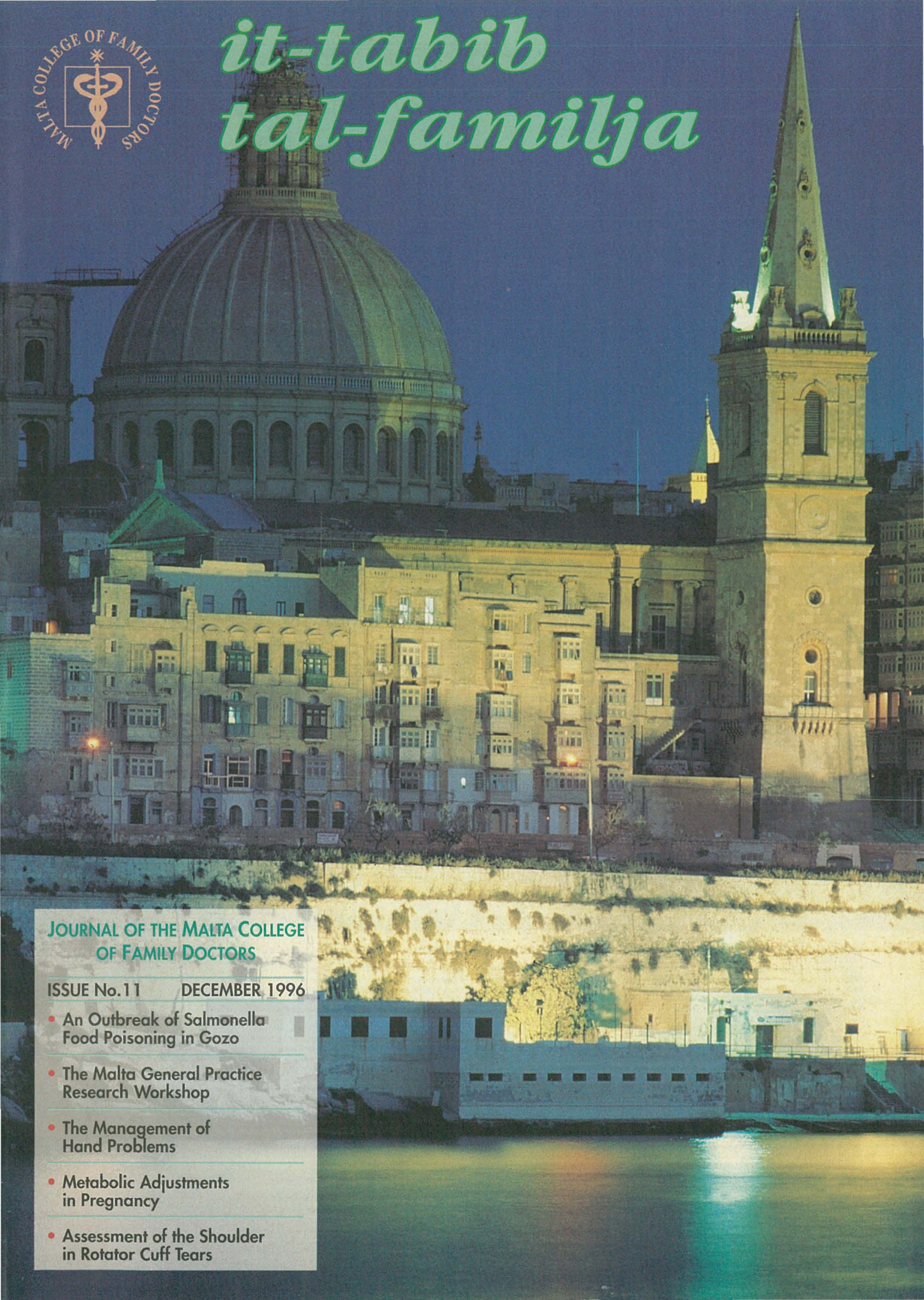

it-tabib tal-familja

JOURNAL OF THE MALTA COLLEGE OF FAMILY DOCTORS

ISSUE No.11 DECEMBER 1996

PRESCRIBING INFORMATION

INDICATIONS

Upper Respiratory Tract Infections e.g. Sinusitis, tonsillitis, otitis media. **Lower Respiratory Tract Infections** e.g. Acute and chronic bronchitis, lobar and bronchopneumonia, empyema, lung abscess. **Skin And Soft Tissue Infections** e.g. Boils, abscesses, cellulitis, wound infections, intra-abdominal sepsis. **Genito-Urinary Tract Infections** e.g. Cystitis, urethritis. pyelonephritis, septic abortion, puerperal sepsis, pelvic infections, chancroid, gonorrhoea. **Other Infections** e.g. Osteomyelitis, septicaemia, peritonitis, post-operative infections. AUGMENTIN intravenous is also indicated for prophylaxis against infections which may be associated with major surgical procedures involving gastro-intestinal, pelvic, head and neck, cardiac, renal, biliary tract and joint replacement surgery.

DOSAGE

Adults and Children Over 12 Years. Oral:- Mild-moderate infections: One 375mg AUGMENTIN tablet three times a day. Severe infections: One 625mg AUGMENTIN tablet three times a day or two 375mg AUGMENTIN tablets three times a day. The 625mg AUGMENTIN tablet is not available in all countries. **IV Injection/Infusion:-** Usually 1.2g 8 hourly. In more serious infections increase frequency to 6 hourly intervals. **Children. Oral:-** Children 7-12 years: 10ml AUGMENTIN 156mg syrup three times a day* or 5ml AUGMENTIN 312 mg syrup three times a day*. Children 2-7 years: 5ml AUGMENTIN 156mg syrup three times a day*. Children 9 months-2 years: 2.5ml AUGMENTIN 156mg syrup three times a day*. Children 0-9 months: No suitable oral presentation is currently available for this age group. *In severe infections these dosages may be doubled. Treatment with AUGMENTIN should not be extended beyond 14 days without review.

CONTRA-INDICATION Penicillin hypersensitivity.

PRECAUTIONS Changes in liver function tests have been observed in some patients receiving AUGMENTIN. The clinical significance of these changes is uncertain but intravenous AUGMENTIN should be used with care in patients with evidence of severe hepatic dysfunction. In patients with moderate or severe renal impairment AUGMENTIN dosage should be adjusted as recommended in the Package Insert Leaflet.

USE IN PREGNANCY AND LACTATION Use of AUGMENTIN in pregnancy is not recommended unless considered as essential by the physician. During lactation, trace quantities of penicillins can be detected in breast milk.

SIDE EFFECTS Side effects, as with amoxycillin, are uncommon and mainly of a mild and transitory nature. Diarrhoea, pseudomembranous colitis, indigestion, nausea, vomiting, and candidiasis have been reported. Nausea, although uncommon, is more often associated with higher oral dosages.

If gastro-intestinal side effects occur with oral therapy they may be reduced by taking AUGMENTIN at the start of meals. Urticarial and erythematous rashes sometimes occur but their incidence has been particularly low in clinical trials. An urticarial rash suggests penicillin hypersensitivity and treatment should be discontinued. Erythematous rashes are frequently mild and transient but may be severe when associated with infectious mononucleosis, in which case treatment should be discontinued. Rare cases of erythema multiforme, Stevens-Johnson syndrome and an occasional case of exfoliative dermatitis have been reported. Serious and occasionally fatal hypersensitivity (anaphylactic) reactions and angioneurotic oedema have been reported in patients on penicillin therapy. Although anaphylaxis is more frequent following parenteral therapy, it has occurred in patients taking oral penicillins. These reactions are more likely to occur in individuals with a history of penicillin hypersensitivity and/or a history of sensitivity to multiple allergens. Hepatitis and cholestatic jaundice have been reported.

AVAILABILITY 375mg AUGMENTIN tablets: White oval film coated tablets engraved "AUGMENTIN" on one side. Each tablet contains 250mg amoxycillin and 125mg clavulanic acid. 625mg AUGMENTIN tablets: White oval film coated tablets engraved "AUGMENTIN" on one side. Each tablet contains 500mg amoxycillin and 125mg clavulanic acid. 156mg AUGMENTIN syrup: Powder for preparing fruit flavoured syrup. When dispensed each 5ml contains 125mg amoxycillin and 31.25mg clavulanic acid. 312mg AUGMENTIN syrup: Powder for preparing fruit flavoured syrup. When dispensed each 5ml contains 250mg amoxycillin and 62.5mg clavulanic acid. In oral presentations amoxycillin is present as the trihydrate and clavulanic acid as the potassium salt. Not all presentations are available in every country.

Further information is available on request to:
SmithKline Beecham International, SB House,
Great West Road, Brentford, Middlesex TW8 9BD, England.

AUGMENTIN is a trade mark. 011/AUG/33/93.

SB
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International

Editorial

Dear Readers,

I would like to dedicate this issue's editorial to the new College web page, and the new site for Health Professionals in Malta, "The Synapse" (http://www.synapse.net.mt).

