positive immediate reaction but no delayed reaction. A C.T. Scan and hepatic angiography were interpreted by a radiologist as almost diagnostic of hydatid disease of the liver.

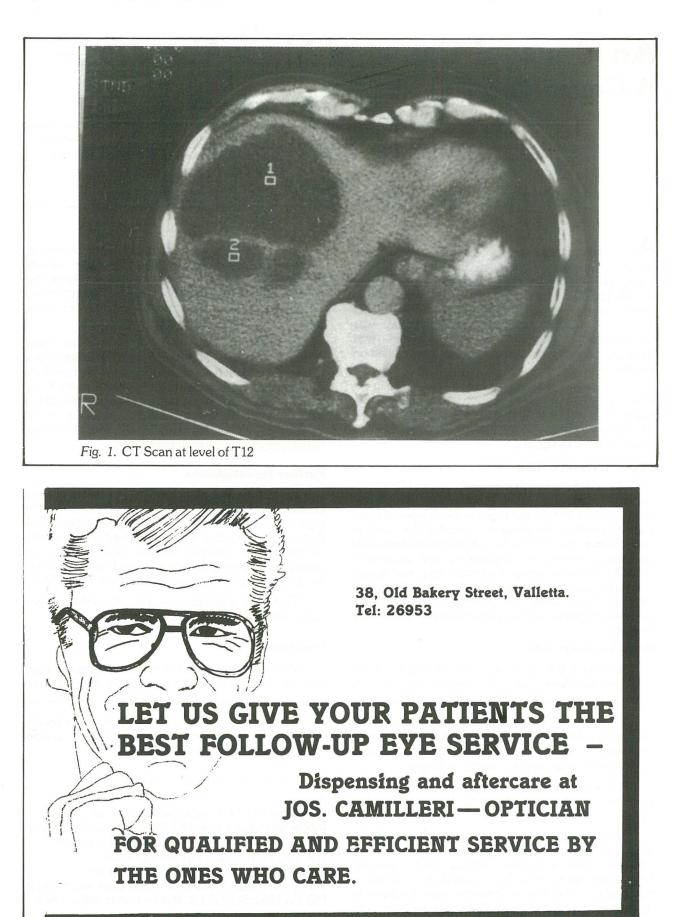
Tests for  $\alpha$ -fetoprotein and Australia antigen were negative.

### Progress

While these investigations were being carried out, the patient's condition varied from day to day. At

All health and most educational facilities should be declared smoke-free areas and mechanisms instituted to enforce these provisions.

Approved by: DR JOSEPH M. CACCIATTOLO MD MRCP PROF MICHAEL CAMILLERI MD MPhil MRCP FACP DR LOUIS C. BUHAGIAR MD MRCS MRCP DR VICTOR CAPTUR MD BSc FACC DR HERBERT M. LENICKER MD DCH PROF ANGELO J. PSAILA MD DCH MRCP FRCP DR PATRICK PULLICINO MD PhD MRCP FACP



times he felt very well. There were 3-4 day periodsin which the patient developed spikes of temperature (up to 102<sup>-</sup>F) with rigors and right sided lower chest pain felt mostly on deep inspiration and often radiating to the right shoulder. On physical examination the only positive finding was tenderness on deep palpation in the right subcostal area. The chest remained clear.

Repeated blood and urine cultures were negative as were serological tests for brucella, salmonella and rickettsiae. The white count reached  $15.5 \times 10^{9}$ /L (with 71% neutrophils). Liver function tests showed mild fluctuations of enzyme levels around the normal values.

After three weeks of variations in the patient's condition a right sided pleural effusion developed with rapid deterioration in the general condition. A laparotomy, previously judged undesirable in the light of the radiological findings, was performed.

### **Operative Findings**

The abdomen was opened through a right paramedian incision. A large intrahepatic collection of pus was found in the right lobe. A search for a primary cause for this liver abscess lead to the discovery of an inflammatory mass with multiple diverticula in the sigmoid colon. The hepatic abscess was drained, the inflammatory sigmoid mass resected and a temporary end-colostomy was performed.

Gram positive cocci including obligate and facultative anaerobes were cultured from the pus drained from the liver. During histological studies on the resected colon, a matchstick was found in one of the diverticula. The microscopic features were typical of diverticular disease with no evidence of malignancy.

#### **Post-operative Progress**

The patient's condition improved gradually. The temperature, ESR, LFT's and white blood count returned to normal. He was discharged 20 days after the operation. A re-anastomosis was planned for 3 months after.

### Abscesses of the Liver

There are three types of abscesses usually encountered in the liver.

1) Pyogenic Abscesses: The source of infection in these cases is usually either from a bile duct infection with ascending cholangitis or from a pylephlebitis resulting from any infectious process in the abdomen but especially from complicated diverticulitis. Less commonly hepatic abscesses are the result of a generalised septicaemia, a suppurating cholecystitis, penetrating peptic ulcer, subphrenic abscess or as a complication of trauma to the liver. Histologically these abscesses contain areas of hepatic cell necrosis surrounded by a white cell infiltrate. Eventually a fibrous capsule forms around the pus. Antibiotic treatment frequently results in a solid mass of inflammatory cells, dead hepatocytes and fibroblasts and may easily be confused with a tumour.

2) Amoebic Abscesses: This is a complication of amoebic dysentery but a clinical history of intestinal amoebiasis is not always present. Ulceration in the bowel wall allows the protozoa to reach the liver via the portal vein. Most amoebae lodge in the interlobular veins and degenerate while others invade the portal tracts leading to hepatic necrosis and eventually abscess formation. At first the abscess is solid with pus appearing later. This typically resembles anchovy paste. Occasionally amoebic abscesses become secondarily infected with pyogenic bacteria.

3) Hydatid Cysts: The liver is the most common site for these cysts but they may occur practically anywhere in the body. The causative organism is a tapeworm Echinococcus, the most common species being E. granulosus. The natural life cycle of this parasite involves sheep and dogs. Man is a secondary host and becomes infected by ingesting vegetables or water fouled by dogs or by handling parasite infested dogs. After ingestion the shell of the egg is destroyed by gastric acid and hatching occurs within the duodenum. The liberated embryos migrate through the gut wall, into the mesenteric circulation and lodge within the liver where each embryo is converted into a small vesicle which as it grows establishes a germinative epithelium eventually evolving brood cysts. As with amoebic abscesses secondary bacterial infection may occur in these custs.

An interesting point about this patient is that the root of his evil was the matchstick found in one of the diverticula. This appeared to have provoked the diverticular complication which spread to the liver and eventually caused the liver abscess. This matchstick must have been swallowed inadvertently with food, possibly even months before. It was a whole, used matchstick found anchored in a diverticulum. The patient did not have the habit of chewing matchsticks.

Prognosis following operation is good. Infact the patient continued to progress well, remained afebrile and had put on weight steadily 3 months after the operation. A foreign body reaction followed by stasis caused by the obstructing matchstick could be enough to start the process which could have had fatal consequences.

It is worthy to note the similarity to Hydatid disease. The patient coming from an endemic area, having a multilocular cystic swelling in the liver and having a positive Casoni Test left very little doubt about the diagnosis of Hydatid disease. In fact the laparotomy was intended to remove surgically the infected part of the liver - the best treatment for hydatid disease of the liver. At laparotomy the abscess of the liver was unlike the classical hydatid abscess and the inflammatory mass involving multiple diverticula and particularly the matchstick in one of the diverticula were certainly unexpected findings.

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### **Intestinal Fistulae**

An intestinal fistula can be defined as an abnormal communication between two epithelized surfaces, one or both forming part of the gut. Fistulas are primarily classified into:

- Internal connecting two hollow viscera or potential spaces
- External connecting hollow viscera to body surface.

External fistulae can furthermore be subdivided into:

Low output - less than 500ml of drainage High output - more than 500ml of drainage.

Although various studies have yielded different results it is generally agreed that mortality lies around 10-20%.

### Aetiology

1. CONGENITAL - these are rare and usually follow failure of normal embryological maturation. Eg tracheo-oesophageal fistula accompanying oesophageal atresia.

### MICHAEL A. BORG EX-MEDICAL STUDENT NEOGRADUATE

- 2. TRAUMATIC may follow diverse types of trauma including gunshot wounds, foreign bodies or even closed injury. Eg. retroperitoneal duodenal rupture.
- 3. INFLAMMATORY predominantly internal. May follow both septic as well as aseptic inflammation. Eg. post-T.B.
- NEOPLASTIC the great majority follow malignant neoplasms and arise as a result of invasion or obstruction with proximal perforation and abscess formation.
- 5. DEGENERATIVE usually develop on a background of senility. Eg. aorto-duodenal fistula.
- POST-IRRADIATION follow deep X-Ray therapy often in relation to gynaecological malignancies.
- 7. POST-OPERATIVE responsible for 95% of cases. Predisposing factors include tension on suture lines; ischaemia; sepsis; obstruction or involvement with malignant growths. They may also occur due to inadvertant bowel injury in connection with endoscopies.

		Sites of Intestinal Fistulas
Oesophagus	Congenital Acquired	<ul> <li>Tracheo-oesophageal fistula</li> <li>Carcinoma of oesophagus</li> <li>Pressure necrosis from NG tube</li> <li>Swollowed foreign bodies</li> <li>Post-pneumonectomy</li> </ul>
Stomach	Spontaneous	— Intra-gastric (neoplasms, ulcers) — Extra-gastric (colonic or pancreatic lesions) — Trauma
	Post operative	
Duodenum	External	— post Billroth II — duodenal ulcer
	Internal	<ul> <li>Duodenocolic (due to Ca colon, duodenal diverticuli, Crohn's)</li> <li>Duodenorenal (due to right nephrolithiasis or carcinoma)</li> <li>Duodenobiliary (following cholecystitis)</li> <li>Duodenovascular (after prostetic aortic grafts)</li> </ul>
Biliary	Spontaneous	— biliary calculus erosion — duodenal ulcer — biliary malignancy
	Post-operative	<ul> <li>— primary surgery</li> <li>— reconstructive surgery</li> </ul>
Bowel	Deliberate Spontaneous	<ul> <li>colostomies and ileostomies</li> <li>Crohn's disease</li> <li>Tuberculosis</li> <li>Diverticular disease</li> <li>Malignancy</li> </ul>
	Post-operative	<ul> <li>breakdown of intestinal anastomosis</li> <li>repair of traumatised bowel</li> <li>abscess formation</li> </ul>
Pancreas	Spontaneous	— pseudopancreatic cyst — Abscess
	Post-operative	— biopsy

### Diagnosis

Few problems are usually encountered in the diagnosis of external fistulae as the skin breach discharging bile, enteric contents or gas is usually clearly visible. In the occasional problematic case, oral markers such as carmine dye will definitely prove helpful. As in every clinical condition a thorough history and examination are essential paying particular attention to any predisposing abdominal pathology and/or previous operation. Internal fistulae often prove somewhat more difficult to diagnose as they tend to present with non-specific symptoms such as abdominal pain, diarrhoea etc. but the one universal complaint is weight loss. Investigations are essential adjuncts in diagnosis. Radiological studies in particular whether plain or using contrast are very useful to show:

- origin of fistula
- complexity and size of fistula track
- condition of G.I.T. from where fistula commences
- disruption of bowel
- prescence of distal obstruction.

Fistulography using contrast media may also be utilised. Ultrasound, CT scan, bacteriological examination or biopsy procedures may also prove useful. Laparoscopy or laparotomy may ultimately be resorted to in order to obtain the full diagnosis.

### Management

The currently adopted rationale of therapy is summerised in Table 2. **Resuscitation** should *not* be carried out using blood unless the fistula is connected to a blood vessel (e.g. aorto-duodenal) or the patient is severely anaemic. Neither is the use of plasma popular. The best way seems to be the administration of 500 - 1000 ml of Dextran 70 followed by Normal Saline.

Once resuscitation is complete full attention must be given to **Fluid and Electrolyte management**. A strict fluid balance chart as well as daily plasma electrolyte concentrations are required. An initial daily regimen for the adult patient would be:

5% Dextrose 200 ml N-Saline 500 ml Potassium Chloride 80 mmol Subsequent administrations must then be tailored to the particular person in the light of the electrolyte levels.

**Nutrition** of the patient with intestinal fistula is initially parenteral. A central venous catheter usually into the subclavian vein is set up. The basic idea is to provide calories, nitrogen compounds as well as vitamins and trace elements. Various proprietary products are available. Nitrogen is provided by means of amino-acid preparations. Initially 0.5g nitrogen/kg body wt./day may be started and subsequently long term management involves the calculation of the daily urea excretion in the urine.

