May, 1993 Vol. 11, No. 2

ISSN 1015-0870

Journal of Bangladesh College of Physicians and Surgeons

Official Journal of the Bangladesh College of Physicians and Surgeons

JOURNAL OF BANGLADESH COLLEGE OF PHYSICIANS AND SURGEONS

Vol. 11. No. 2, May, 1993

Official Publication of the Bangladesh College of Physicians and Surgeons

Mohakhali, Dhaka-1212

Chairman

Dr. Matiur Rahman

Editor-in-Chief

Dr. Shafiqul Hoque

Executive Editor: Dr. Syed Kamaluddin Ahmed

Members:

Dr. K. M. H. S. Sirajul Haque

Dr. Sadiqa Tahera Khanam

Dr. M. Nazrul Islam

Dr. Moazzam Hossain Dr. A. K. M. Anisul Haque

Dr. U. H. Shahera Khatun

Published by :

Dr. Shafiqul Hoque, on behalf of the Bangladesh College of Physicians & Surgeons

ASIAN COLOUR PRINTING. 130, DIT Extension Road (Fokirapool). Dhaka-1000, Bangladesh.

Phone: 407656

Address for Correspondence: Editor-in -Chief, Journal of Bangladesh College of Physicians & Surgeons. BCPS Bhavan, Mohakhali, Dhaka-1212, Tel: 600454, 882836

Annual Subscription: Tk 300/- for Local and US \$ 30 for overseas subscribers.

INFORMATION FOR CONTRIBUTORS

The Journal of the Bangladesh College of Physicians and Surgeons is published thrice a year in the months of January, May and September. The Journal publishes original papers, case reports and reviews in all branches of medical science. The style of the paper should be in the modified Vancouver style (Ref: J Bangladesh Coll Phys Surg 1991; 9(1&2): P I-VII).

Papers should be submitted to the Editor-in-Chief, The Journal of Bangladesh College of Physicians and Surgeons, BCPS, Mohakhali, Dhaka. Papers Should be written in English and three copies must be submitted with three sets of illustrations. Manuscripts should be typed on one side of white paper (size 8.5 X 11 inches) with margins of at least one inch. Double spacing should be used throughout. Each of the following sections should begin on separate pages as: title page, abstract and key words, text, acknowledgements, references, individual tables and legends. Pages should be numbered consecutively beginning with the title page. The title page should carry (a) the title of the article, (b) name of each author with highest academic degree (s) and institutional afiliation, (c) name of the department and institute where the work was carried out and (d) name and address of the author to whom correspondence should be addressed and to whom reprints should be sent.

manuscripts must be accompanied by a covering letter. This must include: (a) a statement that the work has not been published or submitted for publication elsewhere, (b) a statement of financial or other relationships that might lead to a conflict of interests and (c) a statement that the manuscript has been read, approved and signed by all authors. Any work which has been carried out in part or fully abroad, must be accompanied by a letter from the head of the institution where the work was done, stating that the work has been carried

out in that institute and that there is no objection to its publication in this journal.

If the article is a whole or a part of the dissertation or thesis submitted for a diploma/degree should be mentioned in which case the name of the worker and the guide must be mentioned and must be permitted for publication by the competent authourity of the Insitiute where the work has been done.

A summary/abstract of the work should be of less than 200 words. Each table should be typed double spaced on a separate sheet. These should be numbered in Roman numericals consecutively in order of their first citation in the text. A brief title of each table should be supplied. Figures should be professionally drawn and photographed. Xray should be photograhed. Photographs should be on glossy papers (usually 5 X 7 inch) in black and white. These should not be inserted into the text but marked on the back with the figure numbers, title of the paper and name of author. The top of the figure should be indicated. All Photograps, graphs, diagrams should be referred to as figure and numbered consecutively in the text in Arabic numericals. The legends for figures should be typed on a separate sheet.

Ethical aspects will be considered in the assessment of papers and authors should indicate in methods whether permission of relevent ethical committee have been taken if needed (see the World Medical Association's code of ethics, Brit Med J., 1964; 2: 177). Statistical methods used should be described in enough detail to enable a knowledgeable reader with access to the original data to verify the reported results. Study design should be stated with details about randomisation.

Usually 10 copies of reprints are supplied to the author free of cost. Additional reprints may be obtained by prior arrangements but must be paid for.

JOURNAL OF BANGLADESH COLLEGE OF PHYSICIANS AND SURGEONS

Vol. 11. No. 2. Page 38-68

May, 1993

CONTENTS

Editorial	
Malaria Situation in the Hill Tracts of Bangladesh	38-39
Original Articles	
 4520 Cases of Malaria in Chittagong Hill Tracts—A Clinical Analysis Anis waiz, Rabiul Hossain, Barendra Chakraborty, Md. Shahjahan 	40-43
 Acute Cerebrovascular Disease—Some Clinical and Epidemiological Correlates HAM Nazmul Ahasan, Halima khatun, Saiful Islam, Merina Ara Goutam Kumar Shaha 	44-47
 Omental Transposition in End-stage Buerger's Disease—Personal Experience Hasan Mohd, Abdur Rouf 	48-51
Post-tonsillectomy Haemorrhage—A Study of 1230 Cases M. Alauddin, Khabiruddin Ahmed	52-55
Review Article	
 Use of Monoclonal Antibody in Cancer Therapy-Problems and Perspective Rezwanul Hoque, Nazma Akhter, Md. Aslam Hossain 	56-60
Case Report	
Successful Pregnancy Outcome in a Patient of Ectopia Vesicae Shamsun Nahar Begum	61-63
College News	
College News	64-68

Malaria Situation in the Hill Tracts of Bangladesh

Malaria has been a recognised cause of morbidity and mortality in certain parts of Bangladesh namely the north and the east for a good part of the century. There had been an appatent decline in the late fifties due to rigorous eradication measures particularly in urban areas 1.2.

Resurgence of malaria was experienced in the midseventies³ when the members of the security forces were committed to the hilly terrains of Chittagong division.

Severe forms of falciparum malaria including cerebral malaria became a common event among the nonimmune recent arrivals to that area. The unway physicians treating these cases in the base hospitals far away from the area of exposure were perplexed with the protean symptoms and manifestations of severe falciparum infection and made erroneous alternative diagnosis to typhoid state, meningitis, encephalitis etc⁴. The country having gone through a period of upheaval during the war of liberation did not have the technical continuity to provide accurate diagnostic facilities.

However, in the early part of 1975 it became clear that along with high fever, manifestations such as pulmonary oedema, renal shutdown and grade IV coma were but the various manifestations of severe form of falciparum malaria. At that stage several strategies were adopted in order to prevent and a rational approach to the treatment of clinical cases^{5.6}.

A retrospective analysis in 1985 revealed a three fold increase in malaria cases despite strict administration of chemoprophylaxis, the preponderance of falciparum malaria in the hill the security forces on returning to the plains on cessation of chemoprophylaxis. It became clear that chemoprophylaxis had no falciparum malaria but effectively prevented clinical vivax malaria with liability to clinical attacks on withdrawal of chemoprophylaxis^{7,8}. The very high mortality (54%) of cerebral malaria was assumed to be due to the standard practice of administering dexamethasone and came down to 10% on withdrawal of dexamethasone 9.10. The experience gained and the lessons learnt in Chitagong Hill Tracts led to the decision of withdrawing chemoprophylaxis for the long term visitors, recognition of bad prognostic features of cerebral malaria (jaundice, convulsion, shortness of breath, oliguria, haemoglobinuria etc), adoption of triple drug therapy (quinine, cotrimoxazole and tetracycline)11, recognition of hypoglycaemia as an immediate cause of deterioration of previously improving cases and recognition of deleterious effects of steroids, resulting in the happier trend today in the reduction of incidence, development of immunity and effective management of malaria 12. Chloroquine has still retained its efficacy in uncomplicated malaria13.

tracts and occurrence of vivax malaria among

Malaria remains a major problem among the inhabitants of the endemic region, creating a diagnostic dilemma for the unaccustomed physicians with erroneous diagnosis in certain cases.

MAJOR GENERAL ANIS WAIZ (Retd)
Principal, Bangladesh Medical College,
Former Consultant Physician, Bangladesh Armed Forces

J Bangladesh Coll Phys Surg 1993; 11: 38-39

References:

- WHO sixth report on the world health situation 1980, Geneva.
- Waiz A, Hossain R, Hossain M.Review of Malaria situation in a special Risk group. BAFMJ 1989; 13: 12-18.
- World Health Organisation, Technical Report Series No 680, 1982 (Malaria Control and National Health Goals. Report of the seventh Asian Malaria Conference).
- Waiz A. Malaria: Magnitude of the problem in Bangladesh. Editorial, Bangladesh Med J 1984; 73: 47-50.

- Cook GC. Prevention and treatment of Malaria, Lancet 1988; i: 32-37.
- Krugsted BJ, Herualdt BL. Chemoprophylaxis and treatment of Malaria. N Eng J Med 1988; 319: 1538-1540.
- Waiz A, Chakraborty B. Malaria: An analysis of 55 cases, Bang Med Resch Coun Bull 1990; 15 (2): 46-51.
- Waiz A, Chakraborty B. Malaria: Ten years retrospective study. BAFMJ 1986; 1(XI): 7-11.
- Res P. Dexamethasone deliterious in Malaria. BMJ 1983; 286: 978.
- 10. Warrell DA. Looaraesuwan S, warrell MJ et al. Dexamethasone proves deliterious in cerebral malaria. A double blind trial in 100 comatose patients. N Eng J Med 1982; 306: 313-319.
- Waiz A, Hossain R, Cotrimoxazole and Tetracycline in the management of Cerebral malaria, A review of 254 cases. BAFMJ 1992.
- White NJ, Miller KD, March K et al. Hypoglycaemia in African children with severe malaria. Lancet 1987; i: 708-711.
- Waiz A, Siddique M, Hossain M. An analysis of 7318 Malaria cases. BAFMJ 1990: 14: 15-19.

4520 Cases of Malaria in Chittagong Hill Tracts —A Clinical Analysis

ANIS WAIZ FCPS, FRCPI, FRCPED[®]. RABIUL HOSSAIN FCPS[®].

BARENDRA CHAKRABORTY FCPS[©]. MD SHAHJAHAN MBBS^d

Summary:

In a prospective study the authors analysed 4520 cases of malaria in an endemic zone during a three years period. Most of the cases (99.03%) were of falciparum malaria. Only 44 patients (0.97%) were detected to have vivax variety. Majority of the cases (80.80%) of falciparum malaria responded to chloroquine and the remaining (18.86%) did not. Quinine was administered in nonresponding cases. Two cases were

found resistant to quinine and were successfully treated with Mefloquine. One hundred and forty five cases of cerebral malaria were treated during this period, out of which 18 patients died. Case fatality was around 12.41%. June, July and August are the three months which appear to be the peak season for cerebral malaria. Majority of the (40.14%) total cerebral malaria cases occurred during this period.