If you have a computer and a modem, you can connect to the Synapse network. This is a computer information system dedicated to health professionals. Doctors and pharmacists, and soon other professionals in our field, may log in to the network using a modem and a unique *username* and *password* which are supplied free of charge. The site is also accessible via any internet connection, at www.synapse.net.mt.

Doctors have access to a number of free services, including access to recently published Department of Health notices, noticeboards with lists of upcoming conferences (which may be downloaded and read), on-line medical journals including "Plexus" and "it-Tabib tal-Familja", lists of internet links with medical interest, the most up-to-date version of the "Directory of Social Welfare Organisations" which was published last year, and many more items of interest. There is also the facility to post a notice or message on the "Forum", and others may append their answers or comments! A useful member directory lists the electronic mail addresses of many doctors and pharmacists in Malta, and this is a very convenient way to send colleagues a message.

Of particular interest is the recently re-designed College homepage. This web page is a collection of information about the College, the Council, and College projects and activities, including our CPD programme. The pages were originally hosted on Grazio Falzon's web site in the USA, to whom we are greatly indebted. Grazio's "Malta Virtwali" (http://www.fred.net/malta/) still hosts our mirror site till this day. The College web page should be in the *bookmarks* or *favourites* or your internet world wide web browser, as it is the best source for up-to-date information about our CPD and other activities.

I would recommend a visit at your next opportunity. The College web site currently holds copies of documents published by the Council, including the latest summary document of our Specialist Training Programme. This is certainly worth a read!

Jean Karl Soler

Cover Photo taken by J.K. Soler:
Valletta skyline at dusk.

Printed on Recycled Paper

An Outbreak of Salmonella Food Poisoning in Gozo

Dr Peter Muscat
Medical Officer of Public Health – Gozo General Hospital

SUMMARY

An outbreak of gastroenteritis due to Salmonella berta *occurred following a social function (barbeque) affecting 12 out of 16 guests. A birthday cake bought from a catering firm in Gozo was the most likely vehicle of infection implicated. Three patients were admitted to hospital with severe diarrhoea and abdominal pain.*

NARRATIVE

On Wednesday 4th September 1996 at 11.00 a.m. the Disease Surveillance Branch of the Department of Public Health was alerted of a food poisoning event through two notifications of gastroenteritis by a local general practitioner. An investigation team was set up comprising the Medical Officer of Health, the Principal Health Inspector for the region of Gozo together with the respective District Health Inspectors.

The cases followed a barbeque held at the backyard of the home of one of the index cases at the village of Sannat. This was held on Sunday, 1st September from 5.00 p.m. onwards.

Over the next 24 hours 12 out of the 16 guests developed symptoms of gastroenteritis, primarily diarrhoea and abdominal pain. All the 12 symptomatic guests stated that they had eaten from the birthday cake at the function the previous day. The other 4 guests did not.

A 22 year old man who attended the function was admitted to Gozo General Hospital the day following the function, while two girls (ages six and nine years) were admitted 2 days later with gastroenteritis.

METHODS

(a) Epidemiology

A preliminary enquiry started by visiting the two index cases, one at the hospital and the other at home to verify the cases. The Disease Surveillance Branch obtained the names and addresses of guests at the function, identified the caterer, and procured a list of catering staff. A customised questionnaire was prepared for guests based on the information provided. Specific inquiry was made about consumption of individual foods and included questions on any resulting symptoms, and their respective time of onset. Relevant questionnaires were filled up by District Senior Health Inspectors and stool sample bottles were distributed to the persons involved.

(b) Investigation of the Premises and Food Preparation

(i) *Caterer:* The caterer's premises were inspected. Only sweet confectionery was prepared on site. No other party food was prepared at this place.

The caterer suspended his activities voluntarily for a few days until the investigation was complete. The general state of hygiene of premises was good. Working tops were in good state of repair and clean. Rubbish bins were properly covered. Equipment used for the manufacture of confectioneries was unremarkable.

The following deficiencies were noted;

1. No hand washing facilities were present in the preparation area.
2. Some shelving was not impervious.
3. Lime stone washing was lacking.
4. Fridges and prepared food were within reach of the general public.
5. Electric insectocuters were not functioning.

As regards to personal hygiene of the staff, no skin lesions were evident. None of the staff (two) were symptomatic and they did not report illness in the previous two weeks.

Environmental swabs (working tops and mixer), as well as hand swabs of the catering staff were taken. There were no party leftover food items. Six raw eggs and samples of ready-to-eat food items found at the premises were taken for analysis.

No other known cases of salmonellosis had been recently linked to food poisoning from the same premises.

(ii) *Household:* The house where the function was held was located in a new residential area.

The drain was connected to public sewers. The water system operated from the Government main supply. The working area in the kitchen was separated from the storage area. Raw and cooked foods in the fridge were noted not to be properly separated, predisposing to cross contamination. A sample of service water as well as two leftover slices of birthday cake that were saved were taken for microbiological analysis.

(iii) *Meat Supplier:* The meat used was bought from a butcher shop. The place was well equipped. The freezers and the equipment used was clean and well maintained. The personal hygiene of the food handlers was noted to be good, including proper use of head covers and washable clean overalls.

(iv) *Egg Supplier:* An inspection of poultry layers farm revealed good general state of hygiene of premises. Lime washing was in good condition. Premises were free from accumulation of chicken dung. Lighting and ventilation were good. The farm was adequately supplied with service water. A sample of 12 eggs and service water were taken for analysis.

(c) Microbiology

Stool samples were obtained from all kitchen staff and from 9 symptomatic guests.

The environmental swabs collected from the kitchen area as well as the food samples obtained, including the remnants of the birthday cake, were examined by standard methods (PHLS 1974) using selenite F selective broth enrichment for *salmonellas*. No effort was made to quantify *salmonellae* in foods. The food items were also tested for the presence of *Klebsiella, Streptococci, Campylobacter, Staphylococcus aureus,* and *Escherichia coli.*

(d) Temperature

Ambient temperatures (maximum and minimum) for dates of the outbreak were obtained (Meteorological Office, Department of Civil Aviation). This Office is located 15 km as the crow flies approximately 40 degrees due South-East from the outbreak site.

RESULTS

(a) Foods Served

- The foods served at the function are listed at table 1.
- The steak was cooked at the barbeque site.

Birthday cake Steak Raw vegetable salad: cucumber, tomatoes, onions and lettuce.

Table 1: Foods served at the function

(b) Questionnaires

- All guests attending the function completed the questionnaires. (16/16, 100%).

(c) Attack rates

- 12 out of 16 guests were ill, an attack rate of 75%.

A guest or staff member was defined as a **case** if he or she developed diarrhoea or vomiting plus or minus any other symptom within 24 hours of the function.

The principal symptoms reported by cases by frequency of occurrence are at table 2.

Symptoms	Frequency
diarrhoea	100%
nausea	83%
abdominal pain	75%
fever/chills	67%
headache	67%
weakness	67%
vomiting	50%

Table 2: Main symptoms reported by cases by frequency of occurrence.

The initial symptoms were diarrhoea and fever. The mode duration in hours from consumption till onset of symptoms was 14 hrs. The arithmetic mean for the same time period was 13.6 hrs. Range of incubation periods were 8-16 hrs.

Three persons required admission to hospital. Many guests lost time from work as a result of gastroenteritis.

(d) Food histories

Table 3 is a 2x2 table for exposure (consumption of cake) to development of illness i.e. becoming a case. There was an association between illness and the consumption of cake.

The Null hypothesis stated that there was no association between illness and the consumption of foods at the barbeque.

		exposure		
		+	-	
cases	+	12	0	
	-	0	4	
				16

Table 3: 2x2 table for exposure to development of illness.

Odds ratio is infinite. The null hypothesis is thus rejected.

(e) Bacteriological outcome

(i) *Guests*
Nine out of the twelve cases submitted stools, seven of which (78%) yielded *Salmonella berta.*

(ii) *Kitchen Staff*
The two persons who were involved in the preparation of the cake submitted stool specimens. These were positive for *Salmonella enteritidis.*

(iii) *Foods and Environmental Swabs*
The results of culture for salmonellae carried out on various foods, both remnants and food un-consumed at the function are shown in Table 4.