Grms. nitrogen/day = 24hr urea excretion x  $\frac{100}{80}$  x  $\frac{28}{60}$ 

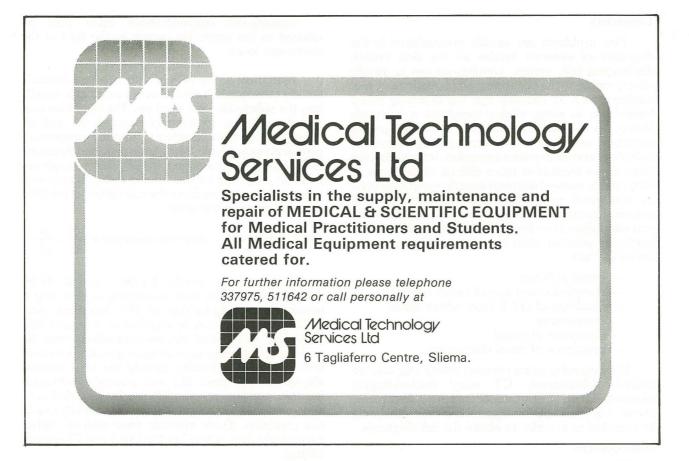
Daily calorie needs border around 40-50 kcal/kg/day. A large part is supplied as fats upto a maximum of 2-3g/kg/day of 10% Intralipid. Any additional calories can be supplied by means of 10% Dextrose. Daily blood glucose estimations must be undertaken and any signs of hyperglycaemia treated with short-acting Insulin. Initially the total volume should be low (about 2L) and gradually increased. Vitamin supplements should also be provided with additional doses of folate as this is particularly low in this condition. Trace minerals must also be added particularly Zinc (12mg/l of fluid lost) and Chromate ( $20\mu$ g).

Once parenteral nutrition is safely established, the patient's gastrointestinal function is reviewed with a view to using it for the provision of nutrients. This is normally in the form of low residue elemental diets of which several types are commercially available. However this may result in a number of problems including gastric stasis, diarrhoea, and hyperosmolar dehydration all of which must be looked for and corrected immediately. Should these prove troublesome the patient can be maintained solely on parenteral nutrition.

As soon as the nutritional needs have been satisfied, it is essential to ensure correct **protection of the skin** (in external fistulae), as well as collection of all fluid discharge. This is carried out efficiently by the use of an adhesive STOMA BAG. Besides protection

Table 2.	
Phase 1 (immediate)	<ul> <li>Restore blood volume</li> <li>Correct fluid and electrolyte imbalances</li> <li>Control fistula and protect skin</li> <li>Drain abscesses with/out antibiotic therapy</li> </ul>
Phase 2 (till day 2)	<ul> <li>Continue fluid &amp; electrolyte therapy</li> <li>begin parenteral nutrition</li> </ul>
Phase 3 (day 3-5)	<ul> <li>Start enteral feeding</li> <li>Demonstrate anatomy of fistula</li> </ul>
Phase 4 (day 5 -)	<ul> <li>Continue nutritional treatment until fistula closes or patient fit for surgery</li> <li>Operate to eliminate sepsis if necessary.</li> </ul>

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and collection of drainage, it improves patient comfort, facilitates early mobilisation and reduces the risk of cross-infection.

Surgery. One of the most debatable query as regards intestinal fistula management is when to stop conservative measures and operate. Although, until recently, thirty days following the control of sepsis has been more or less the baseline it is now felt that this is too short a time. This is especially the case in external fistulae showing signs of improvement such as reduction of discharge, weight gain and return of defaecation. In fact in a study of 27 cases of intestinal fistulae, Parsa et al reported a 100% spontaneous closure in uncomplicated external fistulae. Failure to close occurred in internal and external fistulae with exposed or everted bowel mucosa, very large opening or distal obstruction. The criteria for operative closure of external fistulae are that of failure to close on conservative therapy when investigations have revealed a reason for it. With internal fistulae, spontaneous closure is extremely unlikely although surgery is rarely urgent. Torrential diarrhoea with electrolyte and nutritional problems is one of the major indications for emergency surgery once the patient has been rendered fit for the operation.

Surgical treatment can be palliative or curative. Palliation includes drainage of abscesses, establishment of feeding enterostomies and proximal diversion. Curative operations aim at removing diseased bowel and associated fistulae usually followed by restorative anastomosis. In external fistulas in which failure to close is the result of an area of mucocutaneous fusion, a local dissection to separate the mucosa and infold it into two layers will suffice.

### Complications

In external fistulae failure to close is the major complication. Several reasons may be responsible including distal obstruction, discontinuity of bowel ends, chronic abscess or malnutrition. Other complications include metabolic and nutritional distruption, sepsis, pulmonary problems, deep vein thrombosis, gastrointestinal bleeding as well as psychological problems.

### **References:**

**Acknowledgement:** *I* would like to thank *Mr. A.* Zammit *MD* FA Chir for his help in the preparation of this manuscript.

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Rains and Ritchie. Bailey and Love's Short Practice of Surgery. London. Lewis. (1981).

### **Therapeutic Ultra-Sound**

MR. MARK SACCO SRP (Lond), Cert. Ed., Dip TP PHYSIOTHERAPY TUTOR SLH

The results of research, the use of sophisticated equipment and the contribution of electronics to the field of medicine have contributed in a big way towards a change in the management of various medical/surgical conditions, and especially so in Orthopaedic practice. In a general population whose pace of life is getting fast and whose degree of daily activity is increasing, the need of an efficient treatment that aims to return the patient back to his/her activities in as short a time as possible is felt. This is not possible in all instances but there is a great number of conditions such as sprains, bursitis, tendonitis, capsulitis, etc, that can be treated in such a way as to keep the patient mobile rather than restrict him to bed, chair or immobilise him in plaster! Obviously some other form of treatment in lieu of rest must be applied and this can take the form of ultra-sonic waves, heat energy, electric therapy etc. Of these, ultrasound plays a special role, and proof of this is the number of patients successfully referred to the physiotherapy department for ultrasound treatment. It was with this view in mind that I became attracted to this well documented account of the discovery of ultrasound, its physical properties, and its effects on the tissues, its indications and contra-indications. Every doctor who refers a patient for ultrasonic therapy and every physiotherapist that applies the treatment must be conversant with its properties, indications and contra-indications. Those who are not, are well advised to read this article carefully.

CHARLES J. GRIXTI MD FRCS CONSULTANT ORTHOPAEDIC SURGEON

Ultra-sound is defined as sound with a frequency above the audible limit of 20KHz. The frequency used for therapeutic ultra-sound ranges from 500KHz to 3MHz (500,000 cycles per second to 3 million cycles per second).

Way back in 1883 Galton used a whistle to measure the upper limit of the audible acoustic spectrum. Quartz transducers used to transmit and receive ultrasonic waves first came into use just after the first world war. Langerin used low frequency ultrasound propagation in water as a means of detecting submarines, and for communication. Early experiments using high intensity ultra-sound observed effects including the death of fish which swam through the beam, and painful sensations in the hand if placed in the beam. The fact that effects were obtained in tissues led to the use of ultra-sound in the modification of tissues, both in surgical and therapeutic applications.

### Generation of Ultra-sound

A device which converts energy from one form to another is known as a transducer. For medical purposes, ultra-sound is usually produced by a piezoelectric material. Crystals of some materials have the property that, when a voltage is applied across them, they change shape, and conversely, when they are compressed, a voltage develops across them. This is the piezo-electric effect. The most commonly occurring piezo-electric material is quartz, but artificially made ceramics such as lead zirconate titanate or barium titanate are now being used in therapeutic ultra-sound.

A rapidly varying sinusoidal voltage is applied across the flat surface of the ceramic. Thus the ceramic expands and contracts with simple harmonic motion. As the transducer face moves forward, the medium in which it is immersed, is compressed and as it moves back, a rarefaction is created.

### Transmission of Ultra-sound

This depends on the case and speed with which the media can be deformed and is indicated by the acoustic impedance of the material. Sound waves travel more easily through a medium with a high rather than a low characteristic impedance, for example more easily through steel than through water.

Ultra-sound energy is propagated in soft tissue as longitudinal mechanical waves in a directional beam whose shape depends upon the diameter of the transducer relative to the wavelength of the ultrasound in the tissue. These longitudinal waves will be converted to a transverse wave when something solid, like bone, lies in the path of the sound beam. For therapeutic purposes it is important to note that a transverse wave will not travel through fluids. When a sound wave encounters a different medium from the one in which it is travelling it may be reflected, refracted and/or absorbed or scattered.

### **Reflection**:

When an ultrasonic wave is incident on an interface between two different types of tissues some reflection will occur. The amount of reflection will depend on the characteristic acoustic impedance (z) of the tissues involved. (*z* is the product of the density of the medium and the sound velocity in that medium). The reflected beam interacts with the incident beam and this may lead to the formation of standing waves. This is important when irradiating over bone which has a considerably higher acoustic impedance than soft tissue. Standing waves may affect the flow of blood<sup>3</sup>.

### **Refraction**:

Waves may continue to travel through the new medium; if it strikes the new medium at a right angle it will continue to travel in a straight line, otherwise refraction occurs and this depends on the relative velocities of the two media. The greater the difference in velocities the greater the angle through which the wave bends.

### Absorption:

As the waves travel through any medium some are absorbed, resulting in a reduction in intensity and heat being produced.

The amount of absorption that is likely to take place in a tissue is characterised by this absorption coefficient. Tissues with a high collagen content absorb most strongly. Most soft tissues have similar absorption coefficients (0.5 dB cm<sup>-1</sup> MHz<sup>-1</sup>) but muscle has a slightly higher coefficient (1.5 dB cm<sup>-1</sup> MHz<sup>-1</sup>) and lung and skull bone have high absorption coefficients (20 dB cm<sup>-1</sup> MHz<sup>-1</sup>).<sup>8</sup>

The beam is reduced to half its intensity in a certain distance i.e. the *half value distance* and this depends on the nature of the medium and the frequency of the waves. In general, attenuation increases with rising frequency, thus, a 3 MHz beam will travel less than a 1 MHz beam. For example the half value distance for 1 MHz in air is 2.5mm, in water 1.5 m and in skin 40 mm.

Ultra-sound is rapidly attenuated in air, and only 0.1% of the incident energy is transmitted across the air/tissue interface. Thus ultra-sound is always applied via a coupling medium. This coupling agent must not absorb much ultra-sound and must provide a good acoustic match with the tissues so that reflection at the skin surface is minimised<sup>6</sup>.

### Scatter:

Most tissues contain numerous acoustic inhomogeneities. The incident ultrasonic beam thus suffers multiple reflections while being transmitted through the tissue. Some of these reflections carry energy out of the main beam. Thus the effect of scatter would be to diffuse the heating effect of the main beam.

#### Intensity:

This is the energy crossing a unit area in a unit time (watts per centimetre squared  $W \text{ cm}^2$ )

### **Ultrasonic Field**

This can be thought of as being composed of two distinct regions. The near field and the far field. Close to the transducer, in the near field or Freznel zone, the beam is mainly confined to a cylinder having the diameter as the transducer. The intensity within this zone varies considerably both along and across the beam and it is not until the far field or Fraunhofer zone that the intensity becomes regular without marked changes in intensity. The near field extends a distance  $r^2/$ from the transducer face, where r is the transducer radius and is the ultrasonic wavelength in that medium. In the far field the ultra-sound beam diverges, consequently the use of a smaller transducer or ultra-sound with a longer wavelength will lead to a less directional beam and inaccurate treatment. In therapeutic use the transducer is

typically 15mm in radius, therefore the extent of the near field in water with a 1 Mhz transducer will be 150mm.

As has been discussed above the intensity in the near field can be very *peaky*, and although the spatial average of intensity may be low, the peak intensity can be considerably higher. This is one reason why the treatment head must be kept moving during treatment (Hill, 1970). (See graph at the end of the article).

### Mode

The ultrasonic beam can be continuous or pulsed. For example 1:1; this exemplifies that the time ratio the ultra-sound is on is for 2 milliseconds and off for 2 milliseconds, with a resulting decrease in thermal effect<sup>7</sup>.

### Physiological and Therapeutic Effects of Ultra-sound.

Physiological effects at a chemical, histological level

- 1. There is a metabolic increase in the tissues<sup>2</sup>.
- 2. DNA synthesis is increased in cells, particularly fibroblasts.

3. Fibroblasts can increase their collagen production and the endoplasmic reticulum becomes swollen with newly formed collagen.

4. Polymerisation of collagen is held up, and the excess laying down of collagen is prevented in the isonated tissues.

5. Softening of a cement type substance which attaches fibres to the basement membrane in connective tissue.

6. Tissue regeneration/repair is speeded up.<sup>4</sup>
7. Decreased nerve conduction in C fibres giving an analgesic effect.<sup>1</sup>

8. Endothelial damage in the small blood vessels with little blister-like formations which are usually reversible.

9. Parathrombosis/Stasis; a therapeutic dose with a static transducer for 15 minutes will cause clumping together of the red blood cells at every half wavelength. The clumps are always surrounded by plasma but may cause blockage in small vessels and ischaemia. Occasionally the clumping and stasis is not reversible on cessation of ultra-sound. It occurs more readily in larger bore veins. This is another reason why the treatment head must be kept moving.