(J Bangladesh Coll Phys Surg 1993;11: 40-43)

Introduction:

Malaria remains one of the major health problems in the tropics. In Bangladesh, malaria is hyperendemic in Chittagong Hill Tracts. Chittagong Hill Tracts has been proved to be an insurmountable and impenetrable bastion of plasmodium falciparum and its consort anopheles dirus. The unique terrain offers the ideal conditions appropriate for continuation of virulent species. Dense forest, monsoon rains, abundance of streamlets and rivulets and the presence of illiterate and economically backward tribal population helps in the perpetuation of malarial species. Semi-immune and non-immune individuals from other areas of

Bangladesh who have to reside in this malaria zone are subject to increased mortality and morbidity due to malaria as compared to the local residents¹. Malaria has become one of the major medical problems in South East Asia, part or Africa and Central and south America, specially because of the emergence of drug resistant strains of *P. falciparum*². Falciparum malaria can lead to dreadful complications like cerebral malaria which has a 10-40% risk of mortality³. There is no effective chemoprophylactic agent against *P. falciparum*. So active treatment of clinical cases of malaria rather than mass prophylaxis needs fresh consideration^{4,5}.

The present study is a simple analysis of malaria cases attending a peripheral hospital. Efforts have been taken to find out the types of the disease and their treatment response.

Maj. Gen. (Dr.) Anis Waiz, Consultant Physician, Bangladesh Armed Forces.

Correspondence to :

Maj. (Dr.) Barendra Chakraborty FCPS. Classified Specialist in Medicine Dhaka Cantonment.

Received: May 31, 1992, Accepted: January 10, 1993.

Materials and Method:

This is a prospective study of all malaria cases admitted in Combined Military Hospital, Chittagong during the period January 1989 to December 1991. A total of 4520 cases of malaria were studied. All patients were from hill areas. Diagnosis was established on clinical suspicion and confirmed by the microscopic demonstration

Lt. Col. (Dr.) Rabiul Hossain, Classified Specialist in Medicine, CMH Chittagong.

C. Maj. (Dr.) Barendra Chakraborty, Classified Specialist in Medicine, CMH Dhaka.

d. Maj. (Dr.) Md. Shahjahan, Graded Specialist in Medicine, CMH Chittagong.

of malarial parasite in peripheral blood. Diagnosis of cerebral malaria was established on clinical criteria which includes history of residence in endemic area, fever, alteration of level of consciousness, abnormal behavior, convulsion and finally by laboratory demonstration of peripheral parasitaemia. Other causes of coma were excluded by careful history taking and relevant investigations. Cerebrospinal fluid (CSF) examination was done in all the cases of cerebral malaria. Initial treatment was uniform for all patients with uncomplicated malaria. After confirmation of diagnosis, all patients were treated with chloroquine 600 mg of initial dose followed by 300 mg after six hours and then 150 mg twice daily for next three days. A single dose of 150 mg sulphamethoxazole and 75 mg pyrimethamine was given to all falciparum malaria cases on fifth day and a single dose of 45 mg primaquine to vivax malaria patients on completion of dose of chloroquine. Quinine resistant cases were treated with single dose of mefloquine 750 mg provided by courtesy of Roche Pharmaceuticals. Cerebral malaria cases were treated with triple therapy including intravenous quinine dihydrochloride 10 mg/kg of body weight eight hourly in 5% dextrose in aqua, parenteral tetracycline 250 mg eight hourly along with cotrimoxazole (800 mg sulphamethoxazole + 160 mg trimethoprim) twice daily. Parenteral quinine and tetracycline were continued till the patients became conscious and were able to retain oral medication. Duration of treatment in cerebral malaria ranged from 7-10 days.

Results:

Out of total 4520 cases of malaria 4476 (99.03%) cases were of falciparum variety and 44 patients (0.97%) had vivax type (Table-I). Chloroquine is still effective in the treatment of uncomplicated malaria. 3617 (80.8%) cases of falciparum malaria responded to chloroquine and only 857 (18.86%) patients required administration of quinine (Table-II). In this series

mortality from cerebral malaria was 12.41 % (Table-III). June, July and August appear to be the peak season for cerebral malaria. Out of total 145 cerebral malaria cases. 40.14% occured during these three months. C S F pressure was found raised in only 13 cases (8.97%) of cerebral malaria. None had papilloedema. C S F examination was unremarkable in all cases of cerebral malaria.

Table-ITypes of Malaria (n=4520)

Year	Falciparum (%)	Vivax (%)	Total
1989	1360	26	1386
	(98.12)	(1.88)	
1990	1369	11	1380
	(99.2)	(0.8)	
1991	1747	7	1754
	(99.6)	(0.4)	
	4476	44	4520
	(99.03)	(0.97)	

Table-II Chloroquine sensitivity of falciparum malaria (n-4476)

Year	Responded (%)	Not responded (%)	Total
1989	1106	254	1360
	(81.32)	(18.67)	
1990	1077	292	1369
	(78.67)	(21.33)	
1991	1434	313	1747
	(82.88)	(17.92)	
	3617	859	4476
	(80.8)	(19.19)	

Table - IIIDeath due to cerebral malaria (n-145)

Year	Total cerebral malaria cases	No. of death	Percentage
1989	33	6	18.18
1990	57	5	8.77
1991	55	7	12.73
Total	145	18	12.41

Discussion:

Malaria has become a major public health problem for the people of Chittagong Hill Tracts. No chemoprophylactic agent appears to be effective in preventing falciparum malaria6. In our series 99.03% patients had falciparum malaria. This indicates falciparum malaria has a firm root in Chittagong Hill Tracts. 80.8% of falciparum malaria in this study was chloroquine sensitive. It is in agreement with another study done in recent past in Bangladesh where 85% of falciparum malaria cases were found chloroquine sensitive7. So authors predicts that chloroquine is still effective and the treatment of choice for cases of uncomplicated malaria. However, quinine has to be used in case of complicated falciparum malaria and also in chloroquine resistant cases. In this series 145 patients of falciparum malaria developed cerebral malaria, attack rate is 3.24% which is higher than other reports available. In a recent study done in Malawi attack rate is about 1%8. In the present study death rate from cerebral malaria is about 12% which is much lower than recently published studies. A study done in Kenya showed fatality to be 20%9. Another study conducted in Chittagong Hill Tracts during the period 1986 to 1988, fatality was about 18%10. It may be mentioned that since 1989, authors are using triple therapy (quinine, tetracycline and cotrimoxazole) in the treatment of cerebral malaria 11. So it may be concluded that reduction

in mortality from cerebral malaria may be due to benificial effect of co-administration of tetracycline with quinine and cotrimoxazole. The effectivity of cotrimoxazole in the treatment of falciparum malaria has already been established with a cure rate of about 98%12. Tetracycline is a slow acting blood schizonticide effective against all species of malaria parasites. it has also some tissue schizonticidal action against P. falciparum13. Raised intracranial pressure was not found to be a predominant feature in crebral malaria in this study. This view is in aggrement with the results of other studies that raised intracranial pressure and cerebral oedema are uncommon in adults with cerebral malaria14,15. In our series two cases did not respond to quinine and were treated with mefloquine. Emergence of quinine resistant falciparum may be an alarming phenomenon for our country. Recently published data reveal that although there is no unequivocal case of a complete failure of quinine, resistance is increasing. In South-East Asia especially, there is a real possibility that falciparum malaria could become untreatable within this decade 16.

Although the incidence of malaria in general population in our country is unknown, but malaria is a common health hazard for visitors to a malaria zone. Falciparum is the predominant type of malaria in hill tracts and it is the most dangerous strain of malaria because of it's unpredictable clinical presentation. It can lead to cerebral malaria which carry high risk of fatality. Therefore, falciparum malaria warrants early intervention and skilled treatment. Clinicians working in an endemic zone must be aware of unpredictable clinical presentation of falciparum malaria, so that correct diagnosis and management may be accorded.

References :

 Waiz A, Chakraborty B. Malaria: Tenyears retrospective study. Bangladesh Armed Forces Medical Journal 1985; 11: 7-11.

- Cook GC. Prevention and treatment of malaria. Lancet 1988: 2: 32-37.
- Bruce Chwatt LT. Recent trends of chemotherapy and vaccination against malaria. Br Med J 1985; 91: 1072-76
- Krugsted BJ, Herualdt BL. Chemoprophylaxis and treatment of malaria. N Engl J Med 1988; 319: 1536.
- Peter W, Hall AP. Treatment of severe falciparum malaria. Br Med J 1985; 1291: 1146-47.
- Cook GC. Plasmodium infection; problems in prophylaxis and treatment. Quart J Med 1986; 61: 1091-1115.
- Waiz A, Siddique M A, Hossain SM. An analysis of 7318 malaria cases. Bangladesh Armed Forces Medical Journal 1990; 14: 14-19.
- Molyneux ME, Taylor TE, Thomas CG et al. Efficacy of quinine for falciparum malaria according to previous chloroquine exposure. Lancet 1991; 337: 1379-80.
- Newton CRJC, Kirkham FJ, Winstaley PA et al. Intracranial pressure in African children with cerebral malaria. Lancet 1991: 337:573-76.

- Waiz A, Chakraborty B. Cerebral malaria, an analysis of 55 cases. Bangladesh Medical Research Council Bulletin 1990; 16 (2): 46-57.
- Waiz A, Hossain R, Chakraborty B et al. Quinine, Cotrimoxazole and Tertracycline in management of cerebral malaria. Abstract Book. Royal college of Physicians of Ireland 1992; 178.
- Peter BR, Hedd JC, Khazembe P et al. Cotrimoxazole for childhood febrile illness in malaria endemic regions. Lancet 1991; 337: 518-20.
- Warensdorfer WH, McGregor I. Principles and practice of malariology 1988; Ist ed. Charchill Livingstone: pp 223-25.
- Looareesuwan S, Warrell DA, White NJ et al. Do patients with cerebral malaria have cerebral oedema? A computed tomography study. Lancet 1983: 13: 434-37
- Warrel DA, Looareesuwan S, Philips RE et al. Function of the blood cerebrospinal fluid barrier in human cerebral malaria: rejection of the permeability hypothesis. Am J Trop Med Hyg 1986; 35:88-89.
- Editorial. Rediscovering wormwood qinghaosu for malaria. Lancet, 1992; 339: 649-51.

Acute Cerebrovascular Disease—Some Clinical and **Epidemiological Correlates**

HAM NAZMUL AHASAN FCPS*, HALIMA KHATUN MBBSb, SAIFUL ISLAM MBBSc, MERINA ARA MBBSd. GOUTAM KUMAR SHAHA MBBSe

Summary:

A prospective hospital based study on the clinical and epidemiological aspects of acute cerebrovascular disease was undertaken. Acute cerebrovascular disease was found to be common from fifth to seventh decades of life, the mean (±SD) age being 58 (±13) and 51 (±14) years for males and females respectively. It was twice as common in men as in women. Service holders, businessmen and housewives were common victims of this disease. A positive family history of

Introduction:

Cerebrovascular disease (CVD) is presently the third leading cause of death in adults and accounts for a large segment of the problem of chronic illness and disability in the society1.

This paper reports the findings of prospective study designed to find out the common epidemiological and clinical features of cerebrovascular disease. From an investigation of the personal traits, habits and environment of those who have developed acute erebrovascular disease, it is possible to identify susceptible individuals and assess the factors which appear to predispose to cerebrovascular disease.

