The

Indian Journal of

Surgery

1962

January - 24-1

The Indian Journal of Surgery

Vol. XXIV JANUARY 1962 No. 1

THE INDIAN JOURNAL OF SURGERY

JANUARY 1962

(MONTHLY)

Annual Subscription Rs. 24

The Indian Journal of Surgery

Vol. XXIV | JANUARY 1962 | No. 1

CONTENTS

NOTICE TO CONTRIBUTORS

The Indian Journal of Surgery publishes original articles in all branches of surgery and allied subjects. Papers for publication and all editorial communications should be addressed to the Editorial Secretary, Indian Journal of Surgery, 462, Poonamallee High Road, Madras 10. Papers sent to the journal are sent to a panel of referees and are accepted for publication on their recommendation. The accepted papers are published on the understanding that they have not been, and will not be published in whole or in part in any other journal.

Orthopaedic Section : Articles intended for publication in the Orthopaedic Section of the Journal may kindly be sent to the Sectional Editor, Dr. N. S. Narasimhan, F.R.C.S., 221, Thambu Chetty Street, Madras 1.

Thoracic Surgery Section : Articles pertaining to Thoracic Surgery may be sent to the Sectional Editor, Dr. P. K. Sen, M.S., F.A.C.S., K. E. M. Hospital, Bombay-12.

Plastic Surgery Section : Articles intended for publication in the Plastic Surgery Section of the Journal may kindly be sent to Dr. R. N. Sharma, M.S., F.R.C.S., Reader in Surgery & Specialist in Plastic Surgery, K. G. Medical College, Lucknow.

Style : Manuscripts must be typewritten on one side on quarto or foolscap sheets, doublespaced and with a margin of not less than two inches. They should bear the name and full address of the author to whom the proofs are to be sent. The paper should conclude with a brief summary stating the conclusions described in the paper.

Tables and Illustrations : Tables should be numbered in Roman numerals and set out on sheets separate from the text. The illustrations should be kept separate from the text and should be numbered consecutively in Arabic numerals. Each figure should be referred to in the text and the descriptive legends should be typewritten on a separate sheet of paper. Nothing should be written or typed on the back of the illustrations. Photographs and photo-micrographs should be printed on glossy paper and generally should not be larger than 6·5 × 6·5 cms. If transmitted through the post they should not be folded and should be protected by cardboard paper.

For every article 12 blocks shall be printed free of cost. Cost of preparing extra blocks will have to be borne by the author.

Illustrations will not be accepted unless they reach a certain standard of excellence technically present an attractive appearance and add significantly to the value of the presentation.

References : These should be arranged alphabetically by authors. For citation of a book, give in order author's name, initials, complete title, place of publication, name of publishers, year and page. For an article author's name, initials, full title of paper exactly as it appears, name of journal (abbreviated as in the Quarterly Cumulative Index Medicus), volume number (in heavy type Arabic numerals), inclusive page numbers and year. The following examples illustrate the desired punctuation and arrangement.

Christopher, F.: Minor Surgery, 5th Edition, Philadelphia and London: W. B. Saunders Company, 1945, p. 348.

Fowler, R. H.: Cysts of the Spleen, Ann. Surg., **57**: 638, 1930.

Citations in the text should give the author's name followed by the number listed in the reference.

Proofs : Contributors will receive one proof in galley by air mail, if necessary, which should be immediately corrected and returned by air mail to the Editor. Only minor alterations will be accepted in the original manuscript at the time of the correction of proofs.

Reprints : 25 reprints of an article will be sent to the author or divided among the authors. Additional reprints can be supplied at cost price if application is made early.

All communications regarding membership of the Association, subscription to the Journal, and advertisements may be addressed to the Hony. Secretary, Association of Surgeons of India 462, Poonamallee High Road, Madras 10.

THE

INDIAN JOURNAL OF SURGERY

Vol. XXIV JANUARY 1962 No. 1

HYPOTHERMIA BY DIRECT COOLING OF BLOOD STREAM

(Report of 55 animal experiments)

BY

G. B. PARULKAR, A. J. DHRUVA, P. M. JAVERI and P. K. SEN, Bombay.

The interest of our group in the subject of artificial hypothermia has been mostly concentrated in its application to open intracardiac surgery. Many hypothermic techniques have been described, but the cooling method of choice would seem to be one which affords the central nervous system adequate protection from anoxia during inflow occlusion without causing increased myocardial irritability and gross physiological alterations produced by depression of general body tissue temperature.

Artificial hypothermia was first used for obtaining dry field during intracardiac surgery by Bigelow[4] (1950). He induced hypothermia by application of ice to the body surface. Surface cooling can also be produced by immersing the patient in cold water or by circulating cold water through cooling mattresses wrapped round the patient. Even at present the surface cooling is a more widely adopted method of artificial hypothermia. In the past few years the surface cooling technique has been successfully employed by us for creation of artificial hypothermia both in our experimental work and in clinical practice. Following observations were made during our experience of this method :

It is a technically simple procedure and is generally applicable. The rate of cooling is slow. Body temperature continues to fall after surface cooling is stopped and this fall is not always predictable. Shivering interferes with the rate of cooling and therefore complete curarisation is necessary. Cardiac arrhythmias and cerebral oedema as a direct consequence of hypothermia occurred in very few cases. A marked haemoconcentration and significant depression of plasma potassium was noted during surface cooling[5]. It was possible to cool experimental animals to very deep hypothermic levels with fairly high survival rate[7]. Surface cooling has to be started prior to thoracotomy and is definitely somewhat cumbersome procedure.

Internal and body cavity cooling originally described by Khalil[4] (1954) has not been very extensively used in clinical practice.

Extracorporeal cooling, namely, direct cooling of blood stream outside the body has been used by Delorme[2] (1952), Ross[6] (1954), Sir Russel Brock[2] (1956) and others with gratifying results.

To off set certain disadvantages of the surface technique of hypothermia we have looked forward to the development of the extracorporeal method. The present study was undertaken in order to assess the efficiency of this method of hypothermia and to obtain familiarity with the technical aspects of this method and further, to study the various physiological changes during such a procedure. In this paper a preliminary report of 55 animal experiments carried out in the Experimental Laboratories of the Department of Surgery of K. E. M. Hospital, Bombay, is presented.

Apparatus

A simple apparatus consisting of a polyvenyl coil immersed in ice and a constant perfusion pump (Sigma motor pump) was designed as a refrigerating system in which circulating blood could be cooled. The length of the polyvenyl tube was about 6 yards, the diameter was half a centimeter and the capacity was 100 c.c. The pump could be adjusted to delivery through the polyvenyl coil about 250 to 1250 c.c. of blood per minute. During the experimental study flow rates of 30-50 c.c. per kg. body weight were used. The coil was cleansed carefully with 1 per cent cetavlon and distilled water, autoclaved and filled with heparinised 5% glucose before starting the perfusion.

Material

Fifty-five mongrel dogs weighing between 14-18 kg. unselected as regards age and sex, were used for the experimental study during which venovenous cooling was performed.

Anaesthesia

Dogs were anaesthetised by injecting sodium thiopentone (25-30 mg. per kg. body weight) intravenously followed by largactil 1 mg. per kg. body weight. Respiration was maintained by connecting the intratracheal tube to a respiratory pump through which oxygen was supplied. Hyperventilation was maintained during the experiment. Before cannulation of the veins 0·5 mg. per kg. body weight of heparin was injected intravenously, only in the last 20 experiments.

Following temperatures were recorded :

(Fig. 1) Rectal temperature; oesophageal temperature at the level of the heart;

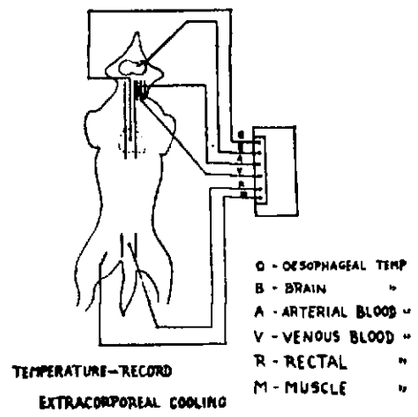

Fig. 1

Temperatures recorded during venovenous cooling.

muscle temperature; carotid artery blood temperature; jugular vein blood temperature; brain temperature was recorded by a needle electrode introduced into the frontal lobe, through a small hole in the skull drilled by using a Kirshner wire drill.

Method

(Fig. 2) In the first 5 experiments both the femoral veins were exposed and cannulated. The blood received from distal part of one femoral vein was pumped through the polyvenyl coil immersed in ice and salt mixture into the proximal part of the opposite femoral vein by means of a Sigma motor pump (Fig. 2-A). Maximum flow rate of blood was about 100-150 c.c. per minute and therefore the rate of cooling was slow (0·13 to 0·15° C per minute). The range of hypothermia established after one hour of perfusion was 31·32° C (Fig. 4). There were no cardiac irregularities or deaths in this series (Table 1).

In the next series of 5 experiments the procedure was modified. Through a longitudinal neck incision the jugular vein was

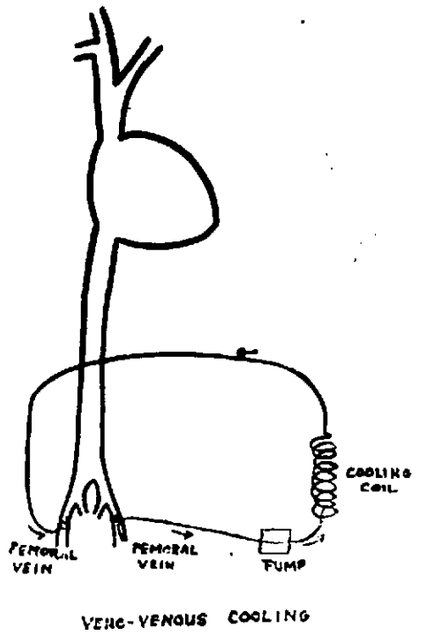

Fig. 2-a

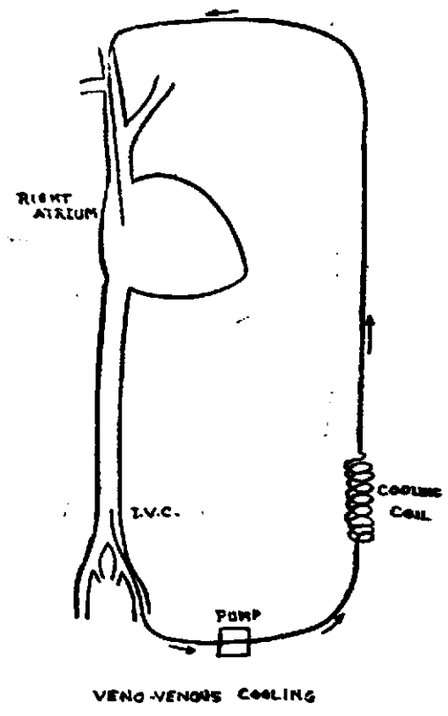

Fig. 2-b

Diagrams illustrating the different methods of venovenous cooling.

TABLE 1

Results of Venovenous cooling (Femoral vein to Femoral vein)

Dog No.	Weight Kg.	Rectal Temperature °C		Oesophageal Temperature °C		Period of rewarming	
		Initial	Final (after one hour)	Initial	Final (after one hour)	Hours	Minutes
1	14	38·5	31·8	38·0	31·0	1	10
2	16	39·0	31·5	38·5	31·0	1	10
3	15	38·0	32·0	37·0	31·5	1	5
4	13	38·0	31·0	37·8	31·0	1	5
5	18	39·0	32·0	38·0	32·0	1	10

TABLE 2

Results of Venovenous cooling (Inferior vena cava to Right atrium)

Dog No.	Weight Kg.	Initial Temperature °C			Cardiac Irregularity	Restitutive Measures	Revival	Temperature (°C) at which ventricular fibrillation occurred		
		R	O	B				R	O	B
6	14	38·2	37·5	37·2	Ventricular Fibrillation	Massage Defibrillation	No	32·0	25·0	31·5
7	15	38·0	37·0	36·5	do.	do.	No	31·5	22·5	32·0
8	14	38·0	37·0	36·5	do.	do.	No	32·5	22·0	32·0
9	16	39·0	38·2	36·8	do.	do.	No	32·0	23·0	31·5
10	13	38·5	38·0	37·0	do.	do.	No	31·8	24·0	31·0

TABLE 3

Results of Venovenous cooling (Inferior vena cava to Superior vena cava)

Dog No.	Weight Kg.	Initial Temperature °C			Cardiac Irregularity	Restitutive Measures	Revival	Temperature (°C) at which ventricular fibrillation occurred		
		R	O	B				R	O	B
11	14	38·0	37·5	37·0	Ventricular Fibrillation	Massage Defibrillation	No	30·0	25·0	29·05
12	16	38·5	38·2	37·8	do.	do.	No	30·2	25·0	30·0
13	17	38·0	38·0	37·0	do.	do.	No	31·5	22·0	30·8
14	15	38·0	37·8	37·0	do.	do.	No	30·0	23·0	30·2
15	14	38·5	38·0	37·5	do.	do.	No	30·2	24·0	30·0

exposed and cannulated. The polythylene catheter was pushed through the innominate vein and the superior vena cava upto the right atrium. The femoral vein was cannulated and the cannula was pushed into the inferior vena cava so as to facilitate increase in the rate of blood flow through the cooling coil. Cool blood was directly introduced into the heart (Fig. 2-B). Within 15 minutes the oesophageal temperature recorded a fall upto 22°-24° C from initial 37°-38° C. Rectal and brain temperatures however remained between 31°-32·5° C. (Fig. 5). There were marked changes in the electrocardiographic pattern soon after the cooling started due to considerable interference with myocardial action and ventricular fibrillation occurred in all the 5 animals at the rectal temperature between 31° and 32° C. Immediate cardiac massage and use of defibrillator failed to revive the heart in all the animals (Table 2).

Following this in a series of 5 experiments instead of introducing the cool blood directly in the heart it was introduced in the superior vena cava (Fig. 2-C). In all

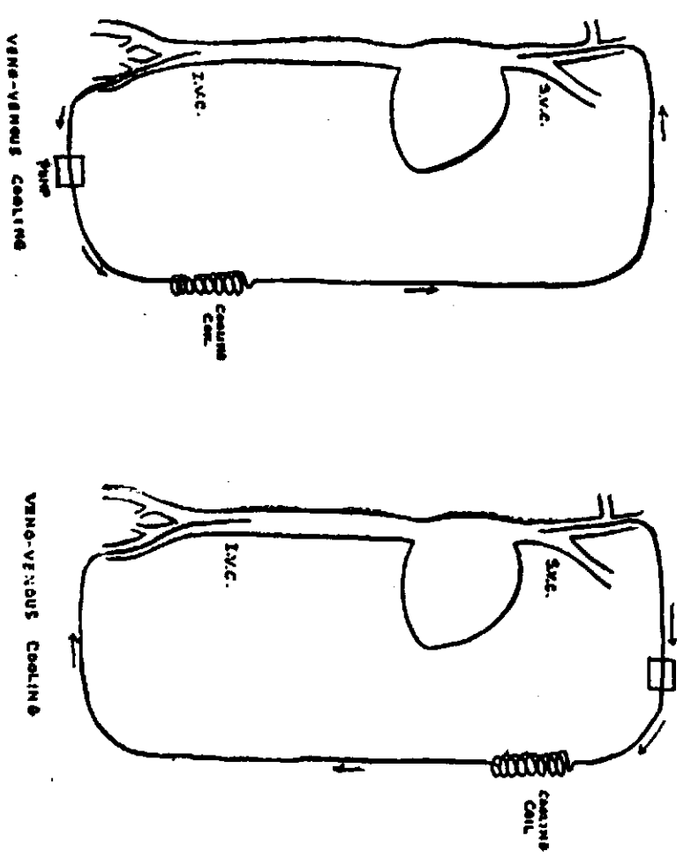

Fig. 2-c

Fig. 2-d

Diagrams illustrating the different methods of venovenous cooling.

these dogs ventricular fibrillation occurred by the time the rectal and brain temperatures were only 30°-31° C (Table 3). In only one case the normal cardiac rhythm could be established after a prolonged massage and use of the defibrillator. The dog later died of brain damage.

In order to lessen the myocardial irritability as a result of direct contact with very cool blood it was decided to introduce the cool blood into the inferior vena cava (Fig. 2-D) (which has longer length than the superior vena cava) after receiving it from superior vena cava. However, in the series of these 4 consecutive experiments ventricular fibrillation occurred in all by the time the rectal and brain temperatures were lowered to 28°-30° C (Table 4).

Fig. 2-c Fig. 2-d

Diagrams illustrating the different methods of venovenous cooling.

TABLE 4

Results of Venovenous cooling (Superior vena cava to Inferior vena cava)

Dog No.	Weight Kg.	Initial Temperature °C R	Initial Temperature °C O	Initial Temperature °C B	Cardiac Irregularity	Restitutive Measures	Revival	Temperature (°C) at which ventricular fibrillation occurred R	Temperature (°C) at which ventricular fibrillation occurred O	Temperature (°C) at which ventricular fibrillation occurred B
16	18	38·4	38·0	37·0	Ventricular Fibrillation	Massage Defibrillation	No	29·5	25·0	29·0
17	16	38·0	37·5	37·0	do.	do.	No	28·0	25·5	28·2
18	14	39·0	38·8	37·05	do.	do.	No	28·8	24·5	28·0
19	14	38·5	38·0	37·05	do.	do.	Yes	30·0	26·0	29·5

TABLE 5

Results of Venovenous cooling (Moderate hypothermia) and spontaneous rewarming (Superior vena cava to Femoral vein)

Dog No.	Weight Kg.	CLINICAL HYPOTHERMIA Initial Temp. °C R	Initial Temp. °C O	Initial Temp. °C B	Final Temp. °C R	Final Temp. °C O	Period of	Period during which clinical level of hypothermia was maintained	Cardiac Irregularity	Survival
20	14	38·0	37·5	37·0	29·0	28·0	Minutes 10	Hours Minutes 1 00	Nil	Yes
21	13	39·0	38·0	37·5	30·0	28·0	8	0 55	Nil	Yes
22	15	38·0	37·5	27·2	30·0	27·5	8	0 50	Nil	Yes
23	14	38·5	38·0	37·5	29·0	28·0	10	0 50	Nil	Yes
24	16	38·0	37·5	37·2	30·0	28·2	10	1 00	Nil	Yes
25	13	39·0	38·0	38·0	30·5	28·0	9	0 55	Nil	Yes
26	12	37·8	37·2	36·5	30·0	29·0	8	0 45	Nil	Yes
27	14	38·0	37·0	36·5	30·0	28·5	10	0 50	Nil	Yes
28	15	39·5	38·5	36·8	29·5	29·0	9	1 00	Nil	Yes
29	18	39·0	38·0	37·0	30·0	29·0	9	1 05	Nil	Yes

Thus in a series of 19 consecutive dog experiments were unable to establish a rapid and a safe procedure for veno venous cooling.

In the next 10 experiments the blood received from the *superior* vena cava was cooled and pumped into the femoral vein instead of inferior vena cava (Fig. 2E). There was a rapid and uniform fall of the oesophageal, rectal and brain temperatures at the average rate of about 0·75° to 1° per minute, and within approximately 10 minutes of cooling the rectal and brain temperatures were lowered to 28°—30° C. Cardiogram showed gradual slowing of the heart (Fig. 6). No cardiac irregularities occurred in this series. The dogs were allowed to rewarm spontaneously and the range of clinical hypothermia was maintained for 45 minutes to one hour in all these dogs. There was no death in this series (Table 5).

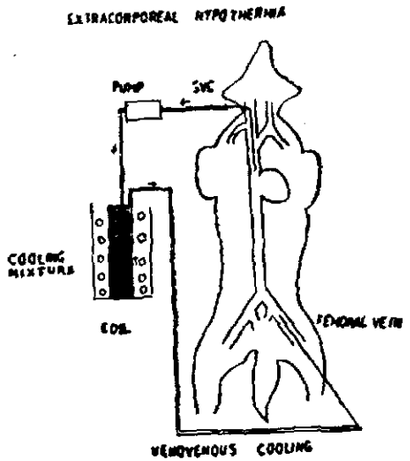

Fig. 3

Technique of venovenous cooling.

Following this in a series of 6 experiments a deeper range of hypothermia namely, 22°—26° C. was attained within a period of 15-20 minutes with the same technique. Cardiac arrest occurred in only one dog where the rectal (Fig. 7) temperature dropped to 22° C in 20 minutes. In spite of the resuscitative measures heart rhythm could not be revived. In rest of the dogs there were no cardiac irregularities during cooling. They were allowed to rewarm spontaneously and all of them survived (Table 6).

In the next 10 experiments after cooling the dogs by the same procedure upto 22°—26°C within 15-20 minutes the cooling was stopped. For the following 10 minutes the blood in the cooling coil was recirculated within it so as to prevent its clotting. Extracorporeal rewarming was started after these 10 minutes by immersing the polyvenyl coil in warm water maintained at 44°—45° C temperature. The dogs were rapidly rewarmed at the average rate of 0·5° C. per minute for about 20 minutes, during which time the temperature was elevated to 34°—35° C. Later the dogs were allowed to rewarm spontaneously (Fig. 8) In the very first experiment on starting the rapid rewarming there was ventricular fibrillation and therefore in the subsequent experiments rewarming was done by gradually increasing the blood flow rate from 10 c.c. per kg. body weight initially to about 25-30 c.c. kg. later and no similar

TABLE 6

Results of Venovenous cooling (Deep hypothermia) and spontaneous rewarming (Superior vena cava to Femoral vein)

Dog No.	Weight Kg.	CLINICAL HYPOTHERMIA Initial Temp. °C R	O	B	Final Temp. °C R	O	B	Period of Cooling Mins.	Period of Spontaneous Rewarming Hrs.	Mins.	Cardiac Irregularity	Survival
30	13	38·0	38·0	36·0	26·0	25·5	25·5	15	1	30	Nil	Yes
31	15	39·0	38·2	36·2	26·0	24·0	24·5	15	1	35	Nil	Yes
32	13½	39·0	38·0	36·8	26·5	23·0	23·0	15	1	25	Nil	Yes
33	13	38·0	37·5	36·5	22·0	—	21·0	20	—		Cardiac arrest	No
34	14	38·0	37·5	37·0	24·5	24·0	25·5	20	1	40	Nil	Yes
35	14	37·2	37·0	36·5	24·8	25·0	25·0	20	1	35	Nil	Yes

TABLE 7

Results of Venovenous cooling and venovenous rewarming (Superior vena cava to Femoral vein)

Dog No.	Weight in Kgs.	Initial Temperature °C R	O	B	Final Temperature °C R	O	B	Period of Cooling	Cooling stopped for	Period of Extra corporeal rewarming	Final Temperature after extracorporeal rewarming R	O	B	Cardiac Irregularity During cooling	During re-warming	Revival
								Mins.	Mins.	Mins.						
36	14	39·0	38·5	36·5	26·0	25·0	25·2	15	10	5	27·0	28·0	26·5	Nil	V.F.	No
37	15	38·0	38·0	36·0	25·0	25·2	24·2	15	10	25	34·5	35·0	34·0	Nil	Nil	Yes
38	13	38·5	28·0	36·8	26·0	25·0	25·0	15	10	22	35·0	35·5	34·5	Nil	Nil	Yes
39	14	38·2	38·0	36·2	24·0	23·0	24·0	20	10	28	34·0	36·0	34·2	Nil	Nil	Yes
40	16	39·0	37·5	37·0	24·5	24·0	24·5	20	10	20	34·5	35·0	34·0	Nil	Nil	Yes
41	14	38·0	37·8	36·5	25·0	24·5	24·0	15	10	20	34·0	35·5	35·0	Nil	Nil	Yes
42	13	38·5	38·0	36·5	22·0	20·5	23·5	20	—	—	23·0	28·5	23·0	Cardiac arrest	—	No
43	15	38·0	37·5	36·2	26·0	24·5	25·0	15	10	25	35·0	36·0	35·5	Nil	Nil	Yes
44	14	39·0	38·5	37·0	23·0	20·0	25·0	20	2	25	34·5	34·5	34·0	Cardiac arrest	—	Yes
45	17	38·8	38·0	36·2	25·5	23·0	24·0	15	2	20	35·0	34·8	34·0	Nil	Nil	Yes

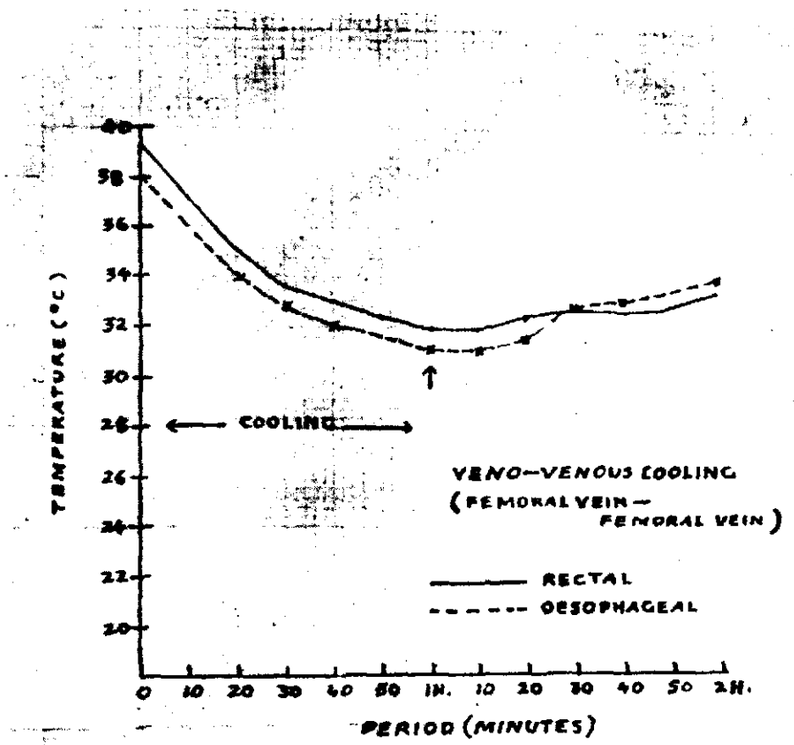

Fig. 4

Graph representing the temperatures during venovenous cooling (Femoral vein to Femoral vein).

disaster occurred. In this series during the extracorporeal cooling cardiac arrest occurred in 2 dogs (Nos. 42 and 44) at rectal temperatures of 22° and 23° C. respectively. In Dog No. 42 the normal cardiac rhythm (Table 7) could not be reinstituted in spite of the usual resuscitative measures. In Dog No. 44, cardiac rhythm was established soon after the extracorporeal rewarming was started, thus proving that extracorporeal rewarming helps to establish the cardiac rhythm.