Environmental swabs obtained from the kitchen premises did not yield *salmonellae* though *Klebsiella* was cultured, indicating deficient hygiene. (Table 5)

Foods tested	Site	Growth
Cake (sample A)	Household kitchen	Salmonella enteritidis detected
Cake (sample B)	Household kitchen	Klebsiella spp detected
Service water	Household kitchen	No growth
Jam tart	Caterer's kitchen	No growth
Apricot jam tart	Caterer's kitchen	No growth
Date turnover	Caterer's kitchen	No growth
Almond cake	Caterer's kitchen	No growth
Apple tart	Caterer's kitchen	No growth
Six fresh eggs	Caterer's kitchen	No growth
Service water	Caterer's kitchen	No growth

Table 4: Results of culture carried out on various foods

Environmental swabs	Site	Growth
Working top	Caterer's kitchen	Klebsiella spp detected
Hobart mixer	Caterer's kitchen	No growth
Food handler (1)	Caterer's kitchen	Klebsiella spp detected
Food handler (2)	Caterer's kitchen	Klebsiella spp detected

Table 5: Results of culture of the various environmental swabs taken

(f) Ambient Temperatures

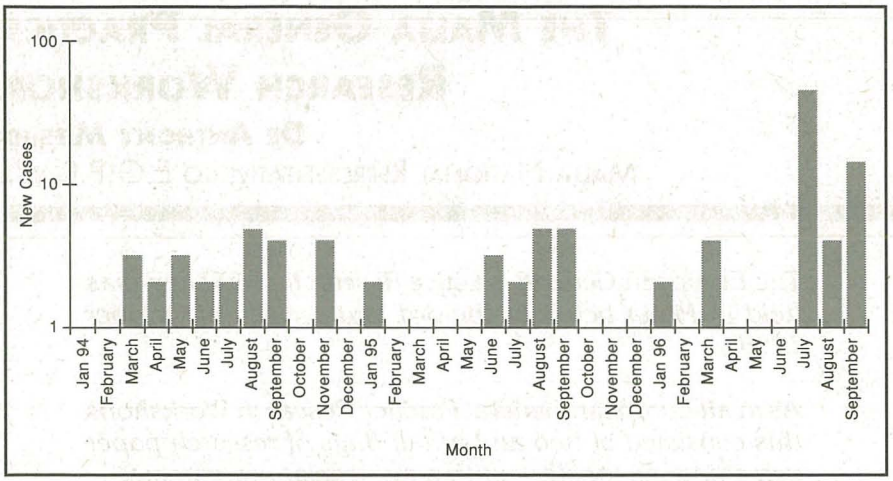
The maximum and minimum ambient temperatures recorded within 15km of the caterer's premises are shown in Table 6.

Day	Maximum	Minimum
31st August	30.5°C	24.3°C
1st September	29.4°C	22.4°C
2nd September	29.0°C	21.6°C

Table 6: Maximum and minimum ambient temperatures recorded within 15Km of the caterer's premises.

Environmental swabs and sampling took place three days after the outbreak occurred. One sample out of the two from the same cake cultured *Salmonella enteritidis.*

Graph 1 shows the number of new cases of salmonella food poisoning by month over the last 3 years. It is noted that the incidence is highest during the months of August and September. The cases were all sporadic except for 2 outbreaks which occurred in July and September 1996 involving 35 and 13 individuals respectively. High ambient temperatures are expected to enhance food spoilage. This factor has been noted before (Roberts 1982).



Graph 1: Time series (logarithmic scale) for notified salmonellae food poisoning in Gozo by month (January 1994 - September 1996).

(g) Action Taken

As *Salmonella enteritids* was isolated from the two sole proprietors and food handlers at the establishment, the place was closed for business for 15 days. The kitchen was thoroughly cleaned. All cakes and food not securely packaged were withdrawn from sale. Staff were required to produced three negative stool samples before being allowed to return to work. The staff were further instructed on cooking and storage techniques, as well as personal and environmental hygiene.

A follow up inspection was carried out at the catering firm to ensure that the licensee had carried out the works recommended.

The residents at household where the function was carried out were advised about proper separation of raw and cooked foods at refrigeration to avoid cross contamination.

DISCUSSION

This outbreak was characterised by its small size and was well circumscribed. This facilitated thorough tracing and follow up of each person involved. Epidemiological and bacteriological evidence suggested that consumption of cake was the most likely means of infection. The cake was probably contaminated by infected egg shells. However, the isolation of *Salmonella berta* from the cake may have been caused by cross contamination at site of preparation by the affected patient following illness ("reverse causality").

CONCLUSION

In summary, this report describes an outbreak of food poisoning involving 12 persons following a barbecue. Epidemiological and microbiological investigations were carried out on cases and food suppliers. The causative organism detected in people and food was *Salmonella berta*. The following action was taken:

1. Deficiencies in the kitchen were highlighted and a supervised cleanup of the premises carried out.
2. All food handlers were suspended for 15 days from work until cleared by 3 consecutive negative stool cultures.
3. Food handlers were further instructed on hygienic measures in production of confectionery.

Early notification in this case resulted in immediate and timely action to be carried out. This case illustrates how notification of infectious diseases by doctors, apart from being a legal obligation under the Medical & Kindred Professions Ordinance, and Prevention of Diseases Ordinance, is key to useful preventive action in the interests of the whole community.

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The Malta General Practice Research Workshop

Dr Anthony Mifsud
Malta National Representative to E.G.P.R.W.

EUROPEAN GENERAL PRACTICE
EGPRW
RESEARCH WORKSHOP

FGPRW is a network organisation within WONCA Region Europe – ESGP/FM

The European General Practice Research Workshop was held in Malta between the 3rd and the 6th of October 1996.

As in all European General Practice Research Workshops this consisted of two and a half days of research paper presentations together with a rich social programme.

Academic Programme

The main theme for the workshop was "Research in Social Problems in General Practice". A number of research papers were presented dealing with various Social problems such as: Aggression and violence, Social distance between doctors and patients, sickness certification, Social consequences of Breast cancer, Drug and Alcohol abuse, Smoking habits in Malta, and Accident prevention in the elderly.

A number of free-standing papers were also presented for discussion. These included papers about Usefulness of desktop analysers in general practice, use of WWH CD-Rom in general practice, Relational Databases, Acute Otitis Media, Morbidity registration in primary care and correct use of contraceptives by Moroccan Women.

The Pilot collaborative Study about Home visits was discussed at length. Certificates were given to the members that participated in the pilot study, and these included the ten Maltese doctors who took part in gathering information about home visiting in Malta.

It was of particular interest to us that the participation of Maltese Doctors in this meeting was high and included 7 presentations.

The Malta meeting was characterised by the rather high attendance (around 100 doctors) and by the fact that members of the Otitis Media Utrecht Group, held a parallel meeting and presented one paper about Otitis media. Also during this meeting presenters for the first time had an opportunity to ask for feedback about their presentations.

E.G.P.R.W. Board Meeting / Council with National Representatives

During these meetings discussed points of particular local interest included:

1 The **evaluation** of the selection process of papers for presentation. For the Malta meeting 50 papers were put forward for presentation, of these 35 were selected. The papers accepted were all of high quality. A question arose whether to choose more than one paper by the same author, and whether to accept papers of inferior quality from countries who put forward only one paper. The consensus was to accept papers **on quality** and not on quantity.

2 Amendments and approval to the new Statute. *Selection of National rep-resentative.*

All **paying members** from the specified Country are eligible for office.

In the case of **retirement at least six months in advance** the existing National representative of the country shall obtain a list of all the country's current E.G.P.R.W. members and notify them of the need to select a new representative.

The term of office for National Representative shall be five years and each representative shall normally be eligible for two terms of office.

3 Treasurer's Report.

National Colleges will **no longer pay** membership fees to E.G.P.R.W.

People who are direct members of the European Society may pay **half price** for E.G.P.R.W. membership.

Social Programme

Various Social events were organised before during and after the meeting.

These included:

1 "The Malta Experience" followed by a Reception at the Palazzo Castellania, hosted by the Parliamentary Secretary for health.

2 Walking tour of Mdina followed by a Maltese Fenkata dinner at Bobbyland.

3 Tour of Valletta special viewing of St. John's Cathedral followed by a visit to Qormi Health Centre and ending at Ta' Maria Restaurant for a Maltese Folk-Evening dinner with participation of foreign doctors in Maltese Folklore dancing.

4 Bus tour and visit to Hagar Qim Temples followed by viewing of the enactment of the entry of the Grandmaster and Knights of St. John into Birgu.

5 Dinner hosted by the M.C.F.D. with the Board members of the E.G.P.R.W. at La Dolce Vita restaurant.

I hope this meeting was a stimulating one and will act as a catalyst for increasing the interest of Maltese doctors to participate in Research in General Practice. During this meeting European doctors were given an opportunity to see how the Maltese family physicians practice.

W.O.N.C.A. '98 DUBLIN

JUNE 14TH - 18TH 1998

The Irish College of General Practitioners

A VENUE AND MEETING TO REMEMBER

Preparations for WONCA '98 in Ireland are well under way. The congress will take place in the historic Royal Dublin Society, in the leafy south of the city. It promises to be an exciting and dynamic conference in one of the most cosmopolitan European cities. The theme of the meeting is "People and the family doctors – Partners in care" and the logo of the conference reflects the importance of that partnership. It depicts the Liffey bridge, a pedestrian bridge known to Dubliners as the Halfpenny bridge as that was the toll when it was erected in 1816. It symbolically links the North and South of the city across the river Liffey, but it is more than an historical monument as it is still a busy thoroughfare of every day Dublin life. It links the heart of the commercial shopping area to Temple Bar, the avant garde centre of Dublin art, culture and street life.

