After lunch Berstock and Firmin scored well till Firmin was unnecessarily run out for 42. Leach and Lloyd's wickets fell cheaply soon afterwards and Bart's were 167 for 8. Sloane joined Berstock and they passed the 200 mark. Edmondson's contribution of 8 enabled the score of 219 to be reached, before Sloane was caught off Goodhart for 29.

Guy's looked set and determined to make the necessary runs, but were shocked after 20 minutes to have Gillham removed by Edmondson. An early change of bowling, and Lloyd replaced Berstock. Bowling well the Guy's batsmen still tried to him around the ground, but Sloane's stumping gave Bart's the useful wicket of Goodhart. Rowland now came on, and bowled well, and little more resistance was offered by the opposition batsmen. Rowland bowled fifteen overs and took four wickets for four runs; he had the batsmen in serious trouble with many unplayable balls.

Score card :-

Bart's—

Purcell, ct. b. Crafts	0
Lindsell, ct. b. Crafts ...	26
Furness, ct. b. Clark	42
Reid, b. Pearson	18
Firmin, run out	46
Rowland, ct. b. Crafts ...	4
Berstock, ct. b. Goodhart ...	30
Leach, b. Pearson	0
Lloyd, ct. b. Goodhart ...	7
Sloane, ct. b. Goodhart ...	29
Edmondson, not out	8
Extras ...	9
Total ...	219

Guy's—125 all out

Edmondson ...	20	11	42	1
Berstock ...	15	6	24	2
Lloyd	21	9	41	3
Rowland	16	13	4	4

Sun., 8th June v. Blackheath. Won by 155 runs.

On the Rectory county ground at Blackheath, Bart's played very well indeed against one of the strongest sides on the fixture list. Batting first, Bart's scored 230, Furness scoring a fine 57, and Husband a very aggressive 57. This included two sixes, one of which was lost beyond the car park, and six fours.

Blackheath were soon in trouble with Edmondson's pace, and at tea were 30 for 3. They only reached 75, mainly due to tight bowling and good fielding.

Sat., 14th June v. Avery Hill. Lost by 105.

A poor performance by Bart's batting, which only produced 82 in reply to Avery Hill's 187.

Junior Cup match v. Guy's. Won by 30 runs.

Bart's batted first and reached 103, Mees scoring 32 and Husband 20. Davis and Husband bowled well, taking four and five wickets respectively. Guy's were all out for 73.

Sun., 15th June v. Horlicks. Won by 7 wickets.

Horlicks were bowled out by Vartan (6 for 8) and Husband (4 for 15) for only 36 runs. Bart's reached this score for the loss of only three wickets, before lunch.

Wed., 20th June v. U.C.H. Lost by 34 runs.

U.C.H. scored 177 for 7 declared, and Bart's were bowled out for 143, Reid scoring 43. Having beaten them easily on in the season, this result was rather unexpected.

Sun., 1st June v. Putney Eccentrics. Matched tied.

This is the first recorded tie in the history of Bart's cricket. Batting first, Bart's scratched around for 120 runs on the best wicket to that date at Chislehurst during the season.

Berstock and Husband bowled very well and the last man was well caught by the captain off Husband at silly mid off.

Berstock 6 for 48; Husband 4 for 9.

Cambridge May week tour

Although only two of the three matches were played and the weather made the other two matches rather unpleasant, the other aspects of the tour were suitably enjoyed by all—except one or two Cambridge inhabitants.

The match against Trinity was rained off; the match against Christ's was lost by 6 wickets.

Tour party: D. Berstock, J. Shepherd, D. Edmondson, E. Lloyd, D. Lindsell, R. Firmin, D. Sloane, K. Jones, P. Rhys Evans, J. Gower and R. Page.

Sun., 22nd June v. U.C.S. Old Boys. Lost by 1 wicket.

Batting first, Bart's scored 221 for 6 declared. Furness making 75, and Lindsell 77: their partnership of 107 was very spectacular.

However, U.C.S. scored very fast and on the last ball of the match just scraped home.

Weds., 25th June v. City of London Police. Won by 81 runs.

Bart's batted first and against the tightest bowling faced all season scored 120 very slow runs. Edmondson at last found his true pace, and bowled well, taking 5 wickets for 18 runs. Smith and Rhys Evans took the other wickets and the Police only managed to score 39.

Sun., 29th June v. Orpington Hospital. Won by 85 runs.

On a very poor pitch, Bart's scored 141 runs, to which Orpington replied with 56 only.

P. H. Rhys Evans

Saint Bartholomew's Hospital

JOURNAL

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Lifemen all ?

by Geoff Hyde

Recent developments in oneupmanship, with apologies and thanks to Stephen Potter whose idea it is.

It is over 20 years since Stephen Potter first coined the expressions Oneupmanship, Lifeman and Gamesmanship. His original and brilliant analysis gave an air of respectability to what some might have called snobbery, pulling a fast one or in some cases even cheating.

Much has happened since then, and an attempt it made here to look again at the whole subject in a more recent context. Lifemanship has become something of a game played by almost all with varying degrees of intensity and skill. In fact so wide-widespread is it that a refusal to participate in the first place (The Riggers Approach) may turn out to be one of the most successful gambits in the long run. In the everyday world around us the simple ploys and gambits of junior and middle grade lifemen are easily countered by their more experienced colleagues. Thus it is to the middle and lower grades[1] that this short review of modern medical[2] oneupmanship is directed. May it speed them over the difficult first steps and bring them to the threshold of the intricate world of upper lifemanship in which those gauche early days are only remembered with a shudder of embarrassment or shouts of seemingly genuine laughter.

brainmanship

This aspect of life is perhaps one of the most serious for the embryo Harleyman/Hammersmithman, and for the simple student there is little freedom of oneup action if he is to achieve the desirable goal of qualification[3].

The enviable ploy of "jolly fellow, one of the lads, never does a stroke but still wins all the prizes", is beyond the capabilities of the majority[4], and those who can genuinely do it have little need for lifemanship anyway. Most attempts at appearing never to work[5] but still doing well are easily seen through in a close-knit academic community.

An alternative ploy is to do very little work and only just scrape through. The casually dropped remark such as, "I could have done better of course, but the rugger/women/vicarage club/Drama Society/drinking take(s) up so much time" is generally considered rather weak. The main disadvantage to this ploy is that it is all too frequently used.

Another ploy to be avoided at all costs is to be seen working all day and every day but still coming near the bottom. To show excessive enthusiasm and effort without any result is poor lifemanship. If one has to work hard to achieve even a moderate pass, as many do, it is best not to draw too much attention to it.

Once the desired qualifications have been gained the field is wide open for the good lifeman. Such gambits as having hundreds of adoring patients, and the doubtful one of publishing as many papers as possible are then mere starting points. One of the most advanced Senior-Doctor Gambits (more accurately a counter-gambit) that has come to my attention is, "Oh I don't like to put my own name to the research we do here. I let the lads in the unit/department do that. After all they do all the nuts and bolts of it."

carmanship

This is one of the more straightforward fields in which the lifeman can excel provided some obvious mistakes are avoided.

Just having a car is a reasonable opening move, although this is easily countered by a remark such as. "Used to have one of those wretched things once, but what with present-day traffic and parking . . .". The sentence can be left unfinished, but " . . . gave it away to my young sister", gives it a little more finesse. This no-car ploy is particularly effective if it can be combined with ostentatiously arriving and leaving College Hall in taxis (only one of many brilliant life ideas filched from G. Chapman). This need not be unduly expensive. To the nearest Underground is about as far as is necessary to go.

A quality vintage sports car is often thought of as a successful gambit (mass produced ones like M.Gs. are becoming passé), although I once heard the following masterly countergambit from a senior lifeman after he had listened for several minutes with a tolerant half-smile on his face (which another good lifeman should at once have recognised as a warning signal) while junior lifeman extolled the virtues of his gleaming Red label Bentley. "Hmm not a bad one. Got an early Hispano S myself—ten litres you know—criminal[6] to use it on the roads though, quite criminal." However this sort of broadside may need back-up evidence to prevent it being classified with the rather underhand life move told to me recently by P. Fairclough (he says he wouldn't risk trying it out himself) of pinning up a scruffy but eyecatching[7] notice, advertising £500 worth of stereo equipment for sale. If a prospective buyer should dare to come and view it is easy to say its sold.

Expensive modern sports cars received as a present from rich father/uncle/grandfather/Mummy is gambit which is difficult to play well as it may arouse genuine envy,—a thing the good lifeman should never do. Better to suggest it is a permanent loan from financially successful brother, or what is probably the best car gambit of all is to let it discreetly be known that it was a gift from a grateful girl-friend. (The author is still awaiting the opportunity to test this one out).

A motor-cycle is a difficult gambit to carry off unless it is outrageously vulgar and flamboyant[8]. Lifemanlike comments such as, "I only have it because it is so quick and easy to park" sound a little hollow unless one can boast a car as well. (It may not be necessary to go to the expense of running it). With scooters and mopeds it is best to dress up in face-hiding P.V.C. gear before going anywhere near the things.

For good, yet practical, carmanship in which lack of money is not a thing to be lightly dismissed, a very large old car of quality is always a safe move (Mk. IX Jaguars, 8-seater Daimlers and so on. R.R. hearses are better but very rare). A neat lifemanlike touch can be added by comments such as, "Of course the big advantage is that the whole team/crew/wine committee can travel down to Chislehurst/Chiswick/Ascot together." This suggests that the owner only bought it[9] out of selfless loyalty to his fellows.

gamesmanship

The lifeman's approach to sport was reviewed by Potter in 1947. His treatise, "The Theory and practice of gamesmanship, or the art of winning games without actually cheating," covered the prewar sports scene. Croquet, billiards, tennis, field-shooting and golf, are dealt with in depth and with a deep understanding his subject. However, team sports such as rugger, hockey and rowing were omitted. probably because a lifeman has less chance to exercise his talents as an individual.

Research into the subtleties of teammanship is continuing and extensive statistical analysis will be needed before meaningful results are obtained. The mind boggles at the possible number of combinations of group oneup action by say thirty dedicated lifemen all pitting their wits to achieve the same objective[10].

Gamesmanship is essentially a man to man confrontation, and the most challenging terrain is sports such as judo, chess and squash, in which a competitor must rely on his own efforts (aided if possible by special equipment and accessory games' gimmicks[11].

Since the war several new sports have grown in popularity and which are of special interest to the lifeman. Predominant among these are Skiing, Judo[12], and small boat sailing. Of these skimanship has so many ramifications besides basic mountain descent that it is worthy of more detailed study.

skimanship

Skiing is no longer a sport for the very rich alone. A fortnight in the Alps costs little more than a fortnight on the Costa Brava. Participants vary from a few hoary pre-war veterans[13] to plump under-the-age of consent typists, but the majority are youngish middling class first and second-timers[14]. The Alpine native is in a class apart and the British gamesman is wise to dismiss them with, "of course they have been doing it since the age of two", and concentrate his life ploys on his fellow-countrymen.

Let us imagine the problems facing a promising young lifeman prior to his first skiing trip. Much thought will be needed before the day of departure arrives.

Firstly, who to go with. Skiing is no team sport and once on the slopes simple gamesmanship degenerates into a cut-throat battle of life-play between the individual and others of roughly the same competence. Lack of skill is no drawback, it only makes the conflict more intense. The socially insecure may find it easier to join a large organised party[15], but let us assume our lifeman chooses to go with a small group of friends. They need to be of strong constitution, markedly individualistic of personality, amusing, garrulous, bi-or-multi lingual and hollow-legged. So indeed does our lifeman, or how else can he stand up to the indignities of a condition resembling severe tabetic ataxia when on skis, a permanent covering of snow, multiple superficial bruising, excess liquor, chronic lack of sleep, and still appear happy and smiling.

The next consideration is what to take. If the journey is to be by air (very cheap and efficient these days) the usual lifeman's ploy of having a monstrous sea-chest with everything one could possibly need, is obviously impractical. A new gambit at present being worked on by the author, is to take nothing[16]. The secret is to either leave all the essential gear like boots, skis and sweaters at the resort the year before, or be prepared to hire it all on arrival. A smart worsted suit and brief-case is all that is needed for the journey. This really puts the hackles up on perspiring fellow-travellers who have put on as much of their heavy clothing as they can to keep their baggage within the weight limit. Anyway the more experienced skiier soon learns that one good nylon anorak (which need not be heavy) is more use than half a dozen Shetland sweaters.

How should the lifeman behave when the Alpine paradise is reached? An important problem always facing him when presented with a novel situation, is what attitude to adopt towards it. Fear, anxiety or bewilderment should never be allowed to break through his aura of quiet confidence. Overconfidence, however, is risky. This is particularly so for the novice skiman, for the good gamesman with strong limbs and an eye for the ball, has little or no inherent advantage over the puny eight-stone weakling when it comes to trying to stay upright on skis.

When on the slopes, falling over is the first skill to be mastered. There are no points to be gained by not falling at all unless one's skill is such that it is almost impossible, for at this level (as in motor-racing) reputations are more likely to be enhanced by the occasional spectacular pile-up. An impressive always-falling-over ploy (recently named "dramatic a.o.t. manship') was accomplished with unintentional brilliance by lifeman Boatman, whose desire to descend every slope the shortest way possible (also an inability to do anything else) invariably led to magnificent disaster. Timid maidens would become weak at the knees at such displays of daring and manly courage.

The more level-headed novice, who may not feel himself to be of such cannon-fodder quality, would do well to take note of a sophisticated counter-ploy used extensively by Gilchrist and Gower during the '68/69 season, which even had L/man Boatman fooled for some considerable time. This was to dismiss the Nursery Slopes with a gay laugh and ascend into the clouds on a cable car. A lengthy Schnapps lunch, followed by two or three hours of sun-bathing then takes place at one of the many delightful high Alpine huts[17] before descending in the cable car. It is only necessary to look exhausted and rug snow into one's ski bindings to complete the deception.

This latter example touches on the essential feature of good skimanship, which is to convert what might have been an uneventful two weeks in wintering London, into an exquisitely pleasurable romp in the Alps. And let us never forget that the pursuit of pleasure is one of the fundamental principles of lifemanship.

summary

Groundwork is being done on some other important aspects of lifemanship. Woomanship, or How to get your girl without her getting you, and Clubman, or How to be invited to membership of the Clubs of your choice without actually asking. Pubman, or How to be seen in the best bars at the right times, is also under study, but as avoidance of buying your round when the time comes is poor lifemanship, more financial backing is needed before this particular line of research can yield results[18].

The good work into the whole vast field of oneupmanship and its derivatives continues apace and I would be most grateful for contributions preferably backed up by experimental results, from fellow workers among our readers.

footnotes

1. Experienced lifemen will of course immediately spot a ploy for establishing themselves as high grade "of course I didn't actually bother to read past the introduction," or better still; "Oh yes, that!—I had to check through the proofs before it went to press."
2. Essentially intramedical. The vast field of Doctor/Patient relationships is not touched upon here.
3. Many subtle examroom gambits, examcraft (or craftiness), have been described, but caution is needed as examiners are invariably high grade lifemen themselves and clumsy attempts at oneupmanship by the student are easily exposed. The "diligent and respectful pupil" gambit is usually found safest and quite effective.
4. An intriguing substitute for ability was suggested by one R. Carruthers-Singh. This was to take time off to study in Calcutta before coming to Barts, but the consequences of being caught out with this one would be so disastrous that it is an idea not worth taking very seriously, and of course a slip-proof cover story to explain away the lost years would be difficult to keep up. Such things as "oh I used to be a Professional wrestler/Fighter Pilot/Reporter in Vietnam," are obviously suspect.
5. The elementary gambit of pretending to go for a fortnight's skiing just before exams while really sitting under a sunlamp with the books in an Earls Court bedsitter is to be particularly avoided, not only because of possible exposure as you surreptitiously slip out for a pint in the Kings Road, but because successful Skimanship requires admiring and garrulous friends to have been with you on the slopes so that one's prowess becomes widely known without actually having to talk about it yourself.
6. This is not of course implying that the car could not pass the M.o.T. test.
7. "Dayglo" flesh-coloured paper.
8. Senior L/Man N. Wagner used to run a large bechromed Harley D. with enormous success.
9. Otherwise the G. Chapman taxi technique would be followed.
10. 1,307,674,368,000 for each team. To be more precise 653,837,184,000 if the efforts of the full-back are discounted.
11. This latter is a rapidly developing field and two sub-divisions have emerged. Firstly, such devices as the talking croquet ball, which is designed to psychologically overawe the opponent. It can be made to scream as the opposition is about to despatch it to the flower-beds. Secondly, there are those gimmicks which are a technical advantage to the player, such as the Dunckley greasy judo suit.
12. The author is at present studying classical Japanese in order to learn gamesmanship tactics from the original masters. It is to be hoped that a preliminary report will be published in the autumn of 1985.
13. Telemark.
14. Statistics show that the intervention of matrimony is responsible for the significant reduction in the number of third-timers.
15. Most travel agencies find it more profitable to handle their customers in these neatly regimented groups.
16. The inspiration came from the example of lifeman Jim Gilchrist, the effect was marred however by his anxiety about the luggage which was entrusted to the care of the
17. The author's personally recommended list can be sent in plain brown envelopes for a small fee.
18. Cheques payable to Life Research Fund and crossed "G. Hyde A/c".

IF MUSIC.....

The true art of making curry has been lost to man for centuries. Here Jake Mackinnon reveals all.

by Jake Mackinnon

The gastric approach to the male heart is one that has been used by ladies for centuries. Why is it traditional for men to be wooed in this manner? Would not a woman be equally susceptible to a marvellous meal cooked by the man of her choice, and eaten by candlelight in his romantic attic? Gentlemen —the time has come; forget the cinema screen and stage set; it must be the oven and the table. Imagine yourself, the dashing immaculate cook, plucking the souffles from the oven at the exquisite moment. How can she resist this wizardry?

Now, let us not be too ambitious! Simple things are forever the most effective. Curries for instance are easy to cook, cheap, and as long as you do not make them too hot everyone enjoys them sooner or later. One particularly good one to try is a keema bhoona—dry mince-meat curry. This is flavoured with cinnamon, lemon, and garlic, as well as curry powder, and need not be particularly hot.

For two people, the ingredients needed are:

2 onions
1 lemon
2 teaspoons cinnamon
2 tablespoons curry powder
2 cloves garlic
1 small tin tomato puree
2 cloves
2 cardomom seeds
¾ lb mince

First slice the onions and fry them in butter until they are soft (not browned). Then add the mince—this should be finely minced as it gives the curry a much less lumpy consistency. It should be stirred and broken up until it is partly cooked otherwise it ends up like a hamburger and spoils the evening. Now add the rest of the ingredients. The garlic should be peeled and crushed; the lemon rind should be pared off and cut into thin shreds, and the juice squeezed out and poured into the curry. Add all the spices—2 teaspoons of curry powder is enough to make it pleasantly hot—if you don't want her to come back you can add some more.

When all the ingredients are in, cook slowly, but make sure the curry does not dry up. You can keep the lid of the saucepan on, but if it looks a bit dry add a small amount of water to maintain the consistency of runny cement.

You can prepare all this beforehand, in fact doing it the night before and allowing the curry to stand overnight most certainly improves the flavour. The final result is a dryish curry which is pungent, fragrant and hot.

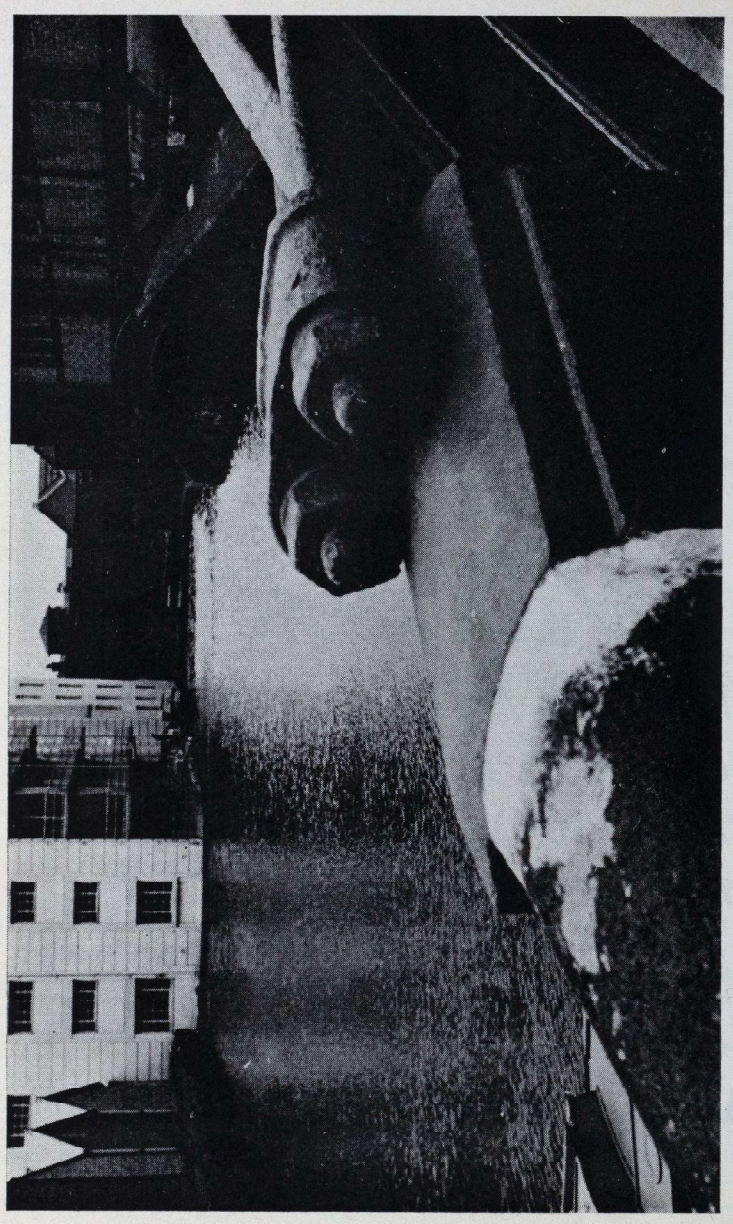
Now, she has arrived, you face the biggest test of your career—the rice. You mustn't fluff it!

There are two ways to cook rice—one in which you allow a lot of water, in which case the rice is drained off from the excess at the end of cooking, the other allowing exactly double the amount of water to rice, so that during the process of cooking the rice swells and absorbs the water and does not then need draining. Either way is equally as good.

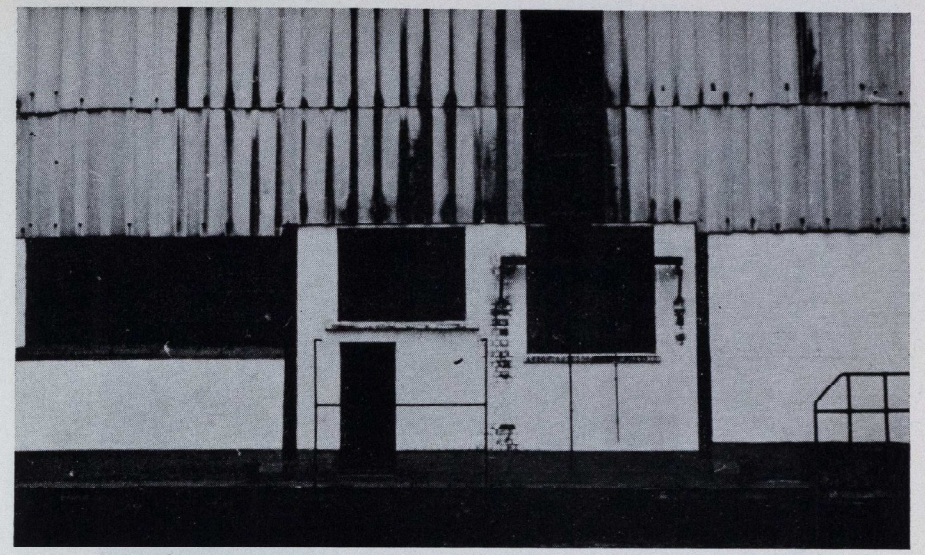
The rice to use is the long grain patna variety, and two teacupsfull should be placed in a fine sieve and washed thoroughly in a fast running stream of cold water. This removes the starchy powder from the outside and this prevents any stickiness when the rice has cooked. Then pour this into a large excess of salted boiling water and cook for 10-15 mins. onwards—it is important not to overcook the rice. It should be firm, not stodgy and sticky. So, when you think it is cooked, drain off in a colander and then you can put it in the warmed oven for 3-4 mins to dry off—remembering to keep the door ajar so that the moisture can escape.

Now you are ready to serve it—remember to warm up the plates and to make everything look interesting. It is usually worth investing in a jar of mango pickle or lime pickle these always make curry more appetising. Any good supermarket or delicatessen will sell these. To drink with it, cold beer or lager, or water. It is interesting that the only time that water "tastes" wonderful is with a curry!

A tin of lychees is a good thing to have afterwards. Again a delicatessen will sell these and they are not terribly expensive. After that, fig flavoured coffee makes a fine finale to what I hope was a great meal prepared by an enthusiastic cook who managed to charm his lovely guest with a few of the delights of the orient.



