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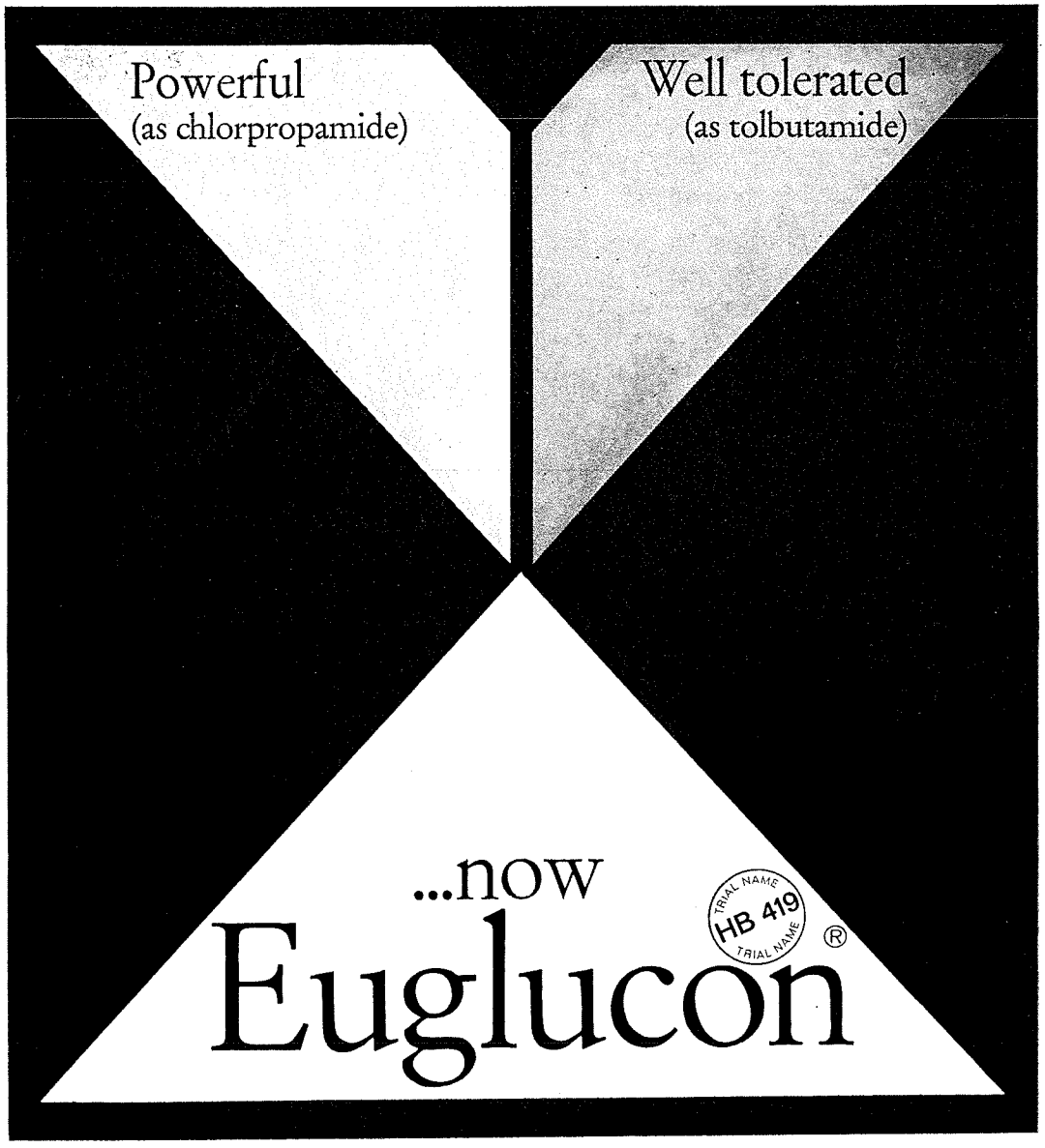

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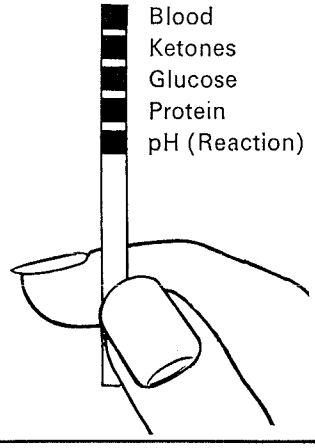

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Reference:
1. Jameson, R.M., Lancet, June 7th 1969, p. 1164.

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journal of the malta medical students association

Five years are a long time to spend in any apprenticeship, but even then one is reluctant to relinquish "the good old student days". Time remembered is grief forgotten, and so a euphemistic description of our student days will linger on with those of us who graduate this year. Sharper memories will however, also evoke thoughts of long hours spent listening, absorbing and noting what countless lecturers, demonstrators and teachers had to say. Recently we have seen a slight diminution in formal lecturing but these still occupy a disproportionate part in our curiculum. In what has been described as a historic event, lectures have been completed in the major subjects six months prior to the Finals. "Superfluity of lecturing causes ischial bursitis" said Osler, and this seems good advice — or is it an even more profound peculiarity in our teachers who as again Osler said "are inclined to teach too much, just as students tend to learn too much — neither, perhaps with great success".

The new sized *CHESTPIECE* cannot pass unnoticed to the least discerning of our readers. In reality we are often pleasantly surprised to note that many who have long graduated still glance through the journal, which alone is sufficient reason for improving as much as possible the text and presentation of the journal.

A second reason is that of further promoting contributions from students. This issue has had to rely, to some extent, on articles from our competent teachers, but we still feel that the bulk of the contents should come from the student.

Since our last issue we note with regret the passing away of two of our teachers. From our brief relationship with such men we not only inherit our academic education, but also a philosophy and way of life. Professor Harry Micallef, who so widely promoted the study of Biology in Malta is remembered for his charm and urbanity towards students and his fellow men. Professor John Craig who for so many years held the Chair of Surgery, cannot be forgotten for his excellence as a clinical and his skill as a surgeon.

A word of thanks must go to our printers at St. Joseph's Home who for a number of years have helped us in the preparation of this journal.

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the problematic pill

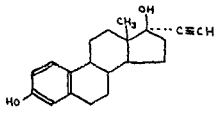
m.g. elder, m.b., ch.b., f.r.c.s., m.r.c.o.g.

lecturer in obstetrics and gynaecology, royal university of malta

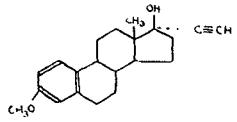
The first oestrogen containing oral contraceptive was introduced in 1957 as a clinical trial involving a few hundred Puerto Rican women; it was first sold on the open market in 1960, and it is now taken by over 14 million women throughout the world.

There are two types of oestrogen containing oral contraceptive, a combined preparation in which both oestrogen and progestogen are present in all 20 to 22 tablets, or a sequential preparation in which all the tablets contain an oestrogen, but only the last 5 to 7 contain a progestogen. Recently a pill containing a progestogen *alone* has been introduced, but as it is not very effective as a contraceptive its use is limited, and there has been as yet little research work carried out on its metabolic effects. Only the first two types, that is those containing an oestrogen, will be considered.

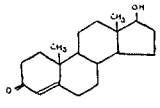
Two *oestrogens* are used at the present time in oral contraceptives. These are *ethynyl oestradiol* and its 3 methyl ether, namely *mestranol*. The chemical formulae of these compounds are shown:



ethinyl oestradiol



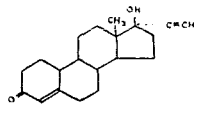
mestranol



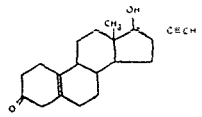
testosterone

The *progestogens* are divided into two groups, the derivatives of 19nor testosterone and those of progesterone.

Removal of the methyl group from the carbon 19 of testosterone reduces its androgenic properties considerably. Substitution of an ethynyl group at the 17 position produces *17 a ethynyl 19 nortestosterone, or norethindrone, or norethisterone.*

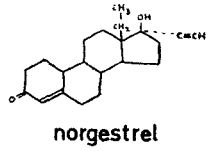


norethindrone/norethisterone

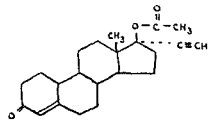


norethynodrel

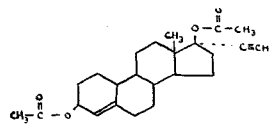
A shift in the double bond from the 4-5 to the 5-10 position produces *norethynodrel,* which in addition to being a progestogen also has some oestrogenic activity. The addition of a methyl group at the 18 position of norethisterone produces *norgestrel,* while acetylation at the 17 position produces *norethisterone acetate.* Finally reduction of the 3 ketone group with acetylation at positions 3 and 17 produces *ethynodiol acetate.*



norgestrel

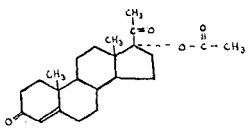


norethindrone acetate

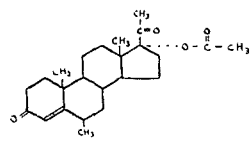


ethynodiol acetate

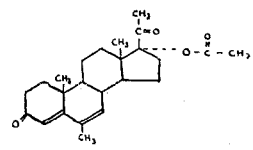
Progesterone itself is inactive when given orally, as is *17a hydroxy progesterone,* but esterification produces an orally active compound with progestational properties. The addition of substituents at position 6 further increases its effectiveness. A methyl group at this position produces *medroxyprogesterone acetate,* while the addition of a double bond between position 6 and 7 produces *megesterol acetate.*



17acetoxy progesterone



medroxy progesterone acetate



megestrol acetate

The progestational agents are metabolised to some extent to oestrogens, and hence they all have a varying degree of oestrogenicity, norethynodrel having moderate oestrogenic properties, while norethisterone and medroxyprogesterone acetate have minimal oestrogenic properties.

The principal mode of action of the pill is inhibition of ovulation by the elimination of the FSH and LH peaks of the menstrual cycle. Both the oestrogen and progestogen are responsible for this occurence. In addition the inhibition of the gonadotrophins by these synthetic steroids leads to a marked reduction in endogenous ovarian steroid production. Finally the progestogen is thought to exert some contraceptive effect by its direct action on the endometrium, causing atrophy of the glands, and also by making the cervical mucus more tenacious, and hence more difficult for sperm penetration.

Having given a brief synopsis of the structure and mode of action of these preparations I should now like to turn to their metabolic effects and the subsequent clinical problems.

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Minor side effects such as breast fullness vaginal discharge and nausea are attributable to the oestrogen component, while apathy, pelvic discomfort, diminished libido and depression are attributable to the progestogen. Nausea during the first few cycles can occur in up to 40% of patients but its frequency diminishes rapidly thereafter. Depression and diminished libido are associated with high endometrial monoamine oxidase activity (Grant and Pryse-Davies, 1968) Another factor that may be responsible for depression is altered trytophan metabolism, leading to a functional deficiency of pyridoxine, which is a coenzyme in the production of 5 hydroxytryptamine. It has therefore been suggested that pyridoxine should be given prophylactically to all women with a history of depression who are taking oral contraceptives, or even that depression is a contraindication to taking these preparations.

Secondary amenorrhoea can follow cessation of the pill, probably being due to a failure of the gonadotrophin release factors to revert to their normal levels. Five per cent of patients who had been taking the combined pill for more than 2 years had a menstrual delay of 70 to 390 days. (Goldzieher, 1970)

The side effect that has recieved the greatest publicity is thromboembolism, with its occasional fatal culmination. Vessey, Doll and Inman in a series of papers during the last two years, and summarised by Doll and Vessey (1970) have concluded that there is a six-fold increase in deep venous thrombosis and pulmonary emboli with an eight-fold increase in mortality from these causes among women on oral contraceptives, together with a six-fold increase in the incidence of, and mortality from, cerebral thrombosis. Opinion in the U.S.A. is slightly sceptical of this work in that the studies were small, retrospective, and involved only hospital admissions. However one of the chief critics in quoting different figures refers to thrombophlebitis and not to deep venous thrombosis, so a comparison becomes less valid. (Drill and Calhoun, 1968) The incidence of coronary thrombosis has also been thought to be raised by oral contraceptives to the extent of being related to the dose of oestrogen used (Inmam et al; 1970) but other aetiological factors such as hyperlipidaemia and smoking have not been adequately excluded. (Oliver, 1970)

The statistical evidence at present is equivocal but nevertheless suggests that the oral contraceptive has some aetiological role in causing thrombosis. The results of large prospective studies currently being carried out will be necessary before this can be conclusively shown.

What then are the effects of oral contraceptives on the blood clotting mechanism? Plasma factors VII and X are raised after 3 months (Poller et al 1968) while factors VIII and IX do not appear to rise. (Poller and Thomson 1966; Hakim et al 1970) Platelet aggregation is increased when the platelets are exposed to ADP but not when exposed to noradrenaline, this being similar to the pattern observed in patients with atherosclerosis. (Bolton et. al 1968). Platelet aggregation was not increased in men who were given a natural oestrogen preparation—namely *Premarin* (Hampton, 1971). None of these changes occur with a progesterone alone hence suggesting that the oestrogen component is responsible for them.

