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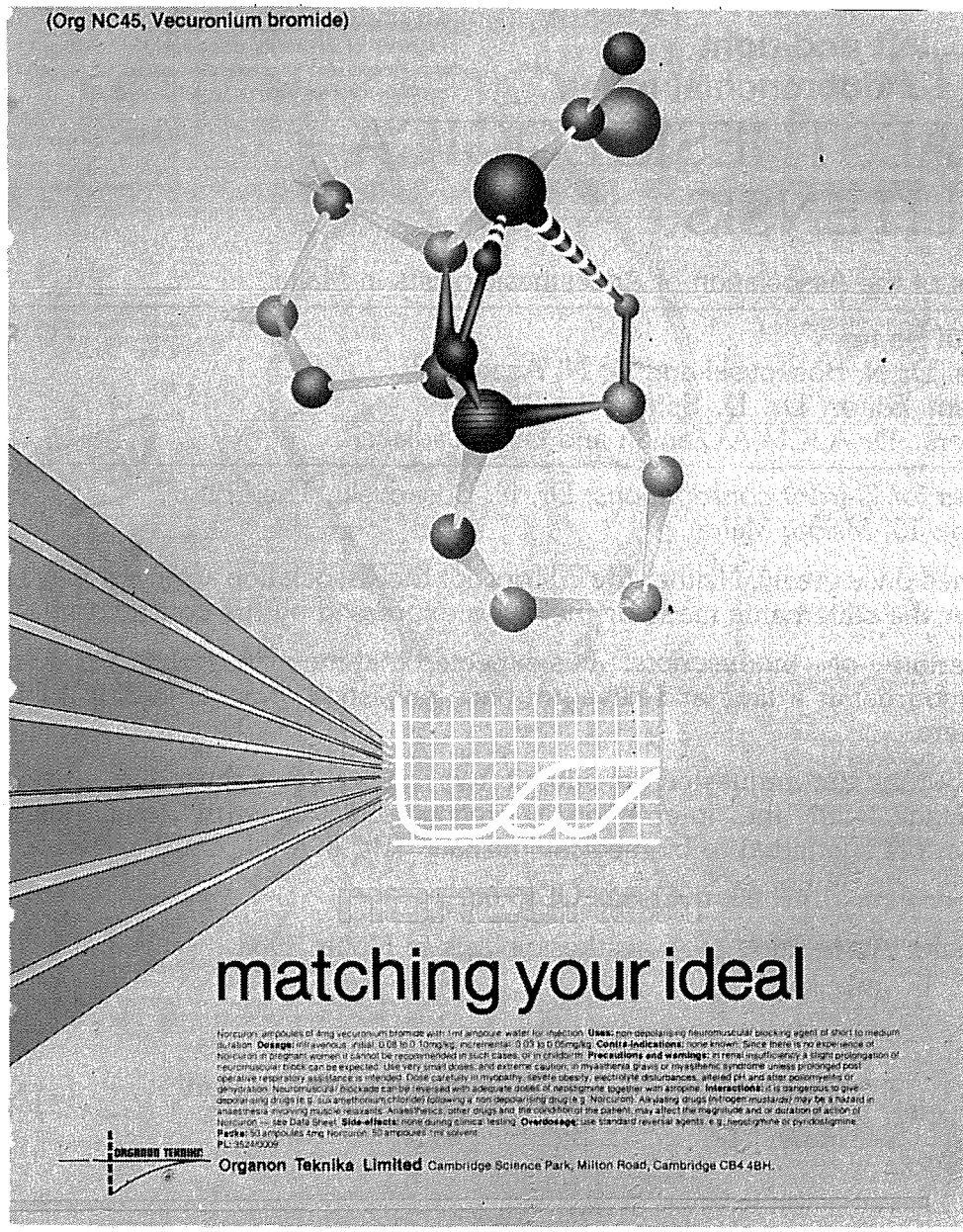

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Editors' Preface

"To those pioneers who, unflinching, have trodden unknown and unsure paths, that those who follow may more clearly see the way ahead."

(*K. Bryn Thomas*)

In November 1982 a group of fifteen anaesthetists working in St. Luke's Hospital in Malta founded the Association of Anaesthesiologists and have applied for membership to the World Federation of Societies of Anaesthesiologists (WFSA). Exactly one year later the Association is holding its Inaugural Scientific Meeting and launching its Journal, "Acta Anaesthesiologica Melitensis". The primary object of the Journal is to encourage clinical research on the Island and to strive for the best possible anaesthetic service, intensive care medicine and pain therapy.

The first issue of Acta Anaesthesiologica Melitensis exclusively comprises the scientific papers of the anaesthesiologists working in St. Luke's Hospital which were presented at the Inaugural Conference.

Our wish is that the coming years will witness continuous efforts to promote the highest and safest standards of care for our patients.

We gratefully acknowledge the support and assistance from the Ministry and Department of Health of Malta, and the local pharmaceutical producers and representatives.

November, 1983

N. Boskovski
N. Azzopardi

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A New Approach to Tetanus*

N. AZZOPARDI and N. BOSKOVSKI

Summary

Following a five year study the authors are convinced that the treatment of established Tetanus is simplified and improved by three weeks controlled respiration with full muscle relaxation and adequate sedation.

Key Words

Established Tetanus. Controlled respiration. Muscle relaxation. Adequate sedation.

"Tetanus appears to have a dangerous period... I have never seen one die where it had existed three weeks" John Hunter (1835).

Malta and Gozo are two small islands in the Mediterranean having a surface are of 122 square miles and a population of a third of a million. As many tourists visit the Islands in the summer season the population swells to over a million, in the peak of the season. No tourists were ever infected during their stay here.

Tetanus spores are present in the faeces of herbivorous and carnivorous animals and so are ubiquitous in the soil and dust. To germinate the spores need strict anaerobic conditions and when the bacteria are lysed by the white cell enzymes a strong neurotoxin is released. This neuro toxin is lethal in a concentration of 0.1 mg/l.

Epidemiology

In the Maltese Islands there is a fear of Tetanus as its fatality has been as high as 55% (Statistics 1966). This was well before the Intensive ward was opened i.e. five years ago. All cases of supsected Tetanus are immediately refered to St Luke's Hospital: the Islands' main hospital. The total number of cases admitted during this five year study was 37 but Tetanus spores or bacilli were only isolated in 26. None of these cases had ever had Tetanus Toxoid Injection though 4 had had Tetanus Serum (ATS). Out of these 37 cases there were 8 deaths - less than 20%.

The majority of the cases were farmers or part time gardeners and it is the writers opinion that this illness should be classed as an occupational one, in Malta, unlike in other countries.

There were no cases of maternal or neonatal Tetanus.

It is during the summer months that tetanus cases mostly occur mainly because most farmers go barefooted in the fields. The worst month is October when manure is spread onto the soil for preparation for the winter rain. The site of infection is most often a forgotten injury in the sole of the foot though one woman, a nun, cut her finger whilst peeling potatoes.

Presentation

The patients had called on their general practitioner for strange headaches, backaches and mild dsyphagia. It is so easy to attribute these symptoms to hard work in the fields but if a proper examination is carried out there is also a slight temperature, tachycardia, stiffness of the lumbosacral muscle group and increased tendon jerks.

Maybe the patient will draw the doctor's attention to a small sore somewhere in the sole of the foot - an old injury that refuses to close. The injury is usually a puncture wound with raised keratinised edges and oozing serous fluid.

Clinical Features

Trismus and increased rigidity of the lumbosacral muscles develop in 24 hours from the onset. Swallowing becomes difficult and breathing tiring and exhausting. Opisthotonos (back arching) is another development because the back muscles in tonic spasm are stronger than the abdominal ones. In another twelve hours the muscle rigidity may develop into painful clonic muscular spasms - a tetanic fit - that is sometimes so severe as to cause tearing of a muscle. Spasm of the facial muscles leads to the characteristic mask of "risus sardonicus" - rasied eyebrows, semi-closed eyes, lips drawn back to reveal clenched teeth. In the untreated case severe spasms of the laryngeal and respiratory muscles will produce cyanosis. Death is due to exhaustion and congestive heart failure due to severe acidosis. Besides its effect on the skeletal musculature the tetanus toxin has also a marked effect on the sympathetic nervous system causing tachycardia and

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* This paper was presented at the 6th European Congress of Anaesthesiology, London 1982.

arrythmias, temperature changes, and vascular instability usually with peaks of hypertension. If uncorrected these cathecolamine induced crises may cause myocardial damage and also ventricular failure.

Prognosis

It is common knowledge that if the symptoms of tetanus start within 10 days of the injury the prognosis is very bad. The later the onset of the symptoms the better the prognosis.

There were no cases of deaths in patients who had had Tetanus Toxoid.

If the patient suffers from a concomittant illness (and diabetes is a very common disease in Malta), then the prognosis is more guarded.

Clinical Course

Before the opening of the ITU ward mortality was considerably high. Since the ITU ward was opened this mortality rate has been reduced to 20% which is still high compared to what is reported from Oxford U.K. In this series four patients died in the first week of admission to the ITU Ward; one because of respirator failure, one of cephalic tetanus and two because of uncontrolled diabetes and old age. The other four patients died of complications in the third week of the illness from *Pseudomonas* bronchopneumonia.

The patients discharged in the third week from the ITU all left hospital soon after, free of any muscle rigidity or nerve damage.

Three weeks duration of treatment seem to be essential before all manifestations of the illness stopped. It could be that despite widespread local excision of the suspected wound of entry, tetanus spores germinate and produce the toxin from other sites.

Management

It is the local custom to rush off to hospital all cases of suspected tetanus. At Admission Ward the Surgical registrar sees the patient and on the least suspicion admits him immediately to the ITU ward. This is a defensible reaction when the fear the Maltese have of the outcome of the illness is appreciated.

At the ITU ward mild sedation is prescribed while the usual monitoring equipment is set up and blood taken for the usual parameters.

If with further observation and consultant advice the diagnosis is confirmed then without further delay treatment is started. The grave risk of developing a tetanic fit and aspiration of vomit with consequent bronchopneumonia will worsen the prognosis of this serious but otherwise curable illness.

If despite mild sedation the patient develops signs of spreading muscle rigidity then muscle relaxation and intubation with intermittent positive pressure ventilation is performed. This is continued together with heavy sedation for around three weeks. Experience has taught us that stopping the above regimen earlier than three weeks may lead to an unexpected fit.

Any drugs previously taken by the patient are continued but the dosage is individually regulated as drug interaction and enzyme induction may grossly upset the balance. If the patient was on oral hypoglycaemics then plain insulin in appropriate dosage is administered.

The benzodiazepenes hold the field as regards sedation, sleep induction, anticonvulsant and muscle relaxation. Flunitrazepam (Rohypnol) is the preferred drug used in heavy doses of 0.3 mg per Kg body weight.

Tubocurarine chloride 0.5 mg per Kg body weight is the preferred muscle relaxant drug because it is longer acting and its peripheral vasodilatory action is useful to counteract the sympathetic overactivity.

Chlorpromazine (Largactil) in doses of 0.5 mg per Kg body weight is used to limit the hypertension and cardiac irregularities. ß adrenergic blockers are used if hypertension and tachycardia are still not brought under control by chlorpromazine alone. If a stronger α and ß blockade is necessary then Labetolol is administered IV with 1 mg at a time boluses till adequate control is attained.

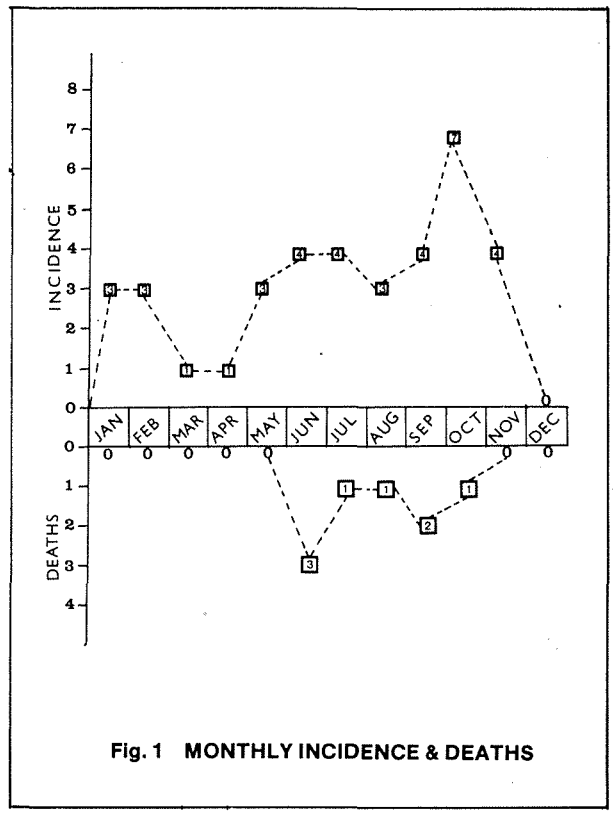

Fig. 1 MONTHLY INCIDENCE & DEATHS

Intravenous feeding is started from the second day of the illness, special attention being given to the high catabolic effect that sympathetic overactivity produces. Strict correction of acid base imbalance is maintained. In this second week it is common to find a hypochromic anaemia that often needs two pints blood transfusion for correction. Could this anaemia be due to an effect as yet undescribed, of the toxin on the bone marrow?

Antibiotics in high doses are given both to control any secondary infection and bronchopneumonia as well as to limit the number of bacilli sporing off. Penicillin remains the drug of choice in a massive dose of 12 million units daily. If the patient is allergic than Erythromycin is given. Additional antibiotics are prescribed depending on sputum and blood cultures.

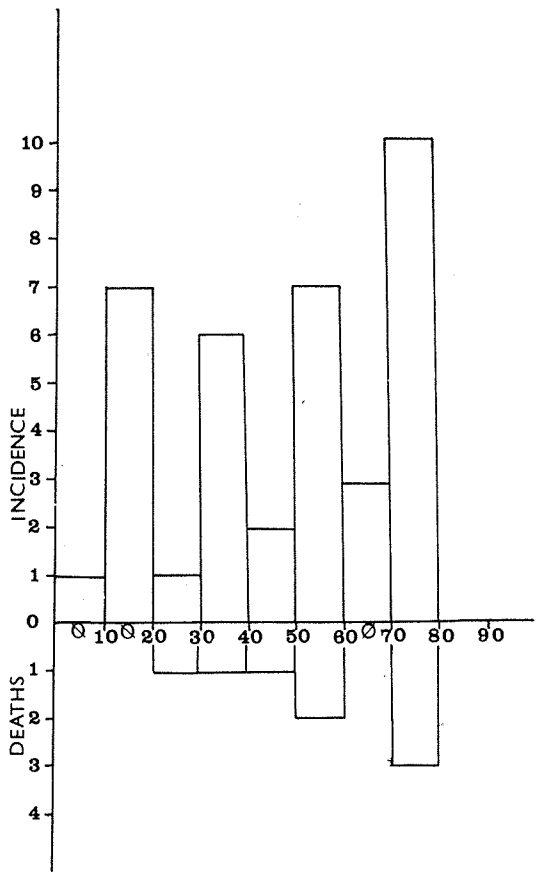

Fig. 2 AGE GROUP INCIDENCE & DEATHS

Anti tetanic serum is no longer given. Human Anti-Tetanus Globulin in a strong dose of 3000 Units is prescribed once only IV.

The wound is attended to on the second or third day. Thorough cleaning with excision of the edges is performed. Peroxide soaked gauze is packed every six hours to try and limit the number of germinating spores.

After three to four days of intubation a formal tracheostomy is performed. This helps the patient and nurse enormously. Phsiotherapy is simplified, mouth hygene is facilitated and the patient feels more comfortable. In the third week the patient can usually talk and his morale is improved when sips of fluid are partaken.