In the following 3 experiments the blood received from superior vena cava after passing it through the cooling coil was pumped into the saphenous vein. However as the calibre of saphenous vein being very

small, the flow rates were restricted, resulting in slow rate of cooling as compared to the immediately preceding series and therefore both the saphenous veins were cannulated in the following 7 experiments in which fairly rapid cooling was possible. (Fig. 2F) Dogs were cooled up to 22°–25° C. Cardiac arrest or ventricular fibrillation occurred in 2 cases during this procedure. In 2 cases where the temperature was lowered to 22°—23° C. during the process of extracorporeal rewarming ventricular fibrillation occurred (Table 8). In one of the cases during the process of rewarming there was clotting of blood in the polyvenyl tube probably due to inadequate heparinisation.

The Pulse rate during Hypothermia.—There was a proportionate slowing of the heart

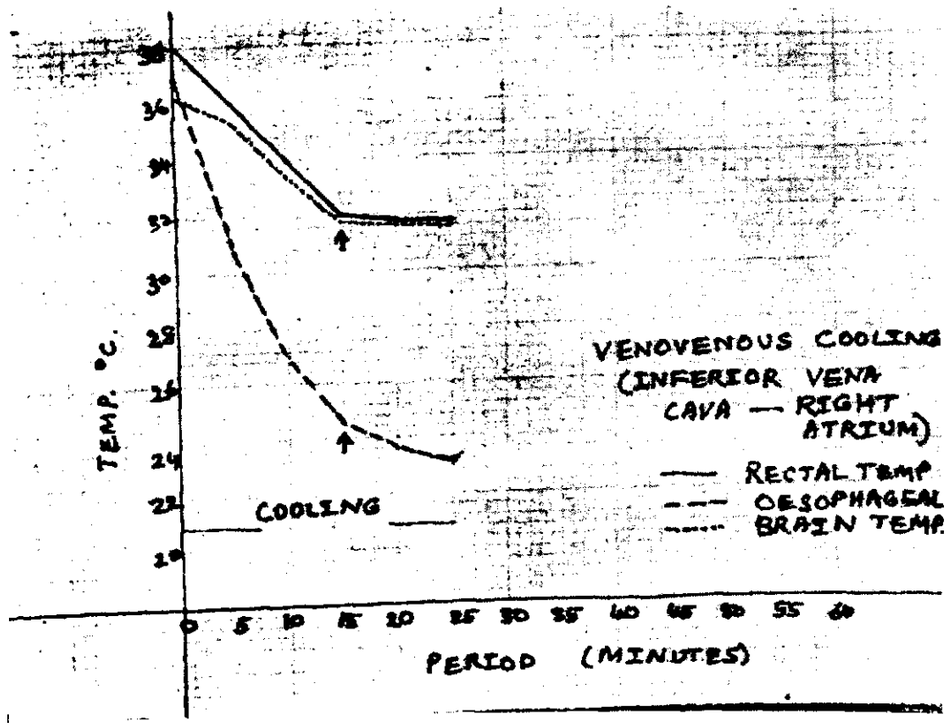

Fig. 5

Graph representing Rectal, Oesophageal and brain temperature during venovenous cooling (Superior Vena Cava to Right atrium).

The Mark represents the temperature at which ventricular Fibrillation occurred.

rate during the extracorporeal cooling which was lowered from initial 150 to 175 per minute at 39° C. to 10-30 per minute at 22°—25° C. As soon as the cooling was stopped there was a rise in the heart rate. During the extracorporeal rewarming there was a proportionate rise in the pulse rate as shown in the graph (Fig. 9).

Blood Pressure.—Arterial blood pressures were recorded throughout the experiment by connecting a metal cannula introduced in the femoral artery to the manometer. During the cooling the initial systolic blood pressure of 120-140 mm. Hg. was lowered rapidly to 80-100 mm. Hg. as soon as the perfusion was started but in a short time it was stabilised and later it showed a fairly steep fall as the cooling continued. The arterial blood pressure was about 25-30 mm. Hg. when the temperatures were lowered (Fig. 10) to 23° - 25° C. thus proving the enormous diminution in the cardiac output due to the venovenous cooling. The marked temporary hypotension during this procedure of venovenous cooling is a definite

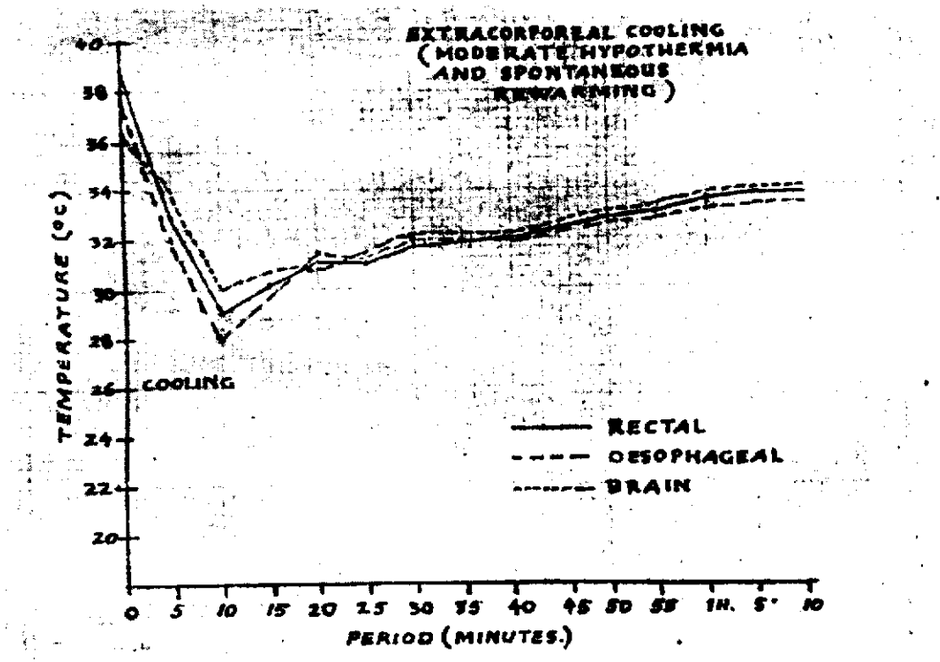

Fig. 6

Graph representing the temperature fall and rise during venovenous cooling (moderate hypothermia) and spontaneous rewarming (Superior vena cava to femoral vein).

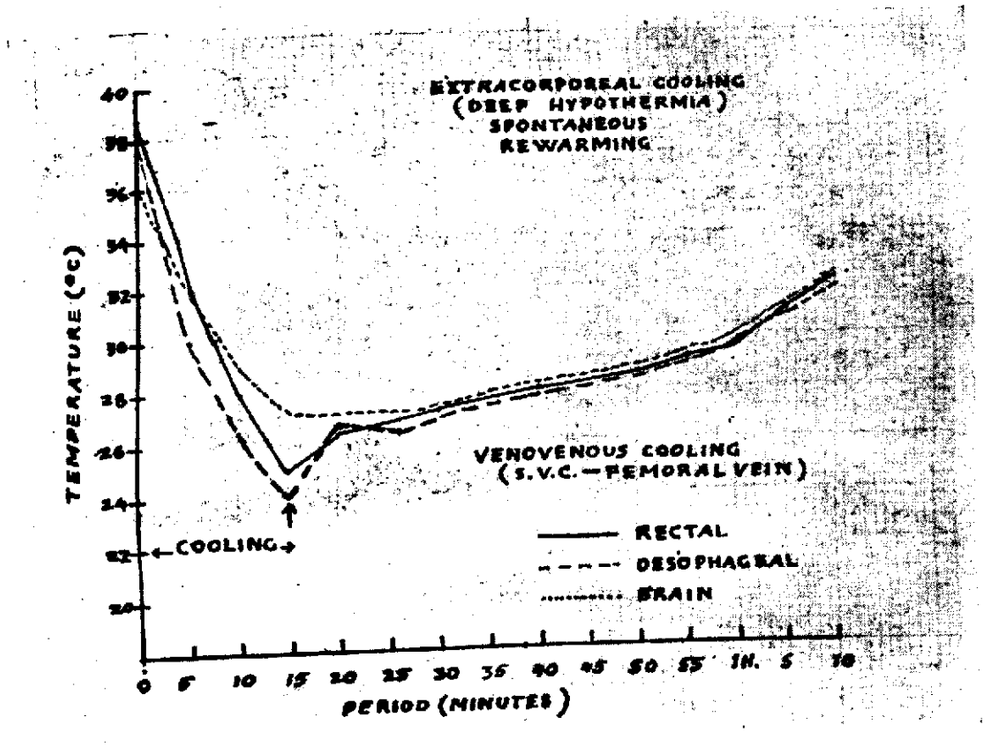

Fig. 7

Graph representing the temperature fall and rise during venovenous cooling (deep hypothermia) and spontaneous rewarming (Superior vena cava to femoral vein).

disadvantage of this procedure and has got to be counteracted. During the rewarming the blood pressure was rapidly restored.

E. C. G.—Following changes were noted in the cardiographic pattern during the extracorporeal hypothermia:

Bradycardia; slight prolongation of PR interval; slight prolongation of QRS complex: marked prolongation of ST segment: (Fig. 11) nodal rhythm was noticed in some cases; cardiac arrest occurred in 5 cases and ventricular fibrillation occurred in 16 cases. The incidence of ventricular fibrillation was very high (100%) in those cases where the cool blood was introduced close to the heart (Fig. 12). The incidence of ventricular fibrillation was high also in those experiments where attempt was made to lower the temperature below levels of 24°—25° C.

Pathological and Biochemical Studies.—The detailed pathological and biochemical studies during extracorporeal hypothermia will form a subject for our further communication. However, a few observations made so far will be presented here—viscocity of blood increased during this method of venovenous cooling and the initial P. C. V. of 40%—45% was increased to 50%—55% at 25° C.

Coagulation time.—The procedure of venovenous cooling was possible without heparinisation of the system. But in those experiments where venovenous rewarming was done after the venovenous cooling there was

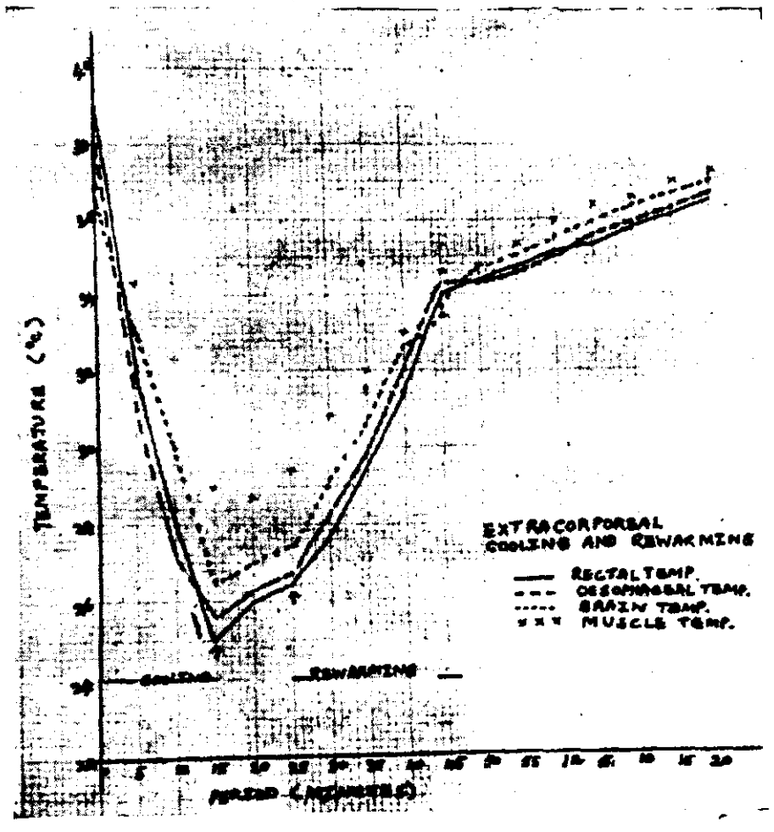

Fig. 8

Graph showing the oesophageal, rectal, brain and muscle temperature during venovenous cooling and venovenous rewarming (superior vena cava to femoral vein).

a small but definite danger of clotting occuring in the polyvenyl coil and therefore a small dose of heparin was exhibited intravenously in most of our later experiments and thus changes in coagulation time during the extracorporeal hypothermia could not be studied. The pH of blood was somewhat lowered to levels of 7·3 to 7·5 (from initial 7·7) at the height of hypothermia but at the end of the rewarming, however (Fig. 13) the pH was restored to normal limits. The hyperventilation maintained during this procedure was probably responsible for the minimal variation of pH during this study.

Hemolysis.—The plasma haemoglobin estimations revealed insignificant amount of hemolysis in most of our later experiments and therefore prove that the rapid direct blood stream cooling and rewarming within the range of temperature used during this

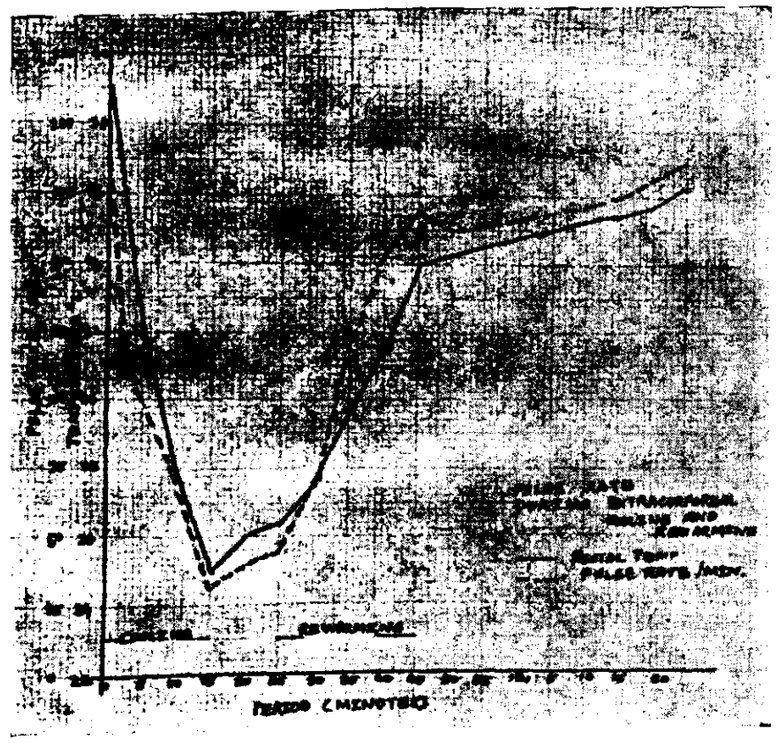

Fig. 9

Graph showing the pulse rate during the method of venovenous cooling and rewarming.

series was *quite safe regarding* the blood element destruction.

Serum Sodium and Potassium.—Serum electrolyte determinations on serial blood samples revealed no significant changes of serum sodium and potassium during venovenous cooling or rewarming.

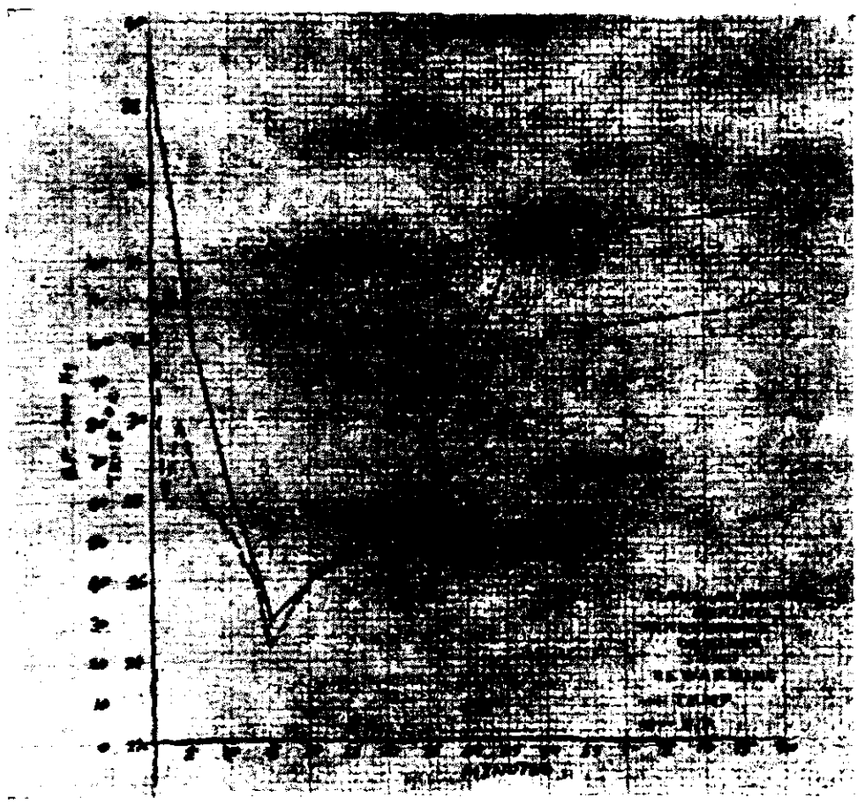

Fig. 10

Graph representing the changes in arterial blood pressure during venovenous cooling and venovenous rewarming.

Results

Direct blood heat exchange by venovenous cooling provides for a fairly uniform rapid cooling (at the average rate of 0·8°—1° C. per minute) of vital organs such as brain, heart and other internal organs up to the temperature range of 24°—25° C. The skeletal muscle temperature and the skin

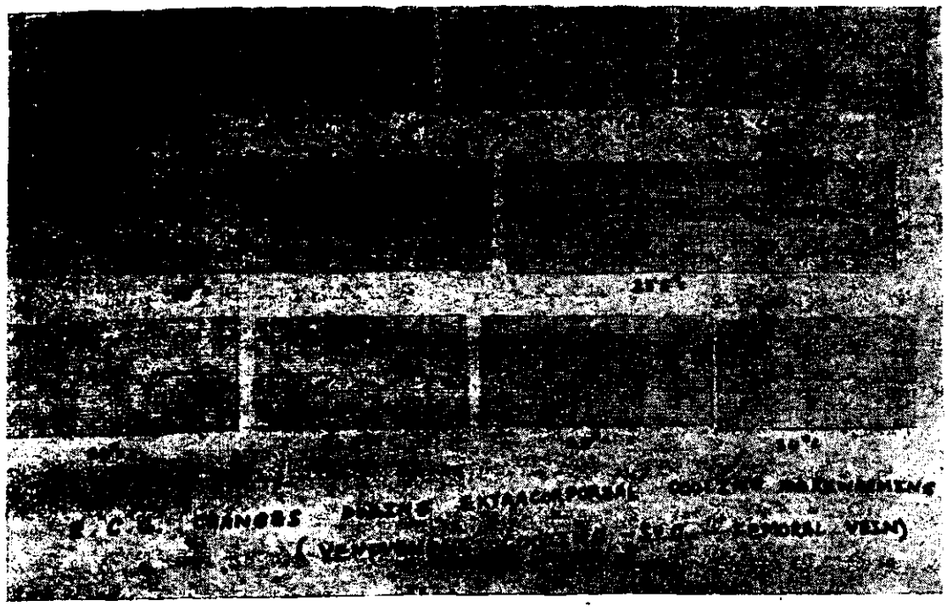

Fig. 11

Graph showing the typical E. C. G. record during venovenous cooling and rewarming (superior venacava to femoral vein).

temperature never attains the low range of internal organs and the responses to temperature (Fig. 8) changes in them are rather less marked as compared to vital organs which are richly supplied with blood. The oesophageal temperature fairly accurately depicts the heart temperature which was lowered most markedly during this procedure of venovenous cooling as the cooled blood first reaches the heart before being circulated to the rest of the organs. The brain temperature and the rectal temperature were lowered fairly uniformly as the cooling continued, but as the temperature was lowered below 24° C. the myocardial action was considerably weakened resulting in lowering of cardiac output and the rate of cooling of vital organs like brain was diminished considerably. In most of the experiments the rectal temperature stabilised between the oesophageal and muscle tissue temperature and has been used in this communication to express the general body temperature.

Rapid rewarming (at the average rate of 0·5° C. per minute) was also possible by this technique. Extracorporeal rewarming

TABLE 8

Results of Venovenous cooling and venovenous rewarming (Superior vena cava and saphenous vein)

Dog No.	Weight in Kgms.	Initial Temperature °C		Final Temperature °C		Period of Cooling	Cooling stopped for	Period of Extra-rewarm	Final Temp. °C Rectal	Cardiac Irregularities		Survival
		R	O	R	O					Cooling	Re-warming	
						Mins.	Mins.	Mins.				
45	30	39	38	38	38	30	10	5	26	Nil	V.F.	No (L.V. clotting)
47	30	39	38	38	38	30	10	25	34	Nil	V.F.	Yes
48	25	38	38	38	37	25	10	30	34	Nil	Nil	Yes
49	20	38	37·5	37	37	20	10	25	34·5	Nil	Nil	Yes
50	15	38	38	38	37	15	15	20	34	Nil	Nil	Yes
51	20	38	38	38	37	20	12	25	34·5	Nil	Nil	Yes
52	20	38·5	38	37	37	20	2	—	—	V.F.	—	No
53	22	39	38	38	37·5	22	16	10	26·5	Nil	V.F.	Brain damage
54	20	38	37·5	38	37	20	10	20	34	Nil	Nil	Yes
55	18	37·5	37	37	37	18	10	20	35	V.F.	Nil	Yes

TABLE 9

Summary of Results of different methods of venovenous cooling and rewarming

Technique	Number of Experiments	Range of Hypothermia Rectal °C	Rate of Cooling °C/min.	Shivering	After fall of Temperature	Extra corporeal re-warming	Cardiac Arrythmias	Revival	Neurological Disorders	Pyrogen Reaction	Renal Disorders	Shock	Death
1. Femoral Vein—Femoral Vein ...	5	31–32	0·13	Nil	Nil	No	Nil	—	Nil	Nil	Nil	Nil	Nil
2. Inferior Vena Cava—Right Atrium.	5	32–32·5	0·50	Nil	Nil	No	V. F. (5)	Nil	—	—	—	—	5
3. Inferior Vena Cava—Superior Vena Cava ...	5	30–31·5	0·50	Nil	Nil	No	V. F. (5)	One	One	—	—	—	5
4. Superior Vena Cava—Inferior Vena Cava ...	4	28–30	0·52	Nil	Nil	No	V. F. (4)	One	—	—	—	—	3
5. Superior Vena Cava—Femoral ...	10	29–30	0·80	Nil	Nil	No	Nil	—	Nil	Nil	Nil	Nil	Nil
6. Superior Vena Cava—Femoral ...	6	24·5–26	0·80	Nil	Nil	No	Cardiac arrest	Nil	Nil	Nil	Nil	Nil	1
7. Superior Vena Cava—Femoral ...	10	24·5–26	0·80	Nil	Nil	Yes (0·5 °C min.)	Cardiac arrest (2) V. F. (1) during warming.	One	Nil	Nil	Nil	Nil	2
8. Superior Vena Cava—Saphenous Vein ...	10	24–25	0·75	Nil	Nil	Yes (0·5 °C min.)	V. F. (4) Cooling (2) Rewarming (2)	One	One	Nil	Nil	Nil	2

was quite safe and was found to help early recovery of cardiac rhythm.

The incidence of ventricular fibrillation was extremely high in those experiments where the cooled blood was directly introduced close to the heart (Table 9). The incidence was also high in those experiments where the temperature was lowered below 24° C. inspite of introducing the cooled blood as far away from the heart as possible. Further studies are being carried out at present to find out the factors leading to high incidence of ventricular fibrillation in these experiments and to modify our method in different ways so as to prevent these cardiac arrhythmias during such a procedure.