The fibrinolytic system is also affected by oral contraceptives. Fibrinogen levels do not seem to be raised but fibrinolytic activity seems to be increased, because raised plasminogen levels have been found and the euglobulin lysis time is shortened in women taking oral contraceptives. (Brakman et al. 1970) Cryofibrinogen, which is an intermediate product in the partial breakdown of fibrinogen and fibrin, is increased. (Pindyck et al. 1970) Thus it is possible that the dynamic equilibrium between coagulation and fibrinolysis is maintained, but at an increased rate. However the pill by its action on the liver can raise the levels of a2 macroglobulin and of serum triglycerides, both these states diminishing fibrinolytic activity. (Barton et. al; 1970) In addition to raised serum triglycerides there is a rise in serum cholesterol levels, and there is some evidence that both these rises are dependent on the dose of oestrogen. Finally there is an increase in low density lipoproteins which may be caused by diminished lipoprotein lipase activity. It is this factor that may cause an alteration in platelet aggregation.

From this evidence one can postulate that the synthetic oestrogen in the pill, by inhibiting endogenous ovarian oestrogen production and by altering lipid metabolism, reduces the pre-menopausal woman's natural immunity to atherosclerosis and its complications, while substituting a state of hypercoagulability which is kept in check by increased fibrinolysis. A slight diminution in the latter may then be the cause of the thromboembolic complications.

The clinical significance of this is that no woman should undergo elective surgery while taking oral contraceptives. These should not be prescribed for anyone with a history of thromboembolism, nor for anyone with an increased tendency to clot formation, such as those with rheumatic heart disease.

Another aspect of altered metabolism which appears to be related to the amount of oestrogen used, is cortisol metabolism. Mestranol was found to increase unbound, protein bound and total cortisol levels. (Burke, 1970) The increased tissue exposure to unbound cortisol is small and probably not signficant, but it is this fraction of plasma cortisol that is responsible for hypothalamic control, and consequently any alteration of this level raises the question of long term interference, at the hypothalamic level, of cortisol homeostasis. Cortisol bound to a globulin may have access to hepatic cells, induce enzyme changes, and thereby be responsible for the altered production of proteins from the liver. This is perhaps one of the most fundamental physiological alterations that the oral contraceptives cause.

Carbohydrate metabolism is also altered by oral contraceptives, probably by the oestrogen component, and maybe secondary to increased cortisol levels. Mean fasting plasma glucose levels appear to be unchanged, but

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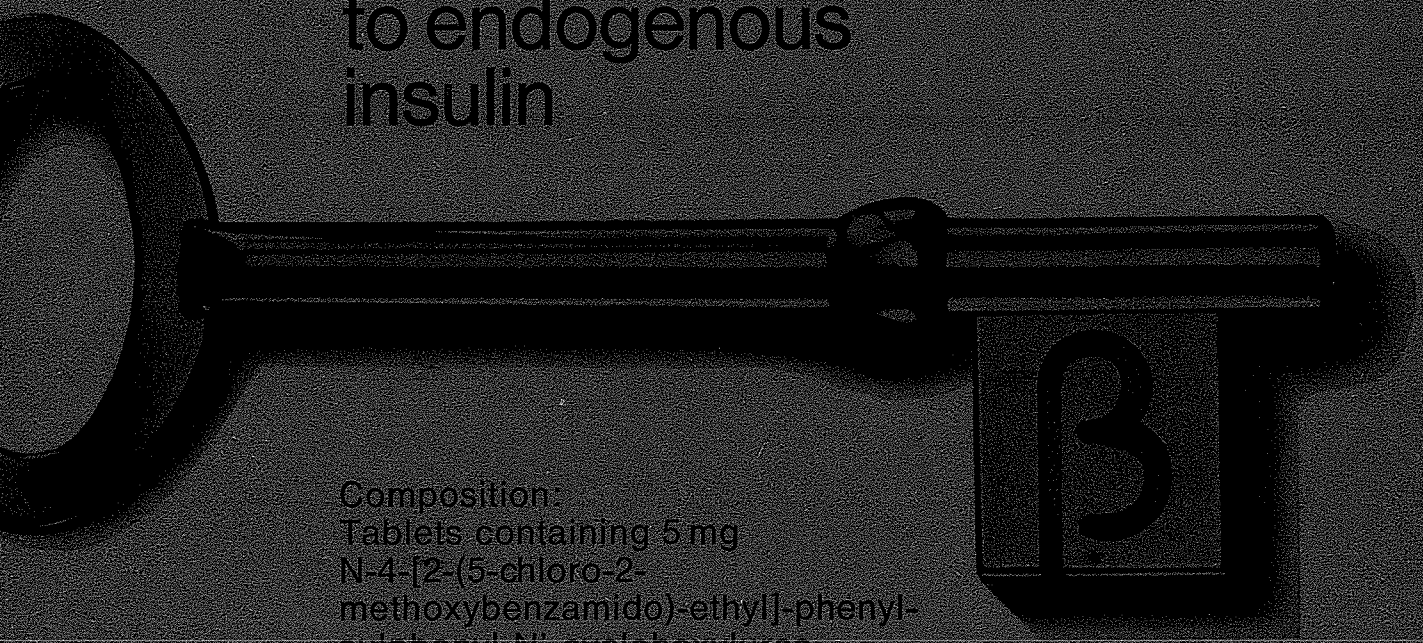

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both intravenous and oral glucose tolerance deteriorates. (Wynn and Doar, 1970) However 90% of cases rapidly return to normal on cessation of therapy. There is doubt about mean fasting plasma insulin levels, some authors showing a rise while others found them unchanged. There is no doubt, however that mean plasma insulin levels after both oral and intravenous glucose administration are higher during oral contraceptive therapy than those before therapy started.

There is evidence that as many as 77% of women taking oral contracptives for more than 8 years will develop abnormal glucose tolerance (Spellacy et. al; 1968) A family history of diabetes, previous large babies, or diminished carbohydrate tolerance during a previous pregnancy predispose to these changes, and certainly diabetes or a history of abnormal carbohydrate metabolism during pregnancy are absolute contraindications.

Liver function is altered as we have already seen, to the extent of producing increased amounts of certain proteins. In addition there is some impairment of transfer of substances from the liver to the bile, as manifested by up to a 12% retention of bromsulphthalein (Kleiner et. al; 1965) while 2% of patients have raised alkaline phosphatase levels due to alterations in the hepatic excretory mechanism. (Schaffner. 1966) A cholestatic jaundice occurs in some patients, preceeded by anorexia, nausea and pruritus, the serum bilirubin being in the range of 3 to 10mg.%. Microscopy of the liver at this stage shows dilatation of the bile canaliculi, with bile stasis. Histories of pruritus or jaundice in pregnancy are therefore contraindications to oestrogen containing oral contraceptives. A history of jaundice unrelated to pregnancy, or of infective hepatitis, is not a contraindication provided liver function is normal.

Finally there seems to be a slight rise in blood pressure among women on oral contraceptives. The mechanism involved is as yet unknown, but there is a rise in plasma renin substrate, which may in turn be caused by the oestrogenic stimulus to its hepatic production. Patients who are already hypertensive should not take oestrogen containing oral contraceptives, lest their hypertension is worsened.

Note: This paper was read at the Annual Clinical Meeting of the Association of Surgeons and Physicians of Malta, Nov. 1970.

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From the evidence described it is obvious that the oestrogen containing oral contraceptives have a fundamental and diversified effect on the patient's physiology. What the outcome of this in terms of iatrogenic disease will be only time and long term prospective studies will tell. However it is important to put the risk at the present time in perspective, first to deaths related to other contraceptives, and secondly to deaths from other causes.

Table I illustrates the relative risks of various contraceptive methods, with the death rate associated with their failure.

TABLE I

METHOD	Pregnancies per 1 million users	Deaths per 1 Million users caused by Pregnancy	Method	Total
Oral contraception	20,000	5	21	26
Intrauterine device	40,000	10	20	30
Condoms and diaphragms	150,000	33	0	33
Spermicides, coitus interruptus, etc.	250,000	56	0	56
Sterilisation, male and female	1,000	0	15	15
Unprotected intercourse	800,000	223	0	223

Table II compares the risks of death from thromboembolism due to oral contraceptives with the risks of death from other causes and in the group more susceptible to thromboembolism i.e. 35-44, the risk is comparable to that of dying in a car accident and much less than the risks associated with pregnancy, delivery and the puerperium.

TABLE II

Death per 100,000 Women from	AGE 20-34	35-44
Thromboemoblism in oral contraceptive users	1.5	3.9
Cancer	13.7	70.1
All risks of prenancy delivery and puerperium	22.8	57.6
Motor Accidents	4.9	3.9

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the abo blood groups and disease

martin anthony - williams
final year medical student

The discovery of definite associations between the ABO blood groups and disease, apart from certain antigenic phenomena, has only been made in the last two decades. In a few conditions an association has been very well established. *Aird, Bentall and Roberts* (1953) were the first to produce strong evidence to show the disadvantage of group A in cancer of the stomach, and *Aird et al* (1954) that of group O for peptic ulcer.

The advantages and disadvantages of belonging to one or other ABO blood group are being slowly established but the cause of the association between blood groups and certain diseases is still unknown, although, in the case of peptic ulcer it has been suggested that group-specific substances (A, B or H) in the gastrointestinal secretions may exert a protective influence.

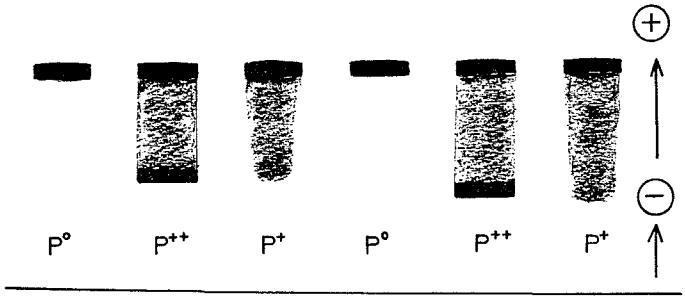
Secretor Character

Some individuals not only have the ABO blood group antigens on their red cells but also in their body fluids, and when this is the case they are termed "secretors". Secretion and non-secretion are inherited characters, the former being dominant to the latter. In Britain about 78% of the population are secretors and 22% are non-secretors. Saliva, gastrointestinal juice and semen have particularly high concentrations of the antigens.

Group O secretors secrete H substance only. Secretors of other blood groups secrete not only their group-specific substance i.e. A, B, or AB, but H substance as well. Non-secretors of ABH do not however have "nothing" on their red cells but usually possess the equivalent amount of the Lewis blood group antigens, i.e. the non-secretors of ABH are generally secretors of Lewis (Le^a).

Alkaline Phosphatase, diet and the ABO blood groups

Using starchgel electrophoresis two different patterns of alkaline phosphatese (both under genetic control) have been discovered, although the method of inheritance is not clear *(Arfors et al., 1963)*. All human sera show one alkaline phosphatase band, but sera from some individuals show also a second slowly moving band. This double band is found much more commonly in those subjects who are group A or AB. Furthermore double band persons are nearly always secretors of their particular ABO blood group substances.



Some electrophoretic patterns of serum alkaline phospatase. (adapted from **Bamford** *(1965), Lancet* **I**, *530.)*

Bamford et al (1965), using a more delicate technique showed on electrophoresis of the serum of 468 British blood donors that about 33% had a weak second band which had not been previously detected (FIG. 1). The population could therefore be divided into three groups — those with no second band (P°, about 50%), those with a weak second (P+, about 33%), and those with a strong second band (P++, about 17%). This second band is found much more commonly in individuals of blood group O or B than those of A or AB, and people of these blood groups had on the average much higher serum level of alkaline phosphase than those who were group A. Furthermore, double band individuals whether P+ or P++were nearly always secretors of ABH substances.

Following earlier observations that serum alkaline phosphatase levels may vary with time in the same individual, the effect of a fatty diet was studied by *Langmen et al* (1966). In 24 secretors of blood groups O or B, one individual was graded as P++ in the fasting sample, five were P++ four and a half hours after a fatty breakfast, and 18 P++ seven and a half hours after, having also eaten lunch. In five secretors of blood groups AB the changes were similar but less marked. In 14 secretors of blood group A there was very little change — as was also seen in the non-secretors.

These findings suggest that there may be differences in the physiological behaviour of the epithelial cells of the small bowel in people with differing ABO blood groups and that the blood group antigen has something to do with the formation of an enzyme. If this is true of alkaline phosphatase it might be equally well be so of other enzymes of the gastrointestinal tract and its appendages.

The enzymes responsible for the first band P° originates principally in the liver and it is probable that the second band (P+ and P++) is quantitatively different and derives from the jejunal mucosa (*Clark*, 1969).

It seems likely that this may provide a clue to the cause of the association between blood groups, secretor status, and certain gastro-intestinal diseases, such as duo-

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denal ulcer with group O, cancer of the stomach and pernicious anaemia with group A.

ABO blood groups and disorders of the gastrointestinal cancer of the stomach

In a search to pin-point the mechanism by which certain genes can influence the susceptability of an organ for cancer, *Aird et al* (1953) investigated the relationship between ABO blood group and cancer of the stomach. This disease is more prevalent in northern parts of England than in the South, as is blood group O. The expected association between cancer of the stomach and blood group O was not found. It was found however, that both in the North and South blood group A was more common among patients with cancer of the stomach than among the general population or than hospital controls (see Table 1). Individuals who are group A are 20% more liable to the disease than those of other blood groups. The association with pernicious anaemia, which is also high in group A, is of interest.