The overall conference theme is developed through five main strands which offer a field of interest for everyone. These are: Clinical practice, including health gain and quality assurance; Care in Context – the importance of personal characteristics; Rights and Responsibilities and Medical Ethics; Practice management and health care delivery and Education and Research. Each morning and evening session will begin with a keynote address by a speaker of international repute and lead into a series of workshops, seminars, lectures and small groups. We extend an invitation to all our colleagues to actively participate by presenting your work or by leading parts of the academic programme, but we assure an equally warm reception to everyone.

Your stay will not be all work, and we hope you will have time to enjoy our culture and experience some of our music, dance, theatre and night life. The Social Programme has been designed to reflect many aspects of traditional Ireland and we know that you will enjoy these organised formal events. Ireland is alive with singing, dancing and lively conversation and we hope that you will also make time to enjoy the informal delights of traditional Irish hospitality and the welcome you will receive as you explore our capital city. In Ireland, the Pub is the meeting place, with one at almost every corner. It is here that you will hear the most erudite philosophers, aspiring writers and political commentators, for everyone here is a genius yet to be discovered. You may also stumble upon a session of Irish music. Dublin is also alive with literary and medical tradition. You can follow the footsteps of Joyce as you explore the city and visit landmarks made famous in Ulysses. Indeed we celebrate Bloomsday party in the programme. You could visit our National Theatre, the Abbey Theatre, where the great dramas of the Irish Literary tradition were first performed with both acclaim and controversy, or step through the hallowed portals of Trinity College and see the Book of Kells. Dublin has a rich medical legacy so you may see the origins of some of the great eponymous figures of medicine such as Colles of the Colles fracture, Corrigan of the pulse, Graves, Cheyne or Stokes.

The academic and social programme promise to make this a meeting to be remembered, and the tremendous enthusiasm of members the Irish College of General Practitioners will ensure you have a most wonderful welcome.

We look forward to welcoming you to Dublin. For further information contact:

I.C.G.P., Corrigan House, Fenian Street, Dublin 2, Ireland.

The Management of Hand Problems

Mr Carmel J. Sciberras
Orthopaedic Surgeon

This is a presentation delivered by Mr. Sciberras at our October CPD Meeting.

Almost 12 years ago, I started the HAND CLINIC. Since then, thousands (literally) of hand problems have been managed. I sincerely hope that, because of this clinic, the management of these problems has improved though I acknowledge that it could be even better.

In this short presentation, I shall try to outline my philosophy when dealing with a hand problem. Obviously, like most philosophies, mine is not perfect and I should be most grateful if any of you in the audience could point out any flaws and help me to correct them.

To start with, the hand problem may be a reflection of what is happening elsewhere in the body e.g. nail pitting because of psorasis, discoloured fingertips due to Raynaud's phenomenon, red palms due to liver disease.

The hand may also be the site of *Referred pain* e.g. paraesthesiae on the ulnar border of the hand due to a compression neuropathy of the ulnar nerve behind the medial epicondyle of the humerus, or from nerve compression in the neck.

Therefore, *the hand should not be examined in isolation,* excluding the rest of the body.

Let us now concentrate on local hand problems with particular emphasis on injuries which are by far the commonest hand problems. Proper management presupposes a sound knowledge of both the *anatomy and the functions of the hand.* Please do not squirm! I am not going to put you through a detailed description of the anatomy of the hand, but I would like to take you with me on a short description of its surface anatomy. I believe that surface anatomy is not given the importance it deserves both in the preclinical and in the clinical undergraduate curriculum.

Let's start by looking at the hand's dorsum. I am always amused when I see or hear the expression "knowing something or someone like the back of my hand" – meaning intimate knowledge. But have we really looked enough at the back of our hands to know them that intimately?

The skin is rather loose ... it can stretch by 40% when a strong fist is made. You can pick it up easily when the fingers are extended but not so easily when the fingers are tightly flexed. This property of the dorsal skin helps in flexibility. It is specialised and not easily replaceable.

The veins coursing underneath the skin differ from one hand to another even in the same person. They are important for drainage especially when flaps are present. They are also a godsend to anaesthetists the world over for the safe administration of intravenous anaesthetics.

The extensor tendons can also be seen ... unless, that is, one is very well covered! The index and the little fingers have 2 tendons each, one the proprius and the other a slip from the Extensor digitorum. This fact is important when we have to transplant tendons e.g. after extensor pollicis longus rupture following a Colles' fracture.

The anatomical snuff box at the base of the thumb is bordered by the extensor pollicis longus and the abduction pollicis longus and extensor pollicis brevis tendons. The latter two tendons are enclosed in tight tendon sheaths in the 1st dorsal compartment. This is the site of the common de Quervain's tenosynovitis which seems to be getting more common, possibly related to increased use of computer keyboards.

We now turn to the palm. Here you see a completely different kind of skin ... tight, hairless, sweaty, and with a lot of creases criss crossing it. To a palmist, these lines reveal a lot about one's life past, present and future, but to me, they indicate the site of important anatomical structures e.g. the distal palmar crease indicates the origin of the tough flexor tendon sheath which many students and doctors still think starts at the crease at the base of the fingers. The thenar crease indicates the medial extent of the thenar muscles and the site of origin of the recurrent muscle branch of the median nerve, a nerve to avoid cutting while doing a carpal tunnel release.

Speaking about the Carpal Tunnel, please remember that this is wholly in the palm i.e. from the distal wrist crease to about 2-3 cm distally.

So much for surface anatomy.

I would like now to outline the various functions of the hand. The hand can be looked upon in 2 ways;

1. As a fixed, non-prehensile end on a mobile arm ... for pushing furniture and lifting.

2. As a prehensile organ at the end of a mobile arm ... for various other functions which can be reduced to 2 basic functions, none of them pure:

   a. *Grip* ... i. *Precision* e.g. pushing a spray gun nozzle.
   ii. *Power* e.g. gripping a hammer.

   b. *Pinch* ... i. *Key* ... significant effort required.
   ii. *Pulp* ... holding artist's brush.
   iii. *Tip* ... picking up small objects.

Having looked at the basic functions of the hand, it will be clear that the management of hand problems must aim at restoring the FLEXIBILITY, the STABILITY, and the SENSITIVITY of the hand. I have not mentioned SHAPE, not because it's not important but because it's secondary to function.

FLEXIBILITY

The hand has got a lot of gliding surfaces. Injuries, infections etc. carry with them the risk of adhesions. These must not be allowed to interfere with the gliding surfaces by early intervention to treat infection, or by meticulous surgical technique in treating injuries.

STABILITY

The bony skeleton is the main stabiliser of the hand. However, ligaments, muscles, and tendons do play a very important part in this stability especially around the thumb and finger joints.

SENSITIVITY

This aspect of hand function was stressed a lot by Moberg (1971). It is extremely important. Almost one quarter of the touch corpuscles in the body are found in the hand. We all remember the brain homunculus with the very large area on the sensory cortex devoted to the hand. Truly the hand can be considered an additional eye of the body.

The management of a hand problem follows the same lines as all other medical problems.

History remains all important and usually gives you the diagnosis on a plate. The history must include the age, sex, occupation and hobbies, and hand dominance, apart from the aetiology of the problem ... how did it happen? Why? When? Where? Was there a previous hand injury / deformity? (This is important in medicolegal practice). The Medical history can be important e.g. Diabetes in trigger fingers and Dupuytren's disease, psychosis in a suicidal attempt.

The clinical examination takes each tissue one at a time.

Skin Is the wound tidy or untidy?
Is the colour pink or dusky?
Has there been any skin loss?
Remember hand skin is irreplaceable.
Are there any contractures?

Tendons Can you see triggering?
Any abnormal position of the fingers, signifying tendon injuries?

Nerves Any paraesthesiae complained of?
Any loss of movement?
Any loss of sensation and sweating?

Bones and joints Any obvious deformity?
Old or new?
Open or closed fractures?

Blood vessels The hand is a very vascular structure.
Bleeding is stopped by localised pressure and elevation. Please do not poke with artery forceps ... you could do more harm.

Special investigations may include high quality X-Rays with, for example, stress films to investigate ligamentous injuries. One may have to carry out blood investigations.

After making a diagnosis, a treatment plan should be drawn up. But before any treatment is started, one should ask oneself 3 questions:

1. Am I competent enough to treat myself or should I refer to someone more experienced?

2. How should I treat?

3. Can the definitive management be safely postponed?

If surgery is carried out, it must be with a gentle, minimally traumatic technique. One must remember that what is possible may not always be what is required ... sometimes the BETTER IS THE ENEMY OF THE GOOD.

I always keep in mind something I read many years ago:

A GOOD surgeon knows HOW to operate.