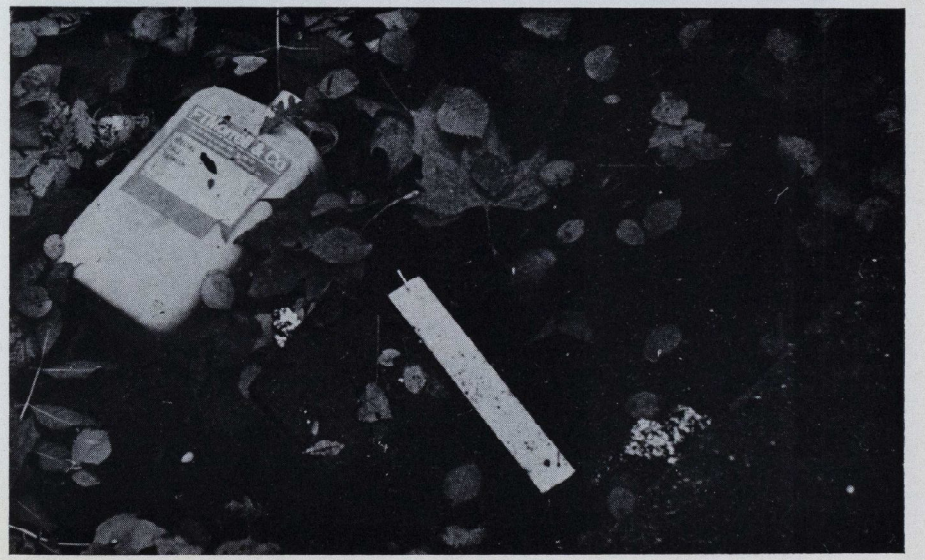
Six Views of a Dead Snake. *This is the Regent's Canal. On a Saturday when the wind is in the east, bringing the first snow of the year. It begins in the Pool of London amid the cranes of scrap metal and timber yards. Past Stepney and Bethnal Green it tunnels for nine hundred and sixty yards under Islington and climbs the Camden locks to end as a piece of park furniture near the Zoo. Nothing moves on it except what the wind can move. The towpath is lonely; a few fishermen sit, and dogs come barking from shacks to guard a pile of planks or some shaggy pine trunks. The staring backs of warehouses and workshops are the only view. Electricity cables plunge into conduits. Pigeons—vultures in this desert—shamble out of dark pipes. The waterway is above all a museum for itself, lying still and quiet in a sunken curving grave.* **Julian Toms.**

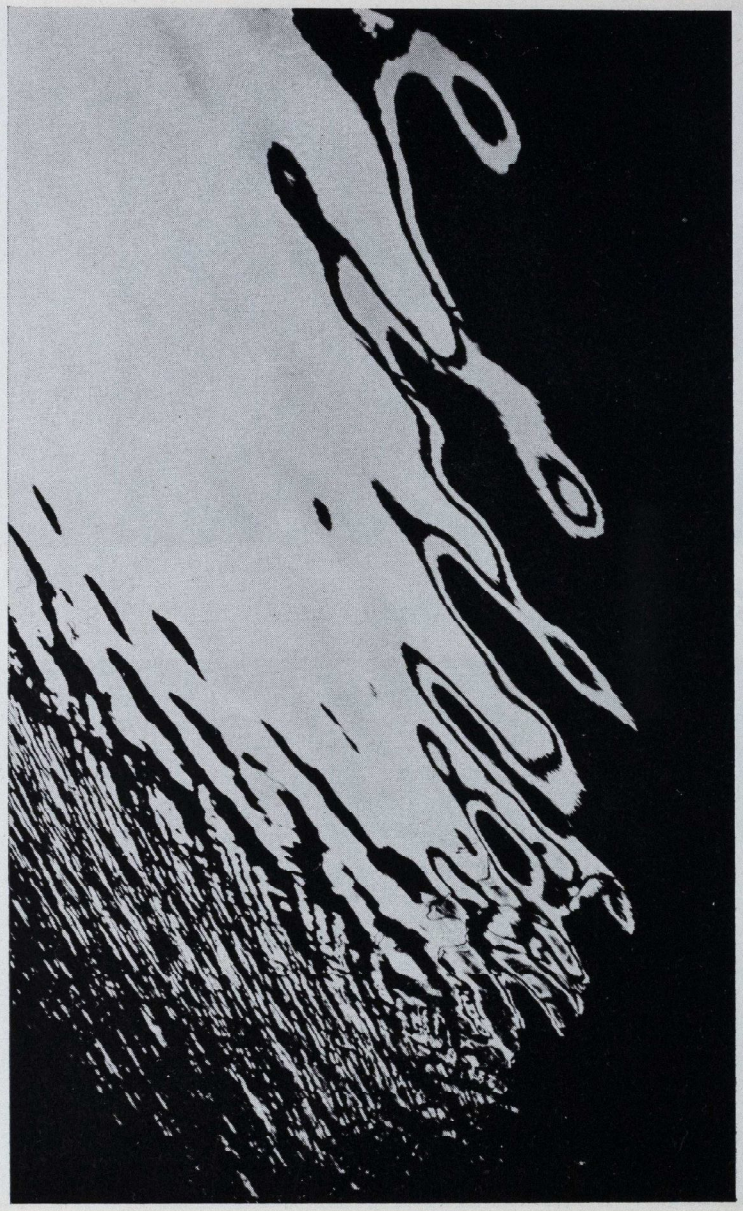


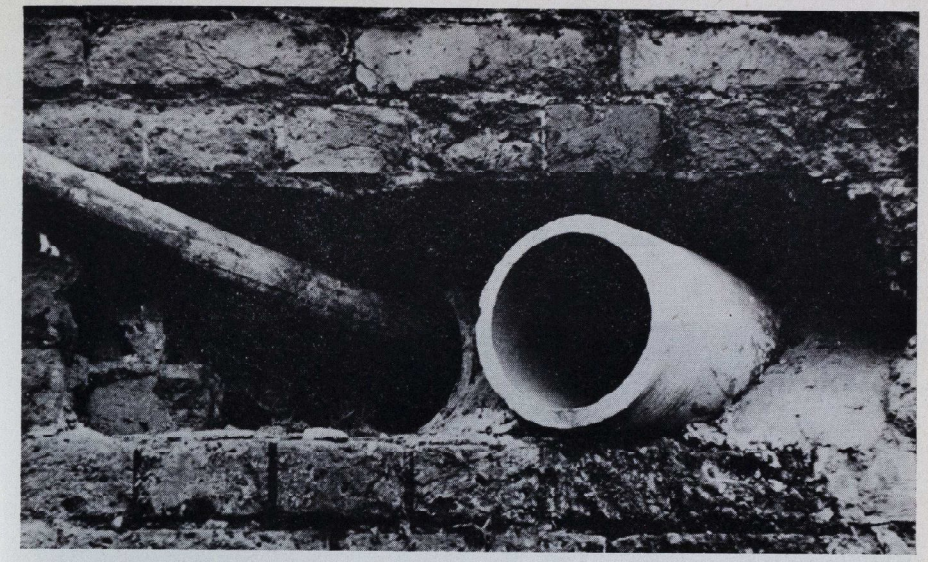
six views

by Julian Toms

part 1

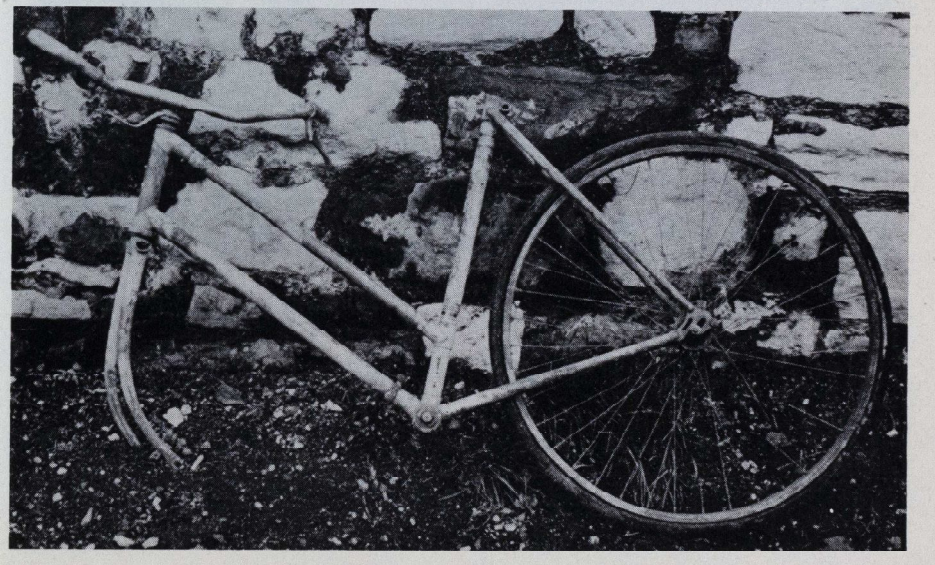






six views

part 2



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immunology

by

Anthony Newman Taylor
and Paul Dieppe

with

Colin Reisner (Text)
Jake Mackinnon (Diagrams)

the authors acknowledge the help of :

Dr. A. G. Stansfeld
Dr. H. Wykeham Balme

Defence against invader or a threat to the host? Modern immunology leaves the textbooks far behind.

In its infancy, immunology was regarded as synonymous with "immunity to infectious disease." Characteristic of this "immunity" was the body's memory for a previous encounter, which was absolutely specific for a particular infection. Once initiated by exposure to an infective agent, the reaction served to protect the host from second and subsequent attacks.

But it was soon shown that not all such "immune responses" were beneficial to the host; they were also responsible for reactions which could be extremely damaging to the host, the so-called "hypersensitivity reactions." In other words, the body's reaction to foreign material (or antigen) could either evoke a state of resistance or of hypersensitivity.

At the turn of the last century von Pirquet pointed out that what was common to these reactions was a changed reactivity of the body, in the form of specific sensitisation, to the particular antigen. He appreciated that although the outcome could be either beneficial (resistance) or deleterious (hypersensitivity) to the host, they both represented the ability of an organism to recognise and respond specifically to antigenic material. As Holborow put it "the capacity to elaborate an immune response is one thing, the results are quite another."

From this work of von Pirquet has sprung the modern study of immunology, which regards immunity as only one facet of immunological phenomena. It is the purpose of this article to discuss the principles underlying immunological reactions, both normal and abnormal, and to relate these principles to certain disease processes in which immunological phenomena are thought to play a part.

part 1

the immunological reaction

(a) *The genesis of tolerance and competence*:

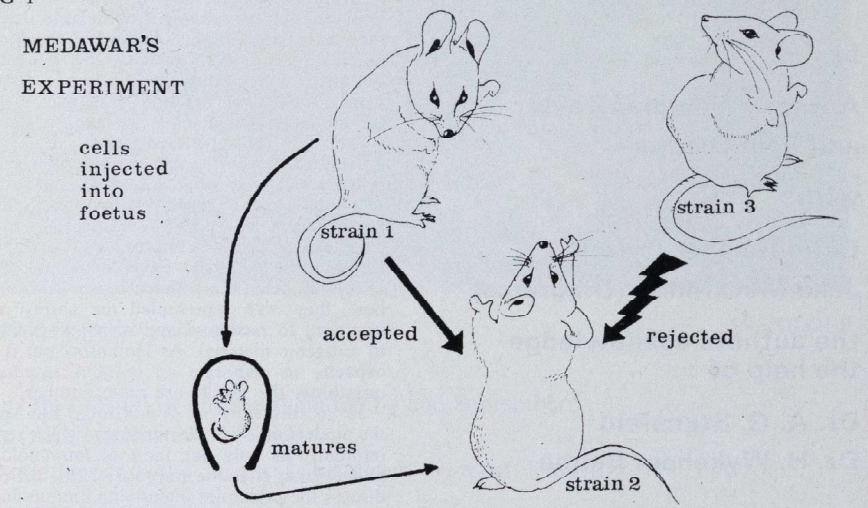
Immunological reactions are concerned with first the recognition, and second the rejection of foreign material present in the host's tissues. This implies the ability to distinguish between "self" and "non-self." Burnet suggested that the ability to recognise and tolerate self antigens developed in foetal life; he postulated that any antigens to which the developing lymphoreticular system was exposed during foetal life would be recognised as self and therefore tolerated. This hypothesis was confirmed experimentally by Medawar. He innoculated foetal mice of one pure line strain with cells from an adult mouse of a second pure line strain. He then showed that after birth, mice from the first line would accept homografts from the second line mice, but would reject homografts from any other line. He termed this phenomenon "acquired immunological tolerance" (Diag. 1).

There appear to be two types of immune response: antibody mediated and cell mediated. The thymus has been implicated in the origin and the development of immunological competence (i.e. the ability to distinguish what is self and what is not self) in the cell mediated response. This is supported by the fact that thymectomy in neonatal mice, and thymic dysplasias in man, are associated with a decreased ability to reject homographs. Work on birds suggests that the bursa of Fabricicius (which may be represented by the lymphoid tissue in the gut in man) may have an equivalent role in the antibody-mediated system.

(b) *Characteristics of an Immunological Reaction*:

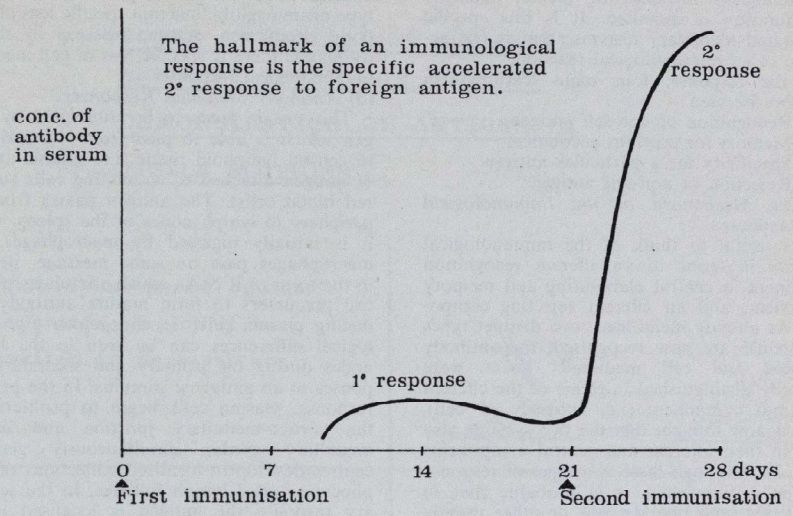
The characteristic features of an immunological reaction are seen in the well known primary and secondary responses which follow exposure of the body to a foreign antigen (Diag. 2). Following initial exposure to antigen a response of low intensity and short duration

DIAG 1

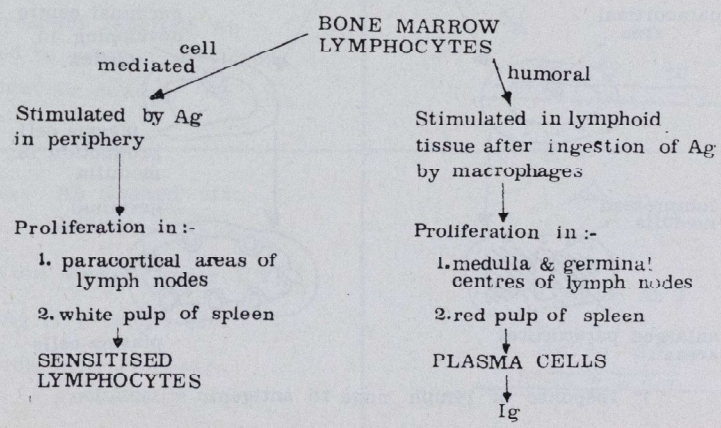


Antigens which an animal encounters in foetal life are regarded as "self"

DIAG 2



DIAG 3



is observed after 10 to 14 days. However, on second and subsequent exposure to antigen, an immediate response of greater intensity and duration is mounted. It is this specific accelerated secondary response that is characteristic of an immunological reaction.

In the response, four main features can therefore be seen:

1. Recognition of non-self antigens
2. Memory for previous encounters
3. Specificity for a particular antigen
4. Rejection of non-self antigens

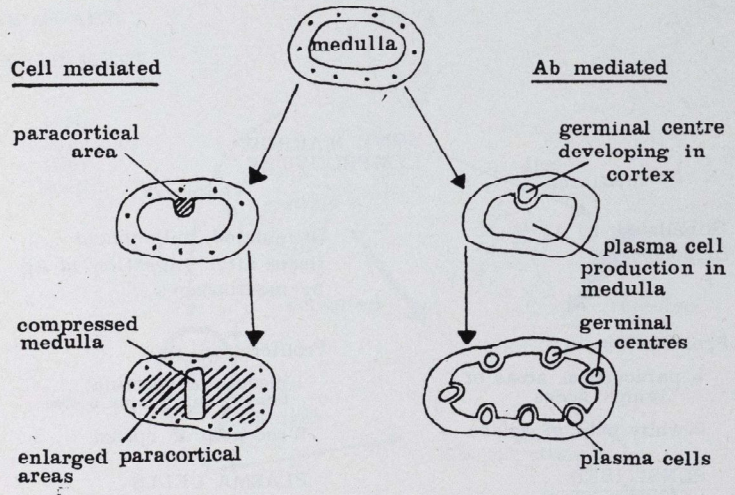
(c) *The Mechanism of the Immunological* Response:

It is useful to think of the immunological response in terms of an afferent recognition component, a central elaborating and memory component, and an efferent rejecting component. As already mentioned, two distinct types of response are now recognised, the antibody mediated and cell mediated. These were originally distinguished in terms of the efferent (rejecting) component (i.e. antibody or cell), but it is now thought that the two systems also differ in their afferent and central components (Diag. 3). Although these two types of response may be distinguished, it is probable that in most cases both operate, one or other usually predominating.

Good clinical evidence exists for this distinction, as one or other response is specifically affected in certain disease processes. In Bruton type agammaglobulinaemia specific loss of antibody production occurs, whereas in thymic dysplasias there is specific loss of cell mediated immunological responses.

(d) *Antibody Mediated Responses*:

This system tends to be stimulated by antigen which is able to pass from the periphery to central lymphoid tissue (i.e. soluble antigen or antigen attached to circulating cells such as red blood cells). The antigen passes from the periphery to lymph nodes or the spleen, where it is initially ingested by macrophages. The macrophages pass on some message, perhaps in the form of R.N.A., which stimulates plasma cell precursors to form mature antibody producing plasma cells. In the guinea pig, histological differences can be seen in the lymph nodes during the primary and secondary responses to an antigenic stimulus. In the primary response, plasma cells begin to proliferate at the cortico-medullary junction and in the medullary cords; simultaneously germinal centres develop in localised collections of lymphocytes called lymph follicles. In the secondary response the antigen is localised in the germinal centres (which may contain the



1° response of lymph node to antigenic stimulation

DIAG. 5

COONS' FLUORESCENT LABELLED ANTIBODY TECHNIQUE FOR THE DEMONSTRATION OF ANTIGEN IN TISSUE SECTIONS

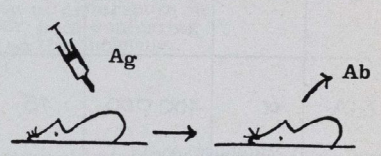
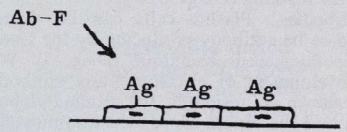
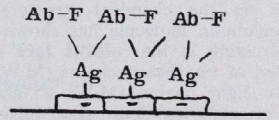
Antibody prepared against the antigen which it is required to demonstrate.	
Fluorescent compound attatched to antibody.	Fluorescin + Ab ⟶ Ab–F
Fluorescent labelled Ab added to section of tissue thought to contain Ag.	
Excess Ab washed off Section viewed under U.V., if Ag is present the section will fluoresce.	

DIAGRAM 6

Ig class	Antigenic component	molecular weight	% of total serum Ig	biological properties
IgG	γ	150,000	80	• found in serum • recognised in vitro as precipitating Ab • possible functions – neutralisation of toxin &c
IgA	α	400,000	15	• found in serum & extracellular secretions • possible main immunological component in external secretions
IgM	μ	900,000	5	• found in serum • recognised in vitro as precipitating Ab • possible functions – agglutination opsonisation

proliferation of plasma cells occurs here as well "memory" cells) and a much more extensive as in the medulla (Diags. 3, 4).

Antibodies: Plasma cells can be demonstrated to be antibody producing by the Coon's immunofluorescent technique (Diag. 5). With the development of electrophoresis antibodies were shown to reside in the gamma-globulin fraction of the serum. More recent immunoelectrophoretic techniques have further differentiated serum antibodies both on physico-chemical and immunological grounds. The groups into which antibodies (or more properly immunoglobulins) are divided by these means have been shown to reflect other and more important differences, particularly functional ones (Diag. 5).

Work on the chemical structure of the immunoglobulin molecule has shown that the basic structure is that of the IgG molecule, composed of two heavy chains (M.Wt 50,000) and two light chains (M.Wt 25,000). Other classes of immunoglobulin (Iga, IgM etc.) are essentially built up from this IgG structure unit (Diag. 7).

(e) *Cell Mediated Responses*:

This type of reaction (Diags. 3, 4) is characterised by the production of immunologically competent lymphocytes. The reaction tends to be stimulated by antigen fixed in peripheral tissues, such as homograft, a tuberculous focus or a modified part of host tissues (of possible relevance in autoimmunity).

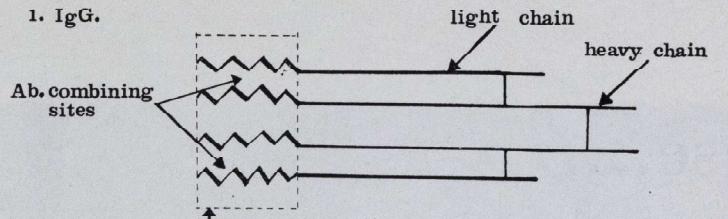
It would appear that lymphocytes are sensitised in the periphery, travel to the para-cortical areas of lymph nodes and the white pulp of the spleen, which provide a *milieu* suitable for their proliferation into immunologically competent lymphocytes.

Considerably less is known about cell-mediated than about antibody-mediated responses. The mechanism of sensitisation of lymphocytes peripherally, and the means whereby they are able to reject an antigen have yet to be understood; however, lymphocytic infiltration in homograft rejections can be seen, and their importance in this and other forms of delayed hypersensitivity is undisputed.

DIAG 7

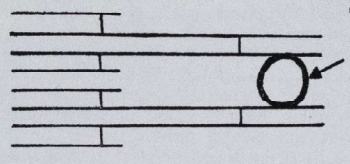
Structure of Ig. molecules

1. IgG.



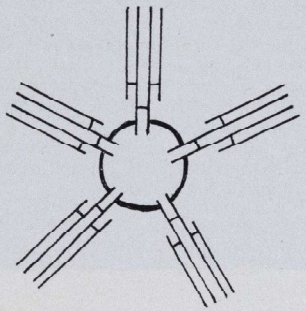
This part of the molecule is for combination with antigen. The majority of differences in amino-acid structure occur here, conferring the necessary specificity on the molecule.

2. IgA



T. piece - probably related to function of IgA as the ab. of secretions.

3. IgM



5 IgG units linked together. This gives great agglutinating power to the molecule.

IgG is the basic subunit of IgA and IgM but both molecules are antigenically distinct from IgG

EDITORIAL · OPINION · EDITORIAL · OPINION · EDITORIAL

noise....

The decibel replaces sterling as the unit of power. Were Britain not surrounded by water (a fact rejoiced in by Shakespeare and ignored by Hitler) neighbouring countries would doubtless by now have complained at the volume of noise our "sceptred isle" produces as the Mecca of grooved sound.

Competition is intense. Record enthusiasts gladly scrap old amplifiers to build bigger and louder versions. Stereo is a neat ploy to produce twice as much sound for four times the outlay. The manufacturers are pleased.

Concorde is the world's fastest passenger aircraft. It is also the most audible. Britain proudly leads the world, by supplying France with the engines.

Beatlemania has produced even more sound than Frank Sinatra. At least the boys run the world's quietest car—another product Britain is inclined to shout about.

American scientists have correlated decibels with brain damage. The Home Secretary does not appear to have heard about this.

Medical Students are reputed to be the noisiest contributors to the current scene . . . the next *Journal* will be on tape.

EDITORIAL · OPINION · EDITORIAL · OPINION · EDITORIAL

Ancient Charterhouse

part 2

immunological abnormalities

There are three types of immunological abnormality:

1. Deficiency in the production of immunologically competent cells and/or antibody.
2. Reactions of antibody or cells with foreign antigen, that in certain circumstances leads to tissue damage in the host following normal antigenic stimuli (i.e. hypersensitivity).
3. Formation of immunologically competent cells of antibody which reacts with "self" antigen (i.e. autoimmunity).

(a) immunological deficiency diseases

There are three basic varieties:

1. Isolated deficiency of antibody production. This occurs in Bruton-type agammaglobulinaemia. In this case there is great susceptibility to acute infections, but delayed-type hypersensitivity reactions, such as homograft rejection, are maintained.

2. Isolated deficiency of cell mediated response. This occurs in thymic dysplasias in which there are normal immune responses to acute infections, but homografts are accepted.

3. Deficiency of both mechanisms. This occurs in Swiss-type agammaglobulinaemia, and may occur in the reticuloses. Immune responses to acute infections are impaired, and homografts are accepted.