TABLE 1

Total of	Cancer cases %				Controls %			
	O	A	B	AB	O	A	B	AB
North of England; (1406 cases)	42.9	46.4	7.6	3.1	50.7	39.3	6.8	3.2
London (478 cases)	43.1	46.0	7.9	7.9	45.8	42.2	8.9	3.1

Percentage distribution of blood groups in cases of carcinoma of the stomach compared with that in equal number of controls. (Aird et al, B.M.J., 1953)

Working on an immunological basis *Barber and Dunsford* (1959) demonstrated a very high concentration of A substance in the serum of a patient dying of carcinoma of the stomach. However more recently a "normal" non-secretor frequency has been found in carcinoma of the stomach (*McConnell* 1966).

Several other associations with blood group A have been suggested particularly for diabetes mellitus, pernicious anaemia already mentioned above, cholelithiasis and portal cirrhosis. A moderately significant excess of blood group A has been observed in carcinoma of the pancreas. (*Aird et al*, 1960). *Cameron* (1958) has also found a high incidence of blood group A in patients with salivary gland tumour. A slight increase of blood group A in 610 patients has been noticed in patients with cancer of the oesophagus.

Peptic Ulcer

The relationship between blood group O and duodenal ulcer is well documented. It is complicated however, and not yet fully understood.

It is known that there are more peptic ulcers in relatives of patients with gastric and duodenal ulcer than would be expected in a study of the general population. If a gene or genes were responsible, no known mechanism of selection or inheritance could have caused such a rapid increase in frequency.

TABLE 2
PERCENTAGE GROUP FREQUENCY

Group	Peptic Ulcer (3011 Cases)		
	Control	Disease	Increase or Decrease on Control
	%	%	%
O	47.00	55.40	+17.9
A	40.99	34.67	−15.4
B	8.98	7.44	−17.1
AB	3.03	2.49	−17.9

adapted from Aird et al.

In duodenal ulcer two genetic components are of importance; those at the ABO blood group, and the secretor status of the patients. Thus blood group O individuals are about 40% more liable to duodenal ulcer than are those of group A, B, or AB (Table 2), and non-secretors are about 50% more prone than are secretors.

TABLE 3

% of non-secretors in 1014 cases of Duodenal ulcer and 851 controls

	Group O	Group A, B, & AB
	%	%
Male ulcer	37.20	35.10
control	21.10	24.90
Female ulcer	40.00	36.20
control	24.20	26.20

adapted from Clarke et al.

If the characters are considered together, it is evident that individuals who are both group O and non-secretors are about two and a half times more prone than the least susceptable groups, i.e. groups A, B, or AB. (Table 3). Stomal ulcer has the highest association of all with group O, and also shows an excess of non-secretors (*Doll et al*, 1960) Table 4.

TABLE 4

Distribution of ABO Blood group among stomal ulcer, duodenal ulcer, and control subjects.

Disease Category	Blood groups of subjects				Total number of subjects
	O	A	B	AB	
Stomach ulcer	298	214	39	13	564
Duodenal ulcer	181	96	18	5	300
Control subjects	4,578	4,219	890	313	10,000

Adapted from Doll et al.

The way in which the blood group genes influence susceptability to duodenal ulcer was not previously known but recently *Hanley* (1964) has produced evidence to show that blood group O patients have a higher serum pepsinogen level and a greater gastric acid secretion than average, besides a larger secretory cell mass. It has also been suggested that Lea substance or its degeneration products may be absorbed from the stomach and stimulate hyperplasia of the parietal cell mass.

Various large series indicate that blood group O is associated with severe or complicated duodenal ulcers,

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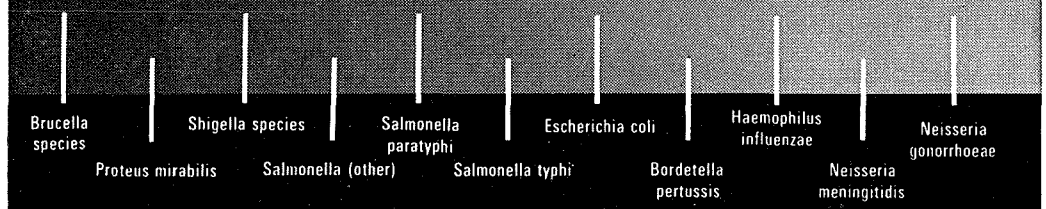

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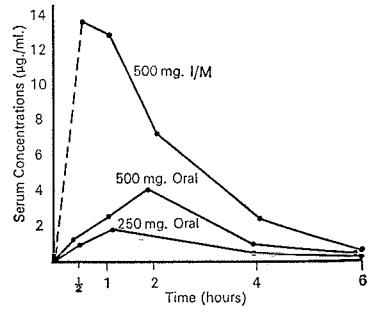

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TABLE 5

BLOOD GROUP ASSOCIATIONS WITH DISORDERS OF THE G.I. TRACT

Condition	*Blood Group*	Remarks
Cancer of the stomach	A	*Firm association.*
Duodenal ulcer	O	*Firm association but more marked in non-secretors.*
Gastric ulcer	O	*Firm association but less marked than in duodenal ulcer.*
Stomach ulcer	O	*Firm association. The highest association of all with blood group O.*
Pernicious Anaemia	A	*Marked increase of group A, but number very small.*
Diabetes Mellitus	A	*Evidence equivocal.*
Salivary gland tumour	A	*Evidence fairly strong.*
Cholelithiasis	A	*Fairly firm association.*
Carcinoma of pancreas	A	*Moderately significant excess of group A.*
Portal Cirrhosis	A	*Marked increase of A, but numbers very small.*
Chromophobe adenoma of the pituitary (derivative of the foregut)	O	*Not proven.*
	A	*Not proven.*

Adapted from Genetics for the Clinician — C.A. Clark.

that is to say duodenal ulcers that bleed, giving rise to haematemesis and/or melaena, or perforate. Some workers doubt this relationship but they appear to be in the minority.

Other associations with blood group O have been noticed. *Mayr et al* (1965) found a large excess of blood group O in chromophobe adenoma of the pituitary gland (a derivative of the foregut) but *Damon* (1957) in a smaller series, and *Aird et al* (1960), in the largest series, found no such excess.

Rheumatic fever and rheumatic heart disease

Host susceptability obviously plays a significant role and evidence is now strongly in favour of there being a genetic constitution which favours the rheumatic response to streptococcal infection. *Glynn* and his collegues (1956) pointed out that the growth of streptococci in the throat might be modified by the secretor status of the patient. The features of the rheumatic constitution show a significant excess of ABH saliva non-secretors and also an excess of group A.

The most susceptible individuals seem to be females who possess blood group A or B with non-secretor status.

Antibodies and Resistance to infection

It has been shown that antibody levels vary between the blood groups. Antibody to strain 086B7 of E. coli is universally present in man. However, the serum levels of the antibody have shown to be highest in individuals of blood group AB and lowest in group O.

This may be because group O is less able to produce the antibody or less susceptible to infections by E. coli. It has been suggested that if there were cross antigenicity, individuals of a particular ABO blood group might be at a selective advantage in combatting infection due to an organism carrying a cross-reaction antigens.

It is well established that some strains of micro-organisms have heterophile antigens, e.g. some strains of E. coli. 086 have a powerful group B-like blood antigen and following infection with this organism, greatly elevated levels of anti-B may be found in the sera of the patients.

Susceptibility to infectious diseases

One study has shown that children of blood group A in the west of Scotland are at a greater risk of death from bronchopneumonia than those of other blood groups. An excess of group O and a deficiency of group A have been demonstrated among sufferers from A2 influenza virus infections, and an opposite trend has been shown in the case of adenovirus infections. An excess of group A has also been shown in sufferers from infectious hepatitis.

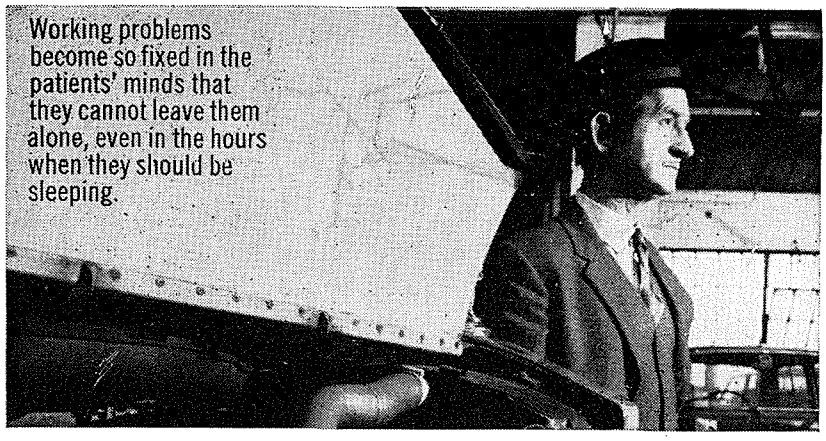
Helmbold, in India (L960) pointed out that smallpox scars appear to be more severe in group A and there is a slight excess of smallpox cases with blood group A. He also pointed out that vaccination induced-encephalitis is two times more common in group A individuals.

Vogel et al (1960) have shown that individuals of blood group O are much more likely to develop plague, as geographic evidence in areas such as India and Outer Mongolia bears out.

Susceptibilty to Chronic Diseases

Lewis and Wood (1961) found that there was a significant difference in blood group distribution between patients with sarcoidosis and with tuberculosis, seen at the Brompton Hospital. The 164 sarcoid patients showed an excess of blood group A and a deficit of group O, 40% belong to group O and 51% to group A, as compared with 48.8% and 38.9% respectively for 894 tuberculous patients.

Fergeusen and Wurm (1964) studied the ABO blood groups in 518 sarcoid patients in Germany. They found that the percentage of group A was 4.81% higher than was found in the unspecified German control groups, this excess was at the expense, principally, of group B, and to a less extent of group O. From these figures it was



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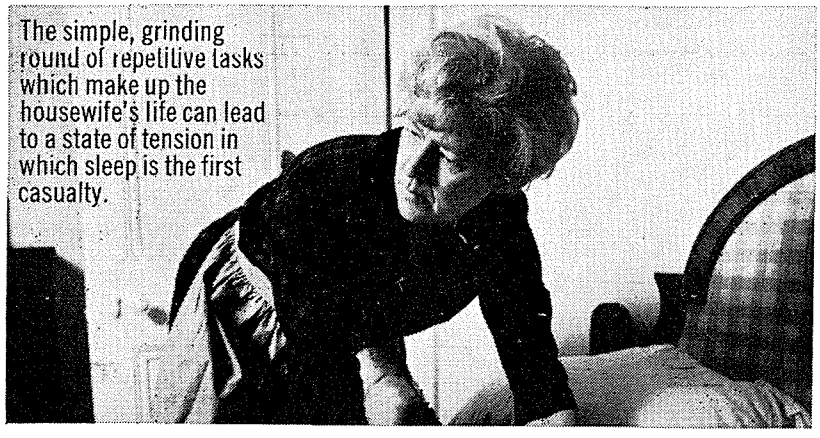

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estimated that group A subjects were 14.2% more likely to have sarcoidosis than group O. Unlike *Lewis and Wood*, they found a slight predominance of group A in tuberculosis as well as in sarcoidosis.

The other association mentioned in Vogel's paper is a possible relationship between syphilis and the ABO blood group. He pointed out that people with A, B, or AB group have a 67% higher chance to stay seropositive after treatment than group O. As treatment is increased, this proportion increases further. This shows that syphilites with blood group O are more likely to develop a milder disease and therefore there would be more group O in areas of epidemic syphilis.

Conclusion

Finally, conclusive evidence of an asociation between the blood groups and disease has been shown. Most of the diseases which show an association with a particular ABO blood group appear to be *diseases of the upper part of* the *gastro-intestinal tract and its appendages*. Carcinoma of the stomach and pernicious anaemia, occur more frequently in persons with blood group A than in those with other blood groups; peptic ulcer more frequently in those of group O. In addition to the association for gastric cancer and pernicious anaemia, there is evidence of the association between *rheumatic heart disease* and blood group A.

The relationship between the ABO blood groups and susceptibility to virus infection and chronic infection is not a very specific one, but there is enough evidence to show that the blood groups are not genetically neutral but may actually have a bearing on disease suscepitibility in general.

Similar interrelationships between other blood groups and disease may also exist and need to be studied further.

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malignant change in gastric ulcer

alex felice

final year medical student

The co-existence of ulcer and cancer in the same stomach may be revealed by examination at autopsy or at surgery. The histological features of chronic gastric ulcer and gastric cancer may be described in the same lesion under microscopic examination.

Two questions arise:

What proportion of gastric cancers are casually related to chronic gastric ulcers?

Secondly, what proportion of chronic peptic gastric ulcers run the risk of malignant change?

Muir estimates that there is evidence of chronic gastric ulceration in about 18% of gastric carcinomas. In judging of the relationship, there must be clear evidence of cancer in connection with an ulcer, as there is often irregular growth of epithelium and even displacement of epithelium or heterotopia at the margin of a healing ulcer, apart from any malignancy. As to the pre-existence of chronic ulceration in an undoubted case of carcinoma, the important points are that there is a long history of chronic ulcer pain and that the muscular coat has been entirely destroyed and replaced by fibrous tissue in which there is often obliteration of arteries. Carcinomatous change begins in the margin of the ulcer crater and tends to encircle it, spreading outwards into the submucosa and the muscular layer, but not invading the fibrous floor to any extent.

On the contrary, whilst primary carcinoma of the stomach often invades the muscularis, it practically never destroys it entirely and even in advanced cases, remains of muscle may be detected in between the neoplastic tissue. Characteristically; Ulcer *destroys* muscle, Cancer *invades* it.

Though most cancer ulcers are large, usually being larger than 4 cms., at least 10% of benign ulcers may be larger than that.