A BETTER surgeon knows WHEN to operate.

The BEST surgeon knows WHEN NOT to operate and / or WHEN to QUIT.

These are the PRIORITIES when dealing with hand injuries:

1. SKIN ... A good skin cover
   a. avoids infection and
   b. Protects surgical procedures done underneath it, acutely or later.

2. BONES and JOINTS ... Stabilisation of the skeleton before vascular, nerve or tendon operations can be carried out.

3. VESSELS ... In the hand itself. They can be safely treated usually. Veins plantations are controversial.

4. NERVES ... especially important for the recovery of sensibility. Even digital nerves.

5. TENDONS ... repaired or transferred later to restore joint movement.

The management of a hand problem is incomplete unless the WHOLE person is considered. A *"Physical"* hand problem does not occur in a *"Psychological"* vacuum. Here are some of the *personality problems* that may be associated with a severe hand problem injury that may entail a long period of enforced idleness and possibly repeated surgical interventions.

1. Loss of confidence ... more shelter seeking.

2. Enforced change of job – usually something inferior.

3. Deterioration of body image with cosequent psychological symptoms such as anger, anxiety and depression.

4. Constitutionalities. A problem too large to be dealt with here.

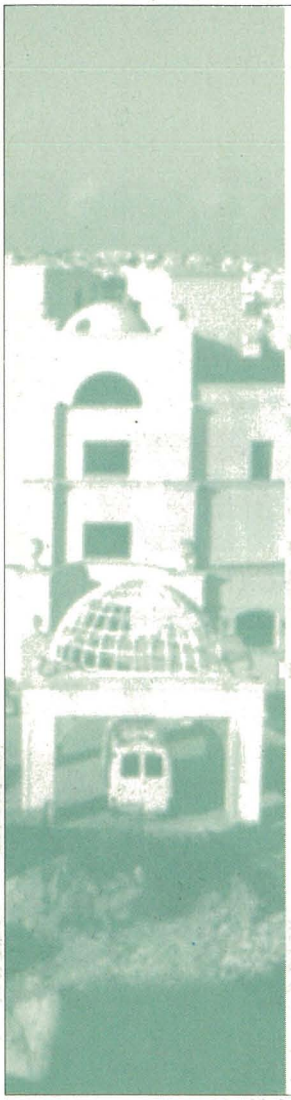
As clinicians, we therefore must have a *2-pronged approach* to the management of hand problems ... or any other medical problems, for that matter.

We must have the "CURING" function – Surgical expertise, drugs, physical therapy and the "CARING" function i.e. attention, hope, support-all part of the art of COMMUNICATION. Patients who are satisfied with the communication they've established with their medical providers are more likely to comply with treatment.

Because we must remember that *"People don't care how much you know until they know how much you care"*.

This is, in brief, my philosophy.

Thank you



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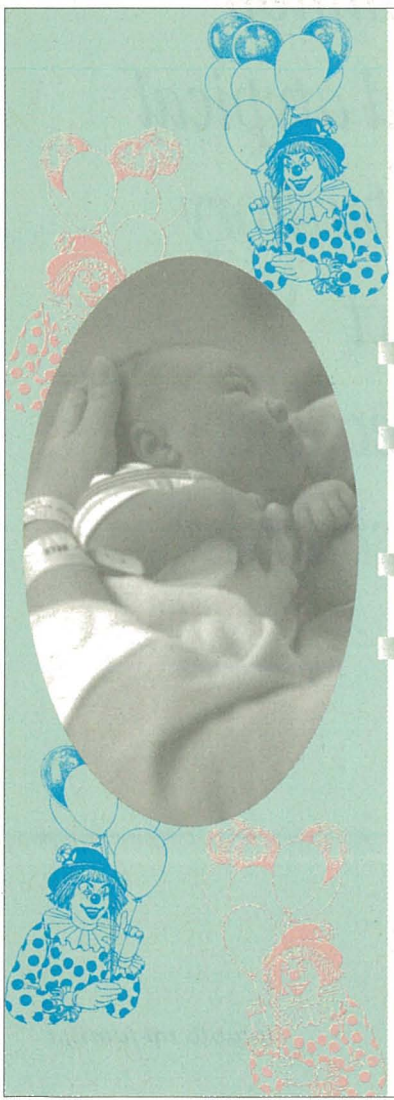

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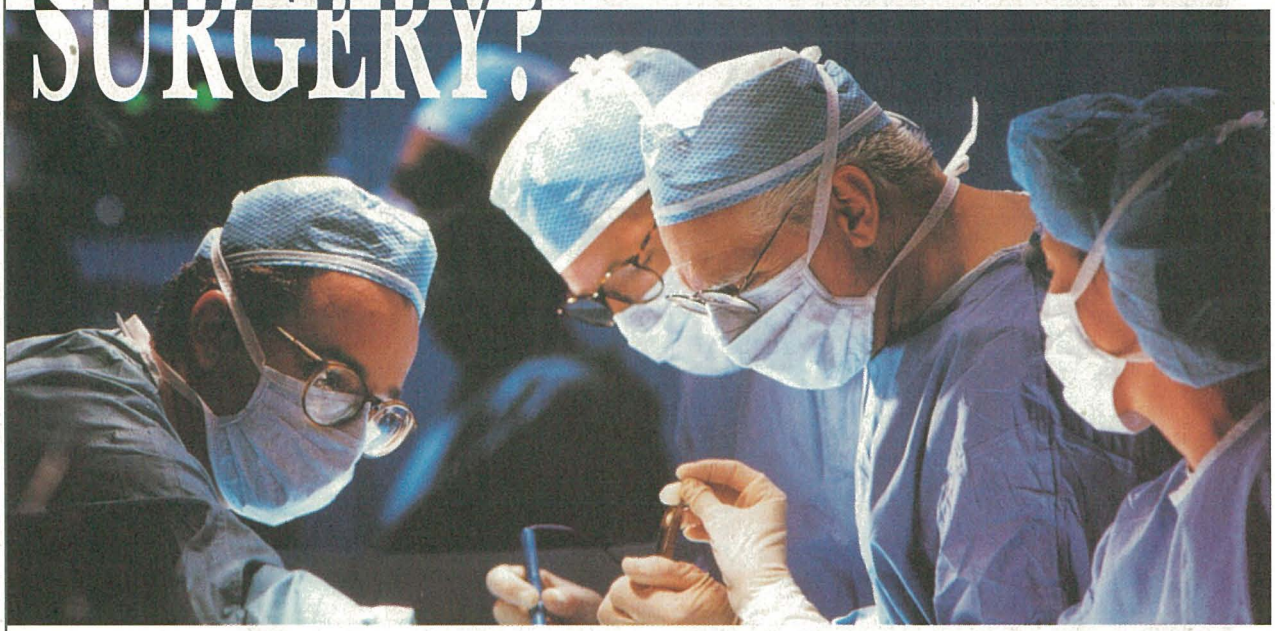

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METABOLIC ADJUSTMENTS IN PREGNANCY

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INTRODUCTION

Physiologically, the mother becomes almost a new person during the nine months of pregnancy. Virtually every system undergoes some change. In this respect, the pregnant woman is a natural laboratory in which to observe the effects of pregnancy hormones and nutritional demands of the fetus. Moreover, understanding the maternal adaptations to fetal growth has an important practical aspect in the management of the abnormal pregnancy. The question may be posed whether the fetus acts solely as a parasite draining fuels from the mother or whether the mother adapts to augment the supply of energy fuels to the fetus.

The Maltese woman is characterised by a high incidence of abnormal glucose metabolism during pregnancy when compared to other populations. Gestational impaired glucose tolerance, defined by the W.H.O. criteria, has been shown to affect about 14% of pregnant Maltese women[1]. Women with gestational impaired glucose tolerance have been shown to exhibit specific metabolic adaptations during their pregnancy[2]. This study attempts to identify the metabolic alterations which occur during the last trimester of pregnancy in this high risk population.

MATERIAL AND METHODS

One hundred and forty pregnant women booking for antenatal care at Karen Grech Hospital (Malta) were selected randomly, excluding only patients known to be diabetic. Karin Grech Hospital accounts for about 90% of all the deliveries which occur in the Maltese Islands. The study population thus constituted about 2.5% of the total population in Malta during the year of the study. Blood was taken in the fasting state from all the subjects who subsequently underwent a 75-gram glucose load for oral glucose tolerance testing at 24 and 34 weeks of pregnancy. One mother was found to have diabetes mellitus and was excluded from the study. The non-pregnant population included the data of the 645 non-diabetic women aged 15-44 years who participated in the National Diabetes Programme in Malta[3], besides data from other studies measuring haemoglobin levels in 1008 women of childbearing age[4]. The data were statistically tested using the standard error of the means, with significance being accepted at a probability value of less than 0.05.