Discussion

Despite all precautionary measures cases of tetanus occur at the rate of 8 a year in the Maltese islands. With the modern approach the infection is not fatal in itself but the complications can indeed be.

It is our hope that as more people become immunised the disease will be eradicated completely, because as our study shows none of the cases had ever had tetanus toxoid injections. In 1982 a law has been passed whereby children attending kinder garten have to produce an immunisation certificate for Tetanus, Diphteria and Polio.

It is our wish that this illness will be recognised as an occupational one by all governments.

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The use of Naloxone in Critically-ill Patients*

N. BOSKOVSKI, M. COSIC, A. LEWINSKI, D. SPITERI and N. AZZOPARDI.

Summary

Ten critically-ill patients with prolonged hypotension or unrecordable systolic blood-pressure, impaired mental status and respiratory depression in this study. Naloxone, 0.8 - 1.2 mg was injected intravenously in all patients. in six hypotensive patients, systolic blood-pressure rose in range of 26-35%, within 5-15 minutes following the naloxone injection. In four patients systolic blood-pressure was unrecordable and after naloxone injection rose to a value of 100-120 mm Hg. Naloxone was effective in three patients in which dopamine could not maintain the systolic blood-pressure at optimal renal perfusion level. It seems that naloxone had a lesser effect in three patients treated with high doses of corticosteroids. The therapeutic effect of naloxone is suggested on the assumption that endorphins are mainly responsible for the hypotension and respiratory depression in critically-ill patients.

Key words

Opioid antagonist; naloxone.
Intensive care; critically-ill patients.

Recent discovery of the endogenous opioids, their possible role as neuromodulators and the diverse localisation of specific receptors for endogenous ligands in the body[1] initiated the current widespread interest to explore various clinical implications. It is suggested that the endogenous opioids, particularly (ß-endorphin) a sequence of pituitary ß-lipotropin contribute to the hypotension in septic shock[2] and we presume they may have the same effect in all critically-ill patients.

It was demonstrated that the hypotension resulting from intravenous[3] or intracisternal injection[4] of ß-endorphin in animals can be readily reversed by the specific opiate antagonist, naloxone.[5]

Peters et al.[2] in a clinical trial, observed an increase in systolic blood-pressure within minutes following injection of 0.4–1.2 mg naloxone in septic shock patients with prolonged hypotension.

Burnie[6], claims that specific opiate receptors exist in cardiac muscle in the rat, so the situation is more complex than appears.

If endorphins are mainly responsible for the hypotension and respiratory depression then naloxone would be expected to be of significant therapeutic value in the treatment of critically-ill patients.

Patients and Methods

All patients included in the study had prolonged hypotension or unrecordable systolic blood-pressure, impaired mental status and respiration and were being nursed in the I.T.U.

Observations were made before giving the naloxone injection and in the period after receiving naloxone.

In three patients, dopamine, which was infused at a rate of 7-20 µg/kg/min over a period of 7-12 h, was stopped just before naloxone administration.

Seven patients were on controlled or assisted ventilation. All patients were receiving intravenuous fluids and other supportive measures.

Arterial blood-pressure was measured with mercury manometer, with the cuff on the upper arm, every 1-5 min by auscultatory method. Central venous pressure, hourly urine output and blood-gases were monitored in all patients. (Table 1) Naloxone hydrochloride ("Narcan", Winthrop) was injected intravenously at a dose of 0.4 mg initially. If in 5–10 min the systolic blood-pressure did not rise to 100 mm

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* This paper was presented at the 6th European Congress of Anaesthesiology, London 1982.

Hg, naloxone was repeated.

Special emphasis was put on the naloxone effect upon systolic blood-pressure, respiration and changes in the mental status. Hourly urine output and blood-gases were also measured.

Results

Eight patients out of ten survived and were then transferred to the respective general ward and later discharged. Two patients died, one (seven days after naloxone injection) in the operating theatre on induction of anaesthesia for planned tracheostomy, and the other one (two days after naloxone administration) from liver failure. Seven patients, were on artificial respiration at the moment of naloxone injection. Five of them were put off the ventilator either the same day or in the next 48 h following the naloxone administration. Two patients died whilst still on the ventilator after 2 and 7 days respectively as mentioned above. Patients 1, 2 and 3 had received high doses of corticosteroids (1.5–2.0 gr of Solu-Cortef) and following naloxone injection, the initial rise in blood-pressure was in range of 11-25%, significantly less than in the patients who were not given exogenous corticosteroids.

The following is a more detailed presentation of four most demonstrative patients:

Patient 1 (Fig. 1) with septic shock, had sustained hypotension for 12 h before dopamine was stopped and naloxone 0.4 mg was injected intravenously. Blood-pressure rose from 80/60 to 100/70 mm Hg in ten minutes time. Hourly diuresis was 16 ml/h and in the moment of naloxone administration Lasix 40 mg was given i.v. which improved significantly hourly diuresis in the next 8 h., but the blood-pressure did not alter.

Pulse rate from 145/min decreased to 100/min in the next few hours following the naloxone injection.

Table 1 CLINICAL CHARACTERISTICS

AT TIME OF NALOXONAL ADMINISTRATION

patients	N history	sex	age	DIAGNOSIS	BP before NALOXONE	BP after adm. N	BP stability	on ventilator	DOPAMIN	Transfer to the ward
1	103367	F	67	SEPTIC – SHOCK	80/60	90/65	110/70	ON	stop	yes
2	134897	M	40	HAEPATIC COMA	80/60	100/70	100/70	ON	stop	DEAD
3	132781	F	40	ANAFILACTIC SHOCK CARDIAC AREST	90/60	100/70	120/90	ON	stop	yes
4	119765	F	61	RESPIRATORY FAILURE CARDIAC AREST	0	120/70	120/80	ON	start	DEAD
5	066849	F	79	ACUTE PANCREATITIS	0	90/50	120/80	ON	no	yes
6	137038	M	60	POST-OPERATIVE SHOCK	0	120/100	160/100	ON	no	yes
7	133952	F	48	SUBTOTAL COLECTOMY	0	100/70	110/80	no	no	yes
8	005924	F	77	SEPTIC SHOCK	95/65	100/80	110/80	no	no	yes
9	018630	M	50	POST-OPERATIVE SHOCK	90/60	130/75	120/70	no	no	yes
10	000106	M	72	RESPIRATORY FAILURE MYOCARDIAL INFARCTION	70/40	130/80	120/80	ON	no	yes

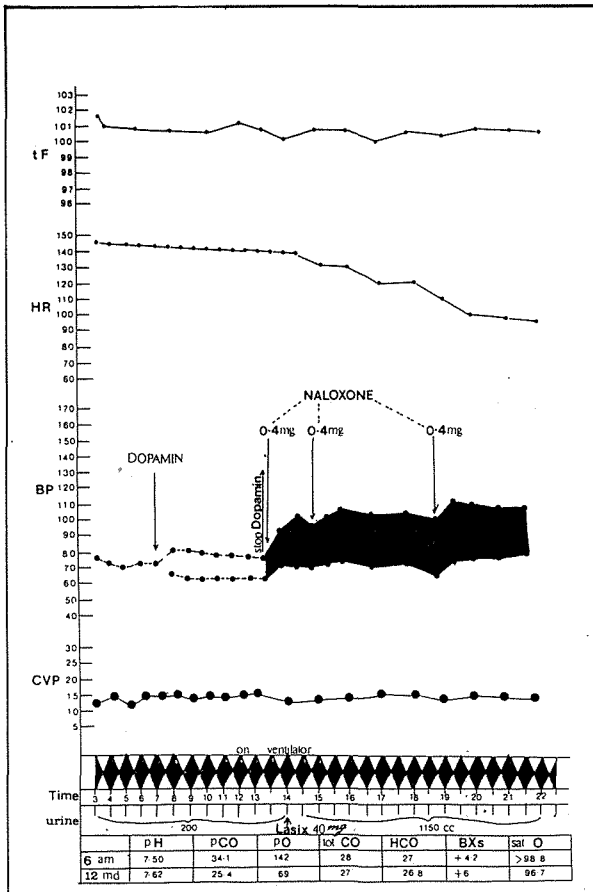
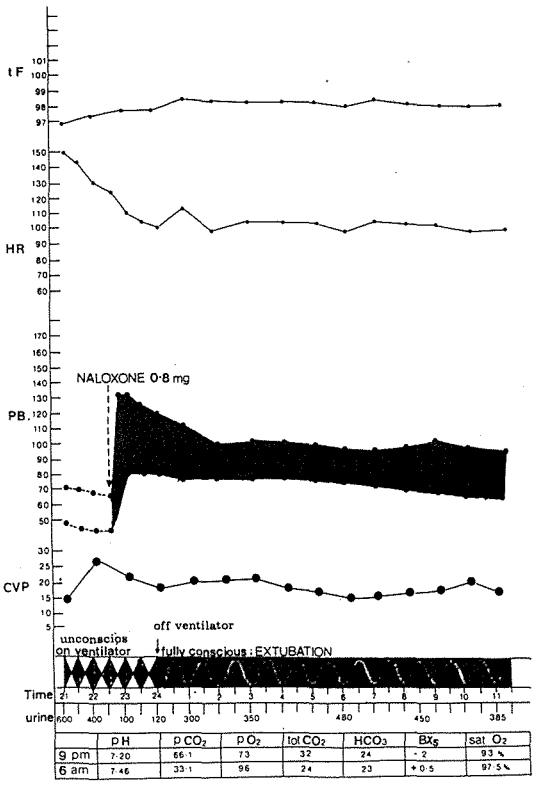


Fig. 1 G.A. No. 103367 female 67 yrs. SEPTIC SHOCK

Controlled ventilation was maintained for five days. Blood-gases improved the next day after admission to ITU. After ten days patient was transferred to the ward in stable general condition.

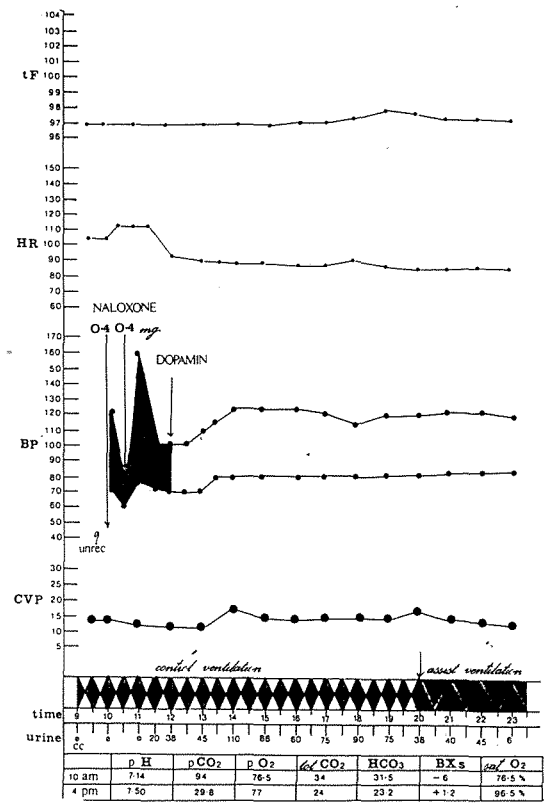
Patient 10 (Fig. 2) was admitted unconscious with a respiratory failure and myocardial infarction. Blood-pressure was 70/40, heart rate 150/min. Endotracheal intubation was carried out immediately in the Casualty Dept. He was put on controlled ventilation and subclavian vein cannulation was done simultaneously. Naloxone 0.8 mg was injected intravenously and in 5 min BP rose to 130/70 mm Hg. After 12 min. the patients became fully conscious. Shortly after that spontaneous respiration (V_t 600 ml) enabled us to extubate the patient, It was the most striking improvement in BP, mental status other clinical parameters in our study. After 48 h patient was transferred to the Coronary Care Unit for further treatment.

Patient 4 (Fig. 3), a 61 year old female, referred to Casualty dept. with cardiac arrest and respiratory failure was given naloxone 0.4 mg subsequent to



	pH	pCO₂	pO₂	tot CO₂	HCO₃	BXs	sat O₂
9 pm	7·20	66·1	73	32	24	- 2	93 %
6 am	7·46	33·1	96	24	23	+ 0·5	97·5 %

Fig. 2 F.B. no. 000106 male 72 yrs.
RESPIRATORY FAILURE: MYOCARDIAL INFARCTION

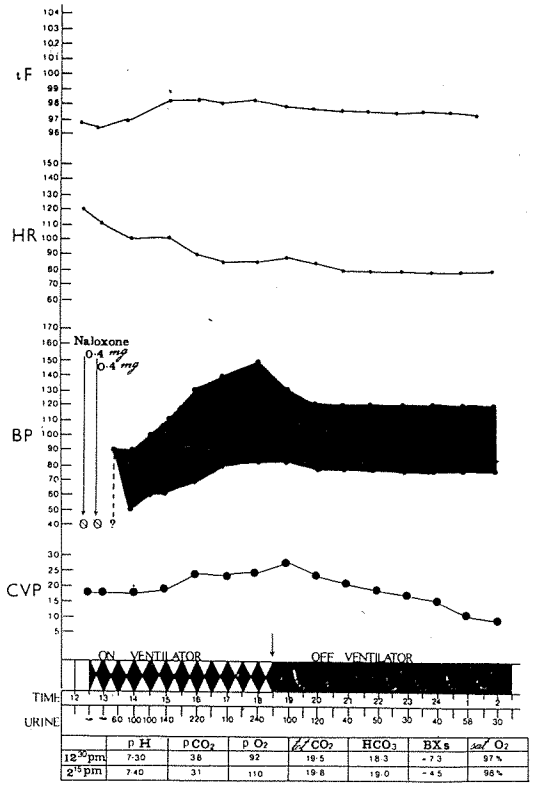


	p H	pCO₂	p O₂	tot CO₂	HCO₃	BXs	sat O₂
10 am	7·14	94	76·5	34	31·5	- 6	76·5 %
4 pm	7·50	29·8	77	24	23·2	+ 1·2	96·5 %

Fig. 3 H.E. No. 119765 female 61 yrs.
RESPIRATORY FAILURE: CARDIAC ARREST

emergency intubation. Systolic blood-pressure was unrecordable and following naloxone injection rose to 120/80 mm Hg. Forty minutes later BP fell to 80/60. Another injection of naloxone 0.4 mg increased the BP to 160/80 mm Hg. Forty minutes later BP fell to 80/60. Another injection of naloxone 0.4 mg increased the BP to 160/80 mm Hg. Later it was decided to give dopamine at a rate of 7μg/kg/min and blood-pressure was maintained at the level of 120/80 mm Hg. After 8 hours controlled ventilation was turned to assisted ventilation. Patient had only one lung due to pulmonectomy 21 yr before. Assisted ventilation was employed for seven days because of concomitant bronchopneumonia. Patient died after seven days in the operating theatre on the induction of anaesthesia for planned tracheostomy to facilitate bronchial toilet.

Patient 5 (Fig. 4) a 79 yr old female, transferred to ITU from the operating theatre after partial gastrectomy, partial pancreatic resection, cholecystojejunostomy. Systolic blood pressure was unrecordable. Naloxone 0.4 mg given intravenously did not increase the blood-pressure. In ten minutes another dose of



	p H	pCO_2	pO_2	$\not{C}CO_2$	HCO_3	BXs	$\not{S}O_2$
12^{30}pm	7·30	38	92	19·5	18·3	−7·3	97%
2^{15}pm	7·40	31	110	19·8	19·0	−4·5	98%

Fig. 4 L.M. No. 066849 female 79 yrs.