Both cancer and ulcer most commonly inhabit the prepyloric region, though the former prefer, the greater curvature and the latter the lesser, however, as many as 30% of gastric carcinomas are found on the lesser curvature, where they tend to be larger, some having huge excavations.

The ulcerative type of gastric carcinoma differs anatomically in many respects from its benign counterpart.

Classically they have a heaped up, beaded, firm, overhanging margin on a shaggy necrotic base. Histologic evidence of neoplasia is found in the margins and in the base of the ulcer. The ulcer defect seems to be carved out of the tumour.

When a peptic ulcer becomes malignant, the transformation occurs at the mucosal margin and very often, if the neoplastic process is not far advanced, the base remains free of tumour infiltration. The presence of anaplastic glands in the base of the lesion is one of the distinguishing features that helps to differentiate the ulcerative type of gastric cancer from the malignant transformation of a previously benign peptic ulcer.

One may perhaps draw an analogy between cancer arising in the scar tissue of a chronic peptic ulcer and chronic burn scar carcinoma. Differences in treatment which account for delayed re-epithelisation is thought to explain the rarity of the condition in the U.S.A. and its relative frequency in the Orient and the Middle East. Repeated or persistent ulceration of the gastric mucosa may take a similar course.

Most authorities would agree that in an ulcerating gastric cancer, evidence that it arose from a previously benign chronic ulcer is given by:

1. Complete destruction of the muscle layers of the stomach in the base of the ulcer.
2. Fusion of the muscularis mucosa and the muscle wall at the margin of the ulcer.
3. Intimal thickening of the blood vessels
4. The presence of cancer in only one part of the wall and its infiltration in the base of the ulcer and in other parts of the wall.

Differing criteria of diagnosis have led to widely different estimates of the incidence of ulcer-cancer. Presently, the pendulum seems to have swung to lower values averaging below 10%, sometimes much below this. This is a far cry from original estimates of over 7%. *Robbins* considers the incidence of malignant change in a benign ulcer as being of the order of 1%. He concludes that it is probably not justified to call peptic ulcer a pre-malignant lesion.

Willis suggests that chronic ulceration perhaps does no more than determine the site of neoplasia in a stomach already prepared for it. He also takes into consideration the fortuitous existence of ulcer together with cancer.

Hellsingen and Hillestad in Norway have shown that in patients with peptic ulcer, the later development of gastric cancer was three times as high as the expected or anticipated incidence in the general population within the same age group.

Bailey and Love suggest that malignant change occurs in 4% of gastric peptic ulcers and when it does so it is limited to the ulcer and the mucosa around it.

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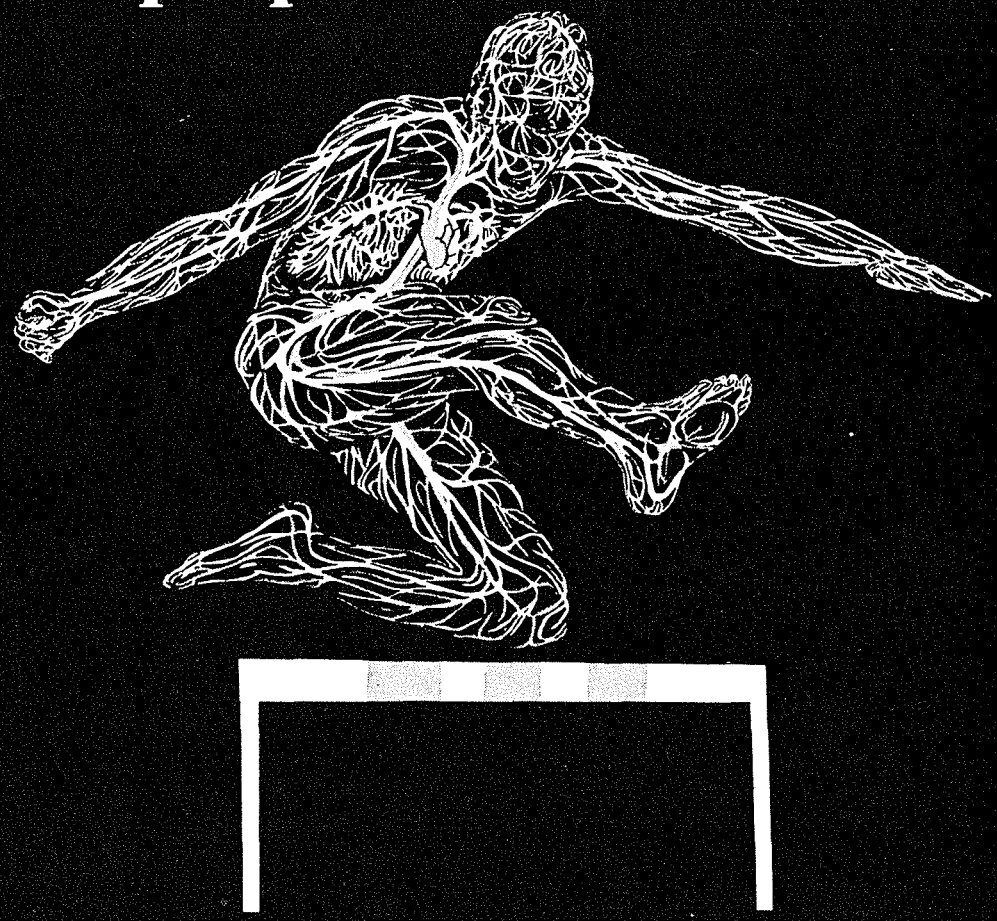

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However much one may argue for or against the relative incidence of malignancy occuring in a gastric ulcer, the possibility in the individual case must be considered.

Recent investigations have been directed towards the study of enzyme activity and mucopolysaccharide content of the gastric mucosa and the gastric juice in cases of gastric carcinoma and atrophic gastritis. Though controversial, some authorities regard the intestinal metaplasia as seen in atrophic gastritis as precancerous and others view hypertrophic gastritis similarly. A marked increase in the glucose content of the gastric mucus and an increase in the concentration of gastric juice B-Glucuronidase is described in malignant aspirates. Other observations correlate with the general concept that cancer arises in stomachs that have been the site of previous injury. Mucosal injury might initiate cellular mutation or facilitate contact between tissue and ingested carcinogen.

The differential diagnosis between benign and malignant ulcer and malignant change in a previously benign ulcer, raises several difficulties. All stomach derangements evoke a fairly limited range of clinical manifestations. At least 10% of gastric ulcers defy clinical classification as benign or malignant.

A duodenal ulcer may be safely assumed to be benign and is more closely correlated with hyperchlorhydria. Similarly, when a gastric and duodenal ulcer are present simultaneously the lesion in the stomach is invariably benign.

Adequate medical treatment may be a valuable therapeutic test. An uncomplicated benign peptic ulcer should heal in three weeks under a suitable therapeutic regimen.

Age of the patient is not of great diagnostic value.

A long history is said to favour the diagnosis of benign ulcer whereas a short one favours malignancy.

With the onset of cancer in a person known to be suffering from chronic gastric ulceration of some duration there is loss of periodicity or regularity of daily painful episodes. The attacks apear to worsen progressively, and intervals between attacks to shorten. The pain is mild but constant and without postprandial relief.

Anorexia and nausea are usual features of malignant disease. Vomiting no longer gives relief. Early diagnosis may be possible if the growth gives rise to obstructive symptoms. Emaciation, anaemia and tumour mass appear at a late stage in the disease. Loss of weight is initially slight but is prorgessive with advance of the disease.

Gastric chemical analysis shows that 51% of primary carcinomas of the stomach are associated with achlorrhydria, and 33% of ulcer-cancer and 81% of chronic ulcer patients are isochlorrhydric. The persistence of achlorrhydria in the presence of an ulcerating gastric lesion is highly suggestive of carcinoma.

The characteristic sign of gastric ulcer is the crater on radiology — the niche of Haudlek. An ulcer crater on the lesser curvature, smooth in outline, projecting beyond the normal outline of the lumen with uninterrupted rugae converging towards it is most likely to be benign. It is claimed that the smaller the diameter the more likely it is to be benign.

Cancer developing in the edge of a previously benign ulcer cannot be detected in the early stages. At a first examination, the ulcer which has undergone malignant change reveals a large crater which should arouse suspicion if larger than 4cms. If seen, the meniscus sign of Carman, which apears as a halo, is pathognomonic of gastric carcinoma. The crater, then protrudes inwardly as a filling defect with irregular contours. Stewart proposes serial radiography in suspect patients as the most satisfactory assessment of ulcer change to cancer.

Gastroscopy will reveal and define a new growth. The points in favour of malignancy are;

1. A raised irregular edge.
2. An uneven base containing blood and necrotic tissue.
3. Irregularity or nodularity of the converging folds.
4. Raising of the whole ulcer above the level of the surrounding mucosa.

Early diagnosis is important if the results of surgery are to be improved. Sometimes this may be impossible even after radiological, endoscopical and cytological investigation. Most important in this evaluation is the probability of the disease in terms of its frequency.

The medical treatment of a supposedly benign peptic ulcer should be followed up by repeated roentgenographic and endoscopic examination. The reliable data of healing in a benign peptic ulcer are;

1. Complete absence of occult blood in the faeces.
2. Progressive decrease in the size of the ulcer and its disappearance on X-ray examination.
3. Complete healing of the crater.

Delaying surgery in an uncomplicated, non-healing ulcerating lesion of the stomach involves serious risk to the patient and is, in all probability not justified, if the patient is operable.

communication

tonio j. bugeja
third year medical student

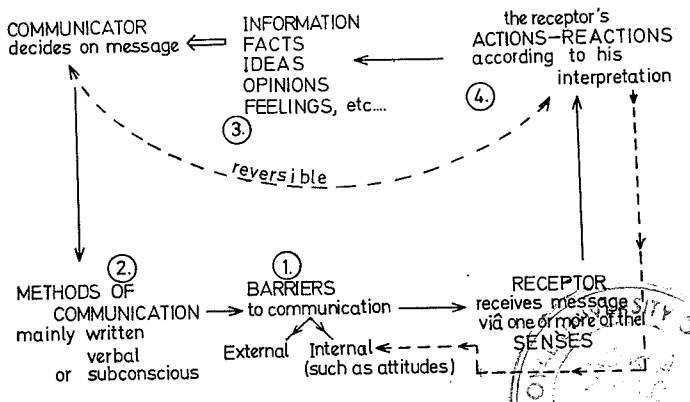
INTRODUCTION: *Peter Little* has called "communication" a chameleon word — one that changes its meaning and application with each person who uses it. The *Concise Oxford Dictionary* definies the word as follows: "Communicate — to import, transmit, share". The nature of communication is very complex and we shall here discuss only the practical aspect of communication as the *PROCESS* governs human relations. The value of efficiency in practical communication to the medical profession cannot be overemphasised; this is inclusive of such specific activities as reading a paper, conducting a meeting or an interview (a viva voce or oral examination, or what you may choose to call it) apart from the all important confrontation with the patient.

THE COMMUNICATION PROCESS: This on analysis proves to be a closed loop or feedback mechanism which may fail or disrupt at one or more points.

This model implies that any communication essentially entails a communicator and a receptor, and that these positions are reversible during the process, so that a discussion of one reveals as much of the second. We will therefore review positions one to four in the diagram under the following respective headings:—

(1) Barriers to Effective Communication.
(2) Clear Thinking; Verbal and Written Expression
(3) Information Sources
(4) Interpreting and Recording Information.

1. *BARRIERS TO EFFECTIVE COMMUNICATION:* This is the weakest link in the loop and hence reflects not only the strength of the process but also the general causes of so much "interference with our transmission". These barriers are in general either external noise, heat and cold; or internal pressures (arising out of lack of knowledge about ourselves and other people) that make us at odds with ourselves and others. Our contacts as individuals are so few and so varied that we have little real experience of people. Moreover human behaviour is like an iceberg — always only barely visible.



The cause of many unexpected reactions to a message is the conflict in a person between his thinking and his emotional side. The ability to think rationally varies a lot from individual to individual, and both thoughts and emotions have a say on his eventual behaviour. But it is when the latter are in control that he distorts information and reacts to a message in a way not intended. This may be subconscious but if the person is aware he will try to justify himself by "reasoning" and finding excuses for his behaviour.

Emotional reactions are the source of irrational behaviour which comes into action when we meet with something quite new. Our personal survival is at stake, and accordingly we either fight or flee. It is here obvious that we need to control our instinctive desire for otherwise we would be at the mercy of every circumstance. Moreover if one could not exert social control, society itself would become a jungle since more of us live a life apart from other people. When the receptor takes in the message he makes a mental pattern of the available information. This, however, is not a photographic reproduction but a picture largely determined by experience, expectations and wishes. Concepts are therefore highly personal and this limits each of us. Our previous experience is in its turn limited by such things as abilities and opportunities.

People also act strangely because of preconceived ideas associated with previous experience. These attitudes often determine the character's behaviour in a specific circumstance. It is important to realise that however rational an attitude appears it has an emotional support. This emotion supplies the driving force of our behaviour and can create the most difficult barrier to effective communication. It follows that we can never ignore attitudes in ourselves or others. When we wish to impart something to someone we must identify attitudes as early as possible so that we can predict what reactions he is likely to have if we approach him.