RESULTS

Some maternal adaptations in metabolic functions can be understood from the pattern of weight gain in pregnancy. The last trimester is the time when nutritional demands by the conceptus on maternal fuel supplies are greatest and the main contribution to maternal weight is the growth of the conceptus. In the first trimester there appeared to be a gain in mean body weight of approximately 8.3% from 60.32 kg in the non-pregnant female to 65.31 kg at 24 weeks of pregnancy. As the pregnancy proceeded in the third trimester, there appeared to be a gain in body weight of 3.55 kg, a 5.4% rise over the mean body weight at 24 weeks. This increase was not solely contributed to by conceptual growth, since a 12.4% gain in fat area could be demonstrated (Table 1). The regulation of fuel metabolism in general is dependant on the relationship of insulin with other regulatory hormones.

Table 1: Body Weight Accumulation (mean ± s.d.)

	24 weeks	*34 weeks*	*% change*	*p value*
Maternal weight (kg)	65.31±13.66	68.86±13.55	+5.4	p<0.05
Skinfold thickness				
midarm triceps	20.30±5.89	24.04±6.86	±18.4	p<0.001
suprailiac	23.66±7.0	27.79±7.75	±17.5	p<0.001
Fat Area	193.52	217.42	±12.4	–

The mean fasting blood glucose levels at 24 weeks appeared to be at a lower level than the mean value in the non-pregnant female. The level subsequently rose by a significant proportion (12.4%) by 34 weeks. The blood glucose level two hour post-glucose load was markedly higher (28.5%) at 24 weeks than pre-pregnancy levels. The level at 34 weeks showed a non-significant drop. The basal insulin at 24 weeks showed a marked drop of 53.2% from pre-pregnancy levels, but by 34 weeks rose to 70.4% of the pre-pregnancy level. The mean fasting plasma C-peptide at 24 weeks showed a 22.9% rise from the pre-pregnancy level and subsequently showed a further 12.5% rise from the pre-pregnancy level throughout the third trimester. Glycosyated haemoglobin levels showed similar trends with a fall in mean levels in the first trimester and a rise during the third trimester (Table 3). As gestation proceeded from 24 to 34 weeks there appeared to be an increase in the stimulated insulin secretory responses in association with an increase in the basal insulin and glucose concentrations. Thus there appeared to be a 45.0 and a 103.9% rise in the early 30 minute and the 2 hour insulin secretory response to a 75g oral glucose load respectively. The response to the glucose load suggests a delay in insulin response at 34 weeks with higher 30 minute blood glucose and insulin levels. There was however a discordant response at 2 hours with no significant change in glucose load in the presence of elevated insulin levels (Table 2).

The cholesterol and triglyceride levels rose persistently throughout gestation from the pre-pregnancy levels with the rise being more marked in the triglyceride levels. The serum albumin concentration on the other hand showed a significant fall of about 10.5% with advancing gestation while the haemoglobin level rose after a 15% fall from the pre-pregnancy level. The products of metabolism in the form of blood urea and serum creatinine showed a fall in the mean values at 24 weeks from pre-pregnancy levels but increased subsequently in the third trimester. No significant change was noted during pregnancy in the urine nitrogen excretion calculated from the appropriate formula involving the urine urea nitrogen (Table 3), though increasing degrees of body weight seemed to be associated with higher levels of urine nitrogen excretion so that whereas in lean women there was a -1.5% change, in the obese woman the change was +9.3% (Table 4). These differences were however not statistically significant.

Table 2: Response to 75g Oral Glucose Load (mean ± s.d.)

	24 weeks	*34 weeks*	*p value*
Blood Glucose mmol/l			
Fasting	4.05±1.61	4.55±1.35	p<0.01
30 min	5.97±1.81	6.62±1.68	p<0.01
2 hrs	6.04±1.86	5.69±1.72	p>0.1
Serum Insulin uIU/ml			
Fasting	10.74±10.00	16.18±13.38	p<0.001
30 min	55.88±43.82	87.50±57.65	p<0.001
2 hrs	45.88±42.50	64.27±51.18	p<0.01
Insulin Secretory Response			
Early 30 minute	22.64	32.83	–
Late 2 hours	16.38	33.39	–

Table 3: Changes in Blood Biochemistry (mean ± s.d.)
[* based on Katona et al, 1983; ** Fenech, 1968]

	Pre-pregnancy	*24 weeks*	*34 weeks*	*p value (24vs34wks)*
Body weight kg	60.32*	65.31±13.66	68.86±13.55	p<0.05
Insulin uIU/ml	23.0*	10.74±10.00	16.18±13.38	p<0.001
C-peptide nmol/l	0.48*	0.59±0.33	0.65±0.10	p<0.05
Fasting Glucose nmol/l	4.3*	4.05±1.61	4.55±1.35	p<0.01
2-hour Glucose nmol/l	4.7*	6.04±1.86	5.69±1.72	p>0.10
HbA glyc %	6.7*	5.75±2.56	6.67±2.81	p<0.01
Cholesterol nmol/l	4.81*	6.60±1.45	7.72±1.65	p<0.001
Triglycerides nmol/l	1.14*	1.85±0.71	3.87±1.56	p<0.001
Albumin g/l	–	31.57±4.84	28.26±5.20	p<0.001
Haemoglobin g/dl	13.6**	11.56±3.91	12.93±3.62	p<0.01
Urea mg/dl	30.4*	24.10±12.16	30.21±11.84	p<0.001
Creatinine umol/l	47.71*	30.21±13.18	33.55±14.23	p<0.05
Urine N_2 excretion	–	7.08±5.32	7.43±5.15	p>0.5

Table 4: Urine Nitrogen Excretion (mean ± s.d.)

	24 weeks	*34 weeks*	*% change*	*p value*
Lean	7.16±4.6	7.05±4.6	-1.5	p>0.5
Overweight	7.24±5.2	7.62±5.8	+5.3	p>0.5
Obese	7.86±6.3	8.59±6.8	+9.3	p>0.5

DISCUSSION

Pregnancy is characterised by major physiological adjustments affecting every system of the body. The changes are frequently on a scale otherwise unknown in healthy adult life and have led to diagnostic confusions. The altered conditions characteristic of pregnancy allow maximum efficiency of fetal growth and metabolism, and are initiated and controlled by the various placental hormones. The changes noted to occur in body fat storage, and insulin secretion and action suggest that there is both a maternal "push" as well as a fetal "pull" with respect to nutrient flow across the placenta.

The average healthy pregnant woman, eating to appetite, has been reported to gain about 12.5 kg of bodyweight, considerably more than can be accounted for by the product of conception, the growth of the uterus and breasts and the expansion of the blood volume. Approximately half this weight gain is accounted for during the first two trimesters, while a further third is accumulated from 24 to 34 weeks of pregnancy[5]. With a total mean weight grain of 8.5 kg, the Maltese pregnant woman appeared to follow a similar pattern but with a slight decrease in the overall weight gain. This weight gain is significantly contributed to by an increase in the fat area. The discrepancy to reported averages may be accounted for by the fact that a significant proportion (55.8%) of women participating in the study were overweight or obese. It has been shown that previously overweight women gain less adipose tissue mass overall in pregnancy, possibly because the hypothalamic appetite centre becomes somehow "awakened" to the degree of maternal adiposity[5]. It appears therefore that the main stimulus to fat storage in the pregnant woman is not simply through the appetite-satiety centres driving the mother to eat more but by a more fundamental change in the control of energy balance. There is animal evidence to suggest that the total quantity of body fat is controlled by some central "lipostat" which is set higher during pregnancy by the influence of progesterone. The new level of body fat is achieved by eating more and expending less energy[6].

Insulin is central to adipose tissue metabolism as well as the regulation of fuel metabolism in general. The fasting blood glucose level fell in the first two trimesters by about 5.8%, while the fasting insulin level fell by 53%. During the third trimester both the fasting glucose and insulin levels rose significantly to approximate pre-pregnancy levels. During the glucose tolerance test, it appears that as pregnancy proceeds, there is a mild deterioration in blood glucose levels in spite of the increased secretion of insulin, thus resulting in a rise in both the early and late insulin secretory response. The changes demonstrated in the Maltese pregnant women are similar to those described in other populations, reflecting the recognised balance between the "accelerated catabolism" in the fasted state and the "facilitated anabolism" of the fed state[7,8]. The apparent paradox of decreased glucose tolerance of pregnancy in the face of a greatly raised level of circulating insulin suggest a "resistance" to insulin action. The nature of the insulin resistance in pregnancy is not yet fully elucidated, but appears to be related to changes in the insulin receptors which occurs during pregnancy. This insulin resistance serves to maintain glucose concentrations within a reasonable range and reduce maternal glucose utilisation, thereby maintaining an adequate plasma glucose supply for the fetus. In this manner, glucose in the circulation is "pushed" to the fetus in addition to the passive "pull" of nutrients by the fetus from the mother[9]. Other hormonal alterations during pregnancy affect carbohydrate metabolism. Free cortisol concentrations are increased in pregnancy and have been associated with reductions in insulin effectiveness. Growth hormone reduces insulin binding to receptors, while the analogous placental hormone – human chorionic somatomammotropin – could also reduce insulin receptor concentrations. Hyperprolactinaemia has been shown to produce a hyperglycaemic hyperinsulinaemic state similar to that seen in late pregnancy. Glucose intolerance and insulin resistance could also be mediated in pregnancy through increased secretion of pancreatic glucagon or increased insensitivity of the liver to glucagon effects[10].