These diseases, although of considerable theoretical interest, as they highlight the differences between the two immunological mechanisms, are of rare occurrence, and will not be discussed further. The remainder of this article will be spent in discussing the other conditions in which immunological reactions in the tissues can be directly responsible for disease, i.e. hypersensitivity and autoimmunity.

(b) hypersensitivity

Hypersensitivity reactions are of two basic types depending on whether they are mediated by antibodies or cells. A major point of distinction between the two types is that whereas antibody mediated hypersensitivity reactions may be transferred passively using serum alone, passive transfer of cell mediated responses can only be effected using a suspension of lymphoid cells.

Classically the two types of response were distinguished by the time taken for a secondary response to appear. Reactions occurring rapidly (immediate type) were generally antibody mediated; those developing after 24 to 48 hours were usually cell mediated.

Antibody Mediated Hypersensitivity:

The type of tissue damage caused by antigen-antibody interaction will depend upon the nature of the antibody and the site of the reaction.

(a) Nature of Antibody: Antibody may either be reaginic (IgE) which rapidly fixes to tissues, or non-reaginic (IgG, IgA, IgM etc.) which are not tissue fixing.

(b) Site of Antibody-Antigen Interaction: Reaginic antibody always reacts with antigen on cell surfaces. Non-reaginic antibody may react with antigen free in the circulation, in tissue spaces, or with antigen attached to cell surfaces.

There are therefore three basic types of antibody mediated Hypersensitivity:

1. Free antigen reacting with antibody which has become fixed to a cell surface (i.e. reaginic antibody). This type of reaction is responsible for anaphylactic reactions.

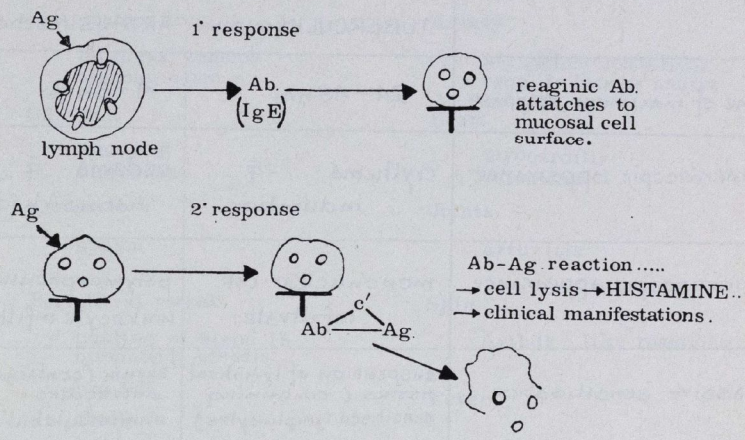
2. Free antibody (IgG, IgA, IgM etc.) reacting with antigens on a cell surface. This type is responsible for blood transfusion reactions.

3. Free antigen reacting with free antibody. This type of reaction occurs in the Arthus reaction, and is responsible for acute glomerulonephritis and many other lesions.

Anaphylaxis:

Anaphylactic reactions (Diag. 8) are characterised by the formation of reaginic antibody (IgE) in response to antigenic stimulation. IgE is fixed in mucosal and other cell surfaces; on second and subsequent exposure to antigen, a complement-fixing reaction occurs at these surfaces. This causes tissue lysis and thereby the release of chemical mediators (e.g.. histamine) which give rise to the characteristic

DIAG 8 ANAPHYLAXIS



features of these reactions (i.e. wheal and flare in the skin, bronchospasm etc.). Another, but ill-understood feature of these reactions, is the associated eosinophilia.

Anaphylactic manifestations may be local or systemic depending on the portal of entry of the antigen. In local anaphylaxis, antibody-antigen reactions may occur at exposed mucosal surfaces such as the respiratory tract, where exposure to pollen or animal dander in susceptible individuals can give rise to rhinorrhoea and bronchospasm, as in hay fever and allergic asthma. If the antigen enters the circulation (e.g. a drug or an insect bite) the subsequent release of histamine may be localised, as in skin atopies, but may cause such widespread vasodilatation and oedema, due to increased capilliary permeability, that a state of peripheral circulatory collapse ensues (i.e. shock).

It is important to appreciate that this, as are all hypersensitivities, is an immunological reaction, and characterised as such by a specific accelerated secondary response. Anaphylaxis can only occur in an individual previously exposed to that particular antigen (either consciously or unconsciously). Desensitisation of anaphylactic individuals may be produced by multiple subcutaneous injections of low doses of the antigen. As a result of this treatment the patient can develop non-reaginic antibody, which, by combining with antigen before it is able to react with reaginic antibody, can prevent anaphylaxis.

Cytotoxic Reactions:

These reactions occur when antibody reacts with an antigen on a cell membrane. Complement is fixed and cell lysis results. The best known examples of this type of hypersensitivity are blood transfusion reactions and haemolytic disease of the newborn.

Imune Complex Reactions:

These reactions occur in one of two situations:

1. Where there is high concentration of free antigen in the tissues together with a high concentration of free circulating antibody: For reasons not fully understood antigen and antibody diffusing towards one another may lead to an antigen-antibody reaction within the walls of small blood vessels, complement is fixed with associated polymorphonuclear infiltration (several components of complement, C5, C6, C7, have been shown to be positively chemotactic for polymorphs) and tissue necrosis. The resulting vasculitis leads to vessel damage with haemorrhage and oedema into the tissues. The classical model of this type of reaction is the Arthus reaction (Diag. 9).

2. The second type of immune complex reaction occurs when there is a high concen-

DIFFERENCES between TUBERCULIN REACTION (cell-mediated) & the ARTHUS REACTION

	TUBERCULIN reaction	ARTHUS reaction
time of maximum response	24–48 hrs	4–8 hrs
macroscopic appearance	erythema & induration	oedema & haemorrhage
microscopic appearance	mononuclear cell infiltrate	polymorphonuclear leukocyte infiltrate
passive sensitisation	suspension of lymphoid tissues (containing sensitised lymphocytes)	serum (containing antibodies – immunoglobulins)
systemic manifestations	fever	deposition of "immune complexes" in organs

tration of both antigen and antibody within the circulation. Insoluble free circulating complexes are formed which can be deposited in the basement membranes of small blood vessels, e.g. in the glomeruli in acute glomerulonephritis and possibly in synovial membranes in acute rheumatic fever.

Serum Sickness:

Serum sickness is a reaction to the injection of foreign serum. It was first described soon after the introduction of diphtheria antitoxin serum prepared in the horse. The reaction which occurs in serum sickness may be the result of both reaginic (IgE) and non-reaginic antibody. The lesions that develop will depend upon relative amounts of each antibody produced, i.e. IgE will result in anaphylactic manifestations, whereas non-reaginic antibody production may lead to immune complex reactions (Diag. 10).

Cell Mediated Hypersensitivity:

This group was originally distinguished clinically from the other types of hypersensitivity by the time taken for a secondary response to develop, being known as delayed hypersensitivity. However, it is now appreciated that all delayed type hypersensitivities are examples of cell-rather than antibody-mediated reactions, and several other distinguishing features are also recognised.

Very little is known about these reactions although the tuberculin reaction and homograft rejection, which are the classical examples, have been studied in great detail. As may be seen from the table many other examples of cell-mediated responses are now recognised.

(c) autoimmunity

Autoimmunity may be defined as a reaction in which cells or antibody react with host tissue antigens, tending to cause rejection of part of the host. This implies that tolerance must either be evaded by antigenic change, or lost via lymphoreticular tissue change. Once tolerance is either lost or evaded, so that cells and antibody can react with a part of the host, the immunological reactions that follow are hypersensitivity reactions of the type considered above, e.g. cytotoxic reactions and immune complex formation with host antigen.

DIAG 10 SYMPTOMS AND SIGNS OF SERUM SICKNESS

1. Anaphylactic.	2. Immune Complexes.
Respiratory tract	Kidney
laryngeal oedema bronchospasm	ac. glomerulonephritis renal failure & anuria
G.I. tract	Heart
vomiting diarrhoea	myocarditis valvulitis
Bladder	Joints
spasm	arthritis
Peripheral vessels	Skin
pooling of blood in mesenteric vessels	"Arthus like" reactions
low BP	Fever
shock	
Skin	Leucocytosis
urticaria maculo-papular eruptions	

In autoimmune reactions the antigen is part of the host, and thus always present and available to the lymphoreticular system. This means that the classical specific accelerated secondary response, by which an immunological reaction is recognised, cannot be demonstrated. Autoimmunity is suggested whenever cells or antibodies that react with host tissues are found in the circulation, but this may be of no significance to the disease-process in question, and it is therefore vital that we find other criteria to define whether an autoimmune reaction is taking place or not. Waksman suggests these criteria:

1. An initiating event and latent period prior to the development of the lesions.
2. Presence of antibody and/or cells that react with host tissues.
3. Lesions compatible on histological grounds with others produced by immunological reactions.
4. Passive transfer of the disease by cells or antibody.

These criteria are difficult to apply to diseases in man, especially the crucial fourth criterion, and thus no disease can be considered a proven case of autoimmunity. One way of surmounting this problem is to obtain experimental models of human diseases in laboratory animals, and applying the criteria to them; much of the recent work on autoimmunity has had this aim.

Two Types of Autoimmunity:

After Burnet and Medawar had established the concept of tolerance to host antigen, possible mechanisms of autoimmunity could be visualised. Antigen-specific autoimmunity could develop via a peripheral change in antigenic stimulation, i.e. evasion of tolerance; and generalised autoimmunity could develop via changes in the central lymphoid tissues leading to defects in tolerance (Diag. 11). This theoretical concept seemed to have some clinical justification; there are a number of diseases with these features in common:

Familial incidence,
Higher incidence in women than in men,
Progressive inflammatory or cytotoxic lesions
Unknown aetiology,
Serum autoantibodies present.

In addition some are associated with changes in the thymus, and there are often associations between different members of the group, and of members of this group with other immunological abnormalities. Some of these diseases, such as Hashimoto's thyroiditis and pernicious anaemia, involve tissue specific lesions and equally specific autoantibodies; others, such as systemic lupus erythematosus and rheumatoid arthritis, are generalised diseases, and a wide and variable spectrum of autoantibodies may be found. It was suggested that the former group was produced by peripheral evasions of tolerance, and the latter group be central defects of tolerance. Much work has been done with this concept in mind, and it seems to be becoming apparent that while the concept of evasions of tolerance is a valid one with clinical significance the concept of defects of tolerance is a doubtful one, and possibly need never be involked to explain human disease.

The Significance of Autoantibodies:

One of Waksman's criteria of autoimmunity is demonstration of autoantibody; it is the one criterion that is easily applied to human disease, and unfortunately many diseases have been labelled "autoimmune" on this basis alone. There are three possible explanations whenever autoantibody is found:

1. It is the cause of the disease.
2. It is produced secondarily to other damage to the host, and then contributes to a disease process.
3. It is produced secondarily, and has no bearing on the disease process at all.

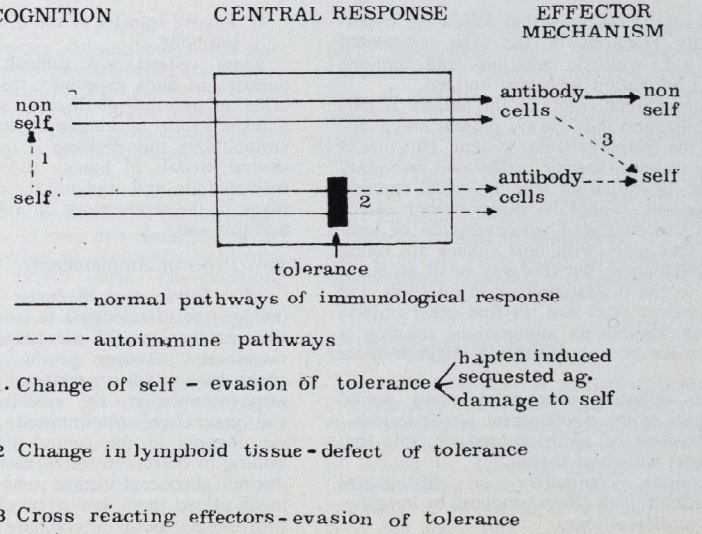
As more work is done on autoantibodies, more seem to fall into one of the second two categories rather than the first.

Evasions of Tolerance:

Evasions of Tolerance may be divided into "false evasions" where the reaction is dependent on the presence of foreign material; and "true evasions" which are initiated by host tissue alone.

a. Hapten Induced: Attachment of simple chemical groups can change the antigenic nature of a protein. It would appear that the attachment of the drug sedormid to platelets can form a new antigenic structure in the platelets and thus lead to the production of autoantibody that destroys the platelets and causes thrombocytopaenic purpura. Other, similar reactions, particularly in the skin, may occur in this way.

DIAG 11 PATHWAYS OF AUTO-IMMUNITY



b. Cross-reacting Antigens: Sensitised cells or antibodies produced in reaction to foreign material might cause damage to the host tissues. The work of Kaplan has produced compelling evidence that the cardiac lesions of acute rheumatic fever are produced in this way. Haemolytic streptococci were isolated from patients with rheumatic fever, and injected into rabbits, serum antibody produced by the rabbits was found to cross react with human cardiac tissue. But why does this reaction only follow throat infections, and why only in certain individuals? Why is it that instead of a cross reaction occurring with host tissues, a cross tolerance to the streptococcus is not found? These questions might be answered to some extent by saying that the efferent limb of the immunological reaction is more specific than the afferent, or that antigenic stimulation is as dependent on the environment in which the antigen exists as on the antigen itself. However, no fully satisfactory answers to these questions can yet be given.

c. Altered self: Any disease process might so alter the tissues of the body as to render them antigenic. That this can occur is demonstrated by the presence of autoantibodies following myocardial infarction, and in any chronic tissue destoying disease such as tuberculosis and syphilis. Autoantibody formed in this way might cross react with normal host tissues, and might in this way contribute to the continuation of a disease process. One immunological theory of cancer aetiology involves the concept that some form of trauma (perhaps viral in some cases) so changes the antigenic structure of cells as to deprive them of the normal immunological mechanism that protects them from neoplastic proliferations.

d. Sequestered Antigen: Development of tolerance seems to be dependent on the presence of antigen in the circulation during the foetal and neonatal period. If an antigen of host tissues was first released into the circulation after this time, an immunological reaction might occur. This may explain some of the inflammatory lesions that can follow trauma to the eye, parts of which are avascular, and some cases of male infertility, as the sperm do not develop until after the period of development of immunological maturity. It has also been suggested that this is the mechanism for formation of anti-thyroglobulin antibody found in Hashimoto's disease.

Defects in Tolerance:

The idea that some abnormality of the lymphoid tissues allows production of widespread immunological reactions to host tissues is now an old one. This theoretical concept has proved difficult to justify.

An inbred strain of mice—NZB/BL—spontaneously develop a disease that is characterised by Coomb's positive haemolytic anaemia, variable lymphocyte infiltrated inflammatory lesions and a generalised proliferation of lymphoid tissues with the presense of autoantibodies. It was suggested that this was analagous to S.L.E., and an example of a disease caused by a defect in tolerance. However, a virus has recently been found in the lymphoid tissue of these mice, and in some of the lesions, thus casting doubt on the concept of a primary immunological aetiology. Other diseases and experimental models have been proved equally disappointing.

combined abnormalities, S.L.E:

Most diseases have a multifactorial aetiology. Autoimmunity was greeted with great enthusiasm when first suggested, and became "the cause" of many diseases; it is likely that it will slowly find its place as a contributory factor in a limited number of lesions. However, immunological abnormalities are undoubtedly important in many diseases, and it is interesting to note that the different types of abnormality described above are often associated—this is apparent from the case histories given below. These points may be illustrated by quoting one of the many unproven theories of the aetiology of S.L.E.

The model of the NZB mice suggest that the initiating event might be something like a viral infection. This causes widespread tissue damage resulting in the formation of a number of autoantibodies, and perhaps in a derangement in the central lymphoreticular component of the immunological response. Resulting autoimmune and hypersensitivity reactions are variable and unpredictable, but seem to contribute to the disease process; for example immune complexes may form with host antigen and thus cause damage to the glomeruli and synovial membranes to explain the nephritis and arthritis. Thus the lesions could be due to a combination of infection, hypersensitivity and autoimmunity.

Case Histories

By kind permission of Dr. H. Wykeham Balme

These histories are from patients suffering from conditions thought to be at least partly due to immunological abnormalities; they illustrate the close relationships between hypersensitivity and autoimmunity.

hypersensitivity

Case 1. Z. A. age 29.

Presented in June 1969 with acute oedema of face and hands, and an erythematous rash, with vesicles, on the face and on the arms distal to the elbows. There were no other findings on examination and all investigations were normal. The patient gave a history of working in a chemical re-crystallisation plant, and vividly described the way in which he had wiped his gloved hand over his face. The chemical that he was most exposed to was dinitrophenol. His symptoms slowly disappeared, and treatment was limited to use of chlorphenyramine (piriton). The unconfirmed diagnosis was of hypersensitivity to chemicals used at work, especially dinitrophenol.

henoch-schonlein purpura

Case 2. A. H. age 67:

Developed a cough and wheeze in April, 1969. On the second day his ankles became red, swollen and painful, 3 days later his knees were affected in the same way, and a rash appeared on his calves. Two days after this his elbows and left wrist also became involved. Ten days after the onset of the cough all his joints had recovered, but the rash on his legs, and swelling of the ankles remained. He then became short of breath, and three weeks after the onset of the illness was admitted to hospital. On examination he was dyspnoeic, and had swelling and a purpuric rash over his ankles. Investigations: ESR 63; urine contained albumin, red cells and casts. A diagnosis of Henoch-Schonlein purpura with renal involvement was made. The albuminuria and red cells in the urine failed to disappear with prolonged bedrest, and the patient was therefore given prednisolone. Soon after this all investigations were normal and the patient was discharged.

rheumatoid arthritis

Case 3. F. McC. age 51:

First presented in 1960 with fleeting joint pains mainly affecting the hands, elbows and feet. Suffered from intermittent attacks of arthritis for the next four years, and since then has had almost continuous trouble, especially with her knees, in spite of treatment. Was admitted to hospital in 1967 for a synovectomy of the left knee joint; at this time the ESR was 35, and the latex fixation test for rheumatoid factor positive. While in hospital she developed a macula-erythematous rash for which no satisfactory explanation was found, it was thought to be a drug reaction and it was noted that she is prone to this type of rash. She was readmitted to hospital in 1969 for synovectomy of the right knee. Hb 70%, ESR 40. Latex fixation test positive, nodules were present over the flexor tendons of the hands. A diagnosis of chronic rheumatoid arthritis was made.

S.L.E.

Case 4. L. M. age 60:

First presented in 1958 with vague pains in the arms and knees and a persistently raised ESR. Was next seen in 1967 when she had pain and stiffness in the neck, shoulders and knees and swelling of both hands. At the same time she developed a fever and rash over exposed areas after sunbathing. A diagnosis of systemic lupus erythematosus was made after L.E. cells had been found. In December, 1968 she had a severe episode of pneumonia that responded slowly to antibiotics. In June 1969 she was admitted to hospital with left pleuritic pain and evidence of pulmonary hypertension as well as infection. She developed a severe dermatitis, thought to be a reaction to mefanamic acid, and for which she had to be treated with steroids. Investigations at this time: Hb 68%, ESR 106. L.E. cells present Antinuclear factor positive.

then and now

by Brian Owen

Disillusioned, Brian Owen looks at Cambridge: dream or reality?

At school my mind was formed by masters who had been at Oxford or Cambridge in the 1930s. Few of them had read Natural Sciences: and those who had seemed dull dogs. They had not, apparently, walked around quadrangles after a good dinner, talking of scabrous Greek verses commemorating the charms of ambiguous recipients: they had not been in clubs which talked wildly and brilliantly of Isherwood and Auden: they did not know people who had spoken with Russell or Wittgenstein: they had not travelled Italy to perfect their language and immerse themselves with other companions in the treasures of the Uffizi, and taken occasional relaxation among the carefree Neapolitans. True, a chemistry master had been forced to sit through Lord David Cecil's lectures on Wilde, since his wife read English and insisted on broadening his mind. But his verdict on these lectures was that they were "wet" and worse. For the most part, he and his scientific colleagues had occupied themselves with beer and machinery. The "broad education" passed them by, it seemed.

And the 'real' University, to me then, was revealed in interludes in the classical sixth, when the Senior Master drifted from an exegesis of one crucial line of the Aeneid, or an enthusiastic alteration of our proses into elegant periods—"this is just the spot for a 'quippe qui' and a subjunctive—there—how much better—and now end with an 'esse videatur'."

And then he might talk of the walks, the talks, the leisure to unfold ideas, the bibulous dinners followed by tumbling into a taxi, and off to the cinema to hurl cabbages at the organist (who played with one hand and hurled them deftly back with the other). Breakfast parties occurred: and scouts brought tea to your rooms. He lent me "David at Kings"—all warm fires, mullioned windows and mulled wine. I read Evelyn Waugh and Aldous Huxley—their lives too moved in a pattern of frenzy superimposed on a backcloth of leisure.

I bought many books, and arrived at Cambridge to enjoy the leisure, not without hope of enjoying some amiable frenzy as well.

It was a little comfort to find that Max Beerbohm, many decades before, experienced the same disenchantment with Oxford as I with Cambridge. Many hours in the dissecting room seemed poor fare compared with dozing over dead languages in a comfortable library. Learning the perverse course of the third cranial nerve was banal compared with delving into the treasure-troves of European thought of recent centuries. And the calming effect of those calm buildings: where was it? They were points B, C, D passed in scuttling fast from a rushed breakfast at A (my room) to E (the pharmacology practical, which had to be signed for). My mentors had told me of tutorials—one read some books, one had some good ideas and wrote an essay; one read to a pipe-

smoking, avuncular tutor who thought some ideas "rather good" and others meet to be corrected by reference to recent excavations in Delos. In my essays, there seemed to be no opportunities for ideas or anything except knowing which muscles were used in walking —or the recent theories on the aetiology of hypertension: or being ignorant of these. They took the form of "some authorities hold that . . . whilst others have been unable to confirm their results." The art resided solely in the ability to get the lot in, with judicious emphasis to avoid a rag-bag effect.

Social life, too, was not so much frenzy superimposed on leisure, as frenzy on frenzy. Who could be seen at most parties? Who could be most socially viable? Spectacular breakdowns took the place of the eccentric pranks of yesterday; and cannabis began to take the place of alcoholic bouts followed by escapades.

Of course, the old-style life continued, in small pockets, but most in a rather self-conscious cliquey way which put one off. Yet there were occasions when the old picture was realised. Weekend strolls in the summer for tea at Grantchester: motoring to London for the occasional theatre or concert: lazy evenings drinking and chatting and going to the Arts cinema for ludicrously cheap prices. My director of studies was a lover of good wine, and meetings which started out as planning and advice often became wine-tasting episodes. I also escaped, to my great relief, doing biochemistry, in which I did not much believe—perhaps irrationally—since I could not feel that the elucidation of isolated parts of cellular metabolism would provide the key to medicine in my lifetime or my children's. Nor did I feel that the secret of life was wholly to be found in unravelling DNA, as seemed to be the prevailing opinion. Perhaps this was because I was too ignorant: but I also felt that there was too much top-heavy fanaticism about it all, and escaped thankfully in the Philosophy of Science as a minor subject—the only part of the course where I felt there was some taste of education as she had been.

All around were signs of the technological revolution, as buildings were pulled down for new laboratories and life was increasingly streamlined. The place was determined to be a backwater no longer. Malcolm Muggeridge appeared to me an increasingly sympathetic figure. Personalities were out: functionaries were in (but at least not in my college, where the most lively and personal contacts were maintained by enlightened fellows).

Once, my erstwhile masters came up to dinner. They could not believe there was so much competition. They could not believe that here, of all places, the demands of earlier and more intense specialisation would be felt so soon. They could not believe that long dinners, served in style with silver, had given way to a cafeteria system on *plastic trays*: that tea could no longer be had in rooms: that multiple choice papers were even now being offered in further degrees, with no opportunities for demonstrating one's grasp of the broad scope of the subject by apt phrases, despite certain lacunae where hard facts were concerned. But perhaps DNA has more to offer, in the long run, than the broad scope of life, the telling phrase and the reflective judgment.

Book Reviews

"A Synopsis of Surgical Anatomy," by A. Lee McGregor, M.Ch., F.R.C.S., and D. J. Du Plessis, Ch.M., F.R.C.S., Tenth Edition. Bristol, J. Wright & Sons, 1969, pp. 894 + xi, 802 illus. Price £2 2s.

The appearance of a new edition of this well established work is an occasion of interest to both anatomist and surgeon, for although the book does not prepare the student specifically for any of the present undergraduate or postgraduate examinations, this is probably a fault in the British examination system, rather than any lack of insight of the senior author.

It is disappointing therefore to find that the addition of a co-author has not been accompanied by a substantial re-organization of the extensive text. The division into normal and abnormal anatomical sections is now largely artificial and although a section is devoted to congenital errors, anomalies of the cardiac, venous and biliary systems appear under different headings. This poor organization is only partly compensated for by a comprehensive index.