10. Increases fibroblast movement in the isonated tissues.

11. Myofibroblast activity is increased.

12. Fibroblast membrane permeability is increased. Substances synthesized can pass out more readily.

13. Lysosomal membrane fragility is increased with consequent release of proteolytic enzymes, thus the acute symptoms in an acute inflammation may be exacerbated by ultrasound.

14. Increases tensile strength and elasticity of the scar tissue.<sup>1</sup>

### Mechanical physiological effects

1. *Micro-massage:* the membranes and particles within the cell vibrate but not the whole cell.

2. *Micro-shaking* by means of the alternate compression and rarefaction the particles and membranes vibrate.

3. Acoustic streaming which causes increased membrane permeability. The microstreaming next to a membrane in damaged tissues causes increased absorption and diffusion of the exudate.

4. Acoustic radiation pressure: (the pushing force caused by the alternating compression and rarefaction). The positive pressure is followed by a negative pressure which never quite returns to the pre-pressure situation, and hence the idea that one can drive medication through the skin. A process known as *Phonophoresis*.

5. *Cavitation:* The alternating waves of high and reduced pressure causes the formation, growth and pulsation of gas or vapour filled voids. It can occur in body fluids, cell suspensions or in tissues. Cavitations can occur in two ways: (a) Stable cavitations which are formed but remain intact for many cycles. (b) Transient cavitation, the bubbles grow suddenly and collapse under the changing pressure of the ultrasonic field, with a resulting huge, local rise in temperature. This will cause gross damage to cells and tissues. This will not occur if the transducer is kept moving.

6. *Heat:* The backwards and forwards movement of particles causes friction and this is converted into heat probably sub-threshold heat. Using pulsed beam, the heat generated is so minute that this is not sufficient for a therapeutic effect.

Summary of physiological effects

- Thermal
- Mechanical
- Chemical

#### Summary of therapeutic effects

- Absorption of extravasated tissue fluids
- Heating
- Tissue Repair
- Thinning/Softening of fibrotic tissue
- Analgesic
- Spasmolysis
- Phonophoresis

— Diagnostically. (Ultra-sound with a frequency of 1MHz can be used to diagnose certain early fractures. For example the metacarpals; which do not show up on X-Ray. The suspected site is isonated with continuous ultra-sound and should an ache be felt, then a fracture has to be kept in mind).

### Contra-indications to the use of ultra-sound

- a) eye, ear, ovaries, testes, abdominal organs and CNS
- b) pregnant uterus
- c) tumours
- d) thrombosis or phlebitis
- e) haemorrhage
- f) infection
- g) devitalised tissue following radiotherapy
- h) diminished peripheral circulation
- i) implants/arthroplasties
- i) intra-uterine device

k) cervical ganglion and vagus nerve if patient suffers from cardiac diseases.

### Precautions one should take prior to treatment

a) Diminished skin sensation - is not a contraindication so long as the operator has sound anatomical knowledge and is familiar with the apparatus. Extra care should always be taken with these kinds of patients and a skin sensation test should always be carried out.

b) Should a patient complain of a burning sensation which is compatible with raising of the periosteum then stop treatment and amend.

c) An ache or a strong, uncomfortable vibratory feeling is compatible with heating of the bone. STOP and amend treatment.

d) Damage to transducer due to inadequate coupling will result in shattering of the quartz or ceramic with no out-put of the apparatus. This is the easiest way that ultrasonic apparatus is damaged. The average cost of an ultrasonic transducer is Lm60.

e) Hearing-Aids should be removed.

f) Ultra-sound may interfer with a demand type of pacemaker.

g) The apparatus should be given time to rest as output intensities vary if the apparatus is used for long periods of time.

Common conditions in which Ultra-sound is used:

- sprains and strains
- tenosynovitis
- tendonitis and paratendonitis
- haematoma
- bursitis and epicondylitis
- capsulitis
- fascitis
- Dupuytren's contracture
- adhesions of scar tissue
  - venous ulcers
- minor fractures (disregard the fracture
- and treat the surrounding soft tissues).
- episiotomy scars
- surgical wounds

### Unusual conditions for ultra-sound treatment

 soreness after manipulations or mobilisations

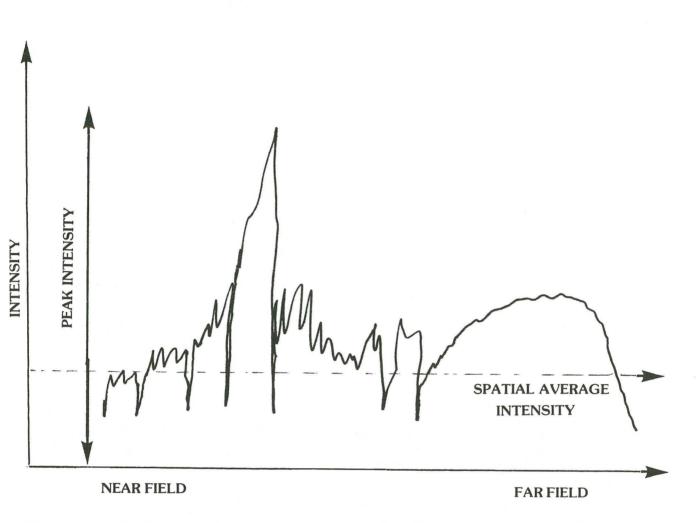


Fig. 1 Beam Profiles, illustrating significance of peak intensity and space average intensity in near and far fields.

- neuroma
- post herpatic pain
- breast engorgement

### **Case Report**

### History:

G.F., a 33 year old female from Mqabba was referred to the physiotherapy department on the 26.12.85 with right Longhead of Biceps Tendonitis.

On examination, the patient presented with generalised pain on flexion of the shoulder joint, a localised tender spot under the right acromion process and limitation of medial rotation. The patient's major complaint was the pain in the right shoulder as she came to clean windows. The patient was treated solely by means of ultra-sound. On her first appointment 27-12-85 she was given ultra-sound at an intensity of 0.5 W/cm<sup>2</sup> for  $1^{1}/_{2}$  minutes, with a pulsed mode for pain relief using a rank sonacel ultrasound unit with a frequency of 3 MHz. Following this she was treated daily for twelve other consecutive treatments using the same apparatus but an increased dose of 0.8 W/cm<sup>2</sup>. It is also interesting to note that the

patient did not rest the shoulder but continued to carry out her daily chores. She was discharged on the 14-1-86 with no pain in the shoulder region, full range of movement at this joint and functionally able to carry out her occupation without any hindrance.

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## Neurological Aspects of Diving

DR. PATRICK PULLICINO MD PhD MRCP FACP

The nervous system is exposed to many unfamiliar sensations in underwater diving, both from the external environment as well as from the body's internal environment.

The underwater environment of a diver is very different from normal: the individual is surrounded by a low-gravity water environment, and the sense of touch is often dulled by a wet-suit and gloves. Sounds are strange and give little indication of direction or distance. The vestibular apparatus has to deal with continually changing body positions in three dimensions, and little sensation of gravity. Visual input is often distorted, reduced or even absent in low visibility diving.

In addition to these sensations that can largely be anticipated, unexpected sensations can also arise if the function of the nervous system (e.g. the vestibular apparatus) is upset by changes in the surrounding pressure or temperature.

The internal environment of the body is altered in diving because of the effects of breathing gases under pressure. Oxygen, nitrogen and the other gases in air are dissolved in the blood and tissues in larger amounts at increasing depths. The extra nitrogen may disturb normal brain activity. This effect should be anticipated on any deep dive. An effect of nitrogen that is not however usually anticipated is that of decompression sickness, when too rapid a return of the diver to the surface causes the extra nitrogen in solution to form bubbles within the tissues of the body.

### Vertigo

Vertigo is a relatively common symptom in diving. The vestibular apparatus in the inner ears work in tandem and the sensation of vertigo may result if the input to the brain from one vestibular apparatus does not match up with that from the opposite side. Vertigo is a sensation of motion of the environment in relation to the body (or vice-versa) and may be experienced as spinning, falling backwards or forwards, or rocking. It must be differentiated from the vaguer sensation of dizziness, which is less specific. Vertigo is a hazard in a diver as it may not only affect his overall performance and cause him to lose his orientation, but it may also be accompanied by nausea and vomiting.

The commonest cause of vertigo in a diver was described by Lundgren<sup>4</sup> and called *alternobaric* 

*vertigo*. It is due to asymmetrical middle ear pressure equilibration during descent or ascent - a frequent problem, particularly among beginners, who have trouble clearing their ears.

Previous disease of the vestibular apparatus which is no longer symptomatic may also cause vertigo during diving. The nervous system can compensate for previous damage to the vestibular apparatus by visual and sensory information. When this compensatory information is not available as when diving, vertigo may result. People with a previous history of vertigo must be screened carefully before they dive.

Apart from these causes, some individuals have one vestibular apparatus inherently more sensitive than the other to changes of temperature and position and these people are prone to develop vertigo when they dive. Vertigo may also be a symptom of nitrogen narcosis or decompression sickness.

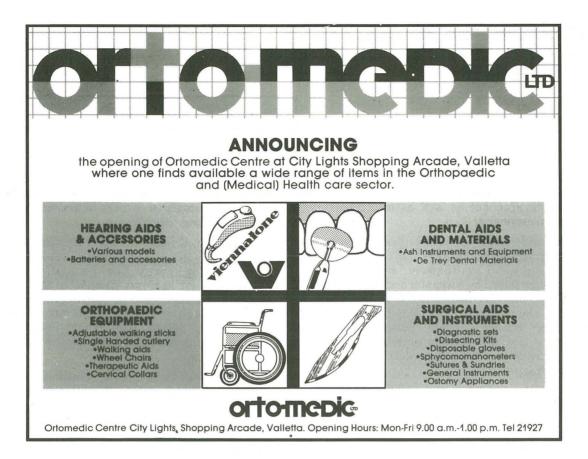
#### Nitrogen Narcosis

Under pressure, nitrogen takes on the properties of an anaesthetic gas. The narcosis it causes is due to its increased fat solubility at high pressures which causes impairment of transmission of impulses at brain synapses. The increased pressure of nitrogen at a depth of 300 feet is sufficient to render a man unconscious. At lower pressures nitrogen causes "narcosis", which is a state that is similar in many ways to alcohol intoxication.

Symptoms start at about 100 feet with a feeling of light-headedness and euphoria. This is combined with a slowing of higher mental functions and a disturbance of short-term memory. The symptoms get increasingly severe as depth increases. Recovery is rapid on returning to the surface but there may be some loss of memory for events during the period of narcosis. Alcohol, hangover or stress may make narcosis worse<sup>1</sup>.

### **Decompression Sickness**

The nervous system is involved in up to 35% of severe cases of decompression sickness, and these are the most serious, as a permanent deficit such as paraplegia may result<sup>5</sup><sup>2</sup>. Symptoms may develop immediately on decompression or may be delayed for up to 15 hours after a dive. Obvious symptoms of nervous system involvement may be preceeded and



overshadowed by symptoms such as limb or muscle pains; a combination of shortness of breath, chest pain and cough called *the chokes*; or just a general sensation of fatigue and feeling unwell. It is important that neurological involvement, which often develops slowly and insidiously, is not ruled out until a careful history and examination are performed, as it is often overlooked. Almost any neurological symptom can be produced ranging from headache and blurring of vision to convulsions and coma, so it is important that any unusual complaints following a deep dive are taken seriously.

The most common site of damage is the spinal cord which is involved in 80% of cases with neurological symptoms<sup>3</sup>. This usually presents with sensory symptoms such as pins and needles in the feet coupled with a slight weakness and unsteadiness of the legs. This may slowly or rapidly lead to complete paralysis of the legs and loss of control of bladder and bowels.

The treatment of decompression sickness is recompression in a decompression chamber as soon as possible. The longer the delay in starting treatment, the more likely is the patient to suffer permanent disability.

### **High Pressure Nervous Syndrome**

Very deep diving (with oxygen-helium to avoid nitrogen narcosis) to depths greater than 500 feet can produce a syndrome consisting of tremors of the arms and legs, myoclonic jerks, fatigue and even convulsions. The cause of the High Pressure Nervous Syndrome is unkown but it is thought to be a direct effect of high pressure on the nerve cell membranes. Much research is being done on this syndrome as it causes problems for very deep diving and sets limits to depths which divers can attain and at which they can work efficiently.

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# Amoxie The thoroughbred antibiotic

### **Prescribing Information**

AMOXIL is a broad spectrum antibiotic suitable for a wide range of infections caused by susceptible organisms.