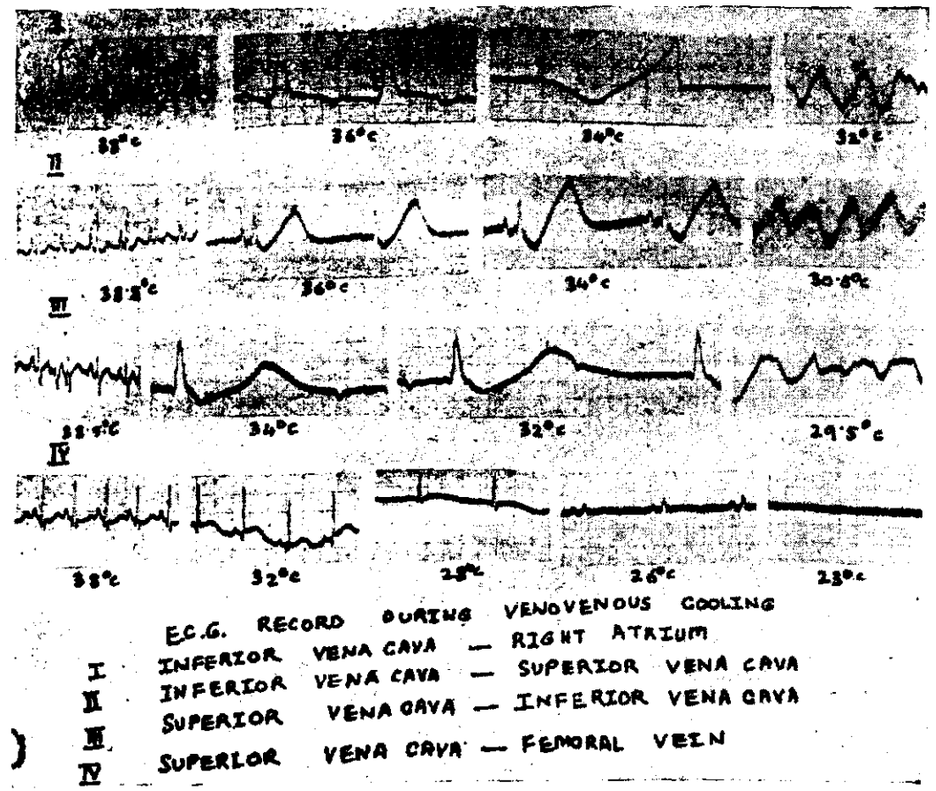

Fig. 12

Graph showing the E. C. G. during different techniques of venovenous cooling.

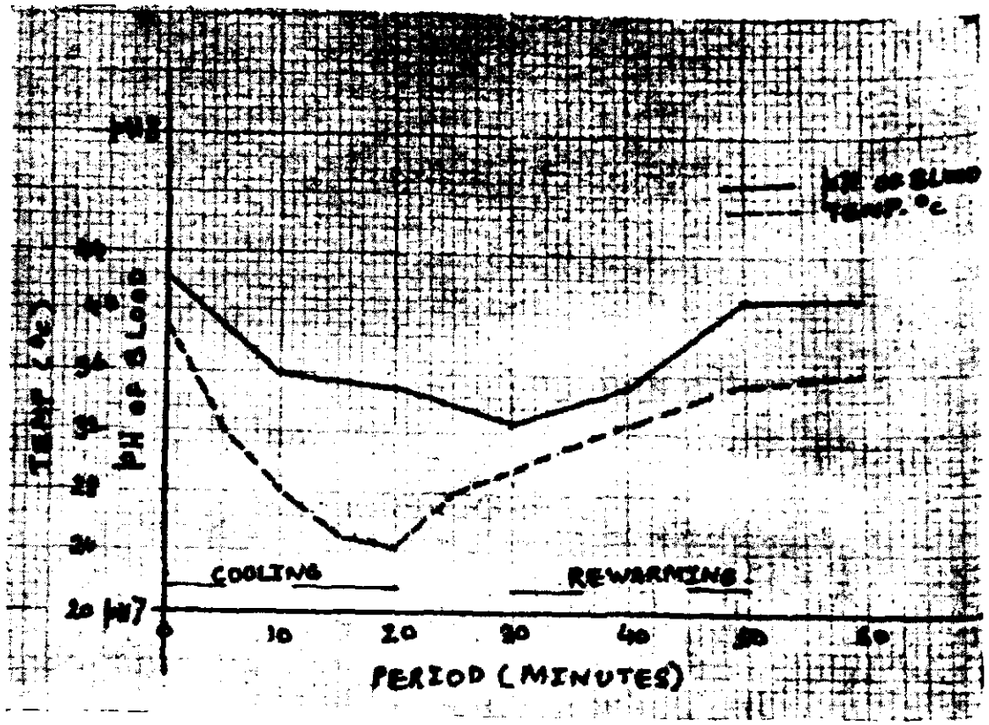

Fig. 13

Graph representing the changes in the pH of blood during venovenous cooling and rewarming.

There was no shivering seen in any of the animals during this technique of hypothermia. There was no significant after fall of temperature. There was no incidence of pyrogen reaction, cerebral oedema or renal shut down in any of the experimental animals.

Comment

This type of thermoregulation provides for a rapid and safe method of cooling of the body up to (Fig. 14) the moderate range of hypothermia followed by rewarming. Certain modifications, however, are necessary to obtain deeper ranges of hypothermia with safety by using such a method. In addition to minimising the total period of anaesthesia during the cooling and rewarming procedures this method supplies a cardiovascular surgeon with a rapid hypothermia without committing to it before thoracotomy.

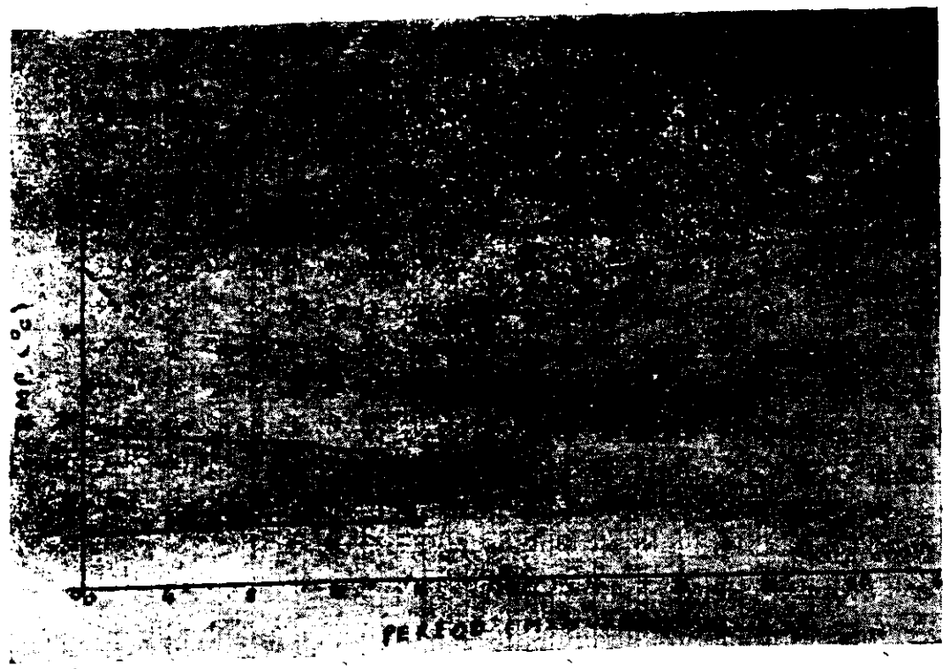

Fig. 14

Graph representing the temperatures of blood entering and leaving the coil during the method of venovenous cooling and rewarming.

Summary

(1) Techniques of venovenous cooling are described.
(2) Results of the experiments are documented.
(3) Physiological changes during this procedure are summarised.
(4) Advantages and disadvantages of the method are discussed.

REFERENCES

1. Bigelow, W. G., Callaghan, J. C. and Hopps, J. A.: General hypothermia for experimental intracardiac surgery, Annals of Surgery, 132: 531, 1950.
2. Brock, R. C.: Hypothermia and open cardiotomy. Proceedings of the Royal Society of Medicine, 49: 347, 1956.
3. Delorme, E. J.: Experimental cooling of blood stream, Lancet, 2: 914, 1952.
4. Khalil, H. H. and MacKeith, D. M.: A simple method of raising and lowering body temperature, Brit. Med. Jour. 2: 734, 1951.
5. Lewis, R. A., Satoskar, R. S., Gaitonde, B. B., Shah, C. B., Datey, K. K. and Sen, P. K.: Biochemical changes during hypothermia in dogs, Journal of Postgraduate Medicine, 2: 71, 1956.
6. Ross, D. N.: Venous cooling—A new method of cooling the blood stream. Lancet, 1: 1108, 1954.
7. Sen, P. K., Fletcher, W., Shah, C. B. and Datey, K. K.: Survival from induced hypothermic cardiac arrest (Studies in extreme hypothermia in dogs). Indian Journal of Medical Sciences, 11: 1007, 1957.

—Received for publication on 15-11-1960.

CARDIAC ARREST

BY

P. N. VYAS, T. THOMAS, R. H. BETTS and G. LEWIS, Vellore (S. India).

Cardiac arrest is one of the most dreadful surgical catastrophies. It usually occurs with dramatic suddenness.

There is no surgical emergency which requires more prompt action than cardiac arrest. It is an emergency which seldom occurs without, at least in retrospect, an ascertainable cause. "An ounce of prevention is worth a pound of cure" finds no better application than here.

The increasing scope of surgery, extending as it now does to include the tiny infant and the aged has posed new hazards and difficulties for the anaesthetist and surgeon and made the opportunities for the development of cardiac arrest greater than ever. Furthermore, the magnitude of excisional procedures has increased to the point that one often wonders whether we are not at times exceeding the natural boundary of extirpation. The frontier in cardiac surgery continues to pose problems in physiology which occasionally exceed our capacity to cope with them, leading to instances of cardiac arrest and ventricular fibrillation. However, one can safely state that as experience is gained with each procedure the number of unexpected emergencies decreases.

Even though it is more than 100 years since the first reported cardiac arrest and inspite of much progress in the field of anaesthesiology and surgery, there are no adequate means of anticipating, preventing or adequately treating this serious complication.

In this paper we are reviewing the 52 cases of cardiac arrest at C. M. C. Hospital, Vellore, from 1955 to September, 1959.

Historical Notes

The first cardiac arrest was reported in 1848 due to anaesthesia when chloroform was used to remove the nail from a toe. (3) In 1874 the Physiologist Schieff (3) described manual compression for cardiac resuscitation in dogs under chloroform and other deaths, but it was only in 1889 that the first cardiac massage was attempted in human patients. (3) Tuffier[16] in 1898 reported the first unsuccessful cardiac massage by the transthoracic approach, and in 1903, Igulsurd reported the first successful transthoracic massage.

Lane in 1902 described the first successful massage by abdominal route which was 54 years after the first reported cardiac arrest under anaesthesia. By 1909, 46 successful cases had been reported but renewed interest in resuscitation measures followed the report by Bailey and Beak[2] in 1941. In 1947 Beak[4] successfully used the electric defibrillator for ventricular fibrillation.

Incidence

Cardiac arrest has been reported as 1 : 2000 operative cases by Stephenson and associates[15]. It is more common in the first decade of life and in the later decades as well. There is a higher incidence in males than females. In our series the incidence of cardiac arrest is 1 : 450 for operations under anaesthesia.

Cardiac Arrest from 1955-1959.

Total number of operations	... 22,936
" " Cardiac Arrests.	52
Incidence of Cardiac Arrest	... 1 : 450

In our series it has also been found to be more common in males than in females, with a slightly higher percentage of survival rate in males. Total number of males operated were 53·2% and females were 46·8%.

Sex Incidence

	Sex % in operated cases	Number of Cardiac Arrests	Total % of Cardiac Arrests	Survival %
Male	53·2	34	65·3	14·7
Female	46·8	18	34·6	11·1

In the present series the age incidence is fairly equally distributed in the 1st, 2nd, 3rd and 4th decades of life. Figure No.1 shows the diagramatic presentation of age incidence and survival percentage.

The highest percentage of survival was noticed in the 4th and 5th decades (*i.e.*) 33·3% and 50% respectively, but the number is too small to be of statistical significance.

Briggs[5] reviewed cardiac arrest cases of the past 30 years and reported an absolute increase in cardiac arrest, due to wide awareness and increasing diagnosis of the condition, and the increasing number of operations on older and poor risk patients.

The figure No. 2 showing the yearly statistics of this series indicates similar findings.

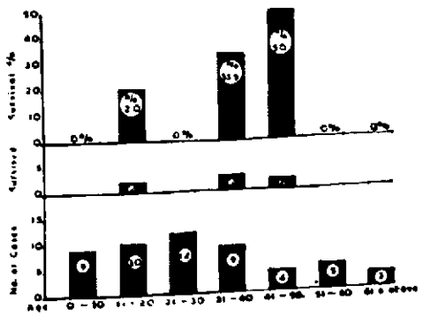

Fig. 1

The diagramatic presentation of age incidence and survival percentages.

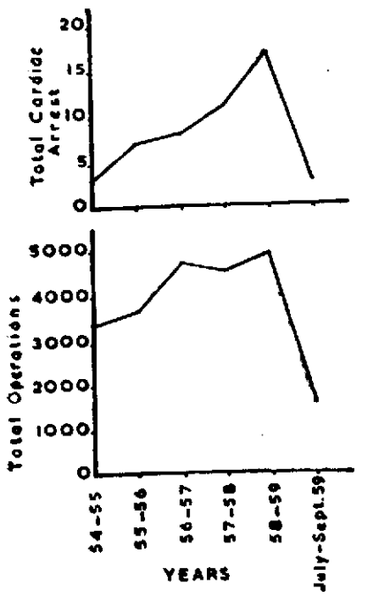

Fig. 2

The graphical presentation of the number of yearly operations and yearly number of cardiac arrest incidence.

According to Briggs[1] cardiac arrest is increased by :

5 fold in cardiac disease patients.
20 ,, elderly patients.
30 ,, poor risk patients.

Etiological Considerations

The fundamental aetiological factor is myocardial hypoxia, and hypoxia is probably the greatest hazard facing any surgical patient today. It strikes the heart and brain swiftly and without warning.

Hypoxia may arise from :

(*a*) Inadequate ventilation of lungs.
(*b*) Circulatory causes :
Hypotension.
Anaemia.
Myocardial damage.

The following tabular presentation shows the aetiological factors that we believe were mainly responsible in the present series. Often one or more factors were simultaneously involved.

Aetiological Relation and Percentages of 52 Cases

			Approximate %
1. Anoxaemia		5	10
(*a*) hypoxia	2		
(*b*) Respiratory inadequacy	3		
2. Circulatory		3	6
(*a*) Bleeding	3		
3. Reflex		12	24
(*a*) Vagal over-activity			
i. Hilar dissection	9		
ii. Bronchoscopy	1		
iii. Pericardial tapping	1		
(*b*) Severe painful stimuli (Light anaesthesia)	1		
4. Anaesthetic drugs		10	20
(*a*) Ethyl Chloride and Ether	2		
(*b*) Cyclopropane	2		
(*c*) Scoline	1		
(*d*) Prostigmine (High dosages)	1		
(*e*) Xylocaine	2		
(*f*) Anaithene	2		
5. Drug sensitivity			
(*a*) Pro-Penicillin	1	1	2
6. Diseases of Heart		4	8
(*a*) Mitral Stenosis	2		
(*b*) Tricusp. regurgitation (Pulmonary insufficiency)	1		
(*c*) Pericarditis	1		
7. Change in position (Sudden hypotension)		7	14
8. Hypothermia		2	4
9. C. N. S. Op. and reflex		2	4
(*a*) IV ventricle	1		
(*b*) Dural shock	1		
10. Unknown factors		6	12

Type of Cardiac Arrest

The heart may be arrested in a systole or may develop ventricular fibrillation. In the present series, on opening the chest for massage initial fibrillation of cardiac muscles was noticed only in 2 cases, but 10 more cases developed fibrillation subsequently. All the 12 cases that had fibrillation at one time or other ultimately succumbed. In the remaining cases the heart was in asystole.

Cardiac Asystole

In this state the heart is toneless, soft, dilated and dark-looking. It apparently is usually due to anoxia, hypoxia or vagal stimulation in the presence of anoxia. In ventricular fibrillation, the cardiac muscle shows fibrillatory movements which may be due to stimulation of the heart when it is in a state of hypoxia. The cardiac muscle may go into arrest during anaesthesia, operation, changing the position after operation or from the injection of drugs to which the patients may be sensitive. The following table shows the different situations at which the arrest was noticed in this series.

Cardiac arrest during Anaesthesia	11	21·1%
Survival	3	27·2%
Cardiac arrest during operation	33	63·4%
Survival	4	12·1%
Cardiac arrest during change of position after surgery	7	13·2%
Survival	...	00%
Cardiac arrest due to drug sensitivity (Pro-penicillin)	1	2%
Survival	...	00%

In our series arrest occurred more often in thoracic cases than associated with other surgery. In thoracic operations there is a greater opportunity for the combination of hypoxia and abnormal stimulation due to handling of the vital organs like heart and lungs. Total thoracic operations were 16·4% out of which 2·6% cardiac operations, 11·3% pulmonary and 2·6% oesophageal operations while 83·6% operations were performed in other departments.

Thoracic Procedures

Bronchoscopy	...	1
Thoracotomy	...	2
Lung Resection	...	11
Empyema drainage	...	1
Oesophagus	...	1
Mitral Stenosis	...	5
Pulmonary Valvotomy	...	1
Blalocks	...	1
P. D. A.	...	1
A. S. D.	...	1
Exp. Cardiotomy	...	1
Pericentasis pericardii	...	2
Total	...	28

Clinical Features

In cardiac arrest the patient is pulseless, the blood pressure is not obtainable, the pupils are dilated and non-reacting, the complexion is ashen, and respiration, if present, is of gasping nature. When cardiac arrest occurs from vasovagal reflex or drug sensitivity the onset is usually sudden, when it occurs from hypoxia there is usually a warning period of cardiac irregularity followed by gradual slowing of the heart rate.[2]

Total Cardiac Arrest

	Total No. of operations in %	No. of cases	Survived
Thoracic Operations ...	16·4	28	4
473 Cardiac operations ...	2·3	13	1
2020 pulmonary operations ...	11·3	13	3
449 oesophageal operations ...	2·6	1	—
Other Operations ...	83·6	24	3

Different types of Operation and Cardiac Arrest Incidence

Laparotomies	...	4
Gastro-intestinal tract	...	5
C. N. S.	...	6
T. B. Spine	...	1
Tendon transplant	...	2
Malignancy in general	...	2
Hysterectomy	...	2
Evacuation uterus	...	1
I. M. Injection (Penicillin)	...	1
Total	...	24

Physiological Approach to Management

Two fundamental steps must be observed and followed rigidly if life is to be preserved. Firstly, the airway should be kept clear and

artificial respiration, preferably with 100% oxygen and with the head in a low position carried out. Secondly, adequate cardiac massage should be started immediately. The objective is to keep up artificial circulation by cardiac massage with fully oxygenated blood, so that oxygen is supplied to the vital organs in adequate amounts until such time as the heart action is fully and durably restored. Lesser procedures like intracardiac drugs and artificial respiration only with 100% oxygen without cardiac massage, are usually ineffective and waste valuable time where every second counts.[9]

It has been proved beyond doubt that interruption of the cerebral circulation for over 3 minutes may lead to permanent changes in the psychic behaviour of the patient and after 8 minutes is usually incompatible with life.[12] Patients with low haemoglobin or low respiratory reserve may not stand interruption of cerebral blood supply even for 3 minutes.[11]

Prevention of Cardiac Arrest

There are no adequate means of preventing cardiac arrest, but the following plan may help to a certain extent.

Make the patient fit for operation.

Haemoglobin should be within normal limits.

Postural drainage to be given for bronchial secretions before surgery.

Heart failure should be corrected by proper therapy pre-operatively.

Adequate premedication should be given.

Meticulous care in anaesthesia in all patients, personal check-up of all anaesthetic agents and clear airway and proper oxygenation to be maintained.

Careful and gentle dissection near the vital structures.

Adequate haemostosis and adequate replacement of blood loss.

Management of Cardiac Arrest

Cardiac resuscitation means full and durable restoration of the essential functions of the heart, which from all appearances had lost its power to recover spontaneously in due time to avoid eventual death.[8]

Cardiac resuscitation implies :

i. Heart must have stopped functioning.
ii. Cardiac function must be fully and permanently restored.
iii. Life must be preserved.

Cardiac resuscitation requires immediate action and every effort should be made to start manual compression of the heart within 3 minutes of the onset of arrest. Any delay is prone to lead to cerebral anoxia with irreparable damage. Effective cardiac massage should produce a radial pulse and a blood pressure approximating 80 mm. of Hg.[11]

Unless a thoracic operation is being done the 4th left intercostal space is usually the best approach. A costal cartilage is divided anteriorly if more space is needed. The lung is retracted laterally and the heart is visualised. Sometimes massage is carried out from outside the pericardium for a few minutes. but routinely the pericardium is opened. The heart is grasped in the palm and is forcibly but rhythmically compressed so that the anaesthetist is able to feel the carotid pulse and there should be a B.P. of about 80 mm. of Hg. The compression is done at a rate of 40-60/min.

In the meantime the anaesthetist passes an endotracheal tube, if it is not already in or has been removed. The head end of the table is lowered about 10-15°. The lungs are inflated with 100% oxygen and all anaesthetic agents are cut off.

The procedure of cardiac massage through a thoracotomy incision may be unfamiliar to some clinicians, and they often hesitate too long before embarking upon what seems to be a drastic procedure. Even when the chest has been opened and the heart exposed there is often much uncertainty about the correct steps to take and valuable time may be wasted in injecting drugs, which may do more harm than good at this stage.[11]

In the present series :

Cardiac Massage done	...	46 cases
,, ,, not done	...	6 cases

The massage was done by		Survived
Thoracic route	43	6 (1 Rt. sided approach)
Abdominal route	3	1

The following chart shows the routine that is now being followed. This chart is posted permanently in each scrub room for ready reference and as a constant reminder of the possibility of cardiac arrest.

The figures No. 3, 4, 5, 6 & 7 shows the incision, mode of massage way to apply electrode of defibrillator, and emergency artificial respiration.

Fig. 3

Thoracotomy incision for cardiac massage.

In the majority of the cases the massage was started within 1-7 minutes, only in 4 cases it took 10-25 minutes, because in these cases the arrest took place outside the operating room and immediate thoracotomy facilities were not available.

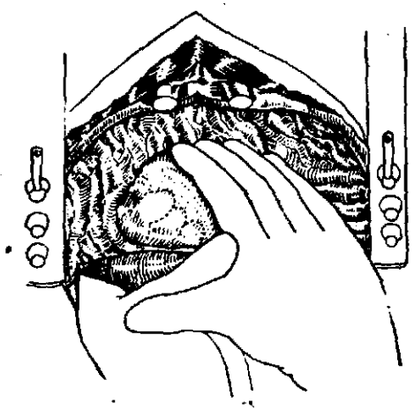

Fig. 4

Showing the method of bimanual compression of heart for cardiac massage.

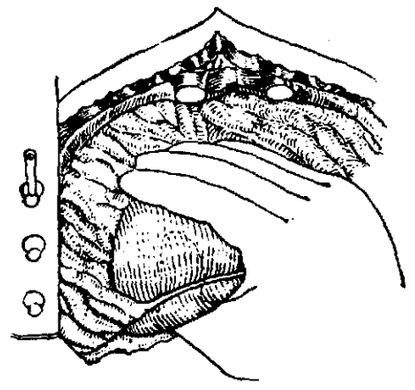

Fig. 5

Showing cardiac massage in progress with flat of the palmar surface of fingers and thenar eminance of the palm.

MANAGEMENT OF CARDIAC ARREST

Don't—Wait for electrocardiograph or cardioscope to diagnose and begin treatment. An absent B. P. and absent pulse in a major vessel (*e.g.*, carotid) means cardiac arrest.

Don't—**Give drug of any type** until type and cause of arrest is determined.

Don't—Fiddle and hope the heart restarts. **You have only 3 minutes to establish circulation before irreversible damage occurs.** So cut and massage.

DUTIES OF PERSONNEL

Anaesthetist	Surgeon	Nurse	Assistant Surgeon	Others
1 When carotid pulse cannot be felt or heart cannot be heard inform surgeon and **ask assistant to record time.**	1 When possible confirm presence or absence of pulsation in aorta.	1 As soon as absent pulse is reported bring emergency thoracotomy set.	1 Insert rib spreader and cut 1 or 2 adjacent cartilages for adequate exposure.	1 One person **call out time at one minute intervals** until adequate massage is established.
2a Stop all anaesthesia.	2a Open left chest in 4th interspace with transverse incision from sternal margin to mid-axillary line.	2 Bring emergency tray of syringes and drugs.	2 Stand by to relieve surgeon massaging the heart.	2 One person start I. V. and assist anaesthetist.
2b Respirate patient with 100% oxygen.	2b Massage the heart.			
2c Tilt patient head down 15 degress.	3 When adequate massage has been continued for 3 minutes pause to see if contractions have returned: if not follow col. 2 below.		3 Start I. V. if no one else is available.	
2d Pass endotracheal tube as soon as possible.				
3a When cardiac massage is established check radial or carotid pulse.				
3b Check B. P.				

POSSIBLE SITUATION EXISTING AFTER ADEQUATE MASSAGE AND OXYGENATION

1. The heart resumes a normal beat

(*a*) Continue massage in rhythm with beat until contractions are vigorous and B. P. is maintained at 80 systolic.

(*b*) If contractions do not improve or if they become weaker give adrenaline 1 : 1,000 0·5—1 ml. diluted to 10 cc. of normal saline in to the cavity of the left ventricle.

(*c*) If the heart is contracting well but the B. P. remains low a noradrenaline (4 mg. in 500 ml. 5% glucose) drip should be started.

(*d*) Observe the heart for at least 30 minutes of adequate action before closing the chest.

(*e*) Close the chest with **adequate haemostasis.**

2. The heart remains in arrest

(*a*) Open the pericardium.

(*b*) Observe the action of the ventricles for diagnosis.

(*c*) Observe the colour of the heart; if it is well oxygenated it will be a good pink colour.