Old anatomical nomenclature is retained and many obsolete operations are described, of particular note being the recommendation of total thyroidectomy for cardiac failure. The surgical approach to hand infections also follows the traditional teachings of Kanaval rather than the current practice. The amputations around the knee joint, which the authors declare obsolete, are now returning to the surgeons' repertoire, although it is not inconceivable that they will again be out of favour with the limb fitters before the book reaches it's next edition.

The omission of that anatomical gem, Marcille's triangle, will be mourned by many, for it remains a treacherous area for even the most skilled vascular surgeon. Yet the authors retain both a 250 word discussion on the outdated perivascular neurectomy and a 1,500 word description of Smithwick's, now rarely performed, lumbodorsal splanchnicectomy.

The book's print is small and tiring to read, while the diagrams are poorly reproduced (and in places too difficult to orientate). Weighted against these latter factors, however, is the very reasonable cost.

Despite many defects, "A Synopsis of Surgical Anatomy" still provides an invaluable collection of surgical and anatomical data and thus remains an important reference book for the shelves of all aspiring surgeons.

JOHN S. P. LUMLEY

"Common Symptoms of Disease in Children," Second edition, 1969, by R. S. Illingworth. Blackwell. Price 42s.

That this second edition has followed so soon after the first is a definite indication of its widespread popularity. Nevertheless, each section has been fully revised and many additions made to the text and references.

This book is of particular value to the General Practitioner and those who have to deal with children. Here they will find a practical account of the many causes of each symptom. Further, the indications for seeking the opinion of a paediatrician are clearly stated. There is definite guidance as to the essential investigations which must be carried out. He also indicates the subsequent investigations which may be carried out in hospital to establish the diagnosis.

Although the author stresses that he does not necessarily list the causes of the symptoms in order of frequency, one is left in no doubt as to the commonest and the most important.

It is the author's intrinsic experience and practical approach to the clinical problem which pervades this book and makes it most valuable to the General Practitioner, House Physician and medical student.

MARGURITE SMITH

Blackwell Scientific Publications. Price 27s. 6d. "Interpretation and Uses of Medical Statistics," by G. J. Bourke and J. McGilvray.

"Statistics can prove anything" is the cry of those with no understanding of the subject, but statistical methods are not concerned with *proving* anything. They are methods of subjecting experimental observations to precise critical analysis and deciding whether the observations support a particular interpretation or theory. The language of statistics is filled with words like "probability" and phrases like "tests of significance". Statistical methods are of great value in biological and medical research where any observations are likely to be clouded in a confusion of biological variability.

G. J. Bourke and J. McGilvray have attempted to fill the need for a book on statistics which may be read (and understood) by the non-specialist. In doing so they have avoided two pitfalls, neither producing a book which requires a knowledge of mathematics, nor one which is superficial. The book does not aim to be a textbook, and in general concentrates on interpretation rather than on calculation; at several points it avoids the details of statistical tests while illustrating the value of them quite clearly. The book deserves careful reading but it is only 160 pages long; it adopts the technique of giving illustrative problems from the medical literature to demonstrate the need for the various statistical tests,

and the meaning of them. There is also a short final section on computers in medical research, but this section tends to concentrate on the mechanics of presenting information to a computer, and is disappointing when compared with the rest of the book. However this book can be recommended to those who wish to understand the value of statistical methods, or to those who may then wish to learn to apply the techniques for themselves.

C. H. MARSHALL

"Gynaecological and Ostetrical Anatomy: Descriptive and Applied," (Fourth edition), by C. F. V. Smout, F. Jacoby and E. W. Lillie, published by H. K. Lewis and Co. Ltd. Price £4 10s. 0d. pp. 430, 182 illus.

There is a place for a textbook of anatomy related to Obstetrics and Gynaecology. The basic knowledge is of value to the undergraduate in trying to understand the mechanics of disorders such as prolapse, and in attaching some meaning to the information gained from bi-manual pelvic examination. At the microscopic level the boundaries between anatomy and physiology are blurred, and physiological aspects have to be emphasised. In such a clinical subject the basic science only becomes intelligible and useful if it is applied. For instance variations in labour can be related to anatomical features in the pelvis, and the natural history of cervical cancer can be related to the topographical anatomy and to the lymphatic drainage.

This is the fourth edition of Smout's Anatomy and the author states that in retirement he has been pressed to produce another edition with the help of a practising obstetrician. This assistance has not, however, proved sufficient to instil new ideas into the book, without which no progress can be made. It is precisely because obstetricians need to glean inspiration from allied disciplines, that such a book should have an up to date anatomical background. The majority of the references are some twenty years old. Views on anatomy and physiology of pelvic organs, particularly the bladder and rectum have evolved considerably during this time, but little account is taken of these views in spite of the fact that modern surgical treatment may be based on them. These omissions are so grave that this book can no longer be recommended for M.R.C.O.G. candidates. It remains, however, a book that may well be consulted by undergraduates who wish to revive their coarse anatomy during their relevant clinical study.

The sections on the more detailed anatomy of the endometrium and ovary, together with the physiology are very different. These are clear, well written and well supported.

C. N. HUDSON

"Lecture Notes on Histology," by William A. Beresford, Blackwell Scientific Publications. Price 25s.

Those readers familiar with other works in the "Lecture Notes" series will not be disappointed by the latest addition to the rapidly growing family. "Lecture Notes on Histology" continues the now familiar pattern of presenting accurate and extremely concise information in a volume of reasonable size at a reasonable price.

This book covers virtually all aspects of Histology (some in considerable detail considering the title of the work) and includes practical aspects such as exactly how to use an optical microscope to the best advantage without first acquiring a degree in optical physics.

A refreshing aspect of the book is the inclusion of sufficient physiology, pathology, and clinical information to capture the interest of the medical student and remind him of the inseparable association with these subjects, a fact painfully forgotten in the great majority of standard works on histology. Useful also to the bewildered first year student is the fact that the author includes all the common synonyms of important words, thus eradicating a common source of confusion.

One marked feature of the book is the absence of drawings and photographs. This inevitably limits the work in its appeal, for histology is essentially an illustrated subject. The author himself explains that the book is a "brief supplementary text," and I think it important that a student contemplating buying this book should realize that it will need to be supplemented by reference to a larger textbook of histology. As an introduction to the subject, however, or for last minute revision it is ideal.

One last word of warning for those with weak literary digestions who like to take their information between generous slices of "padding" and who may not be familiar with the "Lecture Notes" style. This book, like the others, is solid fact.

DAVID ALLISON

survey

Students Union Teaching Committee and Journal. Paediatrics and Obstetrics. Appointments Survey.

Bart's gives its students the opportunity to go abroad for 3 months to study paediatrics. At the time when students arrange where they will spend these 3 months, they have little information to help them decide which are the best centres to visit. It was felt that written reports from every student who has completed the appointment would be of great help to later students and that these reports could most conveniently be collected in questionnaire form.

Accordingly I have been sending, and shall continue to send out questionnaire forms to those who have completed the paediatrics appointment. I am also running a similar survey of obstetric appointments. If there is sufficient agreement between different people who have completed appointments in the same hospital, conclusions drawn from their reports may be published in the *Journal* at a later date.

Copies of the questionnaires which have been returned will be kept in a file in the hospital library where it will be possible for them to be consulted by students planning to visit the hospitals concerned.

Obviously the success of these schemes depends on you who receive the questionnaires. Please help by returning them. I hope you will feel that it is a valuable service to other students and I hope they will use it.

notice

XIIIth DECENNIAL CLUB OF
ST. BARTHOLOMEW'S HOSPITAL

Preliminary Notice: The 1969 meeting of the XIIIth Decennial Club will be held at St. Bartholomew's Hospital on the second Saturday in December (13th December, 1969).

In view of the particularly successful 1968 Meeting in the Great Hall of the Hospital, and bearing in mind what we thought was the consensus of opinion at the time, we have asked for the use of the Great Hall again for the 1969 Meeting.

However, it appears that last year we displaced the League of Nurses who hold a meeting in the Great Hall annually on the first Saturday in December. It has therefore seemed to us only courteous to give up the first Saturday to the Nurses, and transfer our own meeting to the following week, the second Saturday in December.

Further details about times and speakers, etc., will be sent individually later, as usual, and when the time comes we will hold a short dinner-time discussion on our future statutory date of meeting. We hope all this is not too arbitrary on the part of your secretaries, and complaints will be humbly accepted.

JAMES O. ROBINSON,
R. F. McNAB JONES,
Secretaries

July, 1969

Letters to the Editor

Dear Sir,

Dr. John Howkins says in his interesting article entitled "Meminisse Juvabit" (June journal) "In 1938, so far as I know, Sir Thomas Dunhill had been the only extramural in the closed shop of St. Bartholomew's Hospital." This is not correct. In 1920, Dr. (Sir) Francis Fraser and Dr. (Sir) Thomas Dunhill, C.M.G.[1], were appointed assistant directors to the newly created medical and surgical clinics. The previous appointment of an extramural was in 1877, Dr. (Sir) Matthews Duncan[2] who had trained in Edinburgh, was elected lecturer in midwifery. He made a great name for himself and for the hospital by his practice and teaching. His name is commemorated in the Matthews Duncan Medal.

Again, between 1868 and 1875 no less than three assistant physicians were appointed. Dr. Samuel Gee[3], who had trained at University College Hospital and Great Ormond Street; a year later Dr. (Sir) Dyce Duckworth[4], an Edinburgh man; and in 1870 another vacancy occurred and Dr. Philip Hensley, a Cambridge man, who had trained at the hospital was appointed. Five years later, in 1875, Dr. (Sir) Lauder Brunton, another Edinburgh man, was elected[5]. His portrait is now in the Great Hall. I came to this hospital in 1904, Gee and Brunton had just retired, but Duckworth did not retire until 1905. I heard many stories about him and knew that they had all been trained elsewhere. The explanation is simple. Between 1861 and 1868 no less than four physicians or assistant physicians had died and one resigned after a severe illness. William Baley[6] was killed in a railway accident in 1861. Henry Jeafferson[7] died of typhus in 1866. William Senhouse Kirkes[8] died of pneumonia and pericarditis in 1864. George Nelson Edwards[9] died of a chronic renal disease in 1868. He had a convulsion while teaching in his ward. While Robert Martin[10] had a severe illness which affected his mind. He resigned in 1867, but he recovered his health. Although he had no appointment, he used to visit the hospital and post-mortem room regularly.

Until 1786 all the physicians had done their medical training elsewhere, but in that year William Austin, who had trained in the hospital, was elected. Between that date and 1865, nineteen physicians were appointed; eleven had trained in the hospital, while eight had not.

I do not believe any extra-mural surgeons were appointed as they had all served as apprentices to a surgeon before being appointed to the staff. I sincerely hope that Dr. Howkins suffered no inconveniences when he started work and that he has long felt well and truly adopted by the hospital.

GEORGE GRAHAM, M.D., F.R.C.P.

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* Reprints received and herewith gratefully acknowledged. Please address this material to the Librarian.

OBITUARY*

Walter Reginald Bett (1903 - 1968)

**Remarks made at a meeting of the Osler Club on June 5th, this year.*

Walter Reginald Bett was born in Riga on April 8th, 1903. He died in Norwich, N.Y., on November 17th, 1968 after a severe myocardial infarction that, had he lived, would have made him a cardiac invalid. Brought up in a country where he was considered an enemy alien, he came to London at the end of the First World War and entered Saint Paul's School. Here too he seemed an alien with his Germanic appearance and the accent which never left him. Small wonder that after formative years when he failed to be assimilated into any group, he became a "character". Yet out of his eccentricity grew his genius.

founder

We remember him tonight as the Founder of the Osler Club of London. I cannot say what converted him to Oslerolatry. Certainly by 1927, when I first encountered him, Harvey Cushing's Life of William Osler had become his Bible and he had made friends with many of Osler's litrary heroes. While a medical student at Saint Bartholomew's he adopted the name of Willie and signed his letters with W.O.'s initials. He did not however, follow his great hero into the Wards with any pleasure, nor, I think, did he share the same kind of love for his fellow men. Heroes he had; at this time, I recall, Sir James Paget and Sir John Bland-Sutton, whose biography he wrote. Later he found a niche in his Valhalla for Dickson Wright. But he set them high standards and was toughly outspoken when they failed to live up to the fantasy that he had woven round them. He could be devastatingly rude and when displeased with a speaker at The Osler Club he was more than ready to vent his pique in the minutes.

He did not take people as he found them. He seemed to me rather to invent a *persona* based on a living being but given a special existence in Walter's mind and a quality, a character which corresponded little with reality. This gave an unexpected twist to his judgements.

career

His Bart's contemporaries, of whom I was one, could not understand why he had ever embarked on a medical career. His own explanation told of a death-bed promise to a dying father. Obediently he qualified. Later he spent some years in residence at the East London Children's Hospital, Shadwell, now part of the Queen Elizabeth Hospital, Hackney. Here he composed some verse which he had privately printed. In 1935 he accepted an invitation, which came through the good offices of John Fulton and Archibald Malloch, to be Medical Librarian at Columbia University. He and Columbia did not make an altogether happy mixture and so in 1937 he joined the first of the half dozen chemical companies with which in succession he came to be associated. He left the Winthrop Company in 1939 for the RAMC in which he served for the duration of the second World War in Ceylon, India and the Dutch East Indies.

After the war, he settled once again in Chiswick becoming a medical consultant for Menley and James and their successors Smith, Kline and French. This allowed him in 1948 to be secretary of The Osler Club, which had resumed active life in the previous year and

of which he had been Foreign Secretary from the foundation in 1928 until his first departure to the United States. During this post war period, a secure job behind him, Bett built up a practice of library research and immersed himself into references and bibliography and the medical journals of the world abstracting and helping many doctors to prepare their papers.

books

Osler held men and books in a balanced reverence. For Bett books rather than men provided his centre of gravity; literature weighed more than life. His writings belong mainly to the species Ephemeridae. John Thornton reports that there are three hundred and seventy two of his reprints in the Saint Bartholomew's Hospital Library and this is but a fraction of what he wrote. It is sad that he did not find the inspiration for a more solid historical work. He did write four books: The Infirmity of Genius, A Short History of Nursing, Osler the Man and the Legend, and The Life of Sir John Bland-Sutton, and he edited The History and Conquest of Common Diseases.

In the Autumn of 1959 he yielded to the tempting offer of a medical editorship from the Eaton Laboratories Division of the Norwich Pharmaceutical Company. In December he left London once again for the United States hugging a Georgian tankard and the DNB which his friends of The Osler Club had presented to him in gratitude for his years of service. Many will remember the dinner at the Royal College of Surgeons given by the Club in honour of Dr. and Mrs. Bett, and many his Oslerian Oration in 1962 entitled The Epitaph of Adrian's Horse. While to some listeners he seemed an enigma orating upon a mystery, his sentences delighted all our ears.

Back in the United States he continued his library work, editing also International Urological News Bulletin until it ceased publication. From March 1961 until December 1966 he was Medical Editor of Pfizer's Spectrum, bringing it to a high standard of excellence. From January 1967 until March 1968 he was Medical Editor of The Physicians News Service, New York.

It was in New York in September 1967 that Bett and I had our last meeting. The scene was The Gate of Cleve, a large German restaurant under the Sheraton Hotel, an excellent place for over-eating. He was clearly an habitué and we made a triumphal progress to his usual table where we were greeted—he with a kiss—by his favourite waitress. He chose the wine and the food with devotion and three hours later we emerged stumbling into the night air of New York. We had lived again our early Oslerian days, and had devoured a memorable banquet and the reputations of our common friends.

Bett spoke and wrote in a style of his own and hard to classify. For those nurtured in classic simplicity his was an acquired taste. Baroque is perhaps the word most apt, and like most things baroque his style can brilliantly succeed. Sometimes it fidgets, sometimes disconcerts, yet it never, never bores. His writings occupy some feet of shelf. There is much literary mining left for the ardent collector of Bettiana.

perfectionist

He claimed always to be shedding illusions. He must have begun life with a large supply to be able to shed so many for so long. Isn't this the mark of the perfectionist who always sees performance—his own and other people's—falling so far short of his high hopes? He had a fancy for Ambassadors and notabilities and invited them to grace our meetings whether he knew them personally or not. If over this he seemed to present a brazen façade, behind it dwelt a sensitive idealist. Talented above most in tongue and pen, he founded and refound our Club*. To provide a century of successful meetings in a single decade he served us without stint and with distinction. In return let his and our Osler Club of London keep ever green the memory of Walter Reginald Bett.

Dr. Alfred White Franklin

* The story of the founding of The Osler Club of London is told in this *Journal* (Franklin, A. W. S.B.H.J., 1961, 65, 53-4).

sport space

TENNIS

This year, with the tennis remarkably uninterrupted by the weather, the club has enjoyed as good a season as it has had for years.

U.H. Cup Competition:
Having disposed of Guy's 6-1 in the opening round, the Middlesex, last year's finalists, put up little opposition in the second, the score being 8 1. The semi-final against King's College Hospital was a much closer match, the final score being 6-3, with John Ussher making a welcome return to the team. The final against St. Thomas's was eventually played at Cobham on hard courts and between showers of rain. They proved a much stronger side on the day, and retained the cup 6-1.

U.L. Cup Competition:
King's College II were beaten 9-0 in the first round, and a weakened Mary's side 8-1 in the second. We surprised a strong King's College I side, complete with two university players, in the semi-final to beat them 5-4. This match was the high point of the season, and unfortunately we could not produce the same standard in the final, losing 5-4 to University College, going down in three sets in the last two matches.

The U.H. League was again played this year, enabling the team to play singles matches. Although we could not always produce a full cup side, the hospital's final position was third. This League is open to anyone, medical or otherwise, male or female, working in the hospital, and if anyone is interested, please contact the captain or the secretary of the tennis club.

After one postponement, the Past v. Present match was played at Chislehurst. The staff side, headed by Messrs. Dowie and Lettin and Dr. Kelsey-Fry, and ably assisted by Chris Garrard, Martin Savage, Marcus Setchell and Gav Danovitch, put up a sterling performance before adjourning to the bar where all were most generously refreshed by the staff. The final score was 6-3.

Finally the club would like to thank Mike Carruthers who, having taken over from Lawrie White this season, has worked so hard to keep the courts in the superlative condition that we find them whenever we play.

C. G. E. HUNT

CRICKET

senior cup

In the Junior Cup Final, St. Thomas's Hospital could not raise a side on the date agreed, so Barts won by a walk over. However, victories over King's and Guy's in previous rounds justified this win.

Sun. July 6th: Bart's Present vs. Bart's Past —

As somewhat of an anti-climax after the final and its good weather, this match was abandoned before lunch, due to rain. Bart's Present had scored 72 for 4, against the 'older' generation.

Eat, July 12th: Bart's vs Incognito at Chislehurst Lost by 75 runs —

Incognito elected to bat on another good wicket, and scored 209 for 6 dec. in two and a half hours. After tea, Bart's started well, Purcell and Firmin putting on 62 for the first wicket in 42 minutes. However, a collapse then occurred and the remaining batsmen could only take the score to 131.

Sun, July 13th: Bart's vs Hampstead at Chislehurst Won by 1 wkt.

This was a very good victory against one of our strongest opposing teams.

Hampstead batted first, and were routed by Berstock, the Bart's captain. He took all three wickets before lunch, and ended up with nine wickets for 34 runs. Hampstead were all out for 110 runs.

The Bart's innings did not start as smoothly as hoped, losing both openers for 2 runs. However, Husband batted well, and held the side together, with a fine 39 not out, aided by Berstock's useful 27. The required total was reached with one wicket to spare; an exciting game and good cricket.

Sun, July 20th: Bart's vs Dartford at Dartford Lost by 4 wkts.

A weaker Bart's side could not do the same the following Sunday. Batting first, Bart's had useful innings from Purcell, 47, and Sloan, 34, but there were few other contributions. Total score 147. Dartford reached this with 4 wickets to spare.

Sat, 26th July: Bart's vs R.N.V.R. at Chislehurst Won by 8 wkts

A weak R.N.V.R. side elected to bat first, and were skittled for 61 runs. Jones and Husband bowled well, taking four wickets apiece.

The opening pair for Bart's were soon dismissed, but then Berstock (26) and Hopkins (27) soon reached the required score.

Sun, 27th July: Bart's vs Wimbledon at Chislehurst Won by 119 runs

Bart's were put in to bat on a fine wicket, and started at a good rate. At lunch 130 runs for 3 wkts were on the board. Purcell being the main contributor. After lunch he continued, and reached his second century of the season. His late cutting and cover driving came to the fore in this innings; he scored 108 in 2½ hours. Bart's declared at 3.30 p.m. with 237 runs for 7 wickets.

With his second ball, Edmondson removed the opening Wimbledon batsman, and from then on, they were struggling. The bowling was tight, and Berstock took 6 of the wickets for 32 runs.

After a very good season, the best for many years, the club goes on its annual August tour to Sussex as this report goes to Press. This promises to be a fine finale.

Congratulations to D. Berstock, D. Edmondson, P. Furness, D. Lindsell and C. Reid on representing the hospital in the United Hospital's team during the last month.

junior cup

The highlight of this season, was reaching the finals of both the senior and junior inter-hospitals cup.

Senior Hospitals Cup Final July 3rd, 4th, 5th. vs St. Mary's Hospital at Teddington.

Winning the toss, Bart's elected to bat first, and were soon in trouble, losing four wickets before lunch, for 77 runs. The slow scoring continued after lunch despite the hard pitch and good weather; St. Mary's bowling the Goodwin twins for 57 overs, of which 24 were maidens. Rowland batted well for Bart's, defying the opposition for 3 hours for his unbeaten 47. The middle order batsmen all scored a few runs, while he acted as "anchor-man" at the other end.

At ten to six, Edmonson, the eleventh man, was dismissed, and Bart's were all out for 204. At the close of play St. Mary's were 21 for no wicket.

The second day was again very hot, and Bart's were in the field. The majority of the bowling was done by Edmondson and Berstock, the latter taking four wickets. At ten to four, Mary's declared at 237 for 9.

Bart's replied, in their second innings, with 81 for 3. Thus at the close of the second day, the match could have gone either way. On the final day, Bart's scored fast, mainly due to good innings from Rowland (30) and Sloan (59). At 2.30, the innings closed at 206, leaving Mary's 174 runs to score in 205 minutes. This they did, with half an hour to spare, despite keen fielding by Bart's; they lost 3 wickets in the process.

So for the second year in succession, Bart's lost in the final, last year to Thomas's and now to Mary's. However, a good run was had to the final, and some good standard cricket played.

SCOREBOARD

Bart's 1st innings		**2nd innings**	
Purcell b P. R. Goodwin	7	ct Williams b Goodwin P. E.	13
Lindsell b Patterson	22	ct Evans b Goodwin P. R.	19
Furness ct Williams b P. E. Goodwin	18	ct Goodwin P. E. b Hollebone	31
Reid ct Pennells b Patterson	6	ct and b Patterson	6
Firman b Patterson	21	ct and b Hollebone	31
Berstock b Pennells		ct Bartlett b Hollebone	5
Rowland NOT OUT	47	ct Isaac b Pennells	30
Sloan ct Hallebone b P. R. Goodwin	32	ct and b Goodwin P. E.	59
Hann l.b.w. Pennells	15	l.b.w. Hollebene	6
Lloyd ct Evans b Pennells	0	ct Pennells b Hollebone	1
Edmondson b P. R. Goodwin	4	NOT OUT	3
Extras	7		2
TOTAL	204		206

Bowling:									
P. R. Goodwin	26	8	51	2	31	6	66	1	
P. E. Goodwin	26	14	29	2	18	10	20	2	
Patterson	22	10	44	3	7	2	34	1	
Pennells	20	7	42	3	6	0	26	1	
Hollebone	8	1	28	0	18	3	58	5	

St. Mary's 1st innings		**2nd innings**	
Bartlett b Lloyd	27	ct Sloan b Berstock	19
Schneerson l.b.w. Lloyd	29		
Goodwin P. E. ct Furness b Lloyd	33	ct Lindsell b Lloyd	34
Evans ct Furness b Berstock	25	NOT OUT	59
Patterson ct Sloan b Berstock	9	NOT OUT	30
Isaac run out	10	b Berstock	34
Goodwin P. R. ct Sloan b Edmonson	31		
Williams l.b.w. Berstock	0		
Bullow ct Firmin b Berstock	22		
Hollebone NOT OUT	38		
Pennells NOT OUT	3		
Extras	9		1
Total for 9 wkts dec.	237	for 3 wkts	177

Bowling:								
Edmonson	24	9	49	1	6	1	31	—
Berstock	22	5	90	4	23	4	70	2
Lloyd	18	5	45	3	21	7	56	1
Howland	10	3	27	—	5	—	19	—
Hann	5	—	18	—				

ANNOUNCEMENTS

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Engagements

BROOKS—GODFREY.—The engagement is announced between Mr. Nicholas Brooks and Miss Wendy Godfrey.

GUTHRIE—OSBORNE.—The engagement is announced between Dr. Trevor Guthrie and Miss Victoria Frances Osborne.

SILVERTON—CHAPMAN.—The engagement is announced between Dr. John Saunders Silverton and Miss Anne-Marie Chapman.

Marriage

LISTER—MARTIN.—The marriage took place on Saturday, June 28 of Dr. Andrew Lister and Miss Sarah Martin at Chelsea old church.