Materials and Method: The patients:

All of the 100 consecutive cases of acute cerebrovascular disease admitted into the cerebrovascular disease was found to be significantly related to future development of this condition. Acute cerebrovascular disease commonly developed when the patients were in sleep. Arcus senilis in both sexes and smoking in men were found to be significant clinical markers, whereas physical inactivity and obesity did not appear to be contributory factors. Amongst the various systemic diseases, hypertension was found to be the commonest association.

(J Bangladesh Coll Phys Surg 1993:11:44-47)

General Hospital, Khulna during the period June, 1989 to February, 1991 were studied to find out some epidemiological information like age, sex, occupation, family history, smoking habits, physical inactivity, body weight of the patients and also to determine the association of CVD with systemic conditions like hypertension, diabetes mellitus etc. Glucose tolerance test was done in all cases.

The controls:

During the same study period, 100 (70 male, 30 female) clinically healthy controls were studied for comparison. The controls were selected from the relatives of other ward patients admitted for non-neurological diseases. None of the controls did suffer from hypertension, diabetes mellitus. ischaemic heart disease or any other systemic diseases. The mean (±SD) age of the controls was 51 (±11) and 49(±9) years for males and females respectively.

Statistical analysis:

Chi-square test was applied to determine the significance of difference where applicable. Statistical significance was considered when p<0.05.

Results:

The age and sex incidence of 100 patients is shown in Table-I. The mean (±SD) age of the

Correspondence to :

HAM Nazmul Ahasan

Assistant Professor (Medicine), Rajshahi Medical College,

Received: September 27, 1992 Accepted: April 9, 1993.

a. HAM Nazmul Ahasan, Cosultant, General Hospital, Khulna (now working as Assistant Professor, Rajshahi Medical College, Rajshahi.)

b. Halima Khatun, Medical Officer,

c. Saiful Islam, Medical Officer,

d. Merina Ara Medical Officer

e. Goutam Kumar Shaha, Medical Officer, Medicine Unit, General Hospital, Khulna, Bangladesh.

patients was 58 (±13) and 51 (±14) years for males and females respectively. Maximum number of cases (75%) were found between 41 and 70 years. Males comprised 67% as opposed to 33% females giving a 2:1 ratio.

Table-IAge and sex distribution of CVD patients (n=100)

Age group in years	Male	Female	Total (%)
21-30	1 1	2	3
31-40	5	7	12
41-50	16	10	26
51-60	18	7	25
61-70	20	4	24
71-80	5	2	7
81-90	2	1	3
Total	67	33	100

In this study, out of 33 females, 30 were housewives and rest three were from other occupational groups (Table-II). Service holders (17%) were most commonly affected male victims of actue cerebrovascular disease. Sixteen percent of patients gave a positive family history of cerebrovascular disease as opposed to .3% of controls (Table-III).

In 26 (26%) patients, stroke developed during sleep (Table-IV). In 24% CVD developed when the patients were at work and others had the attacks when they were awake and active.

Incidence of physical inactivity, smoking habits, obesity and arcus senilis in patients and controls is shown in Table-V. Sixty one percent of CVD patients were hypertensive and 10% diabetic (Table-VI). Further 8% had both hypertension and diabetes mellilus. In 14%, no systemic disease was evident.

Table-IIOccupational distribution of CVD patients
(n=100)

Occupation	Number of
	patients
Housewife	30
Service holder	17
Businessman	13
Cultivator	12
Retired from service	8
Different categories of	
medical practitioners	4
Mill worker	4
Teacher,	2
Advocate	2
Rickshaw puller	2
Machine operator	2
Day labourer	2
Others	2

Table-IIIFamily history of CVD in patients and controls

Study group	Positive family	Negative family
	history	history
Patient (100)	16	84
Control (100)	3	97

Numbers in the parentheses indicate the number of patients/controls. X^2 (with Yate's correction)=8.38; P<0.01

Table-IVTime of occurrence of CVD (n=100)

Time of occurrence	Number of patients
During sleep	26
During work	24
During rest	6
During meal	6
On road	6
At toilet	4
During agitation	4
During various other activities	24

Table-VOther clinical markers in CVD patients and controls

Clinical markers	Patient (n=100)	Control (n=100)	Statistical significance
Physical inactivity	64	55	NS
Obesity	15	15	NS
Smoking	36	25	NS
			(but signifi- cant, only when male smokers are taken into
Arcus senilis	19	7	account) Significant

NS= not significant

Table-VISystemic diseases associated with CVD (n=100)

Diseases	Male	Female	Total	
Hypertension	42	10	62	
Diabetes mellitus	9	1	10	
Hypertension and diabetes mellitus together	5	3	8	
Rheumatic valvular heart disease with atrial fibrillation	1	2	3	
Ischaemic heart disease	2	1	3	
Normotensive (without any systemic disease)	7	7	14	
Congenital heart disease (Fallot's	1	0	1	
tetralogy)				

Discussion:

Cerebrovascular disease is one of the leading causes of death in many countries. Several factors have been reported to increase the risk of cerebrovascular disease^{2,3}. The mean age of

the patients of this study was lower than that of the western findings but was consistent with studies from India4. The reason behind early development of CVD in this su continent is perhaps due to (i) lower life expectancy of the people of this region and/or (ii) failure to recognise the risk factors of CVD earlier and to take appropriate preventive measures. The sex distribution of the present study is not consistent with other studies⁵, where an equal distribution in both sexes were found. The predominance of the male patients in this study was most probably due to the fact that (i) commonest risk factors of CVD like hypertension and diabetes mellitus were found three times more common in men than in women in the present study (Table-VI), (ii) women are not equally represented due to less number of hospital beds for the female patients and womenfolk is to some extent neglected in poor developing countries like ours. Occupational distribution of CVD patients reveals that 91% females were housewives. This is due to the fact that the socio-cultural and religious context of our society hardly allows the females to engage in outwardly occupation. Amongst men, service holders, businessmen and cultivators were commonly affected by CVD. History of CVD in the family appeared to be important for future development of stroke and the findings are consistent with other studies6. It is likely to be the result of inherited tendency of the risk factors of CVD like hypertension, diabetes mellitus, hyperlipidaemia etc. to be expressed. In the present study, in 26% of the patients, stroke developed during sleep and the finding is consistent with other studies7. The cause of development of stroke during sleep is probably secondary vasoconstriction as a consequence of physiological hypotension. hypoxaemia or some other unknown reasons8. Presence of arcus senilis in both sexes and smoking habit in men appeared to be significant whereas physical inactivity and obesity were insignificant clinical risk markers as it was found in other studies9. Like many other studies 10,11, hypertension was found to be the commonest systemic disease associated with CVD. Glucose tolerance test revealed a close relationship of the elevated levels of blood sugar with cerebrovascular disease. Besides 18 patients who were diabetic, in a further 10 cases, impaired glucose tolerance was observed. Similar observation was reported by other authors 12.

Thus, despite many limitations in a peripheral district hospital, our findings point out the presence of certain risk factors of cerebrovascular disease in Bangladeshi population which need further large scale epidemiological confirmation.

References :

- Kistler JP, Ropper AA, Martin JB. Cerebrovascular disease. In: Wilson JD, Braunwald E, Isselbacher KJ. Petersdorf RG, Martin JB, Fauci AS, (editors). Harrison's Principles of Internal Medicine. New York, 1991: 1977-8.
- Kannel WB, Blaisdell FW, Gifford R, Hass W. Risk factors in stroke due to cerebral infarction. Stroke 1971; 2: 423.
- Nill SE. Economic cost of stroke in Massachussetts. New Eng Med J 1978; 249: 415-18.

- Abraham J, Rao PSS, Inbaraj SG, Shetty G, Jose CJ.
 An epidemiological study of hemiplegia due to stroke in South India. Stroke 1970; 1:477.
- Britton M, Deffaire V, Helmer C. Arrythmia in patients with acute cerebrovascular disease. Acta Med Scand 1979; 25:425-28.
- Alter M, Kulzink J. Genetics of cerebrovascular accidents. Stroke 1972; 3:41.
- Marswall J, Shaw DA. Natural history of cerebrovascular disease. BMJ, 1959; 1: 1614-17.
- Dunbabin DW and Sandercock PA. Preventing stroke by the modification of risk factors. Stroke 1990; 21: 36-9.
- Agarwal JK, Soman PN, Katiyar BC. A study of risk factors in nonembolic cerebrovascular disease. Neurology India 1976; 24: 125-33.
- Aulikki N, Juha P, Seven P, Martti JK. Risk factors for cerebrovascular disease among 55 to 74 year-old Finnish men: a 10-year follow-up. Ann Med 1989; 21: 239-40
- Roy C, Richard P, Stephen M, Patricia H. Blood pressure, stroke and coronary heart disease. Lancet 1990; 335: 827-38
- Poungvarin N, Kanluan I, Chawalithithikul U. Risk factors of cerebrovascular disease in urban community of Thailand. J Med Assoc Thai 1990; 73: 653-7.

Omental Transposition in End-stage Buerger's Disease—Personal Experience

HASAN MOHD, ABDUR ROUF, FCPS (Surgery)

Summary:

Omental transposition in lower extremity was done in twenty five cases of end-stage Buerger's disease as a limb salvage procedure. These patients were candidates for midthigh amputation. Twenty three (92%) patients had symptomatic relief with salvage of lower extremity. Two patiens who had no improvement of progressive gangrenous

change after omental transposition had midthigh amputation later on. The technique of the procedure is easy and rewarding although little time consuming, because a very meticulous dissection is necessary. The procedure is described here along with a short historical background.

(J Bangladesh Coll Phys Surg 1993;11: 48-51)

Introduction:

Leo Buerger (1879-1943), an urologist, first described thromboangitis obliterans (TBA) which is also named after him as Buerger's disease in 1908¹. Since then surgical management of this condition remains problematic. Due to diffuse and intermittent occlusion of small and medium sized vessels, peripheral arterial reconstructive surgery is singularly impossible (only rarely possible). Sympathectomy help the patients but only for a short period of time with recurrence of symptoms in most of the cases.

Since long it is believed that omentum has got some purposeful function. Hippocrates had the idea that in some way greater omentum is responsible for the control of peritoneal fluid. Aristotle believed that omentum protects the abdominal viscera from cold². Senn did an experimental work with omentum in dogs in 1888. His reported results were rewarding and he later used omentum in some of his cases. Same type of experimental work was done by Thomson and Pollock in early part of this century³. Rutherford Morrison, in 1908, called omentum as "abdominal policeman" for its

protective function in various types of abdominal catastrophy and also for its role in sealing off hernial offices and blood supply to detached uterine fibroid by neovascularisation⁴. In 1933 O'Shaughnessy and his associates started cardiovascularisation by omentum in cases of ischaemic heart disease and in 1938 they reported 20 such cases^{5,6}. It was suggested that graft may serve as a bridge between the right and left coronary arteries. Cannady in 1948 used omentum in nonhealing fracture of radius and ulna³. Casten and Alday in 1971 proposed a new method of revascularisation of the lower extremity by omental transfer. The procedure was also used for chronic lymphoedema7. Alday and Goldsmith describing the method of lengthening and extraabdominal mobilization of the omentum, the circulation being maintained by a secure and predictable pedicled omentum8. Since 1980 VK Agarwal in India has been perfoming omental transposition in end-stage Buerger's disease and his result is excellent in 96% cases2. In May 1991, this procedure was started in a surgical unit of Sher-E-Bangla Medical College Hospital and till now 25 cases have been performed with excellent results. This paper presents an account of this

Hasan Mohd. Abdur Rouf Associate Professor of Surgery (Current Charge) Sher-E-Bangla Medical College, Barisal.