A. The heart is in fibrillation	B. The heart is in asystole
1. Procaine 1% 2-10 ml. into cavity of left ventricle.	1. Continue adequate massage.
2. Massage for 3 minutes awaiting action of procaine.	2. Give CaCl 10% 5–10 ml. into cavity of left ventricle.
3. If no result use electric defibrillator (one of the thoracic surgical unit or a senior anaesthetist will be responsible for this).	3. If asystole persists and the heart is a good colour, give adrenaline 1 : 1,000, 0·5-1 cc. diluted to 10 cc. normal saline.
4. If shock is ineffective (or defibrillator is not available) give adrenaline 1 : 1,000 0·5–1 ml. diluted to 10 cc. of normal saline into cavity of left ventricle.	4. If heart beat returns follow col. 1.
5. If spontaneous beat returns follow col. 1.	5. If asystole persists continue massage up to two hours or until the heart fails to fill in diastole.
6. If heart is in asystole follow col. 2 B.	

CARDIAC MASSAGE

Adequate Cardiac Massage should produce a palpable peripheral pulse and should be carried out thus:

Grasp the heart with the whole hand, apply pressure with the palmar surfaces of the fingers and the thenar eminence. Do not use the fingertips. If the heart is large use both hands, 80–90 contractions per minute is the most effective rate but this may be difficult to maintain. The rate must be slow enough for the heart to fill during diastole, and the maximum rate possible may be only 50–60 per minute.

The cerebral and coronary circulations can be improved by occluding the aorta below the origin of the left subclavian artery. The clamp should be released every 20 minutes to allow circulation to the kidney.

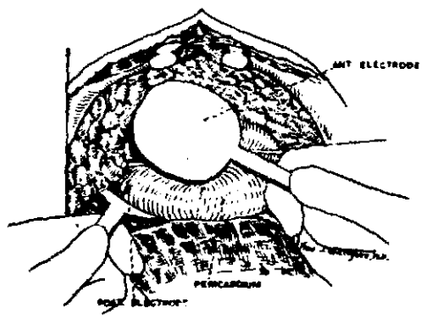

Fig. 6

Shows the method of applying electrodes to the fibrillating heart muscles for defibrillation.

Time taken to start the Massage after detection of arrest.

Time	Number of cases	Survived
1– 3 min.	24	7
4– 7 min.	18	—
10–25 min.	4	—

(It is evident from the figures that earlier the massage better are the chances of survival).

In our present series restoration of cardiac action was in 36 cases *i.e.*, 78·04% while 10 cases *i.e.*, 21·7% did not respond at all, but ultimately 7 cases *i.e.*, 13·7% survived permanently.

In the majority of cases the heart action was restored in 1-4 minutes and in this group were the maximum number of survivors. The longest period before restoration of cardiac action after massage was 112 minutes but the patient expired after

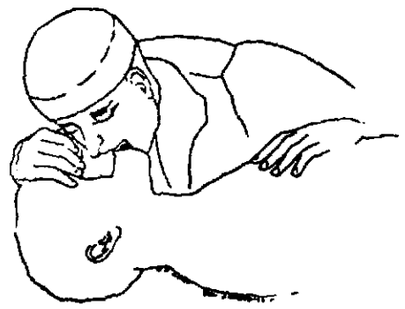

Fig. 7

Mouth to mouth breathing and artificial respiration in case of emergency.

4 hours. In the other two survivals, *one* was after 12 minutes and another after 25 minutes of massage.

Time of restoration of cardiac action after starting massage

Time	Number of cases	Survived
1– 4 min.	19	5
5–10 min.	9	—
10–20 min.	2	1
20–30 min.	4	1
40– min.	1	—
112 min	1	—

(This again proves earlier the massage and earlier restoration of cardiac activity, gives more of survival).

Following table shows the time interval in which cardiac activity was restored in the cases that survived permanently.

Survival Rate

No. of cardiac arrest	No. of Massage	Temporary restoration of cardiac action	Permanent restoration and Survival	% Survived
52	46	29	7	13·7

Restoration of Cardiac Action

(Survived cases)

Time		No. of cases
1– 4 min.	...	5
12– min.	...	1
25– min.	...	1

During massage intracardiac injection of calcium chloride, 10% solution, or adrenaline 1 : 1,000 or 1 : 10,000 may be given depending upon the state of the heart. Occasionally 1% solution of Procaine hydrochloride was given to attempt to abolish fibrillation. In one case a total of 50 cc. of calcium chloride and 12 cc. of 1 : 1,000 adrenaline was given over a period of 3 hours and 15 minutes the patient developed fibrillation and did not survive. Electric defibrillation was used in 12 cases.

Time to Persist in Massage of Heart

It is always a question how long to persist in carrying on the massage, and when to abandon massage as a hopeless case. The literature shows :

1. Lahey and Ruziks[16] continued massage for 2-45 minutes as long as they were getting some response from the cardiac muscle.

2. Adams[1] reported a case of cardiac arrest during P. D. A. division. He started massage at 60-70 beats/minute with the head in a low position and 0·5 cc. of 1% procain hydrochloride were injected into the left ventricle. The electric defibrillator was used 20 times with varying voltages and 10 cc. of 1% procain hydrochloride was given in the right ventricle, as well as 100 mgm. of pronestyl hydrochloride intravenously. After 90 minutes visible rhythm was seen in the left atrium, but it was not transmitted to the ribrillating left ventricle. Ultimately cardiac action was restored after 110 minutes and the patient made an uneventful recovery.

3. Haight and Sloan[7] reported complete and permanent recovery after 3 hours and 15 minutes of massage. During massage a right ventricular tear occurred at the site of an old infarct which was controlled by a gauze piece and later sutured. During all this time massage was continued.

In the present series massage was abandoned in 10 cases after 22 minutes to 3 hours and 40 minutes. In the majority of cases the massage was continued for at least 1 hour. Massage should be continued as long as even a little response from the cardiac muscle is obtained, and a good palpable pulse and reacting pupils are present.

Stephenson[18] and his associates in 1953 reviewed 1,200 cases of cardiac arrest and gave their statistics which is compared with our present series of 52 cases, below :

	Present series	Stephenson and associate statistic
Cardiac arrest	52	1200
Heart action restored	78·2%	56%
Permanent survival	13·7%	28%
Cardiac arrest outside operation room	7·7%	14%
% Survival of above	00%	17%

	Present series	Briggs series (5)
Last 10 years survival %	—	37%
Last 5 years survival %	13·7%	50%

In both the series the majority of cases who did not survive, died within 24 hours.

Restored Cardiac action but patient expired in

Few min. to 12 hours	13 cases	28·2%
12 to 24 hours	12 cases	26·1%
1 day - 8 days	4 cases	9·1%

After care

Every attempt should be made to keep the blood pressure at 80 mm. of Hg. or above. Oxygen is given continuously. Digitalis may occasionally be administered for tachycardia. Hosler" advises 20% dextrose and 3% saline intravenously to counteract the increased potassium ions in blood and possible accompanying arrhythmias. The patient should be kept in the operation room until spontaneous breathing is established and the blood pressure and pulse are satisfactory or cardiac arrest may recur at any moment. Only 7 cases survived in our series and were discharged from the hospital in good health.

Cardiac Arrest Outside operation room

In 4 cases cardiac arrest occurred outside the operation room. Massage was done in all these cases but it took 10-25 minutes to start massage, and ultimately all cases succumbed.

Causes of Failure

Failure to revive the arrested heart, according to Fautex (16) may be due to :

1. Hyperirritability of heart muscle from :
   (*a*) Cardiac ischemia.
   (*b*) Over stimulation of heart muscle by improperly balanced massage.
   (*c*) Injudicious use of adrenaline (epinephrin).

2. Absence of cardiac tone as evidenced by the heart being dilated and flaccid. Massage with overenthusiasm may bruise the cardiac muscle and the ensuing ischemia may impair conduction. Barium chloride or calcium chloride at this stage may be helpful.

3. Increased temperature of cardiac muscles. Due to repeated shocks for defibrillation, heat increases which in turn increases conductivity and a state of fibrillation becomes established with it is impossible to terminate. Pouring on of cold saline has been advocated.

4, Peripheral failure: If forceful compression does not bring about a palpable pulse and recordable blood pressure and the ventricle remain empty, 500 cc. of blood may be injected into both ventricular cavities.

Discussion

As soon as the cardiac arrest is noticed no time should be wasted in the fond hope of revival of heart which has lost its power to propel blood and all appearances is unable to recover spontaneously. Failure to resuscitate heart action is usually due to failure to appreciate the significance of the pulseless patient quickly enough to get cardiac massage started within the vital 3 minute period. Lesser procedures like intra-cardiac drugs are of little value and should be used only after massage has been started. Artificial respiration is inadequate by itself, but combined artificial respiration and manual cardiac massage may save many lives.

The importance of the following points should be noted :—

(1) The thoracotomy incision must be large enough to allow good exposure of the heart. (2) Because of the danger of rupture of the ventricles the finger-tips must not be used to massage the heart ; if the heart is compressed between the thenar eminence and the palmar surfaces of the fingers this danger is avoided. (3) Drugs must not be given until adequate massage has been continued for at least three minutes and until the type of arrest has been diagnosed. (4) Adequate cardiac massage produced a palpable peripheral pulse and a systolic pressure of 70-80 mm. Hg. ; (5) The rate of massage depends chiefly on the speed with which the ventricles fill during diastole. (6) Electrical defibrillation should not be attempted by anyone who is not familiar with this procedure (11).

Summary

1. 52 cases of cardiac arrest have been reviewed.

2. In 46 cases massage was done, the majority within 7 minutes of the onset of cardiac arrest.

3. In all cases that survived, massage was started within 3 minutes after detection of cardiac arrest.

4. Seven cases out of 52 survived giving a survival rate of 13·7%.

REFERENCES

1. Adams, R. in discussion of Sealy, W. C., Yound, W. G. and Harris, J. S.: Studies on cardiac arrest; The relationship of hypercapnia to ventricular fibrillation, J. Thoracic Surg., **28**: 461, 1954.
2. Bailey, H.: Cardiac massage for impending death under anaesthesia, Brit. M. J., **2**: 84, 1941.
3. Barber, R. F. and Madden, J. L.: Historical aspects of cardiac resuscitation, Am. J. Surg., **70**: 135, 1945.
4. Beck, C. S.: Resuscitation for cardiac standstill and ventricular fibrillation occurring during operation, Am. J. Surg., **54**: 273, 1941.
5. Briggs, B. D., Sheldon, D. B. and Beacher, H. K.: Cardiac arrest. Study of a thirty-year period of operating room deaths at Massachusetts General Hospital, 1925-1954, J. A. M. A., **160**: 1439, 1956.
6. Fautex, J. of Thoracic Surgery **16**: 627, 1947. Cardiac Resuscitation.
7. Haight, C. and Sloan, H.: Successful cardiac resuscitation despite perforation of the heart during massage, Ann. Surg., **141**: 240, 1955.
8. Hosler, R. M. Six years' experience with the Cleveland Cardiac Resuscitation course, A. M. A. Arch. Surg.
9. John Brorris: Emergency in Thoracic Surgery.
10. Lahey, F. H. and Ruzicks, E. R.: Experiences with cardiac arrest, Surg., Gynec. & Obst., **90**: 108, 1950.
11. Lewis, G. M, and Macpherson, N. S.: B. M. J. 58.
12. Ruzicks, E. R.: J. A. M. A., **135**: **622**: 47 cardiac arrest under anaesthesia.
13. Schiff, M. Recueil des Memoires physiologique, Lausanne, 1874. (Quoted by Milstein, B. B. in ref. 4).
14. Starling, E. H. and Lane, W. A.: Report of communication to society of Anaesthetists, Lancet, **2**: 1397, 1902.
15. Stephenson, H. E., Reid, L. C. and Hinton, J. W.: Some common denominations in 1,200 cases of cardiac arrest, Ann. Surg. **137**: 731, 1953.
16. Tuffier, T. and Hallion, L.: De La compression rhythmee du coeur dans la syncope cardiaque par embolie, Bull. et Mem. Soc. Chir. de paris, **24**: 937, 1898.

—Received for publication on 6-10-1960.

A TECHNIQUE FOR COMBINED TOTAL LARYNGECTOMY AND RADICAL NECK DISSECTION FOR CANCER OF THE LARYNX

BY

A. G. FLETCHER, Jr. and D. K. GOSAVI, Miraj.

Cancer of the larynx has a high incidence in India, comprising 12·1 per cent of a series of 5,640 cancer cases observed at the Tata Memorial Hospital[11]. The problem is complicated by the fact that most of these lesions (132 out of 139 in the Tata series)[11] arise in the so-called extrinsic larynx, or laryngo-pharynx. Formerly, practically all of these cancers, if they were treated at all, were treated by radiotherapy. However, the last decade has seen a definite swing back to radical surgery in the treatment of many oro-pharyngeal cancers and the same has been true of cancer of the larynx. At the Miraj Medical Centre we have increasingly resorted to total laryngectomy in the treatment of cancer of the extrinsic larynx. Our experience in a small group of 47 patients operated upon to date has led to the development of a standardized technique of combined radical neck dissection and total laryngectomy suitable for the type of lesion commonly encountered in India. It is the purpose of this paper to present the rationale and the technique of this operation.

Rationale

Cancer of the extrinsic larynx is a term which is commonly used to denote tumors arising from the mucosa of the pyriform sinus, the arytenoid, the aryteno-epiglottic fold, the epiglottis, and the post-cricoid area. The post-cricoid lesions really form a separate group, since they frequently require resection of the hypopharynx and cervical esophagus as well as the larynx. The surgical technique for this group will be presented in a separate report. Isolated lesions of the epiglottis are very rare in our experience and these likewise present a rather different problem. The same is true of cancer of the intrinsic larynx. The following discussion, therefore, deals with the type of lesion which is most commonly met with in India, namely, cancer of the pyriform sinus, the arytenoid, and the aryteno-epiglottic fold. Naturally, such tumors may extend, in their late stages, to involve the epiglottis, the base of the tongue, the esophagus or the endo-larynx.

Due to the location and rich lymphatic drainage, these cancers of the extrinsic larynx are characterized by early metastasis to the upper deep cervical lymph glands located along the upper part of the internal jugular vein. Taylor and Nathanson[9] have stated that the incidence of such metastasis may be as high as 71 per cent. Probably it was for this reason that such cancers were formerly considered unsuitable for surgical treatment. Certainly, the limited techniques for laryngectomy which were commonly used until a few years ago, and are described in most of the text books,[2,7,10] are quite inadequate for dealing with cancer of the extrinsic larynx. In recent years a number of surgeons[3,4,6,8] have led in the application of the basic principles of cancer surgery to this problem. The result has been a variety of suggested techniques for

radical resection of the larynx and laryngopharynx and radical neck dissection, either in continuity or in separate steps. In presenting our technique we do not make any claim of originality, though we have modified various details on the basis of our own experience and have laid stress on two points: (1) Wide resection of the tumor, the larynx and the adjacent tissues which might be involved by the tumor; (2) Radical neck dissection on the side of the lesion to remove the neck contents *in continuity* with the larynx.

Indications

The operation which is about to be described is indicated in all cases of cancer of the pyriform sinus, arytenoid, or aryteno-epiglottic fold which fulfil the following criteria. Extensive cancers of the intrinsic larynx may also be included, since metastasis to the cervical glands is likely to be present in this group also in the later stages.

(1) The larynx is freely movable.

(2) The growth is not fixed to the overlying skin.

(3) Metastasis to cervical lymph glands is palpable on one side only.

(4) Cervical glands are not fixed to the overlying skin or to deeper structures such as the carotid artery or the mastoid bone. Fixation to the sterno-cleido-mastoid muscle is acceptable.

(5) The base of the tongue is not involved.

(6) The esophagus and hypopharynx are not so extensively involved as to require circumferential excision.

(7) The general condition of the patient is such as to permit of major surgery with reasonable safety.

It will be seen that the assessment of a patient for operation will require:

(1) A careful external examination of the neck.

(2) Examination of the lesion by both indirect and direct laryngoscopy.

(3) Biopsy of the lesion.

(4) Evaluation of the general condition of the patient, including an x-ray of the chest to rule out metastasis or any other lesion.

If bilateral metastases are present, consideration can be given to the possibility of bilateral radical neck dissection if the primary lesion is not too far-advanced. This can be done in one stage but is probably better handled in two stages. If it is necessary to excise the esophagus circumferentially, the resection can be carried out very much as outlined here, but special techniques have to be employed for the reconstruction of the esophagus. This technique will be discussed in a separate communication. Even in cases in which the cervical lymph glands on the side of the lesion are not enlarged to palpation, radical neck dissection in continuity with radical total laryngectomy is indicated, since there is a strong probability that microscopic metastases are already present in these glands.

Pre-operative Preparation

These patients usually present themselves with a history of increasing hoarseness of voice, dysphagia and dyspnea. If there is marked respiratory obstruction it is better to do an immediate tracheostomy under local anesthesia before proceeding with routine investigations. The tracheostomy should be done at a high level, through the cricothyroid membrane or the cricoid ring, in order to cause minimal interference with

the later operation of laryngectomy. A low tracheostomy leaves only a short tracheal stump for formation of the tracheal stoma and also interferes with the skin incision.

The routine diagnostic procedures are mentioned above. Biopsy should never be omitted, since it is possible for tuberculosis of the larynx to present an appearance very similar to that of cancer. If dysphagia is marked, a nasogastric tube may be passed for purposes of pre-operative nutrition or a gastrostomy may be done. Adequate vitamins should be given. If the hemoglobin is very low, one or two blood transfusions may be given before operation. As a general rule, at least 1000 cc. of whole blood should be kept on hand for transfusion during the operation.

The night before operation the entire neck, face and upper chest are carefully shaved and washed. The shaving is carried out up to a level above the ears. In the morning a nasogastric tube is passed into the stomach and securely taped at the nose. This is essential for post-operative feeding and it is much better to put it in place before operation. An injection of streptomycin and penicillin and adequate premedication are given and the patient is sent to the operation theatre.

Anesthesia

The subject has been discussed by Boyan and Howland[1] on the basis of experience with 1,029 laryngectomies. In general the principles suggested them have been followed. In most cases, even where preliminary tracheostomy has been necessary because of respiratory obstruction, it is possible to pass an ordinary endotracheal tube with a balloon cuff through the larynx into the trachea. This is very desirable, since the tracheostomy tube can be removed and the field left clear for the required procedure. Under these conditions we have found the following technique very satisfactory :

(1) The trachea and larynx are topically anesthetized by dropping 2 per cent anethane into the tracheostomy tube.

(2) Anesthesia is induced with intravenous medium pentothal.

(3) A temporary connection is made between the tracheostomy tube and the anesthesia machine and surgical anesthesia is obtained with ether and oxygen using a closed system.

(4) The orotracheal tube with inflatable cuff is introduced through the larynx in the usual manner and the tracheostomy tube is withdrawn. The cuff is inserted in such a way as to block the tracheostomy opening and to prevent blood and secretions from going down the trachea.

(5) A light plane of anesthesia with ether and oxygen has proved very satisfactory for maintenance.

If there has been no necessity for previous tracheostomy, the orotracheal tube is introduced the balloon cuff inflated, and anesthesia maintained in the usual way, leaving the neck free for the surgeon. If the growth in the larynx prevents the passage of the endotracheal tube, it is necessary to introduce a cuff tube through the tracheostomy opening, which may be enlarged if necessary, for maintenance of anesthesia. This is done after induction of anesthesia as outlined above. An ordinary endotracheal tube may be used and passed upward near the patient's ear on the side opposite to the neck dissection for connection to the anesthesia machine outside the surgical field. The initial stage of the operation must then be carried out with this tube in place.

Whichever method is used, the essential point is that anesthesia must be administered through an endotracheal tube provided with an inflatable cuff to occlude the trachea and prevent the passage of blood and secretions into the lung.

Technique of Operation

Position.—The patient is placed in the supine position with a thin pillow under the shoulders. The neck is extended and the head turned toward the side opposite the lesion, thus exposing the neck on the side of the lesion. The table is tilted somewhat downwards at the feet and up at the head to bring the surgical field into a horizontal plane. An intravenous infusion is started in the saphenous vein at the ankle by venepuncture or venesection. A very wide area from the nipples to the lower half of the face and including both sides of the neck and both shoulders is prepared.

Incision.—The preferred incision is shown in Fig. 1. The transverse area of the incision begins near the mastoid process and extends across the neck to the opposite tip of the hyoid bone. The vertical arm starts downward and then curves laterally to meet the central part of the clavicle. This incision provides adequate exposure and leaves the suprasternal area free for the formation of the tracheal stoma. Naturally, a modification of the incision may have to be made when there is a previous tracheostomy. The incision is deepened through the platysma muscle and flaps consisting of skin, subcutaneous tissue and platysma are widely dissected in all directions. The limits of dissection are the margin of the mandible above, the clavicle below, the edge of the trapezius muscle on the side of the neck dissection, and the sternomastoid muscle on the opposite side. Care is taken to preserve the mandibular branch of the facial nerve, if possible. Throughout the operation, bleeding points are grasped with fine mosquito forceps and controlled with fine cotton ligatures, or with the electric cautery on the specimen side. Sharp dissection with a scalpel or fine scissors is used throughout. Care is taken to avoid rough handling of tissues or excessive manipulation of the tumor (Fig. 2).

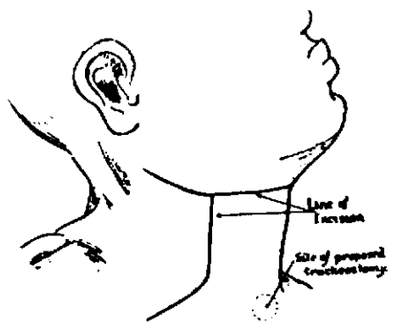

Fig. 1
Skin Incision.

Lower Neck Dissection.—The neck dissection is now begun and follows in general the technique so well described by Martin[6]. The sternomastoid muscle and the internal jugular vein are divided and reflected upward for a short distance, exposing the

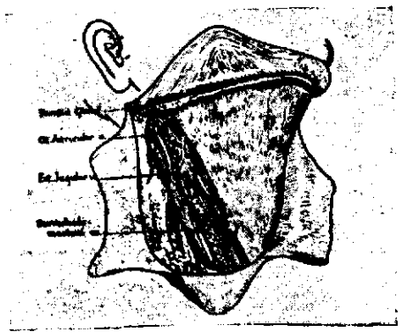

Fig. 2

Flaps of skin and platysma muscle reflected to expose the field of operation.

common carotid artery and the vagus nerve and, lateral to these, the phrenic nerve lying on the anterior scalene muscle. Laterally, the omohyoid muscle and the spinal-accessory nerves are divided and the fatty contents of the posterior triangle are dissected upward and medially, exposing the brachial plexus. Laterally, this dissection is carried upward along the anterior edge of the trapezius muscle to the level of the mastoid. As this is done, the specimen is reflected medially instead of turning it upward as in the routine neck dissection. In this way, the neck contents remain attached all along the lateral border of the larynx, but the carotid artery is exposed to a point well above the bifurcation, bringing the hypoglossal nerve into view. The superior thyroid branch of the external carotid artery is divided and ligated (Fig. 3).

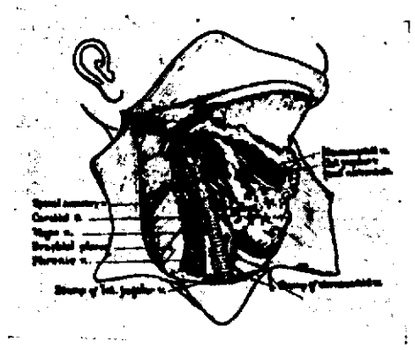

Fig. 3

Lower part of neck dissection completed. Neck contents reflected medially and still attached to larynx.

Upper Neck Dissection.—The dissection now begins at the margin of the mandible, reflecting downward the contents of the submental and the submaxillary triangles. Laterally, the lower pole of the parotid gland is exposed but usually is not resected. The upper end of the sternomastoid muscle is now transected just below the mastoid process. This exposes the belly of the digastric muscle, which is retracted upward to provide a maximal exposure of the upper end of the internal jugular vein. The vein is doubly ligated and divided at a high level, including with the specimen as much as possible of the surrounding areolar and lymph-node-bearing tissue. The accessory nerve is again sectioned at this level and the hypoglossal nerve identified and preserved. The entire neck dissection specimen is now free inferiorly, superiorly and laterally, remaining attached only to the larynx and thyroid medially (Fig. 3).

Ipsilateral Mobilization of Larynx and Thyroid Lobe.—Attention is now turned to the mobilization of the larynx. On the side of the lesion (*i.e.* the side of the neck dissection) the entire thyroid lobe is mobilized with the larynx and remains attached to it, since there is a danger of involvement of the upper pole of the gland by direct extension of the tumor. The sternothyroid and sternohyoid muscles are first transected just above the sternal notch and reflected upward, exposing the thyroid lobe. The inferior thyroid veins and the inferior thyroid artery are divided and ligated to mobilize the gland. The recurrent laryngeal nerve is divided. The inferior constrictor muscle is divided along the posterior margin of the thyroid cartilage, swinging the neck dissection specimen medially to obtain exposure of this area. Above the cornu of the thyroid cartilage, the superior laryngeal nerve and artery are divided and ligated (Fig. 4).