Births

MCLAUGHLIN.—On July 3, to Ann (née Wheldon Williams) and Dr. James McLaughlin, a daughter (Sarah Elizabeth).

SOPER.—On July 8, to Susan (née Bennett) and Dr. Richard Soper, a daughter (Rebecca Jane).

Deaths

CUNNINGHAM.—On May 24, Dr. Frederick Hugh Lester Cunningham, M.C., M.B.B.S., aged 55. Qualified 1914.

HARRISON.—On June 22, Dr. William Robert Eric Harrison, M.R.C.S., L.R.C.P., aged 73. Qualified 1927.

KENNAWAY.—On July 6, Lady Kennaway, aged 86, devoted wife and widow of Sir Ernest Kennaway, F.R.S.

Changes of Address

Dr. E. Colin-Jones' address is now 29 Mallory Road, Hove, Sussex, BN3 6TD.

Group Captain and Mrs. A. Klidjian now live at 16 Beranburh Field, R.A.F. Hospital Wroughton, Swindon, Wilts. Tel. No.: Wroughton 291, ext. 329.

Dr. A. E. Lorenzen's address is now 60 Balmoral Road, Kingsdown, Deal, Kent.

Dr. A. Geoffrey Dawrant's new address is Professional Wing, Meadowlark Park Shopping Centre, 156th Street and 87th Avenue, Edmonton 52, Alberta, Canada.

ERRATUM.....

✻

A junior nurse was told that the patient having rectal fluids was also to have nothing by mouth. She had obviously assimilated this and on taking the tea trolley round . . . calmly poured a cup of tea into the infusion . . . Comment from the patient? "You forgot the two sugars, nurse!"

Another junior nurse had been having a trying time with a somewhat irate consultant. When called upon to chaperone a patient for him she was determined to be a success.

The examination was conducted without a fault. At the end she thought he said . . .

"And now I want to look at the patient's thighs."

Thinking that this was a rather peculiar mode of speech, the nurse prepared the patient for the embarassing gynaecological examination.

The position set, and everything laid out, the consultant appeared. He assessed the situation in a glance, turned to the nurse and said: "Eyes, nurse . . . EYES!"

Saint Bartholomew's Hospital

JOURNAL

contents

Vol. LXXIII No. 10 1st October, 1969

journal staff

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apathy

In this issue of the *Journal* complaints are made about student apathy. But, from our account of American medical education, it would seem that apathy is not an American problem. In America, for example, questionnaires seem to be returned in sufficient quantity for meaningful evaluation of aspects of the curriculum to be made. Perhaps the cure for English apathy is an American length of day? Or if, after three months, a consultant could identify by name all the eleven students on his firm, would this be the start of education becoming more a collaboration and less of a one way information transfer?

reactionary teachers

Teaching staff are now eager to receive comments from students. The barrier to progress in the development of medical education is not reactionary teachers but disinterested students. Teaching committees may become one man's attempt to overthrow a total system. Let Bart's have functional committees which will immediately convey student opinion on matters large and small to receptive authorities, and cause useful changes to be made without delay.

the Journal

Such a system of immediate complaint needs vocal students. Many occasions would arise when the complaint could not be handled by a teaching committee. Only one other vehicle for wide expression exists at Bart's—this *Journal*—till now insufficiently used for complaint and suggestion.

welcome

A warm welcome to those who are new to the Hospital or Medical College. We wish you a profitable, enjoyable, dynamic time at Bart's. May disillusionment, disinterestedness and apathy not spoil your stay!

announcements

Births

GIBBON—On August 14th to Ruth (née Alexander) and Dr. Raymond Gibbon a son (William Peter), brother for Frances, Robert and Rachel.

KNILL-JONES—On April 28th, to Dr. Robin Knill-Jones and Dr. Jennifer (née Sykes) a daughter (Sarah Gabrielle), sister for Alison and Philippa.

Marriage

GALLANT—COX—On August 2nd Dr. Michael Gallant and Miss Rosamund Cox at Hampstead Parish Church.

Deaths

CANE.—On June 8th, Dr. Maurice Hereward Cane, M.R.C.S., L.R.C.P., aged 81. Qualified 1915.

CORFIELD—On August 20th, Dr. Carruthers Corfield, M.R.C.S., L.R.C.P., aged 95. Qualified 1897.

DONKIN—On August 9th, in India, William Donkin, M.B., B.S., M.R.C.S., L.R.C.P., I.M.S. Qualified 1939.

HUNT—On August 19th, in Mallorca, Richard Hunt, M.R.C.S., L.R.C.P., aged 49. Qualified 1944.

JAMES—On August 21st, Ernest T. James M.B., B.Ch., M.R.C.S., L.R.C.P. D.Ph. Qualified 1927.

PRIDHAM-WHIPPEL—On June 13th, Dr. William Pridham-Whippell, M.B.B.S., aged 81. Qualified 1914.

Change of Address

KNOX—Dr. A. J. S. Knox, to 6 Hanning Court Road, Pinhoe, Exeter, Devon.

Appointments

Charing Cross Hospital, Medical School

Professor T. W. Glenister has been appointed to the Chair of Anatomy tenable at Charing Cross Hospital Medical School.

St. Bartholomew's Hospital Medical School

The title of reader in virology has been conferred on Dr. R. B. Heath in respect of his post at St. Bartholomew's Hospital Medical School.

The Cambridge Graduates' Club of St. Bartholomew's Hospital

The 79th Annual Dinner of the Cambridge Graduates' Club of St. Bartholomew's Hospital will be held on Friday, 31st October, 1969, at 7 p.m. for 7.30 p.m. in the Great Hall of the Hospital.

The Honorary Secretary is Dr. T. B. Boulton, Anaesthetic Department, St. Bartholomew's Hospital, London, E.C.1.

letters to the editor

Bart's Teaching

Dear Sir,

It is fruitless merely to coerce lecturers to teach well and students to learn. Humans unlike rats probably respond better to positive than negative reward. Certainly the latter has its place, but it should be reserved for those who seem immune to all forms of encouragement and whose continued presence in the medical or teaching profession constitutes a threat to its standards and a danger to other human beings. The teaching system at Bart's is notably lacking in immediate reward for teachers and pupils alike. It is a rare pupil who needs only the thought of reward three years hence to maintain his industry. It is a rare teacher who is content with verbal praise as his only reward. Based on these considerations I would suggest the following ways of improving the standard of education at Bart's.

(1) More responsibility for the treatment of patients should be undertaken by the students.

(2) More small group teaching. Tutorial groups should be instituted on all firms.

(3) More periods of residence at other hospitals, particularly during the summer months.

(4) Greater flexibility in inter-firm teaching, or shorter periods with each firm.

(5) Consultants and junior staff alike should be paid adequately for their time spent teaching.

(6) If necessary, meaningful sanctions to be taken against students and teachers, in the form of an effective system of referral with increased supervision for students, and a polite request to some teachers to concentrate on non-educational activities.

Yours etc.,
Jolyon Oxley.

A "Footnote" on John Thurtell

Dear Sir,

Many years ago, when the late Sir Arthur Keith was Conservator of the Museum to the Royal College of Surgeons, he demonstrated in one of his Demonstrations in the Theatre of the College, the articulated skeleton of a foot, which, he told us, was "Thurtell's foot".

Yours faithfully,
A. L. MORETON.

Wilton House, 33 High Street, Hungerford, Berks.

MEDICAL EDUCATION in the U.S.A.

by Dana Hershey

Dana Hershey, medical student at the University of Pennsylvania, recently spent a few weeks on the Medical Unit. Here he describes the changes now taking place in American Medical Education, discusses them and comments on what he has seen at Bart's.

As an American medical student and a visitor to Bart's for the past four weeks, I have been asked to write an article concerning the British and American systems of medical education. I hesitate to do so on two accounts, firstly the brevity of my experience at Bart's and my consequent lack of familiarity with the total programme and secondly the turmoil that is now taking place in American medical education which has led to the appearance of a wide variety of programmes in the different schools and to some degree of experimentation and revision within any single programme. Thus it is difficult to characterise American medical education as a single entity, and it should be understood that I have drawn my impressions from my experience of three years —two of them preclinical and one clinical—at the University of Pennsylvania. In the following paragraphs I will deal mainly with medical education as I know it in the States and then make a few comparisons with what I have seen of the programme at Bart's.

When I entered the University of Pennsylvania School of Medicine three years ago, the curriculum had not yet been changed, and the programme involved a rather fixed series of compulsory courses which required four years for completion. The first two years were preclinical with lecture courses in the basic sciences—that is anatomy, biochemistry, physiology, pathology, bacteriology and pharmacology; the last two years were clinical, requiring 12 weeks of surgery, 12 weeks of medicine and 12 weeks for paediatrics, psychiatry, obstetrics and gynaecology in the first clinical year. The second clinical year was essentially a repeat of the first, except that one's grades and recommendations—very important for getting an Internship (Housejob) in a good hospital—had been determined by that time and the external pressure for study was off.

The old curriculum, then, left little room for choice on the part of the student. He had little opportunity to select his courses or his instructors and everyone who gained an MD degree from Penn had almost the same rather broad educational background. By the 1960s however, physicians, faculty and students were intensely examining both the *aims* and *methods* of American medical education.

The *aims* of American medical education were in the past geared to the production of general practitioners and consequently emphasis was placed on a well rounded training programme. The advocates of reform argued that the demands of medical practice had changed, that GPs simply could not keep up with the new developments in each area of medicine, that more specialists would be required and that more doctors were in fact specialising. Accordingly post-graduate Intern programmes were changing from comprehensive "rotating" programmes to "straight" programmes in single medical disciplines such as surgery, medicine or paediatrics. The graduating doctor had to have fairly well formed ideas about what area of medicine he wanted to learn and the old medical school curriculum left him little opportunity to explore his interests.

If the demands of medical practice were changing from general practice to specialisation, then surely the aims of medical education should also change. Prerequisite courses should be abbreviated in order that the student might have elective time to explore his interests and to study several subjects in depth.

The "new curriculum" at the University of Pennsylvania allows for this elective time. The time allotment for the basic sciences has been cut from two years to one, and the extra year remaining may be devoted entirely to such electives as cardiology, endocrinology, renology, research or respiratory intensive care to name only a very few.

While studying the aims of American medical education, critics are also evaluating the *methods*. This question involves the effectiveness of course presentation and the psychology of student motivation. The lecture system, when used sparingly, can be a very useful means of education but unfortunately the system is only as good as its lecturers, and when used to excess is more conducive to somnolence than retention of information. Medical textbooks are generally excellent in organisation, expression and thoroughness and the best a lecturer can do is to present material not available in the books, or to reorganise textbook material into a new and more meaningful form.

The trend in the new curriculum, then, is to cut down on the number of lectures to allow students more time to read on their own, and to set up small discussion groups to handle uncertainties in the reading. For example, the pathology course had scheduled about 10 lectures a week in 1966, but only about 3 in 1967. Our two-month course in medicine now has absolutely no lectures. Each student has aproximately 6-8 patients on his firm at one time and he is expected to know everything about those patients—history, physical findings, laboratory data, diagnosis, aetiology and pathology of the disease, and hospital course. The theory is that one remembers the various facets of a medical problem much better when one has worked through them in practical experience than if one has merely read about them. A written test at the end of the course in medicine makes it necessary for the student to read about all the major disease entities on his own, even though they may not be manifested in his own patients.

Another change in the structure of the curriculum at Penn is earlier exposure to clinical medicine in order to make the basic sciences seem pertinent and important to medical practice at an earlier time. First and second year students will be able to take clinical electives along with their basic science with the idea that the experience in practical medicine will help them integrate and remember their anatomy, biochemistry, physiology, pharmacology, etc.

In addition, students are taking a greater role in the development of medical education at Penn. Students sit on the committee which is formulating and revising the new curriculum, and, after the completion of each course, the students are encouraged to fill out questionnaires, composed by fellow classmen, which ask for evaluations of the course format, lectures, consultants, interns and residents (registrars). This kind of feedback is essential to the evaluation of both the individual courses and the new curriculum as a whole.

The institution of these changes in the medical programme at Penn has not been without problems. The reduction in the time allowed for the requisite basic sciences has forced some courses to be abbreviated to a rather extreme degree. First year students do not now study the limbs in anatomy although they may do this subject as an elective if they so desire. Other departments have responded to the curtailment of their course time essentially by throwing th same amount of material at the students in a much shorter time period, producing a good deal of misery and confusion.

Another problem that has arisen is the difficulty in scheduling popular electives. As word gets round which courses and which instructors are good or bad, students apply for the same favoured courses, with the result that some are unable to take electives they feel are necessary to their medical education.

There is also, of course, the risk that students will limit their electives to fairly narrow areas of medicine and fail to gain adequate background in other important areas. Thus it

would seem that some control of electives might be necessary to avoid this over-specialisation.

Despite these and other difficulties, the new curriculum offers important advantages over the old. It better meets the new demands of medical education both in allowing the student elective time in which to pursue his interests and in providing an opportunity to study subjects in depth. By the time a student has completed his third year of medical school, he has a good idea of where his deficiencies in knowledge lie, and he can improve these with the proper elective choices; the new curriculum offers this flexibility.

Furthermore the new curriculum has done away with several outmoded conceptions of medical education (for example the belief that every student should learn the minutiae of the human anatomy), and it has eliminated some of the academic isolation of the preclinical years. The student is now more a participant in his own education, where before he was so often the passive pupil drowsing while the noise of sequential lectures droned in his ears. Medical education is more a collaboration and interaction between faculty and students than a one-way bestowal of information. And finally, there is sufficient pressure on each department to provide interesting, well-taught electives, since they will be vacant if students do not feel they are worthwhile.

My own medical education has taken place in the three years in which the Medical School has been phasing in the new curriculum. It is with this background that I contrast my experiences during the four weeks I spent with first year clinical students on Professor Scowen's firm.

One striking difference between the American and British systems is that there is a definite element of competition and compulsiveness in the former and little, if any in the latter. In order to obtain an Internship at the better hospitals in the States we must have good grades and recommendations and consequently we are evaluated fairly constantly through the first three years of education with the result that American students experience a great deal more anxiety than British during this time but achieve a higher rate of learning. In contrast the British student seems to have few really important evaluation periods until his qualifying examinations at the end of his fifth year and knowledge is acquired during the first few years more by a process of osmosis than of active transport.

There is also a corresponding difference in what the British and American medical school staff expect of their students. The American student's day generally starts at 8.00 a.m. with firm rounds, and he is expected to be on or near the firm until 5.00 or 6.00 p.m. He has night duty every fourth or fifth night, and if a new patient comes in at midnight, he is expected to clerk the patient that night and be able to present the case orally and coherently on rounds the next day. When he has night duty he generally does not go to bed before the Intern. This type of schedule is a great deal more exhausting than the one I have seen at Bart's, but the extra work load is often most rewarding. We are encouraged to be an integral part of the care of our patients and the personal working contact with the interns, residents and patients can be quite stimulating.

In contrast, the isolation of the British medical student from the active practice of medicine on the firms has a negative influence on his motivation. Whereas our clerking is checked by our residents and placed on the patient's charts, the clerking by Bart's students seems to be ignored. Thus, there is little feedback to the Bart's student about his progress and his incentive to do a thorough job of clerking is generally quite low. He misses the challenge of working *with* the house staff and the stimulus of being involved with real human beings with real medical problems.

Medical education in the clinical years, then, is more informal at Penn than at Bart's. Much of what a Penn student learns is a result of a kind of symbiosis between himself and his interns and residents, whereby he helps them with their daily duties (venepunctures, IV injections, filling out lab. slips etc.) and they discuss with him his patients and review his clerking. The programme at Bart's, however, involves a schedule of formal presentations of one or two diseases, generally with clinical examples, and I must say that I have never experienced better formal teaching sessions than those at Bart's. They have been superbly thorough, relevant and interesting, and the emphasis on physical diagnosis is something that is often lacking in medical education in the States. This thorough, systematic approach to the examination of the patient has been most impressive.

In conclusion, my brief stay at Bart's has been instructive and enjoyable. Both staff and students have been warm and welcoming to me, and I have enjoyed, in particular, the Bart's sense of humour. I only hope that British students going to the States have as great an experience as I have had here.

Stemming the Tide of Apathy

One cannot help cynically observing that, despite the recent striving of students and teachers to talk to and understand each other, there has been remarkably little in the way of educational advance. Either in spite of or indeed because of this intense desire to evaluate and communicate, the relationship between the medical student mass and the teaching body has in fact been deteriorating. Undoubtedly the additional enforced lethargy of the summer period in the Hospital, with its half empty timetable, summer holidays and the newly introduced surgical residencies have aggravated the situation on both sides; but the pall of apathy which hangs over the Hospital this summer really marks a new low in student-teacher relations.

I would like to put forward here at least a partial answer to this disconcerting deterioration. Once having established a new "status quo", it may be possible for the succession of students at this and other medical schools to use this basis of understanding to allow them to construct new teaching techniques and programmes in co-operation with their teachers.

Perhaps a better word than apathy with which to describe the mood of clinical teachers would be disillusionment. One cannot entirely blame Consultants for the exasperation induced by poor student attendances, when they have just been talking to the students' Teaching Committee, which is trying to improve the standard of teaching. It is, however, for the Consultants at this Hospital at least, their job to teach. I feel that they and the rest of the clinical staff who teach the students, who are "Dei gratia" tomorrow's doctors, would find their task easier and more rewarding if they could appreciate the simple point of my thesis.

I do not know if it is a new phenomenon or if it has always been so, but it is obvious to me that there are two distinct groups of Medical students, not relating to any of the established social groups within the College, or even to intelligence brackets.

There is the aggressive group, eager to learn, who become angry and disheartened at the cancelled ward rounds and indifferent teaching occasioned by their teachers' reaction to the other group. This other group regularly fail to attend teaching sessions, and would rather glean a few hasty, uncorrelated facts from the Houseman's notes than perform the trying task of formally clerking a patient. I would appeal to those who teach us to recognize this important difference, as it is only by segregating attitudes that this vicious circle can be broken and a rapport established. The former group must be encouraged and the latter group discouraged if any improvement in our education is to be achieved.

The way to encourage the former group is simply to offer stimulating teaching sessions, and to organize alternative teaching or to clearly publicise cancelled rounds when holidays, illness or a conference make keeping an appointed teaching session impossible. The way to discourage the habits of the latter group clearly involves firmer sanctions against these students. I have no desire to see the traditional freedom of the medical student tampered with, as I firmly believe that it is to this environmental factor that the British, and especially the London medical student owes a great deal of his finesse and ease as a medical practitioner. However I believe just as firmly that our ability to provide society with competent doctors must be seriously compromised if the present situation persists. Such sanctions as statutorily exist are seldom invoked and as such have become standing jokes among the students. The threat of referral because of poor attendance on a firm, for instance, has so often proved an empty one that it is no longed heeded. I realize that the College is obliged to produce a certain number of students each year, but it is only by using tougher measures that many students reaching finals can truly be said to have completed the course.

In essence then, I would ask teachers, like actors, to play to their audiences, though they may be small at times. The students who have come, come because they wish to learn, and if the students' respect is returned by the consultants then each will find his own reward in the ideal student-teacher relationship. As for the malingerers, it is they who should be whipped, not the good and faithful servants.

George Lodge

THE 1969 HORDER MEMORIAL LECTURE

Given by PROFESSOR E. B. ADAMS

of the University of Natal, Durban, S. Africa

Bacterial Endocarditis and other infections in an African setting

It was with surprise and considerable pleasure, about a year ago, that I received an invitation from Sir Max Rosenheim, on behalf of the Trustees, to be the Horder Travelling Fellow for 1969; and it is indeed a privilege and a great honour for me to be asked to come to this famous hospital to deliver a lecture to commemorate one of its greatest sons.

I am going to start by using some of Lord Horder's own words, taken from the introduction to his Lumleian Lectures on Endocarditis in 1926 (Horder, 1926), by saying that the "gratification which I feel is tempered by humility" when I realize how unlikely it is that my contribution will be worthy of so distinguished a clinician and teacher. But if I may again borrow his words and interpolate some of my own to explain my purpose, "I shall be satisfied if a few of my audience, generously disposed, may feel helped by reconsideration of old facts" viewed against an African medical background quite unlike yours at St. Bartholomew's Hospital "to discriminate between what we do and what we do not know."

An examination of Lord Horder's medical writings from 1894 to 1955 reveals immediately how catholic his interests were and how prolific his pen. It also shows the dominant part infection played in the clinical practice of his day—which should not surprise us if we remind ourselves that when he retired from the staff of this hospital in 1936 the antibiotic era had not yet begun. Although he ranged widely, some subjects obviously attracted his attention more than others—and he left behind for our benefit a legacy of writings especially rich in papers on pyogenic and other infections, pyrexia of obscure aetiology and bacterial endocarditis. If he were in my hospital today I believe Lord Horder would have been fascinated to see in such profusion the old diseases about which he wrote, and to be able, as we are to tackle them with effective new tools which he was denied. And I am sure we would be all the better for his incisive mind, his wealth of knowledge and his great clinical acumen.

Let me tell you something about my hospital, its students and its patients, before I draw on my own clinical experience and record some observations on infections of various kinds and bacterial endocarditis in particular.

For reasons which would be inappropriate for me to discuss in this lecture, our undergraduate students are almost entirely African and Indian—but my house physicians, my registrars and senior staff are of all sorts, in the South African context, and the multiracial society which we form is a delight to work in, despite its problems; it is also an enriching experience.

The Medical School is adequately built and well-equipped for teaching and research, but like so many African hospitals, the teaching hospital next-door, which bears the name of King Edward VIII, suffers under the burden of an enormous patient-load with which it is

ill-fitted to deal. Durban is a bustling city much in the American idiom. It was founded by British people, however, and still shows strong ties with this country, links of an enduring nature in language, education and morality, which political change cannot eradicate. The city is multi-racial, almost equal in its content of Africans, Indians and white people; and it is indeed the meeting place in Africa of the West and the East.

The influx of rural Africans to the cities leads to the development of shanty towns, with their grave social and health problems: but vast housing schemes have been built or are under way to relieve the evils of slum-dwelling and I am glad to say they are succeeding. People from the shanties and the housing estates, all of them African or Indian, are the poorest in the community and are poorly served by general practitioners. For almost all their medical needs they come to our hospital in their thousands—many with trivial complaints, but many more with gross disease, much of it infective, often left unattended for longer than should have been.

In the Britain of today, the transformation of which to the welfare state I have been most interested to see at regular intervals since I was at Oxford just after the war, the hospital conditions we know in Durban have largely disappeared; but they must have been familiar to Lord Horder when he was a student in the 1890s, and later in his progression at St. Bartholomew's Hospital from house-physician in 1897 to physician in 1921. And I have therefore thought it appropriate in this memorial lecture to remind you of one or two facets of infective diseases with which he must have been familiar in London by relating some of my experiences at King Edward VIII Hospital, Durban. I shall also, however, introduce other diseases with which you are probably unfamiliar, and which Lord Horder, I believe, would have found novel and fascinating.

Although I am the first to agree with the criticism that hospital statistics may be misleading, I have collected some figures of admissions to my own ward, which handles about a fifth of the intake of medical patients in the hospital. In a period of 14 years we have dealt with 18,000 patients. Their case records have been summarised by my registrars, discussed at staff meetings, and filed in punch-card form in my department—and on these I have drawn to give you an idea of our clinical practice.

Intestinal infections

As can be seen in table 1, the biggest group consists of the intestinal infections—5,000 patients. Let me point out straight away that the preponderance of amoebiasis—caused by infestation with the parasite *Entamoeba histolytica*—is in part due to the research interests of my two colleagues A. J. Wilmot and S. J. Powell.

In particular, I want to mention two diseases from this list—typhoid fever and amoebic liver abscess. One in every hundred of the patients in my ward has typhoid. Apart from the rarity of rose spots, Lord Horder would have had no difficulty whatever in recognising typhoid on clinical grounds among Africans. Because of its gravity we often start treatment with chloramphenicol before blood cultures are available—and seldom regret the decision. Blood culture, of course, is the cornerstone of diagnosis. It is far more valuable than the Widal reaction; and I would agree with Lord Horder when he said in 1926, that "agglutination tests supplied relative data to which undue weight should not be attached."

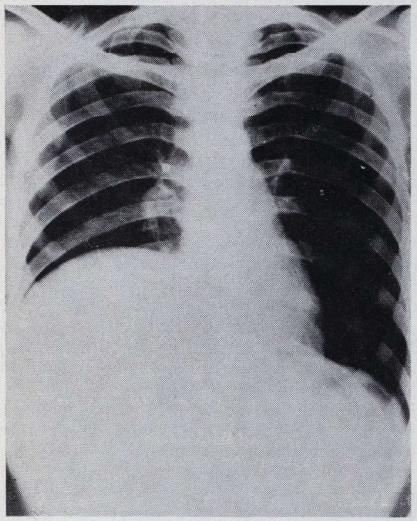
Lord Horder would, however, have been puzzled by amoebic liver abscess, one of our commonest diseases. The patients are usually African and almost always male. The onset is generally insidious and symptoms may have been present for weeks or months before the patient decides that it is time he came to hospital. Dysentery is only present in 10% of cases. Pyrexia, hepatic pain, an enlarged tender liver and intercostal tenderness are the usual symptoms and signs and suggest the diagnosis; and X-ray may show a suspiciously high diaphragm (see figure 1).