Dg. GASTRIC ULCFR; PSEUDOPANCREATIC CYST; PANCREATITIS

naloxone 0.4 mg increased the systolic blood-pressure to 90 mm Hg. Fifteen minutes later BP was 90/50 mm Hg and after one hour 110/70 mm Hg. The patient was initially unconscious and on controlled ventilation. Seven hours later she was on spontaneous respiration. Blood gases improved markedly. Hourly diuresis was 30–240 ml/h in the next 12 h following naloxone injection but CVP remained stable.

No corticosteroids or vasopressor drugs were given. After 72 h patient was transferred to the ward.

Discussion

Prolonged hypotension may lead to complex biochemical, cardiovascular and haemodynamic derangements thus making cellular perfusion inadequate for normal cellular function i.e. shock. Adequate fluid therapy plays a part in the compensation for some of these derangements. When renal function is impaired management becomes more complicated. Regaining an adequate cellular perfusion is the aim in the treatment of the shock state.

If all these derangements, including the CNS activity, are partly associated with endorphin release than it would be expected that naloxone could delay the physiologic homeostatic breakdown imminent in critically-ill patients. How exactly neurotransmitters act when the body is in an emergency state is still a medical enigma. The diverse localization of specific receptors for endogenous ligands in the body make it possible to postulate their probable role in stress and near-death situations. Naloxone has been used extensively in man and has had no adverse effects, even in high doses.[7,8]. Demonstrating that intravenous naloxone is effective in raising the systolic blood-pressure, improving the mental status, respiration (and secondary to this also diuresis and acid-base status) itsuse in critically-ill patients of different pathology may be justified.

Naloxone was effective in raising and maintaining the blood-pressure even when dopamine was stopped although Peters and others were giving naloxone concurrently with dopamin.

In three patients corticosteroids were given in high doses and naloxone did not promptly increase the systolic blood-pressure as it did in the other seven patients which were not injected with exogenous steroids. This is in agreement with the findings that exogenous steroids may supress endorphin release from the pituitary[2]. More clinical data are needed to elucidate the exact pharmacological mechanisms of naloxone in critically-ill patients and to define the criteria and exact indications for its clinical application.

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The use of Spinal Anaesthesia in Diabetic Patients

I. SMAILBEGOVIC, N. BOSKOVSKI, M. COSIC and S. JOOS.

Summary

The modifying effect of low spinal anaesthesia on hyperglycaemic and haemodynamic response to surgery in 22 insulin dependent diabetic patients (IDDM) was studied. No significant alterations from the preoperative valves of blood glucose levels, arterial blood pressure, and ECG pattern were noticed during surgery. These studies indicate the possible advantages of conducting surgery in diabetic patients under regional anaesthesia.

Key Words

Insulin dependent diabetic patients
Stress response to surgery.

Diabetes mellitus is a metabolic disorder caused by many environmental and genetic factors, usually acting mutually.[1] Surgery and trauma induce profound changes in endocrine function, characterized by an increase in the circulating concentrations of the catabolic hormones such as catecholamines, glucagon and cortisol and a concomitant decrease in plasma concentration of the anabolic hormones, insulin and testosterone.[2] The hyperglycaemia of surgery is the result of an increase in glucose production compared with the rate of utilization. This increase in blood glucose concentration is proportional to the severity of the surgical trauma.[3] According to a recent survey (W.H.O. 1981) the prevalence of diabetes mellitus in Malta is 7.7%. It has been concluded that diabetes mellitus is a major health problem in Malta.

It seemed appropriate to investigate and assess the effects of spinal anaesthesia on the perioperative glucose homeostasis in a population with a high prevalence of diabetes mellitus. This was the aim of our clinical study.

Patients and Methods

Twenty two insulin dependent diabetic patients (IDDM) with an ASA physical status classification II and III were given low spinal anaesthesia for lower limb surgery.

Premedication to all patients consisted of 5mg nitrazepam (Mogadon) orally at hour of sleep the night before surgery and 10 mg diazepam (Valium) administrated orally one hour before anaesthesia.

Table 1 summarises the surgical procedures and

TABLE 1. *Summarized kind of surgery, age and sex of the patients.*

No.	Kind of Surgery	Sex	Age
	Amputation of lower limb:		
1.	below knee	F	52
2.	"	M	83
3.	"	F	78
4.	"	F	56
5.	"	F	70
6.	above knee	M	60
7.	"	F	68
8.	"	F	64
9.	"	M	63
10.	"	F	71
11.	"	F	73
12.	"	F	74
13.	"	M	57
14.	"	F	75
15.	"	F	74
16.	"	M	81
17.	"	M	72
	Amputation of great toe and toes		
18.	"	F	84
19.	"	M	65
	Necrectomy of the foot and sole		
20.	"	F	59
21.	"	M	58
22.	"	F	61
Total 22		**Sex ratio F-M 14-8**	**Mean age: 68**

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patients data. Prior to the spinal anaesthesia all patients were infused with 500 ml of 1/2 Ringer lactate in 5% dextrose, with 8 i.u. plain Insulin, followed by 3ml kg^{-1} h^{-1} during surgery and in the first postoperative hour while patients were kept in the recovery room. In the ward patients were left free to take liquids orally.

Spinal anaesthesia: all patients were positioned in the lateral horizontal position. A midline approach was used to insert a spinal needle G 22 in the subarachnoid space and 3 ml 0.5% plain bupivacaine (Marcain) was then injected.

ECG was continuously monitored. All our patients preoperatively showed signs of some of the following: elevation of ST segment, left bundle branch block (LBBB), right bundle branch block (RBBB). Blood was drawn from an ante-cubital vein for determination of blood glucose (1) prior to the induction of spinal anaesthesia, (2) half an hour after commencing of surgery, (3) and one hour after surgery. Blood pressure was measured with an aneroid sphygmomanometer before induction of spinal anaesthesia and every five minutes thereafter during the surgery.

Results

No significant haemodynamic changes were recorded in any of the patients during the induction, surgery, and in the first hour following surgery. The maximum recorded fall in systolic blood pressure was approximately 4kPa (30 mm Hg).

Continuous ECG monitoring showed no significant alterations in the heart rate and the ECG patterns in all cases.

The values of blood glucose taken during the study period correlated well with preoperative levels.

Discussion

Pathophysiology of diabetes mellitus

Diabetes mellitus is a permanent disorder of glucose metabolism in which failure to use glucose properly leads to hyperglycaemia, glucosuria, hyperosmolarity, polyuria, and dehydration. Associated abnormal fat metabolism produces ketonaemia and ketonuria. Disordered protein synthesis and enhanced catabolism, is associated with muscle wasting.[4 5]

Glucose and free fatty acids are the primary and the immediate sources of energy for the body. In diabetes mellitus not only is carbohydrate metabolism seriously deranged, but simultaneously fatty acid synthesis is impaired. The diabetic relies, therefore, for his energy needs, on the metabolism of lipids, (stored or dietary) and so produces an excessive amount of ketone bodies. Ketosis and acidosis result. In the neutralization and excretion of these organic acids, potassium and sodium are lost. Some of the accumulated Acetyl-CoA is diverted to excess synthesis of cholesterol. Thus hypercholesterolaemia is a prominent feature of diabetes mellitus and associated with the grave consequences of accellerated atherosclerosis. Anabolic processes such as synthesis of glycogen, proteins and triglicerides are slowed while proteins and glycogen are catabolized for gluconeogenesis and energy. The impaired carbohydrate, fat and protein metabolism eventually involves all the endocrine glands but principally the anterior pituitary and adrenals. Growth hormone is necessary for metabolism of stored triglycerides but, unfortunately, the liberated fatty acids act as insulin inhibitors. Adrenal steroids are involved in gluconeogenesis. These metabolic alterations lead to pathologic changes in organs and tissues. At post mortem the pancreas in 90% of diabetics, shows hyaline (amyloid) degeneration, fibrosis, atrophy and lymphatytic infiltration of the islets. The liver of long-standing poorly controlled diabetics is often fatty and enlarged. The kidneys are usually the most severely damaged organs in the diabetic. Renal failure, usually due to renal microvascular disease, accounts for many of the diabetic deaths in both juveniles and adults. Any one or any combination of the following lesions may be found:

1) Glomerular involvment with three distinctive patterns: diffuse glomerulosclerosis, nodular glomerulosclerosis (Kimmelstiel-Wilson disease) and exudative lesions.
2) Arteriolosclerosis inducing so-called benign nephrosclerosis.
3) Pyelonephritis, sometimes with necrotizing papillitis.
4) Glycogen accumulation in the tubular cells and
5) Fatty change of the tubular cells.

One of the most threatening aspects of diabetes mellitus is the developoment of blindness as a consequence of retinopathy, cataract formation or glaucoma. Diabetic retinopathy is characterized by microangiopathy, exudates, proliferative changes and vitreous hemorrhages.

Diabetes mellitus is a significant cardiovascular risk factor, with higher levels of blood glucose directly related to the extent of vascular disease. This relationship is true for both the juvenile and adult onset diabetic, and on both groups premature microvascular disease is the major cause of death.[6]

Surgically-induced adverse hormonal and metabolic changes, if not prevented or rapidly treated may seriously harm the patient and prolong his convalescence. Modification of the neuroendocrine response to surgery may be attempted in two ways. This may be achieved either by afferent neuronal blockade with local anaesthetics, for example extradural or spinal anaesthesia, or by inhibition of hypothalamic function with large doses of opiates.[7 8]

Painful stimuli from the site of operation can be partly suppressed by profound anaesthesia but only drugs blocking the spinal cord itself or the afferent or efferent pathways in its neighbourhood can block it completely.[9 10]

In all our patients surgical analgesia was excellent. Haemodynamic stability was another feature during and after surgery in all our patients.

Absence of a significant hyperglycaemic response in our patients, we assume, was due to the afferent neuronal blockade.

Another beneficial effect of spinal anaesthesia in our patients was a prolonged postoperative pain-free period and a decreased requirement for opiate analgesia.

Our results would suggest that lower limb surgery under spinal anaesthesia is associated with mild changes in haemodynamic and metabolic response to surgery and could be a safer method of anaesthesia particularly in diabetic patients.

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The use of Thalamonal in Acute Myocardial Infarction

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Summary

Intravenous administration of narcotic analgesic drugs provides effective pain relief in patients with acute myocardial infarction. Thalamonal, a premix of 50:1 droperidol and fentanyl, was given intravenously in dose of 2 ml. to 20 patients with acute myocardial infaction. Effective pain relief, haemodynamic stability and low incidence of cardiac dysrhythmias were noticed in the patients studies.

Key words

Acute myocardial infarctionl; thalamonal

Pain relief should have high priority in the management of patients with acute myocardial infaction. Prompt and effective relife of pain serves to alleviate the patient's anxiety and therefore may limit catecholamine activity. There is experimental evidence that the persistent pain and associated between myocardial oxygen demand and oxygen supply and consequently increase the size of infarction and the risk of death[1].

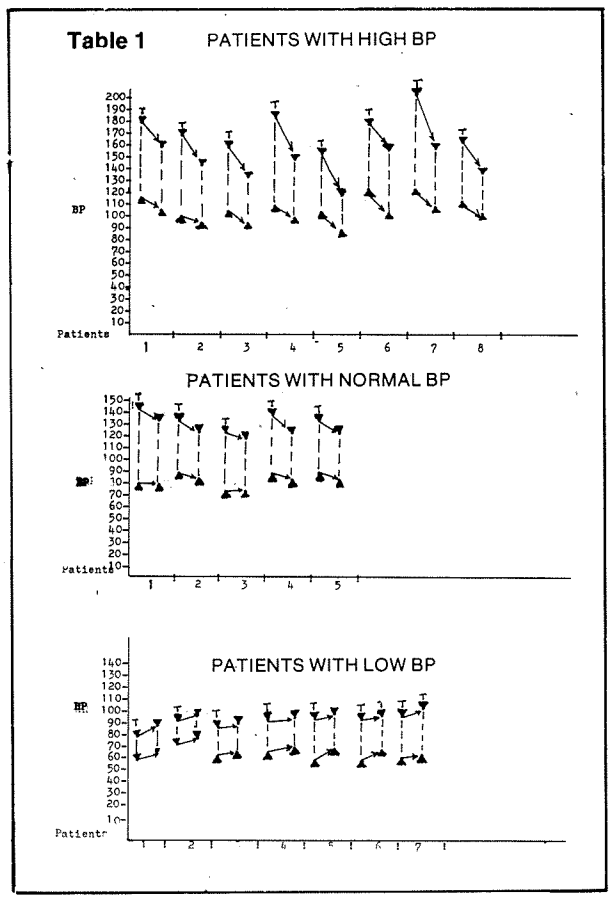
Sublingual nirtoglycerin may be employed for pain relief if the systolic blood pressure is higher than 100 mm Hg: (13,3 kPa). However it has been shown that this drug may reduce mean arterial blood pressure, with a resultant fall in coronary perfusion pressure, and cause a reflex tachycardia which may result in increased myocardial oxygen deman.[2] If pain is severe, morphine should be administered in small doses, 2-5 mg intravenously and repeated as required. Narcotic analgesics exert favourable haemodynamic effects by increasing venous capacitance, thereby reducing venous return, and by reducing systemic vascular resistance, hence diminishing the impedance to left ventricular emptying. The result of both effects is a reduction in myocardial oxygen demand.

This prompted the present study, in which the effect of thalamonal in the relief of pain and on haemodynamic stability were studied in a small group of patients with acute myocardial infarction.

Patients and Methods

Twenty consecutive patients with clinical and ECG signs of acute myocardial infarction were studied. Their ages ranged between 37 and 72 years (mean 54.5 years). 8 were female, 12 male. To each patient a dose of 2 ml Thalamonal was injected intravenously over a two minutes period.

Table 1



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A contraindication for giving Thalamonal was congestive heart failure.

Following the Thalamonal injection the relief of chest pain was assessed on the basis of patient's subjective pain ratings as have no, slight, moderate, or complete relief.

Arterial blood pressure and heart rate were frequently measured and charted.

Patients who did not have pain relief following the first injection were given repeated doses of 2 ml thalamonal 10-15 min apart.

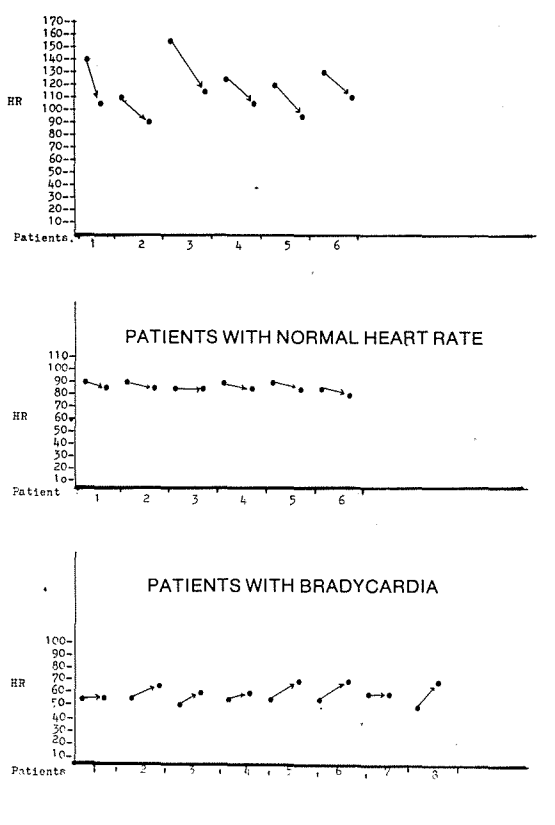
Results

Eighteen patients reported moderate to complete pain relief following the first Thalamonal injection. In two patients repeated doses of Thalamonal were required to provide adequate pain relief.