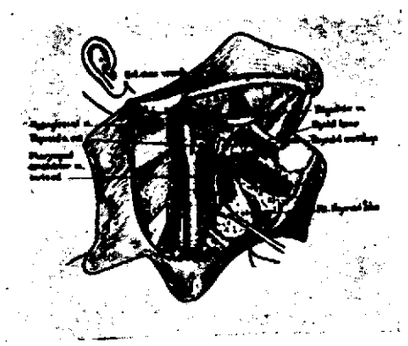

Fig. 4

Upper part of neck dissection completed. Mobilization of larynx on the side of lesion completed, including right thyroid with the specimen.

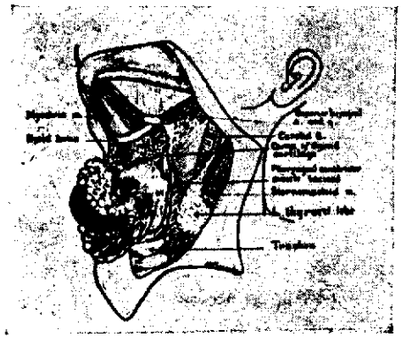

Fig. 5

Mobilization of larynx on side opposite to the lesion completed, reflecting left thyroid lobe away from the specimen and preserving it.

Contralateral Mobilization of the Larynx.—On the side opposite to the lesion, the entire thyroid lobe together with the attached parathyroid is to be preserved. The sternomastoid muscle is retracted laterally and the sternohyoid and thyrohyoid muscles are transected just above the sternal notch and reflected upward, freeing them from the anterior surface of the thyroid capsule. The thyroid isthmus is transected and the thyroid lobe is reflected laterally and separated from the trachea and the larynx. Many small branches of the inferior and superior arteries which enter the trachea and the larynx, must be clamped and ligated. The upper pole and the superior thyroid artery and vein are also reflected laterally away from the larynx. The inferior constrictor muscle fibers and the superior laryngeal artery and nerve are divided exactly as on the opposite side (Fig. 5).

Suprahyoid Dissection.—Attention is now turned to the region above the hyoid bone, where the mylohyoid and hyoglossus muscles are transected. The hyoid bone is divided on either side just medial to the attachment of the digastric tendon, thus removing the central part of the bone with the specimen and leaving the tips *in situ*. Formerly, we used to preserve the hyoid bone but it has been found much more satisfactory to remove most of it for several reasons: (1) It makes it easy to do a wide excision around the epiglottis, removing part of the base of the tongue, if necessary; (2) It facilitates closure of the hypopharynx after laryngectomy; (3) removal of the hyoid causes no demonstrable interference with swallowing.

The larynx is now completely mobilized, remaining attached only by the hypopharyngeal mucosa above and the trachea below. The neck dissection specimen is still attached on the side of the lesion (Fig. 6).

Transection of the Trachea.—The balloon of the endotracheal tube is now deflated, the tube withdrawn somewhat, and the trachea transected. Unless there is subglottic extension of the tumor, this is done at a high level, usually just or below the first tracheal ring. The orotracheal tube is now removed and a sterile right-angled cuffed tube, designed specially for this purpose[1], is inserted into the distal tracheal

Fig. 6
Division of suprahyoid muscle and hyoid bone completed.

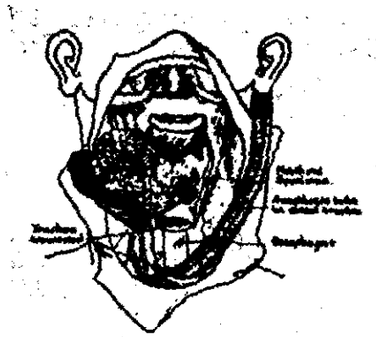

Fig. 7
Trachea transected and anesthesia tube placed in distal tracheal stump.

stump and the other end passed up to the anesthetist for connection with the anesthesia machine. Stay sutures in the margin of the tracheal stump are tied around the tube to secure it in place and the cuff is firmly inflated to provide a closed system and to block the trachea against blood and secretions (Fig. 7).

Transection of Hypopharynx.—The mucosa of the hypopharynx is now opened near the cornu of the thyroid cartilage on the side opposite the lesion. As this incision is enlarged and the larynx rotated, a good view of the lesion is obtained. Under direct vision the mucosa is now divided all the way around, carrying the incision at least one centimetre beyond the grossly visible or palpable limits of the cancer. The specimen is removed (Figs. 8 and 9).

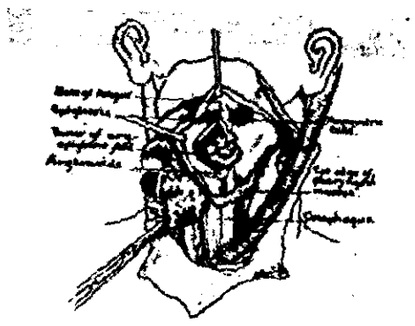

Fig. 8
Hypopharynx opened on left side, opposite to the lesion, exposing tumor of arytenoepiglottic fold as larynx rotated.

Fig. 9
Larynx and neck contents removed after incision of mucosa of hypopharynx. Defect of hypopharynx exposed.

Fig. 10
Hypopharynx closed using "T"-shaped suture line. A transverse suture line is possible in many cases and provides a more satisfactory closure.

Closure of the Hypopharynx and Esophagus.—This step is extremely important, since carelessness or poor technique will result in troublesome post-operative fistulae. Often the hypopharynx can be closed transversely, and this is probably ideal. If this is not possible, a "T"-shaped closure is generally the most useful. In either case the first layer of the closure is a continuous inverting Connell suture of 000 atraumatic chromic catgut. This suture includes the mucosa and some of the supporting tissues. It is re-inforced by a second layer of interrupted sutures of fine cotton. In placing these sutures, the posterior belly of the digastric, the muscles of the tongue, the suprahyoid muscles and the pharyngeal constrictor muscles are made use of to provide support to the suture line. Naturally, tension on the sutures must be avoided (Fig. 10).

Closure of the wound.—After closure of the hypopharynx, the entire wound is washed out thoroughly with warm saline solution, taking care to prevent fluid from entering the trachea, which is still occluded by the special tube and inflated cuff. Hemostasis is carefully checked and penicillin and streptomycin powder is placed in the wound. Two small catheters, near the tip of which many holes have been cut, are placed in the wound and brought out through small stab wounds in the skin flaps on either side. These catheters are secured in place with cotton skin sutures and are used for post-operative suction drainage of the wound. The skin flaps are carefully approximated by means of interrupted fine cotton sutures for the platysma muscle and a second layer of interrupted fine black cotton skin sutures (Fig. 11).

Tracheal Stoma.—The completion of the tracheal stoma is left until the last, since to accomplish this the anesthesia tube must be removed and, the patient is likely to start waking up and moving soon afterwards. Naturally, it is important to insure that effective, spontaneous respiration is present before the anesthesia tube is removed. The open end of the trachea is now cut somewhat obliquely, with the long lip posteriorly, in order to provide for a wider stoma. A large, round hole is now cut in the skin flap in the suprasternal area, and the trachea is brought through this hole

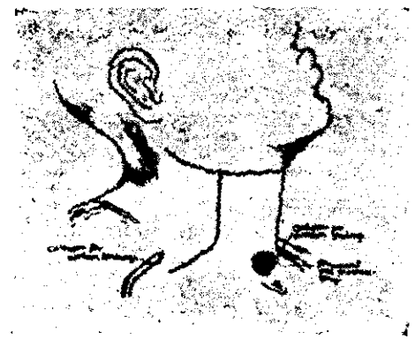

Fig. 11
Operation completed by closure of skin incision and formation of tracheal stoma. Note catheters in place under the skin flaps for suction drainage.

for suturing to the skin margins. It is important to have a wide-open stoma, since there is always a tendency for the stoma to contract post-operatively, and an unduly narrow stoma is often very troublesome and difficult to correct later on. The tracheal margin is secured to the skin edges with numerous interrupted sutures of fine black cotton. A Martin-type laryngectomy tube[3] is placed in the stoma and a light dressing applied to the wound. A bulky pressure dressing is not necessary since suction under the skin flaps is to be used.[2]

Post-Operative Care

After operation, continuous suction is applied to the catheters which were placed beneath the wound flaps. This is maintained for about forty-eight hours after which the catheters may be removed. In most cases the total drainage of blood and serum amounts to about to 150 cc. Suction may be applied by means of a Wangensteen apparatus or by means of the pump used for intra-thoracic suction drainage.

As soon as the patient has come out of anesthesia, the head of the bed is elevated somewhat, to promote drainage from the face and neck and thus minimize edema. The tracheostomy is aspirated at least once every hour for the first twenty-four hours and thereafter as required. The tracheostomy tube is cleaned, boiled and replaced several times daily and the area around the tracheal stoma is kept as clean as possible. The tracheostomy tube may be dispensed with after the fourteenth day, when the tracheal stoma is well-healed.

Suction is applied to the nasogastric tube for the first twentyfour hours until any tendency to nausea or vomiting has subsided. Then feedings of milk, tea, raw eggs, soup, etc., are given through the tube. If the condition of the wound is satisfactory, oral intake is cautiously begun on the eighth day after operation. At first only glucose water is given, one ounce every hour. If this is swallowed well, without evidence of leakage, the quantity is increased next day. On the third day the tube may be removed and a full liquid diet may be given. Solid food is started gradually from the fourteenth day after operation. Occasionally, a small fistula may be noted after oral fluids are started. If this occurs, oral intake is stopped and feeding is continued by means of the nasogastric tube. Usually, such fistulae will close within 8 to 10 days. We have no experience with large fistulae requiring plastic surgery for closure.

A combination of 400,000 units of penicillin and ½ Gm. of streptomycin is injected twice daily for the first six days and once daily thereafter, till the fourteenth day, to prevent infection of the extensively contaminated wound. The patient is encouraged to sit up and move about from the first day after operation. Most of the skin sutures may be removed on the seventh day, but those around the tracheal stoma are left until the tenth day.

Clinical Experience

The technique presented in this paper was developed as a result of experiences with forty-seven total laryngectomies for cancer performed at the Miraj Medical Centre and the Wanless Tuberculosis Sanatorium over a period of several years. In fourteen cases (the earlier ones in the series), only a "wide-field" total laryngectomy was performed, without complete radical neck dissection. In fifteen cases, resection of the larynx, hypopharynx and cervical esophagus was done for cancer of the hypopharynx, and various abdominal viscera were employed for reconstruction of the cervical esophagus. In two cases, radical neck dissection and radical total laryngectomy were performed in separate staged operations. In another case total laryngectomy was combined with hemiglossectomy, hemimandibulectomy and radical neck dissection in the treatment of cancer of base of tongue. Fifteen patients have been operated upon with the technique described in this paper.

Fourteen of these 15 patients were males. In 12 cases the cancer originated in the pyriform sinus. In two cases the primary site was the vocal cord and in one case it was the post-cricoid area. In eleven out of the 15 cases (73·3 per cent) tumor metastasis was found in the glands removed by radical neck dissection, thus emphasizing the need for radical neck dissection whenever total laryngectomy is performed in the treatment of extrinsic cancer of the larynx.

The operation consisted of a radical total laryngectomy combined with a radical neck dissection on the side of the lesion in each case. In six of the fifteen cases tracheostomy had been performed prior to the main resection. The mucosa was closed transversely in five cases and a "T" shaped closure was employed in ten cases. The average time required for the operation was about five hours. An average of 800—1200 cc. of whole blood was given during the course of the operation.

Post-operative complications consisted of a small wound infection in two cases and a temporary salivary fistula in three cases. Two of the fistulae were very small and closed spontaneously within eight days. The third fistula was larger and required six weeks for complete closure. However, it closed spontaneously, without further surgery, and a good functional result was obtained. There was no mortality. All patients were discharged in good condition, eating a normal diet. In spite of the loss of voice, the patients are uniformly happy with the results of this operation, since they are relieved of a painful and foul-smelling tumor with its attendant difficulties in breathing and swallowing. These patients were operated upon too recently for significant evaluation of results in terms of cure of cancer, but it is reasonable to suppose that an extended, carefully planned operation of this type would offer a better chance of cure than more limited surgery or radiotherapy.

Discussion

It is the opinion of the authors that the operation described in this paper represents the best available treatment for cancer of the "extrinsic larynx" (pyriform sinus, arytenoids, aryteno-epiglottic fold) and extensive cancer of the intrinsic larynx. In all such cases ipsilateral radical neck dissection should be done even when grossly enlarged glands are not palpable.

The operation is a painstaking and exacting one. It must not be performed hastily or roughly. Time should not be wasted, but the objective is not speed but rather complete eradication of the cancer. At least four hours will be required, even after considerable experience. If hemostasis is accurate and complete, the blood loss is only moderate and the patient remains in excellent condition throughout the operation.

In India, where the incidence of oropharyngeal cancer is high, there should be more emphasis on the surgical treatment of these lesions by modern methods and steps should be taken to train younger surgeons in the detailed and exacting techniques required.

Summary

1. Cancer of the larynx, especially the "extrinsic" type originating in the pyriform sinus, arytenoids and aryteno-epiglottic folds, is common in India.

2. Since these cancers metastasize early to the upper deep cervical lymph glands, the ideal surgical treatment is radical total laryngectomy combined with ipsilateral radical neck dissection in continuity. This is true even where enlarged lymph glands are not palpable.

3. A technique for performing this operation is presented in detail.

4. This technique is based upon an experience with total laryngectomy in forty-seven cases, the last fifteen of which were performed according to this technique with gratifying results.

REFERENCES

1. Boyan, C. P. and Howland, W. S. : Management of Anesthesia for Laryngectomies, Cancer **11**. 550, 1958.

2. Clerf, L. M. : Laryngectomy, Surg. Gynec. & Obst. **86**, 197, 1948.

3. Hoover, W. B. : The Technique for "Wide Field". Total Laryngectomy, Surg. Clin. N. America **36**, 589, 1956.

4. Jackson, C. L., Blady, J. V., Norris, C. M. and Robbins, R. : Carcinoma of the Larynx, J. Am. Med. Assocn. **163**, 1567, 1957.

5. Martin, R. : Surgery of Head and Neck Tumors, Paul B. Hoeber, Inc., New York, 1957.

6. Martin, H., Del Valle, B., Ehrlich, H. and Caham, W. G. : Neck Dissection. Cancer **4**, 441, 1951.

7. New, G. B., Figi, F. A., Havens, F. Z. and Erich, J. B. : Carcinoma of the Larynx, Surg. Gynec. & Obst. **85**, 623, 1947.

8. Pollock, R. S. : Laryngectomy and Radical Neck Dissection, Cancer **8**, 1177, 1955.

9. Putney, F. J. : The use of Continuous Negative Pressure after Laryngectomy and Radical Neck Dissection, Surg. Gynec. & Obst. 103, 244, 1956.

10. Ward, G. E. and Hendrick, J. W. : Tumors of the Head and Neck. The William and Wilkins Company, Baltimore, 1950.

11. Wynder, E. L., Bross, N. J. and Day, E. : A Study of the Environmental Factors in Cancer of the Larynx. Cancer **9**, 86, 1956.

—Received for publication on 1-11-1960.

PRIMARY PYLORIC HYPERTROPHY IN ADULTS

(A report of 2 cases)

BY

R. S. GUPTA and S. S. BAIJAL, Calcutta.

Primary pyloric hypertrophy in adults is an interesting clinical entity. Since its first description by Portal and Curveilheir in 1885, more case reports have appeared in the world literature. A number of investigators consider this condition as a manifestation in adulthood of a congenital disease, and hence the name "achalasia" of the pylorus has been applied.

Case Report

Case 1.—A female patient aged 21 years was admitted for vomiting 7 to 8 times a day for the past 6 days. The patient would not eat anything for fear of vomiting. The vomits contained ingested food matter with little bile. The patient was having pain in the epigastric region for the past 6 months with occasional vomiting.

Past history did not reveal much. The patient had no difficulty in taking milk in the first year of birth or since, till the present complaint 6 months ago.

On examination, a thinly built, obviously dehydrated female markedly anaemic, was seen with a Pulse rate of 90 p.m., B. P. of 110/70 and a temperature of 98°F. On palpation of the abdomen, a mass $\frac{1}{2}'' \times \frac{1}{2}''$ was felt in the right hypochondrium just to the right of the midline. Visible peristalsis could be seen going from left to right in the epigastrium. The lump was intra-abdominal and could not be moved from side to side, and was tender on palpation. The rest of the examination was essentially negative. Laboratory examination showed a-Hb. of 70%, a W. B. C. count of 5,900; urine - NAD, stool - NAD; B. T. 46 secs., C. T. 3 min. 41 secs. An F. T. M. showed absence of free hydrochloric acid. A Barium Meal examination showed pyloric obstruction. Very little barium trickling through after 6 hours.

The patient was treated by stomach washouts, and milk diet for 6 days and was put up for operation on 16-3-59. The abdomen was opened by a right paramedian incision and the pylorus was felt to be firm in consistency giving the impression of a tumour formation. No evidence of scarring due to any ulcer was seen. A partial gastrectomy with a Polya-Hoffmeister antecolic antiperistaltic anastomosis was done.

On opening the removed stomach, marked narrowing of the pyloro-antral junction was seen, so much so, that a probe could be passed through only with difficulty. On incising the pylorus the pyloric canal was markedly narrowed and the wall was thickened (Fig. 1.) The mucosa of the pylorus appeared hypertrophied. There was no evidence of any ulcer. Histological examination of the pyloric canal showed —"Formation of papillae of mucosa with thickened muscular coat without any evidence of inflammatory cells." This is shown in (Figs. 2 & 3.)

The post-operative course was uneventful and the patient was discharged on the 14th post-operative day. At follow up, one year after the operation the patient was well with no complaints, having put on about 18 lbs. in weight.

Fig. 1

Case 1—Specimen showing marked narrowing of the pyloric canal.

Fig. 2

Case 1—Microphotograph showing formation of papillae of mucosa (H. E. × 200).

Fig. 3

Case 1—Microphotograph showing hypertrophy of the muscular layer without any inflammatory cells (H. E. × 200).

Case 2.—A male patient aged 24 years was admitted for pain in the epigastrium with vomiting off and on for the past one year. The patient had been well till one year ago when he started getting vomiting off and on after meals with mild pain in the epigastrium. The vomiting gradually progressed and for the last one month he had vomiting after each meal. There has been loss of weight of about 14 lbs. in the last one year.

The patient had been well previously and there was no history of any complaints in childhood.

On examination a thinly built adult male slightly anaemic was seen. On palpation of the abdomen, tenderness on deep palpation, to the right of the midline in the epigastrium was elicited. There was no palpable lump. Visible peristalsis in the epigastrium from left to right was seen after giving a drink of water. Charcoal retention in the stomach was present after 24 hours. Laboratory investigations showed: Hb. 72%, W. B. C. 7200; urine - NAD; stool - NAD. Fractional analysis showed achlorhydria. A Barium meal examination showed pyloric stenosis. There was almost complete retention of barium in the stomach after 6 hours.

The patient was operated on by a right paramedian incision and the abdomen opened. The pylorus was found thickened giving the impression of a tumour. There was no scarring or evidence of any ulceration in the stomach. A partial gastrectomy with a Polya Hoffmeister antecolic antiperistaltic anastomosis was done.

On opening the stomach marked stenosis at the pyloro-antral junction was seen. The opening was narrowed to the size of about 2 m.m. (Fig. 4). The pyloric canal was found narrowed with thickening of the pyloric muscle, and the mucosa of the pylorus was hypertrophied (Fig. 5).

Histological examination of the pyloric region showed "marked thickening of the muscle walls with the presence of thickened nerves. There are numerous mucosal folds without any evidence of inflammation" (Figs. 6 & 7).

The Post-operative course was uneventful, the patient being discharged on the 14th post-operative day.

Fig. 4

Case 2— Resected specimen showing marked stenosis of pyloro-antral junction.

Fig. 5

Case 2—Showing marked narrowing and thickening of pyloric canal.

Follow up

The patient is well 6 months after operation and is doing his normal duties. He has no complaints, can eat a normal meal and has put on 12 lbs. in weight.

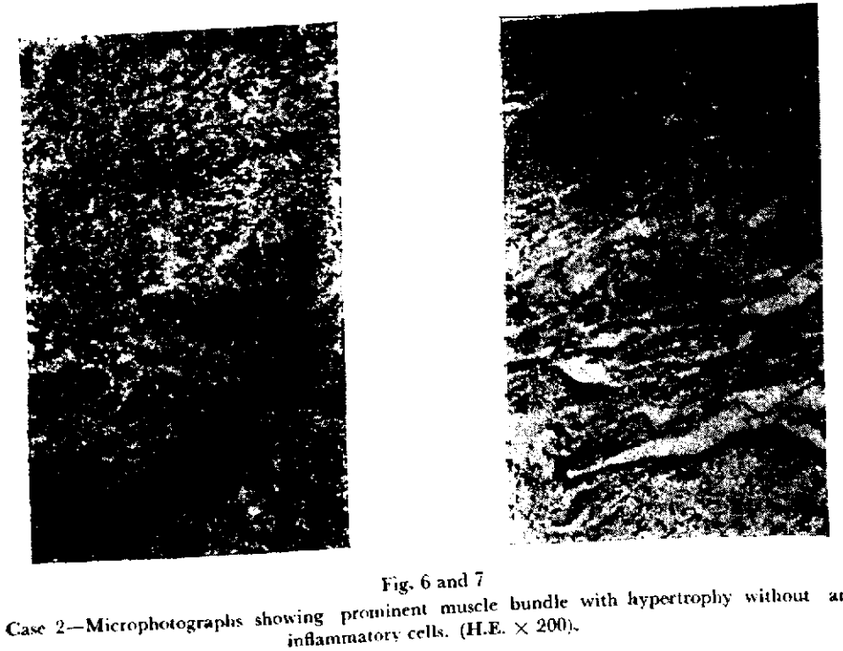

Fig. 6 and 7

Case 2—Microphotographs showing prominent muscle bundle with hypertrophy without any inflammatory cells. (H.E. × 200).

Discussion

Correa Netto, in a detailed clinical study attempted to identify this condition with achalasia of other sphincters of the digestive tract. Raia described lesions in the myenteric plexuses of the pylorus which could lead to complete destruction of the nerve elements which were replaced by connective tissue. The pathogenesis of this disease may therefore be explained on the basis of Hurst's theory of achalasia. A relationship may also be drawn between the development of primary pyloric hypertrophy in adults and the structure and function of the pyloric canal. Cole and Wernstedt's work tends to support the above view. The pyloric canal in man contains a specialised structure of circular muscle fibres comprising the pyloric wing muscle. This muscle which is fan-shaped consists of two sphincteric loops enclosing interpositioned circular fibres. The fibres of these two sphincteric loops converge on the lesser curvature forming a muscular prominence called the muscle torus. Torgersen and Cunningham considered also the structure of the longitudinal musculature of the pyloric canal as supporting evidence for the specialised function of the pyloric wing muscle. Torgersen further suggested a functional imbalance between the circular and longitudinal musculatures of the pyloric canal in the causation of infantile pyloric hypertrophy. This may persist into adult life either asymptomatically or with mild symptoms. These mild symptoms may be aggravated later on by some secondary factors. It is also possible that a functional lesion alone is present at birth with the organic phase developing in adult life. In both of the cases under review, there was marked hupertrophy of the proximal sphincteric loop of circular muscle fibres resulting in an extreme degree of stenosis at the pyloro-antral junction. The mucus membrane in both cases showed oedema and numerous sinusoid vessel conglomerates in the submucosa. There was a complete absence of fibrous tissue in the histopathology slides, which would negate a previous inflammatory process.

Bockus divided cases of pyloric hypertrophy in adults into three groups according to the duration of symptoms: (1) those in whom symptoms dated from infancy, (2) those in whom symptoms have been present for a long duration but beginning in adult life, (3) those with symptoms of short duration starting in adult or late life. In both the cases under review, the symptoms started off in adult life and were of relatively short duration. A very searching history could not trace back the symptoms to more than 6 months in case 1, and 12 months in case 2. Yet at operation a marked degree of stenosis was seen. This would tend to support Torgensen's suggestion of functional imbalance between the circular and longitudinal musculatures of the pyloric canal present since infancy, without symptoms, with the sudden development of a more pronounced hypertrophy of the circular muscle of the pylorus, triggered by some secondary factors in adult life, causing acute symptoms. We are, therefore, inclined to feel that the aetiology of primary pyloric hypertrophy of adults can be explained more on the structure and function of the pyloric canal, than on any developmental defect in the myenteric plexuses of the pylorus.

The symptoms of the condition are indistinguishable from those of stenosis due to ulcer of the pyloric canal. The main symptoms being a mild pain in the epigastrium with nausea and vomiting, resulting in definite weight loss and a low gastric acid curve. Both the cases showed achlorhydria.

On radiological examination a marked degree of pyloric stenosis is seen. An elongation of the pyloric canal is usually described. In the majority of cases an abrupt narrowing of the pyloric canal in contrast to a well distended antrum is present. The hypertrophied musculature of the circular coat may produce a concave impression on the base of the duodenal cap, a sign described by Kirklin and Harris. This sign would be absent in cases where the hypertrophy was more in connection with the proximal sphincteric loop and was not seen in the two cases under review.

Summary

1. Two cases of primary pyloric hypertrophy in adults are described.

2. An attempt is made to correlate both the infantile and adult pyloric hypertrophy on the basis of a functional imbalance between the circular and longitudinal musculatures of the pyloric canal.

3. The anatomical structure of the pyloric canal is described.

4. The radiological signs helpful in diagnosis are discussed.

Acknowledgements

Acknowledgement is made to Dr. K. P. Sen Gupta of the Pathology Department of S. S. K. M. Hospital for the histopathology examination and report.