2. *CLEAR THINKING, VERBAL AND WRITTEN EXPRESSION:* The expression "clear thinking" does not tell us a great deal; it would be more instructive to observe that unclear thinking may stem out of supersti-

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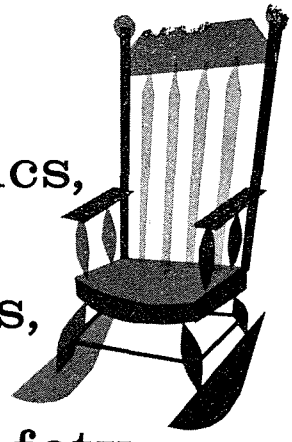
pediatrics,
to
geriatrics,
when
speed and safety
are desired



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tion, self-interest, laziness or fallacies of relevance or of ambiguity. The first two of these are simple; as for the third, all of us tend to grow lazy and prefer to accept certain traditional beliefs without adjusting to changing times; we tend often enough to fall with the majority and cease of think independantly. *Jepson* in his book "Teach Yourself to Think" says: "As long as we neglect the duty of thought, all sorts of beliefs will appear in our minds... we shall be at the mercy of anyone in whose interest it is to manufacture them".

In fallacies of relevance the communicator introduces ideas that have nothing to do directly with the latter being discussed. In doing this he plays on emotions such as pity, fear, reverence, disapproval, enthusiasm or *force*: At the Yalta Conference, Churchill told Stalin and Roosevelt that the Pope suggested a certain course of action to which the Russian leader replies "And how many divisions has the Pope?".

In fallacies of ambiguity the speaker changes the meanings of words he uses during the course of an argument, to gain his own ends. Thus it is obvious that most of our thinking is not good enough, the trouble being that the arguments are often the result of personal wishes and the energy we put into trying to make them come true. It is only by reasoning — the making of inferences and the drawing of accurate conclusions — that enable us to support and sustain our arguments and demolish unfounded ones.

Verbal communications can break down and often does for a number of reasons: The major one is overconfidence; we know that we have managed to get by with the aid of the spoken word for so long and through so much of life that we take the spoken word for granted: one is then shocked to learn that over 50% of our communication is misunderstood, rejected, distorted or forgotten. Through haste we often open our mouths and think afterwards so that it is only when the results are obvious that we realise a mistake. Clear thinking is not only required but required before one does or says something and not as he does so. Poor presentation (both in the literal sense as say talking at a distance, and in the metaphoric meaning) is another vital consideration. The very words we use must be well chosen, something which is not so difficult with experience; the sentences we compose must be simple and straight forward with emphasis laid in the proper places. Something more positive is to check whether one has understood your communication or that you understood what you are told. As to the latter case, listening is an art difficult to acquire. It needs patience, flexibility of mind, willingness to forget for the moment what you are thinking and to focus your attention on what the other person is saying. A determined effort must be made to understand the massage and to check if necessary on any equivocal or doubtful points. Verbal communication is even more advantageous if it is to face-to-face for this gives the communicator his best chance of making sure that the listener knows what he has to do and that he is able to do it. Provided one takes advantages of this, uses the feedback that comes back through the looks, changes in the face and answers to questions, verbal communication improves considerably.

In the medical world paper-work procedures are an important way of reading a wide, scattered and varied number of receptors; people to whom it is impossible to speak personally. The piece of paper may also be used as a record to be read by you or there later on. This system though essentially a good one, depends however on the "writing skill" of the people using it. Through this medium one can build a picture with the relevant information, one can analyse a situation or persuade readers on to his way of thinking; the communicator can alternatively easily baffle, annoy and frustrate the receptor and you cannot be at the readers's elbow to explain or develop the ideas behind your words — this would otherwise boil down to face-to-face contact. The whole trouble is that you have to use words and often even worse symbols. Sometimes the meaning is quite clear, and yet the desired effect is not reached. This is in sharp contrast to verbal expression when one can modify what he said to get where he wants or alternatively inch himself slowly till he leads the receptor precisely to the reaction intended. For effective written communication one must have a purpose and he must frame his information on one good structure with the right approach and layout Thus the purpose of note-making may be that of an aid to memory, analysis and clear thinking.

3. *INFORMATION SOURCES:* These are varied but the information one requires in any circumstance does not fall into his lap if he merely sits back and waits for it. One must develop and exercise good powers of observation. Knowledge must be sought out with something next to the newspaper reporter's "nose for news" and yet one must behave in such a way as to attract incoming communication. The reporter's standard questions: What? Who? When? Where? How? Why? often come in handy.

Information required whether in a committee room, a lecture room, the ward or the patients' house comes from four main sources:—

1. *Physical objects and events* — one must become very observant of his physical surroundings especially when they look the ordinary. Methodical accurate and complete observation comes with self-training.

2. *People and their actions* — A "Sherlock Holmes" attitude is here likely to breed a similar attitude to people. One could actually go on for ever on this; it is however worth pointing out that provided you do not jump to conclusions (for first impressions may be misleading) one can observe people's conduct to pick up clues to their thoughts and feelings. It is useful to notice the communication which people send out despite themselves: an

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involuntar gesture perhaps, a fixed or a fleeting facial expression or fidgety movements.

3. *Reading* matter: this can be as varied as the fish in the ocean and I do not propose to discuss anything except the ways whereby one can communicate effectively with this information source. These are in ascending order of thoroughness:—

Reference — as for dictionaries, directories and statistical compendia;

Scanning — looking for some topic which may be in a book especially by using the index;

Skinning — picking up the general drift without absorbing all the details; here one makes use especially of any headings and illustrations; of the openings or "signposts" to the paragraphs and of just the "food" in the chapter.

Reading for meaning — getting a broad impression but leaving examples to fall by the wayside.

Study reading — reading and rereading so completely that you can reproduce it in your own words. Note making or summaries often come in here.

Critical reading — forming an opinion of a text after a good study of it to compare your ideas with those of the author.

One can even apply these methods consecutively to the same reading material.

4. *Finally the mass media*: These today are no less important as a source of medical information and communication.

4. *INTERPRETING AND RECORDING INFORMATION:* Making sure of the facts is one of the marks of the scientific mind. As far as other people's statements go if these arouse strong feelings in you because you have other views, you may fail to register exactly what they would like to convey to you. It has been truly said that "A mark of the mature mind is the ability to read and understand statements which are in contradiction to one's own beliefs". Vagueness often characterises many expressions which come readily to lazy or hasty minds and these expressions must therefore be probed, measured and inquired into.

Though this tends to apply mostly to verbal expression it can be equally important in written communication. To make sure of the facts and possibly of the emphasis in some statements (that is if these have been framed well enough) one ought to reread and stop to consider parts of this (if this warrants such a treatment of course). The fact that we are living in the twentieth century when time is reckoned in seconds only means that the communicator should present his message concisely and in a coherent manner to facilitate understanding, interpretation and recording; it also implies that the true value of the communication is to be weighed as accurately as possible to eliminate serious waste.

In this second step — that of evaluating the significance of the facts and seeing what valid inferences if any can be drawn from them, a scientific approach is also worthwhile. What scientists do is to form a provisional theory — a "hunch" which is then tasted, modified or even rejected. In practical everyday life one hardly needs to go to such lengths, though the scientifically minded do most of this subconsciously and in good order.

CONCLUSION: The meaning of practical communication has been analysed very briefly and in rather perhaps a too superficial manner. Yet the reader is expected to appreciate its exact value; indeed the topic derives its worth from the current social and economic waste, inaccuracies, and trouble, it can eliminate when well understood and practised; it is the subject to be introduced in any university course with no little gain.

Observation: No apology is made for lack of examples, any member in the medical field has sufficient personal experience which amply illustrate the points being made.

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"The Doctor who smokes is like a clergyman with a mistress" — *Professor Lawther, of Industrial Medicine of London University.*

malta medical students' association

EXCHANGE DEPARTMENT

The Exchange Department of the MMSA this year has been active from the start, both in its Outgoing section — in trying the very hard task of persuading the Maltese medical student to clerk abroad in summer, and in its Incoming section — in trying to meet all the demands of foreign medical students clerking at our Hospital.

One of the aims this year was to try and seek more clerkships for our preclinical students. No co-operation in this field could be offered by foreign medical students' associations pending our re-membership with the IFMSA, though they all offered to help this coming year. We had thus to ask help from certain medical schools with whom our Association has private contact, and Professor K. Hill kindly offered us two places for six weeks each at the Pathology Laboratories of the Royal Free Hospital. The eagerness of the preclinical students to clerk abroad was shown by the 14 applications for the two vacancies, which had to be drawn up by lots. Mr. J. Sant-Cassia and Miss K. Pearl were the two fortunate students.

Though clinical clerkships were available in nearly all European countries, only six *clinical* students took advantage of the offers, all six being in Great Britain. Mr. R. Farrugia-Randon, Mr. G. Agius and Mr. A. DeBono each spent a month at the Royal Free Hospital in London; Mr. Anthony Felice spent a month with Professor Ellis at Westminister; and Mr. J. Grech-Attard and Mr. A. Grixti-Soler (through the co-operation of Prof. G.P. Xuereb) each spent eight weeks with Professor Forrest and Professor Williams in Cardiff. Another three students viz: Mr. M. Tabone, Mr. C. Gauci and Mr. Arthur Felice arranged their own clerkships in Edinburgh, Cardiff and Rome respectively. Mr. C. Brincat's aim to clerk in Israel had to be abandoned owing to unforeseen circumstances.

This year the Incoming section of the Exchange Department had about 40 foreign medical students clerking at St. Luke's Hospital. About half of these came form the British Isles and the rest from other European countries including Holland, Belgium, Germany, Eire and Finland.

As in previous years the foreign collegues clerked in the various departments available at our hospital, every morning, six days a week. This year we also had the first pre-clinical clerkship attached to our Physiology Department. The majority stayed for a period of a month but we also had some students staying for two months or more.

Besides work and learning, the students also enjoyed a holiday of sun, sea and sightseeing. A number of activities were organised for them in order to make their stay here more enjoyable; these included a typical Maltese 'Fenkata', Bar-B-Ques and Folk Singing gatherings at the old University cellar. Some also took the opportunity of joining the many excursions organised by the National Students' Travel Service.

We would like to take this opportunity in thanking both our professors and consultants for the interest they showed in taking round and teaching the foreign students, and for the kind help and understanding they have shown us. A special thanks goes to the various Heads of Departments: Professor V.G. Griffiths, Professor J.V. Zammit Maempel, Professor G.P. Xuereb, Professor A.P. Camilleri and Professor W. Bannister.

All in all this has been a very busy year for the Exchange Department. We are sure that both foreign and local medical students have ended their clerkships with a high opinion of the hospitals they have worked in and with a better knowledge and experience of their medical students. We are very thankful that our Medical Faculty actively encourages these student exchange programmes and we hope that this year's Exchange Department has served both sides in the best way.

BERNARD ANASTASI
JOE GRECH-ATTARD
(Exchange Officers)

SPORTS —

WEDNESDAY SPORT

In the spirit that Sport is part of education, the Education Department of the M.M.S.A. organised a Wednesday afternoon snooker, table- tennis, darts and draughts competition for sixth-year medical students.

The big number of competitors showed that competition sport is of major interest in the Medical Faculty. Thanks must be given to Prof A P Camilleri who made free Wednesday afternoons possible, as it was on such days that the competitions were held at the Games-Room starting on the 3rd December 1969. This was also a good opportunity for a student get-together, one afternoon a week. The competitions went on in great spirit up to March 1970 and on the 7th of the same month a Prize giving, Pizza-and-Wine Party was held for both teaching staff and medical students. During the party Prof. A.P.C. presented the prizes, kindly donated by the Royal University Sports Club, to the winners. These were:

Draughts: Bernard Anastasi (1st), Richard Miller (2nd).

Table-Tennis: Benjamin Anazodo (1st), Anton Bencini (2nd).

Snooker: Arthur Felice (1st), Bernard Anastasi (2nd).

Darts: Richard Miller (1st), Bernard Anastasi (2nd).

BERNARD ANASTASI.

EDUCATION DEPARTMENT

IFMSA Symposium 1966

A Summary of Student Attitudes in Some European Countries

This summary of student opinions in some of the Europe's major medical faculties is presented with the hope of stimulating further discussions among the members of our own medical school on current developments in medical education. It is based mainly on data supplied by IFMSA bullettins.

It was presented to the MMSA in April 1970.

FINLAND

With an average of five students per teacher the student has the opportunity to listen and discuss a variety of individual opinions. As almost 75% *of the work is practical,* student interest is increased through participation, and learning is facilitated.

Students would like to see their anatomy course shortened and part of the time set aside to go deeper into subjects of which they felt that they have a rather sketchy knowledge.

As in many other countries, there is a tendency to compartmentalise preclincal studies resulting in a lack of integration and cohesion.

The entire new curriculum of the three Finnish Faculties of medicine is the work of student-staff committees.

Finnish medical students hope to sponsor closer integration between the basic sciences and clinical studies.

GERMANY

One notable and prominent feature of the German medical system is its *liberalism.* Within reason the student can alter his time-table to suit himself. He is free to attend other faculties and other universities. It is claimed that this liberalism enables the student to develop his interest and knowledge in particular facets of the course as he chooses.

Studies are not systematic and the failure rate is low.

Overcrowding at lectures and bedside teaching is a common complaint. Before the student can be granted the title of Dr. Med he must not only pass his examinations (which in Germany means a four-month period of assessment covering the whole curriculum) but also write a dissertation. The final examination may be taken as often as desired.

The NETHERLANDS

One great advantage which certain Dutch universities offer is a housemanship in Social Medicine. Under this scheme, students must work for a few weeks under the supervision of a general practitioner learning social practical medicine.

An integrated system of teaching in Biochemistry, Anatomy and Physiology is working well and students would like to see it extended to other branches of the curriculum as well. There is also a common feeling for closer integration of clinical and preclinical subjects.