Table 1 shows the changes in the systolic and diastolic blood pressures following the thalamonal injection. Analysis of these results indicates that Thalamonal has beneficial haemodynamic effects.

The effect of Thalamonal on heart rate (HR) is shown on Table 2.

Table 2



Discussion

Following the introduction of Coronary Care Units (CCU) in the early sixties, the in-hospital mortality in patients with myocardial infarction (MI) has fallen from 30-40% to 10-20%. This reduction is due predominantly to vigorous treatment of ventricular dysrhythmias. Monitoring of disrhythmias is of prime importance in patients with MI and in uncomplicated cases is usually required only for the first 24-48 hours. In all but one of our patients serious disrhythmias were absent following Thalamonal injection. It has been suggested that the use of thalamonal is associated with lowered cathecholamine levels in plasma[7] and a decrease in peripheral vascular resistance[5], which contributed in preventing serious dysrhythmias in our patients.

Thalamonal acts on brain stem structures and also possess an antiemetic effect[6]. Its alpha-adrenergic blocking properties[4] could have been partly responsible for the beneficial haemodynamic response in our patients.

It has been indicated, from posmoretem findings, that when 40% or more of the left ventricule is destroyed death is inevitable. Hence, therapeutic approaches must be directed towards reducing the infarct size.

Control of pain is one of the most important aspects of therapy and should be achieved promptly with adequate doses of effective drugs. The pain of infarction is often short-lived and slow IV injection of Thalamonal which is a potent narcotic analgesic drug, has been effective in alleviating the pain in our patients.

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Current Trends and Developments in the Field of Local Anaesthetics

NIKOLA BOSKOVSKI

Summary

Local anaesthetic agents belong to a surprisingly homogenous family of drugs that are able temporarily to interrupt impulse trnasmission along nerves in a relatively predictable and reversible manner.

During the past hundred years, more efficacious, safer, and varied types of local anaesthetic agents have been developed. Since the mechanisms by which these drugs block impulse conduction are becoming better understood, many responses to nerve block can be readily explained in physiologic terms[4]. Such basic understanding is important to the clinician, especially when complications arise or his block is only partly successful. The purpose of this paper is to summarize current knowledge regarding the areas of drug development, basic mechanism of local anaesthesia, clinical pharmacology of local anaesthetic drugs, and comparative effects of regional and general anaesthesia. It is emphasized that the bibliography is not a complete list of the vast literature but merely intends to list major reviews on the subject.

Key Words
Local anaesthetics
Current trends

Areas of Drug Development

There is no doubt that the local anaesthetic drugs currently available are highly effective for the majority of surgical and obstetrical procedures in which regional anaesthesia is indicated.

The situation in regard to postoperative pain alleviation is, however, much less satisfactory[2]. There is no drug or preparation that can provide ultra-long duration of analgesia, not for several hours but, preferably for the first 24-28 hours. Such an agent should selectively block sensory fibres with no or minimal motor blockade and sparing autonomic function. The pelvic parasympatheic control of bladder and sphincter function should be particularly retained.

So far, inspite of considerable efforts, minimal success has been achieved in developing a clinically useful, ultra-long-acting local anaesthetic preparation.

Attempts to prolong the action of procaine by the use of peanut-oil vehicle, which would provide a depot type preparation, failed because of its unacceptable neurotoxicity.

Dextran has been utilized as vehicle for local anaesthetic drugs in order to produce a depot-type, slow releasing anaesthetic preparation. It has been shown that dextran could extend the duration of conventional local anaesthetics by a few hours although conflicting data exist.[3,4]

In recent years two interesting approaches have been pursued. In early 70's the concept of cyclizing compounds was introduced.[5] The idea was that a tertiary amine compound would be injected and than taken up by the nerve membrane, where it would be converted to a quaternary-ammonium agent which could not readily diffuse out of the membrane, providing long duration of anaesthetic activity. Although preliminary results in animals were encouraging, no suitable agent was developed for human trials.

Another hope for an ultra-long-lasting local anaesthetic agent was sought in the biotoxins i.e. tetradotoxin and saxitoxin.[6] Unfortunately, the very high systemic toxicity of tetradotoxin and saxitoxin and their unreliability in terms of predictable anaesthetic duration limit their potential usefulness. The biotoxins do not penetrate neural sheaths easily, so that results of peripheral nerve blocks in animals were not satisfactory. However, spinal anaesthesia of 24 hours duration was obtained in sheep with tetradotoxin, where no neural barrier is present to obstruct the diffusion of the biotoxins to the receptor site in the nerve membrane. Currently, efforts are focused to modify the structure of tetradotoxin or

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saxitoxin in order to provide acceptable agents of ultra-long duration.

At the present time, the use of epidural and intrathecal opiates in prolonged pain relief have opened up a new area of applied neuraxial pharmacology, not only for narcotics, but for adrenergic agonists as well. The analgesic properties of intraspinal narcotics are uniquely powerful, since they appear to act mainly on primary afferent nocioceptive synapses in the dorsal horn.[7] Although intraspinaly applied narcotics offer the greatest promise for prolonged pain relief at present, they are not free of side-effects. Delayed respiratory depression and urinary retention are the main danger, but both are treatable by i.v. naloxone

Most recently Scurlock & Curtis (1981) have opened a new area of promise by using derivatives of tetraethyl-ammonium.[8] A phenomenal duration of sensory analgesia of approximately 400 hours (16 days) was observed following infraorbital nerve block when compounds with a chain length of C-12 or longer were used in rats. Studies of the mode of action suggest that the ultra-long duration of action of these agents is produced by binding to a receptor site in the potassium channel[9]. Important features include reversibility of the blocks and absence of persistent neurotoxicity.

Further studies are required to establish the ultimate efficacy of these tetraethyl-ammonium compounds.

The concept of temporarily blocking the sodium and potassium transport at the membrane channels to produce ultra-long selective sensory analgesia should provide the basis for developing new agents that might fulfill the clinical criteria for use in control of chronic pain.

Basic Mechanism of Local Anaesthesia

In terms of impulse transmitting properties, the membrane is the most important part of the nerve fibre.

Local anaesthetic drugs block a nerve without damaging it and are unique in that their action is reversible.

The main pharmacological action of local anaesthetics is by interfering with the impulse conducting properties of the nerve membrane.[10]

Conclusive evidence indicates that local anaesthetic agents act at the sodium channel of the nerve membrane, probably by physically occluding the transmembrane sodium channels.[11] A local anaesthetic block is a nondepolarization block, resembling in some ways the action of curare at the neuromuscular junction. It is suggested that with conventional agents, such as lidocaine, binding occurs at the receptor sites located on the inner surface of the nerve membrane. In contrast, biotoxins, such as tetradotoxin and saxitoxin, act at receptor sites located on the external surface on the membrane.

Agents such as benzocaine and benzyl alcohol act by penetrating the nerve membrane, causing membrane expansion and subsequently diminish the diameter of the sodium channel[12].

The current concept of the mechanism of action of local anaesthetic drugs is based on the following sequences: binding of local anaesthetic molecules to receptor sites in the nerve membrane; inhibition of sodium permeability; reduction in the rate of depolarization; inabilty to achieve threshold potential level; lack of development of a propagated action potential; and block of nerve conductivity.

Clinical Pharmacology of Local Anaesthetic Drugs

Thorough knowledge of the clinical pharmacology of local anaesthetics enables the performance of safe and effective regional block.

Currently, the choice of agents is wide, and it is now possible to select a drug that will suit the particular regional block technique in each individual case.

Basically, all local anaesthetic agents have the following chemical structure; aromatic portion, intermediate chain and an amine portion.

There are two major groups of local anaesthetics: amides and esters. Procaine, chlorprocaine, and tetracaine represent the ester group while the amide group includes mepivacaine, bupivacaine and etidocaine. The esters are rapidly hydrolysed in the plasma, whereas the breakdown of amides depends on hepatic metabolism.

Another significant difference between the ester and amide compounds is their allergic potential. Para-aminobenzoic acid is one of the metabolites formed from the hydrolysis of ester-type agents. This substance has a potential of inducing allergic-type reactions in a small percentage of the general population.

Allergic phenomena with amide group compounds are extremely rare.

All local anaesthetics have certain common characteristics but their anaesthetic profile is determined by their: lipid solubility, protein-binding, pK_a, non-nervous tissue diffusibility, and intrinsic vasodilator activity.

The lipid solubility of a particular anaesthetic drug appears to be a primary determinant of its anaesthetic potency. Procaine has a low lupid solubility as determined by partition coefficient measurements, and this drug is least potent in supressing conduction in an isolated nerve.

The partition coefficient of bupivacaine,

tetracaine, and etidocaine vary from about 30 to 140, indicating a high degree of lipid solubility and a high potency in supressing conduction in an isolated nerve. Indeed these drugs block nerve conduction at very low concentrations.[13]

It has been shown that approximately 90% of the axolemma consists of lipids, which is in accord with the relationship to lipid solubility. The anaesthetic agents which are highly lipid-soluble penetrate the nerve membrane more easily and this is reflected biologically in increased potency.

The duration of action of a local anaesthetic agent is primarily dependent on its protein-binding slightly faster onset of anaesthesia *in vivo*. The factors tetracaine, and etidocaine are highly bound to proteins and possess a relatively long duration of action.

The relationship between protein-binding of local anaesthetic drugs and their duration of action is consistent with the basic structure of the axolemma. Proteins account for approximately 10% of the nerve membrane. Thus, local anaesthetics which penetrate the axolemma and attach more firmly to the membrane proteins will tend to possess a prolonged duration of action.

The onset of anaesthesia is directly related to the rate of epineural diffusion which, in turn, is correlated with the amount of drug in the base form.

The pK_a values of the common local anaesthetic drugs are all greater than the physiological pH value, which means that the drugs will exist in the body predominantly in the ionized form. The percentage of a specific local anaesthetic drug which is present in the base form when injected into tissue whose pH is 7.4 is inversely proportional to the pK_a of that agent. For example, lidocaine, which has pK_a of 7.74 is 65% ionized and 35% nonionized at a tissue pH of 7.4. On the other hand, tetracaine, with a pK_a of 8.6, is 95% ionized and only 5% non-ionized at a tissue pH of 7.4. Both, *in vitro* and *in vivo* studies have confirmed that local anaesthetic drugs such as lidocaine, whose pK_a is closer to tissue pH, have a more rapid onset time than agents with high pK_a, such as tetracaine. In an isolated nerve, onset time is a function of the rate of diffusion of a compound through the epineruium which, in turn, is related to percentage of drug in the base form. However, *in vivo*, a local anaesthetic must diffuse initially through non-nervous connective tissue barriers. Naturally, differences exist between the rate of non-nervous tissue diffusion for various agents. For example, procaine and chlorprocaine have similar pK_a's of 9.1 and similar onset times for conduction blockade in an isolated nerve. However, *in vivo*, the onset of anaesthesia for chloroprocaine is significantly shorter than that of procaine, which is indicative of a more rapid rate of non-nervous tissue diffusibility. Similarly, in the amide group, lidocaine and prilocaine possess the same pK_a and onset of action in isolated nerves, whereas lidocaine has a slightly faster onset of anaesthesia in vivo. The factors that determine diffusibility through non-nervous tissue are still unclear.

Intrinsic vasodilator activity of different local anaesthetic drugs could significantly influence their potency and duration of action *in vivo*. The degree and duration of nerve block is related to the amount of local anaesthetic drug which diffuses to the receptor site at the nerve membrane. Following injection of a local anaesthetic drug, some of the drug will be taken up by the nerve and some will be absorbed by the vascular system. The latter is related to the degree of blood flow through the area in which the drug is depositied. All local anaesthetic drugs, except cocaine, possess vasodilator properties. However, the degree of vasodilation produced by the various drugs differs. Studies *in vitro* have shown that the intrinsic anaesthetic potency of lidocaine is significantly greater than that of mepivacaine, while their durations of action are similar. On the other hand, *in vivo*, mepivacaine is similar in potency and produces a longer duration of anaesthesia than lidocaine. These differences between *in vitro* and *in vivo* results are probably related to the greater vascular absorption of lidocaine such that less is available for nerve blockade.

In summary, on the basis of differences in anaesthetic potency and duration of action, it is possible to classify the local anaesthetic agents into three groups: 1. Local anaesthetic drugs of high anaesthetic potency and long duration of action, i.e. tetracaine, bupivacaine and etidocaine; 2. Local anaesthetic drugs of intermediate potency and duration of action, i.e. lidocaine, mepivacaine, and prilocaine; 3. Local anaesthetic drugs of low potency and short duration of action, i.e. procaine and chloroprocaine.

Comparative Effects of Regional and General Anaesthesia

Our main task, as practising anaesthetists, is the safety of our patients.

The metabolic consequence of the neuro-endocrine response to surgical trauma are characterized by the following triad: increased resting energy expenditure, negative nitrogen balance, and altered glucose homeostasis.

Recent interest has been focused on the influence of different anaesthetic techniques in modifying the endocrine-metabolic response to surgery.

Considerable number of studies have been published in an attempt to determine the relative

advantages of general and regional anaesthesia in suppressing the surgical stress.

These studies generally fall into four categories: 1. haemodynamic effects; 2. metabolic effects of general vs. regional anaesthesia; 3. postoperative convalescence; and 4. morbidity and mortality studies.

Haemodynamic effects

The initial differences observed in the haemodynamic changes are those of a significantly greater fall in systolic, diastolic and mean arterial pressure in subjects receiving epidural anaesthesia as compared to those undergoing general anaesthesia.[15] The fall in blood pressure during epidural blockade is due almost entirely to decrease in systemic vascular resistance.

However, a significantly greater decrease in cardiac output and stroke volume is observed in those patients receiving general anaesthesia with halothane.

Supplementing epidural anaesthesia with light general anaesthesia usually results in a somewhat greater fall in systolic, diastolic, and mean arterial pressure. However, cardiac output and stroke volume remain basically unchanged, so that the fall in blood pressure is due mainly to the fall in systemic vascular resistance.

Metabolic effects of general vs regional anaesthesia

Surgical trauma evokes a neuro-endocrine response which results in substrate mobilization, a change in metabolism towards catabolism with a negative nitrogen balance and retention of salt and water[16]. A number of studies indicate that the metabolic response to surgery seems to be obtuned to a greater degree by regional anaesthesia as compared to general anaesthesia.

Postoperative convalescence

There are several studies pointing at the benefits of regional anaesthesia on postoperative organ function, especially pulmonary and cardiac. The use of regional anaesthesia is associated with decreased requirements for postoperative opiate analgesia, imporved pulmonary function, earlier ambulation, and earlier hospital discharge than the patients receiving general anaesthesia plus postoperative opiates.[17]

Morbidity and mortality studies

There are only few reports concerning the relative morbidity and mortality of regional vs general anaesthesia on surgical patients. It appears that incidence of thrombo embolism is lower following surgery under epidural anaesthesia as compared to general anaesthesia. It has been also shown that blood loss following total hip replacements in patients under epidural anaesthesia is significantly reduced as compared to those patients under general anaesthesia.[18]

However, further investigations should be undertaken to determine the relative advantages of regional vs general anaesthesia.

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Preoperative Evaluation of Electrolyte Status

J. SEDLACEK and N. AZZOPARDI

Summary

The metabolism of Na^+, K^+ and Cl^- and its disturbances described. The importance of monitoring the balance of thse electrolytes stressed. When treating the patient with electrolyte infusions calculation of the amounts given with respect to osmotic state, acid-base balance and the patient's metabolic situation have to be performed.

Key Words
Preoperative evaluation;
Electrolyte status.