REFERENCES

1. Atkinson, M., Edwards, D. A. W., Honour, A. J. and Rowlands, E. N.: Comparison of cardiac and pyloric sphincters, Lancet, Lond., 2, 918, 1957.
2. Belding, H. H. and Kerwohan, J. W.: A morphologic study of the myenteric plexuses and musculature of the pylorus with special reference to the changes in hypertrophic pyloric stenosis. Surg. Gynaec. Obst. 97, 322, 1953.
3. Bockus, H. L.: Gastro-enterology; Esophagus and Stomach, Vol. 1, Philadelphia and London, W. B. Sunders Co., 1946.
4. Cole, L. G.: Physiology of the pylorus, fillens ventriculi and duodenum as observed roentgenographically. J. Am. M. Ass., 61, 762, 1913.
5. Correa Netto, A.: Achalasie du pylore chez L'adulte. Presse Med., 1, 148, 1940.
6. Cruveilhier, 1842, quoted by Mack, H. C.: Bult. Hist. Med. 12, 465, 1942.
7. Greenfield, H.: Pyloric hypertrophy and stenosis in adults. Ann. Int. M., 34, 492, 1951.
8. Horton, B. F.: Pyloric muscle with special reference to the pyloric bloc. Am. J. Anat., 41, 197, 1928.
9. Keet, A. D. Jr.: Focal hypertrophy of pyloric wing muscle in adults. Arch. Path. Chic., 61, 20, 1956.
10. Kirklin, B. R. and Harris, M. T.: Hypertrophy of the pyloric muscle in adults; a distinctive roentgenologic sign. An. J. Roentg., 29, 437, 1933.
11. Mc. Cann, J. C. and Dean, M. S.: Hypertrophy of pyloric muscle in adults; experiences with conservative and medical surgical treatment. Surg. Gynaec. Obst. 90, 535, 1950.
12. North, J. P. and Johnson, J. H.: Pyloric hypertrophy in adult. Ann. Surg., 131, 316, 1950.
13. Raffensperger, E. C.: Time required for the development of pyloric muscle hypertrophy in an adult. Gastroenterology, 28, 458, 1955.
14. Raja, A. Curti., P. DeAlmida, A. C. and Fry, W.: The pathogenesis of hypertrophic stenosis of the pylorus in the new born and the adult. Surg. Gynaec. Obst. 102, 705, 1956.
15. Skoryna, S. C., Dolan, H. S. and Gley, A.: Development of primary pyloric hypertrophy in adults in relation to the structure and function of the pyloric canal. Surg. Gynaec. Obst. 108, 83, 1959.
16. Wallensten, S.: Pyloric hypertrophy in adults. Acta Chir. Scand. 104, 285, 1952.

—Received for publication on 10-9-1960.

OBSERVATIONS ON CLINICAL APPLICATION OF ARGINASE INHIBITOR IN URAEMIA

BY

DHRUBA KUMAR SEN, ASIT KUMAR MAITRA and

ANANTAPRASAD SINHA, Calcutta.

Introduction

Much controversy and confusion still exist regarding the development of toxic symptoms and manifestations in uraemic cases. In all cases of uraemia, be it of a pre-renal, renal or of a post-renal type, urea is retained in the body primarily due to renal insufficiency. The treatment of uraemia so far was directed towards elimination of urea from the body by methods involving intestinal and peritoneal dialysis, artificial kidney, and by excluding the intake of exogenous, protein (Bull's regime). Synthesis of urea in vivo takes place in the liver mainly and the kidneys are responsible for an effective elimination from the body (Mann et al 1924). That it is possible to stop urea synthesis in the body was first pointed out by Sen (1959). Intravenous administration of L-lysine HCl, the strongest arginase inhibitor, increases the survival time in bilaterally nephrectomised dogs. It is also postulated that in uraemic cases, if the synthesis of urea can be minimised or totally stopped in the body, the retained urea of the blood will be gradually eliminated *via* the kidneys, though at a slower rate, with simultaneous marked improvement in the clinical symptoms of the patient. Results of clinical trial on three human uraemic cases were reported by Sen (1959). All the three cases of that series recovered completely following administration of L-lysine HCl. The series of cases now being presented in this paper, having received treatment for uraemia with l-lysine HCl, show encouraging results and thereby lend further confirmation to the observations first elicited by Sen (l. c.) on experimental dogs.

Methods

All the patients now being presented, were admitted to the Surgical Ward of the N. R. S. Medical College and Hospital, Calcutta, for various surgical conditions.

Selection of cases for treatment was made at random. During the treatment with arginase inhibitor, the patients received a completely protein-free diet and Glucose solution intravenously calculated to supply thirty calories per kilogram of body weight of the patient. In milder cases, however, Glucose solution was not injected intravenously, but the patient was put on a completely protein-free diet in the form of boiled rice, sugar, whey fruit juice, fresh fruit etc. L-lysine HCl was injected intravenously 1 Gm. in 10% solution once in every 24 hours. No other specific therapeutic measures were adopted in order that the action of L-lysine HCl may be assessed without any bias.

Case Reports

Case 1.– S. B., a woman of 65, was admitted on 4-8-59, with transitional cell carcinoma of the urethra. She was operated on four days later and local excision of the growth was done. Post-operative recovery was uneventful, on 24-8-59, however, she became slightly drowsy and apathetic. Estimation of blood urea on that day revealed a figure of 124 mg%. The patient was put on a protein-free diet and Glucose drinks. One Gm. of L-lysine HCl was injected intravenously daily. The total urinary output during the next 24 hours was 1200 c.c. The blood urea fell down to 84 mg% after 24 hours with marked improvement in the clinical state. Treatment with L-lysine HCl was continued for a further period of three days, at the close of which the blood urea was 78 mg%. The patient got discharged on risk-bond and further treatment could not be continued.

Case 2.—S. P. D., a man of 75 was admitted on 18-8-59, with acute retention of urine due to benign hypertrophy of the prostate. The bladder was drained by means of a suprapubic catheter. Clinically, the patient was not uraemic and estimation of blood urea on 24-8-59, revealed a figure of 74 mg%. The patient was put on a protein-free diet and Glucose drinks. He was given an injection of L-lysine HCl intravenously and during the next 24 hours the blood urea came down to 28 mg%. Treatment was discontinued. Subsequent recovery of the patient was uneventful.

Case 3.—P. M., a man of 60, was admitted on 2-7-59, with an old un-united fracture of the neck of the right femur. He was operated upon on 11-7-59; an osteotomy was done and a Whitman's plaster spica applied. Four days after the operation, the patient showed early signs of uraemic manifestations. Blood urea estimation revealed a urea level of 54 mgm%. On 20-7-59, L-lysine HCl was started intravenously, the dose being 1 gm. in 10% solution, every 24 hours. 600 ml. of 10% Glucose solution were given by drip at the rate of 30 drops per minute and the patient was kept on a protein free diet. He received, on an average, 2500-3000 ml. of 25% Glucose solution intravenously daily. On 21-7-59, the blood urea came down from 54 mg% to 30 mg% and on 23-7-59, it was estimated at 25 mg. % with simultaneous improvement in the clinical state of the patient. The patient received only 3 gm. of L-lysine HCl in all.

Case 4.—B, R., a man of 44, was admitted on 16-7-59, with 'acute abdomen'. He was operated on the very day of admission and a subdiaphragmatic abscess was drained. The post-operative period was rather stormy as the patient developed effusion in the right pleural cavity. On 26-7-59, he became confused and uraemic. Blood urea was estimated at 60 mg% on 27-7-59. 1 gm. of L-lysine HCl was injected intravenously on the morning of 29-7-59, and the patient received protein-free diet and Glucose solution by means of a Ryle's tube. Blood urea estimation on 30-7-59 revealed a figure of 30 mg%. Urinary output during the previous 24 hours was 900 ml. In this particular case a drop in the blood urea level by 30 mg% was recorded after giving only one injection of L-lysine HCl, though there was no marked improvement in the clinical state of the patient due to the pleural condition, he did come back to full consciousness.

Case 5.—B. D., a woman of 60 was admitted with Papillary Carcinoma of the bladder. Uretero-colic anastomosis on the left side was performed on 22-8-59. Following the operation the patient gradually became drowsy and was in a semi-comatose condition on 31-8-59, when the blood urea was estimated at 92 mg%. The patient was put on a 25% Glucose drip, and a completely protein-free diet. L-lysine HCl was injected intravenously in doses of 1 gm. every 24 hours. Estimation of the blood urea on 1-9-59, revealed a figure of 90 and remained at that level on 2-9-59 also. The total amount of approximate urinary output was between 300–400 ml. each day. The patient expired on 3-9-59, in a comatose condition.

Case 6.—B. M. R., a man aged 65, was admitted with subacute intestinal obstruction (Carcinoma Caecum). Clinically, he was not uraemic, but his blood urea level estimated on 28-9-59 was at 82 mg%. He was put on a protein-free diet and a Glucose drip. The total quantity of urine passed during the previous 24 hours was 520 ml. 1 gm. L-lysine HCl was injected intravenously daily for 2 days and on 30-9-59, the blood urea was recorded at 54 mg%. The total quantity of urine passed gradually increased to 800 ml. per 24 hours during the period of treatment. The patient was operated upon on 30-9-59, and an Ileo-transverse anastomosis was done.

Case 7.—R. B., a man of 80 was admitted with Benign enlargement of the prostate, his only symptom was frequency of micturition. On 30-10-59, his blood urea was estimated at 72 mg%. He received 1 gm. L-lysine HCl intravenously daily from 31-10-59, a protein-free diet and on an average he passed 900 ml. of urine every 24 hours. Clinically, he was not uraemic. He received five intravenous injections of L-lysine HCl and over these five days of treatment, his blood urea came down gradually from 72 mg% to 44 mg%, well within the safe limit to undergo an operation.

Case 8.—B. R. C., a man of 55 was admitted with benign enlargement of the prostate with the symptom of frequency of micturition. His pre-operative blood urea level, estimated on 2-11-59, was 70 mg%. He received a protein-free diet, Glucose drink and 1 gm. L-lysine HCl intravenously on 2-11-59. During the first 24 hours of treatment he had passed 1000 ml. of

urine and the blood urea level was estimated at 44 mg%. During the next 24 hours it came down to 32 mg%. Treatment was discontinued at this stage as the patient was well within the range of safety for surgery.

Case 9.—J. K. P., a man of 32 with stricture of the urethra was operated upon on 7-11-59, when the bladder was drained by suprapubic tube and excision of the stricture with reconstruction of the urethra was done. Three days later, the total urinary output fell to 390 ml. and the patient became uraemic. Blood urea estimated on 10-11-59 revealed a figure of 100 mg%. He was immediately put on an intravenous Glucose drip (25%) and 1 gm. of L-lysine HCl was administered intravenously on two successive days. During the period of treatment the total quantity of urinary output rose from 390 ml. to 550 ml. An estimation of blood urea on 12-11-59, recorded a figure of 30 mg%. The general condition of the patient markedly improved with the fall in the blood urea level.

Case 10.—S. C. S., a man aged 65 years was admitted on 14-11-59, with painless haematuria and with an irregular firm lump in the left loin. Clinical diagnosis of hypernephroma of the left kidney was made. He was extremely anaemic (Hb 40%) and received 280 ml. of blood on 15-11-59. Following the transfusion he became comatose, vomited once, and passed 300 ml. of urine over the next 24 hours. His blood urea on 16-11-59, was 94 mg%. 25% Glucose drip was immediately started intravenously and 1 gm. of L-lysine HCl was injected intravenously daily. Over the next three days, though his daily total urinary output rose from 300 to 790 ml., his blood urea remained at 96 mg% Injections of L-lysine HCl were continued for two more days and on 21-11-59, the blood urea came down to 56 mg%. His total daily urinary output reached 900 ml. The patient was conscious at the end of the treatment and recovery was uneventful.

Case 11.—S. G., a man of 68 was admitted on 3-12-59 with frequency of micturition due to benign hypertrophy of the prostate. Clinically, the patient was not uraemic, and his pre-operative blood urea level estimated on 5-12-59, was 78 mg%. He was put on a protein-free diet, and 1 gm. of L-lysine HCl was injected intravenously daily from 8-12-59. He received three successive injections of L-lysine HCl and on 11-12-59, his blood urea was recorded at 30 mg%. The total quantity of urine passed by the patient on an average was between 800-900 ml. per day.

Case 12.—K. K. P., a man of 60 was admitted on 12-12-59, with acute abdomen. He was operated on the same day and at operation the peritoneal cavity was found to be flooded with bilious material. A perforated, gangrenous gall bladder was removed and the peritoneal cavity was drained. The patient was slightly incoherent in his speech from the same evening and the suction material from the stomach contained altered blood. Over the next couple of days his general condition deteriorated and on 15-12-59 his blood urea was estimated at 120 mg%. The patient was kept on a Glucose drip and injections of 1 gm. of L-lysine HCl were given intravenously daily. His urinary output was well within normal limits. On 17-12-59, his blood urea came down to 114, on the 18th to 88 mg% but it rose again to 108 mg% on the 19th. L-lysine HCl injection was continued and on 22-12-59, the blood urea was recorded at 98 mg%. All these days, altered blood was aspirated through the gastric tube, though the patient's systolic blood pressure did not fall below 100 mm. of Hg. From 23-12-59, the patient's condition gradually deteriorated and he expired in a state of deep coma on 28-12-59, at a blood urea level of 124 mg%.

Case 13.—K. N. P., a man of 45 with appendicular perforation and early peritonitis was admitted on 27-2-60. He was operated on the same day, and at operation a gangrenous appendix with perforation at its tip was removed. The patient did not show any clinical manifestations of uraemia. His blood urea estimated on 5-3-60, was 72 mg%. The urinary output was within normal limits. He was put on a protein-free diet and received 1 gm. of L-lysine HCl intravenously for three successive days. The blood urea came down to 28 mg% on 8-3-60, when the treatment was discontinued. Recovery was uneventful.

Case 14.—K. B. D., a woman aged 65 was admitted with intertrochanteric fracture of the left femur on 25-2-60. The patient was put on skin traction three days later. After about two weeks, i.e., on 12-3-60, the patient became slightly drowsy and apathetic. Her blood urea estimated on the same day was 104 mg%. She was put on a protein-free diet and Glucose drinks and received 1 gm. of L-lysine HCl intravenously daily. Her daily urinary output was between 780 and 950 ml. On 15-3-60, the blood urea was reduced to 78 mg% and the injections of L-lysine HCl were continued for three more days. After three days the blood urea was recorded at 52 mg%. The symptoms of uraemic manifestations passed off completely at this stage when

treatment was discontinued. It is to be noted that the patient received 7 gm. L-lysine HCl over a week and the fall in the blood urea level, though steady was rather slow.

Case 15.—C. S., a man aged 60 had undergone one stage prostatectomy with primary closure of the bladder on 12-3-60. On the third post-operative day he suddenly became incoherent in speech and drowsy. The total quantity of urine passed by the patient on the third post-operative day was 1980 ml. but his blood urea level was recorded at 68 mg%. The patient received a protein-free diet and Glucose drinks as usual and 1 gm. of L-lysine HCl was injected intravenously for three successive days. On the third day following administration of L-lysine HCl his mental symptoms disappeared completely and the blood urea came down to 50 mg%. Recovery was uneventful.

Case 16.—S. C. B., a man of 48 was admitted on 23-1-60 with retention of urine due to impassable stricture of the urethra. On admission he was grossly uraemic with mental confusion and incoherent talk and his general condition was extremely low. Suprapubic cystostomy was done. Following the operation no improvement in the mental state was noted and on the day after the operation the patient passed 870 ml. of urine. On the next day (23-1-60) the urine volume was reduced to 300 ml. and the blood urea was recorded at 94 mg%. 25% Glucose drip and a protein-free diet were instituted and 1 gm. L-lysine HCl was injected intravenously. The urinary volume increased to 500 ml. in the next 24 hours and over a period of another 24 hours the volume rose to 1300 ml. Blood urea came down to 68 mg% on 28-1-60 and 47 mg% on 29-1-60. With the drop in the blood urea level to within normal limits, the patient's general condition improved and the uraemic state passed away completely.

Case 17.—C. D. D., a man of 40 was operated upon on 16-8-59, for a huge amoebic liver abscess. Following operation, from the same evening, the patient became grossly uraemic, remained in a semi-comatose state and gastric suction revealed presence of altered blood in fair amounts. The total quantity of urinary output on the day of operation was only 200 ml. Blood urea estimated on 17-8-59, was 116 mg%. Continuous gastric suction was carried out, 25% Glucose drip was started intravenously and 1 gm. L-lysine HCl was injected intravenously. The urinary volume did not increase and the patient expired the next day, i.e. on 18-8-59. Blood sample collected immediately before his death had an urea level of 138 mg%. The patient died in a state of coma.

It is interesting to note that there was a significant rise in the blood urea level in spite of injections of L-lysine HCl, the interpretation of which be discussed later.

Case 18.—H. L., a man of 58 was admitted on the evening of 9-4-60, with intestinal obstruction. He was operated on the next morning (10-4-60) and at operation a solitary stricture in the lower jejunal loop was detected. Obstruction was relieved by short circuiting the stricture. The patient's condition gradually improved following the operation, but on 15-4-60, he developed acute abdominal pain with rigidity. A diagnosis of rupture of the anastomotic stoma was made and the abdomen was re-explored. At the second operation, the peritoneal cavity was found to be flooded with small intestinal contents and a round worm about 15 cm. long was seen peeping through the site of the anastomosis which was obviously the cause of the mischief. The round worm was removed and the opening at the anastomotic site was repaired and reinforced by a patch of omentum. The patient's general condition, however, did not improve and he sank gradually due to peritonitis. During the whole post-operative period he received broad spectrum antibiotics. On 19-4-60, his blood urea was estimated at 24 mg% but on the 24th he passed into a state of coma and blood urea estimation on 25-4-60, revealed a figure of 238 mg%. The patient was in a moribund state and was gasping. The total quantity of urine passed during the last 24 hours was 400 ml. approximately. The patient was put on 25% Glucose drip and 1 gm. of L-lysine HCl was injected intravenously. The patient expired two hours after the injection. Blood sample collected a few minutes before his death showed a urea level of 224 mg%. It will be noted here that the blood urea level dropped by a 14 mg% over a short period of 2 hours only following the injection of L-lysine HCl, 1 gm. intravenously.

Case 19.—M. C., a woman of 21 was admitted on 21-4-60, with a huge Colloid goitre. Routine examination of blood urea on 23-4-60, revealed a figure of 58 mg%. The patient was put on a pure carbohydrate diet, to meet the daily calorie needs (at 30 calories per kilo of body weight) and one injection of L-lysine HCl was given on the same day. Blood urea level came down from 58 mg% to 40 mg% after only one injection of L-lysine HCl.

Case 20.—M. A., a woman of 68 was admitted with an old fracture of the neck of the femur for which an osteotomy was done on 7-5-60, and a Whitman's plaster spica applied. Three days later, *i.e.* 10-5-60, the patient became drowsy and delirious. The total quantity of urine passed daily came down to 260 ml. Blood urea estimated on 10-5-60, was 88 mg%. The patient was put on a Glucose drip and injection of L-lysine HCl 1 gm. was given intravenously daily for two days. On 12-5-60, her blood urea level was 46 mg%; one more L-lysine HCl was injected on that date and on the following day the blood urea level was estimated at 42 mg%. The general condition of the patient improved remarkably after the first injection and 24 hours after starting the treatment the patient passed about 800 ml. of urine. Subsequently, the urinary volume was within normal limits.

Discussion

The intricate problem of the "uraemic state" is worth studying, as retention of urea in the body alone may not be responsible for the production of uraemic manifestations. At the same time the authors would like to emphasize that retention of urea in the body does play a great role in the production of toxic manifestations. It has been pointed out by Shackman (1959) that in the state of uraemia, even when the patient is put on pure carbohydrates (in the form of 40% Dextrose by intravenous drip) in order to supply sufficient calorie needs and to avoid endogenous protein breakdown, there is a daily increase of blood urea by 20-30 mg%, serum potassium by ·5 mEq/l and an average daily fall of serum bicarbonate by 1·5 mEq/l, which would obviously denote the degree of acidosis consequent on phosphate and sulphate retention. He further pointed out that in cases complicated by fever or intestinal bleeding with absorption of blood protein from the bowel, or whenever there is endogenous protein breakdown following severe tissue trauma (*e.g.* major surgical operation or severe lacerated injury—the "katabolic phase"), the daily blood urea may rise by 80 mg%, serum potassium by 1·5 mEq/l and the fall of serum bicarbonate may be as great as 3 mEq/l. From the above data the cause of production of acidosis in uraemic subjects is obvious, and it also becomes clear how the conservative regime for the treatment of acute renal failure proves insufficient. Moreover, studies on extracellular potassium concentration show that a rise to 7 mEq/l or more may be responsible for the development of arrythmias and sudden death in uraemia (Shackman l. c.).

So far the treatment of uraemia has been directed entirely towards elimination of urea and correction of electrolytic imbalance by various methods such as peritoneal and intestinal dialysis and artificial kidney.

Sen (1959) attacked this baffling problem from an entirely different angle. Taking for granted that urea synthesis in the liver could be stopped by use of l-lysine monohydrochloride, the most potent arginase-inhibitor, he succeeded in prolonging the life of bilaterally nephrectomised dogs. The observation was not only thrilling, it was satisfying indeed being beset with promises of hope for the uraemic subjects of the future. The fact that the lives of bilaterally nephrectomised dogs can be prolonged by keeping urea synthesis at the minimum, goes to prove indirectly that in uraemic subjects the unchecked synthesis of urea, coupled with its retention due to kidney failure, is the crux of the problem in treatment.

At the outset, it must be emphasized that treatment of uraemic cases with arginase-inhibitor does not claim to eliminate urea from the body in any way, but it only aims at stopping urea synthesis in the body. Thus,the prognosis of uraemic cases treated by arginase-inhibitor will depend upon the ultimate renal functional integrity. Assessment of the value of this form of treatment is therefore naturally dependent on the recoverability or otherwise of the underlying renal pathology.

In the present series of experiments on 20 human subjects treated only by intravenous injections of 1 gm. of l-lysine HCl in 10% solution daily, 16 of them recovered while 4 patients died. While the cause of recovery is obvious, the cause of death for the 4 patients (cases 5, 12, 17 and 18) needs clarification. In case 5, following ureterocolic anastomosis on the left side only, the patient gradually became uraemic and her blood urea was 92 mg%. After the first injection of l-lysine HCl it remained at 90 mg%, a fall in the urea percentage too insignificant to be taken into account and on the following day it still remained at 90 mg%. The total quantity of urine passed by the patient was approximately 300-400 ml. daily. This case goes to show that l-lysine HCl succeeded in minimising urea synthesis in the liver, though it could not probably stop complete synthesis of urea in the body. It is obvious that the daily total urinary excretion was not sufficient enough to eliminate the retained urea in the blood, which remained more or less steady at the level of 90 mg%. Ammonia intoxication (McDermott et al 1954) was probably the cause of death in this particular case.

In case 12, where the patient had a perforated gall bladder with bilious peritonitis, blood urea level was 120 mg%. The patient was bleeding from the gastric mucosal surface and the suction material from the stomach contained altered blood. With injections of l-lysine HCl, it gradually came down to 88 mg%, to go up again to 108 mg% on the third day. After three days it came down to 98 mg% again, but on the next day the patient expired at a blood urea level of 124 mg%. The cause of death in this particular case is easy to explain. As the patient was having gastro-intestinal haemorrhage, an effect more or less similar to giving the patient a high-protein diet was produced, with the production of ammonia intoxication (McDermott & Adams, 1954).

In case 17, a case of amoebic liver abscess, the blood urea was estimated to be 116 mg% at the time the patient became comatose. Altered blood was continuously being aspirated from the stomach following the operation. The patient received only 1 gm. of l-lysine HCl and he expired 18 hours after the injection in a state of coma. Blood sample collected immediately before his death revealed a urea percentage of 138 mg. The cause for this rise is obvious. The presence of altered blood in the gastro-intestinal tract acted as a protein meal and the patient passed only a very small quantity of urine daily (200 ml. approx.), a volume which could not eliminate urea from the body efficiently. The cause of death was probably ammonia intoxication as a result of breakdown of the ingested blood by intestinal micro-organisms.

In case 18, the patient developed a severe degree of peritonitis following rupture at the anastomotic site caused by a round worm. The patient was given only one injection of l-lysine HCl at a blood urea level of 238 mg%. The patient expired after two hours and a sample of blood collected immediately before his death showed a urea level of 224 mg%. The fall of blood urea level by 14 mg% was naturally due to inhibition of further urea synthesis in the body and simultaneous excretion of the same *via* the kidney. In this case, death was probably due to a combination of factors,—severe peritonitis and surgical trauma leading to endogenous protein breakdown, in association with high blood urea.

In rest of the cases, the urea level was successfully brought down from the original high level after use of l-lysine HCl. The authors have used 25% Glucose instead of 40% solution for transfusion since in a tropical country like ours, water is greatly eliminated *via* sweat and respiration.

The cases for treatment with l-lysine HCl were selected at random and whoever had the slightest chances of recovery was given a full trial. Lastly, it may also be mentioned that no side effects were noticed in any one of our cases following l-lysine HCl administration.

Acknowledgement

The authors express their grateful thanks to Dr. R. N. Guha Mazumdar-Principal-Superintendent, N. R. S. Medical College Hospital, Calcutta, through whose courtesy the case reports are presented. The authors are also grateful to Dr. S. K. Chatterji, F.R.C.S., Hony. Senior Visiting Surgeon, for allowing treatment with l-lysine HCl to be carried out on some of his patients included in this paper.