Correspondence to :
Associate Professor of Surgery (Current Charge)
Sher-E-Bangla Medical College, Barisal.

Recived: December 7, 1992, Accepted: March 16, 1993

Materials and Method:

work.

Seventy three cases of Buerger's disease were admitted from January 1991 to October 1992 in the surgical Unit-II and IV of Sher-E-Bangla Medical College Hospital. Among those 21 had bilateral lumber sympathectomy few months to few years back. Other 52 had unilateral lumber sympathectomy or no sympathectomy at all previously. All the 21 cases with previous bilateral lumber sympathectomy were candidates for amputation for end-stage Buerger's disease of lower limb either in right or left side. Among the rest 52, two were candidates for amputation with no history of sympathectomy at all. In another two cases sympathectomy of the opposite side of presently affected limb was done and they were also asking for amputation rather than sympathectomy. Clinical features of these 25 cases are shown in table-1.

Table-IClinical features of patients of Buerger's disease.

Cli	nical features N	No. of patients
1.	Burning pain in the foot	25
2.	Intermittent claudication	
	a) Foot claudication	25
	b) Calf claudication	22
3.	Rest pain	25
4.	Superficial thrombophlebitis	16
5.	Oedema	9
6.	Ulceration / Ischaemia of dig	its 20
7.	Gangrene of one or more digi	ts 4
8.	Pulp atrophy	1

All the patients were smoker for about four decades of their life. Twenty three patients (21 both sides, two opposite side) had undergone lumber sympathectomy for Buerger's disease three months to 17 years ago. All of them left smoking after sympathectomy only to start again after improvement of symptoms. So they all were smoker or just left smoking at the time of admission. They were of low socio-economic condition. None was hypertensive or had cardiovascular disease. Blood chemistry were normal (serum chlolesterol and fasting blood

sugar). VDRL was non-reactive in all the cases. Skiagrams did not reveal any arterial calcification in any of the cases. Angiography was not possible in our hospital. The four cases who had no lumber sympathectomy on the affected side were also investigated preoperatively in the same line as the patients of end-stage Buerger's disease.

Surgical anatomy and the operative procedure:

The blood supply to the omentum comes from right and left gastroepiploic arteries. These two arteries form an arcade on the greater curvature of the stomach within the omental apron. From this arcade many small vessels arising out of it supply blood to the stomach. Three othervessels named right, middle and left omental artery also arise from this arcade to supply the omentum.

These vessels form another arcade at the free border of the omentum named Barlow's arcade. Vascular pattern of this arcade is not constant and has got several variations. It is to be mentioned that if this arcade is not at the free border and remains high up then mobilization and lengthening of the omentum becomes problematic. Fortunately this type of high arcade is uncommon.

Abdomen is opened by left paramedian incision. Then vascular pattern of the omentum is checked. Omental lengthening has got three stages. In stage I, omentum is separated from the transverse colon. It should be started from the left side and in the vascular plane. Special precaution should be taken that no blood vessel of the omentum is injured during the procedure. Last part of the separation is difficult as because the right posterior layer of the omentum is attached with the anterior surface of the head of the pancreas. But with patience it is possible to separate it. In stage II, gastroepiploic vascular arcade is separated from the greater curvature of the stomach after ligation and division of the

right or left gastroepiploic artery and the small arteries supplying the stomach depending upon the pedicle to be formed either from the left or right. Arcade will remain within the omental apron. The author prefers the pedicle on the right as because right gastroepiploic artery is larger than the left. After completion of stage II, the omentum can be reached upto midthigh.

In stage III, lengthening of the omentum is done based upon the vascular pattern. This is shown in the diagram. (Fig 1 and 2)

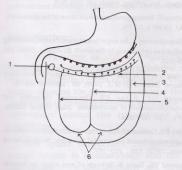


Fig-1: 2nd stage of omental lengthening.

Ligation and division of left gastroepiploic artery and small arteries (supplying greater curvature of stomach) arising from marginal artery:

- 1. Right gastroepiploic artery.
- 2. Marginal artery.
- 3. Left Omental artery.
- 4. Middle Omental artery.
- 5. Right Omental artery.
- 6. Barlow's arcade.

After completion of lengthening, mobilization is done under the subfascial tunnel making multiple transverse incisions, two in the abdomen, three in the thigh and three in the leg. At the level of inguinal crease and knee joint, the lengthened omentum is passed over the fascia, otherwise throughout the route it would be passed into the subfascial tunnel.

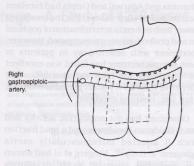


Fig-2: 3rd stage of omental lengthening. Interupted line is the line of division of the omentum depending upon the vascular supply.

In present study, mobilization was possible upto ankle in five cases, in fifteen upto midcalf and in rest five just below the knee joint.

Results :

Symptomatic improvement occurred in 23 cases (92%). Oedema subsided and ulcers healed readily with improvement from coldness, cyanosis, calf claudication and rest pain. Amputation of digits were required in some cases. In one case Syme's amputation and in another mid-tarsal amputation were done afterwards. Two patients had no improvement at all and needed midthigh amputation. However, it is very difficult to predict long term outcome at this moment.

Discussion :

Sympathectomy sometimes gives good result in Buerger's disease, but recurrence is very common^{2,9}. Reconstructive surgery like endarterectomy and by-pass graft are rarely possible⁷ and feasibility of these procedures deemed to be poor^{2,9}. Casten and Alday showed improvement of symptoms with remarkable

healing in 70% of their patients with lower limb ischaemia and Agarwal and Gupta had excellent result in 96% of their cases². For the treatment of lower limb ischemia arterialisation of popliteal vein and profendoplasty are suggested. However, experiences with omentopexy in patients in whom sympathectomy has failed was excellent and rewarding for limb salvation^{8,9}. Our experience is for a very short period of time but, so far, the result is excellent.

Omentum has got angiogenic activity and from it Goldman has discovered a lipid fraction which if injected intramuscularly exerts angiogenic activity 10. Vingerg et al had shown that pedicled omental graft developes anastomoses at capillary level within three days and arteriolar sized channels within eight days 11. Vessels of omentum thus invade ischaemic muscle tissue supplying adequate blood to the muscles and once the anastomosis has developed, the diseased segment of original blood vessles are filled up with blood to supply the ischaemic tissue and the omentum has pronounced capacity to increase vessel size.

While describing the original lengthening procedure Alday and Goldsmith foresaw that the clinical use of the intact omentum would be increasing and they predicted that this trend would continue8. From the world literature it is found that lengthened omentum are now in use for various clinical conditions like end-stage Buerger's disease and atherosclerotic conditions where amputation was the only alternative, for revascularisation of CNS, for lymphoedema, for repair of bronchopleural fistula and oesophageal perforation etc12. Turner Warwick used omentum for reconstructive surgery of urogential system and also for idiopathic retroperitoneal fibrosis leading to hydronephrosis 13. This paper likes to present a small work started from May 1991 based on the lengthening and mobilization procedure of intact omentum for limb salvage surgery.

References:

- Rains AJH, Mann CV (Eds): Bailey and Loves Short Practice of Surgery: 20th edn. ELBS/HK Lewis, London, 1988; pp 232-232.
- Agarwal VK, Gupta RL. Use of omentum in surgery. In: Recent Advances in Surgery - II. Jaypee brothers, New Delhi 1988; pp 199-209.
- Cannady JE. Some uses of undetached omentum in surgery. Am J Surg, 1948; 76: 502-505.
- Morison R. Remarks on some functions of the omentum. BMJ, 1906; 1:76-78.
- O'Shaughnessy L. An experimental method of providing a collateral circulation to the heart. Br J Surg. 1936; 23: 665-668.
- O'Shaghnessy L. The surgical treatment of coronary disease. Edinburgh M J, 1939; 46: (cited in ref.-3).
- Casten DF, Alday ES. Omental transfer for revascularisation of extremities. Surg Gynae Obstr. 1971: 132: 301-304.
- Alday ES, Goldsmith HS. Surgical technique or omental lengthening based on arterial anatomy. Surg Gynecol Obstr. 1972; 135: 103-107.
- Agarwal VK, Bajaj S. Salvage of end stage ischaemic extremity by omentopexy in Buerger's disease. Ind J Thor Cardiovas Surgery, 1988; 5: 12-17.
- Goldsmith HS, Grifith AL, Catsimpoolas N. Increased vascular perfusion after administration of an omental lipid fraction. Surg Gynae Obstr. 1986; 162: 579-586.
- 11. Vineburg AM et al. Myocardial revascularisation by omental graft without pedicle experimental background and report on 25 cases followed 6 months later. J Thorac Cardiovas surg 1965; 49:1965 (Cited in ref. -9).
- Thomson SA, Pollock B. The use of free omental grafts in the thorax. Am J Surg, 1945; 70: 227-231.
- Turner Warwick R. The use of the omental pedicle graft in urinary tract reconstruction. J Urol, 1976; 116: 341-343.

Post-tonsillectomy Haemorrhage—A Study of 1230 Cases

M. ALAUDDIN FRCS, FACSa, KHABIRUDDIN AHMED FCPS (ENTD)b

Summary:

Haemorrhage following tonsillectomy in a series of 1230 patients over an eleven and a half year period have been studied. Thirteen patients had post-operative haemorrhage (1.06%), out of which eight had reactionary and the other five patients had secondary haemorrhage. The low incidence of

haemorrhage is attributed to the use of catgut suturing of the bleeding points instead of ligation or diathermy coagulation and experience of the operating surgeon. Per-operative use of intravenous pethidine and routine use of antibiotic appears to play a favourable role.

(J Bangladesh Coll Phys Surg 1993;11: 52-55)

Introduction:

Tonsillectomy is still a frequently done surgical procedure, but the regional statistics varies in different parts of the world. There are many reasons for this variation and the most important of these is perhaps a genuine lack of agreement about the indications for the operation.

The most important complication of tonsillectomy is haemorrhage and the death associated with tonsillectomy are directly or indirectly related to this complication. In almost all cases the real contributing factor remain masked although several causes have been claimed to be of importance. Thus several authors assert that the surgical technique and the operative experience of the operating surgeon are important. Post-operative infection in the tonsiller bed, menstruation and pregnancy are stated to be predisposing factors².

Haemorrhagic diathesis may naturally cause haemorrhage post-operatively, but in surgical

practice this is of little importance as tonsillectomy is usually contraindicated in such cases³.

The aim of this study was to record the incidence of post-tonsillectomy haemorrhage and to correlate it with some factors especially focusing on the operative experience of the surgeon and using suturing instead of ligating or use of diathermy coagulation for haemostasis.

Materaials and Method:

The operations were performed between July 1979 and December 1990 by the author himself in three different hospitals in the Dhaka city. The indications were:

- a) recurrent tonsillitis, more than six times in a year for two to three years in children;
- b) one or more attack of peritonsiller abscess;
- c) obstructive tonsils causing sleep apnoea;
- d) chronic suppurative otitis media which failed to respond to conservative treatment and where tonsils were thought to be the source of infection;
- e) acute exacerbation of chronic tonsillitis in adult causing loss of valuable working hours.