It is the anaesthetist's task to prepare the patient for the operation under the best conditions. Preoperative preparation may include many important investigations, but in this paper we look only at the electrolytes Na , K and Cl. . Estimation of these ions is a part of routine examination. We want briefly to draw the attention to some aspects of their evaluation.

One may be misled in trying to evaluate electrolyte concentration if the total solvent volume is not considered:

Total amount of ions = vol. x conc.

Because the total body Na^+, K^+ and Cl^- are difficult to estimate, we normally have to rely on their levels in the plasma.

	Na^+	K^+	Cl
Plasma concentration	138 ± 7mmol/l	4.5 ± 0.7 mmol/l	101 ± 5 mmol/l
Intracellular conc.	3-35 mmol/l	110-150 mmol/l	appr. 30 mmol/l
Total amount in EC fluid	2000 mmol	60 mmol	1400 mmol
Total amount in IC fluid	1200 mmol	3200 mmol	1000 mmol
Daily turn over	140-260 mmol/ 24hrs	50-100 mmol/ 24hrs	140-260 mmol/ 24hrs
Renal excretion	120-240 mmol/ 24hrs	45-90 mmol/ 24hrs	120-240 mmol/ 24hrs

To enable estimation of concentrations and total amounts of these electrolytes we have to measure the intake from diet and/or infusion therapy and the output in the urine and from other body fluids. The normal composition of some of the body fluids is shown in table 1.

The electrolyte levels before the operation should be normalised as much as possible. Special note should be made of an existing catabolic state which may sometimes be present for a long time before operation and may persist after the operation for at least 3 - 7 days. During catabolism 300g of muscle tissue on average is broken down daily. But following injury, operation etc., the loss of tissue can amount to 20 - 30g of nitrogen, or a loss of 1000g of body tissue daily. Even if the weight of the patient is checked daily, it may not be possible to discover this loss immediately because the kidneys are not able to eliminate sufficiently rapidly the overwhelming amount of catabolites. The local oedema due to local hyperosmolality e.g. in the operating field area may mask the muscle tissue break down. The oxidation of broken down tissues leads to the formation of water and this increases its normal daily production from 330 - 380 cc to 600 - 1000cc. From 300g of tissue another 200cc of water is released which had been previously bound in intracellular structures. There is an increased excretion of urea if renal function is normal, but sometimes the urea level in plasma increases in spite of this.

The destruction of cellular proteins leads to release and loss of K from the intracellular space. For every gram of nitrogen lost there is a loss of 2,0 - 3,0 mmol K^+ but this increases fivefold during the postoperative period. However if the urine output is higher than 300cc/24 hrs no dangerous increase in potassium level of the plasma occurs.

The energy consumption of a healthy man is 1500-6000Kcal/24hrs (6300-25100KJ/24hrs). The requirements in operated patients are about 2000-8000Kcal/24hrs (8000-33500KJ/24hrs). This need for energy cannot be satisfied by the infusions of 5% Dextrose frequently used postoperatively! This impaired energy supply situation has a great influence

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on the metabolism of electrolytes, especially on potassium.

Sodium is the most important cation in plasma as it regulates the osmotic pressure. The normal osmolality in adults is 285 ± 10 mOsm /l. We can calculate the osmolality approximately from the relation:

Plasma Osmolality in mOsm/l = 1.86 (Na + K conc. in mmol/l) + glucose conc. + urea conc. in mmol/l

But patients in poor physical condition show large discrepancies between their real and estimated values for plasma osmolality.

Hypernatraemia of more than 145 mmol/l can appear if there is: 1. An absolute lack of water (decreased volume of total body fluid). In this situation one simultaneously finds increased haemoglobin and total protein values. The weight of the patient falls and urine hyperosmolality follows. Na^+ values higher than 155mmol/l result in encephalopathy.
2. Relative lack of water: Here one finds a normal haemoglobin level, normal total protein and body weight. Signs of extracellular fluid retention (oedemas) are usually present.
3. Absolute and relative lack of water, where the loss of water is higher than that of sodium.

Increased sodium stores lead to an increase of total body water. This happens after excessive salt intake (NaCl, NaHCO3) or after therapy with sodium salts of some antibiotics (2) following decreased elimination from the kidneys (3) in pituitary hyperfunction (due to ACTH), in hyperaldosteronism, after steroid treatment, diseases leading to oedemas and eclampsia.

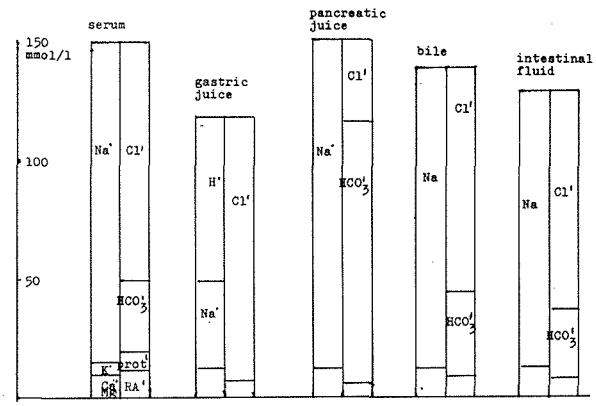
A hyponatraemia lower than 130 mmol/l is usually accompanied by hypoosmolality if there is no simultaneous hyper-glycaemia and/or uraemia. Water intoxication can occur and is manifested by cramps if the sodium decrease happens rapidly. In chronic diseases the sodium falls slowly, parallel to the fall in protein resulting from reduced intake. The body becomes accustomed to the new lower osmolality and any increase in intake of sodium can cause oedema, an increased extracellular space and may lead to circulation overload. Therefore the catabolic state has to be corrected before sodium replacement.

Decreased stores of sodium lead to decreased amount of total body fluid and it can be seen mostly in old cachectic patients. The severe state of these patients is often in contrast with their "normal" values of Na, total protein and haemoglobin. When therapy is begun all these values initially fall and this may falsely give a picture of overloading, but is in fact the result of dilution and the opening of tissue spaces. The loss of Na^+ is accompanied with a loss of anions. Their relation in plasma is:

Na:Cl = 138:101 = 1.37

If the concentration of Na^+ and Cl^- in the urine is in the ranges 1,28 - 1,42 the acid-base balance is not influenced. If the chloride output is lower the body fluids become acidotic and if higher alkalotic.

● table 1.



The relationship $Na^+ : K^+$ concentration in serum is 138 : 4,5 = 30 : 1. In urine it is usually 2 : 1. The renal clearance of Na or K manifest the intensity of the electrolyte elimination by the kidneys. The value of "excretion fraction" (EF) is the amount of the eliminated ions in percentage of its filtrated amount. The renal clearance of Na or K can be calculated from the formula:

$$C_x = \frac{U_x \times V}{P_x}$$

C = clearance, U = concentration in urine in mmol/l, V = amount of urine during 1 min., P = concentration in plasma in mmol/l X = Na or K ion
the value of C_{Na} is 0.5 - 1,0 ml/min. and C_K is 5-15 ml/min.
Excretion fraction of sodium can be calculated from the formula:

$$EF_{Na} = \frac{C_{Na} \times 100}{C_{Cr}}$$

C_{Cr} = clearance of creatinine in ml/min. EF_{Na} is usually in ranges 0,40 - 1,25% and EF_K is 4,0 - 19%
The resorption fraction RF is:

$$RF_X = 100 - EF_X \qquad \text{where X = Na or K}$$

RF_{Na} is 98,75 - 99,6%, RF_K is 81 - 96% normally

From the formula we can see that the value for the excretion fraction can be calculated by means of the concentration indices of electrolyte and endogenous creatinine even without the knowledge of urine output:

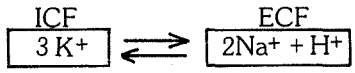
$$EF_{Na} = \frac{\frac{U_{Na}}{P_{Na}} \times 100}{\frac{U_{Cr}}{P_{Cr}}}$$

U_{Cr} = urinary level of creatnine
P_{Cr} = plasma level of creatinine

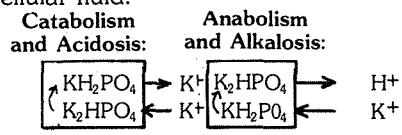
The values of resorption fractions give us the effective resorption of these electrolytes as a percentage of their filtered amounts. Monitoring of these values enables us to check very quickly the elimination of both electrolytes and renal function.

A check of sodium balance may demonstrate sodium retention which leads to an increase of the extracellular space, which can be followed by circulation overload. On the other hand an increased sodium loss leads to a decrease in extra-cellular fluid volume which can be followed by cirulatory and renal insufficiency. Large swings in sodium levels can lead to disturbances in acid-base balance. An evaluation of the ratio of sodium to protein concentration (especially albumin) in plasma gives an indication of the osmotic and oncotic pressures in blood capillaries.

Potassium is the main intracellular cation and is important in maintaining intra cellular osmotic pressure. Its presence in the cells is directly related to the metabolic status of the cell. During catabolism potassium is released from the cells and the cells let in passively sodium and protons:



The decrease of K^+ in the cells could be estimated easily from erythrocytes studies. However the potassium content in these cells is lower than in other tissues. Diabetes, injuries and cachexia are the most common examples of decreased cell potassium levels. The binding capacity for K^+ is dependent especially on hydrogen and phosphate ions. During acidosis there is a shift K_2PO_4 to KH_2PO_4. Therefore in acidosis potassium is released from the cells into the extracellular fluid:



In a similar way potassium is released from the protein bonds. Protein exchanges K^+ for H^+, the K^+ being freed to pass into the ECF.

In the first phase of catabolism there is an increased potassium elimination in the urine from the raised plasma potassium. The plasma potassium level is decreased when the supplies of K^+ in the cells become significantly lowered. On the contrary, anabolism increases not only glycogen supplies but also potassium levels inside the cells. The high concentration gradient for potassium across the cell membrane helps the transpot of K^+ to the extracellular fluid. Vice versa passage of K^+ against the concentration gradients i.e. into the cell, needs energy (sodium pump). This means therefore that the administration of K^+ ions only, by infusion cannot solve a severe decrease of K^+ in the cells as it is only eliminated from the extracellular fluid in urine without passing into the cells. For the good function of the sodium pump which is clearing Na out of the cells and transporting K^+ into the cells the following functions are needed: 1. anabolism 2. energy intake (Dextrose), 3. its utilisation (insulin), 4. potassium intake and 5. increased protein intake. The function of the sodium pump is dependent on the intact cell membrane, on the functioning enzymatic system and on the correct hormonal regulation (hypothalamus, pituitary gland and adrenals).

Hyporkalemia means values of K^+ higher than 5,2 mmol/l. The level of potassium concentration depends on the plasma pH:

pH	level of plasma K conc.
7,1	6,0 ± 0,5 mmol/l
7,3	5,2 ± 0,5 mmol/l
7,4	4,5 ± 0,7 mmol/l
7,5	3,8 ± 0,6 mmol/l

The potassium values obtained while an infusion is still running are often incorrect and therefore such studies are useful only for checking relative changes in the plasma level. The correct value of K^+ can only be obtained 30 min. after the drip has finished.

Hyporkalemia means plasma K^+ lower than 3,8 mmol/l.

The K^+ plasma fall can be very quick and dangerous especially 1. on dextrose intake with insulin during treatment of diabetic ketoacidosis, 2. rapid improvement of chronic hypoxia after inhalation of oxygen, 3. external losses (bleeding, burns) 4. high fluid intake without K^+, etc.

Potassium stores are decreased by catabolism and diuretics. Low potassium level and therefore impaired K^+ gradient across the cell membrane leads to weakness, tiredness, apathy and loss of appetite and paralytic ileus. The cardiac output is decreased and tachycardia with ectopics, atrial fibrilation and impaired conductivity occur. If hypokalaemia is prolonged the microscopic picture shows myocardial necrosis. Digoxin treatment is not effective and makes the arrythmia worse. Therefore during changes from catabolism to anabolism the patients have to receive infusions containing K^+ with frequent checks of their K^+ plasma concentration and the K^+ balance. As long as the K^+ intake is higher than the output (i.e. retention of K^+) the K^+ treatment has to be continued.

The amount of K which should be given to the patient can approximately be calculated from the formula:

mmol K = extracellular volume x (4,5 - plasma level) x 3 + potassium loss during 24 hours

When the improvement of the patients condition is apparent and the urinary output of K^+ is rapidly increasing lower doses can be given.

Potassium treatment is contraindicated during renal insufficiency. The highest concentration in the infusion should be 40 mmol/l, i.e. 3g/1 litre and the maximum rate the infusion should run is 20 mmol/l hr. The potassium losses should be checked especially at the beginning of the treatment because even relatively large deficits (e.g. 30%), the K^+ excretion may not differ too much from the normal valued and plasma K^+ levels have thus to be checked.

Chloride is the main extracellular anion maintaining the acid/base balance and osmotic pressure. It is present in high concentration in gastric juice and less in intestinal fluid. Chlorides are antagonised by bicarbonate ions. Severe vomiting leads usually to alkalosis which decreases the activity of the respiratory centre. The pCO_2 rises and the hypoventilation makes the hypoxia worse. Combined Cl^-, Na^+ and K^+ losses can lead to paralytic ileus. If the chloride level in serum decreases below 96 mmol/l we have to check the output in urine and it is usually very low in these cases and replecement is called for.

Physiological relation is: $(Na + K) : Cl = 1{,}41$

Sometimes we have to monitor also the so called anion gap:

$$(Na + K) - (Cl + HCO^3) = 10 - 18 \text{ mmol/l}$$

Pathological increase of this anion gap occurs during uraemia, diabetic ketoacidosis, fasting, severe dehydration, after diuresis and steroid therapy.

Decreased anion gap may occur during unbalanced i.v. infusion. Monitoring of the anion gap is a useful paramenter in esixtent combined chloropenic metabolic alkalosis with metabolic acidosis due to fasting or hypoxia. This combination of disturbances cannot be detected otherwise.

Hyperchloraemia is present in similar conditions as in hypernatraemia and include severe dehydration after 36 - 38 hours duration (while in children after 12 24 hours), increased chloride intake in renal diseases (sodium is eliminated sufficiently, but chlorides which are in a relatively higher amount of Na^+ are not. Also production of NH_4 and H^+ ions in renal tubuli is decreased), in hypochloraemic acidosis after brain injury (stimulation or damage of hypothalamus), (after a high dose of steroids (by higher losses of Na^+ relative to Cl^+, (severe diarrhoea or intestinal fistula, excessive reabsorption of Cl^-, after ureterocolic anastomisis and (respiratory alkalosis).

Increased stores of chloride are found when the volume of extracellular fluid is increased or in chronic catabolic states. The correction of this hyperchloraemia is harmful. During disturbances of renal excretion, hypothalamic disturbance and after excessive treatment with isotonic saline solution, the stores of Cl^- are increased too.

Decreased stores of chlorides are present after treatment with diuretics, after vomiting, sever sweating, in extracellular fluid deficit, in severe catabolism and in adrenal insufficiency.

Acknowledgements

We would like to express our thanks to Dr. V. Holecek CSc for his assistance and his permission to use the tables from his book, and to Dr. M. Falzon for his help in writing this script.

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Brain Death

D. TALAR and N. BOSKOVSKI

Key Words
Brain Death;
Diagnosis of Brain Stem Death.

The problem of determining death has been fascinating humanity through the ages. Specific criteria have been changing with development of medical science and practice.[1]

In the eighteenth century, for example, the only certain sign of death was putrefaction! Until quite recently death was determined by the cessation of heart-beat and respiration. The invention of the mechanical lung ventilator, in the early fifties, radically changed the treatment of critically-ill patients. In cases of brain-damaged patients this only served to prolong the process of dying and to complicate the medico-legal definition of death. Most medical and legal authorities today identify death with brain death, in particular brain stem death.[2] Damage to cerebral cortex caused by hypoxia after cardiac arrest make the patient unconscious and unresponsive, but leaves the brain stem reflexes intact. This clinical condition is refered to as "appalic syndrome", "social death", or "persistent vegetative state".