If a point of maximum tenderness can be found over the liver or between the ribs the diagnosis becomes highly likely. Aspiration with a wide-bored needle will then generally yield typical pus, stained, in most cases, with blood—and while I deplore the use of similes in medicine which involve thoughts about food like "apple-jelly" granulations and "prune-juice" sputum, I must admit that the "anchovy-sauce" description which is usually applied to this material is very apt.

TABLE 1
Intestinal Infections

		% of 18,000 admissions
Amoebic dysentery	2,800	16
Amoebic liver abscess	1,500	8
Bacillary dysentery	305	2
Typhoid fever	175	1
Gastroenteritis	152	1
Tuberculous peritonitis	88	0.5

Figure 1
Amoebic liver abscess. Note height and contour of diaphragm



Entamoeba histolytica can be identified in the pus in most cases and the gel diffusion test, developed in Durban in the Amoebiasis Research Unit is positive in 99% of cases (Powell *et al.*, 1965). The point about this test is that it is most unwise to diagnose hepatic amoebiasis if it is negative.

We know that amoebic liver abscess will respond very well to treatment with emetine, dehydroemetine or metronidazole—probably the best of the three—and that aspiration is only indicated when there is clear clinical evidence of a localised collection of pus. Let me add immediately that we regard blind needling as a most unwise procedure. We also know that these abscesses may rupture into lung, pleura, pericardium, peritoneal cavity or the abdominal viscera and of all these complications, amoebic pericarditis is the most dangerous. Skin involvement or distant metastases are also possible. What we do not know about this disease is why there should be so heavy a preponderance of males over females—15 to 1, and whether the parasite in the bowel, its portal of entry, invades the tissues because of some circumstance in the host-parasite relationship other than the pure weight of infection; and these problems are exercising my colleagues.

Cardiovascular diseases

Next to the intestinal infections in frequency come cardiovascular diseases, of which we have seen about 3,000 examples, with a distribution of the commoner ones as shown in table 2.

Top of the list, as a group, are the rheumatic disorders, rheumatic fever, chronic rheumatic endocarditis and bacterial endocarditis (about which I shall have more to say later). This group makes up the biggest section of our cardiological practice.

Not all these patients, of course, are admitted with congestive cardiac failure. The commonest single entity is cardiomyopathy, which is the most important single cause of congestive cardiac failure in my wards. Cardiomyopathy is an African disease—indeed all but 12 of the 529 cases listed here were African. There is no evidence that the disease is infective. Some Johannesburg workers believed it to be a collagen disease but the basis for this view is tenuous. There is in fact very little definite evidence about aetiology. Recently, however, J. V. O. Reid, our professor of physiology, has done two rather elegant studies in Durban—on dietary supplements in the case of women with twin pregnancies (Reid, 1969), and on rats fed the staple maize diet of the African (Reid, 1966); and these suggest dietary deficiency of tryptophan as the cause.

Having just pointed out an overwhelmingly African disease, let me comment in passing on myocardial infarction. This is a relatively insignificant condition among Africans (see table 3). Almost all our cases are Indian, with an actual ratio of 1 African to approximately 6 Indians but a true ratio of 1 to 26 if the preponderance of African admissions (4 : 1) is taken into account. Here one is tempted to speculate that if diet plays a big part in the myocardial infarction of Western man, the African's different dietary habits may well protect him from this disease; but perhaps these same dietary customs lead to the replacement of coronary artery disease by cardiomyopathy as a major cause of cardiac disability.

Despite the rarity of myocardial infarction in the African, hypertensive heart disease,

TABLE 2
Cardiovascular Diseases

Cardiomyopathy	529
Chronic rheumatic endocarditis (520) Bacterial endocarditis Rheumatic fever	877
Hypertensive heart disease	302
Syphilitic aortic incompetence	218
Myocardial infarction	143
Cor pulmonale	141
Pericarditis (T.B., amoebic)	113

malignant hypertension and strokes are by no means uncommon, as table 3 shows.

Tuberculous and amoebic pericarditis among the African are also commoner than myocardial infarction. These two infective diseases are the most important causes of pericarditis leading to congestive cardiac failure in my wards. They may be difficult to distinguish from one another unless there is clear evidence of hepatic amoebiasis (which leads to pericardial involvement by direct spread through the diaphragm); here the gel diffusion test may be helpful, since amoebiasis is most unlikely if it is negative.

There were 8 deaths among the 25 cases of amoebic pericarditis studied by my colleagues (MacLeod, 1966) and this is a considerable improvement on the results in other series. Their success, I believe, was due to awareness of this complication of hepatic amoebiasis and to early recognition of the dangers of cardiac tamponade, one of the 2 modes of death. The other great danger of amoebic pericarditis is constriction—but it is quicker in onset than that of tuberculous pericarditis and it is reversible without surgery. If the diagnosis is missed, death is almost invariable. If recognised and treated by appropriate tissue amoebicides—such as metronidazole or dehydroemetine—and also by aspiration of the pericardium or the liver abscess which caused it, or both, the results are good.

Tuberculous pericarditis, which is commoner than the amoebic variety, is not infrequently confused with cardiomyopathy by the general physician. Both come on insidiously, both lead to heart failure for which no cause is immediately apparent, both give radiological pictures of large silent hearts, and both may produce rather similar electrocardiographic changes, consisting of low voltage and flattened T-waves. A pleural effusion only helps if it is large enough to be aspirated, a high protein content suggesting infection. In differentiating tuberculous pericarditis from cardiomyopathy, a raised sedimentation rate and pyrexia favour tuberculous pericarditis; pulsus paradoxus adds strong weight to this diagnosis and a rub is conclusive.

We know that in time, without appropriate treatment, tuberculous pericarditis leads to constriction. What we do not know is how many patients treated properly with streptomycin, isonicotinic acid hydrazide and P.A.S. can be saved from this fate. I believe that the proportion is quite high, but in our hospital environment in which follow-up is unsatisfactory, an extensive field study would have to be done to give the answer.

My colleague Mervyn Gotsman, professor of cardiology in Durban, uses cine-angiography to distinguish between cardiomyopathy and tuberculous pericarditis when there is difficulty. The dye only occupies a small part of the cardiac outline in tuberculous pericarditis. He has also shown the usefulness of catheter studies: left and right atrial pressures are much the same in constrictive pericarditis as compared with cardiomyopathy in which the left atrial pressure is raised. The pulmonary artery pressure is also much higher in cardiomyopathy.

Chest diseases

I included amoebic pericarditis in this lecture because of its novelty to you and its importance to us, but it is a much less common entity than tuberculous pericarditis. Indeed, tuberculosis in almost all its forms is a very common disease. Our inpatient hospital statistics underestimate its incidence because many patients are recognised in the outpatient department and sent off to the chest clinic or to a tuberculosis hospital.

Nevertheless, as seen in table 4, my own unit has handled a large number of cases of pulmonary tuberculosis and tuberculous pleural effusion, comparable in frequency with lobar pneumonia. I am not going to dwell on tuberculosis, other than to say that we constantly

TABLE 3
Relative Incidence of Myocardial Infarction and other Cardiovascular Diseases among Africans and Indians

	Total	African	Indian	Corrected ratio*
Myocardial infarction	143	19	124	1.26
Hypertensive heart disease	302	204	98	
Malignant hypertension	104	61	43	
Cerebrovascular accidents	488	324	164	
Pericarditis (T.B., amoebic)	113	110	3	9.1

*African: Indian admissions 4:1

TABLE 4
Some Common Chest Infections

Pulmonary tuberculosis	(630)	
Tuberculous pleural effusion	(201)	831
Lobar pneumonia		813
Pyogenic lung abscess		145
Chronic bronchitis and emphysema		114

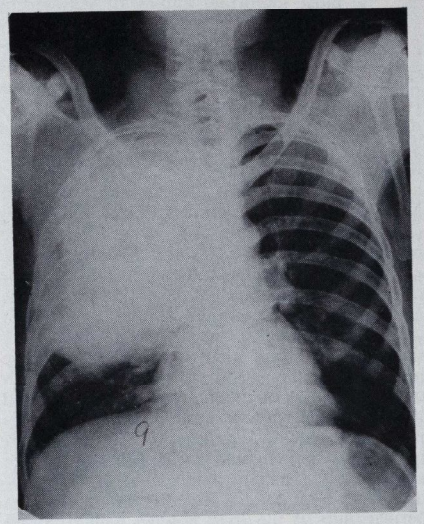
have to bear it in mind since it is a common cause of chest disease, meningitis, peritonitis, paraplegia, and pyrexia of obscure aetiology.

Cavitating pulmonary tuberculosis and pyogenic lung abscess are sometimes confused, but the more localised nature of lung abscess, the absence of tubercle bacilli in the sputum and the good results which follow treatment with penicillin and postural drainage readily distinguishes it from tuberculosis in most cases. Unlike your experience here in Britain where, I believe, almost all cases of lung abscess have a much graver origin, being the result of bronchial carcinoma, among Africans in Durban the majority by far follow extensive dental caries (so common in these patients) or alcoholic debauchery and inhalation of vomitus. Conservative treatment gives good results, with a death rate of only 3%.

Pneumococcal lobar pneumonia is a disease which is always with us, and my students never lack cases on which to learn and to practise the art of eliciting physical signs. In my unit my colleagues and I have seen over 800 patients with this disease in 14 years. Often they come in rather late, but the disease is otherwise typical and responds extremely well to treatment with penicillin, generally with defervescence in 24 hours. The case fatality rate is low—3%—and what a difference this is from Lord Horder's time! I do not have any of his own figures, but at the turn of the century his colleague, Sir William Osler, gave the death rate as 30% in a series of over 600 cases at John Hopkins Hospital (Osler *et al.*, 1938).

I have learned the hard way, however, not to be complacent about lobar pneumonia but to suspect any deviation from the usual clinical and radiological picture of the disease because of the risks involved in missing Friedlander's pneumonia. If the patient is overwhelmingly ill on admission; if his sputum is mucoid, tenacious and red rather than rusty; if the chest X-ray, as in figure 2, shows bulging of a fissure; and if response to penicillin is not a satisfactory one in 24 hours—my suspicions are aroused. The presence of *Klebsiella pneumoniae* in the sputum does not necessarily mean that you are dealing with Klebsiella pneumonia, but a heavy growth is suggestive. Your patient and you can ill afford to await proof in so serious a condition, and I add streptomycin, chloramphenicol or tetracycline whenever I am suspicious of the diagnosis. Nevertheless the case fatality rate in Friedlander's pneumonia is still very high. It was 30% among our 24 patients, the usual figure given, but this is certainly much better than the 80% which has been recorded when the disease is unrecognised or treated too late (Beeson, McDermott, 1967).

Figure 2
Klebsiella pneumonia. Note the bulging lower border.



The anaemia of infection

I should like to digress from further consideration of the common infections we encounter to comment on the anaemia which so often accompanies them. Although usually moderate in degree, the anaemia may at times be severe and warrant immediate treatment with blood transfusion, so it is perhaps appropriate for me to say something about its recognition.

The first point worth making is that the anaemia of infection is by no means always normochromic. Indeed as often as not it is hypochromic. Lord Horder noted this in 1926 in relation to endocarditis when he said that "the reduction in haemoglobin seems to be invariably greater than the reduction in red cells". In recent years however, there has been an unfortunate tendency to use the terms hypochromic anaemia and iron-deficiency anaemia as synonymous—which of course they are not.

A few years ago we were impressed by the frequency with which the hospital laboratory reported hypochromic anaemia in patients with chronic infection. We thought this a surprising finding in a hospital population composed largely of Africans, in whom, in South Africa,

TABLE 5
Iron Content of Bone Marrow and Liver

	Bone Marrow (μg. Fe/g)		Liver (total tissue Fe., g.)	
	No.		No.	
Africans	180	345	200	2.09
males		390		2.33
females		155		1.12
Indians	59	85	37	0.36
Europeans	19	122	16	0.47

(Mayet, 1964)

body iron stores are so frequently excessive. Table 5 demonstrates this point. Iron overload in the African appears to be due to his dietary habits and his taste in beverages—brewed in such a way that the iron-content is high. As a result iron-deficiency anaemia among Africans is a comparative rarity.

To investigate this apparent anomaly we studied the haematological features of a random group of 65 African patients admitted with various long-standing infections such as pulmonary tuberculosis, amoebic liver abscess, tuberculous peritonitis, pyogenic lung abscess and bacterial endocarditis; and for comparison we used 51 subjects in apparent good health (Mayet and Powell, 1964; Adams and Mayet, 1966).

We took particular care with the haemoglobin and packed cell volume estimations, doing them all in duplicate. We also investigated the plasma-iron pattern, and when there was anaemia, we examined the bone marrow for cell-morphology and iron content.

I have summarised the results of this study in table 6 which shows, among other things, the percentage of cases with hypochromia, by which I mean an m.c. h.c. of 30% or less. It is seen that anaemia is not only common but may be severe; in general, I might add, severity is related to the duration of the infection—and as a corollary, I would remind you that acute infections such as lobar pneumonia are usually not accompanied by much anaemia.

Then there are two other important points. The first of these is that hypochromia is a frequent finding. 60% of our patients had mean corpuscular haemoglobin concentrations of 30% or less, sometimes as low as 24%. The second point is that haemosiderin granules can invariably be demonstrated in marrow smears in the anaemia of infection, and their presence distinguishes this cause of hypochromia from true iron-deficiency, in which, of course, anaemia only ocurs when the body's stores are depleted of iron.

Examination of the bone marrow after staining with potassium ferrocyanide is the quickest test and quite the best for differentiating these two causes of hypochromic anaemia among patients such as those at King Edward VIII Hospital where chronic infection is common.

While it is true that the two tests which make up the plasma-iron pattern—the plasma-iron itself and the total iron binding capacity—are also useful, they take much longer to do and are not so reliable. The plasma iron is usually low in the anaemia of infection as it is in iron-deficiency. But in infection the total-iron-binding capacity also falls, so that the normal percentage saturation of the iron-binding protein transferrin is preserved—at about 25% in our cases. In contrast, in iron-deficiency anaemia the total iron-binding capacity rises, giving saturation percentages below 16 and often under 10. But sometimes the plasma-iron pattern in the anaemia of infection is similar to that of iron-deficiency anaemia and in this case only marrow examination at the time or subsequent response to treatment with iron will settle the question about the kind of hypochromic anaemia with which you are dealing.

With regard to the research project I have just described, haematologists might well argue that we should have considered two other causes of hypochromic anaemia besides iron-deficiency and chronic infection. We excluded one of these, sideroblastic anaemia (which is a rather rare disease), on the grounds that the plasma iron was low and not high as it should be in sideroblastic anaemia; and also that ring sideroblasts were not seen in a sample of 20 cases taken at random. We felt justified in ruling out the other well-known cause of hypochromia, thalassaemia, because it is so uncommon in South Africa—in fact I have seen very few cases in Durban in 14 years.

To summarise these remarks, what we know about the anaemia of chronic infection is that it is frequently hypochromic and may be confused with iron-deficiency; that it is related to the duration of infection and therefore in long standing cases may be severe; and that examination of the marrow for stainable iron readily distinguishes it from true iron-deficiency anaemia. In some way the incorporation of iron into haemoglobin is interfered with but what we do not know is how this takes place.

TABLE 6
The Anaemia of Chronic Infection

	Hb (g./100 ml.)	m.c.h.c. (%)	% with hypochromia
65 patients	10.0 (4.1 – 15.2)	29.9 (24 – 34.5)	60*
51 controls	15.0 (12.4 – 16.8)	33.3 (30 – 36)	2

* Marrow iron present in all 65 cases

Bacterial Endocarditis

Lord Horder wrote 12 papers on bacterial endocarditis and no other subject seems to have attracted more of his interest if one is to judge from his publications. I found three of his papers of particular interest; his comprehensive review of 150 cases of infective endocarditis which appeared in the Quarterly Journal of Medicine (Horder, 1909); his Lumleian Lectures of 1926 to which I have already referred; and the 9th Oslerian Oration delivered to the Osler Club (Horder, 1936). It was because he seemed to be so interested in the disease that I decided to comment on bacterial endocarditis in this memorial lecture.

Twelve years ago my colleagues and I began to suspect that we were missing the diagnosis because we followed the current practice which depended on a positive blood culture or at least demanded the presence of very strong clinical indications.

It is true that we often *suspected* the disease because a patient with chronic rheumatic endocarditis or a congenital heart lesion had some of the following features:—pyrexia (which was often low-grade), anaemia, splenomegaly, clubbing, and microscopic haematuria or some other embolic phenomenon. Yet we were loath to make a firm diagnosis which committed us to *treat* the patient with penicillin in high dosage for many weeks if we failed to obtain the infecting organism or had not observed most of the well-known clinical signs. Moreover we were usually unwilling to accept the validity of a blood culture which yielded an organism besides *Strep. viridans*, regarding most other organisms as probable contaminants.

On several occasions, however, we had the unhappy experience of finding necropsy evidence of bacterial endocarditis without having treated the patient because convincing proof of the diagnosis was lacking. We began to realise that our views were too rigid. We therefore adopted the more liberal practice of treating patients in whom bacterial endocarditis seemed a possible diagnosis, even if the criteria were tenuous; and we did this after we had taken three specimens of blood for culture without waiting to see if they were positive or negative. To put it bluntly, we did not waste time trying to make up our minds. What we had not appreciated was that we were witnessing the changing face of bacterial endocarditis, about which much has been written lately.

Some idea of incidence of this disease among Africans and Indians in Durban may be gained from a review of my own experience over a twelve year period. I examined the case records of 124 patients admitted consecutively to my ward, each of them diagnosed at some time as suffering from bacterial endocarditis. I rejected 24 of this total because in retrospect there seemed to have been insufficient evidence. In the remaining 100 cases the diagnosis was supported by blood cultures or proved at necropsy (42 cases); or appeared to be highly probable although blood cultures were negative (58 cases). During the same 12-year period, 14,000 patients came into the unit, giving an incidence of one case of bacterial endocarditis to every 140 admissions. Perhaps a better idea of frequency is obtained by comparing this total of 100 cases with other diseases usually considered to be fairly common. Bacterial endocarditis proved to be a fifth as common as lobar pneumonia and chronic rheumatic endocarditis; its incidence was the same as that of typhoid; and it was twice that of leukaemia.

I also obtained a measure of its incidence by looking at necropsy records for the whole hospital. Over a four-year period 9,000 necropsies were performed. Overall cardiac deaths accounted for 1,055; rheumatic heart disease for 188; syphilitic heart disease for 37; and there were 40 cases of bacterial endocarditis. This makes it just under a quarter as common as rheumatic heart disease as a cause of death.

Post-mortem examinations were performed on 16 of this series of 100 cases. Several are worth mentioning, either because they illustrate how minimal the clinical evidence of bacterial endocarditis may be, or because they show how easy it is to miss the diagnosis.

Twelve of the 16 cases had been correctly diagnosed during life, although blood cultures were positive in only three. One of this necropsy group was a patient with congestive cardiac failure due to aortic incompetence; the only clues to the correct diagnosis were slight pyrexia and a raised sedimentation rate (which of course should have been low because of congestive cardiac failure). Another patient, who had been admitted with dysentery, was found in addition to have aortic valve disease, anaemia and a raised sedimentation rate. Bacterial endocarditis was suspected but not treated. The diagnosis became almost certain with the rapid development of congestive cardiac failure due to a ruptured cusp, but it was now too late and death occurred before treatment could be started.

In four of the 16 patients the correct diagnosis had not been reached before death. One patient had congestive cardiac failure, atrial fibrillation, hemiplegia, a raised sedimentation rate, anaemia and meningitis. The second presented with what appeared to be acute nephritis, but he also had splenomegaly and anaemia. The third patient had a systolic murmur and anaemia without any other evidence of bacterial endocarditis. The fourth, who was admitted with amoebiasis, was found to be in congestive cardiac failure, apparently due to aortic incompetence; the only clinical clue to the correct diagnosis was clubbing.

It will be noted that blood cultures were positive in only three out of 12 necropsy cases diagnosed during life. In the clinical material under review, the blood culture was positive in 26 out of 100 cases, virtually the same proportion as in the necropsy material. We were, of course, worried by this low culture rate, although we invariably did at least three blood cultures. We therefore ran a parallel series on lobar pneumonia as a check and recovered the pneumococci in 50%. What surprised us at the time was how few of the positive cultures in bacterial endocarditis yielded *Strep. viridans* (see table 7), and what a bizarre collection of other organisms we found. There was generally no proof that these other organisms were causative; like ourselves at the time, many would have been most suspicious that they were contaminants. For comparison, however, in this table I show the broad distribution of organisms given recently in a Boston series.

TABLE 7
Organisms in 26 cases of Bacterial Endocarditis

	No.	%	*%
Str. viridans	5	20	27
Other streptococci	3	12	29
Staphylococci	10	38	23
S. typhi	4	15	1
Others	4	15	20

*Results from 86 cases of Lerner and Weinstein (1966)

Against this background of my own experience as a general physician who has no research interest in the disease, it seems worthwhile to examine the picture of bacterial endocarditis seen elsewhere at the present time by the experts, and to compare it with observations made by others in the past. There are several differences.

As regards the infecting organisms, a comparison of four large series published in the last 20 years shows quite clearly how *Strep. viridans* is losing its place as the commonest causative organism (see table 8). Note the progressive drop from 87% to 27%. What is not shown in this table is the wide range of organisms, bacterial, fungal and rickettsial, which once again come into the reckoning; "once again", because the range was a large one in Lord Horder's papers.

Another point which emerges from a review of the literature is that the high proportion of positive cultures in the earlier series, such as the 92% of Cates and Christie (1951) has not been the rule in recent years. Hickie (1961) gave the figure as 57%, Hampton and Harrison (1967) reported it as 59%, and others have recorded even lower percentages. This drop is probably the result of the widespread use of broad-spectrum antibiotics before the patient's admission to hospital, but diagnostic criteria used in different series may well invalidate comparisons.

Evidence that the pattern of bacterial endocarditis is changing also comes from an examination of its age distribution (see table 9). It used to be a disease of the young. 76% of Lord Horder's patients were under 40 years of age—and only 3% of Christie's cases were over 50. Recently, however, the proportion of older patients has risen considerably—partly, no doubt, due to a changing age distribution in the general population; but there are other reasons. Note that just under half the patients of Hughes and Gauld were over 50.

The pattern has also altered with regard to the underlying lesions (see table 10). When Cates and Christie reviewed their cases, the overwhelming majority suffered from acquired heart disease, almost always chronic rheumatic endocarditis. More recently bacterial endocarditis has been found in an appreciable

TABLE 8
Organisms in 4 series of Bacterial Endocarditis

	No. of cases	Str. viridans (%)	Other Strep. (%)	Staph. (%)	Others (%)
Cates & Christie (1951)	408	87	7		6
Toh & Ball (1960)	49	57	16	10	17
Hughes & Gauld (1966)	68	45	14	12	29
Lerner & Weinstein (1966)	86	27	29	23	21

TABLE 9
Age Distribution

Horder (1909)	(150)	range	3 – 63 years
		76%	under 40 years
		8%	over 50 years
Cates & Christie (1951)	(442)	range	7 – 74 years
		3%	over 50 years
Pankey (1961)	(167)	range	1 – 77 years
		25%	over 50 years
Hughes & Gauld (1966)	(68)	range	12 – 87 years
		46%	over 50 years

TABLE 10
Underlying Lesion in Bacterial Endocarditis

Cates & Christie (1951)	87%	acquired heart disease
	13%	congenital heart disease
Pankey (1961)	57%	acquired heart disease
	21%	congenital heart disease
	22%	no underlying valvular lesion*
Hughes & Gauld (1966)	57%	acquired heart disease
	7%	congenital heart disease
	36%	no underlying valvular lesion*

*occasionally atheroma

TABLE 11
Commonest Clinical Features (after Pankey, 1961)

Murmurs	99%
Fever	95%
Raised E.S.R.	94%
Cardiomegaly	75%
Petechiae	70%
Anaemia	64%

Note: Clubbing, splenomegaly, haematuria, splinter haemorrhages occur less frequently.

number of patients with no underlying lesion, particularly in the older age groups. Despite earlier teaching bacterial endocarditis occasionally follows syphilis of the aortic valve, as it did in two of my cases, proved at necropsy. Moreover, as the British Medical Journal (1967) put it a few years ago, "cardiac surgery has brought iatrogenic endocarditis, usually a staphylococcal infection introduced during operation".

There seems little doubt that the pattern of bacterial endocarditis has changed over the past 20 years. The underlying lesions are less frequently rheumatic, although rheumatic endocarditis still has pride of place; older patients especially may suffer from bacterial endocarditis with no evidence of previous valvular disease; a variety of organisms besides *Str. viridans* have now become important; and blood cultures are negative in a higher proportion of patients. Although the factors responsible for these changes are unknown, it is likely that prominent among them are increasing awareness of bacterial endocarditis as a hazard in patients with chronic rheumatic lesions; the prophylactic measures which we take in such cases; the relative sensitivity of *Str. viridans* to penicillin when compared with other organisms known to cause the disease; and the widespread use of broad-spectrum antibiotics for febrile illness of obscure aetiology: all of these favour the emergence of organisms which are less sensitive than *Str. viridans* to penicillin. The low proportion of positive blood cultures in our series is probably largely due to the indiscriminate use of antibiotics before patients come to hospital. But we do not deny that the use of pencillin when patients are first seen in the outpatient department or poor technique in blood sampling are other factors which should be considered in a retrospective analysis of this sort.