Correspondence to:

M. Alauddin

Professor & Head of the Department of E.N.T.D. Dhaka Medical College, Dhaka.

Received October 24, 1991. Accepted: February 16, 1993.

M Alauddin, Professor & Head of the Department of E.N.T.D, Dhaka Medical College., Dhaka.

Khabiruddin Ahmed, Registrar, Surgical Oncology Cancer Institute & Research Hospital, Mohakhali, Dhaka.

All operations were done under orotracheal or nasotracheal general anaesthesia. No premedications were used. In case of a frightened child, one of the parents was allowed to remain with the patient during induction. All the patients received injection pethidine through intravenous route (1 mg/kg body weight) along with the anaesthetic agents.

The tonsillectomies were done by blunt dissection and haemostasis was secured by gauze packing and suturing of the bleeding point with atraumatic chromic I/O catgut having 1/2 circle 26 mm needle.

No diathermy coagulation or silk ligation were used for haemostasis. All the patients were extubated on their side with the head down position-the "tonsiller position." Parents were allowed by the side of the patient as soon as the child recovered from the anaesthesia. No postoperative sedation was given in any of the patients. All the patients routinely received antibiotics parenterally peroperatively and continued orally in the post-operative period for a minimum of five days.

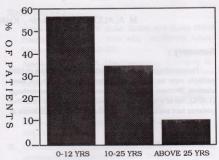
All the children except few with postoperative rise of temperature were discharged from the hospital after 24 hours and adults after 48 hours. Patients with pyrexia were discharged after they had been apyrexial for 48 hours.

Results:

The age of the 1230 patients included in the study was from 20 months to 55 years. Out of them 789 (64.15%) were male and 441 (35.85%) female. Among the males the highest number was in 0-12 years age group (62.99) but in females 13-25 years age group comprised the largest fraction (46.93%) (Fig. I).

Recurrent tonsillitis was the commonest indication and equally represented in both sexes. Obstructive tonsils were solely confined within the 0-12 years age group and mainly in the

TONSILLECTOMY AGE GROUP



AGE GROUP

Fig.1: Age distribution of the patients.

boys. Peritonsiller abscess was an indication predominantly in the 13-25 years age group and here also the males were the usual sufferer. In all the cases of peritonsiller abscess, tonsillectomies were done as a cold procedure after an interval of four to six weeks.

In this series only 13 patients had posttonsillectomy haemorrhage (1.06%), out of which eight were reactionary and the other five were secondary. In case of reactionary haemorrhage, all happened within the first four hours of operation. Reactionary haemorrhage occurred in 0-12 years age group, particularly in the boys, and the secondary haemorrhages mainly in the 13-25 age group equally in both sexes.

All the reactionary haemorrhages were controlled by gargling with ice cold water and removal of clot from the fossa under direct vision. None of the patient needed second anaesthesia and surgical treatment. The secondary haemorrhages were reported by the patients in between the sixth and tenth post-operative day. All of those were minor bleeding and none required second hospitalization.

No other serious complication was recorded in this series.

Table-IIncidence of post-tonsillectomy haemorrhages in patients of different age and sex

Age group	Ma	le la DIA	Fen	nale	То	tal
0-12 years	7/497	(1.40%)	1/186	(0.54%)	8/683	(1.17%)
13-25 years	2/220	(0.90%)	2/207	(0.96%)	4/427	(0.93%)
> 25 years	1/72	(1.39%)	0/48	(0%)	1/120	(0.83%)
Total	10/789	(1.26%)	3/441	(0.68%)	13/1230	(1.06%)

Discussion:

With the advent of modern anaesthesia and blood transfusion system, haemorrhage itself can no longer be responsible as a major cause for post-operative mortality in surgical practice. But this is not true in case of tonsillectomy. Clearly the most important factor and the cause of most of the deaths associated with tonsillectomy is haemorrhage and delay in treating it. In the present series haemorrhage was the only significant complication in the post-operative period and there was no mortality. Haemorrhage occurred in 13 out of 1230 patients (1.06%) amongst which eight (0.65%) were reactionary haemorrages and five (0.41%) were secondary heamorrhages. The incidence reported for post-tonsillectomy haemorrhage varies, ranging from 0.1%4 to 8.1%5. Kristensen and Tveteras2 reported that the range of posttonsillectomy haemorrhage was one to 10 percent. All the operations in the series were performed by the principal author himself and any minor bleeding was also brought to the notice and was recorded meticulously.

In fact, all the haemorrhages were minor and none required surgical treatment under general anaesthesia (G/A). This finding is inconsistent with the retrospective study of Kristensen and Tveteras², who noted a rate of 2.8% posttonsillectomy haemorrhage requiring haemostatic surgery under G/A. The second

anaesthesia for controlling post-tonsillectomy haemorrhage is always hazardous⁶.

Most of the haemorrhages occurred postoperatively within first 24 hours, which is in accordance with the literature. No particular indication for tonsillectomy appeared to be of significance in precipitating post-operative haemorrhage although cold procedure after peritonsiller abscess sometimes causes troublesome bleeding during operation. In one such case blood transfusion was needed during operation. Four patients required obliteration of tonsiller fossa by pillar suturing for haemostasis after tonsil had been removed.

In our series the incidence of post-tonsillectomy haemorrhage is lower than that of many other studies^{8,9,10,11,12}.

The low incidence of post-tonsillectomy haemorrhage in our series is probably attributed to the use of catgut suture instead of ligation of the bleeding point or diathermy coagulation and increasing experience of the operating surgeon. Injection pethidine (1mg/kg body weight) along with the anaesthetic drugs is also appeared highly beneficial in preventing post-operative haemorrhage. In the post-operative period the sedative action of pethidine wears off but the analgesia persist which keeps the child quiet. Peroperative and post-operative use of antibiotic probably has got a role in preventing secondary haemorrhage, as secondary

haemorrhage in this series is one of the lowest so far reported. In our obsevation suturing of the bleeding point with catgut does not increase the post-operative pain. Rough dissection through improper plane not only causes excessive bleeding during operation but also increases post-operative scarring in the fossa.

None of the patient in our series required second hospitalization for secondary haemorrhage which reduces the cost of the treatment also.

Acknowledgement:

We are grateful to Dr. B.H. Siddiquee, Registrar, E.N.T.D. Dhaka Medical College Hospital, Dhaka, for his help in preparing this paper.

Referrences:

- Hibbert J. Acute infection of the pharynx & tonsils. In: Scott Brown's Otolaryngology. 5th edn. Vol-5. Butterworths, London. 1987, pp 76-98.
- Kristensen S, Tveteras K. Post-tonsillectomy haemorrhage. Aretrospective study of 1150 operations. Clin Otolaryngol, 1984; 9: 347-350.
- Segal C, Berger G, Basker M, Marshak G. Adenotonsillectomies on a surgical day clinic basis. The laryngoscope, 1983; 93: 1205-1208.

- Allen TH, Steven IM, Sweene DB The Bleeding Tonsil-Anaesthesia for control of Haemorrhage after Tonsillectomy. Anaesth Intensive care, 1973; 1(6): 517-520.
- Kerr AlG, Brodie SW. Guillotine Tonsillectomy: Anachronism or pragmatism. J Laryngol Otol., 1978; 92(4): 317-323.
- Davis DD. Reanaesthetising cases of tonsillectomy & adenoidectomy because of persistent post-operative haemorrhage. Brit J Anaesthesia, 1964; 36: 244-49.
- Mawson SR. Disease of the Tonsils & Adenoids (excluding neoplasm). In: Scott Brown's Diseases of the Ear, Nose & Throut, 4th edn, Vol-4, Butterworths, London, 1979; pp 123-170.
- Carithers Jeffrey S, Gebhart Don E, Williams Jill A, Columbus. Post-operative risks of pediatric Tonsilloadenoidectomy. Larynogoscope, 1987; 97: 422-29.
- Carmody D, Vamadevan T, Cooper SM. Posttonsillectmy haemorrhage. Laryngol Otol. 1982; 96: 635-38.
- Kennedy KS, Strom CG. A comparison of postoperative bleeding incidence between general & local anaesthesia Tonsillectomies. Otolaryngology-Head & Neck surgery (Abstract). 1990; 102(6): 654-7.
- Stage J, Jensen JH, Bonding P. Post-tonsillectomy haemorrhage & analgesics. A comparative study of acelylsalicylic acid & paracetamol. Clin Otolaryngol (Abstract), 1988; 13(3): 201-4.
- Guida RA, Mattucci KF. Tonsillectomy & Adenoidectomy : an inpatient or outpatient procedure?. Laryngoscope (Abstract), 1990; 100 (5): 491-3.

Use of Monoclonal Antibody in Cancer Therapy Problems and Perspective

REZWANUL HOQUE MBBS; FCPS (surgery)^a NAZMA AKHTER B.Sc (Hons), M.Sc; Mphil (Biological Science)^b MD. ASLAM HOSSAIN MBBS^c

Introduction:

Modern era of cancer chemotherapy began in 1941 when Huggins and Hodges demonstrated that administration of diethylstilbestrol ameliorated carcinoma of prostate1. Zbar et al demonstrated that intradermal tumour could be regressed and lymphnode metastasis could be prevented in experimental animals by intradermal injection of living Mycobacterium bovis2. Active non-specific immunotherapy began with the use of BCG, methanol extracted residue of BCG(MER), Corynebacterium parvum etc3. Active specific immunotherapy involving immunization of the patient with either unmodified or modified (e.g. Neuramidinase treated) tumour cells or extracts of tumour cells either alone or with an adjuvant such as BCG were used in the early seventies4. Adoptive immunotherapy using cells or products of the immune system have been used to restore or augment general immunocompetence⁵. Passive immunity involves administration of antitumour antiserum or purified antibodies. The antitumour antibody can be cytotoxic or in some cases have been shown to augment lymphocyte or macrophage mediated cellular destruction.

The 'Sinkovics hybridoma' was the earliest concept of natural hybridoma recognised in1967-19686. The technique for making hybridoma was described in 1975 by Cesar Milstein and Georges Kohler in England7. Hybridomas are produced by fusing B cells, which are genetically programmed for producing a particular (single) antibody, with a cancer cell, which is capable of proliferating rapidly under laboratory conditions. Cesar Milstein and Georges Kohler in their experimental model injected antigen into a mouse whose B cells began to produce antibodies against that antigen. They then removed the mouse's spleen which contained many B cells. These cells were incubated with cancerous tumour cells derived from myeloma tumours of bone marrow, some of the B cells fused with the tumour cells to produce the hybrid cells. The hybrids are screened to select ones that produce a desired antibody. Grown in culture, the descendants of 'clones' of a single hybridoma cell continued to produce only the type of antibody characterstic of the parent B cell, hence the name 'monoclonal antibody'. The hybridoma formed is a permanent union. Hybridoma cells are uniform, highly specific and can readily be produced in large quantities7.

The recent advances in the molecular biology immunogenetics have opened up numerous new facets for the use of monoclonal antibody. Monoclonal anti-T cell antibodies have been used to delineate different subpopulation of T-lymphocytes and in diagnosis of various disorders of immune system⁸. The success of monoclonal antibodies in clinical practice is dependent on good design. Finding a suitable target is the most important part as other

- Rezwanul Hoque, Resident Surgeon 200 Bedded Hospital, Narayangani, Bangladesh
- Nazma Akhter, Research Fellow in Molecular Biology, Department of Zoology, University of Dhaka, Bangladesh
- Md. Aslam Hossain, Medical Officer, Tongibari Health Complex, Munshiganj, Bangladesh

Received: 18 March 1993. Accepted: 24 April 1993.