It is also evident that co-ordinated reflex activity in the spinal cord can persist long after all the brain stem reflexes have disappeared.

For these reasons the modern concept of brain death focuses on the brain stem. Jennett's (1981)[1] concept "if the brain stem is dead, the brain is dead, and if the brain is dead, the person is dead" would seem to gain widespread acceptence.

There is accumulated evidence that it is the death of the brain stem which indicates that the process of death has passed the point of irreversibility.[2]

Causes, circumstances and frequency of brain death

In the following three groups of patients extreme care should be taken before brain death can be safely declared: 1. Patients poisoned by barbiturates, sedatives, narcotics, carbon monoxide, alcohol, and other CNS depressants; 2. patients with acute CNS infection and other intracranial conditions, such as tumour or abscess; 3. infants and small children.

Such patients can frequently recover following prolonged and appropriate treatment. Caution is also required to exclude other reversible causes of brain stem depression such as hypothermia and gross metabolic imbalance.

About 4000 diagnoses of brain death are made annually in Britain.[3,4] More than half of all cases of brain death are a result of head injury with obvious structural brain damage. Spontaneous intracranial haemorrhage accounts for another one-third of cases The remaining cases are usually patients in whom treatment for other intracranial conditions such as brain abscess or tumour, has failed or patients who have not responded to resuscitation after cardiac arrest or profound hypotension.

Occasionally, persons may be found unconscious with no witnesses able to explain what happened and in spite of an obvious head injury it is prudent to wait long enough to allow exclusion of drugs as the cause of brain stem depression.

Criteria for diagnosis of brain death

According to the most recent, so-called "U.K. criteria", the time of death is when is when brain death is diagnosed, not some time later when the heart stops.

The Harvard criteria require absence of all reflex motor activity which implies death of the whole central nervous system: cortex, brain stem and spinal cord.

It is now agreed that permanent functional death of the brain stem constitutes brain death.[2] It implies that brain stem death would always be followed by cessation of the heart-beat. This concept does not redefine death but only changes the criteria by which death can be identified. The "U.K. criteria" have two

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components: the pre-conditions and the tests. It has to be emphasized that only when the pre-conditions have been satisfied is it appropriate to apply the tests.[1]

The pre-conditions require that the patient is: 1. in coma; 2. apneic; 3. has suffered irremediable structural damage of the brain; and that 4. reversible causes of brain stem depression have been excluded.

The tests are aimed to determine if there is any evidence at all of residual or returning activity in the brain stem. The following reflex responses are tested: response of the pupils to light; of the eyelids to corneal touching; responses of the facial muscles to painful stimuli applied to the face and forehead; responses of the muscles of the throat to movement of the endotracheal tube; injecting 20 ml of ice-cold water into the external meatus of the ears and observing for eye movements. If any of these responses is present, than the brain stem is still functioning.

The most important test, however, is to confirm that there is no return of respiratory drive from the brain stem. The patient is disconnected from the ventilator for long enough i.e. from 3 to 10 minutes, to ensure a threshold value of Pa co_2 (c 8 kPa), whilst maintaining diffusion oxygenation to avoid hupoxaemia. This is the apnoea test. An important safeguard against a false-positive diagnosis is to permit sufficient time to elapse.

Provided enough time has passed to satisfy the pre-conditions, the tests can be repeated after an interval of 30 min. Important safeguards include also, the two following tests: angiography and monitoring the electrical activity of the cerebral cortex by EEG. If absence of blood flow to the brain for 15 min can be recorded it is considered that the brain is dead, but because of the equipment and expertise involved, angiography is regarded as impractical test for general use. The E.E.G. assesses function in the cerebral cortex whilst brain death dependes on loss of function in the brain stem. There are quite a number of reports of patients recovering from deep coma whose E.E.G was at one stage iso-electric[4]. This is the strongest argument against its use and "U.K. criteria" explicitly state that E.E.G. is not needed for the diagnosis of brain death.

There are another three tests for diagnosis of brain death which are worth mentioning.

Failure to increase heart rate by more than five per minute following 1 mg atropine intravenously is compatible with brain stem death, as it shows that the Vagus nuclei in the brain stem are not functioning. This is called the atropine test.[5]

Another simple and useful test is the so-called ophtalmic test.[6] 10% solution of fluorescein hydrochloride is injected in the cephalic vein of the arm and its appearance in the retina is noted. The normal arm-retina time is 10-15 sec. Delay of more than one minute in appearance of the fluorescein on fundoscopy indicate absence of blood flow in the brain. And finally, it has been suggested that the endogenous opiate system could be responsible for the breakdown in sympathetic discharge which may occur in brain death.[7] One of the authors (N.B.) has used and proposed the so-called naloxone test[8] for diagnosis of brain death, in order to determine whether the nocioceptive system, which is a function of the brian stem has retained any activity.

In summary, the brain is the ultimate site of the human personality. It is also the target of resuscitation and various treatment modalities. Our main concern is the false-positive diagnosis of brain death in a patient who might survive with continuing mechancial ventilation and medical care.

In these controversy-laden and litigious times, it is prudent that physicians who are involved in making the diagnosis of brain death familiarize themselves with the material pertinent with the subject.

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Anaesthetic Circuits

N. SHAH

Key Words:
Anaesthetic circuits

Anaesthetic Circuits

An anaesthetic circuit or system can be defined as that part of anaesthetic apparatus which is used to connect the patient to the anaesthetic machine deployed to deliver predetermined volumes and concentration of oxygen, anaesthetic gases and volatile agents. This can easily be achieved by using a length of tubing, supplying the anaesthetic mixture from the machine to the patient. But it is highly desirable to have a proper circuit or system between the patient and the anaesthetic machine due to the following reasons:

(a) The patient is to be provided with a desired, balanced and congenial breathing atmosphere. Some anaesthetic circuits may tend to modify the concentration of gases and vapours which are actually delivered to the patient.
(b) The re-breathing of exhaled anaesthetic mixture requires to be prevented or minimised. Accumulation of CO_2 due to re-breathing also reduces the inspired concentration of O_2 and anaesthetic mixture in use.
(c) The mixing of inspired gases with room air as well as the pollution of theatre atmosphere from expired gases has to be prevented.
(d) The resistance to expiration needs to be minimised.

An anaesthetic circuit should, therefore, achieve the functional objectives as stated above and have the following characteristics:

(i) Simple:— should not complicate the overall function of an anaesthetic machine.
(ii) Light weight:— because a heavy circuit will cause dragging on the endotracheal tube or mask.
(iii) Versatile:— which can be used for all age groups as well as with spontaneous, controlled or assisted breathing.

Furthermore, this circuit should be easily sterilized. A large number of circuits or systems have been designed but, considering all the functional objectives one has in mind, none can be described as perfect. For descriptive purposes, however, Conway (1970)[1] has classified anaesthetic circuits into the following four groups:—

1. Open circuits.
2. Semi-open circuits.
3. Closed circuits.
4. Semi-closed circuits.

His classification allows a rigid definition of any system under any condition of use. The purpose of this article is to describe these circuits and to discuss new developments which have taken place since then.

1. Open Circuits

These circuits are those which have indefinite boundaries and no restriction upon the entry of anaesthetic mixture, e.g. open drop method. They offer little control over inspired concentration of gases. Anaesthetic mixture usually mixes with atmospheric air and for this reason, open circuits are nowadays rarely used. No re-breathing occurs in this system.

2. Semi-open Circuits

This type of circuit is partially bounded with some restrictions on fresh gas entry, e.g. gauze-covered Schimmelbusch mask. Depending on its volume, the mask applied to the face increases the patient's dead-space, causing re-breathing, the degree of which is dependent on the mask volume and the thickness of material around it. A reduction in inspired oxygen concentration usually occurs in this system. Supplying a stream of oxygen beneath the mask reduces the

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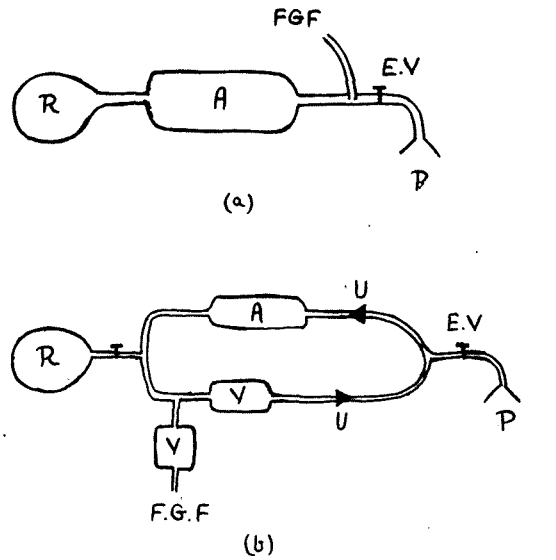

Fig. 1
CLOSED CIRCUITS
(a) To and Fro system.
(b) Circle system.
A = Absorber
E.V. = Expiratory valve.
I.V. = Inspiratory valve.
FGF = Fresh gas flow.
P = Patient end (mask).
R = Reservoir bag.
U = Unidirectional valve.
V = Vaporiser.

danger of hypoxaemia, flushes the mask and reduces re-breathing.

3. Closed Circuits

These are fully bounded circuits with no provision for any gas overflow. In this sort of apparatus total or partial re-breathing is intended because a facility for CO_2 absorption exist within the system. However, fresh gas flow (FGF) is necessary to allow for O_2 utilization and a certain degree of leakage which is unavoidable. They have two basic types:—

(a) Water's To and Fro system

The patient breathes to and fro from a re-breathing bag and through a carbon-dioxide absorber. Fresh gas is introduced into the circuit near the patient's mouth. There are no uni-directional valves, (Fig. 1).

(b) Sword's Circle system

The direction of gas flow is controlled by two uni-directional valves. Expired gas is allowed to pass into the CO_2 absorber through an uni-directional valve and then to a re-breathing bag, the reservoir. The bag also receives fresh gas flow which is directed to the patient through another uni-directional valve. Numerous variations of this arrangement can be made and are available depending on the site of fresh gas entry, relative positions of valves, reservoir bag and CO_2 absorber. However, the position of any vaporiser used and its efficiency is of greater importance than arrangements of components in closed circuits.

The principal reasons for the introduction and early popularity of both these closed-circuit systems were their economy in the use of gases and anaesthetic vapours, the conservation of heat and water vapour, less risk of explosion and minimum pollution of theatre atmosphere. Nevertheless, both systems suffer considerable disadvantages. The to and fro system, although simple and less cumbersome, has more inherited deficiencies than the circle system. Prediction regarding the concentration of gases within the circuit is impossible due to varying degree of anaesthetic uptake, efficiency of CO_2 absorption, and the difficulty in estimating basal O_2 requirements. Resistance to breathing is greater in to and fro system. Apart from this it has a relatively large dead-space. There exists also the danger of inhalation of irritant alkaline dust.

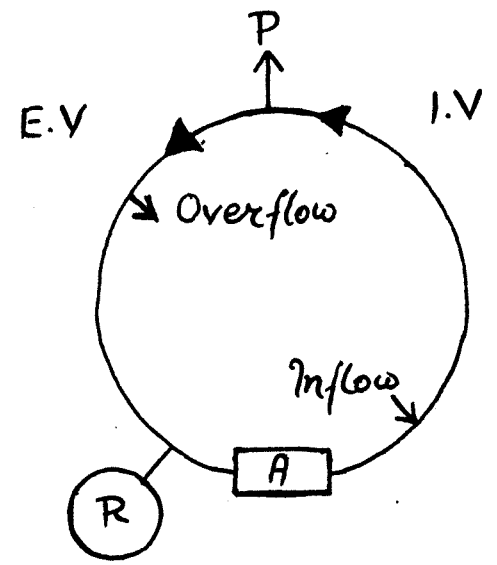

Fig. 2
SEMI-CLOSED ABSORPTION CIRCUIT
Overflow and unidirectional valves are placed close to the patient.

4. Semi-closed Circuits

These are fully bounded circuits having provision for venting of excess gas. They are the result of development carried out over the years on the previously stated systems. They can be described as absorption, re-breathing or non-rebreathing circuits.

(a) Absorption Circuits

A closed absorption circuit can be converted into a semi-closed system by increasing fresh gas flow (FGF) above the basal level. Thus, the inspired gas

mixture is controlled at the expense of fresh gas supply. Besides wastage the efficiency of the system is dependent on the relative position of various components, e.g. inspiratory, expiratory and overflow valves, Eger & Ethans (1968)[2], have demonstrated that, if overflow and the uni-directional valves are placed close to the patient (Fig. 2), the efficiency is at optimum during spontaneous as well as under controlled ventilation. However, the system suffers from various disadvantages. It is cumbersome and exerts high resistance to expiration.

(b) *Re-breathing Circuits*

Mapleson (1954)[3] has classified these circuits into five groups (A, B, C, D and E). The diagram (Fig. 3) adopted from Mapleson illustrates the differences between them.

(i) *The Mapleson A or Magill Attachment*

This attachment has been extensively studied and is the most commong anaesthetic circuit in use. During spontaneous breathing, re-breathing of alveolar gases will not occur unless FGF is reduced to the alveolar ventilation level. To prevent re-breathing FGF must be at least 70% of respiratory minute volume. The efficiency of the system depends on the flushing effect of FGF to expel expired gases. If FGF level falls below alveolar ventilation, the degree of CO_2 retention will be an inverse function of FGF, and

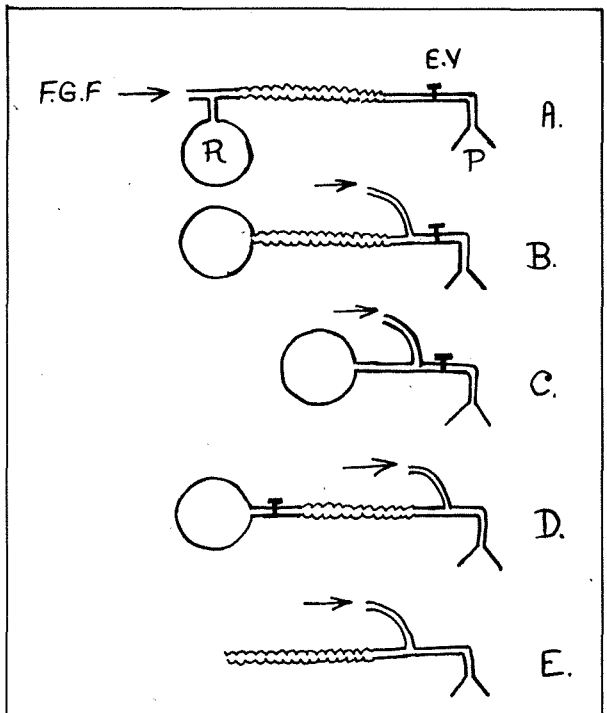

Fig. 3
SEMI-CLOSED RE-BREATHING CIRCUITS
Mapleson classification.

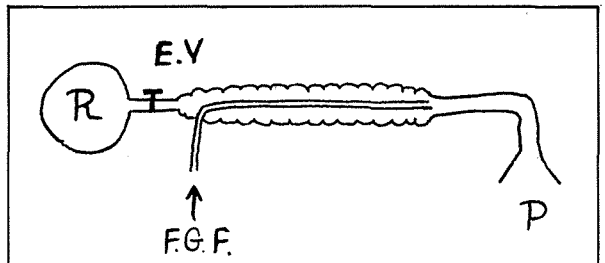

Fig. 4
BAIN CIRCUIT
Mapleson type D modification.

therefore, hyperventilation by the patient will not affect alveolar gas composition.