### Indications

### Respiratory, ENT, genito-urinary and skin and soft tissue infections. **Dosage**

Children: Oral and injectable – up to 2 years: 62.5mg-125mg every 8 hours. 2-10 years: 125mg-250mg every 8 hours. Based on bodyweight (including neonates) 35-100mg/kg/day. Adults: Oral – 250mg-500mg every 8 hours. Injectable – I.M. 250-500mg every 8 hours or more frequently if necessary. IV. 500mg-2g every 4-6 hours. (Doses in excess of 1g should be given by infusion over 30 minutes).

#### Presentations

Capsules: maroon and gold capsules, each containing 250mg or 500mg amoxycillin.

Syrup: 125mg amoxycillin per 5ml in 60ml or 100ml bottles. Syrup Forte: 250mg amoxycillin per 5ml in 60ml or 100ml bottles. Paediatric drops: 125mg amoxycillin per 1.25ml in 10ml bottles with calibrated dropper.

Injection Viale containing 250mg or 500mg amountailling

### Precautions

Reduced dosage is required in patients with impaired renal function.

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### Contra-indications

Penicillin hypersensitivity.

### Side-effects

Side-effects, as with other penicillins, are usually of a mild and transitory nature; they may include diarrhoea, indigestion or an occasional rash, which may be either urticarial or erythematous: in either case it is advisable to discontinue treatment.



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(clavulanate-potentiated amoxycillin)

### **NEW SYRUP** PRESENTATION

### Augmentin

clavulanate-potentiated amoxycillin

100 ml syrup in powder form

156 mg/5 ml clavulanatepotentiated amoxycillin

Expiry date is d dry powder. Beecham Rest Laboratories. Brentford, Eogan Augmentin and

Each 5 ml contain amoxycillin as the trihydrate and 3 th clavulanic acidist potassium sat

Date of dispensi

#### PRESCRIBING INFORMATION Indications:

Chest, ear, nose, throat, genito-urinary, skin and soft tissue infections including those caused by B-lactamase producing

### organisms. Dosage:

Adults and children over 12 years one AUGMENTIN tablet (375mg) three times daily. Children 7-12 years 10ml AUGMENTIN syrup (312mg) three

times daily. Children 2-7 years 5ml AUGMENTIN syrup (156mg) three

times daily. Children 9 months – 2 years 2.5ml AUGMENTIN syrup (78mg) three times daily

In severe infections these dosages may be doubled. Treatment should not be extended beyond 14 days without review.

### Contra-indication: Penicillin hypersensitivity. Precautions:

Safety in human pregnancy is yet to be established. Oral dosage need not be reduced in patients with renal impairment unless dialysis is required Side-effects:

Uncommon, mainly mild and transitory, eg diarrhoea, indigestion, nausea, vomiting, candidiasis, urticarial and morbilliform rashes.

If gastro-intestinal side-effects do occur they may be reduced by taking AUGMENTIN at the start of meals.

Presentations: 375mg AUGMENTIN tablets each containing 250mg amoxycillin (1) and 125mg Clavulanic acid. (2) 156 25mg AUGMENTIN syrup. Powder for preparing fruit flavoured syrup. When dispensed each 5ml contains 125mg

amoxycillin (1) and 31.25mg clavulanic acid. (2) (1) as the trihydrate. (2) as the potassium salt. Not all presentations are available in every country.

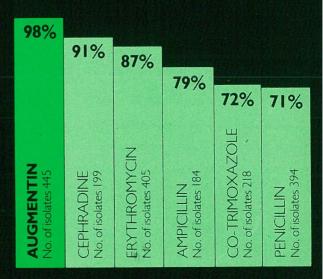
## A CHANGE FOR THE BETTER IN CHILDREN'S INFECTIONS

### **Broader in Spectrum**

- A broader spectrum than ampicillin, erythromycin, co-trimoxazole and oral cephalosporins
- Excellent activity against Haemophilus influenzae, Strep. pneumoniae<sup>1,2</sup> and Branhamella catarrhalis<sup>3,4</sup>

# Outstanding in Practice

- Rapid relief from symptoms
- Excellent success rates in ear, nose and throat infections<sup>6,7,8,9,10</sup>
- Well tolerated, with a low incidence of side effects<sup>6,11</sup>



(General Practice isolates from Ear, Nose & Throat infection collected during 1979-80)<sup>5</sup>

Paediatric infections (and no. of assessable patients)	Favourable Response	% Clinical Success
Otitis Media, <sup>9</sup> (133)	129	97%
Tonsillitis Pharyngitis <sup>9</sup> (109)	106	97%
Bronchitis <sup>9</sup> (91)	85	93%
Urinary Tract <sup>9,12</sup> Infections (50)	48	96%

### AUGMENTIN A performance that sets it apart

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Further information is available from: Beecham Research Laboratories

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### **PRESCRIBING INFORMATION**

**Presentation** BACTROBAN ointment: A presentation of mupirocin 2% weight/weight in a white, translucent, water-soluble, polyethylene glycol base. Available in 15g tubes.

Activity BACTROBAN is a topical antibacterial agent, active against those organisms responsible for the majority of skin infections, e.g. Staphylococcus aureus, including methicillin-resistant strains, other staphylococci, and streptococci. It is also active against Gram-negative organisms such as Escherichia coli and Haemophilus influenzae. Indications Acute primary bacterial skin infections, e.g. impetigo and folliculitis.

**Dosage and Administration** Adults and children: BACTROBAN ointment should be applied to the affected area up to three times a day, for up to 10 days. The area may be covered with a dressing or occluded if desired. **Precautions** When BACTROBAN ointment is used on the face care should be taken to avoid the eyes. Polyethylene glycol can be absorbed from open wounds and damaged skin and is excreted by the kidneys. In common with other polyethylene glycol based ointments, BACTROBAN ointment should be used with caution if there is evidence of moderate or severe renal impairment. Use in Pregnancy: Studies in experimental animals have shown mupirocin to be without teratogenic effects. However, there is inadequate evidence of safety to recommend the use of BACTROBAN during pregnancy. **Contra-indications** Hypersensitivity to BACTROBAN or other ointments containing polyethylene glycols. BACTROBAN ointment formulation is not suitable for ophthalmic or intra-nasal use.

**Side Effects** During clinical studies some minor adverse effects, localised to the area of application, were seen such as burning, stinging and itching.



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# The Development of Speech and Expression

DR. C.J. BOFFA, BChD BPharm FICD PhD DENTAL SURGEON, PART-TIME LECTURER, DEPARTMENT OF HEALTH

Speech and language as a means of communication are marvellous qualities. Speech is used between man and man or on occasions, it is used in, what can be termed as, a collectivised speech. Speech is part and parcel of everyday life and we tend to take it forgranted. However, we can safely assume that in primitive man, speech and vocabulary was limited and rather crude. In the long history of man's development, his progress, though slow, has been remarkable; from a crude ape to an intelligent being in a million years or less, from hunter to agriculturist, from stone to metal user, to citizen in about twenty thousand years or so. That the primitive man who appeared some half-a-million years ago should have had within him the potentialities of civilization with all its achievements in various fields and cultures is an amazing thing.

There are various characteristics which differentiate man from animal. These include the power of thought - thinking that solves problems and difficulties and involves concepts, plans, ideas, reflection and a strong will to survive. We find only the simplest beginnings of any such faculties in animals, but man, from the start, plans and devises to improve his lot and his life. One very significant difference between man and animal is speech and the use of language. Many animals make signs to each other, uttering cries of warning and the like, but they do not use names for things and actions: they do not converse. There is the use of countless sophisticated tools and machinery devised for a purpose. Furthermore man lives in a community.

Over a period of perhaps half a million years or so, there has been an immense development in the brain of man as compared with the brain of animals. The difference is not merely quantitative, it is qualitative. There are new specialized structures, not merely more brain cells. The cortex, which exercises the central regulation of all actions, consists of a dense network of about 10,000 million nerve cells and their interconnecting branches. No new neurons (nerve cells) are ever added to those with which each human being is born. It is mainly in the new connections and patterns that accumulated knowledge resides. The larger (in comparison with other creatures) efficient brain offers increased possibilities of reacting positively in various ways, a greater capacity for developing talents and improving skills and knowledge. Thinking is the basis of speech and the human being is able to speak because he possesses not only the vocal mechanisms but the cortical accompaniment. If this is missing or injured, speech is impossible. As far as I know, no exact statistics are available, but I am informed that the percentage of those in Malta with serious speech defects is not high, and probably less than 0.4% (less than 1 per 250).

With the development of man, gradual organic changes passed into a new phase of operation - one which is shrouded in mystery, but as striking an innovation as the beginning of life itself. It is difficult to define how this came about, but probably the result of a number of factors acting together. Development no longer proceeded just by the transference of the physical genetic material - the hereditary genes and related matter, but also by the handing on and the development of ideas. Acquired skills and knowledge was passed on; at first it was transmitted in speech, then in writing, next in printed books and in our agevia the new means of communication. Speech and words were being reinforced by experience and by necessity. There must have gradually emerged in the stream of life a new element: the verbal inheritance of acquired experience and ideas.

To some extent, speech is a reaction to a particular environment as is skin colour or body shape; muscles tend to develop stronger in certain trades where more use is made of them. Speech probably developed further when communities came into existence, as speech was, as it still is, the medium through which human co-operation could be brought about. It also co-ordinates and correlates the diverse activities of men for the attainment of common ends. One may say that language is not always synonymous with race, it may start that way, but the more movement there is the less it will stay so.

The nerve is one of the most marvellous of creations in Life. Embodiment of sensitivity and directiveness, it will convey messages both of feeling from the outside world and of command to action in response. It is indeed remarkable how a nerve network evolves into a nerve system, with as its hub a ganglion that is best thought of, although not wholly accurately, comparable to an automatic telephone exchange.

It is impossible to claim full understanding of the extraordinary, intricate process of how Man acquired the unique faculty of expression and speech. Man represents the highest form of organisation of matter and energy that has ever appeared on Earth. Recognition of this kinship with the rest of the Universe is necessary for understanding him, but his unique essential nature is defined by qualities found

### ERRATA

The opening paragraph of the paper titled "The Development of Speech and Expression" by Dr. C.J. Boffa Consultant Dental Surgeon, should read as follows:

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Speech and language as a means of communication are marvellous qualities. Speech is used between man and man or on occasions, it is used in, what can be termed as, a collectivised speech. Speech is part and parcel of everyday life and we tend to take it forgranted. However, we can safely assume that in primitive man, speech and vocubulary was limited and rather crude. In the long history of man's development, his progress, though slow, has been remarkable; from a crude **man** to an intelligent being in a million years or less, from hunter to agriculturist, from stone to metal user, to citizen in about twenty thousand years or so. That the primitive man who appeared some half-a-million years ago should have had within him the potentialities of civilization with all its achievements in various fields and cultures is an amazing thing.

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nowhere else, although of course physiologically similar to those present in other living creatures, such as other mammals.

During the ascent of Man, his mind - mental activities and potential, emerged slowly but surely, with greater clarity and intensity and came to play a more important role in individual lives. Eventually it broke through to become a sort of driving force, a basis for further evolution, cultural besides genetic and physiological. It was to a considerable extent this breakthrough brought about by a combination of factors - automatic mechanism of natural selection, by an effort for survival and better nutrition, together with an underlying supernatural force, that Man owes his dominant position.

What we are looking at is mainly the physical skeleton of speech and expression and along with it may flow, so to speak, a mysterious paraphysical element which is only partly understood. Mist shrouds the corridors of the very distant past, while the human brain was slowly developing. Before it could be an apparatus for action, it had to be one of preparation. For that, quite specific areas are involved; for example intact frontal lobes. But more deeply, it depends on the long preparation during human childhood.

The study of man's brain and its faculties, including speech is a tantalizing subject. Such a study leads us into a fascinating field, so vast and difficult, that no one can fully explore. And here my glance goes backwards to merely two million years. I refer to the remains of an early man, brought to light during excavations by the famous Dr. L. Leakey and his team, from the lowest level of that fantastic fossil mine, Olduvai Gorge in Tanganyika, now known as Tanzania.

remains of this unusual man are The approximately 1.8 million years old and he was named Homo habilis (handyman). His face is still rather primitive, but the back teeth are narrower and the brain shows significant change: at 680 ml, it is half again larger than that of Homo africanus, who was another early man, although still only half as large as the average brain to-day. The brain and skull of Homo habilis is more developed and much more similar to that of modern man. Dr. Ralph Holloway of Columbia University and other brain experts see for the first time in the cast of the H. habilis brain, the so-called Bulge of Broca, which anatomically is essential to speech. Thus in the experts' view, this primitive man already had the neurological mechanism for at least rudimentary speech. Increased brain power could have meant not only the beginnings of speech but also the all important advantage of stone tools. Its jaw, though not identical shows several human characteristics.