What is quite clear from my own experience, backed by the evidence of others, is that many cases will be missed during life if the criteria for diagnosis are made too strict. Although desirable, a positive blood culture is certainly not a *sine qua non*, nor must the practising doctor insist on the presence of most of the clinical signs of the disease before he starts treatment. Diagnosis in medicine is usually made on grounds of probability; and when the consequences to the patient are likely to be grave if we fail to offer appropriate treatment, as they certainly will be in bacterial endocarditis, we cannot afford to withhold treatment even when the probability of the diagnosis being correct is not very high. I believe that when you have any doubt, your patient should be treated for bacterial endocarditis soon after blood has been taken for culture.

Lord Horder said that "the signs of infective endocarditis are three: (a) the presence of valvular disease, as determined by examination of the heart, (b) the occurrence of embolism, and (c) the discovery of micro-organisms in the blood-stream. If these three signs concur in any case the diagnosis of infective endocarditis may confidently be made; the concurrence of any two of these signs makes the existence of the disease highly probable" (Horder, 1909). In his day the physician resisted making the diagnosis because the prognosis was so hopeless: it was worse, in fact, than the prognosis of acute leukaemia is today, because in leukaemia we now have encouraging signs of progress towards a cure. Until many years after he retired from this hospital, Lord Horder had to accept the certainty of death in bacterial endocarditis; yet he never gave up trying to find a remedy. He used, among other things, immunotherapy, diathermy, X-irradiation and radium—but he knew, he said, "of no specific method of treatment that promises, even remotely, any hope of success" (Horder, 1936).

Today we have the means of successfully treating bacterial endocarditis. Making the diagnosis, however tentatively, therefore carries hope and not despair. Accordingly, I believe we should spread our diagnostic net. Table 11 shows the commonest features of bacterial endocarditis. Points particularly worth noting are that murmurs, fever and a raised sedimentation rate are of the utmost importance; and we must remember that some of the time-honoured signs such as clubbing, splenomegaly, haematuria and splinter haemorrhages are rather less important than we once believed.

It is not my purpose to discuss treatment in detail, but several points are worth making. The first is that penicillin in high doses must be given, and for a long time. I would recommend in the first instance 10 million units of penicillin G daily by intra-muscular injection for at least 4 weeks and preferably for 6. The majority of patients will respond with disappearance of fever within a week. If not, much higher doses should be used, preferably intravenously, if necessary increasing the dose to 60 million units. When *Str. faecalis* is the organism streptomycin should be added in a dose of 1 G daily. Finally, for suspected cases —those with a reasonably high index of suspicion on the clinical evidence but a negative blood culture—penicillin should be used from an early stage, for delay while you are trying to confirm the diagnosis will not help the patient although the correct diagnosis may reveal itself in time; and if the temperature does not drop, streptomycin should be added.

In summary, bacterial endocarditis should be suspected in any patient irrespective of age in whom there are cardiac murmurs and unexplained fever. Suspicion should increase if there is a raised sedimentation rate, particularly in the presence of congestive cardiac failure, which normally depresses it. These three signs must be regarded as the major features of the disease.

In making the diagnosis, one must not insist on finding all or even some of the other time-honoured signs—anaemia, petechiae, splenomegaly, clubbing, haematuria and splinter haemorrhages. They do not occur as frequently as we used to think. They certainly help in the diagnosis, but bacterial endocarditis is quite often present without them.

There are two additional features which alert one to make the correct diagnosis: the occurrence of embolic phenomena in young people, and sudden deterioration in patients with valvular lesions or congenital heart disease.

A review of recent literature shows two changes in the pattern of bacterial endocarditis which should be noted. The first is that the disease occurs more frequently in older patients than previously, sometimes when the heart is thought to be otherwise normal. And the second change is that although *Str. viridans* is still the commonest organism, staphylococci and *Str. faecalis* are incriminated in an increasing proportion of patients; and a wide range of other organisms, bacterial, fungal and rickettsial, must also be kept in mind.

May I conclude this tribute to Lord Horder, whom you remember as an outstanding physician and teacher in London in the first half of this century, by drawing your attention to the profound influence his type of clinical medicine has had on the developing countries in the English-speaking world? I believe that the modest success of South African medicine is due in large measure to the firm basis of British medicine on which it was founded.

May I also thank the Trustees for asking me to come here as Horder Travelling Fellow for 1969?

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THE SECRETS OF LADY AUDLEY, MELODRAMA AND THE ENGLISH TOY THEATRE

by
PAUL SWAIN

Photographs by
Steve Warrington

During the extremely successful Barbecue Ball of 1969, the Drama Society of the Hospital was allowed and encouraged by the Wine Committee to perform a short Victorian melodrama, *Lady Audley's Secret*. This play is a classic in a despised genre and commands respect for its sheer and extraordinary concentration of melodramatic incident; its confrontations, discoveries, turning of tables, villains turning to good in death, sentiments, all ending inexorably in the madness and death of the villainess.

A climax among many such comes as the dying Luke, never do well blackmailer, is brought on after being rescued from the fire that Lady Audley raised in a tavern to murder her accuser Robert and her Blackmailer. Just before he is brought on she cries "And who is to be my accuser?", and her rhetorical question is of course answered, as are all the rhetorical questions of villains in melodrama.

LUKE: I thank Heaven! I am spared to do an act of justice before I end my guilty life. I accuse that woman of—

ROBERT: No! hold, hold. It will be better not to cast a stain upon my uncle's name. Say nothing, I beg, I entreat of you.

LUKE: Then I will be silent, silent for ever—ever—ever. (Falls back in the arms of the PEASANTS).

LADY AUDLEY: (aside). He is dead and I shall triumph over them all. (The great bell of the Castle is heard tolling). Enter ALICIA from back followed by SERVANTS.

ALICIA: Robert! Robert! my father is dead. Oh, pity me! pity and protect me! (Goes to Robert).

ROBERT: Sir Michael dead! Now vengeance, take thy own! Friends, hear me—I accuse that woman of the murder of my friend, George Talboys.

LADY AUDLEY: How and where?

LUKE (revives): I-I will tell that. She pushed him down that well, (points to well, all start), but it will be useless to search there now, for George Talboys is—

Enter GEORGE TALBOYS, R.2E.

GEORGE: Here! (LUKE falls back dead).

OMNES: Alive!

LADY AUDLEY (petrified): Alive! Alive! you alive!

GEORGE: Back, woman! and thank that man (points to LUKE). that you have not my death upon your soul. You will be scorned, loathed, and despised by all. The blow you struck me rendered me an invalid for months. I have been silent until today, because I gave my word to that poor, dying wretch. (Points to LUKE). But now I am free—free to tell all. Speak to her, speak to her, Robert, and say I forgive her. (Points to LADY AUDLEY).

ROBERT (to LADY AUDLEY): You hear, woman!

LADY AUDLEY (vacantly): But I do not heed. I have a rich husband. They told me he was dead—but no, they lied—see—see, he stands there! Your arm—your arm, Sir Michael We will leave this place—we will travel. Never heed what the world says—I have no husband but you—none—none! It is time to depart, the carriage is waiting, Come—come—come!

GEORGE: What does she mean, Robert?

ROBERT: Mean! Do you not see that she is mad?

OMNES: (retreating from her). Mad!

LADY AUDLEY: Aye—aye! (Laughs wildly). Mad, mad, that is the word. I feel it

here—here! (Places her hands on her temples). Do not touch me—do not come near me—let me claim your silence—your pity and let the grave, the cold grave, close over Lady Audley and her Secret. (Falls—dies—Music—tableau of sympathy—GEORGE TALBOYS kneels over her).

CURTAIN

Music against the action as a means of heightening the sentimental effect is an important adjunct to melodrama. Indeed it accounts for the derivation of the word melodrama, which meant originally a stage play with music and later came to its present meaning of a sensational play with a happy ending. (Melos-Greek for song, as in melody).

Briefly Lady Audley's Secret is this. When her husband, George Talboys wastes his small fortune and has to leave her for India, duty and more cash, she takes the step of ensnaring rich kind old Sir Michael into a marriage only she knows to be bigamous, having taken the precaution of informing George Talboys that she is dead When George reappears unexpectedly and confronts her she prudently pushes him down a convenient well. unaware that she is observed by the drunken gamekeeper Luke.

The origins of the piece are interesting and typical of many melodramas. Mary Elizabeth Braddon at the age of 25 achieved immediate success with her first published novel *Lady Audley's Secret* which appeared in *The Sixpenny Magazine* in 1862. There were two immediate adaptations of this play appearing on the London stage the next year and the better of these was by C. H. Hazlewood. Originally an actor, he was for many years resident dramatist at the Britannia Theatre, Hoxton, London. In Allardyce Nicoll's authoritative *Handlist of Plays Produced Between* 1850 *and* 1900 Hazlewood is credited with 118 plays or adaptations. *Lady Audley's Secret* is the work of an experienced dramatic hack, economical and confident in the speedy manipulation of incident.

These are the qualities expected of a competent melodrama writer; but amongst the writers who worked in the genre were some who transcended the medium. The French Irishman Dionysius Lardner Boucicault, in his brilliant play *The Colleen Bawn,* combines a more delicate sentimentality with a more complex and impressive motivation than is usual in the medium and has some marvellous stage Irish. Edward Bulwer-Lytton, who later became Colonial Secretary and then a baron, shows in his plays a lightness and wit in his manipulation of plot reminiscent of similar achievements in the best of Restoration Comedy. There are a few impressive writers whose reputation is underrated by our attitude of amused contempt for melodrama and things Victorian.

The fire at the Inn (lit by Lady Audley to consign her accusers to perdition).

The Theatre was built for the Barbecue Ball. The Author stands in the well down which Lady Audley pushed her first husband. Stage construction was by Justin Blake-James and assistants; the several scenes and the proscenium front was finely painted by Susan Lee.

English Toy Theatre

For our production of Lady Audley's Secret we built a small but elaborate theatre. This theatre was a mock up or enlargement of a traditional design, that of the English Toy Theatre, or the Benjamin Pollock Toy Theatre since Benjamin Pollock Toy Theatres and Toy Museum is the only surviving firm of the industry of Toy Theatre makers that was once a flourishing part of the printing trade. They still make their toy theatres and sell sheets of scenes and characters—their best theatre comes beautifully wrapped and amusingly has a Design Centre award label on it. Such a toy theatre was amongst the most devastatingly influential Christmas presents I ever received.

In size the theatres vary a little, but imagine a cube fourteen inches in each of the three dimensions and that would be an average size. It is the facade of these theatres that turns these toys for me and others who have had one almost into an archetypal image instantly recognisable when seen. The shape of the recessed proscenium arch and the forward bowed orchestral pit is shown off by elaborate decoration of the high rococo of proscenium art with its flutings, corinthian columns, centrepieces, unicorns, clouds and theatrical masks gaudily flared out in scarlet and gold. This elaborate decoration is simply a cut out coloured print glued on a frame.

Behind this proscenium there is a simple

grid structure which supports the scenic back drops and the wings which were such an essential part of the design of scenery in the nineteenth century. These scenes and also the characters are again prints which are cut out and glued to card; Benjamin Pollock's delicately dated instructions read: "When you stick the sheets onto cardboard use a thin rigid card (like visiting card) for the characters and a thicker card that won't curl for the scenery." The characters are cut out in their stylised postures, mounted on wire slides, pushed on the stage and the show starts. Pollock explains, "It is a good idea to move gently whichever figure is supposed to be speaking, with a slight shaking of the slide, as this helps the audience to follow the story".

The history of the English Toy Theatre is that of a sudden spectacular flowering in the late Regency and early Victorian period. Between 1815 and 1840 the finest and most elaborate prints and sets of characters were published for this medium. Then there followed a long period of consolidation, imitation and cheapening, making them more available to the children for whom they were intended. The industry dwindled after the Victorian era.

For its origins we must look first to the emergence and popularity of the genre picture which achieved apotheosis in the printed scenes of Hogarth, particularly in his dramatic sequences of prints telling a story—such as his *Rakes Progress or Marriage à la Mode*. At the time of the first outflowering of the Toy Theatre scenes and characters, Hogarth's once revolutionary naturalism had given way to the expressionism of Gillray and Rowlandson, whose fantastic vituperative caricature of skinny clergymen and grossly podgy John Bulls put their characters into dramatic mock heroic poses, arms thrown wide, fists in the air—the posturings of melodrama. And the prints are frequently coloured; coloured by hand. At the same time theatrical prints were being produced, gaudier and more brightly theatrical than any of the delicate portraits of actors in the eighteenth century. The highly coloured profile of Mrs. Siddons and John Kemble in

Two Toy Theatres. The larger shows a tableau from "Blackbeard," in which the pirate is flung to his doom by a British Naval Officer; while the smaller shows a scene from Skelts "Othello."

melodramatic gesture are forerunners of a flood of theatrical prints—they appear in Sheridan's adaptation of *Pizzaro* and bear the text: "Hold!— Pizzaro—hear me! if not always JUSTLY, at least act always GREATLY." The step from this to the production of print sets of characters and scenery, which could be placed in a toy theatre faced with a reproduction of the proscenium at Drury Lane, is not a large one; and soon special books of the play, often suitably shortened, began to appear. In West's *Original Juvenile Drama*, the early plays are headed "written and adapted to West's Theatre, Scenery and Characters, with Songs, Duets, etc. adapted to popular Airs." Piracy was a frequent occurrence in this fringe of the printing industry; and just such a bold pirate was West, who even published on his sheets of *The Black Prince*—"Done without the permission of Mr. W. Barrymore."!

Plays for Toy Theatre

What were the plays published for the toy theatre? Robert Louis Stevenson, whose *Treasure Island* is appropriately published by Penguin as a toy theatre production, wrote a sentimental eulogy of the Toy Theatre entitled *A Penny Plain and Twopence Coloured* and gives his own list. "I have, at different times, possessed *Aladdin; The Red Rover, The Blind Boy, The Old Oak Chest, The Wood Daemon, Jack Sheppard, The Miller and his Men, Der Freischutz, The Smuggler, The Forest of Bondy, Robin Hood, The Waterman, Richard I., My Poll and my Partner Joe, The Inchcape Bell* (imperfect), and *Three Fingered Jack, The Terror of Jamaica*; and I have assisted others in the illumination of *The Maid of the Inn* and *The Battle of Waterloo*."

These plays fall easily into the categories of melodrama which were popular in the first half of the nineteenth century. The Gothic melodramas are of two types the earlier was a castle—dungeon—ghost variety, a form whose archetype is found in the great Gothic novel by Horace Walpole *The Castle of Otranto*. The dreadful tyrant and his crumbling castle, the terrified heroine and her perilous flight, the ominous forest and its colourful robbers, the spectre and the raging storm are the essential ingredients.

The other later sort of Gothic melodrama is the bandit—forest—cottage type. This form includes the most famous of all Gothic melodramas, Isaac Pocock's *The Miller and his Men* (1813). This was by far the most successful production for the toy theatre trade, it appears most constantly in publishers' lists, Stevenson refers to it more than once. The conflict is between Grindoff, a prosperous miller, (really the bandit Wolf, with all his men disguised as robbers) and Lothair, a virtuous young peasant who are in competition for the hand of Claudine, the daughter of the aged Kelmar who lives in his little forest cottage. It is worth quoting the tremendous climax of this piece.

LOTHAIR: And this my triumph! (music).
LOTHAIR throws aside the cloak and his natural complexion is seen; he throws himself before CLAUDINE and receives WOLF'S attack; the robber is wounded and staggers back, sounds his bugle, and the mill is crowded with banditti. LOTHAIR, having caught CLAUDINE IN HIS ARMS (and previously thrown back the bridge upon his release from WOLF), hurries across it, and as he is on it cries, "Now Ravina, now, fire the train!" RAVINA instantly sets fire to the fuse, the flash of which is seen to run down the side of the rock into the gulley under the bridge from which she has ascended, and the explosion immediately takes place. KELMAR, rushing forwards, catches CLAUDINE in his arms, and the whole form a group as the curtain descends.

Similar to the Gothic melodramas are the even more romantic Eastern Plays. *Aladdin and the Forty Thieves* and several of the piratical plays, for instance *Bluebeard* and *Blackbeard*, include colourful Turkish scimitar-swinging figures. These plays relied more heavily on spectacle and highly imaginative setting and hence were obvious candidates for transfer to the toy theatre.

The other popular types of melodrama were the Military and Nautical melodrama. *The Battle of Waterloo* finds Wellington telling his staff that "where danger is, there will I be found. Now for Victory and England! (exit in an immense fire)." Jerrold's *Black-Eyed Susan* was amongst the most successful of the nautical melodramas and was constantly reproduced for the toy theatre. The hero William returns from three years at sea to seek his Susan whom he

hadn't married much more than three years ago. He finds her importuned by a lustful smuggler whom he promptly knocks down: shortly afterwards he commits the capital offence of striking his own captain (without knowing who it is), who is also trying to seduce Susan. At a sensational court martial the admiral who has to sentence him shakes his hand, and after an emotional farewell to a distressed Susan, William is led to his death. Only to be snatched from it on the most far-fetched of pretexts. The play features a curious convention of nautical stage speech—I quote William's response to Susan's Black Eyes. "Oh, Susan, when I look at your eyes you put me in mind of a frigate, with marines firing from the tops," or his reproach to the villain—"Aren't you a neat gorgon of an uncle now, to cut the painter of a pretty pinnace like this, and send her drifting down the tide of poverty, without ballast, provisions, or compass?"

Some of the Shakespearean Toy theatre plays have an extraordinary literary pedigree. Green's *Richard the Third*, for example, was based not on Shakespeare's but on the prevalent adaptation by Colley Cibber first published in 1700. Cibber was an extraordinary flamboyant literary hack who is immortalized as one of the galaxy of villains in Pope's *Dunciad*. He was not above "discovering" Shakespearian plays—his Shakespearean distortion *Fratricide Punished* was broadcast on the B.B.C. as a comic curiosity recently. Cibber presented a simpler and more melodramatic portrait—but perhaps scores by his filling in of more of the historical background. Sir Laurence Oliver re-introduced portions of it into his splendidly overacted film version. The famous lines "Off with his head. So much for Buckingham," and "Richard's himself again" were Cibber's own. Green, in his version for the toy theatre, drastically reduced the play—though he cut only one character and preserved all of Cibber's scenes. His method of cutting was simple—in general he takes the opening two lines and the final two lines of a scene and as little as possible in between . . . The famous soliloquy "Now is the winter of our discontent . . ." which occupies 41 lines in Shakespeare's version takes 37 in Cibber's, while Green gives it only six.

Etchings and Plates

The prints for Juvenile Drama were almost all etchings. Etchings have been done on copper-plates since the perfection of this printing process in Germany in the early part of the 16th Century. An acid-resisting ground (a combination of wax, mastic and asphatum) is laid on the polished metal plate. It is applied either with a roller or a dabber on a warm plate, or poured on in liquid form. The artist draws with an etching needle through the ground, thus creating his designs in lines of exposed metal. Some of the more experienced printers who wanted a quick finished product, would cut down the time spent on this stage by making a very soft acid-resisting ground and placing an original drawing or print that they were perhaps pirating on this ground then could run a hard pencil over the lines of the drawing, thus indenting the ground. The print that they would make was therefore a mirror image of the original; examples of these abound throughout the history of printing but the toy theatre prints have perhaps more than their share. The plate is then immersed in an acid bath (nitric or other mordants), producing incised lines. Etching is a labour-saving technique compared with the fellow technique engraving, where the hard work of pushing with the palm of the hand a tool called a graver or burin produces a sharply pointed cut in the copper.

The great freedom of the etching technique is that one can actually draw on the plate, the lines moving freely in any direction. In an engraving, variation in width or depth of line is achieved by pushing the burin in deeply or shallowly. In etching it is obtained in a different way—by repeated bitings of the plate. After a first faint biting, the artist covers over with an acid resisting "stopping out" varnish, all the lines he wishes to remain light or delicate. The longer the plate remains in the mordant bath the deeper and wider the exposed lines become. In this way a great variety of line can be achieved. If you ever have to distinguish the two in a given print, examine a line with a magnifying glass and if pointed ends to that line are clearly visible, it is an engraving while if they are blunted, the line is etched.

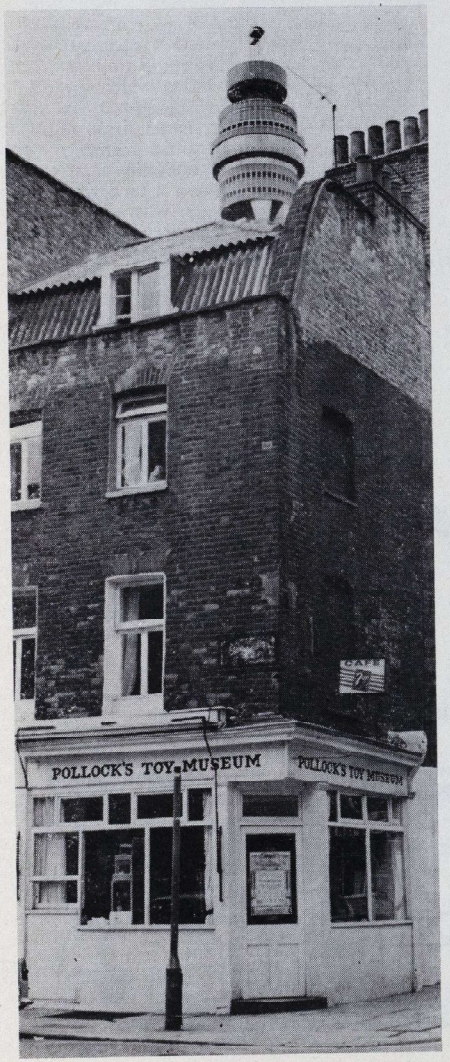
These etchings for the toy theatre run the range from the brilliantly executed, extravagantly imaginative prints to be seen in the British Museum and the Victoria and Albert, to the coarse prints which turn up in Portobello Road with the original colouring on them scarcely dry. But even the coarseness can be charming—these wooden heroines who have become ugly with constant reproduction in press attain a certain comic sadness.

One scene, once produced, might be suitable for another play. Thus one particularly non-descript scene by Skelt is suitable for *Children in the Wood, Forest of Bondy, Rookwood or Turpin's Ride to York, Richard the Third* and *The Life of a Soldier*; while some military wings are suitable for both *The Battle of Waterloo* and *Richard the Third*, a jump of some four centuries.

These sheets were produced for ½d. plain or 1d. coloured, sometimes 1d. plain, 2d. coloured. Now it takes me a good half an hour to complete a single sheet using a restricted palette of say six colours. In professional hands only three primary colours were used to produce the complete range of hand painted colour—they were carmine, gamboge and a light blue. The typical brilliant crimson was produced by applying deep pink over strong yellow. Many were elaborately painted with up to seventeen distinct colours and an incredible amount of brushwork—all for an extra halfpenny or penny. But the fascination of the colouring book was a selling point for these sheets. Stevenson writes: "I cannot deny that joy attended the illumination; nor can I quite forgive that child who, wilfully foregoing pleasure, stoops to "twopence coloured." With crimson lake (hark to the sound of it—crimson lake!—the horns of elf-land are not richer on the ear)—with crimson lake and Prussian blue a certain purple is to be compounded which for cloaks especially, Titian could not equal. The latter colour with gamboge, a hated name although an exquisite pigment, supplied a green of such a savory greenness that today my heart regrets it. Nor can I recall without a tender weakness the very aspect of the water where I dipped my brush. Yes, there was pleasure in the painting."

Green's Characters in RICHARD THE THIRD, 1851

The Pollock Toy Theatre Museum in Scala Street.



Survivor of an Industry

Glancing at a list of the addresses of the printers it becomes clear that toy theatre was a printing industry that flourished all round Barts. Although many small printing firms still work in our catchment area there is no longer the same extraordinary concentration of such firms in the area as existed before the bombing in the last war. Hodgson and Co. at 10 Cloth Fair, West Smithfield; Bernard Hodgson and Co., 5 Cloth Fair, West Smithfield; W. Webb Cloth Fair, West Smithfield; H. J. Webb, 146-121 Old Street; D. Straker, 21 Aldersgate Street; R. March and Co., 18 St. James Walk, Clerkenwell; A. Park, 6 Old Street; W. Love, 81 Bunhill Row near Finsbury Square; W. Harrison, 27 Long Alley, Finsbury; S. Fairburn, 44 Barbican; Dyer, 109 Aldersgate Street; W. Clerke, 265 High Holborn. This is the roll-call of a local industry now sadly dead.

Benjamin Pollock Ltd. is the sole survivor of this pretty tradition. As well as theatres they produce a series of plays at five bob each—in four languages—with scenes and characters for *Aladdin, Harlequinade, Blackbeard, Babes in the Wood* and *Cinderella* for instance. Recently they have produced a set of Skelt's *Characters and Scenes in Othello* in 8 scenes, 8 plates of characters with a Book adapted to the Above —also Green's *Richard the Third* of Kean's famous performance as Richard. They have a shop at 44 Monmouth Street. Puffin, the offshoot of Penguin, have a good *Treasure Island* for toy theatre, but sadly their *High Toby* which I had as a child and remember as one of the best—it was a production of admirable originality in style for the medium—is no longer in print. There is also a curious photographic set of sheets of Olivier's Film of *Hamlet* which I find disconcertingly realistic.