Lipoprotein composition undergoes interesting changes during pregnancy. The triglyceride and cholesterol content of all three lipoprotein fractions is increased. The

endogenous hyperlipidaemia of pregnancy is a progressive one resulting primarily by an increased endogenous tri-glyceride entry into the circulation[11]. The changes noted in the Maltese population followed the previously reported trends. The albumin level on the other hand has been shown to decline as pregnancy advances. It has been suggested that this fall in serum albumin con-centration is principally a haemodilutional effect, but may also be contributed to by a fall in the total circulating albumin mass[12]. Haemoglobin con-centration, in contrast to previously reported trends, rose significantly during the third trimester. This rise was probably due to the routine iron supplementation the women received during pregnancy.

The biochemical parameters of waste-end products in the form of blood urea and serum creatinine showed a significant drop from the pre-pregnancy values by 24 weeks of pregnancy, the values later rising in the third trimester in contradistinction to the generally expected trends. The urine nitrogen excretion similarly rose in the third trimester. These waste-end product parameters are however not accurate markers for metabolic function since they are closely dependant of renal function, which is rather unsatisfactory to study during pregnancy because of the confounding influence of a number of factors.

It appears therefore that the "parasitic" fetus alters significantly the homeostatic mechanisms of its host so that it produces an internal milieu which allows maximum efficiency for its growth and metabolism. Many of these physiological changes occur and reach their full development in early pregnancy when the metabolic demands of the fetus are presumably negligible. Thus the physiological adjustments of pregnancy anticipate possible needs, unlike most other physiological responses which occur after a need has arisen. The pregnant woman is physiologically almost a different species.

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Assessment of the Shoulder in Rotator Cuff Tears

Mr Ray Gatt
Orthopaedic Surgeon – SLH

INTRODUCTION

The rotator cuff consists of four muscles; the subscapularis, supraspinatus, infraspinatus, and teres minor. The long head of the biceps tendon is another important component of the complex. The subscapularis is a head depressor, and in certain positions an internal rotator. The infraspinatus and teres minor are external rotators. The conjoint tendon of these muscles, attached to the tuberosities, anatomically and functionally, works as a unit, to maintain dynamic glenohumeral stability centering the humeral head onto the glenoid articulation. The long head of the biceps attaches to the supraglenoid tubercle of the glenoid and has a stabilising and depressing action on the humeral head.

The rotator cuff gives 50% of the abductor power and 80% of the external rotator power to the gleno-humeral joint.

BIOMECHANICS OF GLENO-HUMERAL MOTION

With the arm by the side, the deltoid acts to pull the humerus up, subluxing the head superiorly. On further abduction, the deltoid pulls the humeral head into the glenoid. When the arm is overhead the deltoid tends to sublux the humeral head inferiorly. The rotator cuff prevents this subluxation by pressing the humeral head into the glenoid. In the presence of a rotator cuff tear, excessive excursion of the humeral head along varying centres of rotation can occur due to loss of the compressive force of the supraspinatus.

INCIDENCE

Cadaveric studies by Fukuda et al. show that full thickness tears are present in about 20% of post-mortem dissections, whereas partial thickness tears are present in up to 32%.

AETIOLOGY

The cause of rotator cuff tears is probably multifactorial, involving some or all of the following points:

- Continued overuse of the shoulder causing attrition of the rotator cuff due to friction between the humerus and the acromion;
- Age-related changes in the integrity of the tendon associated with the constitutional condition of the patient and overuse predisposing to rupture;
- Trauma;
- Heavy overhead work, such as welding;
- Throwing and overhead sports.

A *critical hypovascular zone* is present adjacent to the insertion of the supraspinatus tendon just proximal to its insertion on the greater tuberosity. This avascular area seems to correspond with the area of tendon degeneration and rupture.

Lindholm and Mosely felt that the decreased vascularity in the critical zone represents an area of anastomosis between the vessels derived from bone and those derived from the muscle belly. Apart from this, with the arm in neutral and abducted position, there is constant pressure by the humeral head against the supraspinatus tendon. This compression more or less wrings the blood out of the tendon at the critical area, further compromising the site.

The supraspinatus tendon passes under the coracoacromial arch and may be subject to wear as it is compressed between the acromion and the humeral head. Neer talks about the concept of the *supraspinatus outlet,* through which the supraspinatus tendon passes between the coracoacromial arch superiorly and the superior edge of the glenoid inferiorly. This causes impingement with consequent attrition of the tendon, due to narrowing of the outlet. The latter may be due to:

- variations in shape/slope of the acromion
- acromial spur formation
- prominence of the acromioclavicular joint.

DIAGNOSIS

Diagnosis is usually from the history. The housewife states that she is unable to hang the washing, but is able to work perfectly well at the kitchen worktop. The schoolteacher will

come with a history that he is unable to write on the blackboard. The main symptoms are pain and weakness of the abducted arm.

PAIN

This usually relates to the anterior aspect of the shoulder and deltoid area. The pain is not in the cervical region. Neck pain starts in the neck and is referred distally to the hand; shoulder pain is not referred proximally.

The patient indicates cuff pain by the *palm sign* – putting the palm of his hand over the shoulder cape area to show the painful region. Acromioclavicular joint problems e.g. joint osteoarthritis is usually brought to the examiner's attention by the *pointing sign* directed to the acromioclavicular joint on the anterior aspect of the shoulder.

Cuff problems also give *night pain,* especially so when the patient turns over in bed on the affected shoulder. Night pain is worrying, because apart from cuff tears which is a common presentation, it can also be due to more serious pathology of the shoulder joint e.g. neoplastic problems and infection.

PHYSICAL SIGNS

LOOK

Watch the patient *undress.* This can provide a multitude of pointers that can lead the examiner down the right track. The patient will usually take off the shirt sleeve from the normal side first, protecting the injured arm.

Watch out for the *shrug* of a complete cuff tear. A patient with a complete tear will be unable to initiate abduction of the arm against gravity in any position. The patient will usually resort to trick movements to lift up the arm; by leaning sideways towards the injured arm, the patient will produce passive abduction by gravity until deltoid takes over to continue abduction.

'Fluid Sign' – swelling of the shoulder joint from the presence of synovial fluid in the subacromial bursa secondary to joint fluid in the glenohumeral joint. However, the amount of fluid has to be large for this sign to be present.

Prominence of the acromioclavicular joint due to osteoarthritis.

Look out for muscle wasting around the shoulder girdle. Wasting of the two main motors of the rotator cuff i.e. supraspinatus and infraspinatus is best seen in the plane of the scapula by looking at the seated patient from the top, and comparing both sides.

FEEL

Crepitus which is palpable in complete cuff tears.

Localise tenderness along the anterior edge of the acromion, the greater tuberosity of the humerus and the bicipital groove.

Pointing sign – ACJ problems.
Palm sign – Cuff problems.

Look for the palpable gap in the continuity of the cuff. Wallace reports clinical sensitivity of 91% and specificity of 75%.

MOVE

The *Painful arc of elevation* is a misnomer. The pain is usually felt between 70° and 120°, as the arm is slowly **lowered** actively from the overhead position to the side. Loss of the arc of movement from 0° to 60° is usually due to a complete cuff tear; and a painful arc from 120° to 180° can be due to acromioclavicular joint problems e.g. osteoarthritis.

Positive impingement sign – in this sign, pain is felt at the anterior edge of the acromion with passive, forced, forward elevation of the arm. The examiner should stand behind the patient to stabilise the scapula with one hand. Impingement may occur between 70° and 120°. A less reliable test, is pain felt over the coracoacromial ligament with forward flexion of the humerus to 90°.

An extremely helpful diagnostic tool is the **painful arc injection test.** The aim of this test is to abolish pain in the subacromial space and exclude causes of referred pain from other areas such as the neck and chest. With the undressed patient sitting comfortably in a chair, 10mls of 0.25% bupivacaine is injected in the soft spot at the lateral end of the scapula just beneath the posterior lip of the acromion. Infiltration from this site is safer than from the lateral site and definitely safer then from the anterior site. After an interval of 5 to 10 minutes, the painful arc test is repeated. If pain is completely abolished, then the problem is in the subacromial space, and not referred on from surrounding joints. If pain is completely abolished and the arm is still weak, this is indicative of a rotator cuff tear problem. If pain is abolished, but there is no weakness, a tear is excluded, and the problem could be subacromial bursitis or supraspinatus tendonitis. The test is both diagnostic and therapeutic.