Readers may be interested to know more about Broca's area. This region is situated towards the front of the left hemisphere of the brain and it co-ordinates the muscles of the mouth, throat and tongue as we speak. Another area situated more posteriorly, in the temporo-parietal region of the left hemisphere, known as Wernicke's area is responsible for the structure of

our knowledge. Wernicke's area receives information from the ears and the eyes and is located fairly close to an important area of the cerebral cortex which integrates and compares the incoming information from all the senses. There is a consensus of opinion that it is likely that sentences that issue from our lips have been organized according to grammatical form by the neural mechanisms in Wernicke's area, but the actual muscular movements necessary to produce the sounds are controlled by Broca's area. The left hemisphere is rather larger than the right and there is a detectable lump over the region that houses Broca's area. One other interesting aspect is that in some lefthanded people, the corresponding areas are located in the right hemisphere of the brain. Some have tried to trace the origins of language by looking for the imprint of Broca's area in ancient crania. However, the quest proved to be not so simple.

Dr. Jeffrey Laitman of the Mount Sinai School of Medicine believes that the degree to which the base of the skull is flexed or bent, is indicative of whether the larynx can move up and down. And since the bases of the early Homo skulls are only slightly flexed. Dr. Laitman believes that the full range of sounds was not possible. He suggests that full command of articulate speech did not likely develop until perhaps 300.000 to 400,000 years ago. This tallies with the opinion of Prof. J. Aitchison of Glasgow University. He had expressed the view that two million years ago, Australopithecus (also African) robustus and boisei were living an exceedingly primitive life, without articulate speech and only very poor tools. In my view, Humans, known as Homo sapiens, arose around 40,000 years ago, possibly earlier and a fairly refined distinctive form of language was on their lips.

What conclusions can we draw about the emergence of language in our hominid ancestors? As yet one can only conjecture, however, it is reasonable to assume that the process was a very gradual one. It is possible that a rudimentary form of verbal communication arose as long ago as a million and a half years ago. The study of human speech presents many problems of peculiar difficulty and certain aspects are subject to conjecture. It is not easy to form foolproof functional and taxonomic judgements on early man and the above mentioned scientists deserve a lot of credit for their observations. We cannot claim to understand completely the extraordinary intricate stages in the development of speech. To reach the present advanced state may have taken man one million years. The potential for normal boys and girls to increase their vocabulary is much greater than we can measure in IQ or aptitude tests. There is a strong tendency for growing children and even adolescents to try the words and expressions used by parents.

As a dental surgeon I have had the opportunity to notice what a vital organ the tongue is. It plays an important part (besides other functions) in keeping certain parts of our teeth clean, in swallowing, and in the origin of the spoken word. In order to produce speech one has to be able to stop one's tongue at many different positions in one's mouth. Dr. R. Fouts who studied this aspect, says that there are about fifty different positions and the *stop-stop-stop* process is very rapid. On reflection we realize how highly developed man's neurological system is. Although the main function of teeth is chewing, they are also important in speech and help one speak clearly. The shape of the dental arch and the way the teeth occlude can have an effect on speech sounds. Certain letters of the alphabet need the assistance of teeth to form proper sounds, while some others may not be pronounced fully correctly without the aid of teeth. Of course, teeth also contribute considerably to one's facial shape, form and expression.

The development of articulate speech is not an instinctive process. A child does not arrive in this world with an inborn predisposition to express himself in any particular language. One born of Maltesespeaking parents like us, will gradually learn to speak in a foreign tongue if brought up in an environment where another language is used.

Various anatomical hard and soft structures play a part in speech. It is interesting to reflect on this aspect. There are no special organs designed primarily for speech; by a process of adaptation and over a period of millions of years, man has learnt to make use of apparata which perform other functions, such as the respiratory system, jaws, tongue, lips and palate, the masticatory and facial muscles, etc. These are used primarily for breathing, swallowing, and mastication of food. The larynx evolved to act as a safety valve to guard against the entrance of saliva and food into the airways and the lungs and to regulate the inward and outward flow of air during respiration. Of course we know that it also plays an important part in speech.

Before a child can express his needs, say of hunger and thirst and, in due course, his thoughts in words, he must pass through various stages, during which he gradually acquires the ability to co-ordinate the movements of the various muscle groups, and eventually exhibits articulate speech. This period varies between a normal, intelligent child and a less fortunate one. Genetics, health and environment all play their part. The great majority of children, given the right conditions can learn to pronounce nearly all words during childhood. Speech is usually not established until towards the end of the second year of life, but the actual sounds used in speech are acquired much earlier.

### The Growth of Speech

Both the sensory impressions received and the neuromuscular control of the speech organs are concerned in the growth of speech. The nervous system of man is a marvellous, highly sophisticated structure by which one is able to react to various types of stimuli of the environment and other factors and which controls the activity of the other systems of the body so that the whole may act in harmony.

As this development proceeds, the child is able to make increasing use of the spoken sounds he hears around him. Both the sounds he hears from others

and those he makes himself become important in the process of speech development. Impulses arising in the sense organs such as the eyes or ears reach the brain by means of afferent or sensory nerve fibres, the most important being the auditory nerve. Tactual sensations resulting from contacts between various parts of the speech mechanism, tongue teeth, and lips and kinoesthetic sensations, or impressions of movement enable the child to distinguish the positions and movements of the particular parts of the speech mechanism being used in articulation. Through this system of sensory and motor nerve fibres with the association areas of the cerebrum, speech patterns are gradually built up, and co-ordination of the muscles concerned in speech ensues. These patterns form very definite speech habits, and any alteration in such habits or anatomical defects will influence speech adversely. For example in the case of cleft palate patients, faulty speech may develop because of the child's inability to produce sounds normally owing to the cleft palate and is not the result of auditory imperception or lesion in the afferent nervous pathway.

As the child grows, there is gradually a change from mere vowel sounds to the use of sounds which more nearly resemble language. Parallel with the development of speech there is soon a fairly rapid growth in understanding. Adult brains are not merely in constant chemical and physiological action, they are also small generators producing electrical impulses that make the brain thousands of times busier than a switchboard. Brainwave patterns are not fully understood, but they effect speech and expression considerably.

In a normal healthy individual, voice can be regulated. Meanwhile breathing must be kept up. To win better dominance over his environment and organise the community around him, that individual must be a well-integrated whole and capable of teaching or guiding others by means of his voice or his actions. Voice must have played a vital part in the story of very ancient man.

So man has been blessed with Voice. How is it produced in modern man? It is produced in the larvnx. or voicebox - a structure of cartilage in the throat that can be felt as the Adam's apple and forming part of the wind-pipe. Two thin bands of tissue are stretched across it, one on each side of the opening. The human voice is regulated by the nervous system and results from air being passed over the vocal cords while breathing out. Muscles contract and relax the vocal cords. When we make sounds, these muscles narrow the opening by pulling on the vocal cords. As air from the lungs passes through the larynx, the cords vibrate. The result is audible sound. (The cricket, which makes such a strident call which goes on for several hours, has strong cords and tissues in relation to its small size). The volume of the sound in Man depends on the force with which the lungs push up air, however, the pitch of the sound depends on the tension in the cords and their length. The tighter they are pulled, the higher is the frequency of sound

produced and vice-versa. Generally speaking, women have shorter vocal cords than men; that is why their voices are higher pitched. Men's voices are generally an octave lower than women's. Up to the age of puberty, boys and girls have larynges of the same size, and voices of nearly the same pitch. In boys, the larynx grows larger and the voice breaks.

The vocal cords can produce only simple sounds of varying pitch. Different vowel sounds are produced by varying the shape of the mouth cavity through the use of voluntary muscles and related oral tissues, including the tongue and lips. Consonants are formed by changing or interrupting the air flow through the throat and mouth.

### Conclusion

In the Mediterranean region, Man's life, including his speech and vocabulary, probably blossomed so to speak, in Neolithic and later times. In this living stream, we can imagine to some extent the emergence, conflict, rejection and survival of various ideas which occurred with the passage of time. Few other factors have effected history more than vocabulary and coordinated efforts among settled communities. However, without the evidence of writing we cannot tell exactly what were the languages of the paleolithic and neolithic peoples. We know little on how languages developed. Limited information can be derived from the fragments of early writings and drawings in ancient caves. During the Early Stone Age, man's vocabulary must have been rather scanty.

The development of languages poses many questions. What, for instance, constituted the original

languages of the world? How did they come about? Was it pure chance in the beginning? Shape of palate, mouth and jaw must have, to some extent, contributed to this end. In the dental profession, we notice that people with short upper lips are less likely to use the sound 'p' and some mouths find 's' and 'th' not easy to pronounce. However, there are other factors as yet unknown; probably several factors acting in various ways.

Neolithic Man must have been hardworking and energetic. This is evidence by the temples he built, such as Haġar Qim, Mnajdra, Tarxien, and Ġgantija. It is reasonable to assume that he was an imaginative and capable creature. With new power, he controlled to some extent his environment and adapted himself to it. He roved the countryside and valleys, hunted, reared animals and grew crops. He gave names to strange or unusual birds and animals which he had not seen before.

He had his mind and his skills to help him, his conscience to contend with and his imagination. The weather must have played an important part in his everyday life. He was also concerned with omens, diseases, life and death, and sacrifices to his gods. Life for Neolithic man was likely to have been a very difficult one, one of hard labour in order to survive. Challenges in everyday life must have spurred him on to greater efforts. He probably coined new words for new things and new crafts. With the ups and downs of life, new words and new expressions blossomed.

(a)	Dysphasia	(disorder in the use of symbols for communication whether spoken (motor) or heard (sensory)
(b)	Dysarthria	(disorder of articulation).
(c)	Dysphonia	(disorder of vocalization).
(d)	Dementia	(intellectual deterioration).
(e)	Certain types	of deep cleft palates - speech impairment.
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### Gastroenteritis in the Maltese Islands

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### WHO meeting on Diarrhoeal Diseases (1980)

I he thirty-first World Health Assembly of 1978 urged its member states through its resolution (WHA 31.44) to identify diarrhoeal diseases in the Mediterranean as a major priority area for necessary action. Towards this end a WHO Mediterranean Meeting was convened in Rome in April 1980; besides Malta, eight other Mediterranean countries participated in the meeting, and these were Algeria, Greece, Italy, Morocco, Spain, Tunisia, Turkey and Jugoslavia. The rendezvous was the Istituto Superiore di Sanita in Rome. Malta was represented by Dr. P. Cuschieri and Dr. A. Mifsud.

The main purpose of the meeting was to submit specific recommendations relating to the following:

- Epidemiological surveillance and exchange of information between National Health authorities and WHO.
- 2. The diagnosis and laboratory identification of pathogens, old and new.
- 3. Oral rehydration therapy.
- 4. Support for water and sanitation and related health education programmes.

The countries bordering on the Mediterranean basin are an attraction to tourists from the North mainly because of beach availability and warm climate; the latter, however, encourages microbial multiplication in food and beverages, and this in its turn favours food poisoning. On the other hand, quite a few tourists visit not one but several Mediterranean regions on their holiday round, and spread of infection from country to country is thereby facilitated. Although bordering on the same sea, the various holiday resorts vary in quite a few aspects, particularly in socio-economic development, cultural and educational patterns, food habits, standards of hygiene, sanitation and health services. In the larger countries, differences also exist between one region and another.

Although notification of disease is a pre-requisite to proper and adequate surveillance, it is difficult to ascertain to what extent these measures are adopted by member countries, in view of the detrimental effect this might have on the tourist industry, which always plays a major role in the national economic balance.

Commenting from the bacteriological point of view, Dr. Cuschieri reviewed the enteric pathogens which are common in Malta. Cholera has not appeared since the early 1900's, and this has been supported and confirmed by routine and periodic bacteriological investigations on local sewage and by stool cultures on visitors from overseas who suffer from a diarrhoeal disease.

Bacillary dysentery has been on the decline since 1970; *Shigella sonnei* predominates with an occasional *Shigella flexneri*. Four cases of typhoid fever were reported in 1979, and the consumption of raw food, particularly shellfish, has been incriminated more often than not over the past few years; paratyphoid fever is rare.

The inajority of Salmonella food poisonings were caused by *S. typhimurium* (55%), whilst *S. anatum* accounted for another 31%. Meat and chicken made-up dishes have been the more frequent culprits.

Enteropathogenic *E. coli* account for a minimal amount of diarrhoeal diseases in children. The development of techniques for the detection of Campylobacter and Yersinia is still under way, and it is envisaged that facilities would soon be available for the diagnosis of Rotavirus and Norwalk Agent.