Correspondence to :

Rezwanul Hoque, Resident Surgeon 200 Bedded Hospital, Narayanganj, Bangladesh properties of the antibody can be altered by genetic engineering. Antibodies that target lymphocyte antigens offer less toxic immunosuppressive treatment than currently available drugs and the first monoclonal antibody approved for human use is an immunosuppressive agent for treating rejection of renal transplants. Human trials of monoclonal antibodies to treat septic shock have been done and antibodies are also being developed to target common pathogens such as Herpes simplex virus9. Anticancer antibodies have had a long history in the management of cancer, with major applications having been shown in the immunohistochemistry and immunoassay of Tumour-associated antigen markers. With the advent of hybridoma derived monoclonal antibodies, attempts to use these more reproducible reagents in vivo for cancer detection and therapy have been intensified. Radiolabelled monoclonal antibodies appear to be gaining a role in the management of cancer by means of imaging methods to detect sites of increased radioactivity and several products have been developed and tested clinically 10. Monoclonal antibody have also been used in surgical specimen to detect specific factors for assesment of the nature of the tumour and prediction of prognosis. Brem et al have examined surgical specimen from 52 human brain tumour by immunocytochemical method using a murine (foreign) monoclonal antibody for detection of basic Fibroblast growth factor (bFGF). The presence of bFGF predominantly within the tumour microvasculature indicates a cellular depot for this potent growth factor that mediates angiogenesis and tumorogenesis and a role in the transition from the benign to the malignant phenotype¹¹. Yu et al used murine IgG3 monoclonal antibody (MAb) IH10 which recognises a tumour-associated antigen expressed on the surface of more than 40% of human cervical carcinoma tissues for in vivo localisation and therapy of cervical tumour xenograft in experimental tumour system using immunohistochemical staining method ¹². Sarkis et al have used mouse monoclonal antibody PAb 1801 to detect mutation of P53 tumour suppressor gene (TP53) which is a common occurrence in superficially infiltrative transitional cell carcinoma (T1-TNM) of the urinary bladder. These results suggested that T1 bladder cancers exhibiting nuclear over expression of P53 proteins have a higher probability of disease progression ¹³.

Immunohistochemical detection of mutant P53-suppression gene product have also been used in breast cancer to evaluate the influence on metastasis free survival14. Qian et al have used intraperitoneal injection of 131 I-COL 183B2 monoclonal antibody for radioimaging and clinical evaluation of ovarian carcinoma 15. Present diagnostic techniques do not allow the detection of early metastatic spread of tumour cells, although this spread largely determines the clinical course of patients with small primary cancers. By use of monoclonal antibody CK2 to the epithelial cytokeratin (intermediate filament) component number 18 (CK18) individual disseminated carcinoma cells present in bone marrow of patients can now be identified. CK18 cells present in the bone marrow of cancer patients with apparently operable non-small cell lung cancer exhibit the potential to form solid metastasis and may be used to determine the risk of early relapse in operable non-small cell lung cancer with potential consequences for adjuvant therapy16. Sundin used 125 Ilabelled anti-carcinoembryonic antigen (CEA) monoclonal antibody (MAb) preparation I-38S1, AEC38 and 99-16 for Radioimmunolocalisation (RIL) and Radioimmunotherapy (RIT) of colonic cancer using experimental model of human colonic cancer xenografted in nude rat17. Tumour - associated antigen can be seen as unique targets for the delivery of anti-cancer therapy. Monoclonal antibodies directed at such antigens are being increasingly seen as important biological reagents that will complement the group of existing cytotoxic drugs. Different types of immunoconjugates consisting of combination of monoclonal antibody plus radioisotopes and anticancer drugs, toxins. interferon or cytokines, hormones and anticancer antibiotics have been developed for this purpose. The rationale of using these immunoconjugates remains in the fact that these agents will be delivered at the specific target sites in maximum concentration because of the selective binding of monoclonal antibody to the particular target antigen.

Tumour associated antigen, cell membrane glycoprotein, intracellular substrates, gene and its products, growth factors, membrane receptors etc. have been considered as target for immunoconjugate therapy. The selective binding of these immunoconjugates also reduces the systemic side effects of these drugs and potentiates their effects.

R24 is a mouse IgG3 monoclonal antibody that reacts with ganglioside GD3 expressed by melanoma cells and other cells of neuroectodermal origin (e.g. adrenal medulla). The clinical use of this agent was limited by the development of urticarial rashes, vascular leak syndrome, serum sickness and lymphopaenia 18. Extensive research were undertaken to find out better alternatives with less side effects and minimal immunological reaction. Goodman et al have compared chimeric (human-mouse) monoclonal antibody L6(ChL6) to a murine (foreign) L6 monoclonal antibody directed against a tumour-associated cell surface antigen expressed in non-small cell lung, colon and breast cancer for side effects, localisation of antibody to tumour cells, pharmacokinetics and the development of antibodies against Ch L6. Chimeric L6 appears to be a more suitable antibody for delivering anti-tumour agents because of its low immunogenecity and favourable in vivo tumour binding characterstics 19. The host immune response to the immunoconjugates have led to the development of bispecific monoclonal antibody. Bruynck et al developed a humanised bispecific

monoclonal antibody (bsMAb) with binding specificity for Carcinoembryonic antigen(CEA) on one arm and a radiolabelled chelate (DTPA-90Y) generated by consecutively transfecting the humanised genes of an anti-CEAMAb and the chimerised genes of an anti-chelate MAb into eukaryotic BHK cells using the calciumphosphate co-precipitation technique. This humanised bsMAb may be employed in two phase radioimmunotherapy binding to the tumour via the anti-CEA arm and localising a radiolabelled chelate with the other arm, without inducing a strong immune response observed sometimes with murine MAbs20. Papanastassiou et al successfully used intracavity injection of radiolabelled monoclonal antibody (131-IMAb) for the treatment of recurrent and cystic malignant gliomas21. Greiner et al showed that the radioimmunotherapeutic efficacy of radiolabelled anticarcinoma monoclonal antibody (131-I-CC49) when given in combination with gammainterferon (IFN-gamma) resulted in a statistically significant improvement in therapeutic efficacy when compared with 131-I-CC49 alone²². Kitamura et al showed that intratumoural injection of an antibody drug conjugate MAb-A7-neocarcinostatin (MAb-A7-NCS) resulted in complete remission of established colorectal cancer in five of six antigen-positive xenograftbearing mice. These findings indicate that local injection of immunoconjugate is a promising new field for controlling the local recurrence of colorectal cancer23

Immunotoxins are hybrid proteins in which the potent cytocidal action of a toxin is harnessed for the selective destruction of target cells by attachment to a specific monoclonal antibody or growth factor²⁴. Kreitman et al showed that recombinant toxins containing the variable domains of the anti-Tac monoclonal antibody to the interleukin-2 receptor kill malignant cells from patients with chronic lymphocytic leukaemia²⁵. Mickison et al showed that pseudomonas exotoxin conjugated to

monoclonal antibody MRK 16 specifically kills multidrug resistant cells in cultured renal carcinoma and in MDR-transgenic mice26. A recombinant immunotoxin consisting of Fv region of monoclonal antibody PR1 and a truncated form of pseudomonas exotoxin have been found to exert anticancer effect in prostatic adenocarcinoma²⁷. Antibody 15A8, an immunoglobulin G1 (IgG1) subclass anti-human breast carcinoma murine monoclonal antibody and gelonin, a plant toxin conjugates have been found effective against human carcinoma of breast in vitro studies. The chemotherapeutic agents 5-FU, vincristine and bleomycin and the biological response modifier interferon-alpha and tumour necrosis factor-alpha were shown to augment 15A8-gelonin cytotoxicity. Should in vivo pharmacology and therapeutic studies confirm these in vitro findings, 15A8-gelonin conjugate may be a potent agent for therapy of cancer in man28. 131-I-labelled novel antimucin monoclonal antibody 2G3 have been successfully used in symptomatic chemotherapy-resistant malignant associated with carcinoma of ovary and breast 29.

Although monoclonal antibodies against cancer have been much heralded, their success have been limited by the poor access to the inside of tumours. Treatment of blood cancers has been more successful. Encouraging results have been seen in clinical studies using different immunoconjugates and biological response modifier combination especially in lymphoproliferative disorders. As knowledge about natural immune responses and antibody engineering increases, many more monoclonals are likely to feature in clinical practice.

References :

 Huggins C and Hodges CV. Studies on prostatic cancer, the effect of castration, of oestrogen and androgen injection on serum phosphatases in metastatic carcinoma of the prostate. Cancer Res, 1941: 293-97.

- Zbar B, Bernstein I, Bartlett G, Hanna M(Jr), Rapp H. Immunotherapy of cancer-regression of intradermal tumour and prevention of growth of lymphnode metastases after intradermal injection of living Mycobacterium bovis. J Nat Cancer Inst. 1972; 49: 119-30.
- Morton D, Eilber F, Holmes E et al. BCG immunotherapy of malignant melanoma: summary of a 7 year experience. Am surg. 1974; 180: 635-40.
- Powles R, Crowther D, Bateman C et al. Immunotherapy for acute myelogenous leukaemia. Br J Cancer, 1973; 28: 365-76.
- Thompson RB and Mathe G. Adoptive immunotherapy. Transpl Rev, 1972; 9: 54-72.
- Wainwright M. The 'Sinkovics Hybridoma'-the discovery of the first 'Natural hybridoma'. Perspect Biol Med 1992; 35(3): 327-9.
- Milstein C. Monoclonal antibodies. Scientific American, 1980; 243(4): 66-74.
- Haynes BF. Human T-cell antigen as defined by monolonal antibodies. Immunol Rev, 1981; 57:1.
- Russel SJ, Llewelyn MB, Hawkins RE. Principles of antibody therapy. Br Med J, 1992; 305: 1424-9.
- Goldenberg DM. Monoclonal antibodies in cancer detection and therapy. Am J Med, 1993; 94(3): 297-312
- Brem S, Tsamaclis AM, Gately S, Gross JL, Herblin WF. Immunolocalisation of basic fibroblast growth factor to the microvas-culature of human brain tumours. Cancer 1992; 70(11): 2673-80.
- Yu MH, Roffler SR, Hwang WS et al. Localisation and therapy of human cervical tumour xenografts with radio labelled monoclonal antibody 1H10. Gynecol Oncol. 1992; 47(1): 93-101.
- Sarvin AS, Dalbagni G, Cordon Cardo C et al. Nuclear over expression of P53 protein in transitional cell bladder carcinoma: a marker for disease progression. J Natl Cancer Inst, 1993; 85(1): 53-9.
- 14. Hanzal E, Gitsch G, Kohlberger P et al. Immunohistochemical detection of mutant P53suppressor gene product in patients with breast cancer: influence on metastasis free survival. Anticancer Res, 1992: 12(6B): 2325-9.
- Qian H, Feng J, Cin H et al. Clinical evaluation of radioimaging with 1311-COC183B2 monoclonal antibody against ovarian carcinoma by intraperitoneal injection. Gynecol Oncol, 1992; 42(2): 216-22.
- Pantel K, Izbicki JR, Angstwurm M et al. Immunocytological detection of bone marrow