Unfortunately there is a tendency to overteach data, quite a lot of which may be in fact irrelevant to practice.

One would like to have contact with the clinic at an earlier stage in the curriculum.

It would appear desirable that curricula should be so designed as to facilitate the incorporation of developments in medical science.

SWEDEN

In Sweden as in Finland, personal contact and a willingness on the part of the student to work make the student an effective and influential body. They occupy 50% of the places on the curriculum committee and few situations have been reported on which agreement has not been reached.

From Sweden comes another plea for improvement in the integration of teaching.

After the first four years students may time their courses to individual desires. This is regarded as an advantage after a period of intense and compact studies.

The feeling students have that they are not beeing forced to specialize in an added advantage.

SWITZERLAND

At present Swiss students work 40 hours a week but this is expected to be shortened.

Swiss students enjoy their liberalism and long holidays as these afford an opportunity to taste various branches of medicine, to follow their own particular bent, to prepare their dissertation and to undertake their obligatory military service.

It should be noted that Swiss students are taught Psychology at school.

It is recommended that Physics, Chemistry and Biology be taught with a medical bias in preparatory courses.

More practical work is required including small study groups and bedside teaching groups with a maximum of four students per teacher. A new curriculum was introduced in Switzerland in 1965. The Students' association is represented on the Curriculum Committee.

UNITED KINGDOM

Compared to most European countries the course of studies in the U.K. is short lasting six years with one year of internship.

Students look forward to the expansion of courses in general practice at present available only In Manchester and Edinburgh.

British students repeat Finland's and Sweden's plea for better integration of the subject matter and the former's wish for a reduction in the anatomy course which are held to be too long in most schools. The system achieves its aim of producing a basically educable physician but students hold that it fails at postgraduate level due in particular to young doctors beeing overworked and therefore without time enough to dedicate to further studies.

FINANCING OF MEDICAL STUDIES

FINLAND

Students are mainly privately financed although there are a few state scholarships. Graduates form a society which acts as gaurantors for student loans from either a bank or the University. After the fourth semester, students can legally work, and earn as doctors during the vacation. Medical education is practically free but students must pay for the materials they use.

GERMANY

Fees are between 150-200 DM per semester. Almost 40% of medical students have government grants. These are repayable over a long period after graduation up to 1500 DM. Other scholarships are also available. Both these forms of financial aid are valid for a student studying abroad provided he mantains a fairly high academic standard.

The NETHERLANDS

Some students work to finance their study, but others have state grants some of which are repayable in part or in whole. A government grant automatically includes payment of fees. Students are unable to earn professionally.

SWEDEN

Almost all students have state grants, part of which is repayable. There are no fees, but students must buy their own books.

SWITZERLAND

Fees are 300 Francs per semester plus lab fees in some occassions. There is governmental aid for those of no means. At present the government is trying to spread its grants. Some grants are repayable either in whole or in part.

The U.K.

Fees are mainly paid by the government. All students qualify for a minimum grant of £50, but most students are given grants to cover tuition and living expenses, depending on parental means, to a maximum of £2000.

SOURCE: Proceedings of the first symposium on medical education Giessen, April '66.

ALEX E. FELICE,
Med. Educ. Officer.
now *President* M.M.S.A.

book reviews

IMMUNOLOGY FOR STUDENTS OF MEDICINE
J.H. Humphrey & R.G. White.
Third Edition 1970
Blackwell Scientific Publications
Price £3.00 net.

The authors of this book are in themselves evidence for the high standard of this work, rich in knowledge of the science of Immunology. Immunology is a growing science and these books run the risk of being slightly outdated, but a book of this calibre has the added advantage of providing a deep insight into the study of this stimulating science. It is sometimes overtechnical in its approach; the seven hundred or so pages are however constantly brimming with terse — but interesting facts, plus an abundence of plates showing photographs of electrophoretic patterns, fluorescent techniques, Electron Microscopy, and a few clincal photographs.

The masterpiece of the book is the chapter on Immunoglobulins which was fascinating in all of its fourty pages. Less satisfactory is that on autoimmunity (or as the authors prefer to call it auto-allergy). It unfortunately does not adhere to the Coombes classification which we are accustomed to and which would have resulted in greater clarity. Immunology is not all auto-immune disease (though the association is quite strong nowadays), and a third of the chapters are devoted to antigens, antibodies, and their reactions, discussed to a great length. Another good chapter is that on tissue transplantation. A glossary is conveniently placed at the end of the book.

In short, this book is recommended for the more technical minded who prefer the scientific aspects of medicine. Equally valuable will it be to those who believe that they must obtain their facts straight from the oracle and are prepared to digest the contents for themselves. With the growing emphasis on Immunology this book will certainly occupy a more prominent position in our Library in years to come.

joseph v. psaila.

LECTURE NOTES ON HAEMATOLOGY
N.C. Hughes-Jones
Blackwell Scientific Publications, 1970.
£1.50 144 pages.

A very comprehensive work and an excellent companion to 'Lecture Notes on Pathology', this pocket-sized book contains much more information than a quick glance through it or its mere size would suggest. Its contents are enough to cover the requirements of the Medical Course. It is therefore strongly recommended to any student who is still looking for the right book on Haematology or the one who gets lost on heaps of irrelevant data that much larger books contain.

The first five chapters deal with the various anaemias, comprising the aetiology, clinical presentation, diagnosis and treatment of each. The relevant physiological background is included in each case and this is one factor which helps to make the text clear. One should point out that the only omission is a good aetiological classification of the anaemias. Otherwise the subject matter is adequately covered in the five chapters except perhaps for the haemoglobinopathies.

Chapters Six, Seven and Eight cover Haemostasis, Blood Transfusion and Haemolytic Disease of the Newobrn respectively and hardly need to be supplemented in any way.

The final Chapter is on Haematological Techniques and quite worth while going through.

A useful help to anyone who wants to read further or go deeper, is an abundance of references at the end of each chapter. The inclusion of a number of plates is helpful in identifying the cell types referred to in the text.

The main points dealt with are summarised at the end of each chapter under the heading 'Objectives in Learning' — a very useful aid to memorise the facts.

On the whole this book is an excellent treatise on Haematology and is as lucid as the authors claim it to be.

anton vella.

some reflections on the rhesus problem

p. vassallo agius, m.d., d.c.h., m.r.c.p.
lecturer in medicine, royal university of malta

The problem presented by the Rhesus baby is not usually one of diagnosis — this should have been made by the obstetrician before the baby has been delivered. However, it is not uncommon for one to be faced with the problem of a baby who becomes jaundiced in the first 24-36 hrs. after delivery. In such cases, the baby is deemed to be suffering from haemolytic disease of the newborn until proved otherwise. Estimation of the Hb and serum bilirubin, the Coombs Test, blood group and Rhesus factor of the mother and the baby, examination of a blood film, together with direct cross-match of the baby's red cells will enable one to arrive at the correct diagnosis in the large majority of cases. Besides haemolysis due to Rh incompatibility one must bear in mind ABO incompatibility and other rarer group incompatibility, such as Kell, Duffy and a few others.

In the case of ABO incompatibility there are three combinations which might cause trouble to the foetus —

mother B — infant A
mother A — infant B
mother O — infant A, B, or AB

In actual fact, only the mother O — infant A or B situations, with rare exceptions, leads to haemolytic disease. The offending antigen is almost always A, and what is more, A of the specific subgroup A1. Even so, as in the case of Rh antigens, only a small proportion develop any significent jaundice presumably because the foetus may develop protective mechanisms, or the facility for the mother to develop antibodies is fortunately somehow impaired, or it could be that the liver of most infants is capable of excreting the small additional load of bilirubin so that the jaundice never becomes clinically apparent.

Occasionally, hereditary spherocytosis, hereditary elliptocytosis, hereditary non-spherocytic anaemias, (eg. pyruvate kinase deficiency), congenital toxoplasmosis, cytomegalic inclusion disease (C.I.D. virus) and G6 P.D. deficiency can present an identical picture.

One also sometimes sees a baby with anaemia **at birth** in which the question arises whether he is suffering from haemolytic disease of the newborn. Indeed, anaemia and oedema, singly or in combination, with little or no jaundice, may be the predominant signs of haemolytic disease of the newborn. The absence of haemolysis as a cause of the anaemia is not difficult to settle by a proper evaluation of the results of the investigations already outlined. In such cases, one must exclude extraneous bleeding from the cord or from vasa praevia, or concealed bleeding into the placenta itself or even bleeding into the mother (foetomaternal transfusion) — this latter can be excluded by the Kleihauer test (a test for the presence of foetal RBCS in the maternal circulation). I was recently faced with this problem in a baby who was born at St. Luke's Hospital and was under the care of Dr. E.A. Cachia. The mother was Rhesus negative and had no detectable Rh antibodies during pregnancy. I was called to see the baby who was obviously pale soon after delivery. The Hb was in the region of 10gm% and the blood film showed no evidence of haemolysis. The baby was Rh negative. The Coombs test was negative and the serum bilirubin was 1mg %. The Kleihauer test was strongly positive and the baby was treated by simple transfusion. In this case, the mother was Rh negative but this was a red herring and haemolysis was quickly and decisively excluded as a cause of the anaemia.

Sometimes one is presented with anaemia in the first week or two of life which is due to haemolytic disease, when jaundice, (though present initially) has not been severe enough to draw attention to the underlying disease. Indeed, the jaundice may have been so mild as to be labelled simple physiological jaundice. Again in such cases, a proper evaluation of the routine investigations outlined above should not present any difficulty in diagnosis. The treatment is by simple transfusion.

Bleeding from one twin into the other twin (twin to twin transfusion syndrome) can also present problems soon after delivery — with anaemia in the one and polycythaemia in the other twin. The anaemia can be quite severe and I have seen it cause intrauterine death. Otherwise it may need correction by simple transfusion, soon after delivery if necessary. The polycythaemia twin may become quickly severely jaundiced, with the danger of kernicterus, and the increased blood viscosity can embarrass the circulation and cause various other complications.

Again, the problem of Rhesus disease in the newborn does not really lie with treatment. All the efforts of treatment should be directed at correcting the anaemia and preventing kernicterus. The indirect-reacting serum bilirubin should not be allowed to rise above 20 mg%, especially if respiratory distress, cyanosis and prematurity are also present. There is still no way of doing this except by the tedious procedure of exchange transfusion. Recently, both phenobarbitone and exposure to light of so many candle-power (phototherapy) have been found statistically to lower significantly the serum bilirubin in premature and full term infants. Indeed, I have not seen any significant jaundice in our premature babies at St. Luke's

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Hospital, and, I believe that our well-lighted nursuries are important in this respect. However, these methods (phenobarbitone and phototherapy) have no place at all in the management of haemolytic disease of the newborn. The anchor sheet of treatment is the exchange transfusion, which should be repeated as often as necessary to keep the serum bilirubin below 20 mg%. The provision of disposable exchange transfusion sets has done a great deal to make it less tedious and a lot less messy. With these sets there is no need to wash those glass syringes which are continually stuck! The disposable syringe provided with the set is silicone-coated. I prefer to use fresh heparinized blood, which obviates several of the complications of citrated blood.

In my opinion, in the majority of cases, the real problem in Rhesus factor disease is one of communication and coordination. This is understandable considering the number of people who are involved — the obstetric team, the blood transfusion department, the haematology and biochemistry department and, of course, the paediatric team. The antenatal ward, the maternity ward and the paediatric ward are also involved at one time or another in the care of the Rhesus baby. Cooperation should not be difficult if there is adequate and timely communication and coordination. I have not mentioned the general practitioner — I do not think that the mother who has previously delivered a baby affected with haemolytic disease, however mild, or who has antibodies during pregnancy, should be delivered at home. The delivery should be made in a hospital equipped with the staff and facilities for performing an exchange transfusion, as an emergency if necessary.

I will illustrate what I mean by referring to three cases I have seen at St. Luke's Hospital in the last few months.

Case 1

This baby was delivered at home. He was the third baby. The first baby was normal. The second baby was slightly jaundiced in the first day or two of life. The third baby was referred to this hospital (to the O.P.D.) in the fourth day of life for jaundice. In fact, the serum bilirubin was 26 mg%, Hb 66% and Coombs test strongly positive. The baby was Rh positive and the mother Rhesus negative. The mother had never had the Rh factor determined and this was the first time that her blood grouping was performed, after the delivery of her third baby.