With regards to the situation in Malta from the paediatric point of view, quoting data gathered from hospital records and from the Department of Statistics it was apparent that the vast majority of enteric infections are treated by the general practitioner at home by a variety of proprietary antidiarrhoeal agents and glucose-electrolyte mixture administered orally to replace milk feeds; traditionally, to one litre of sterilised water a tablespoonful of sugar and a teaspoonful of salt are added. With the onset of dehydration, the child is then referred to hospital for fluid replacement, usually in the form of quarter saline in 5% dextrose, and this is more commonly administered through a peripheral vein. Very rarely a cutdown is required when shock has supervened, and blood volume replacement would then also require blood or plasma.

Two pre-graduate medical students surveyed all the admissions of children under the age of ten years during 1979 into the paediatric wards of Karin Grech Hospital; a summary of the statistics prepared from this study follows. With a population of 57,000 children under the age of ten years in the Maltese Islands during 1979, an average of 1.1% required admission into hospital for control of a diarrhoeal disease. Of these, 50% were in the first year of life, and 25% in the first six months. The mean hospital stay was seven days, and intravenous fluid replacement was the rule for the first few days. Antibiotic therapy was guided by stool cultures and sensitivities, but was more usually limited to the more severe enteric infections. The majority of the cases were admitted between June and August (46%), with a nadir between March and May (14%). The mortality rate was among the lowest recorded at the Meeting, and estimated at 1.5 per thousand admission into hospital for diarrhoeal disease.

It is interesting to compare notes with the other participating Mediterranean countries. In Morocco, diarrhoeal disease have a peak incidence in August and mainly effect children under the age of two years; Salmonella is responsible for 72% of stool cultures, and lack of adequate sanitary hygiene plays a major role in pathogenesis. Bacillary dysentery is a problem Yugoslavia where it is reported to effect in 211/100.000 population. Rotavirus is the main offending organism in Spain, accounting for 50-60% of all diarrhoeas of infancy. In Tunisia, enteric infections are a major cause of referrals to hospital; infants between the ages of one and eleven months are mainly effected, and the mortality is extremely high, amounting to 24% of all admissions for diarrhoea. (17% of all deaths in infancy are caused by upper respiratory tract infections). Salmonella is responsible for 60% of cases, Shigella 25%, E. coli 10% and Vibrio cholera 3%. Epidemics come on in waves and also effect hospital populations; resistance to antibiotics is a feature, particularly with S. wien, which has reached Tunisia from Algeria. The cholera vibrio is also showing resistance to tetracycline in some countries like Bangladesh where this antibiotic has been gravely abused.

In Turkey, diarrhoeal diseases run into millions of cases per year, but only 4% of these are notified; this is partly owing to the fact that tourism is still the most important industry. Cholera cases are due to Vibrio El Tor which causes a much milder disease than classical cholera, and is associated with a low fatality rate.

Notification of diarrhoeal diseases is not obligatory in Greece. Typhoid fever is not a problem, and paratyphoid is practically non-existent. In Thessalonica, *Vibrio parahaemolyticus* thrives in shellfish and sea-water. Campylobacter is a problem with pig and chicken handlers. 47% of diarrhoeal diseases under the age of three years are caused by Rotaviruses, whereas Hepatitis A and B viruses are important pathogens in older children under 15. The infant mortality rate in diarrhoeal diseases is high, and ranges at 19 per thousand cases.

Typhoid is a major problem in Italy, effecting 9.2/100,000 population; Shigella is less common. Two recent outbreaks of cholera were attributed to the consumption of raw sea food. The 1973 cholera epidemic was a national disaster; it cut off 75% of the tourist industry, and in 1980 it is still 25% less than it was before the epidemic. The fatality rate of diarrhoeal diseases is very low in adults, but accounts for 5% of the infant mortality rate; the fatality rate in hospitalised cases is next to nil.

A few practical points of interest which came up

during these reports included the following listed hereunder:

- 1. Enteropathogenic E. coli is manifesting a decreasing incidence throughout the world.
- 2. S. anatum may be present in duck's eggs.
- 3. There is a constant increase of Salmonellosis in non-temperate climates, e.g. Germany, i.e. in countries not favouring growth and proliferation of pathogenic micro-organisms.
- 4. *V. cholera* has been cultured from steamed and boiled crabs in Louisiana.
- 5. *Campylobacter jejuni* is on the increase in Europe.
- Yersinia grows well at low temperatures and is a problem with refrigerated food and with veterinary surgeons.
- 7. S. wien and S. agana display a marked resistance to antibiotics.

Breast milk is abundant in antibodies, and my first recommendation at the Meeting was for the promotion of breast-feeding at least for the first six months of life. This was immediately seconded by Italy and Greece. My other recommendation was for discouraging the indiscriminate use of antibiotics, which more often than not cause more harm than good, particularly from superinfection by resistant bacteria and viruses. This too was recorded and adopted.

The commonest pathogens of infantile diarrhoeas are the Rotavirus and Norwalk-like virus (Norwalk agent; Hawaii agent; Ditchling agent; W agent; M.C. agent). Other viral entero-pathogens include Astrovirus, Calcivirus, adenovirus-like agent, Otofuke agent. Small round virus, corona virus, enterovirus and influenza virus. - Antibiotics are obviously contra-indicated. Antimicrobial therapy with bacterial diarrhoeas is controversial; with severe Shigellosis antibiotics with high blood levels are required - resistance to ampicillin is common. In severe cholera, tetracyclines reduce shedding, the duration and the final amount of diarrhoea. Resistance to this antibiotic is encountered in Bangladesh and some parts of Africa because of abuse. With typhoid fever, resistance to chloramphenicol is occasionally encountered.

There exist a variety of drugs which reduce secretory diarrhoea: WHO recommends that they be used with extreme caution if at all. Aspirin and Indomethacin are prostaglandin inhibitors, and it is believed that these prostglandins are important in the pathogenesis of diarrhoea; these drugs may act by influencing the chloride in the fluid output into the gut lumen. Chlorpromazine is another drug used against diarrhoea, but it may aggravate hypotension. Cyclohexamine is a poison and inhibits protein synthesis. Ethacrynic acid paradoxically is an antidiarrhoeal but is also a powerful diuretic and therefore potentially dangerous. Nicotinic acid and loperamide are also included in this list of antidiarrhoeal agents, but their action is unpredictable. Neomycin is frequently incorporated in various preparations, but it may damage the intestinal mucosa and cause malabsorption. Tincture of opium, paregoric and atropine are dangerous in children and dysentery patients because of delayed intestinal transit time. Steroids are expensive, useless and may cause adverse side-effects. Charcoal, kaolin, pectin and bismuth are of no value. *Mexaform* is useless and may be dangerous, whilst *Lomotil* is particularly dangerous in infants (Euro Reports and Studies 44). Nothing much can be left in the therapeutic armamentarium at this stage.

Replacement of fluid and electrolyte losses suffices in most instances, and this can usually be effected through the oral route. Intravascular and extracellular fluid loss of water and electrolytes causes an isotonic hypovolaemia; aldosterone secretion increases and the GFR decreases in order to conserve sodium. There is diminished tissue perfusion and shock causing lactic acidosis. The associated bicarbonate loss in the stool causes a hyperchloraemic metabolic acidosis, and the large potassium concentration in the diarrhoeal stool depletes the potassium stores. The serum K<sup>+</sup> may be normal in acidosis at the expense of low intracellular K; correction of the acidosis will cause K<sup>+</sup>to enter the cells and cause a low serum K<sup>+</sup>. WHO has recommended the following formula for an oral rehydrating solution:-

3.5 G/litre =	90 mmol/litre
	20 mmol/litre
2.5 G/litre =	80 mmol/litre
20 G/litre =	111 mmol/litre
	1.5 G/litre = 2.5 G/litre =

It is slightly hypertonic for small children so that more of diluent is required in preparation. The glucose is metabolised and does not contribute to the osmotic strength of the solution in the intestines.

Water losses and requirements vary with age and these variations must be taken into account when planning replacement and maintenance fluid programmes. Tables 1 and 2 illustrate the differences encountered between infancy and adulthood. It is to be remembered that neonates cannot concentrate their urine as well as older infants and children. After due allowance is made for losses and maintenance, the thirst mechanism can safely be relied upon to maintain homeostasis.

Enteric infection is the greatest killer among children, and it is estimated that 30% of deaths under 5 years are due to diarrhoeal diseases. There is a marked decrease in incidence in breast fed infants, and the trend noticably increases on the institution of mixed feeding. Weight loss is a problem, and if the disease is protracted there is also an arrest of height gain.

One official from the Italian Health Ministry remarked that a child with diarrhoea stood a better chance of survival without the treatment prescribed by the general practitioner. The same cannot be said for the local situation, and it will so remain as long as antibiotics and dangerous anti-diarrhoeals are not

Table 1:		
	aily Fluid Losse by Children and	
	7 kg male	70 kg male
Insensible Renal Faecal Total	45 ml/kg 40 10-15 95-100	22.5 ml/kg 20 2.5 45
	itenance Fluid R	1. S. S.
Age	ml/l	cg
1-3 days 4-10 days 3 months	60-100 125-150 140-165 130-155 125-145 115-135 90-110 70-90 40-50	

abused. Promotion of breast feeding is the best prophylactic, and simple water and electrolyte replacement by oral rehydrating solution (ORS) is calculated to save 70% of admissions for replacement by the intravenous route. Fasting causes disappearance of enzymes from the gastro-intestinal tract; with ORS, acidosis and hypokalaemia are gradually corrected and hunger is re-acquired permitting weight gain and a speedier approach to convalescence. Lactase is particularly very labile, and is one of the last enzymes to be restored to function after enteric infection - hence the importance of avoiding milk and milk substitutes until well into convalescene.

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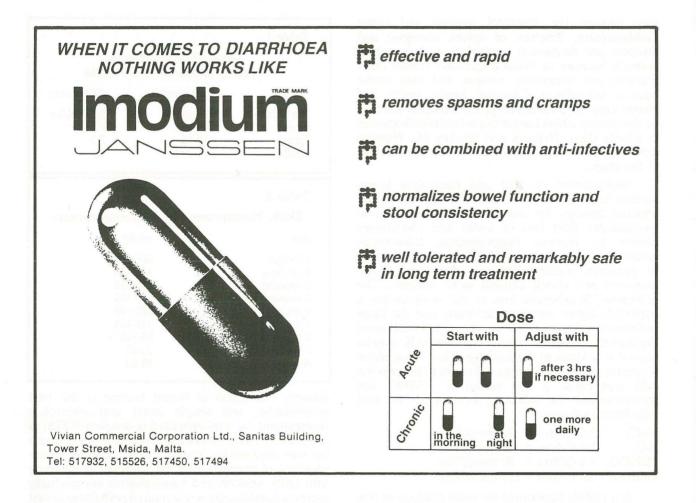


Table 3:

### Assessment of Dehydration and Fluid Deficit in Infants

Degree of dehydration (% loss of body wt)

	5	5	, ·
	Mild (4-5%)	Moderate (6-9%)	Severe ± shock (+ 10%)
Appearance	Thirsty, alert, restless.	Thirsty; restless or lethargic; irritable or drowsy.	Drowsy; limp; cold; Sweat and often cyanotic extrem ties; may be comatose.
Skin elasticity	Normal	Decreased, mild to moderate	Marked decrease.
Eyes	Normal	Sunken (detectable)	grossly sunken.
Tears	Present	absent	absent
Fontanelle	Normal	depressed	depressed
Mucosae	Moist	dry	very dry
Radial pulse	normal	rapid/weak	rapid/feeble/absent
Rectal temp	Nil	present	present
Urine flow	Normal	scanty	Nil for +8 hours.
Respiration	normal	deep/rapid	deep/rapid
Heart	normal	Tachycardia	Tachycardia/feeble
Estimated deficit	40-50 ml/kg	60-90ml/kg	100-120 ml/kg

### A Short Note on the History of Medicine in Malta.

I he University of Malta was founded by the Knights of St. John of Jerusalem. Thirty years after Grand Master La Vallette had founded the City that bears his name, the Jesuit fathers offered to build a College and a Church in Valletta. This offer was accepted and work was started on the 4th of September 1595. The building was completed in 1602. Twelve Jesuit fathers held public courses in Philosophy and Theology, and the degrees of Master of Philosophy and of Doctor of Divinity were conferred on successful scholars.

In 1574, the Order started building the Holy Infirmary also in Valletta, and every now and then improvements were added to the original plan, so that by 1662 the length of what was called The Great Ward was over 500 feet. A laundry and linen store were erected nearby.

In 1769, the Jesuits were expelled from the Island and their colleges and property were, by authority of the Holy See transferred to the Government of the Knights. In the same year a Bull by Pope Clement XIV, dated 20th October, confirmed the foundation of the new university. Three faculties were established: Theology, Law and Medicine.