- micrometastasis in operable non-small cell lung cancer. Cancer Res, 1993; 53(5): 1027-31.
- Sundin A. Radioimmunolocalisation and quantification
 of liver metastasis and subcutaneous tumours from a
 human colonic cancer xenografted in nude rat. Acta
 Radiol (suppl), 1993; 382: 1-29.
- Bazorin DF, Chapman PB, Wong GY et al. Treatment with high dose mouse monoclonal (anti-GD3) antibody R24 in patients with metastatic melanoma. Melanoma Res. 1992; 2(5-6): 355-62.
- Goodman GE, Hillstorm I, Yelton DE et al. Phase 1 trial of chimeric (human-mouse) monoclonal antibody L6 in patients with non-small cell lung, colon and breast cancer. Cancer Immunol Immunother, 1993; 36(4): 267-73.
- Bruynck A, Seemann G, Bosslet K. Characterization of a humanised bispecific monoclonal antibody for cancer therapy. Br J Cancer, 1993; 67(3): 436-40.
- 21. Papanastassiou V, Pizer BL. Coakham HB et al. Treatment of recurrent and cystic malignant gliomas by a single intracavity injection of 1311-monoclonal antibody: feasibility, pharmacokinetics and disimetry. Br J Cancer, 1993; 67(1): 144-51.
- 22. Greiner JW, Ullmann CD, Nieroda C et al. Improved radioimmunotherapeutic efficacy of an anticarcinoma monoclonal antibody (1311-CC49) when given in combination with gamma-interferon. Cancer Res, 1993; 53(3): 600-8.
- 23. Kitamura K, Takanashi T, Kotani T et al. Local administration of monoclonal antibody drug conjugate

- : A new strategy to reduce the local recurrence of colorectal cancer. Cancer Res, 1992; 52(22): 6323-8.
- Wawrzynczak EJ and Derbiyshire EJ. Immunotoxin: the power and the glory. Immunol Today, 1992; 13(10): 381-3.
- Kreitman RJ, Chandhary VK. Kozak RW et al. Recombinant toxins containing the variable domains of the anti-Tac monoclonal antibody to the interleukin-2 receptor kill malignant cells from patients with chronic lymphocytic leukemia. Blood, 1992; 80(9): 2344-52.
- Mickisch GH, Pai LH, Siegsmund M et al. Pseudomonas exotoxin conjugated to monoclonal antibody MRK 16 specifically kills multidrug resistant cells in cultured renal carcinomas and in MDR transgenic mice. J Urol, 1993: 149(1): 174-8.
- 27. Brinkmann U, Gallo M, Brinkmann E, Kunwar S, Pastan I. A recombinant immunotoxin that is active on prostatic cancer cells and that is composed of the Fv region of monoclonal antibody PR1 and a truncated form of Pseudomonas exotoxin. Proct Natl Acad Sci USA, 1993; 90(2): 547-51.
- Resonblum MG, Zuekerman JE, Marks JW, Rotbein J, Allen WR. A gelonin-containing immunotoxin directed against human breast carcinoma. Mol Biother, 1992; 4(3): 122-9.
- Buckman R, De Angelis C, Shaw P et al. Intraperitoneal therapy of malignant ascites associated with carcinoma of ovary and breast using radioiodinated monoclonal antibody 2G3. Gynecol Oncol, 1992; 47(1): 102-9.

Successful Pregnancy Outcome in a Patient of Ectopia Vesicae

SHAMSUN NAHAR BEGUM FCPS (GYNAE)

Summary:

Mrs. R.B., 18 years, primigravid patient with history of previous operation for ectopia vesicae was admitted with prolong obstructed labour. Vaginal delivery of an asphyxiated female baby was done after giving a deliberate incision over the perineum which was repaired in layers with an uneventful recovery.

(J Bangladesh Coll Phys Surg 1993;11:61-63)

Introduction:

Exstropy of the bladder is an embryological catastrophy in which there is an absence of the infraumbilical part of the anterior abdominal and anterior vesical walls. The posterior vesical wall is exposed with continuous dribling of urine from the ureteric orifices. There is an accompanying epispadiac deformity of the urethra and seperation of the pubis (split pelvis). This abnormality occurs in about one in 50,000 birth with male predominance, the ratio being 4:11.

Pregnancy in a patient with ectopia vesicae is very rare because of social inconvenience associated with true incontinence resulting frequently in divorce or seperation and indirectly leading to infertility. This case is presented here for its unusual occurrence.

Case History:

Mrs. R.B., 18 years, fair looking primigravid patient from poor socioeconomic background with history of previous operation for ectopia vesicae in Chandraghona missionary hospital at the age of 11 years, married for one year, was admitted on 9-9-91 in Sadar Hospital, Habigonj with obstructed labour prolonging for 48 hours. She had 38 weeks pregnancy and did not have any antenatal check up.

On examination, she was mildly anaemic, normotensive with slight oedema and dehydration. On abdominal examination there was scar of previous operation over the anterior abdominal wall with deformed umbilicus. Uterus was 36 weeks size, contracted and slightly deviated towards the right side. Foetal pole could not be detected properly for contraction of uterus. Foetal heart sound was feeble and irregular. Vaginal examination showed wide and bulged perineum with small introital orifice through which foetal scalp hair was seen. Both labia were small and widely seperated. There, was continuous dribling of urine through a small opening just above the introitus.

After resuscitation of the patient with fluid, sedation and antibiotic, vaginal delivery of an asphyxiated female baby weighing 2.5kg was done after giving a deliberate incision over the perineum which was repaired later in layers. Postnatal period was uneventful, only there was slight cystocoele on the seventh day of puerperium. Patient was discharged with advice for follow up, but she did not turn up for further followup.

Shamsun Nahar Begum, Consultant Sadar Hospital, Sylhet.

Corresepondence to:

Shamsun Nahar Begum Sadar Hospital, Sylhet.

Received: February 3, 1992. Accepted: March 14, 1993



Fig-1: Appearance of perineum and lower abdomen before delivery.



Fig-2: Generous episiotomy over the perineum.

Discussion:

Ectopia vesicae is a very rare embryological abnormality associated with social beconvenience occasioned by total incontinence.

Frequent upper urinary tract infection often

leads to death ¹. Reconstruction of bladder and urethra may be successful but may need diversion of urine by ureteric transplantation into the large gut or in an isolated loop of the ileum with an external orifice.

Myerscough² recorded two such cases, with ureteric transplant into the colon in childhood, who became pregnant and delivered electively by L.U.C.S. but both developed uterovaginal prolapse in postpartum period.

This patient had reconstruction operation for ectopia vesicae at the age of 11 years, got married, became pregnant and admitted with obstructed labour. As ectopia vesicae is associated with widely seperated pubic bones (postpartum X-ray of pelvis showed split pelvis), engagement of head was easy but obstruction was due to thick broad perineum with small introitus and probably from previous operative scar. So a generous episiotomy over the perineum was adequate to deliver the baby. There was slight cystocoele in post partum period which may be due to weakness in the ligament of pelvic floor.



Fig-3: Post partum X-ray pelvis.

This patient had reconstruction of the bladder and anterior abdominal wall and as there was no sphincter in the artificial urethra, there was continuous dribling of urine. Ideally this patient should be delivered by L.U.C.S. but due to certain unavoidable circumstances, surgery was not possible. So vaginal delivery was justified in this case.

References:

- Rains A J H, Mann CV. Ectopia Vesicae. In: Bailey & Love's Short Practice of Surgery. 20th Edition. London, ELBS, 1988, pp.255.
- Mayerscough PR. Deformity of bony pelvis. In: Munokerr's Operative Obstetrics. 10th Edition, London, Bailliere Tindall, 1982, pp. 141.

College News

Continuing Medical Education

26-11-92 — Dr. Md. Abdul Matin Paediatric Surgeon

Dhaka, delivered lecture on

"Carcinogenesis in Anomalous arrangement of the

Pancreaticobiliary ducts".

01-04-93 — Dr. Paddy A Dewan

Paediatric Urologist

Adelaide Children's Hospital

Australia, delivered lecture on Obstructive Uropathy in

Children.

29-4-93 — Dr. Md. Humayun Kabir

Professor-cum-Director

National Institute of Ophthalmology, Dhaka, delivered lecture on Intraocular lens implantation in Bangladesh.

List of Councillors:

Following is the 20 member list of the Councillors:

For the session 1991-95		For the session 1993-97	
1.	Dr. Nazmun Nahar	9.	Dr. A.H.M. Ahsanullah
2.	Dr. Golam Rasul	10.	Dr. Md. Tahir
3.	Dr. A.H.M. Towhidul Anowar Chowdhury	11.	Dr. Md. Nurul Islam
4.	Dr. Mahmud Hasan	12.	Dr. Md. Abdul Mobin Khan
5.	Dr. Rashid-E-Mahbub	13.	Dr. Tofayel Ahmed
6.	Dr. A.K.M. Mahbubur Rahman		Dr. Md. Abdul Hadi
7.	Dr. Shahjahan Nurus Samad Chowdhury	15.	Dr. Md. Harunur Rashid
8.	Dr. Abdul Bayes Bhuiyan	16.	Dr. Md. Ruhul Amin

Councillors nominated by Government of People's Republic of Bangladesh (1993-97).

17. Dr. M. A. Majed

19. Dr. Anis Waiz

18. Dr. Kazi Masihur Rahman

20. Dr. Matiur Rahman

Executive Committee:

The Election Commission of the Bangladesh College of Physicians and Surgeons has declared the members of new Executive Committee for two years from March, 1993 as follows:

President — Dr. A.H.M. Ahsanullah Senior Vice-President — Dr. Md. Tahir Vice-President — Dr. Nazmun Nahar

Treasurer — Dr. Md Nurul Islam

Members — Dr. Md. Abdul Mobin Khan

Dr. Tofayel Ahmed

Various Committees and Faculties of the Bangladesh College of Physicians and Surgeons:

The Council of Bangladesh College of Physicians and Surgeons has formed the following Committees and Faculties of the College for two years with effect from March, 1993:-