During controlled or assisted ventilation, considerable degree of re-breathing may occur. This can be reduced by venting out a greater amount of alveolar gas from the circuit by deploying higher FGF, reducing respiratory rate, or by increasing tidal volume. The placement of the expiratory valve close to the patient head in this system is its main disadvantage. The valve adjustments and venting of gas can be difficult especially during head or neck surgery.

In circuits B, C and D, fresh gas is introduced close to the patient. This arrangement appears to be advantageous but Sykes (1968)[4] has demonstrated that during spontaneous breathing all systems behave less efficiently than circuit A. With controlled or assisted ventilation most of the fresh gas input is delivered to the patient during inspiration particularly in system D, therefore system D causes less re-breathing than circuits B and C.

(ii) *Mapleson E or T-piece circuit*

This is the only valveless system without a reservoir bag described by Ayre in 1937. In this system during expiration dead-space gas passes down the open end of the tube first and the alveolar gas last. The concentration of fresh gas towards the patient end of the tube increases as expiration progresses. If FGF is greater than the peak inspiratory flow rate (at least 2.5–3 times the minute volume) all the inspiration will consist of fresh gas.

Controlled ventilation can be maintained by intermittent occulsion of the expiratory limb, a principle used in many paediatric ventilators. The main advantage of this system is the absence of resistance to expiration, an important advantage in the case of small children. Many modifications of the T-piece circuit are available, e.g. Jackson-Rees (with an addition of open ended reservoir to expiratory limb) and Rendell-Baker paediatric system.

(iii) Bain circuit (Mapleson D modification)

This system is a modification of Mapleson D type circuit introduced by Bain and Spoerel (1972)[5]. It comprises a 1.8 metre length of light weight conductive corrugated plastic tubing 22 mm in diameter. Through this runs a smaller bore tube of 7 mm diameter. Fresh gas is introduced through the inner narrow tube. Reservoir bag and expiratory valve are placed at the anaesthetic machine end of the circuit (FIG 4).

For spontaneous breathing a FGF of 1.5-2.0 times the minute volume is necessary to prevent re-breathing. During controlled ventilation FGF of 70 mls/kg body weight/minute into the system would produce normocarbia when venting with a tidal volume of 10 mls/kg body weight and a frequency of 12-14 per minute, as demonstrated by Henville & Adams (1976)[6].

This system is useful when access to the patient is difficult e.g. head and neck surgery. It also facilitates the scavenging of the expired gases. Hazards, however, can occur if the inner tube becomes dislodged or broken resulting in a considerable increase in the patient' s dead-peace.

(iv) Lack circuit (Mapleson A Modification).

This is a coaxial modification of the Magill system introduced by Lack (1976)[7]. The main objects were to overcome the disadvantage of accessibility and to prevent theatre pollution. FGF is directed through an outer corrugated tube and exhaled gases are collected and discharged through an inner expiratory limb. The reservoir bag and the expiratory valve are placed at the end of corrugated tubing (fig. 5).

The prototype was evaluated by Barnes et. al. (1976)[8]. The resistance of the system was found to be unacceptably high with marked evidence of re-breathing when FGF equalled minute volume. It was estimated that FGF of $1^1/_2$ times the minute volume would be required to prevent re-breathing of alveolar gas. Subsequent improvements on the production model by increasing the capacity of inspiratory limb to 500 mls and a 50% reduction of expiratory resistance need to be further evaluated in long term usage.

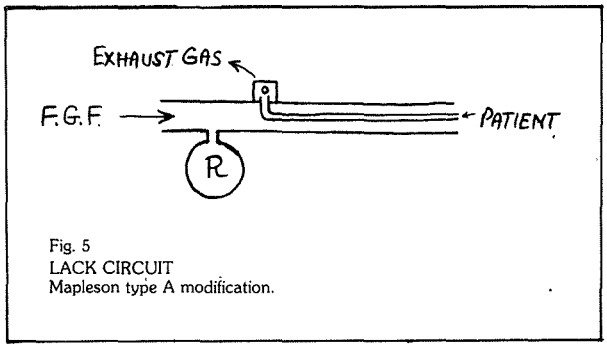

Fig. 5
LACK CIRCUIT
Mapleson type A modification.

(c) Non-rebreathing Circuits.

In this system a non-rebreathing valve is placed to the patient, most conveniently as a modification to Mapleson A,B or C circuits, where it replaces the expiratory valve. Sykes (1959)[9] has described many different designs of such valves. Under ideal conditions (FGF is equal to minute ventilation), the system will allow full control over the inspired atmosphere. In practice, however, reproducibility is difficult to attain under all conditions of ventilation. A small discrepancy between FGF and patient minute volume can cause serious disfunction of the circuit.

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Forty Years of Anaesthesia in Malta

RICHARD LOUIS CASOLANI (1902 - 1981)

PAUL CASSAR

The death of Dr. R.L. Casolani on April 8, 1981 marked the end of an era in the evolution of anaesthesia in Malta.

I often had occasion, during the seventies, to listen to him describing local developments during the formative years of his speciality in our island. At my request he kindly supplied me with notes, written in an *impromptu* fashion, about his experiences. They form the basis of this paper.

The son of a Police Officer, Dr. R.L. Casolani was born in 1902. He was educated at the Lyceum and at the University of Malta from where he graduated M.D. in 1925. Being fourth in order of merit in his course he was, in accordance with the procedure prevailing at the time, appointed Junior Resident Medical Officer at the Central Civil Hospital, now the building housing the Police Headquarters, at Floriana.

Until the end of the first World War, anaesthesia was administered at the Central Civil Hospital by one or other of the four Resident Medical Officers. With the cessation of hostilities, the post of Anaesthetist was created on the recommendation of the eminent surgeon Col. Sir Charles Ballance and other British consultant surgeons who were in Malta during the war years.

The first to fill the post was Dr. Emmanuel Vella (1885-1925) who was appointed on June 20, 1919 and who was previously anaesthetist to Sir Charles Ballance. On May 22, 1922, Dr. Vella was succeeded by Dr. George Busuttil who, like his predecessor, had gained considerable experience as anaesthetist during the war with his attachment to the Royal Army Medical Corps of the British Army[1]. It was through Dr. Busuttil that Dr. Casolani was introduced to the practice of anaesthesia.

Recollecting the conditions prevailing in the Operating Theatre at the Central Hospital in the late twenties. Dr. Casolani described them in these words:- "There were no Surgeons Dressing Rooms or Washing rooms. No Anaesthetic Room. Patients were put under on the Operating Table in full view of all the theatre paraphernalia including including (the sight) of the surgeon washing up" together with his assistants at a number of white porcelain sinks fixed to the wall in one corner of the theatre. The surgeon used "to take off his outer clothes behind the screen in the same theatre... Operating gloves were sterilised by boiling for as long as I can remember as were all the instruments". Anaesthesia was administered by the "Rag and Bottle" method with chloroform and ether mixture dropped on a Schimmelbusch mask. "Patients were held down forcibly (on the operating table) by one or more nurses during induction... and many a struggle developed with the hardier country type of patient".

Casolani's service at the Central Hospital was disrupted when he contracted Undulant Fever (Brucellosis) through an accidental prick in his hand from the needle of a syringe containing blood that had been drawn from a patient suffering from the disease and which threw Casolani out of action for four months.

In 1926, after a competitive examination, he was appointed Clinical Assistant to the Professor of Surgery, Dr. Peter Paul Debono, who modernised surgical methods at the hospital and enforced a strict aseptic technique during operations.

In July 1929 the post of Anaesthetist at the Central Hospital became vacant on the retirement of Dr. George Busuttil. Casolani applied and was selected for the post. In August of the same year he went to London with the support of a letter of introduction to the Dean of St. Bartholomew's Hospital from the anaesthetist of the Royal Naval Hospital at Bighi, Surgeon Commander M. Brown, who was himself a Barth's man. Casolani was

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accepted at this hospital as a trainee under one of the then junior anaesthetists, Dr. F.T. Evans. There were then no Diploma or Fellowship in Anaesthesia; these came years later when the same Dr. F.T. Evans became first President of the Faculty of Anaesthetists of the Royal College of Surgeons.

From Evans, Casolani learned all about the Boyle's machine and how to intube patients under chloroform. Mr. Boyle was then the Senior Anaesthetist at St. Barth's though he was by then an elderly man and reaching the end of his professional career. Casolani met him once or twice. Another renowned anaesthetist that Casolani came across was Sir Francis Shipway, the inventor of the Shipway apparatus, at Guy's Hospital.

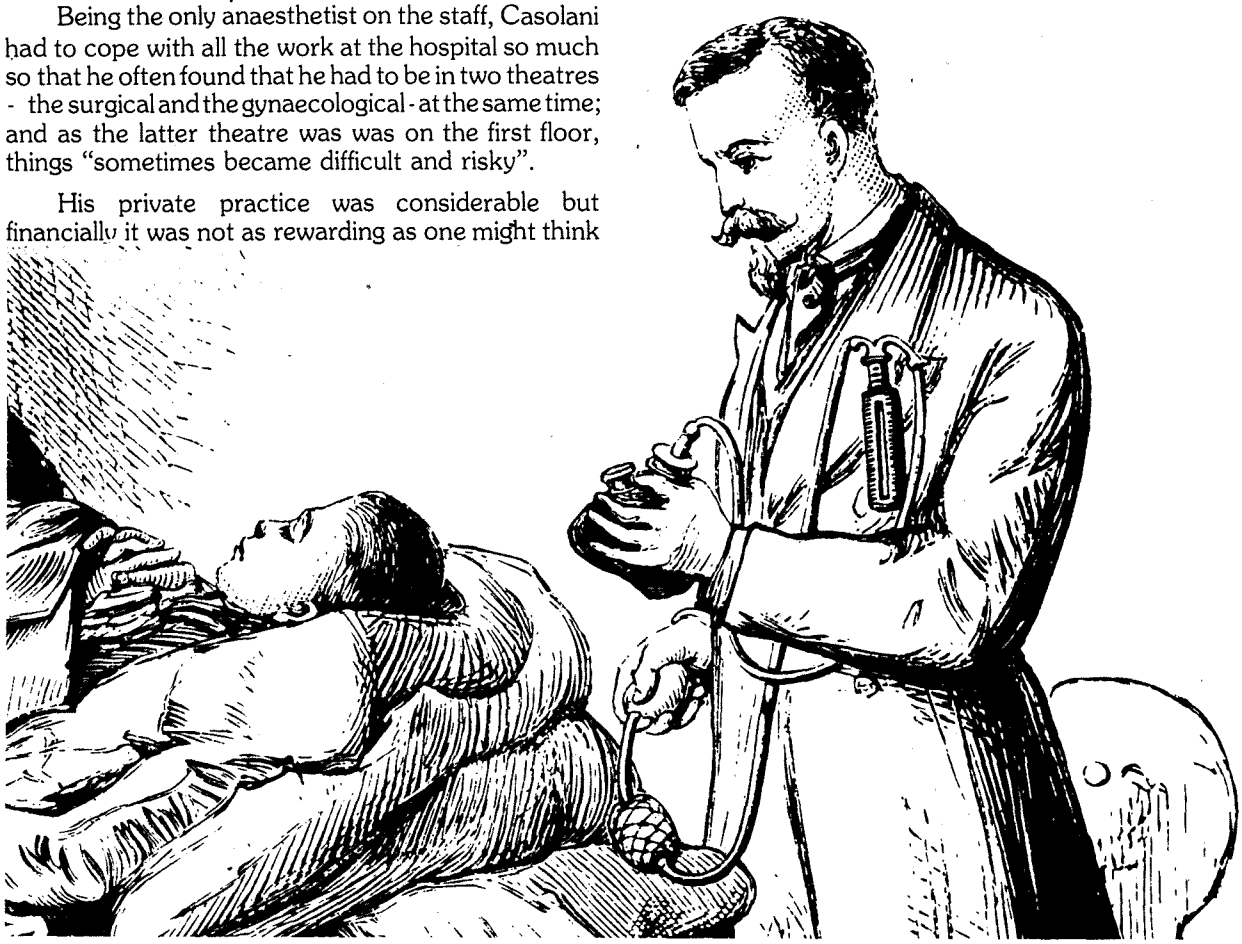
On his return to Malta, Casolani set himself to apply at the Central Hospital what he had learned in London and to bring anaesthesia up to date in our island by promoting the use of Boyle's gas and oxygen apparatus and introducing intranasal anaesthesia. This was started by using a stiff gum elastic catheter which he had to make himself by cutting lengths of rubber tubing as Boyle's intranasal catheters had not yet come on the market.

Being the only anaesthetist on the staff, Casolani had to cope with all the work at the hospital so much so that he often found that he had to be in two theatres - the surgical and the gynaecological - at the same time; and as the latter theatre was was on the first floor, things "sometimes became difficult and risky".

His private practice was considerable but financially it was not as rewarding as one might think because in those days the fees from private patients were collected by the surgeons and the anaesthetist had to to share them with the family doctor and the surgeon's assistant.

In April 1932 the post of Surgeon Lieutenant in the Royal Malta Artillery became vacant. He applied for the post although this meant giving up his intention to specialise in anaesthesia. He was selected from among fifty applicants[2]. He once told me, in his usual tendency to belittle his own merits, that three factors may have tipped the scales in his favour:- (a) he was the grandson of a former officer of the Royal Malta Fencible Artillery - Richard Casolani - Captain and Adjutant of that regiment; (b) he was a trained anaesthetist and the army in Malta needed such a specialist: and (c) his connections with St. Barth's Hospital stood him in good stead as the Deputy Director of Medical Services, who was on the interviewing panel, was a Barth's man himself.

In January 1983 he applied to follow the Junior Officers Course in England as all new commissioned RAMC officers and Medical Officers of the Indian Medical Corps usually did. He was accepted and spent the next six months at the Royal Army Medical



College, Millbank, for lectures in Tropical Medicine and Hygiene, Military Surgery, etc. and at the RAMC depot in Hampshire to undergo a purely military training in such activities as marching and horse riding.

On his return to Malta on completion of this course, he was assigned to Lascaris Barracks, below the Upper Barrakka, the site now occupied by the Ministry of Education. He had to carry out a daily sick parade but in those days few Maltese gunners reported sick because of the prejudice against hospitalisation so that it was only when they were really ill that they went to see him. He observed that a gunner through almost illiterate, could "grasp things quickly and with enthusiasm" and "was, generally speaking, a man of upright character, loyal to his superiors, obedient to the last word" and a very accurate marksman.

Casolani's military commitments were not so time consuming as to allow the practice of anaesthesia to pass out of his hands as he had feared when he joined the RMA. In fact he was on the staff, as anaesthetist, of King George V Hospital, now Boffa Hospital but then a private hospital patronised mainly by British seamen and citizens resident in Malta. He also occupied a similar position at the War Memorial Hospital for Children at the Zammit Clapp Hospital, St. Julian's. These appointments kept him in the running with the principal surgeon of the island - Professor P.P. Debono - and thus he had the opportunity of doing some private work though the lion's share of the practice passed into the hands of his successor, as anaesthetist, at the Central Hospital - the late Dr. Edward Critien who was introduced to St. Barth's Hospital for a course in anaesthesia by Casolani.

When the RAMC became aware of Casolani's skill in anaesthesia, he was called upon to exercise this speciality at the Military Families Hospital, which was a combined women, maternity and children's wing of the main military hospital at Mtarfa.