During this period, the Holy Infirmary was one of the leading hospitals in Europe, and our knowledge of the medical work being performed here was limited to traumatic surgery. This bias in favour of woundsurgery is understandable if we bear in mind that the Order of St. John was constantly engaged in naval warfare against the Moslems. In assessing the nature and value of this work one must consider that European Surgery had not yet freed itself completely from Hippocratic or Medieval ideas. Progress had been made in anatomy in the famous Italian schools, but physiology and pathology were still in their infancy. Nothing was known about the origin and prevention of sepsis, and anaesthesia had not yet been dreamt of.

At this period a pioneer in surgery appeared on the scene, by the name of Michelangelo Grima. He spent his early years of training at the Holy Infirmary and thence went to specialise in Florence and Paris. In 1740 he was appointed Chief Dissector in the Royal Hospital of Sta. Maria Maggiore in Florence, and eight years later Master of Anatomy in the hospital in Messina. In 1761-62 he worked as Military Surgeon in Germany during the 7-year war. During this year he learnt the damaging effects of exposure to cold and of the long journeys in jolting carriages, especially in the case of head injuries.

In 1763 he returned to Malta and was immediately

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> appointed Chief Surgeon and Anatomist at the Holy Infirmary. He died in 1798 and was buried in the Franciscan Church in Valletta. Some of his Works include:

- a. Traumatic Medicine
- b. On the Injuries of the Spleen.
- c. On Popliteal Aneurysms.
- d. On a New and Certain Method of Suturing the Intestines.

A few months after the death of Grima, the Order was expelled from Malta by Napoleon and the educational and cultural life of Malta was disrupted. The new masters abolished the university, and the Holy Infirmary was taken over by the French for their troops. However, in 1800 the first British Royal Commissioner re-established the University, and medical studies re-started. Recognition of local Degrees was accorded on the turn of the century, and a happy association with British Universities began. So much so that although, up to a hundred years ago, the physician was accorded the imposing title of *Excellent Doctor* or *Magnificus Doctor*, his humbler brother, the surgeon, followed the English tradition and was simply called *Mister*.

Malta was becoming a prosperous centre of commerce and a gateway to the East, but also a target to infectious diseases like plague, smallpox and cholera. The quarantine regulations were very strict and annoyed many prominent visitors, and up to the year 1900 letters coming from effected countries were still being disinfected at our Lazzaretto.

The next milestone in our medical history was the discovery of the Micrococcus causing Undulant Fever.

The earliest reference to this illness in Malta is to be found, most probably, in an account of the Island written in the late 16th century by Giovanni Battista Leoni. Leoni was an ecclesiastic from Venice who accompanied Mgr Visconti on his visit to Malta in 1581 to inquire into the causes of the internal dissensions that were agitating the Order. Some time after his arrival in Malta, Mgr Visconti contracted a grave and prolonged fever which had *certain capricious intermissions* by which one was never sure whether he had recovered or was still sick, and which the doctors called *erratic fever*.

Leoni further informs us that the fever was accompanied by an *uncomfortable obstruction of the spleen*, apparently an allusion to the enlargement of this organ and to the pain and tenderness produced by the perisplenitis which is also of common

### occurrence.

For almost 300 years this type of fever continued to prevail undifferentiated from other *intermittent* or *remittent* fevers until the second half of the 19th century when its protracted course and disabling effects among the British troops began to engage the attentions of the military authorities.

The microbe causing the disease was discovered by Surgeon-Major (later Sir) David Bruce while he was working at the Station Hospital in Valletta in December 1886. He found the micrococcus in the spleen of 5 fatal cases of Undulant Fever. A few months later in conjunction with the Maltese Dr. Caruana Scicluna, he cultivated the organism on Agar-Agar.

Recognition of the disease was made easier in May 1899 when another Maltese, Dr. (later Sir) Themistocles Zammit applied Widal's Method to the serum diagnosis of the fever and demonstrated the microscopic coagulation of the Bruce Micrococcus when treated by the blood serum taken from a patient suffering from the disease.

The prevention of the illness, however, still remained a grave problem for, as long as the source of the micrococcus was unknown, no prophylactic measures could be devised.

In June 18, 1905, Zammit discovered the organism in the blood of the goat. The work of the commission set up by the Royal Society, at the request of the Armed Foces worked very hard from 1904 to 1906.

Zammit's discovery was soon confirmed by an unpremeditated experiment on human beings. In the summer of 1905, Mr. Thompson of the U.S. Bureau of Animal Industry obtained a herd of 65 goats from Malta and shipped them to America via Antwerp on the S.S. Joshua Nicholson. During the voyage many of the ship company drank freely of the goats' milk. On arrival at Antwerp the goats were re-embarked on the S.S. St. Andrew and again, during the passage to New York, a larger quantity of milk was consumed by the crew. Bacteriological examination of the milk of several of the goats that reached America resulted in the recovery of the micrococcus.

Exceedingly satisfactory results were obtained by pasteurisation. In the following months the Garrison also changed over from goat's milk to condensed milk. Someone, very wittingly, remarked that a tin-opener saved the British Army from extinction.

During the two World Wars, Malta was the Nurse of the Mediterranean, although during the last War the Island was a battered Nurse taking a very active part in the battle against the enemies of Democracy, and paying heavily for doing so.

In the medical field we are doing our best to carry on the good work at St. Luke's Hospital as did the Knights at the Holy Infirmary, because, like Osler

> We have loved no darkness Sophisticated no truth Nursed no delusions Allowed no fear.

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### Recto-Sigmoid Endometriosis

PROF. J.A. MUSCAT MD FRCS EX-DIRECTOR DEPT. OF SURGERY DR. RAY DELICATA MD DEPT. OF SURGERY

Colorectal endometriosis is sufficiently uncommon to warrant reporting as is evidenced by the number of single case reports in the literature. It is, according to statistics quoted from paper to paper, common enough to present one or more times in a life time of surgical experience. The following is a report of a case which was treated recently at St. Luke's Hospital.

### **Case Report**

Mrs. A.J. a 44 year old Maltese woman was referred on 2-9-83 to the Surgical Out-Patients' Department with pain in the left iliac fossa and constipation. The pain was colicky and intermittent and had been present for some two years. It was not evidently related to her periods, although her constipation, which was of long standing seemed to be worse with periods.

Her periods were regular, heavy and fairly painful. In January 1981 she had been referred to Gynaecology Out-Patients at St Luke's Hospital for vaginal discharge, pain in LIF and constipation. Vaginal examination showed a cervical erosion and a bulky uterus. The adnexae were normal. A PAP smear was taken. When seen in Surgical Out-Patients' clinic in September 1983 her general condition was good. Her abdomen was soft; there was vague tenderness in LIF. An IVP was requested and a note entered that she was to have an investigation of the colon in due course. Her IVP was normal and she was referred to Gynae. Out-Patients' with the possibility of an ovarian cyst. On 29-10-83 she had a D & C and an examination under anaesthesia in the Gynaecological Department. The cervix was now healthy, the uterus bulky and "? fixed". Adnexae were reported as normal. The curettings were reported: Dyssynchrous and hypersecretory endometrium - 2-11-83 (Dr H).

She was then referred from Gynae to the Orthopaedic Department. This was because of the severity and persistence of the pain and its tendency to radiate to the region of the left hip. Her orthopaedic assessment was negative. She was seen again in SOP on 4-11-83. She was not in pain at the time but complained of severe consitipation requiring regular dosing with laxatives. P.R. was negative. A Barium enema was reported on 29-12-83 as follows:

There is a narrow segment about 3" long between the sigmoid and the rectum. The outline is irregular but as the post evacuation film is not satisfactory the mucosal pattern cannot be visualized. Radiologically this is compatible with a Carcinoma but requires confirmation with a sigmoidoscope. The rest of the colon is normal. Sgd Dr Ś P K.

On 9-1-84 she was admitted to the Woman's Surgical Ward for sigmoidoscopy and further treatment. On 11-1-84 sigmoidoscopy was performed. (A.K.) Appearances were described as follows:

Ca sigmoid colon - growth looked like

cauliflower, stiff, 17cms above sphincter. Growth occupied only one side of colon.

A biopsy was taken. This histological report of 2 fragments submitted was:

Two fragments of large bowel mucosa with signs of congestion. No malignancy in these samples.

On 19-1-84 she underwent a repeat sigmoidoscopy by the same Surgeon (A.K.). Again the findings were described: 15-17 cms above sphincter on anterior wall of sigmoid colon Ca - stiff growth with small ulceration. Biopsies were also taken. These were reported thus:

Four fragments of mucosa - Heavy colitis with severe atrophy of mucosa. No malignancy in these samples.

A third sigmoidoscopy was performed this time by another Surgeon (A.S.) The findings were described thus:

At 15cms rigid stenosis especially anterior wall of rectum but without cauliflower formation.

Biopsies were taken. The biopsies were histologically examined and reported by Prof. B thus:

Heavy colitis with severe atrophy of mucosa. Nest of ? mucinous adeno-carinomatous formation in lamina propria. Malignancy must be taken into consideration. The sample is superficially taken.

An ultrasound scan of liver (7-1-84) showed no evidence of  $2^0$  deposits. The liver texture is normal. (Dr A S W).

The patient underwent operation on 12-2-84 under G.A. (J.A.M.). Through a left paramedian incision the abdomen was explored. A right ovarian 'chocolate cyst' was present. The uterus was bulky and densely adherent to rectum above posterior fornix. The anterior rectal wall in this region felt thickened and hardened. No tumours or other pathology were evident in rest of rectum and colon. Rectal endometriosis was considered to be the diagnosis. An anterior resection of the rectum was performed together with a total hysterectomy and bilateral salpingo-oophorectomy with removal of (R) ovarian cyst. An axial colorectal anastomosis was performed using the EEA stapler gun. This was protected by a caecostomy after appendicectomy. The patient made an uninterrupted recovery. The histological report of specimen submitted for

pathological study was as follows:

Uterus with cervix, both adnexae and segment of rectum. Uterus  $11 \times 7 \times 5$  cms. The rectum with the anterior part is closely attached to posterior part of uterus and cervix. No anus in the submitted specimen. Length of rectum 7cms. Circumference of proximal part is 6cms of distal part 4cms, 2cms above distal resection edge, very pronounced stenotic part of rectum 3cms long and 3cms in circumference. Mucosa of rectum smooth, shiny. Muscle sheath of proximal part of rectum highly hypertrophic, 0.8cms. On the anterior wall of rectum corresponding to the adhesions on posterior wall of uterus protruding part of mucosa 3cms long 1-5cms wide. On the cut surface of protruding mass the thickness of muscle sheath 14mm and thickness of mucosa 4mm. Muscle sheath is almost transformed to whitish hard mass occupying both muscle sheaths (no border between longitudinal and circular sheaths). Two sections from rectum.

Smooth wall of uterus up to 2.5cms. Mucosa is shiny, 2mm thick. Left tube  $6 \times 0.5$ cms. left ovary  $2.5 \times 1.5 \times 1$ cm with small haemorrhagic cyst from 2 to 4mm. Right tube 7  $\times 0.5$ cms. Right ovary not present. One section from cervix, one section from uterus. One section from left ovary and tube.

2. Cystic ovary  $4 \times 3 \times 2$ cms with haemorrhagic cyst 2cms in diameter. Cyst with defect on surface  $2 \times 1$ cm.

3. Congested appendix 6 × 0.5cm very narrow lumen. Three sections.

ME: Stenosis of rectum. Endometriosis of rectum. Hypertrophy of muscle sheath of rectum. Recto-uterine adhesions. Left ovary haemorrhagic corpus luteum. Right cystic ovary. Appendix not remarkable. (Prof K B).

Her post-operative course was uneventful. Her drains and urethral catheter were removed on 6th day. By 29-2-84 her wound had healed by primary intention but she was still running a low temperature. A gastrografin enema was requested. This was duly reported as follows:

There is congestion after operation in the rectosigmoid region. Anastomosis is patent. In the region of the anastomosis the filling of an irregular cavity is visible situated on the Rt side and posteriorly. Conculsion: Finding is suspicious of internal fistula. F/up is suggested. (R.K. - 29-2-84)

Her low grade fever subsided on 4-3-84 and she was discharged home on 6-3-84. She was last seen in Out-patients' Clinic on 6-7-84. She was well apart from an episode of short lasting abdominal pain in April 1984 and slight constipation. She was to be reviewed in 6 months' time.