REFERENCES

1. Mann et al : Amer. J. Physiol. 69 : 382, 1924.
2. McDermott, W. V., Adams, R. D. and Riddell, A. G. : Ann. Surg. 140 : 539, 1954.
3. McDermott, W. V. and Adams, R. D. : J. Clin. Invest. 33 : 1, 1954.
4. Sen, D. K. : Ind. J. Surg. 21: 514, 1959.
5. Sen, D. K. : Nature, 184 : 159, 1959.
6. Shackman, R. : Recent, Adv. in Surg. (J. & A. Churchill Ltd.), 166, 1959.

—Received for publication on 10-9-1960.

CHRONIC DUODENAL ILEUS

BY

R. S. GUPTA and Miss RAMA ROY, Calcutta.

Chronic duodenal ileus was first described by Finney in 1906. In 1921 Wilkie gave a masterly description of this condition, which is characterised by recurrent dilatation of the duodenum without any anatomical or pathological cause. The superior mesenteric artery is drawn taut by a pendulous small intestine and the duodenum is compressed in the acute angle between thin vessel and the aorta. Louw reviewing 54 cases with duodenal ileus from 1953 to 1958 believes that malrotation of the intestine is the major cause of duodenal ileus. Neuromuscular incoordination may explain those cases of duodenal ileus where the dilatation extends only to the level of the ampulla of Vater or where it is continued beyond the superior mesenteric artery to the duodenojejunal flexure. For chronic duodenal ileus due to compression by the superior mesenteric artery, or mesenteric ileus Wilkie had stressed a (1) long history of dyspepsia and bilious complaints dating from childhood which might be relieved by position ; (2) occasional acute colicky pain and vomiting ; (3) occasional large pale offensive stools ; (4) associated duodenal ulcer ; (5) roentgenological demonstration of a grossly dilated duodenum ending abruptly in the region of the superior mesenteric artery. Chronic duodenal ileus is an extremely rare disease to-day.

Case Report

A female patient aged 20 years was admitted on 10-7-60 with complaints of pain in the abdomen for the past one year, associated with vomiting off and on. The patient was well till year ago when she had acute 'dysentery' for which she had to be hospitalised. She lost about 20 lbs. of weight during this attack. Following this she started getting pain in the epigastric region, especially after meals, which was relieved by vomiting. The pain became more or less constant, aggravated by food and radiated to the back. The vomits contained bile and relieved the pain to a certain extent.

On examination, a thinly built female, obviously dehydrated and anaemic was seen. There was evidence of the patient having lost weight. On inspection visible peristalsis could be seen in the epigastrium going from left to right. There was tenderness on deep palpation just to the right of the umbilicus. A vaginal examination showed a nulliparous uterus with culs clear. Heart NAD, Respiratory system - NAD ; urine - NAD ; Temp. 98·2°F, Pulse 100 p.m., B. P. 106/80, Hb.%—60%, WBC 5800 ; E. S. R. 10 m.m. 1st hr. ; stool - normal ; X-ray chest - Normal. F. T. M. showed achlorhydria. Weight 68 lbs. A barium meal examination showed a normal stomach, with obstruction in the 3rd part of duodenum in the region of superior mesenteric vessels with dilatation. (Fig. 1) Very little barium trickled through with a substantial hold up of barium after 6 hours.

The patient was prepared for operation by stomach washes and I. V. fluids and her electrolyte balance was corrected. On 22-7-60 a laparotomy was done

Fig. 1

A Barium Meal examination shows obstruction in 3rd part of duodenum in the region of superior mesenteric vessels.

Fig. 2

A Barium Meal one month after operation shows duodeno-jejunostomy working satisfactorily.

by a right paramedian incision. On opening the peritoneum most of the small bowel was found lying in the pelvis and the mesentery of the small bowel was seen to be taut. The small bowel was delivered into the abdominal cavity from the the pelvis and examined. The duodeno-jejunal junction was normal as was the small and large bowel. The right colon was then mobilised by incising the peritoneum on its right side. The attachment of the mesentery of the small bowel to the posterior parieties was incised and the mesentery together with the superior mesenteric vessels was lifted upwards towards the left by the manoeuvre described by Cattel. An excellent view of the 3rd & 4th parts of the duodenum was thus obtained. No tumour or any other lesion could be palpated in the third part of the duodenum. The wall of the duodenum was smooth and no constriction could be made out on its surface. There was distension of the whole of the 1st, 2nd & 3rd parts of the duodenum. After a thorough exploration revealing that no obvious pathological lesion was present in the duodenum, the mesentery of the small bowel was replaced. No attempt was made to resuture the right half of the colon to its normal attachments. The transverse colon and omentum were lifted upwards. The first loop of jejunum was then brought up in front of the superior mesenteric vessels. A 2" horizontal incision was made in the peritoneum between the middle colic and right colic vessels, exposing the third part of the duodenum, taking care not to injure the vessels. The loop of the jejunum was then laid against this exposed part of the duodenum after approximating the peritoneum of the jejunal mesentery and the peritoneum of the posterior abdominal wall, so as to close off the peritoneal tunnel created by the anastomosis. Interrupted '00' silk stitches through sero-muscular coat between the jejunum and duodenum were then applied. A through and through all coats continuous '00' chromic catgut anastomosis was then carried out. The anastomosis was completed by applying an anterior row of interrupted '00' silk stitches. No intestinal clamps were used during the anastomosis. The abdomen was closed without drainage.

•

The post operative course was uneventful. Stitches were removed on the 8th post-operative day.

Follow up

A barium meal examination was done 3 months after operation and is shown in (Fig. 2). The patient is symptomless and has put on 22 lbs. of weight.

Summary

1. A case of chronic duodenal ileus - mesenteric ileus as seen in a 20 year old female is presented.

2. The aetiology of this condition is briefly discussed.

3. A duodeno-jejunostomy between the 3rd part of the duodenum and the first proximal loop of jejunum was carried out and the operative procedure is described in some detail.

4. A follow up at 3 months shows an excellent result.

REFERENCES

1. Aird, I. : A companion in Surgical Studies, 2nd Edition, 1957, E. & S. Livingstone Ltd., Edin. & London.
2. Cattel, R. B. and Brassch, J. W.: A technique for the exposure of the third and fourth portions of the duodenum, Surg., Gynaec. Obst. 3, 379, 1960.
3. Dwight, J. : Anat. Physiol., 31, 516, 1897.
4. Finney : Bull. John Hopkins Hosp., 17, 37, 1906.
5. Fischer, H. W.: The Big Duodenum. Am. J. Roentg., 83, 861, 1960.
6. Glover, D. M. and Barry, F. McA.: Ann. Surg. 130, 480, 1949.
7. Gross, R. E. : Surgery of Infancy and Childhood 1953, Saunders W. B., Philadelphia.
8. Johnson, H. D. : Operative Surgery, Progress, Vol. 1958, Butterworth & Co. Ltd., Lon.
9. Louw, J. H. : Intestinal malrotation and Duodenal ileus, J. R. Coll. Surgeons, Edinburgh, 5, 101, 1960.
10. Wilkie. : Brit. J. Surg., 9, 204, 1921.

—Received for publication on 5-11-1960.

—★—

SERUM POTASSIUM IN SURGICAL STRESS

BY

A. K. BANERJEE, Vellore and B. N. BALKRISHNA RAO, Gwalior.

Introduction

Selye says that anything that causes stress endangers life unless it is met by adequate adaptive responses; conversely anything that endangers life causes stress and adaptive responses. Adaptibility and resistance to stress are fundamental requisites for life and every vital organ and function participates in them.

The effect of stress may be mild or severe, may develop quickly or gradually and may be short lasting or prolonged depending upon the type of stress.

There are series of changes occurring in the blood of a person undergoing an operation.

Many workers have estimated serum electrolytes in persons under various types of stress (*e.g.*) pregnancy, traumatic injuries, ether anaesthesia, artificial fractures etc.

Berry, Hob and Campbell (1948) noticed an increase of the urinary excretion of potassium during the post-operative phase and this was confirmed by Randall, Habit, Dickwood and Werner (1949); Blixenkrone-Moller (1949); Marks (1950); Wilkins, Billing, Bagy and Stewart (1950); Mc Innes, Bodansky, Brunswig (1950) and Cole (1950) Reinberg and Stolkwoski (1953) found increased potassium in plasma and extracellular fluids during stress. Eliel, Pearson and Lawson (1950) studied 32 post-operative cases and they observed that urinary excretion of potassium was maximum on the first 24 hours, gradually diminishing from the 4th to the 7th day. They attribute this to hyperadrenal activity due to surgical trauma.

MacPhee (1953) observed that serum potassium concentration appeared to be variable and showed no pronounced change. In general, the concentration of potassium in the serum was slightly lowered during some part of the post-operative period.

Terail and Elkinton (1949) have demonstrated that in both normal and potassium depleted patients the kidney has a limited minimal excretion of potassium and below the minimal level the excretion does not fall. They believe that continued loss even in the presence of cellular potassium deficiency is an important factor in producing further depletion. The incidence of surgical operation increases further loss.

Manzanilla et al (1953) found that calcium and potassium tend to go down during surgical stress while phosphorus tends to rise.

Evans (1950) has remarked that after accidental or surgical trauma, sympatho-adrenal discharge (Selye's alarm reaction) may result in extraordinary loss of potassium by the urinary route. Continuous gastric suction for 4 or 5 days without potassium replacement may lead to potassium deficiency.

Methods and Material

The patients were taken from the male and female surgical wards of the J. A. Group of Hospitals, Gwalior. No particular disease, sex or age group was selected. Pre-operative blood was taken from each patient under study to establish the normal base line of that person.

In the first fifty cases - five samples of blood were taken, first - 24 hours before operation, second and third - before and after the operation, fourth - after 48 hours and fifth - after 7 days.

In the second fifty cases - the same number of blood samples were taken but time interval was different. The first three samples were taken at the same time as in the first fifty cases. The fourth sample was taken 24 hours after operation and the fifth sample on the fifth day after the operation.

Patients undergoing operations under spinal anaesthesia only were included in this study so as to avoid the chemical effect of a general anaesthetic. Blood was taken without applying any tourniquet or any massage as Farber et al have pointed out that plasma potassium level increases transiently by even slight exercise in veins draining the exercised muscles.

Potassium was estimated by the method described in E. J. King's book on Micro-analysis in Medical bio-chemistry.

Observations

In 72 cases there was a fall in potassium level during the operation. The maximum and minimum fall was found to be 4·5 and 0·1 mgm. respectively. A rise in the level during the observation was observed in 28 cases but most of them showed fall in the post-operative period. Maximum and minimum rise was 3·3 mgm. and 0·2 mgm. respectively.

A. *Age Group : 10-20 years.*—7 cases were studied in this age group. A rise in potassium level was seen in 2 cases and fall in 5 cases.

B. *Age Group : 20-30 years.*—30 cases were investigated in this age group. 23 cases showed a fall and 7 cases showed a rise in potassium level.

C. *Age Group : 30-40 years.*—30 cases were studied, 22 cases showed a fall and 8 cases showed rise.

D. *Age Group : 40-50 years.*—16 cases were studied. Potassium level fell in 12 cases and rose in 4 cases.

E. *Age Group : 50 years and above.*—12 cases were investigated in this group. 8 cases showed a fall and four cases a rise.

Potassium level in relation to duration of Operation

The duration of operation was taken from the time the spinal anaesthetic was effective to the end of operation. The duration of operation had been divided into three different groups (1) 0-30 minutes (2) 30-60 minutes and (3) 60 minutes and above.

1. *Duration of operation 0-30 minutes.*—Twenty six cases were studied in this group. Potassium showed a fall in 17 cases, rise in five cases and no change in four cases. The maximum and minimum fall was 3·4 and 0·3 mgm.

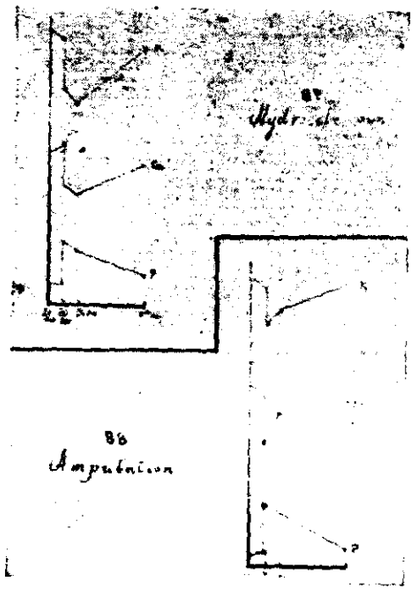

Fig. 1

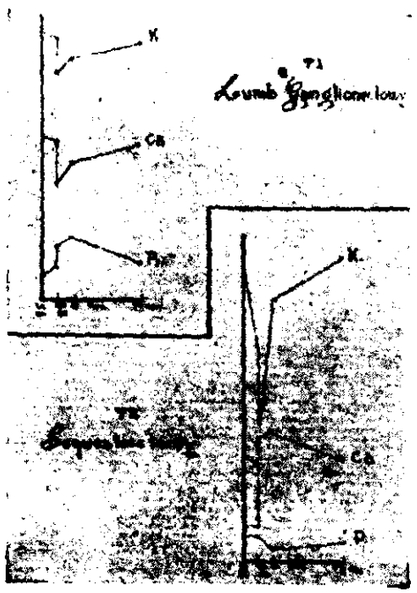

Fig. 2

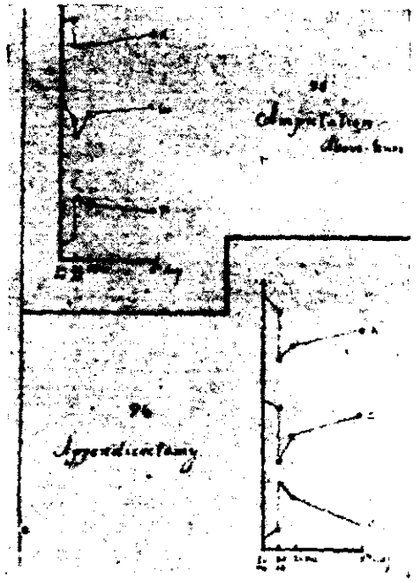

Fig. 3

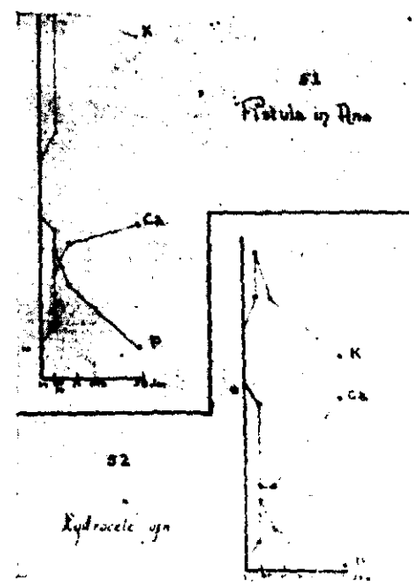

Fig. 4

Photographs 1 and 2 and 3 showing fall of Potassium during Operation.
Photograph 4 shows rise of potassium during Operation.

2. *Duration of operation: 30-60 minutes.*—Forty eight cases were investigated. Potassium level fell *in* thirty five cases, rose in twelve cases and no change *in one case.*

   The maximum *rise* was 3·0 mgm. and minimum 0·2 mgm. The maximum fall was 3·8 mgm. and *minimum* 0·2 mgm.

3. *Duration of operation: 60 minutes and above.*—Fall in potassium level in *thirteen* cases and rise in four cases.

   The *maximum* and minimum rise was 2·1 mgm. and 0·4 mgm. respectively.

   The maximum *and minimum* fall was 2·5 mgm. and 0·3 mgm. respectively.

Discussion

It has been observed *that* there is a *decrease of* potassium during and after the operation. In *some* cases it was observed that there was a rise and in *some* none. Post-operatively potassium level remains *low but comes* to normal on the fifth day onwards; it remained low in some cases *who had diarrhoea* or prolonged sepsis *in the post-operative period.*

This sustained decrease *in* potassium may *be explained by the stimulating* action of stress on the pituitary-adreno cortical and medullo-adrenal systems and *secondly, lack* of ingestion *of potassium* associated with *continued loss* in the vomits and urine *in* the post-operative period. Manzanilla *et al* (1953) also found a fall *in* potassium level and thought that it may be due to stimu*lation* of the pituitary adrenal *axis.*

Cuthbertson (1936) found increased excretion of phosphorus and potassium *in* urine after *injury.* He attributed this to katabolism of tissue, probably muscle. Howard et al (1946) do not agree with Cuthbertson. Blixenkrone-Moller (1949) found that the greatest amount of potassium was excreted in the first twenty four hours after operation. He attributed the loss of potassium chiefly to intra-cellular dehydration.

Randal et al (1949) have shown with balanced studies pre and post-operatively in surgical patients, that the potassium losses were two to three times higher than would be anticipated from nitrogen loss. They said, "therefore this extra potassium come from the cells". The loss occurred not only because of potassium deprivation in the presence of normal urinary excretion but because of removal of potassium by vomit*ing,* suction, drainage and diarrhoea. In addition to this, potassium excretion in urine occurs as a part *of the* alarm reaction that occurs *with disease* or operation.

Eliel et al (1949) observed fall of potassium, which gradually came up by the eighth day. This was accounted for by hyperadrenal activity. This bears a striking resemblance to the findings *in patients* with Cushing's syndrome. Eliel and *Pearson* (1951) induced hyperadreno-corticism by *injecting* ACTH or cortisone and *found* similar changes.

Patients maintained post-operatively on parenteral fluids, low *or lacking* in potassium may develop a syndrome characterized by apathy, lethargy, muscular weakness, abdominal distension and ileus, cardiac arrythmias *and oedema,* occasionally confusion, delirium, muscular twitching and tetany. Evidence was found in these patients of the existence of (1) Hypopotassaemia (2) Hypochloraemia (3) metabolic alkalosis (4) lowering of erythrocyte potassium and increase of erythrocyte sodium. Two factors

were thought to be of primary importance in the pathogenesis of potassium deficit.

1. Adreno-cortical hyperfunction resulting from trauma (Alarm reaction) leading to increase renal excretion of potassium.

2. A low potassium intake with poor renal conservation of potassium in the post-operative period.

Berry, Job and Campbell (1947) say that in the homeostatic readjustment required to meet the obligations placed on an organism by a stress, there is concurrent reactivation of at least two important mechanisms :—

1. The hypothalamico–neurohypophysial and
2. Adreno hypophysial—adrenocortical. The control of the former is located in the hypothalamus and the two systems are activated simultaneously. Evidence is present that the antidiuretic hormone has no effect on potassium excretion in reciprocal fashion to sodium. An unrecognised urinary potassium loss occurs during all operations and is part of the natural response to stress.

Hayes and Coller (1952) observed that there is a rapid and progressive activation of the pituitary adrenocortical axis in response to the stress of anaesthesia and surgical trauma. The endogenous release of pituitary adrenocorticotropic hormone evokes the production of adrenocortical steroids. The physiological response of the adrenal gland in stress produces a reciprocal effect on sodium and potassium excretion inhibiting the former and facilitating the latter, so confirming the observations that protein catabolism does not account entirely for the amount of potassium excreted in the immediate post-operative period.

Manzanilla et al (1953) found decrease in the potassium level after operations and explained it on the basis of the activation of the pituitary cortico adrenal system. Fasting, lack of ingestion of potassium, increased and continued excretion of potassium and possible resultant dehydration were thought to be additional factors. They found a definite increase in young persons ; a decrease or small increase in old people.

In the present series one hundred patients were investigated and it was found that there was decrease in the potassium level in 77 cases and rise in 23 cases. Even those who showed rise during operation, showed a fall in the post-operative period in most of the cases. There was no definite relation found with age groups. However, it was noticed that rise and fall in the age group of 10-20 years was very small in comparison to higher age groups. Berry et al (1948) mention that in older patients, potassium excretion may be less because of renal impairment. Variation in the levels in relation to duration of operation was also interesting. Very little changes were seen in the first 30 minutes. Peak changes were seen between 45 and 90 minutes.

Terail and Elkinton (1949) have demonstrated that in both normal and potassium depleted patients, the kidney has a limited minimal excretion of potassium and below that minimal level the excretion does not fall. If in addition to these other sources of potassium deficit, the incident of surgical operation increases potassium loss, cellular potassium is further depleted and the state of potassium deficiency is hastened.

Conclusions

1. Surgical stress causes fluctuation in the level of potassium which is mostly a fall in the level and sometimes a rise.
2. Potassium level remains low till about 5 days post-operatively.
3. Diarrhoea, vomiting, and sepsis in the post-operative period further lower the potassium level.
4. The variation is probably due to stimulation of pituitary cortico adrenal system due to stress.

Summary

A study of serum potassium in 100 patients who were operated is presented herewith.

REFERENCES

1. Berry, R. E. L., Job, V. & Campbell, K. N. : Arch. Surg. 57 : 470, 1947.
2. Blixenkrone-Moller : Acta Scandinavica, 97 : 301, 1949.
3. Cuthbertson, D. P. : Lancet, 2 : 433, 1942.
4. Eliel, L. P., Pearson, O. H. & Rawson, R. W. : New England J. Med. 243 : 471, 1950.
5. Evans, E. I. : An. of Surg. 131 : 945, 1950.
6. Elkinton, J. L. & Winkler, J. A. : J. Clin Invest. 23 : 93, 1954.
7. Gamble, J. L. : Chemical Anatomy, Physiology and Pathology of Extracellular fluid, Harv. University Press, 1953.
8. King, E. J. : Microanalysis of Medical Biochemistry, 1951.
9. Manzanilla et al. : J. Intern. Coll. Surg. 20 : 573, 1953.
10. Rene Fontanaine, Mendel, P. J. Dano. : Sem. Hop. Paris. Ann. Chir. 32 : 1045, 1956.
11. Selye, H. : Studies in Adaptation—Endocrinology, 21 : 169, 1937.

—Received for publication on 15-12-1960.

ORTHOPAEDIC SECTION

ARTHROPLASTY OF THE ELBOW JOINT

BY

A. D. DIAS, Bombay.

Ankylosis of the elbow can indeed be a very trying disability. This is more so when the joint is ankylosed in a bad position. Arthroplasty plays a large part in the successful rehabilitation of these elbows. This is the opinion of several surgeons like Giuntini[4], Knight[5], Van Zandt[6], Mercer[7], Sanchis-Olmos[9], Wilson[11], etc.

Aim

The aim of arthroplasty is to obtain both free movement and also a stable joint, since mobility alone will not materially improve the function of the limb.

Assessment of a Case

All ankylosed elbows are not suitable for arthroplasty. The following features may be considered useful in assessing a case.

(1) *Nature of the causative disease.*—Arthroplasty should only be performed on joints which are biologically sound *i.e.* joints that contain no traces of active or latent inflammation.

Tuberculous disease of the elbow readily heals under conservative therapy though a stiff and ankylosed joint is the ultimate outcome. The bone ends, however, are so often healthy that arthroplasty can be carried out with a fair prospect of success. Knight[5], however, feels that arthroplasty is contraindicated in tuberculosis.

When joint ankylosis has followed gonococcal or a pyogenic infection, about a year or so should preferably elapse before operation is undertaken, in order to be reasonably sure that all inflammation has subsided (Mercer[7]).

The results of arthroplasty for rheumatoid arthritis are on the whole poorer than in the other types of ankylosis because of the danger of recrudescence.

The best results are obtained when arthroplasty is done for traumatic ankylosis.

(2) *Condition of the muscles controlling the joint.*—If the flexors and extensors of the joint are free from toxic or disuse atrophy or scarring, then there is an excellent chance of a good and useful joint resulting from the operation.

(3) *Condition of the Skin.*—If there has been extensive destruction of the skin at the time of the original affection, the joint may be left with skin of poor quality. It would then be necessary to replace this skin by a pedicle flap graft prior to arthroplasty.

(4) *Whether the ankylosis is bilateral.*—Arthroplasty is likely to be most useful and best appreciated in these cases.

(5) *Position of the ankylosed joint and occupation of patient.*—If the ankylosis is in a good position, this would be quite compatible with the efficient functioning of the limb. Thus in an occupation requiring hard physical labour where strength and stability are more important than mobility arthroplasty should be avoided.

When the position of the ankylosis is bad or where the ankylosed joint is painful or becoming progressively deformed, then arthroplasty would be indicated.

(6) *Temperament of the patient.*—Arthroplasty needs considerable and prolonged post-operative physiotherapy. This can be quite painful especially in the early stages. Hence unless the patient is willing and likely to cooperate, it may not be worthwhile to perform the operation.

(7) *Age of the Patient.*—The operation is likely to give the best results in young patients, but should be performed after active bone growth stops. If performed earlier, trauma to the epiphysis may distort or arrest growth. Good results can be confidentally expected up to the age of 40.

Indications

Thus arthroplasty would be indicated in complete fibrous and bony ankylosis. It is also indicated in partial ankylosis when movement is insufficient or when pain is a disabling factor.

Arthroplasty may also be performed as an early and primary treatment for serious fractures of the elbow in adults, when it is almost certain that normal function will not be obtained by conservative treatment. The conditions mentioned in "assessment of a case" should, however, be satisfied. In these cases there is considerable local trauma to the soft tissues as a result of the force of the injury. Also the triceps insertion may be separated from the bone in a rather undesirable fashion. Again there may be damage to the adjoining surfaces of the radius and ulna resulting in crossunion; this union would diminish or obviate pronation and supination (Fig. 4). All these factors then might vitiate a good result.

Fig. 1
Pre-operative x-ray (Case No. 3) showing complete bony ankylosis with fracture of bony mass due to manipulation.