On June 12, 1940 Malta became directly involved in the hostilities of the Second World War. Casolani was attached to Mtarfa Military Hospital where he remained for the next two-and-a-half years, at the end of which he was posted to another British military hospital - the 39th General - at Mellieha Bay. By this time he had been officially graded as a Specialist by RAMC standards. At the Mellieha Hospital he had to cope with over seven hundred pro-allies Yugoslav Tito partisans who were evacuated to Malta from Bari following the Allied invasion of Sicily and Southern Italy. Most of them - men and women - had compound fractures that had been immobilized in makeshift plaster of Paris " with pus pouring out from everywhere". It took months to get them well but with few exceptions they all eventually returned to their country.

At the age of fifty-five years he retired from the RMA in 1957 with the rank of Surgeon Major. However, as the RAMC was short of anaesthetists, he was taken on by that corps as a full-time Civilian Specialist in anaesthesia. In this capacity apart from doing most of the work, he trained a number of British doctors who were interested in the speciality. When the RAMC left Malta in 1962, he passed on to the Royal Navy in the same grade of civilian specialist, his duties being divided between the Naval Hospital at Bighi and the naval Maternity Wing at Mtarfa. He retired completely from professional activities in 1969 at the age of sixty-seven years.

In his medical career of forty years, Casolani saw anaesthesia grow from the "Rag and bottle" era, when he took over from his predecessor Dr.G. Busuttil in 1929, to what it is to day with such developments as the use of spinal anaesthesia, the introduction of the rapid acting barbiturates, the advent of curare and, later on, the safer muscle relaxants, the use of intratracheal intubation and the sophisticated means for resuscitation now availabe as routine equipment. During all this period he had only two cases of cardiac arrest - at Mtarfa - both of which ended happily.

Although Casolani was unequipped with the diploma and the fellowship of his speciality - which came on the scene late in his career - he enjoyed, and justified, the full trust of his patients and of his colleagues and of the highest authorities of the British military and naval forces then stationed in Malta. In fact during his career he had the honour - and the onus - of having entrusted to his care as anaesthetist a number of distinguished personages in our island, both among the Maltese and British communities, without any mishaps.

An unobtrusive and fortright man, Dr. R.L. Casolani made his way to a high place in the chosen branch of the medical profession by the sheer force of hard work, meticulous competency and the shouldering of responsibility without flinching under circumstances of extreme difficulties in his speciality[3].

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3. I have not succeeded, so far, in tracing any papers from him on anaesthesia. The only two publications I know of have no link with his speciality. They are (a7 *L-ewwel għajnuna lil min ikorri,* being the fourth edition in Maltese of the St. John Ambulance Association booklet *First Aird to the Injured,* published in 1943; and (b) a pamphlet *Off the Beaten Track in Malta* consisting of very short description of a number of excursions for visitors to Malta. It was written jointly with Major H.T. Laycock (late RAMC).

Advanced Preparation for Rescue and Medical Action in an Emergency Landing Disaster in Maltese Waters

J. BARSKI and M. UPTON

Key Words
Emergency landing disaster;
Medical action; rescue

Air traffic into Malta is heavy for such a small island, especially during the busy summer tourist season. Almost every type of aircraft flies in, including Jumbos with up to 450 passengers. Eversince the airport has been the sole responsibility of the Maltese authorities there have been two minor incidents; one in which a small private plane made a crash landing (minor injuries) and another when a commercial aircraft engine caught fire on take-off. Both incidents were efficiently dealt with. The island is only 10 km in width at the widest point and Luqa airport is only 6-7 km distant, from each coast. The flight path along the main runway passes over the Grand Harbour on the east and over high cliffs on the west. At landing speeds this is approximately 1 minute of flying time, so there is a considerable likelihood that, in the event of a disaster it could be at sea.

All those persons involved in the aircraft industry with whom we consulted have stated that, forewarned of an aircraft fault, they would always prefer to come down on land, rather than on the sea. Making an emergency landing at sea would compound the difficulties in rescuing the passengers, and salvaging the aircraft would be almost impossible. Also, Luqa Airport has emergency equipment immediately available and procedures selected according to the likely extent of the disaster. These are normally designated as "Priorities" numbered 1 to 3 following the practices of the International Civil Aviation Organisation (ICAO). Additional to procedures for a crash on land preparations are in hand for rescue at sea.

This paper deals with one special aspect of such a rescue. In the event of a known fault developing in an aircraft, while emergency services are placed on stand-by at the Airport, so also are teams for a sea rescue.

Should the situation develop into an accident, full air/sea rescue procedures would be set in action. The location of the wreckage is the responsibility of the helicopters and patrol boats of the Coast Guard. Immediately after the accident is known to have occured all services likely to be required are put into action by the Emergency Control Centre, such as: Police, Ambulance Service, Casualty Department at the Hospital, etc. One of these services is the Surface and Underwater Volunteer Rescue Organisation (SURVO).

SURVO consists of a number of teams specially trained in techniques of rescuing persons trapped beneath the water, and from the surface. All are experienced aqua-lung divers, highly skilled in approaching a panicking person and in administering respiratory resuscitation to the drowning.

When alerted, the SURVO volunteers assemble at one of three selected centres around the island; the heliport, the coast guard station and diving centre. Equipment is collected from the designated centre. This includes medical supplies - additional aqualungs, regulators with a full face mask/octupus attachment, in addition to their own personal equipment. One member of each team is a medical officer, a doctor from the local hospital who is also a trained aqualung diver.

The Maltese Islands are in the form of a plateau tilted approximately from east to west. The western coast is almost entirely a cliff 150-200 m high from which access to the sea is almost impossible. The

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east coast has several bays. The three centres are on the east coast from which acess to the sea is easy. The sea is very deep close to the coast at most points around the island. Diving operations are very difficult below 50 m, so effective rescue is limited to this depth.

If a perfect landing occurs on the sea, the plane floats for some little time. All passengers should be able to make their escape from the plane complete with an inflatable life jacket. The SURVO teams should be present to take the passengers from the sea and transport them by inflatable boats to the larger boats on which medical teams are in attendance. They are trained to give respiratory resuscitation on inflatable boats using a tight face mask and an Ambu bag. If need be the SURVO divers are able to attach a pressure regulating valve to their cylinders with an output to a soft bag connected to non-rebreathing system with a Ruben Hesee Valve.

The divers work in pairs as is the normal practice. They approach a person in the water, determine if respiratory resuscitation is necessary and if so start it immediately, while towing him to the inflatable boat. Once safely on board major injuries can be attended to while the victim is transported to the hospital boat or coastal mobile casualty unit. While giving resuscitation in the water life saving principles are used. The approach to the subject must always be made from behind (to prevent the rescuing diver from being grasped by the victim). A firm hold is taken with one hand around the nape of the neck, and the other on the chin. The hand holding the chin is normally used to close the mouth, while a seal is made on the nose, so using a "mouth to nose" technique. The use of a "mouth to mouth" method in rescuing passengers would be preferable but it is very difficult to accomplish in the sea. There remains the problem of those injured and unable to leave the plane. In order to rescue them, the plane would have to be entered risking it sinking at any time. It would be impossible for a fully equipped SURVO team member to reach any of the plane exits from the sea. This problem is still under consideration.

In the more likely situation where the plane hits the water and the fuselage is damaged it will become submerged within a very short time. The probability of anyone contriving to escape is extremely remote.

Should the fuselage come to rest at a workable depth and should a pocket of air be trapped therein, there might be a possibility to save those still alive. In a simplified analysis of the situation, we consider that the fuselage is damaged on the underpart and that it rests with this damaged portion towards the sea bed. The water level will rise inside the aircraft compressing the trapped air until the air pressure balances the pressure of the water. Should the plane end up in relatively shallow water, say, 10 m so that with a larger aircraft the roof would still be exposed; the water would rise half way up the fuselage, so it can be imagined that in deeper water a very small air pocket would remain. Any surviving passengers will be exposed to the same physiological conditions as a diver swimming at the depth of the water/air interface.

For an interface above 9 m no decompression complication exists. As the interface goes below 9 m the decompression consideration rapidly becomes very significant.

When a group of people are trapped in a sealed space two principle factors effect the rate of onset of asphyxia – the partial pressure of carbon dioxide and of oxygen. Many other very complex physical and physiological influences are involved, but to make a discussion reasonably simple only these two effects are being considered.

If the sealed passenger cabin is at approximately atmospheric pressure since it is so regulated during flight and unpolluted air was present, the onset of hypoxia would be concurrent with the increase in carbon dioxide partial pressure. If however the aircraft were submerged, the air trapped inside would be compressed to a higher pressure, and carbon dioxide narcosis would occur before hypoxia. To emphasise this simplified analysis, the same mass of air at a higher pressure (under reduced volume) will give enough oxygen for a longer time, but will produce carbon dioxide narcosis in a shorter time. This difference is further exaggerated if the oxygen carried in the aircraft is released into the cabin. The most likely situation is that the aircraft will break up and be scattered over a large area; any survivors being those who by some miracle have been thrown clear. However in planning a rescue operation it is necessary to be prepared for every eventuality that one can imagine, thereby reducing the number of unforeseen situations which will occur. The presence of even a single living person in a submerged aircraft necessitates that a procedure is worked out to rescue him.

An "octupus rig" is a standard piece of equipment used by a diving instructor. It is an additional mouthpiece attached to the instructor's cylinder. By further adding a full face mask it should be possible for an untrained person to breath while being led from the aircraft. Using this as a basis it is necessary to consider methods of ingress. The plane should be entered below but as close as possible to the air/water interface; this level would first have to be determined. It should be noted, that if a part of the cabin is above sea level, opening this to the atmosphere will cause the level of the water in the plane to rise to sea level. Either a suitable door would have to be forced or an opening cut in the fuselage and the inevitable debris would have to be cleared to make a passage.

All of the foregoing still holds true for an accident during the night or in poor weather conditions, but the survival chances would be correspondingly reduced.

To all the injury and rescue problems expected in a disaster on land are added the complications of drowning, asphyxia, hypothermia, decompression sickness and barotrauma. Therefore every person rescued, even those with no apparent injury must be fully hospitalised from the medical boat or mobile casualty unit.

Epidural Anaesthesia for Elective Caesarean Section*

Case report

N. BOSKOVSKI and E.S. GRECH

Summary

Continuous epidural anaesthesia is now established method for the relief of pain associated with labour and childbirth, and for Caesarean section, both elective and emergency. A 34 year old woman at term was electively allocated for Caesarean section because her previous two deliveries were managed in the same manner. The report emphasizes the advantages of epidural over general anaesthesia for safe conduction of Caesarean section.

Key Words

Elective Caesarean section:
Anaesthetic Technique: Epidural

Epidural anaesthesia has been used with increasing frequency for obstetric patients during the past twenty years. When properly applied, efficient analgesia is invariably obtained, maternal satisfaction is high, and serious morbidity is significantly low.[1] Maternal and fetal acid-base status are better maintained under epidural anaesthesia.[2,3] Neonates delivered by Caesarean section under epidural anaesthesia have higher Apgar scores and less fetal depression than those babies delivered by general anaesthesia[3]. In addition, epidural anaesthesia eliminates some of the risks associated with general anaesthesia, namely aspiration pneumonitis and awareness[4]. A further advantage is that the mother is alert and thus an early mother-infant emotional bond is established.

Case report

A 34 year old woman, gravida 3 para 2, was admitted in St. Luke's Hospital at 39 weeks of gestation for an elective, lower segment Caesarean section. Her previous two children were delivered by elective Caesarean section conducted under general anaesthesia. The patient was well nourished and in good health. Her antenatal period was uneventful and she had gained 12 kg in weight durign the course of this pregnancy. On admission, laboratory findings were within normal limits. Her haemoglobin (Hb) level was 11.2g dl^{-1}, white blood cell count 6.1 x 10^9 l^{-1}, and platelet count 173 x 10^9 l^{-1}.

She gave informed consent for the procedure and was premedicated with diazepam (Valium) 10 mg orally one hour prior surgery.

An intravenous infusion of 500 ml $^1/_2$ Ringer lactate in 5% Dextrose, was given in the 15 min preceding the induction of epidural anaesthesia. Left lateral tilt was utilised to prevent aortocaval compression.

Epidural anaesthesia was induced with the patient in sitting position, using a midline approach and a Tuohy needle G 17. An epidural catheter was inserted, advanced 2-3 cm cephalad, and 19 ml of 0.5% plain bupivacaine (Marcain) was used in accordance with the method of Thorburn and Moir (1980).[5] After twenty minutes, the level of anaesthesia was checked bilaterally by pin-prick and was found to extend from T_6 to S_5. Peroperative fluid therapy consisted of 1.5 l cristaloid infusion. A routine lower segment Caesarean section was performed and a baby-boy weighing 2600 g was delivered with one and five minutes Apgar scores of 10.

Surgery was terminated uneventfully and no vascular or pulmonary complications occured in the post-operative period.

Discussion

In contrast to narcotics, epidural anaesthesia produces complete relief of pain and the hazard of pulmonary aspiration of gastric content during general anaesthesia is virtually eliminated.

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*The very first Caesarean section under epidural anaesthesia in Malta was done in St. Luke's Hospital on the 20th of May, 1980.

When properly administered, epidural anaesthesia causes no maternal or neonatal depression. It also permits the mother to remain awake during labour and delivery so she can participate actively in the birth of her child.

Our patient remained awake and was co-operative and enthusiastic during the whole procedure. Significant haemodynamic changes in our patient were prevented by appropriate fluid therapy. Blood loss was insignificant.

The Apgar score of the neonate at one and five minutes were both 10, indicating an undisturbed placental perfussion. Epidural anaesthesia eliminates the progressively increasing maternal metabolic acidosis which occurs in the first stage of normal labour.[6]

One hopes that continuous epidural anaesthesia in Malta will become a more frequent procedure for anaesthesia in Caesarian section and for pain relief during labour and childbirth *. Maternal diabetes mellitus is also a relative indication for use of epidural anaesthesia in labour. According to the recent Survey (W.H.O 1981) the prevalence of diabetes mellitus in Malta is 7.7%. Therefore, it seems appropriate to offer epidural service for labour and delivery to a population with high prevalence of diabetes mellitus.

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* The very first labour and delivery under epidural anaesthesia in Malta was performed in Karen Grech Hospital on 18th of April, 1982.

Intragastric Knot of Gastro-Duodenal Tube

Case Report

NIKOLA BOSKOVSKI MD.

Key Words:

Complications during anaesthesia:
Intragastric knot.

Case report

A 47 yr old female, starved and premedicated, was presented in the operating theatre for elective cholecystectomy. Anaesthesia was induced with thiopentone 4 mg kg^{-1}. Tracheal intubation was carried out using 6mg pancuronium, after the larynx had been sprayed with Xylocain 4% and lungs were ventilated with nitrous oxide and oxygen.

Fentanyl 10 μg kg^{-1} was given i.v. for analgesia, supplemented with N_2O: O_2 6:4 1 min^{-1}, and artificial ventilation commenced. Soon after the skin incision was done a lubricated gastroduodenal tube, with a metal olive (Sonjet, Laboratories, Peters, France) was introduced into the stomach through the left nostril. There was no difficulty in passing the tube. Peritoneal cavity was explored and moderate distension of the stomach persisted. The surgeon could palpate the tube in the stomach but when aspiration was attempted neither gastric content nor air was sucked out. The tube was then repositioned by pulling and pushing but this was to no avail. Finally, it was decided to remove the tube and re-insert it again.

It was a surprise for all to see that a knot on the distal part of the gastro-duodenal tube was formed, so that suction was virtually impossible. The first side-hole in this kind of tube is located at 7cm from the tip. If more side-holes are located higher up, it may be possible that when a knot in the stomach occurs a few holes proximal to the knot remain free thus making gastric suction possible.

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