TESTING INDIVIDUAL COMPONENTS OF THE CUFF

- *Supraspinatus test*

With the arm abducted to 80° in the plane of the scapula and with the thumb pointing downwards, strength is tested with one or two fingers resistance by the examiner. Weakness

suggests a tear of this tendon. Passive motion that exceeds active motion in the abduction plane can be due either to pain or loss of the abduction power. A further corollary of this test is a positive 'drop arm' sign, which is inability to maintain the passively positioned arm in 90° of abduction; this signifies a large tear. Inability to initiate abduction of the arm against gravity in any position signifies massive tears.

- *Infraspinatus test*

Weakness of active external rotation of the shoulder joint with the elbow flexed to 90° and the forearm acting as a pointer to the extent of rotation, indicates a complete thickness tear of the tendon. Compare with the other side.

- *Subscapularis test*

This test is done by asking the patient to internally rotate the arm behind his back, with the hand over the natal cleft, and continuing the internal rotation movement against resistance. A positive *'lift off'* sign signifies subscapularis weakness/tear.

RADIOLOGY

Plain X-rays

AP shoulder with 10° caudal tilt of the source.

This will show an anterior acromial spur which is an osteophyte located along the antero-inferior edge of the acromion and is caused by the traction from the coracoacromial ligament.

AP shoulder

This will show cystic changes and sclerosis with irregularity of the tip of the greater tuberosity where supraspinatus inserts. There will also be superior subluxation of the humeral head with narrowing of the acromio-humeral interval, and gleno-humeral osteoarthritic changes in cuff arthropathy.

The most reliable radiographic procedure to demonstrate tears and associated problems is **MRI**. It can delineate the size of complete tears, hypertrophic changes on the anterior edge of the acromion and acromio-clavicular joint, detection of bursitis and tendonitis and glenoid labral tears; but it is unable to differentiate between partial and full thickness tears. As yet, MRI is unavailable locally, but an equally useful procedure that can be done is **arthrography** with or without double contrast. Visualisation of intra-articularly injected contrast material (in the glenohumeral joint), extending into the subdeltoid region is the single arthrographic criterion of a complete rotator cuff tear.

Neer's indications for arthrography are:

- Impingement syndrome persistent beyond 12 weeks in over 40 years olds.
- Injury with 'sudden marked shoulder weakness'.
- Ruptured long head of biceps with shoulder symptoms.
- Unstable or symptomatic shoulder after dislocation in over 40 years olds.

Ultrasonography is a non-invasive and inexpensive test that permits examination of both surfaces of the cuff. Comparison can be made with the contralateral shoulder and may be of use in the evaluation of the post-op shoulder. This test is user-dependent and does require an interested radiologist.

The role of joint **arthroscopy/ bursoscopy** is controversial and depends on setup availability and expertise.

MANAGEMENT

Treatment of cuff tears depends on several factors including age of the patient, athletic prowess, and associated impingement.

CONSERVATIVE MANAGEMENT

A large percentage do well with conservative management. This will involve decreasing the pain and rehabilitating the rotator cuff, and avoiding harmful and provocative motions. The initial management is treatment of pain and rest. Infiltrations into the subacromial bursa with local anaesthetics are quite useful to ablate the pain. Local infiltration of steroids into the cuff area should be avoided (except in calcific tendonitis problems), as this can lead to tendinous degeneration of the cuff. Nsaids are useful to decrease the inflammation and oedema of cuff tears.

The shoulder is not used for activities of daily living above 60° of abduction and flexion. After the pain is decreased, strengthening exercises of the cuff components are initiated, including supraspinatus and infraspinatus, together with stretching exercises. **Physiotherapeutic rehabilitation** is the keystone of conservative management and this should be carried out for periods of up to 3 months. This is the first stage in the management of rotator cuff tears in non-athletes and athletes with small tears producing minimal signs.

If there is no improvement during the rehabilitation phase after 3 months, the second stage of management comes in; and this will include further investigations, including an arthrogram as well as plain films to exclude impingement.

Surgery is done to decompress the subacromial space and to repair full thickness rotator cuff tears. Decompression will include anterior acromioplasty with resection of the prominent inferior acromioclavicular joint spur and excision of the coracoacromial ligament.

The **goals of surgical treatment of rotator cuff tears** are:

- Tensionless repair with the arm at the side and snug apposition of healthy tissue.
- Restoration of the muscle/tendon unit attachment to the humerus.
- Production of a smooth surface and watertight closure.

RESULTS

Good results have been reported with arthroscopic subacromial decompression, and this is a promising alternative to open acromioplasty in selected patients **without** full thickness cuff tears. Repair of small cuff tears arthroscopically is possible, but in centres where the necessary expertise is lacking, and there are multiple pathologies to sort out, it is probably wiser to perform an open cuff repair together with decompression.

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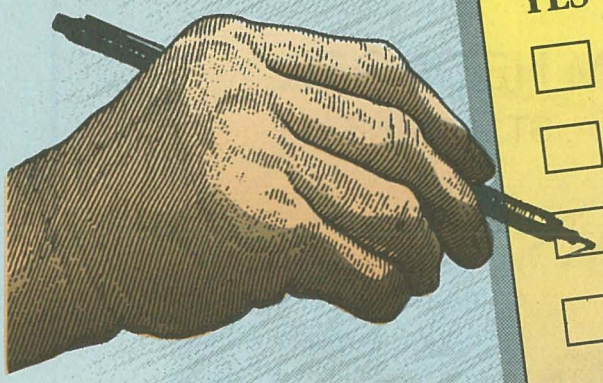

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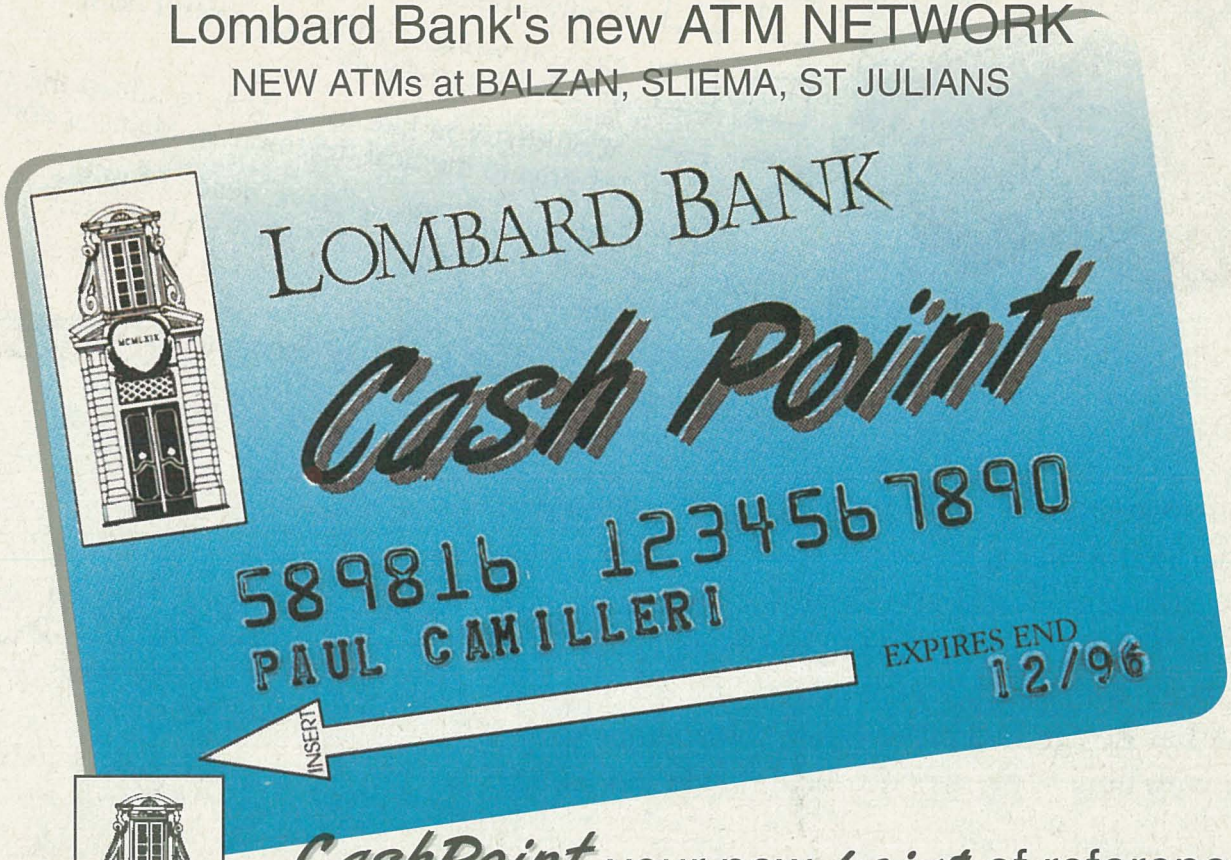

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