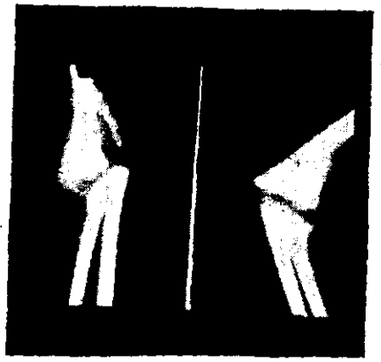

Fig. 2
Post-operative X-ray (Case No. 3)

Fig. 3
Post-operative photograph of Case No. 3 showing range of flexion and extension.

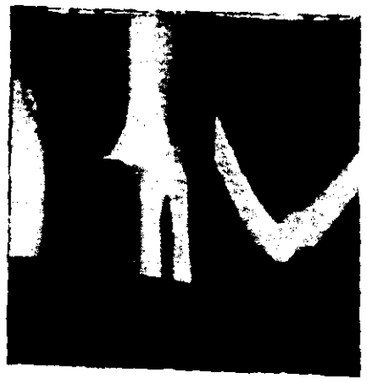

Fig. 4

Post-operative x-ray (Case No. 5).

Hence the performance of an arthroplasty at this stage may be considered controversial. It has actually been frowned upon by Sir Watson Jones[10] who prefers to treat the patient conservatively and subsequently reassess the case.

In this series, there were 2 patients on whom arthroplasty has been done as primary treatment for serious fractures and the results are satisfactory.

Fig. 5

Post-operative photograph (Case No. 5) showing flexion.

Fig. 6

Post-operative photograph (Case No 5) showing extension.

Technique

1. *Exposure.*—The joint is exposed through a vertical posterior incision, 5" in length. This was the incision employed in all the cases.

2. *Triceps insertion.*—Great care should be taken in trying to separate the triceps insertion from the olecranon. In doing this it will be found that it is not possible to strip off the insertion with a periosteum elevator, but the separation requires small strokes with a sharp knife. Unless this care is taken, the triceps insertion will get frayed and mangled and there will be considerable weakness of extension. By subperiosteal dissection, all vital structures will remain undamaged. With the exception of the ulnar nerve, none of these vital structures will even be seen.

3. *Formation of the gap.*—The bone ends are then dislocated out of the joint and sectioned with the gigli saw. The gap between the bone ends should be about 1".

Fig. 7

Pre-operative x-ray of Case No. 7.

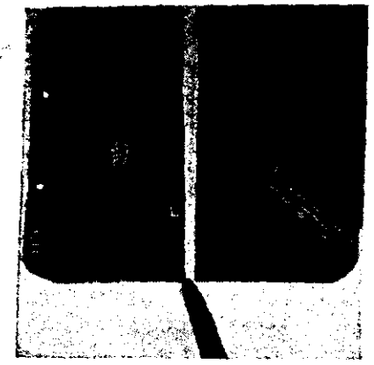

Fig. 8

Post-operative x-ray of Case No. 7.

A large gap is likely to result in a weak elbow. If serial post-operative x-rays are taken, one will notice that the distance between the bone ends gradually gets less and less.

4. *Shaping of the bone ends.*—The humerus when sectioned should have as wide an end as possible to increase stability. In this series, the standard method of shaping the bone ends was not employed. Instead, the bone ends were sectioned transversely—the humerus at or just below the epicondyles and the radius and ulna at the level of the neck of the radius.

5. *Covering of the bone ends.*—It is very essential to cover the bone ends, otherwise, a large amount of granulation tissue and new bone forms between the raw surfaces. This ultimately results in a fusion between the various bones.

Simple application of bone wax over the raw surfaces of the bone, without interposing any other tissue in between them is inadequate to prevent cross union. Various tissues have been used to cover the bone ends. These tissues should be generous and well draped over the raw bone. The interposed tissue acts as a protective cap and mould for the subjacent growth of fibroblats (Albrook and Kirkaldy-Willis-(1). The portion of the interposed tissue between the bone ends becomes liquefied and a sac or cavity lined with adventitious tissue forms which is not unlike that of a physiological joint or bursa.

Fascia lata has been used in all but one of the cases in this series. Removal of these sheets from the thigh does not result in any herniation of the thigh muscles.

Several other interposing materials have also been used. Mercer[7] mentions the use of pedicled muscle flaps, but these are not always convenient to use. Wilson[11] prefers nylon to fascia as he feels that fascia becomes adherent and more time is required to develop motion than with nylon. Giuntini[3] has used gelfoam as an interposing material since it was also haemostatic and is quite satisfied with his results. Sanchis-Olmos[9] has used skin instead with good results.

6. *Prevention of adhesions.*—By maintaining strict asepsis and careful haemostasis and by suitable post-operative care, the incidence of post-operative adhesions can be kept down to the barest minimum. The use of intraarticular hydrocortisone locally is also a useful aid and it does not cause any delay in the healing of the incision.

Post-operative Care

The limb is suspended from a transfusion stand with the elbow at right angles soon after operation. This gives elevation and the necessary traction.

Rehabilitation

Physiotherapy is started about 10 days after operation. Patient and persistent rehabilitation will ultimately reward the patient with a strong, stable and mobile joint. This usually occurs within 4-6 months following operation. A range of motion of from 90-120° in a useful arc is considered ideal (Van Zandt[5]).

Late-Results and Complications

Delayed ulnar nerve palsy may occur secondarily to valgus deformity when transposition of the nerve may be necessary. If the joint is not quite stable, the instability can be considerably reduced by fascial stabilisation (Knight[5]).

Bone absorption may occur as a result of extensive periosteal stripping or infection. New bone formation frequently occurs, but this hardly causes pain or limitation of movement. The x-ray appearance of a joint subjected to arthroplasty does not always parallel the functional results. Even when the x-ray shows irregular joint surfaces, the functional result can be quite good.

Medial or lateral subluxation may occur, but ordinarily if bone absorption is minimal, this subluxation is of little importance.

Analysis of the Cases (Table I)

All the 10 cases were operated on by me at the Lokmanya Tilak Municipal General Hospital, Sion. They were all post-traumatic cases. Eight had bony ankylosis (3 following fracture dislocations and 5 following comminuted fractures of the elbow). One was an unreduced dislocation with myositis and a stiff elbow. Another had fibrous ankylosis.

The ages of the patients ranged between 18 years and 48 years. In 2 cases, arthroplasty was performed as an early and primary treatment for serious compound fractures of the elbow.

In 7 cases, the injury had occurred between 2-12 months prior to operation. In one patient who was 23 years old, the injury was said to have occurred in childhood. In all cases, fascia was used as the interposing material except in one case where the raw bone ends were only cauterised and covered with bone wax. The follow up period varied from 4-25 months. The range of motion varied from 100-135° except in the case where no fascia was used where the range was only 55°. In the latter case, there was quite an amount of new bone formation post-operatively.

In all cases, muscle power was good. In the 2 cases, where arthroplasty was done for recent fractures, the power (especially of extension) was weaker though sufficient for ordinary activity. Stability was satisfactory in all cases.

Conclusion

Even though the number of cases in this series is too small to have any statistical significance, the results do appear encouraging.

TABLE—I

Name	Age	Aetiology	Duration of ankylosis	Interposing material	Follow-up	Range	Muscle Power	Stability	Complications
	Years								
1. O. B. ...	22	Fracture dislocation	1 year	Nil	2 years	55°	Good	Satisfactory	Nil
2. D. S. D. ...	32	Comminuted fracture	5 months	Fascia	8 months	120°	Good	Satisfactory	Nil
3. A. B. ...	18	Fig. 1	2 months	Fascia	7 months	Fig. 3	Good	Satisfactory	Subluxation (Fig. 2)
4. H. A. R. ...	25	Fibrous ankylosis	7 months	Fascia	4 months	110°	Good	Satisfactory	Nil
5. M. J. ...	48	Recent comminuted fracture	—	Fascia	2 years	Fig. 5–6	Satisfactory	Satisfactory	Subluxation and radio ulnar fusion (Fig. 4) (Upper end)
6. P. K. ...	23	Comminuted fracture	Since Childhood	Fascia	13 months	132°	Good	Satisfactory	Nil
7. V. D. ...	24	Fig. 7	4 months	Fascia	25 months	122°	Good	Satisfactory	Nil
8. M. M. ...	26	Unreduced dislocation with myositis	2 months	Fascia	6 months	135°	Good	Satisfactory	Nil
9. L. ...	30	Recent fracture dislocation	—	Fascia	7 months	100°.	Satisfactory	Satisfactory	Nil
10. R. G. ...	29	Fracture dislocation	6 months	Fascia	10 months	130°	Good	Satisfactory	Nil

Knight and Van Zandt[5] who followed up 45 cases, 14 years after operation came to the conclusion that fascial arthroplasty of the elbow properly performed in selected cases offers a reasonable expectation of 70-120° of motion in a useful arc with good strength and stability and with little if any pain.

Summary

The aim, indication, technique and complications of arthroplasty of the elbow have been described. Due stress has been laid on the proper selection of cases and on the need for patient and persistent post-operative rehabilitation. The manner of selecting a case has also been presented. 10 cases have been analysed.

Acknowledgements

I wish to thank the Superintendent and the Chief Surgeon who offered all possible facilities for the completion of this analysis.

REFERENCES

1. Albrook, D. & Kirkaldy-Willis, W. H.: "Restoration of articular surfaces after joint excision", J. of Bone and Joint Surgery, 40B: 742, 1958.
2. Editorial: "Plastics in Surgery", Brit. Med. Journal, 1: 750, 1952.
3. Giuntini, L.: at the meeting of the British Orthopaedic Association, J. of Bone & Joint Surg. 38B: 941, 1956.
4. Hass, J.: "Functional arthroplasty" J. of Bone & Joint Surg. 26: 297 April 1944.
5. Knight, R. A. & Van Zandt, I. L. "Arthroplasty-end result", J. of Bone & Joint Surg., 34A: 610, July 1952.
6. Murphy, J. B.: "Arthroplasty", Ann. Surg. 57: 593, 1913.
7. Mercer, W.: Orthropaedic Surgery, 4th Edition (1950), London: Edward Arnold & Co., pg. 919.
8. Phemister, D. B. & Miller, E. M.: "The method of new joint formation in arthroplasty. An experimental study", Surg., Gynaec and Obstetrics, 26: 406, 1918.
9. Sanchis-Olmos, V: at the 7th Congress of the International Society of Orthopaedic Surgery and Traumatology, J. of Bone and Joint Surg. 40B: 159, 1958.
10. Watson Jones: Fractures and Joint Injuries, Vol. II, IV Edition (1955) Edinburgh and London: E. & S. Livingstone Ltd., Pg, 536.
11. Wilson, P. D.: in the discussion of reference No. (5) Jr. of Bone and Joint Surg. 34A: 618. July 1952.

—Received for publication on 22-4-1960.

THE TREATMENT OF ORDINARY ABSCESS BY INCISION, EVACUATION AND PRIMARY SUTURE

BY

A. K. TALWALKAR and M. N. SAHANE, Bombay.

From the days of primitive surgery till almost today, the old classical treatment of an acute abscess has been to incise, evacuate and drain, followed by regular daily dressings. Granulation tissue creeped in from the walls of the cavity and eventually filled it. The skin grew over the granulations and the cavity was closed. It is essential that the cavity should fill in evenly without pocketing so as to prevent recurrence or fistula formation. This was avoided by packing the cavity with gauze soaked in antiseptic solutions. The pack was changed frequently as the cavity began to fill up. But this would take weeks before the abscess was healed up completely.

We are challenging this classical treatment by *incision, evacuation and primary suture of abscesses* and we think it is a correct modern treatment with the umbrella of antibiotics.

It is almost a hundred years ago, that John Hilton during one of his famous lectures on "Rest and pain" asked a question: Why do surgeons open abscesses? He has given many reasons for it.

(1) To relieve the patient from pain and constitutional disturbance.

(2) To limit destruction of tissues.

(3) To prevent further encroachment upon important organs.

(4) To remove accumulated extraneous fluid; but the most important being,

(5) To permit and secure co-optation of internal surfaces of the abscess, to give its internal surfaces rest, so as to permit of their union and further rest for consolidating the medium of union.

Once the abscesses are opened, and evacuated, the frequent dressings, this continuous interference with nature by the surgeon, might be called "meddle some surgery" in John Hilton's words. Once more to quote Hilton, "Rubbing the two surfaces of the abscess together once or twice a day, we are not only likely to disturb the natural process of granulation, but almost sure by such friction to induce an inflammatory condition in structures which for the purposes of repair ought to be in a comparatively healthy state and quietly taking their own steps towards filling up the whole interior of an abscess in a sound manner.

Frequent dressings are not only painful but often may lead to a secondary infection. And as most of the patients are treated as outdoor cases, they involve many hours of travelling, waiting and much expense.

For many years, in the treatment of acute osteomyelitis, after surgical interference under antibiotic cover, Trueta has been doing a primary closure of the skin with most gratifying results. Healing is almost invariably by primary intention and the incidence of recurrence and sinus formation was much less.

In the Orthopaedic Unit of K. E. M. Hospital, Bombay, we receive many cases from the general surgical units with a diagnosis of ? Acute osteomyelitis, which on opening turn out to be cases of simple abscesses without involvement of bone. Applying the same principle as employed by Trueta in cases of acute osteomyelitis, we have departed lately from the accepted principle of abscess treatment and have done incisions, evacuation and primary suture of the skin instead of incisions, evacuation and drainage.

The object of this paper is not to present a large series of cases but to stress the point, whether the principle of abscess treatment should not be changed now. As the number of cases of pure abscesses received by Orthopaedic Unit is very small, it is decided to present a paper with large number of cases in collaboration with general surgical units at a later stage.

Thirty-six cases of pyogenic abscesses at various sites treated in one of the Orthopaedic Units of K. E. M. Hospital, Bombay, from January 1960 to May 1961 have been included in this series. Twenty-two were males and 14 females.

Age incidence is as follows :—

Table I

Age in years		No. of cases
0—10	...	22
11—20	...	4
21—30	...	2
31—40	...	5
41—50	...	1
51—60	...	0
61—70	...	1
71—80	...	1
	Total	36

The table below shows the various sites where abscesses were located :—

Table II

Site	Right	Left	Total
1. Scapular region	1	1	2
2. Axilla	—	1	1
3. Shoulder	2	—	2
4. Arm	3	2	5
5. Forearm	2	1	3
6. Chest	2	—	2
7. Lumbar region	—	—	2
8. Gluteal region	3	2	5
9. Thigh	5	4	9
10. Popliteal region	3	1	4
11. Leg	2	1	3
12. Foot	1	3	4
Total	24	16	42

A few hours before the abscesses are opened, antibiotics are started. In most cases injection Penicillin 1 lac units 3-4 hourly is sufficient. But if the antibiotic sensitivity tests show that the organisms are resistant to Penicillin then a broadspectrum antibiotic is given.

Abscesses are opened under general anaesthesia after usual preparations. After the abscess is incised and the pus evacuated, a finger is inserted in the abscess cavity, septa if any are broken, hidden pockets are searched for and opened and an attempt is made to remove as much debris and necrotic tissue from the abscess wall as possible. No deliberate attempt is made to curette out the abscess wall with a scoop.

After the surgeon is satisfied that no pockets are left and thorough evacuation is done, local Penicillin is instilled and the skin is closed with interrupted nylon sutures. A compression dressing is given. The pus is sent for smear, culture and antibiotic sensitivity test.

Post-operatively the limb is rested in different ways according to the site of the abscess. In the abscess of the thigh Thomas' traction is given. In the case of leg and foot a posterior slab is all that is necessary. Abscesses of upper limbs are rested by a cuff and collar sling.

Antibiotics are continued for 6-7 days.

The sutures are removed on the 7th or 8th day. Till that time wound is not opened. If the healing of the wound is by primary intention, there is not much oedema, the immobilisation is discontinued and active exercises are started. Patient gets back to normal work in a few days time. If there is slight gaping with little discharge, just a superficial dressing 2-3 times will be snfficient for healing. If, as in occasional case, there is a recurrence of the abscess, a proper antibiotic as determined from the antibiotic sensitivity test is started before the procedure is repeated.

Out of 14 smear and culture reports available, the following table shows the incidence of different organisms :

Table III

Smear Reports

1. Pus cells only	...	7
2. Pus cells with mononuclear cells and R. B. C.	...	1
3. Pus cells with gram positive cocci	...	4
4. Pus cells with coccobacilli and gram negative rods	...	1
5. Pus cells with gram negative bacilli	...	1

Table IV

Culture Reports

1. No growth	...	8
2. Staph. pyogenes	...	2
3. ,, aureus	...	2
4. ,, albus	...	1
5. B. coli	...	1

In one case an entire guinea worm was removed after the incision of the abscess (Case No. 24).

Follow up and results

All the 36 cases were followed up. The following table shows the period of post-operative follow-up. Minimum follow up was 2 months and maximum 20 months.

Table V

Follow up period		*No. of cases*
0 - 6 months	...	23
6 months - 1 years	...	7
1 - 1½ years	...	3
1½ - 2 years	...	3
	Total	36

At the follow up examination the cases were seen to find out whether the union of the skin was by primary intention or there was any gaping of the wound or a sinus which persisted. Also whether there was a recurrence of the abscess at the same site was determined. Quick improvement in function was a noticeable feature.

Table VI

Result	*No. of cases*	*Percentage*
Primary union of skin	30	83·3
Gaping of the wound	1	2·7
Temporary sinus formation	3	8·3
Recurrence of abscess at same site	2	5·5

All the cases that are listed under the heading of 'Primary union' showed complete healing of the skin on the 7th or 8th day. There was no recurrence or oedema of the skin. Criterion of healing was complete healing of wound without surface moisture, granulation tissue or scab. The movements were started immediately and they returned to normal within a few days.

Sutures of one patient were removed on the 4th day by mistake. About 1 inch of the wound in the centre gaped. Only superficial dressing was done without any attempt to pack the cavity and the wound healed on the 16th day (Case No. 35).

Three cases showed temporary sinus formation. It may be due to inadequate drainage of the abscess. These patients also healed up in 8-10 more days (Case Nos. 16, 29 and 31).

There was a recurrence of the abscess at the same site after a few days in two patients. But it should be noted that both these patients had primary union of the skin. In one case the recurrent abscess was very small (Case No. 25) and it subsided after aspiration. In the other patient (Case No. 15) the procedure was repeated and the wound healed by primary intention.

Discussion

Since the discovery of antibiotics, like many other diseases the treatment of abscesses has to be modified with an advantage to the surgeon. Incision and primary suture of the abscesses is a very cenvenient and time-saving procedure. In a very busy general hospital like K.E.M. Hospital, where 20-25 abscesses are opened every day, and of course, dressed regularly thereafter, many hours of labour can be saved, and the house surgeon can have more time at his disposal for the other work if this procedure is followed in every case.

An incidence may be recalled in this instance. In one of the general surgical units, the house surgeon, who had heard about this procedure from his orthopaedic colleagues, did a primary suture of the abscess. On the 3rd day during the ward round, his chief ordered him to open the abscess again and pack it. To his surprise the house surgeon found it difficult to get the original abscess cavity, which by this time was healing satisfactorily.

Ellis of Leeds, has been following this procedure for many years for abscesses around the anal region, with very satisfying results. But he curettes out the lining of the cavity "to allow blood laden with antibiotic to ooze in". He postulates that before operation the lining of the abscess had not only prevented the infection in the abscess from getting into the circulation, but had also prevented Penicillin in circulation from getting into the abscess.

Scott et al had used this method in the treatment of infections of the hand. They called it excision of the abscess because they excised the necrosed tissue completely before suturing the skin. "Excision takes the place of the usual process by which dead tissues are eliminated by discharge. Healing is thereby accelerated. Morever, the best possible conditions are provided for control of residual infection by the striking and immediate improvement in local blood supply after relief of tension".

Immediate skin cover is the best defence against secondary infection and minimises amount of scarring.

There are certain factors which may delay or prevent healing.

1. *Necrosis of the skin.*—This happens when the conservative treatment is continued for a long time and the abscess points to the surface. Even in these cases if the abscess is incised at the comparatively healthy part of the skin, sloughing of the skin and secondary ulcer formation can be prevented.

2. *Inadequate evacuation of the abscess.*—This may be the cause of recurrence or temporary sinus formation.

3. Inadequate antibiotic umbrella.

Summary

Thirty-six cases of pyogenic abscesses at various sites treated in the last 20 months in the Orthopaedic Unit of the K.E.M. Hospital with incision, evacuation and primary suture of the skin are reviewed. 83·3% showed union by primary intention without recurrence. Advantages of this method over the traditional one of incision, evacuation and drainage are discussed.

Acknowledgement

We wish to thank the Dean of the K. E. M. Hospital for allowing us to use the material of the K. E. M. Hospital, Bombay.

Dr. A. K. Talwalkar said the work done under his guidance, the number of cases was small because of faulty documentation which led to exclusion of many cases from the series presented. He advocated removal of the lining membrane, use of a big enough incision, suturing of skin only and sufficiently long use of antibiotics for successful out-come.

Dr. M. R. Chowdhary (Akola) agreed with Dr. Sahane and described his technique of incision and drainage of breast abscesses. He, however, used a drain for 24 hours and sutured the deeper tissues with catgut obliterating all dead-space.

Dr. Dixit pointed out that the series did not include infections of tips of fingers.

Dr. Satyaprakash advocated the use of tourniquet for clear vision.

Dr. Kohli enquired if the recurrences were in any way related to the size of the abscess and absence of drainage. Dr. Sahane replied to say the small series did not allow him to draw definite conclusions in this direction.

REFERENCES

1. Aird, Ian: A Companion in Surgical Studies, 53, 1957.
2. Bailey, Hamilton: Pye's Surgical Handicraft, 461, 1956.
3. Ellis, M.: Incision and primary suture of abscesses in anal region, Proceedings of Royal Society of Medicine, Vol. 53/8: 652, 1960.
4. Hilton, John: Rest and pain, 125, 1950.
5. London, J. B., Miniero, J. D. and Scott, J. C.: Infections of the hand, Journal of Bone and Joint Surgery, 30-B: 409, 1948.
6. Trueta, J.: Discussion on acute osteomyelitis, Journal of Bone & Joint Surgery, 37-B: 722, 1955.

—Received for publication on 25-8-1961.

ASSOCIATION NOTES

GOVERNING BODY

President :

B. N. BALAKRISHNA RAO (Gwalior)

Vice-President :

S. P. SRIVASTAVA (Kanpur)

Three Immediate Past Presidents (Ex-officio Members)

S. K. SEN (New Delhi)
B. N. SINHA (Lucknow)
A. E. deSA' (Bombay)

Hony. Secretary :

A. VENUGOPAL (Madras)

Hony. Treasurer :

C. P. V. MENON (Madras)

Members :

S. S. ANAND (Amritsar)
U. MOHAN RAU (Madras)
R. NIGAM (New Delhi)
R. N. SINHA (Patna)
B. RAMAMURTHI (Madras)
K. S. GREWAL (Amritsar)
A. K. TALWALKAR (Bombay)
B. B. OHRI (Indore)

THE XXIV ANNUAL CONFERENCE

The 24th Annual Conference of the Association of Surgeons of India will be held at Calcutta during December 1962, further details of which will be announced later.

CONTRIBUTORS—JANUARY, 1962

1. Dr. G. B. Parulkar,
   Dr. A. J. Dhruva,
   Dr. P. M. Javeri,
   & Dr. P. K. Sen,
   } Dept. of Surgery, K. E. M. Hospital, Parel, Bombay-12.

2. Dr. P. N. Vyas, M.S., F.C.P.S.,
   Dr. T. Thomas, M.B.B.S., F.A.C.S.
   Dr. R. H. Betts, M.D.,
   Dept. of Thoracic Surgery.
   & Dr. G. Lewis, M.B., B.C.H., F.F.A.R.C.S., Dept. of Anaesthesia, C. M. C. Hospital, Vellore (S. India).

3. Dr. A. G. Fletcher, Jr., F.A.C.S., F.R.C.S.(C), Director of Surgery, Wanless Tuberculosis Sanatorium, Miraj.
   & Dr. D. K. Gosavi, M.S., D.O.R.L., Dept. of Otolaryngology, Miraj Medical Centre, Miraj.

4. Dr. R. S. Gupta, F.R.C.S. (Eng.), F.A.C.S.,
   & Dr. S. S. Baijal, M.D.
   } B. R. Singh Hospital, Calcutta.

5. Dr. Dhruba Kumar Sen, M.S., Visiting Surgeon,
   & Dr. Asit Kumar Maitra, M.B.B.S., Ex-Senior House Surgeon, Nilratan Sarkar Medical College Hospital, Calcutta,
   & Dr. Anantaprasad Sinha, M.B.B.S., Medical Officer, Chittaranjan Cancer Hospital, Calcutta.

6. Dr. R. S. Gupta, F.R.C.S. (Eng.), F.A.C.S.,
   & Dr. (Miss) Rama Roy, M.B.B.S., D.G.O.,
   Dept. of Surgery, B. R. Singh Hospital, Calcutta.

7. Dr. A. K. Banerjee, M.B., M.S., Dept. of Surgery, Christian Medical College, Vellore (S. India).
   & Dr. B. N. Balakrishna Rao, F.R.C.S., F.I.C.S., Principal and Professor of Surgery, G. R. Medical College, Gwalior.

8. Dr. A. D. Dias, M.S., Lokamanya Tilak Municipal General Hospital, Sion Road, Sion, Bombay-22.

9. Dr. A. K. Talwalkar, M.Ch. orth., F.R.C.S. (Eng.),
   & Dr. M. N. Sahane, M.S.,
   Dept. of Orthopaedics, K. E. M. Hospital, Parel, Bombay-12.