Case 2

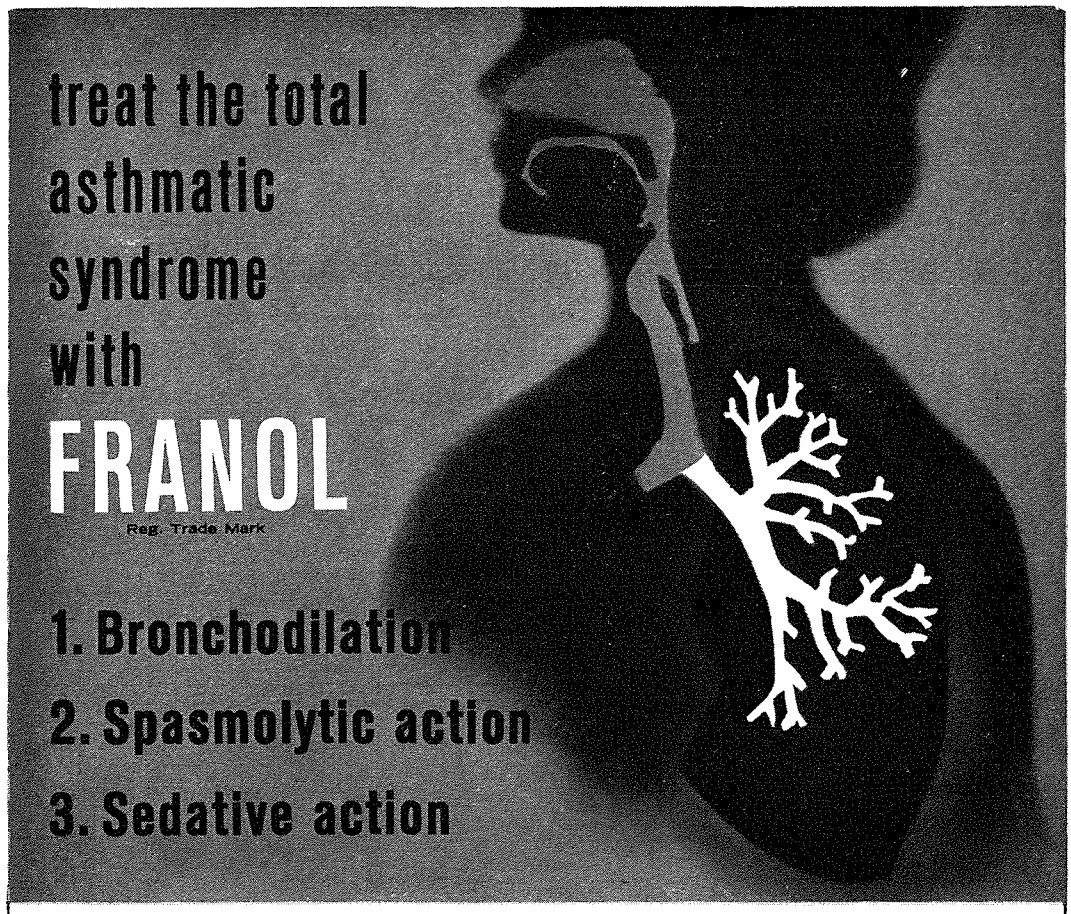
This also was the third baby, and was delivered at another hospital. The mother was Rh negative and had a rising titre of antibodies during her third pregnancy. He was born in the middle of the night three weeks prematurely, and soon after delivery was transferred to St. Luke's Hospital. On examination he was moribund, with marked pallor, oedema, some jaundice and in heart failure. Efforts at resuscitation proved fruitless and he died 4 hrs later. The serum bilirubin was 7.3 mg%, Hb not determined (clotted blood), Baby's group O Rhesus positive.

Such cases of hydrops are of course notoriously difficult to treat. On two or three occasions I have given such babies a mini-exchange transfusion with packed cells, exchanging 100 or 200 cc blood, together with digitalization and diuretics, and then performed a full exchange transfusion for the hyperbilirubin-aemia later on, at 24-48 hours of age. Initially, it is not so much the jaundice as the anaemia which is causing trouble.

Case 3

This was the eighth baby. There were at least 3 previously affected babies necessitating exchange transfusion, all of whom, in fact, died. This baby was also affected, though not severly so, but it took about 12 hours before the exchange transfusion could be started.

In each of these cases there has been a lapse in communication. The difficulties are obvious but I do not think that they are insurmountable. The blood transfusion department and the haematology and biochemistry departments, and paediatrician should be alerted as soon as the mother is admitted to hospital, or at least as soon as labour has started. The inexorable march of labour cannot be halted, but we can do a good deal to be prepared to treat the product of a Rhesus gestation.



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respiratory difficulties in the newborn period

a.j. psaila, m.d., d.c.h., m.r.c.p.
registrar in medicine, st. luke's hospital

One of the major problems in paediatric practice is respiratory distress of the newborn. This problem is frequently met with; the causes are various and the mortality rate is considerably high.

The neonatal period covers the first four weeks of life. Depending upon its basic cause, respiratory distress may make its appearance immediately after birth or at any time thereafter. The severity varies from simple rapidity of respiratory rate (tachypnoea), to the most extreme air hunger. The main physical signs which are found in most moderate to severe cases are: increased respiratory rate, cyanosis, grunting respirations, intercostal and sternal retraction and seesawing of the abdominal wall. Silverman et al. evaluate severity of respiratory distress by assigning a score of O, 1, or 2, to 5 of these physical signs, the sum constituting the infant's score. Silverman score for assessing respiratory distress is used in most units in the United Kingdom and United States of America.

SILVERMAN SCORING

	Severe	Moderate	Nil
Grunting Respiration	2	1	0
Intercostal Recession	2	1	0
Sternal Recession	2	1	0
Seesawing of Chest Wall	2	1	0
Chin lag	2	1	0
1 — 3 mild respiratory distress			
4 — 6 moderate ,, ,,			
7 — 10 severe ,, ,,			

This scoring system has its faults in that it does not include two of the most important physical signs namely increased respiratory rate and cyanosis, but by and large holds good for quick assessment and for comparing series from various hospitals in different countries.

I would like now to discuss briefly some of the physical signs mentioned above.

Cyanosis is a blue colour of the skin or mucous membranes usually due to the presence of an excessive amount of reduced haemoglobin, and this in turn is due to insufficient oxygenation of the infant's blood. It is not present in mild cases of respiratory distress, as for cyanosis to appear, there must be about 5g. of reduced haemoglobin per 100 c.c. Lesser grades of cyanosis may clear completely in low concentration of O_2 but more severe ones may not disappear even when 100% oxygen is administered.

As intensity of respiratory difficulty increases, more and more of the chest wall retracts with each inspiration. At first the intercostal spaces alone are involved, then the suprasternal notch and eventually the sternum. In the most severe cases the whole chest wall retracts during inspiration whilst the abdomen is blown out *(seesawing* of the abdomen). In this phase all the accessory muscles of respiration are brought into play.

Expiration may be short and accompanied *by a grunt,* or may be prolonged and difficult, and may be accompanied by movement of the chin downwards *(chin lag).*

IDIOPATHIC RESPIRATORY DISTRESS SYNDROME OF NEWBORN (Hyaline Membrane Disease)

This syndrome is by far the commonest cause of death among premature infants, but is also seen with significant frequency in infants of diabetic mothers, in babies born by C.S. and occasionally among infants in whom no predisposing factor is present.

The syndrome consists of increasing respiratory distress at or very often a few hours after birth. Rapid respiratory rate is quickly followed by retraction, expiratory grunt and cyanosis. The disease progresses quickly in some infants, so that they become seriously ill with dyspnoea, deep cyanosis and profound retraction within 3 to 8 hours, but in most, progression to the severe phase takes 12 to 24 hours. Thereafter there is either improvement or deterioration with apnoeic and cyanotic attacks. The mortality rate is as high as 50% in most series and the smaller the infant at birth the higher the mortality rate.

CAUSES OF RESPIRATORY DISTRESS OF THE NEWBORN

The commonest causes are:

(1) Aspiration of amniotic, vaginal or oropharyngeal contents.
(2) Idiopathic Respiratory distress syndrome of the newborn (Hyaline membrane disease).
(3) Atelectasis.
(4) Cardiac failure.
(5) Cerebral Birth Injury.
(6) Pneumonia of the newborn.
(7) Congenital abnormalities e.g.
    Oesophageal atresia.
    Diaphragmatic hernia.
    Posterior choanal atresia.

Rarer causes include:

(1) Congenital cysts or tumours obstructing or pressing on pharynx, larynx, and lungs.
(2) Congenital laryngeal or tracheal stenosis.
(3) Emphysema.
(4) Pulmonary hypoplasia.
(5) Accesory and sequestrated lobes.
(6) Chylothorax and pleural effusion.
(7) Pulmonary haemorrhage.

By far the commonest causes of respiratory distress are idiopathic R.D.S. and massive aspiration.

The cause of this syndrome is not known. At post-mortem pulmonary hyaline membranes with atelectasis are the principal findings, (hence the other name of this syndrome i.e. hyaline membrane disease). But it is not known whether this membrane is the cause of the symptoms. It is not always present in premature babies dying from this syndrome, and it is formed in the course of many disorders.

There are many theories on the cause of the idiopatic R.D.S. of the newborn including aspiration of amniotic fluid and alveolar effusion from the pulmonary circulation (left sided heart failure) with resulting transformation of the fibrinogin of the effusion into fibrin. This in turn covers the alveolar membrane and prevents exchange of gases.

The most recent theory is that the I.R.D.S. may result from a deficiency of a surface tension reducing film of lipoprotein (surfactin) normally present in the alveoli. This deficiency is presumably produced by an inhibiting substance derived from damaged pulmonary tissue or inhaled with amniotic fluids. This substance is believed to interfere with fibrinolysis resulting in the formation of the fibrin — containing hyaline membranes.

Treatment is strictly supportive and one must emphasise the importance of prevention of this syndrome by the prevention of prematurity, avoidance of unnecessary cesarean section and careful management of the diabetic mother.

Treatment include management in an incubator, gentle handling, oxygen, humidity, antibiotics (owing to frequency of complicating pneumonia) and intravenous glucose and bicarbonate to counteract resulting acidosis. Some of these infants develop a hyperkalaemia which in turn can cause hyperkalaemic E.C.G. conduction defects. The proper management of these infants must include repeated blood pH, serum K and serum bicarbonate levels and glucose + insulin is given to correct hyperkalaemia.

THE ASPIRATION SYNDROME

This is due to aspiration into lungs at birth of liquor amnii, meconium, blood or vaginal secretion. Aspiration of small quantities of liquor amnii is commonly met with, giving rise to very little discomfort. On the other hand massive aspiration fill the bronchi and bronchioles with resultant respiratory obstruction and severe distress. Conditions producing foetal asphyxia cause the foetus to breathe or gasp in the uterus and birth canal, these movements being caused by foetal anoxia or hypoxia in utero.

The most frequent cause of foetal aspiration is post-maturity (Peterson and Pendleton), other causes being placenta praevia, maternal haemorrhage, cord prolapse and other causes of compromised placental or foetal circulation. Another, often overlooked cause is administration of heavy sedatives to the mother shortly before delivery. These drugs may induce loss of the mechanisms calculated to prevent aspiration.

The aspirated fluid, by obstructing large airways, may give rise to segmental atelectasis (failure of segments to expand) and in turn emphysema. If the fluid is infected as in cases of prolonged rupture of membranes, it may lead to intrauterine pneumonia.

Respiratory difficulty of various severity occurs at or soon after birth. If there is massive aspiration the baby is shocked and apnoeic. These babies might have suffered intrauterine cerebal anoxia and may die soon after birth. The survivors develop quite marked respiratory distress when they recover from the initial state of shock.

The infants who have not suffered cerebal anoxia carry a good prognosis as the respiratory distress, after lasting a few hours to 2 or 3 days, is followed by rapid recovery unless complications such as gross atelectasis with emphysema, pneumonia or pneunothorax occurs.

Treatment consists of oropharyngeal and laryngeal toilet via an endotracheal tube, oxygen and antibiotics. the onset of feeding is delayed.

ATELECTASIS

Atelectasis means incomplete expansion of a lung or a portion of a lung. Atelectasis may be primary due to pulmonary immaturity (prematurity) or inadequacy of respiratory effort as occurs in oversedation or cerebral birth injury. In most cases it is secondary to other disease process as in massive inhalation and idiopathic respiratory distress syndrome of the newborn. It can also be caused by abnormal external pressure upon the lung as in congenital diaphragmatic hernia.

CARDIAC FAILURE

Congenital heart disease may present in the neonatal period as cardiac failure. There is dyspnoea, tachypnoea, tachycardia and cyanosis. Intercostal recession may also be present but it is rarely as great as in pulmonary disease. Enlargement of the liver may be present. Dyspnoea may occur in paroxysms in cyanotic infants who are not in heart failure but who suffer from anoxic spells. Among congenital heart defects giving rise to neonatal respiratory distress are transpositions of the great vessels, total anomalous pulmonary venous drainage, coarctation of the aorta, patent ductus arteriosus aortic atresia. Some of these defects are amenable to early surgery.