### Discussion

A review of the extensive literature on intestinal endometriosis seen shows that the condition is relatively common, essentially benign and must often go unidentified. This latter characteristic is an added reason to warrant the publication of the present case report. No cases of intestinal endometriosis have ever been reported from St Luke's Hospital, Malta, The condition is likely to present to the Gynaecologist as often as it does to the abdominal surgeon. To take the matter of overall incidence first, it is reckoned that about 15% of all women in the reproductive period are effected by Endometriosis i.e. the presence of functional endometrial tissue separate from the uterus. Where such tissue is found ectopically but related to the uterus, superficially or deeply in the myometrium the condition is termed adenomyosis. About 33% of women with endometriosis have it effecting the bowel. This high incidence, which is contested by some, was reported by G. Kratzer & E Salvati in a study of a series, from Allentwon Hospital, Pennsylvania USA of 225 cases (1955). Dr Joseph Pratt in the discussion that followed the presentation of a paper on Endometriosis of the Bowel by Dr L A Gray at a Surgical Conference in Florida in 1972 stated that after conducting a study over a 5 year period of cases seen on his service in Rochester, 280 cases of endometriosis were noted. Of this number, 94 (34%) had endometriosis of the bowel. Further breakdown of these figures were not given but it was confidently stated that the rectosigmoid is the most commonly involved region. In Dr L Gray's reply he pointed out that the figure of about 15% with pelvic endometriosis described only relatively marked lesions.

Jenkinson & Brown reported on a series of 117 patients with endometriosis seen over a period of 3 years (1939 - 1941) at St Luke's Hospital, Chicago. In 47 cases the lesion was situated in the rectosigmoid region of the large bowel (40%). This he compares with Allen's (1933) incidence of 41 in 112 patients (37%) and R. Cathells' series where the incidence was strikingly lower 17 cases in 104 cases (16% - 1937). Counsellor and Masson's series of 162 cases of pelvic endometriosis included 51 lesions of the sigmoid, rectum and rectovaginal septum (31%). Keen & Kinborough's reported incidence of rectosigmoid lesions is the lowest; 6 cases in 118 i.e. 5%. The simultaneous presence of ovarian endometriosis with rectosigmoid endometriosis is less clearly defined. Jenkinson & Brown found 83 cases of endometrial lesions in the ovary and 47 in the rectosigmoid in 117 cases; this suggests that in 13 cases the two lesions were associated. In Counsellor & Masson's series there were 120 ovarian lesions and 51 rectosigmoid in 162 cases of pelvic endometriosis, suggesting that in 9 cases they were associated (i.e. 5.5%).

This fact is of some importance in helping the surgeon to diagnose the condition at laparotomy. In the case we reported a chocolate cyst the size of a grapefruit was present in the right ovary. Considering the figures quoted for pelvic and colorectal endometriosis it is generally argued that 2 to 4% of all menstruating women may develop endometriosis of the sigmoid, rectum or rectovaginal septum.

### Clinical Characteristics & Diagnosis of Colorectal Endometriosis

The extent and severity of symptoms produced by endometrial lesions vary with the size of the lesions and the degree of obstruction they produce. They tend to be present for longer than 12 months before presenting for treatment; in our case for 2 years. The common symptoms are vague lower abdominal discomfort, or cramp-like pains often associated with the menses, and constipation. The constipation may get worse with time and may also be aggravated during the periods. Bleeding per rectum from these lesions is rare as a presenting symptom, nor is passage of mucus a characteristic symptom. Tenesmus is an occasional complaint. The intensity of the pain and the constipation is related to the degree of constriction which becomes more marked as the lesion and its associated inflammatory reaction progresses circumferentially in the large bowel. In the small bowel kinking may induce obstruction.

#### Diagnosis

Many authors emphasize the fact that correct preoperative diagnosis of this condition cannot be minimized (Jenkinson & Brown). Definitive diagnosis prior to operation will permit the avoidance of radical resection of the bowel where castration will suffice. Simple excision of the lesion with part of the wall of the colon or rectum is sometimes also possible. Incorrect medical management would also be avoided, where operation is for some reason being postponed. However, in spite of the fact that some authors claim that certain diagnosis can be established in a majority of cases (162 of 179 cases 90%) from palpation of the disease in the cul-de-sac, uterosacral ligaments and rectal wall (Laman A Gray 1972) a review of the literature confirms our impressions that this is not so. In our case, as in hundreds of others reported, pelvic examinations by a Gynaecologist failed to establish the diagnosis. Nor was certain diagnosis arrived at when aided by radiological, endoscopic and biopsy studies. As Kratzer & Salvati point out the diagnosis of endometriosis of the large bowel is seldom made prior to surgery and when at the operating table it is difficult to distinguish this lesion from carcinoma.

It is fortunate that in a vast majority of cases of constrictive endometriosis of the rectum bowel resection does not necessitate sacrifice of the sphincter although Cattell (1937) cites a case and Lesh and Hatchcock, in 1955 another case where an abdomino-perineal resection was performed with permanent colostomy, carcinoma being mistaken for endometriosis. Amano & Yamada in 1981 reported a case of endometrioid carcinoma of sigmoid colon 13cms from the anus, diagnosed histologically after endoscopic biopsy as benign adenomatous polyp. Resection with end-to-end sigmoid-rectal anastomosis was performed with a satisfactory result.

It is thus evident that in general the most accurate approach that is possible is that of maintaining a high index of suspicion when certain differentiating criteria, favour endometriosis. The following differentiating points are of note:

1. The age in endometriosis is slightly younger (25-45).

2. Colorectal carcinoma is seen in males more commonly (65%).

3. There is often marked weight loss in cancer.

4. Constipation in endometriosis is often of long standing and intermittent and sometimes associated with menses.

5. Blood in the stools is common in cancer: rare in endometriosis.

6. Fertility is often below par in endometriosis.

7. Dysmenorrhoea is also not uncommon.

8. At endoscopy no ulceration is seen but stenosis and fixation are common.

At Barium enema a long stricture with *intact* mucosa may be reported. It is also well to remember that castration with or without colostomy may be a sufficient operation in the older patient or the patient with extensive disease where bowel resection would seem to be unduly hazardous.

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**Bactroban** ointment is a formulation of *mupirocin* 2% w/w in a water soluble polyethylene glycol base. Applied topically, **Bactroban** achieves a good penetration into the stratum corneum<sup>2</sup> giving levels that are well above the minimum inhibitory concentrations (MICs) of the majority of skin pathogens.<sup>3</sup> Only very low amounts of **Bactroban** are absorbed systemically and these are rapidly metabolised into an inactive compound and excreted via the kidneys.<sup>2</sup> **Bactroban** is not suitable for systemic administration and consequently does not compromise the effectiveness of oral or injectable antibiotics. In short **Bactroban** has been developed specifically for topical use. It has proved to be safe<sup>4</sup> and demonstrates a low potential for skin sensitisation.<sup>5</sup>

There is a broad spectrum of activity which includes those organisms commonly associated with skin infections i.e. staphylococci and streptococci.<sup>14</sup> In fact **Bactroban** is active against all clinically important stains of *Staph. aureus* and *Staph. epidermidis*. In tests against 92 skin isolates of staphylococci, **Bactroban** showed a high level of activity compared to fusidic acid and erythromycin: the tests showed about 40% of *Staph. aureus* resistant to erythromycin and 10% resistant to fusidic acid - none of the isolates were resistant to **Bactroban** at a concentration of  $0.5\mu$ g/ml.<sup>1</sup>

**Bactroban** has been shown to have a low potential for the development of resistant bacteria. In addition the novel chemical structure and its unique mode of action means that there is no cross-resistance with other clinically available antibiotics.<sup>1</sup>

**Bactroban** is a thoroughly researched topical antibiotic. Clinical trials have been conducted throughout the world to assess the efficacy in a very wide range of indications. The results have been assessed for the first 1,302 patients treated. Clinical success was achieved in 96% of patients with primary skin infections including - impetigo, furunculosis, pyoderma, cellulitis, folliculitis, ecthyma, balanitis and abscess.<sup>4</sup> A number of the trials are summarized in the Product Book available on request from the company.

In conclusion, a worldwide clinical trials programme has shown **Bactroban** to be safe and highly effective in the treatment of a variety of skin infections.

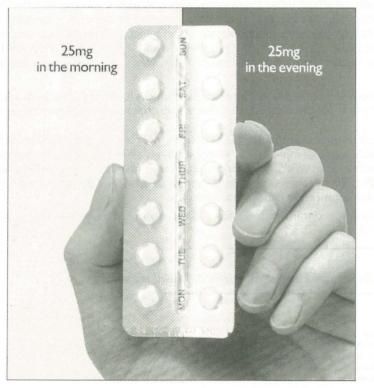
Full information is available from: Beecham Research International Beecham House, Brentford, Middlesex, UK.

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### **KEEPS PATIENTS FEELING WELL**



### Simple BD dosage

The usual maintenance dose is 25mg b.d. The starting dose of 12.5mg b.d. is particularly suitable for elderly or mild hypertensive patients. In mild to moderate hypertension, uncontrolled by a thiazide, the majority of patients will be controlled by the addition of Capoten 25mg b.d. usually combined with a diuretic.

### EFFECTIVE IN MILD TO MODERATE HYPERTENSION

Agents: K & M Limited 148 Melita Str., Valletta - Malta Tel: 221570, 225341

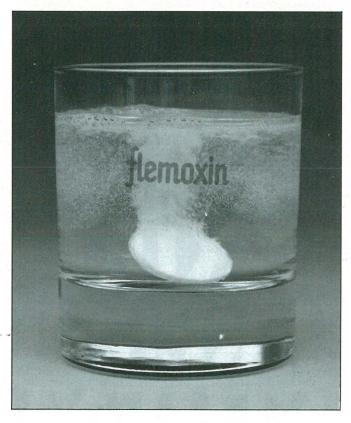




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the first amoxicillin effervescent tablet

rapid effect
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 no aftertaste



### • extensive range of presentation forms: drops, suspension, capsules, effervescent tablets

Drops	0-2 years	100 mg/ml (20 ml)
Suspension	2-10 years	125 mg/5 ml (100 ml) 250 mg/5 ml (100 ml)
Capsules	Over 5 years	100 x 250 mg
Effervescent tablets	Over 2 years	20 x 250 mg

Indications Flemoxin is indicated in the treatment of infections caused by amoxicillin-sensitive gram-positive and gramnegative microorganisms, e.g. respiratory, urogenital, gastrointestinal tract infections of the skin and soft tissues. In severe infections such as sepsis, meningitis, endocarditis, peritonitis, etc., parenteral administration (e.g. am picillin) is to be preferred. Therapy initiated with parenteral administration may be continued with oral Flemoxin when parenteral therapy is no longer required.

Contra-indication Hypersensitivity to penicillins.

*Use in pregnancy As far as known, this drug can be taken safely during pregnancy.* 

Side-effects Macular or maculopapular rashes may occur. Typical allergic reactions, such as uriticaria and purpura are less common. An anaphylactic reaction following oral administration of penicillin or one of its derivatives has only very occasionnally been reported. Gastro-intestinal side-effects, such as nausea, vomiting and diarrhea are sometimes observed, but are generally not serious and of a transient nature.

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# ADA

has many faces but usually presents with complaints such as fatigue, irritability and an inability to concentrate. Your ADA patients may also complain of backache, headache, chest pain, indigestion, insomnia or some other psychosomatic symptom associated with Anxiety or Depression.

ADA is a syndrome characterised by

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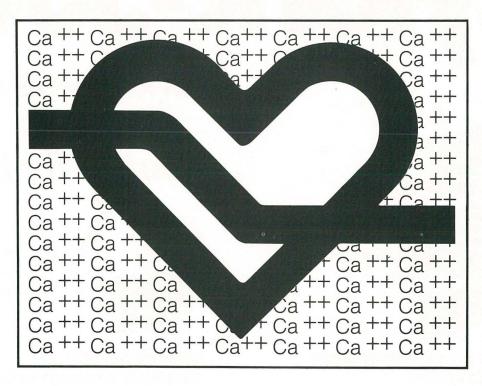
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## once-a-day dosage in gouty arthritis

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ZYLORIC, a product of Wellcome research, offers a radical approach to the treatment of disorders characterised by excess uric acid, such as gouty arthritis. By controlling the production of uric acid biochemically, it lowers both serum and urinary uric acid levels. Thus, apart from the more clinically obvious benefits of relief of joint discomfort, increase in joint mobility and decrease in size of tophi, ZYLORIC also significantly reduces the hazard of kidney damage and stone formation. Moreover, because of its sparing effect on the kidneys, it controls hyperuricaemia even in patients with impaired renal function.

### with the benefits of simplicity and convenience

Easier to remember, less chance of missing a dose - ZYLORIC-300 combines all the established and unique benefits of ZYLORIC therapy with the simplicity and convenience of once-a-day dosage in the treatment of gouty arthritis and excess urate disorders. Continuing Wellcome research has shown that, because of its inherent long action, a single dose of ZYLORIC provides a whole day's therapy without the need for sustained release technology. Wellcome discovered that ZYLORIC is largely converted in the body to a long-acting metabolite which extends the therapeutic effect to a full day. New ZYLORIC-300, with more likelihood of maximum patient co-operation and maximum therapeutic response, adds up over the years to better control of excess uric acid.

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Each ZYLORIC-300 tablet contains 300mg allopurinol ZYLORIC is also available as tablets each containing 100mg allopurinol Additional information is available on request

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