	0		
EXAMINATION COMMITTEE		FINANCE COMMITTEE	
1. Dr. Md. Tahir	Chairman	1. Dr. Nazmun Nahar	Chairman
2. Dr. T. A. Chowdhury	Member	2. Dr. Md. Nurul Islam	Member
3. Dr. Nazmun Nahar		3. Dr. Md. Ruhul Amin	"
4. Dr. Mahmud Hasan	BON BOTOVIDE	4. Dr. Md. Muazzam Hussain	
5. Dr. A.K.M. Mahbubur Rahman	A III Eleganogo	5. Dr. Md. Fazlul Haque	
6. Dr. S. N. Samad Chowdhury	un yasaldanii	6. Dr. U. H. Shahera Khatun	
7. Dr. K. M. Nazrul Islam	dy A Lievann	LIBRARY COMMITTEE	
		1. Dr. Md. Nurul Amin	Chairman
REFERENCE COMMITTEE	e Children's H	2. Dr. Md. Abdul Hadi Faquir	Member
1. Dr. A. H. M. Ahsanullah	Chairman	3. Dr. Sadiqa Tahera Khanam	
2. Dr. M. A. Majed	Member	4. Dr. Kaniz Mowla	
3. Dr. Rashid-E-Mahbub	*	5. Dr. Md. Margub Hossain	90.4 00
4. Dr. Tofayel Ahmed	EN HILLANDET	6. Dr. Rowshan Ara Begum	
5. Dr. Abdul Bayes Bhuiyan	SOBSING WINDS SO	7. Dr. Syed Atiqul Haq	
6. Dr. S. A. M. Golam Kibria	Lio.amirment i	8. Dr. Parveen Shahida Akhtar	
STUDENTS ADVISORY COMMITT	EE	JOURNAL COMMITTEE	G1 .
1. Dr. Golam Rasul	Chairman	Major General Anis Waiz	Chairman
2. Dr. Kazi Masihur Rahman	Member	2. Dr. Shafiqul Hoque	Editor
3. Dr. Md. Jalaluddin	"	3. Dr. Md. Abdul Mobin Khan 4. Dr. Md. Harunur Rashid	Member
4. Dr. Md. Salek Talukder	For Myr sees	5. Dr. Nazimuddin Ahmed	m() mon
5. Dr. Md. Taiabur Rahman		6. Dr. Md. Nazrul Islam	
6. Dr. Quazi Deen Mohammad	PA JOL A	7. Dr. A. K. M. Anisul Haque	M. TO. I
o. Dr. guazi Deen Monaninau		8. Dr. Md. Moazzam Hossain	2 Dr. G
CONTINUING MEDICAL E	DUCATION	9. Dr. Md. Afzal Hossain	AMI . E
COMMITTEE		TENDER COMMITTEE	
1. Dr. Md. Matiur Rahman	Chairman	1. Dr. A. K. M. Anowarul Azim	Chairman
2. Dr. Abdus Shakur	Member	2. Dr. Md. Nurul Islam	Member
3. Dr. Naseem Akhter Chowdhury	7 M = 0 * 01 ×	3. Dr. A. Quashem	Z. "Dr. SI
4. Dr. Hasina Banoo	M -0 - 81	4. Dr. Md. Israfil	A 10" 18
5. Dr. Syed Kamaluddin Ahmed		5. Dr. T. I. M. Abdullah-Al-Faruq	
6. Dr. Sameena Chowdhury	to stickenst at	6. Dr. Md. Abdullah	Councillors
6. Dr. Sameena Chowdhury		7. Dr. Rafiqul Alam	
		FELLOWS WELFARE COMMITTEE	
MUSEUM COMMITTEE		1. Dr. Rashid-E-Mahbub	Chairman
1. Dr. M. A. Awal	Chairman	2. Dr. A.K.M. Mahbubur Rahman	Member
2. Dr. F. A. Azim	Member	3. Dr. Md. Ruhul Amin	evilona.
3. Dr. Kohinoor Begum	Variable " talles	4. Dr. Md. Abdul Mannan Miah	313-3111 -
4. Dr. Ava Hossain	mon sanay o	 Dr. Shafquat Hussain Khundke Dr. Emran Bin Yunus 	the member
5. Dr. A. K. M. Khorshed Alam	A M.H.A TO	7. Dr. Kanak Kanti Barua	. Pres
	Dr. M.d. Tahn	8. Dr. F. Mohammad Siddiqui	Sen
6. Dr. Md. Shahid Hossain	Dr. Masmun	9. Dr. A. P. M. Sohrabuzzaman	" Vice
7. Dr. Abdul Quader Khan	Dr. Md Nuru	10.Dr. Md. Saiful Islam	TITE
8. Dr. A. N. M. Zia-ur-Rahman	Dr. Md. Abda	11.Dr. Syed Mahbubul Alam	TOPA "

FACULTY OF MEDICINE		FACULTY OF OBSTETRICS AND	
1. Dr. Nurul Islam	Chairman	GYNAECOLOGY	
2. Dr. S. G. M. Chowdhury	Member	1. Dr. T. A. Chowdhury	Chairman
3. Dr. Md. Nurun Nabi	Silvery " unfaith	2. Dr. Abdul Bayes Bhuiyan	Member
4. Dr. A. K. M. N. Chowdhury	te Man • world	3. Dr. A. K. M. Anowarul Azim	
5. Dr. M. R. Khan	Symet Med. Coll	4. Dr. Anowara Begum	* 18
6. Dr. Md. Abdul Mobin Khan	See to the second state	5. Dr. Latifa Shamsuddin	22 " 25
7. Dr. K. M. H. S. Sirajul Haque	· ·	6. Dr. Jahan Ara Begum	23 " Dr.
8. Dr. Probhakar Purkayastha	fests on fontilities	7. Dr. Sultana Jahan	25 " Dr.
FACULTY OF SURGERY		8. Dr. Sultana Razia Begum	30 00
1. Dr. S. A. Ashraf	Chairman	FACULTY OF BASIC MEDICAL SO	CIENCES
2. Dr. S. A. Sobhan	Member	1. Dr. K. M. Nazrul Islam	Chairman
3. Dr. K.M. Siraj Jinnat		2. Dr. K. A. Khalegue	Member
4. Dr. M. N. Huda	withet Med. Coll	3. Dr. M. A. Hai	. d . 88
5. Dr. Md. Humayun Kabir	ir Salımıdlah M	4. Dr. M. H. Mullick	70 . DE
6. Dr. A. N. M. Atai Rabbi	her e-Bangla M	5. Dr. Tahmina Hossain	1,70 a 61
7. Dr. M. A. Muhaiemen	dymen . many	6. Dr. Md. Nazrul Islam	
8. Dr. Shamsuddin Ahmed	Mod how sonly	7. Dr. Syed Mokarram Ali	
9. Dr. Selim Md. Jahangir	tymen, agn are	8. Dr. A. K. M. Nurul Anowar	13 " E

The President and the Secretary of the College shall be the ex-Officio Members of all Committees.

Examination News:

Results of FCPS Part I, FCPS Part II and MCPS Examinations held in January, 1993 are given below:

409 candidates appeared in FCPS Part I Examination held in January, 1993 of which 42 candidates came out successful. Subjectwise results are as follows:

Subject Name of the land	Number appeared in theory examination	Number qualified for viva-voce	Number Passed	43
Medicine	75	18 MAJERIE	6	
Surgery	107	44		
Paediatrics	48	13	6	
Obst. & Gynae	64	13 5	2	
Ophthalmology	37	medat unid 4 adixaW mus	0	
ENT Diseases	14	7	M 10 1	
Psychiatry	6	erum Kamu Gl. Lein	0 Det	
Anaesthesiology	26	mrusil L7 refridacisti ib	Mag 3	
Radiology		5 Me vad avia		
Radiotherapy	4	3 IlA variana Ali		
Physical Medicine	6	5 mala reguerte	1	
Haematology	8	4	2	
Biochemistry	1	0	0	
Microbiology	1	0	0	
Histopathology	1	d. Resignal BanO	0	
Total = Deliverage	409	116	42	111

95 candidates appeared in FCPS Part II Examination in different subjects. List of candidates who satisfied the board of examiners is as follows:

Roll No.	Name	Graduated from	Speciality
3	Dr. Mohammad Ashraful Islam	Rajshahi Medical College	Medicine
19	Dr. Faruque Ahmed	Chittagong Med. College	Medicine
20	Dr. Goutam Kumar Roy	Sylhet Med. College	Medicine
21	Dr. Md. Azizul Haque	Mymensingh Med. College	Medicine
22	Dr. Uzzwal Kanti Das	Chittagong Med. College	Medicine
23	Dr. A. H. M. Feroz	Dhaka Medical College	Medicine
25	Dr. S. M. Motahar Hossain	Chittagong Med. College	Medicine
30	Dr. Md. Muzibur Rahman Bhuiyan	Dhaka Medical College	Medicine
34	Dr. Radheshyam Saha	Chittagong Med. College	Medicine
48	Dr. A. H. M. Towhidul Alam	Sher-e-Bangla Med Coll.	Surgery
53	Dr. Md. Sohail Moosa	Dhaka Med. College	Surgery
64	Dr. Selina Khanum	Sir Salimullah Med. College	Paediatrics
68	Dr. Md. Habibur Rahman	Sylhet Med. College	Paediatrics
70	Dr. Begum Sharifun Naher	Sir Salimullah Med .College	Paediatrics
75	Dr. Krishna Banerjee	Sher-e-Bangla Med. College	Obst. & Gynae
79	Dr. Shamima Siddiqua	Mymensingh Med. College	Obst. & Gynae
80	Dr. Md. Zafar Khaled	Sylhet Med. College	Ophthalmology
32	Dr. Abu Ahmed Mohi Uddin	Mymensingh Med. College	Ophthalmology
33	Dr. Md. Nizamuddin Faruque	Sylhet Med. College	Ophthalmology
90	Dr. Mohammad Hamidur Rahman	Rangpur Med. College	Anaesthesiology
91	Dr. Md. Shah Alam	Rajshahi Med. College	Anaesthesiology
92	Dr. Md. Faruq Alam	Chittagong Med. College	Psychiatry
94	Dr. Md. Moyeenuzzaman	Dhaka Med. College	Physical Medicine

95 candidates appeared in MCPS Examinations in different subjects. List of candidates who satisfied the board of examiners is as follows:

Roll No.	Name Name	Speciality
32	Dr. Ruhidas Debnath	Paediatrics
36	Dr. Sarker Md. Rafiqul Islam	Paediatrics
42	Dr. Nurun Nahar Chowdhury	Obst. & Gynae
43	Dr. Selina Akhtar Banu	
48	Dr. Lily Afroza	Obst. & Gynae
59	Dr. Kismat Ara Sheikh	Obst. & Gynae
61	Dr. Mushfique Ahmed Chowdhury	Obst. & Gynae
66	Dr. Tahera Begum	Obst. & Gynae
67	Dr. Nilufa Musharrat Afza Begum	Obst. & Gynae
69	Dr Begum Waziha Akhtar Jahan	Obst. & Gynae
70	Dr. Sayeeda Akhtar	Obst. & Gynae
73	Dr. Niloufar Shameem Afza	Obst. & Gynae
74	Dr. Tarun Kanti Ghosh	Obst. & Gynae
78	Dr. Md. Mahabubu-Al-Karim	Ophthalmology
86	Dr. Vidya Dev Sharma	Psychiatry
87	Dr. Md. Azahar Ali	Radiology
88	Dr. Jahangir Alam	Radiology
92	Dr. Mohammad Zobair	Clinical Pathology
93	Dr. A. K. Md. Musfata Abedin	Clinical Pathology
94	Dr. Md. Saiful Islam	Clinical Pathology
102	Dr. Md. Afzal Hossain Bachchu	Dental Surgery
109	Dr. Md. Rafiqul Bari	Forensic Medicine
110	Dr. Md. Akhtaruzzaman	Forensic Medicine
111	Dr. Md. Monsur Rahman	Forensic Medicine

CONGRESS '93

An International Scientific Conference "CONGRESS" 93 will be held on 10-14 December 1993 in Karachi, Pakistan by the Joint Collaboration of the Bangladesh College of Physicians and Surgeons, College of Physicians and Surgeons Pakistan and International College of Surgeons (Pakistan Chapter).