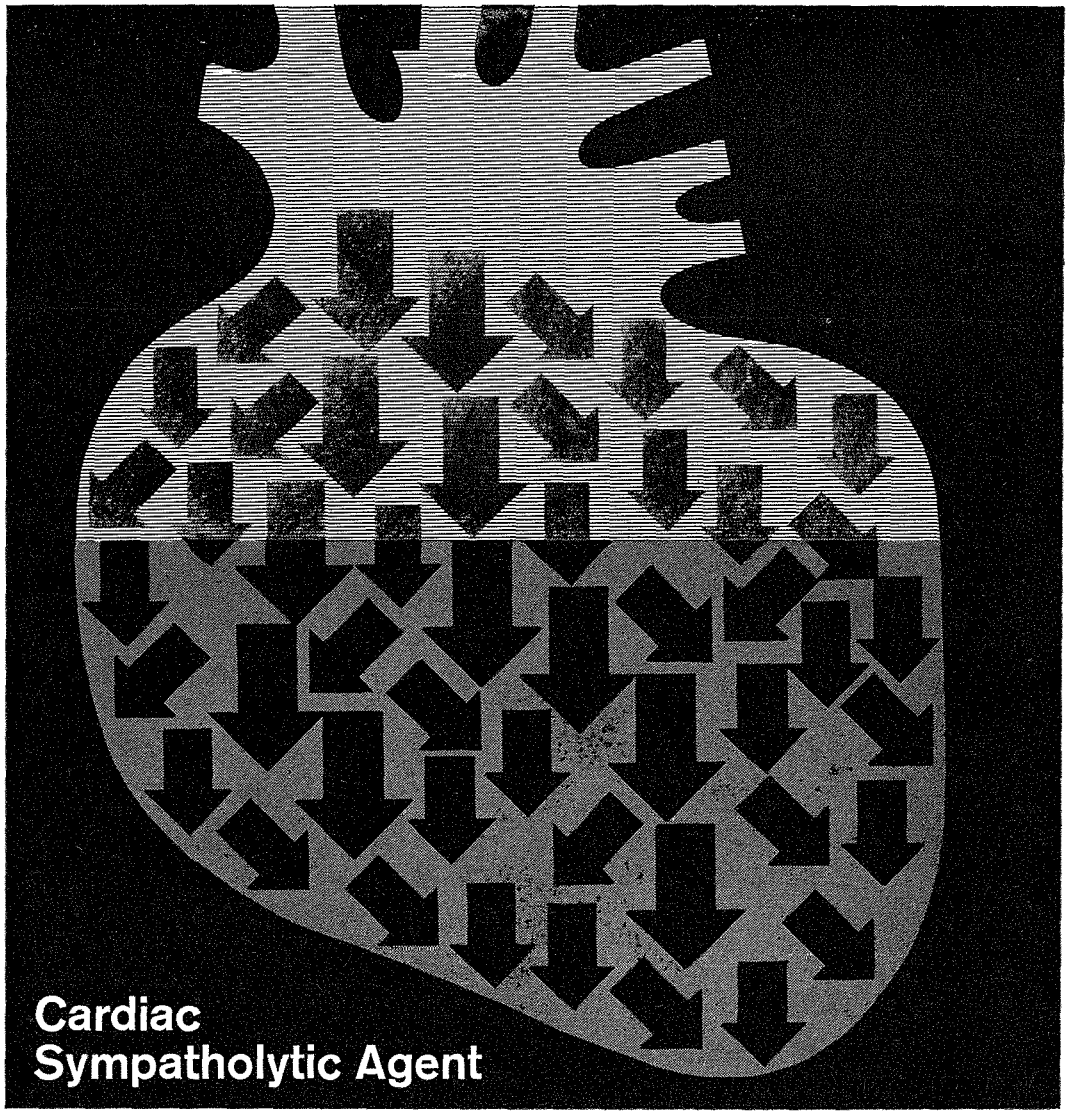
CEREBRAL BIRTH INJURY

This has already been mentioned as a cause of massive aspiration and atelectasis. Intracranial haemorrhage whether due to trauma or to anoxia is very often present. There is bleeding in the area of the respiratory centre which in turn leads to failure of respiratory centre resulting in weakness of respiratory effort and apnoeic attacks.

PNEUMONIA OF THE NEWBORN

Pneumonia is one of the important causes of perinatal death. It may be acquired in utero, during labour and after birth.

Pneumonia can be acquired in utero either transplacentally secondary to maternal septicaemia, or an ascending infection in cases of prolonged rupture of the membranes.



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It can be acquired during labour if baby inhales maternal faecal matter or infected liquor.

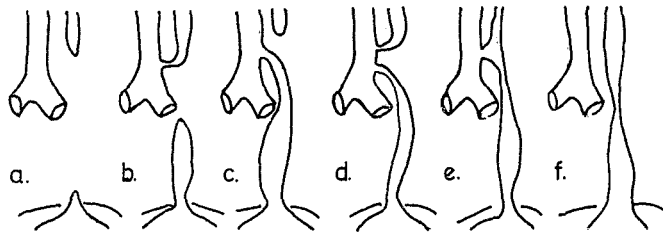
Post-natally it can be due to aspiration of food or gastric juice, it can be due to airborne bacterial, viral and fungal infections, and it can be secondary to a septicaemia. Respiratory distress may be present at birth in cases of intra-uterine pneumonia but usually develops later. Oxygen and antibiotics are the main lines of treatment.

CONGENITAL ABNORMALITIES

In addition to the illnesses described above, there are a number of important surgically remediable congenital abnormalities which can give rise to severe respiratory distress. They are less common than the illnesses already described but are by no means rare. The following conditions should be borne in mind in any infant with respiratory illness. They are the commonest of this group of illness.

(1) Oesophageal Atresia with Tracheo-oesophageal fistula.
(2) Congenital Diaphragmatic Hernia.
(3) Congenital Lobar Emphysema.
(4) Choanal Atresia.

A chest X-Ray is an important help to the diagnosis in all cases of respiratory distress of the newborn but more so if one of the above conditions is suspected.



Six types of congenital anomalies of the oesophagus as described by R.E. Gross (Surgery of Infancy and childhood).
a - d=oesophageal ateresia, b - e=tracheo-oesophageal fistula, f=oesphageal stenosis.

(a) *Oesophageal atresia with Tracheo-oesophageal fistula.*

Recurrent collection of mucus in baby's throat should give rise to immediate suspicion of atresia and diagnosis is confirmed clinically if a small gastric catheter cannot be passed via the oesophagus into the stomach.

Once diagnosis is made early surgery by closing the tracheo-oesophageal fistula and gastrostomy is essential as a life-saving procedure. Reconstructive surgery on the oesophagus is performed at a later date.

(b) *Congential Diaphragmatic Hernia,*

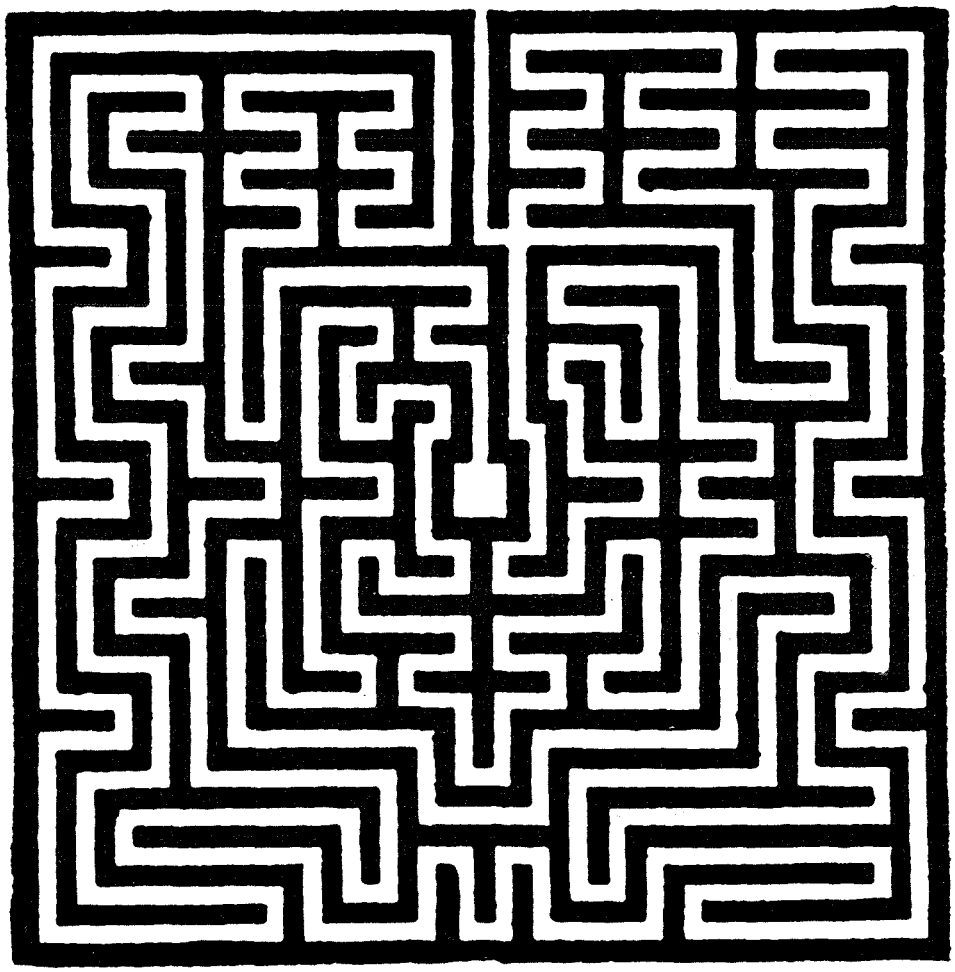
This is another condition in which surgery is essential as a life-saving procedure as soon as diagnosis is made. The small bowels and sometimes parts of the large bowel and spleen are within the chest cavity, the lung on the affected side being completely collapsed. The mediastinum is displaced to the opposite side so the lung on the opposite side may also not be fully expanded due to this pressure. Because of this there is severe dyspnoea and cyanosis which are exacerbated by attempted feeding. A clue to the diagnosis may be a bulging hemithorax (due to the increased contents) and a fairly flat abdomen. Chest X-Ray is diagnostic and only surgery can save an infant's life.

(c) *Congenital Lobar Emphysema* occurs as the result of a congenital deficiency of the bronchial cartilage. The affected lobe expands enormously and compresses the remaining lobe on the same side, and also due to shifting the mediastinum compresses the lung on the other side. Chest X-Ray shows marked translucency of the affected side. Lobectomy will be necessary.

(d) *Posterior choanal atresia.*

In this condition the openings of the nose into the nasopharynx are closed by a septum containing bone. A bilateral case will present with cyanosis and violent inspiratory effort. If the mouth is opened and tongue drawn forward air can be inhaled easily. A newborn baby is unable to mouth breath and some form of tube over the back of the tongue may be used to allow mouth breathing until the choanae can be opened by operation.

For further information about the conditions mentioned in this article the reader is instructed to look up standard works of paediatrics such as *Nelson;* 'Text Book of Paediatrics' and *Schaffes,* 'Disease of the Newborn'.



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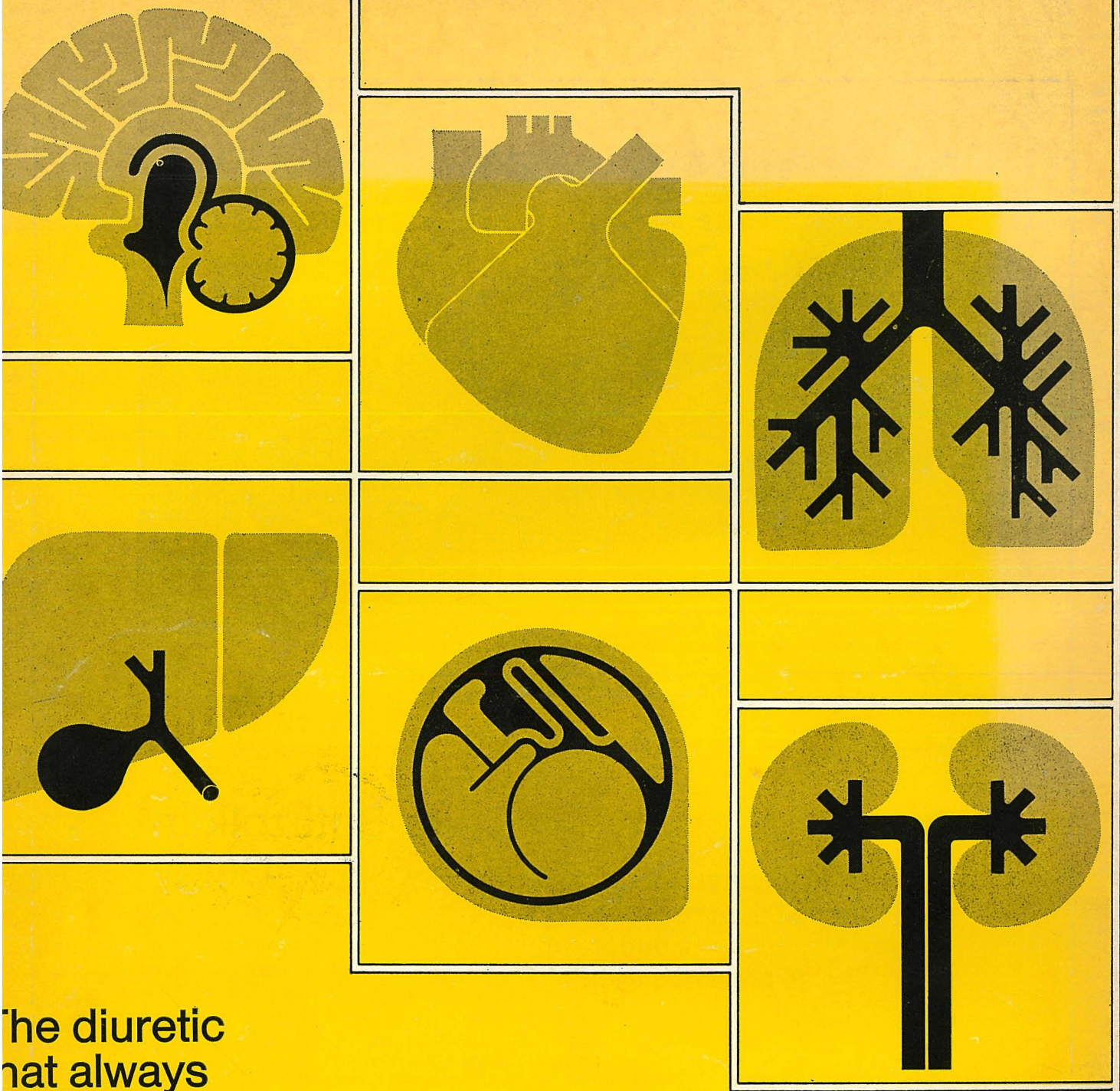

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