Happily Pollocks appears to be reviving. They have opened a new branch near Tottenham Court Road which includes a Toy Museum with many of their old theatres, different sorts of Victoriana and other curios. They do you quite a reasonable cup of tea and sell gingerbread men—each cost ninepence. There are also for sale some amusing pop-up cut out books and magic shells which if you pop in a tumbler of water sprout flowers and a stars and stripes flag (well, they were made in Japan) and make excellent emergency presents for nephews and nieces, etc., who always seem to have their birthdays yesterday.

music at Bart's

As a fairly regular attender at musical functions at Bart's, I feel that the generalization that amateur music-making can produce both highly rewarding and excruciatingly painful moments for performers and audience alike, was largely upheld over the past year. Perhaps the most appealing feature of the musical ethos here is the amazing diversity of activities, ranging from the grand occasion of Carols at the Royal Festival Hall, in which the Bart's choir participated, to the piquant intimacy of flute and clarinet duets at musical evenings in College Hall played to an audience of six. It is inevitable that the performance of the Fauré Requiem by a choir of one hundred and thirty with an imported orchestra and professional soloists should be a more totally pleasurable event in absolute terms, than the first utterances of an amateur orchestra struggling out of the darkness of dissolution; but where else can the Bach Brandenburg Concertos and Beethoven Symphonies be heard interpreted afresh by conductor and players alike?

Although there are many talented musicians at Bart's, it would be absurd to pretend that there are not sizeable deficiencies in their technique. But this is a minor consideration compared to the relative paucity of certain instrumentalists and singers, notably string players and tenors. Thus the influx of new players is vital not only to encourage the existing ones to remedy these deficiencies, but also to extend the repertoire which such an amateur choir and orchestra might hope to encompass. Moreover the inexperienced musician need not fear the humiliation of auditions, as there are none. Many a crime is committed in the back row of the sopranos and at the rear desk of the second violins, and even if noticed is liable to be regarded as a sign of artistic "joie de vivre".

Jolyon Oxley

Editor's Note: Last year there was only *one* desk of second violins and I was in it. Next year I hope that there will be several desks so that I can go back to committing my musical crimes in the back desk!

drama at Bart's

Presenting plays at Bart's is a task of formidable proportions. Unlike most University audiences, the Bart's audience is predictable for its dislike of the esoteric and its predelection for the melodramatic and the salacious. Thus is would be courting disaster to indulge our academic whim and stage King Lear on Ice or Medea in the original Greek, although I have no doubt Lysistrata would be an enormous success. Therefore it is necessary to search for material which is at one and the same time aesthetically and financially rewarding.

Having at last discovered this rare work, the Society is then faced by the eternal difficulties of staging productions at Bart's. Although this issue was elegantly side-stepped on the last occasion by building an entire stage, with proscenium arch, revolving scenery and trap door, the problem remains of either making-do with the rather inadequate facilities of Gloucester Hall, or spending almost one and a half times the Society's annual grant on the hire of the Cripplegate Theatre. Clearly it is a great advantage to present plays in a well equipped theatre, but it is felt by some members to constitute too great a restriction on the Society's activities at other times of the year.

Despite these considerations, the Society expanded its activities considerably last year by the addition of a summer production and took responsibility for one of the Ward Shows at Christmas. Plans are already in hand to improve the stage in Gloucester Hall by the addition of a moveable apron stage, on which it is hoped to produce a new play some time in November which was written originally for television. This production will also be entered for the NUS Drama Festival. Ideas for the rest of the year are as yet unformed—the Ward Show remains to be written, and February's production to be planned—perhaps a combined production with the Music Society or what about a play for marionettes? The possibilities are endless, but they will only be realized by the continuing endeavour of established members of the Society and a wave of uninhibited enthusiasm from new members, who will be welcomed whatever their skill and experience. A drama society consists not only of actors and directors, but also of designers, painters, carpenters, electricians, musicians and publicists. The Society needs all these.

Although the new academic year means new members for the Society, it also means the loss of Benita Wylie, who has appeared in most of the Society's productions since 1963. Having both acted with her and directed her, I can only say that her gaiety and consummate skill as an actress will be sorely missed.

Jolyon Oxley

Miss Benita Wylie relaxes after the exhaustions of her demanding melodramatic role as Lady Audley; with her, in characteristic pose, is the play's producer,

Jolyon Oxley

Sports News

BOAT CLUB REPORT 1969

1969 was not a vintage year for the boat club; it was disappointing, after boating the unprecedented number of four eights during the winter, that so many talented and much-needed oarsmen should forsake the river just as the weather became milder.

For various reasons very little rowing took place during the Easter term, and for the first time for several years we were not represented in any of the head of the river races.

However, in May a 1st VIII was assembled and spent a very enjoyable week in training at Marlow, under Joe Bailey. This crew, though keen, never went very fast: but it was very unlucky to be bumped in the UHRC races, due to a broken slide. We are now lying fourth in this event, which means we are still in a position to recapture next year the headship which we held in 1966 and 1967.

The 2nd VIII rowed very well, and would easily have retained the 2nd VIII's pennant had they not been forced to row an inexperienced substitute on the second night, when they were bumped. This trophy can also be regained next year.

Since it was obvious that there was little future for us in eights, the crews broke up into small boats, and for the rest of the season we boated a Junior-Senior, Junior, and Novice four. These two last, whose personnel were fairly interchangeable, showed some promise, but were unsuccessful purely due to their lack of fitness. It must be emphasised again that the days when one could win by skill and determination alone are now past, and a vigorous programme of fitness training in both the boat and the Gym is a prerequisite for success.

The Junior-Senior IV trained six days a week from May onwards, with heavy weight-training twice a week. At our first two regattas we lost in the first round; but at Windsor and Eton we won the J-S IV's event, beating London R.C. (winners at the Vesta Dashes the day before), by 1½ lengths in the final, having disposed of Henley R.C. in a heat. At this regatta we lost our boat (run down by an umpire's launch!) and our defeats at Reading and Chiswick are largely attributable to the very unsuitable boat we were forced to borrow.

After this we changed our steersman, and were unfortunate in drawing Eton in the first round at Marlow. Although we pushed them harder than anyone else, they won the event easily, and went on to win the Visitor's Cup at Henley.

We then moved up to Henley, where owing to getting stale (we were there nearly 3 weeks) and over-eating at the Old White Hart, we became, if anything, slower; this despite the coaching of Barry Grimaldi, who certainly improved our style and cohesiveness. But we lost to Pembroke in the eliminators, and on reflection I think we deserved it.

This marked the end of serious rowing, though a pair rowed with varying success at several post-Henley regattas. Tim O'Carroll and Barry Grimaldi were selected to represent the university at the BUSF championships, and won the bronze medal in double sculls.

Our thanks are due to Chris Hudson, Joe Bailey, Nick Boyd, and Barry Grimaldi, for spending their valuable time coaching us.

1969 successes: 1st VIII won May & Baker trophy (v. Guys I); 2nd VIII won World Medicine Shield (v. Guys II); 1st IV won J-S fours at Windsor and Eton regatta; Double scull won bronze medal, BUSF championships.

N. J. C. Snell

SAILING CLUB REPORT

REGATTA 1969

This year's regatta was held on Wednesday, July 9th at the Welsh Harp, when a fairly good entry of 12 boats enjoyed a day's sailing marred by only the diabolical weather conditions which prevailed for most of the day.

The racing started at midday with the first race for the Commodore's Cup. This turned out to be the pleasantest sail of the day with a fresh breeze blowing down the Harp although the skies were overcast. John Shaw set off to a good start, and led to the first mark, closely followed by Mike Williams, John Durham, Roger Chapman and Mark Rowntree. Shaw held and increased his lead for most of the first lap, but unfortunately a misunderstanding had arisen over the course, and he rounded the wrong mark at the end of the first lap and subsequently had to retire. Chapman broke his tiller on the reach and also retired, whilst Williams sailed on round to win the race with Rowntree in second place and Durham third.

An increase in the strength of the wind provided some fast planing for boats awaiting the start of the second race for the Single-Handed Trophy. Bruce Noble capsized but managed to right himself and bail out in time for the start of the race. Before the start, however, every drop of wind departed during a very heavy rain-storm and the pattern for the rest of the day was set, with a flat calm interrupted only by periods of heavy rain. Shaw was again away first and was followed to the first mark by Madsen, Chapman and Williams. The following beat and reach taxed every helmsmans' light weather ability to the utmost, as the little wind present was continually shifting in direction. Shaw maintained his lead and won the Single-Handed Trophy; Chapman came second and Williams third.

The Ladies enjoyed bright sunshine for their race, which was sailed after a lunch break, and also provided us with some of the closest racing of the day; so close in fact that the *Racing Rules* and the *International Regulations for Preventing Collisions at Sea* might never have existed.

Favourite for the event was the previous year's winner, Pippa March; and despite being severely handicapped by a bad crew, she managed to retain her trophy after a close fight with Liz Elder. Bad crewing by the male members seemed to be the order of the day in this event, many of them throwing buckets of water around instead of concentrating on the race.

The second race of the three for the Commodore's Cup was sailed in the calmest conditions of the day, with only the faintest of zephyrs to send the boats forwards. Shaw, Williams and Chapman found the quickest way up the first run, when it suddenly changed to a beat. At the same time, the rest of the fleet, still on a run, caught a puff of wind and rapidly closed up with the leaders. Chaos followed on the next reach and round the mark, this being largely started by Chapman tacking on top of Shaw and Williams and he retired for this infringement. Williams was able to clear himself of the confusion and sailed home to win the race with Shaw second.

Williams' two firsts meant that the trophy was his whatever the outcome of the third race, and as there was still no wind and a further storm had drenched everyone just as they were beginning to dry out, the third race was abandoned.

Our thanks are due to the various Colleges which lent us boats for the day; to Jennie Glanville and Laurrie Dougan for starting all the races for us; and to the people who went into Wembley to buy food when it was obvious that we could not make the "Blackbirds" by closing time.

About twenty people attended the first Sailing Club Dinner anyone can remember at "The Rutland" the same evening, where we were pleased to welcome as Guest of Honour our Commodore, Professor Quilliam, who kindly presented the prizes.

SAILING CLUB (contd.)

Bart's v Thames Estuary Yacht Club. Saturday, 2nd August.

An effort has been made by the Sailing Club to arrange team racing at the "home" clubs of its own members. The aim of this is to improve the standard of Bart's sailing by the use of different classes of boats at different venues.

As the first of these meetings, M. Williams arranged for a race in National Twelve Foot Dinghies at the Thames Estuary Y.C. in Southend. As a result of a slight misunderstanding The Bart's team arrived with enough time to spare to admire the Pier and the "Kiss-me-quick" hats before having a pint at the club.

The Bart's team consisted of H. Tubbs, J. Shaw, and R. Chapman crewed by Miss D. Robinson, J. Winner and a local Cadet member. M. Williams and his wife sailed for the home team.

The first race was sailed in a pleasant light breeze starting with a beat along the shore helped by the tide. At the weather mark the Bart's team were trailing at the end of the fleet, and so the position remained at the end of the first lap. Beating along the shore for the second time R. Chapman moved into third place making good use of the changing wind shifts. On the final reach for the line both he and H. Tubbs made a further place each. Final places were 1, Williams T.E.Y.C.; 2, Chapman; 3, T.E.Y.C.; 4, Tubbs; 5, T.E.Y.C.; 6, Shaw. Points 21¼ to 18.

The teams changed boats for the second race which started with a beat into the now ebbing tide. All the boats made a good start and rapidly broke company to form three "matched" pairs. Tacking inshore J. Shaw was quickly marked by one of the home team and unfortunately never recovered. Further offshore a few tacks put R. Chapman ahead of M. Williams. H. Tubbs rapidly found himself being "squeezed" between Williams and the third Thames boat. He was forced to tack out into the stronger tide. At the weather mark Bart's were first, fifth and sixth and the positions remained the same at the leeward mark.

The wind fell to a mere zephyr for the second beat and it was necessary to make short tacks among the swimmers to make over the tide. Chapman made the first break for the weather mark 100 yards offshore and soon discovered that the tide was faster than he had imagined. Luckily, however, he came across a puff of wind which helped him around the mark before the opposing team could head him off. By the time they had enough wind to round the mark as well, Chapman was gybing half a mile away. A similar time elapsed before Tubbs and Shaw rounded.

The final order was 1 Chapman, 2 Williams, T.E.Y.C. 3, and 4, T.E.Y.C., 5 Tubbs, 6 Shaw. Points 21 to 18½.

Result: a win for the T.E.Y.C. by 42½ to 36½.

An excellent supper was provided after the racing and the teams were still to be seen at the bar at closing time after a very enjoyable day. The return match will be on the Welsh Harp in November.

If in the future any one at Bart's, nurses included, can arrange a similar event please inform R. Chapman at College Hall.

M.J.H.W.

RIFLE CLUB REPORT

United Hospitals Cup—Bisley Meeting:

Only three hospitals entered teams in the U.H. Cup: Bart's, Westminster and the London.

The competition, for teams of four, was shot at 200, 500 and 600 yards. Bart's shot well at 200 and 500 yards, but—true to form—had a disasterous collapse at 600 yards, which resulted in our coming third.

Team: Tony Knight (91), Mike Rymer (89), Ian Franklin (89), Gareth Tuckwell (88).

The staff match will be held at Bisley on September 7th, and the following week there will be a friendly (and fairly liquid !) match against Whitbreads brewery.

Small-bore in 1969-70:

It is hoped to provide better .22 facilities for the coming season. The range has been cleared out, thanks to the efforts of Ken Parsons and his colleagues, and all being well it should receive a coat of paint. We have now acquired some more small-bore equipment, and there will be a new system of target frames.

Efforts are being made to obtain more shoulder-to-shoulder matches with a corresponding drop in our pistol match commitments. Many thanks to Gareth Tuckwell for all the work he has put into getting the range cleared.

M. J. Rymer

Saint Bartholomew's Hospital

JOURNAL

contents

Vol. LXXIII No. 11 1st November 1969

journal staff

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Clinical Sub-Editor: **Anthony Newman-Taylor**

Sports Sub-Editor: **Noel Snell**

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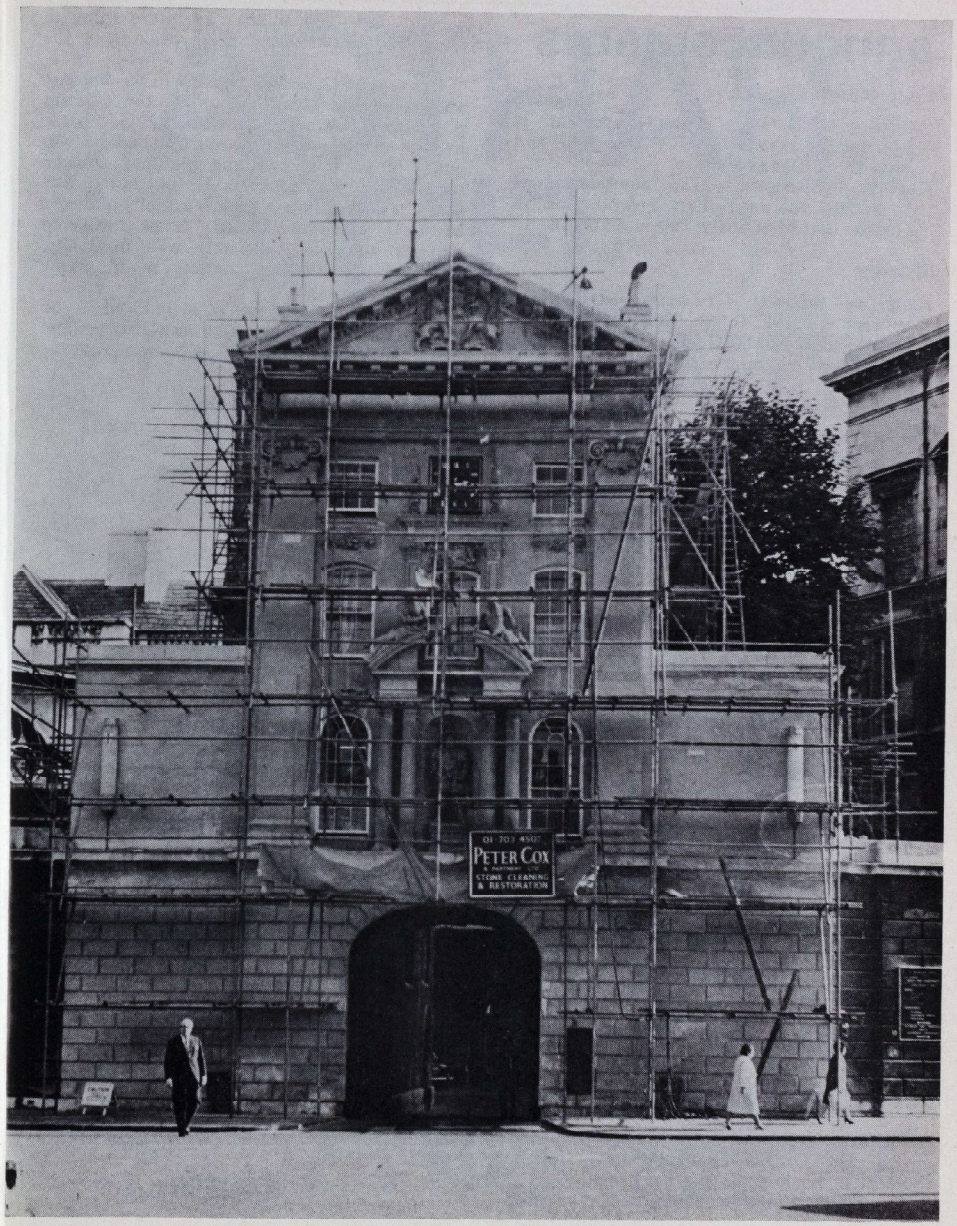
EDITORIAL · OPINION · EDITORIAL · OPINION · EDITORIAL

During the last year there has been a slow burgeoning of vocal student criticism against the constrictions of traditional clinical teaching. Much of the criticism has been constructive; some has found its way into the Journal. The various manifestations of this discontent all reflect the need for an increased involvement and an earlier direct patient responsibility. The means of responding to this need on the part of the hospital seem to be hampered by a disinclination for change as well as administrative and structural difficulties.

At the same time it might be said that many students lack a critical awareness of their own role as *student*. We are in a unique position to question everything and ask anybody their medical opinion. Suffering from an acute lack of academic consciousness how can we hope for academic stimulation. The comfortable conservatism of the average Bart's student makes him a very *nice* person but blunts the edge of his critical perceptiveness.

If students get the Journal they deserve then this Journal can be no more radical than the students it represents.



announcements

Engagements

DAVIDSON — DAVIS — The engagement is announced between Dr. C. Davidson and Miss D. T. Davis.

HUDSON — SIMMONS — The engagement is announced between Dr. Trevor Gordon Hudson and Miss Susan Eileen Simmons.

Marriage

KEIGHLEY—SHEPHEY—The marriage took place between Dr. M. R. B. Keighley and Dr. D. M. Shephey at St. George's Church, Leeds on Saturday, September 27.

Deaths

ELLIS—On September 11, Dr. Rowland Evan Ellis, M.R.C.S., L.R.C.P. Qualified 1940.

HARRISON—On Jne 22, Dr. William Robert Eric Harrison, M.R.C.S., L.R.C.P., aged 73. Qualified 1927.

MORTON—On September 15, Dr. James Aird Morton, M.R.C.S., L.R.C.P. Qualified 1922.

ROWE—On August 18, Mr. Robert Morison Rowe, M.A., M.D., F.R.C.S., age 94. Qualified 1899

SLINGER—On June 24, Dr. Leonard Anthony Paul Slinger, C.B.E., M.B.B.Ch., D.P.H., aged 69 Qualified 1927

VOSPER—On August 10, Dr. Sydney Vosper, M.R.C.S., L.R.C.P., aged 87. Qualified 1907.

APPOINTMENTS

North-East Metropolitan Regional Hospital Board

Dr. W. R. Cattell, M.D., M.R.C.P., has been appointed as consultant in nephrology to the East London group of hospitals and to St. Bartholomew's Hospital.

letters to the editor

Dear Sir,

Re. Dr. George Graham's letter in the September issue of the *Journal*.

He is most concerned that an error he made be corrected—"let students in future speak of 'Sir' J. Matthews Duncan in speaking of the Medal".

I have read G. Graham's letter in the *Journal*, and being the great nephew of the eminent physician-accoucheur, wrote to "G. G." in the interest of accuracy, to point out that he was guilty of error in crediting Matthew Duncan with a "Sir" (in brackets). He did not in fact accept a title, although he was actually offered a baronetcy which he declined, "As he considered that he was not sufficiently well financially equipped to comport himself in the style appropriate to such".

Dr. Graham would be much relieved if you could therefore find space for this corrigendum, and thought you might possibly care to add the quoted explanation.

Yours sincerely,
C. Mathews Duncan,
Skills Cottage,
Dilton Marsh,
Westbury,
Wiltshire.

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LEONARDO DRAWINGS

Dear Sir,

May I recommend, through your correspondence columns, a visit to the "Leonardo da Vinci" exhibition at The Queen's Gallery, Buckingham Palace. This magnificent exhibition (estimated value eight million pounds), consists of Leonardo's drawings from the Royal Collection, and includes the famous anatomical studies.

The drawings give a clear insight into the varied aspects of Leonardo's genius—the restless genius which so often caused him to leave his works unfinished. The exquisite draughtsmanship is the same whatever the subject—portraits, war machines, landscapes, horses, caricatures or maps.

The detailed and interesting explanatory notes to the anatomical drawings are the work of Dr. K. D. Keele.

The exhibition will be open until the end of February. Hours of opening: Tues.—Sat. 11 a.m.-5 p.m., Sun. 2 p.m.-5 p.m. (closed Mondays). Admission 2/6d., students 1/6d.

Yours sincerely,
Yvonne Hibbott.

DUTY DRESSERS?

The teaching of Surgery is a vital and fundamental part of the medical curriculum at Bart's. as at any other hospital: and part of this tuition involves spending the night in residence as the Duty Dresser. The lucky student has his own little room in R.S.Q. equipped with all mod. cons. and a telephone to wrench him from the arms of Morpheus when he is needed.

The point of this preamble is simply to ask the question: Is the Duty Dresser really necessary? Students are told, as Duty Dressers, that they will be called first to see everything brought into the Accident Box, after which they should call the Houseman. However. most of the cases coming in are of almost hysterical insignificance, for which the poor, tired, overworked Houseman has to get up. I give the following examples: I was woken at 2.30 a.m. to come and see a man who had grazed his finger and wanted it looked at; I was woken at 3 a.m. to come to see a chap who had been bitten by a gnat two days previously—when I asked why he had chosen to come to hospital at that hour, he replied that he had happened to be passing, so he had just dropped in. The list of cases of similar trivia must be endless in everybody's experience.

What I would like to ask is this: Could not the Duty Dresser deal with these sort of patients himself, as well as minor cuts for which all he has to do is give Tet. Toxoid and put in the stitches. He could, of course, call the Houseman whenever he was unsure of anything, as well as for all important cases. This simple delegation of responsibility would save the Houseman a lot of unnecessary work. The patients could then be asked to attend Minor Ops the next day during normal working hours for assessment of their condition, and any extra treatment.

In a lot of provincial hospitals night casualty is done by a Staff Nurse alone—if a student goes there to do some resident surgery, he is rewarded by being made Cas. Officer at night, with full responsibilities. Why not here?

If unqualified students can do Locums for Housemen here, and sign for treatments, why not the same for the Duty Dresser, especially if he is a second-time surgery student?

The more responsibility given to students before they qualify, the better. This will make them think and put into practice what they have learnt, especially when they know they can turn to the Houseman for advice when needed.

Why not here?

John W. Frank.

IAN HOWAT MEMORIAL

A former Bart's student, Ian Howat, was killed in a plane crash in the Maluti Mountains, Lesotho, on 25th November, 1968. (Obituary, *St. Bart's Hosp. J.*, March, 1969, p. 97). A number of his friends collected a sum of money to purchase medico-historical books for the Medical College Library as a permanent memorial, and the following items have been added to the Library. The book-plate in each book is inscribed "Presented by his friends in memory of Dr. Ian Howat (1935-1968)".

B.J.S.: *Great teachers of surgery in the past*, 1969.

CLARKE, E., & O'MALLEY, C. D.: *The human brain and spinal cord*, 1968.

CRAIG, W. S.: *John Thomson*, 1968.

DREW, SIR, R.: *Commissioned officers in the medical services of the British Army*, 1660-1960, 2 vols., 1968.

LINDEBOOM, G. A.: *Herman Boerhaave*, 1968.

MARGOTTA, R.: *An illustrated history of medicine*, 1968.

MEADE, R. H.: *Introduction to the history of general surgery*, 1968.

POYNTER, F. N. L., *ed.*: *Medicine and science in the* 1860's, 1968.

PUGH, P. D. G.: *Nelson and his surgeons*, 1968.

SIEGEL, R. E.: *Galen's system of physiology and medicine*, 1968.

TALBOT, C. H. : *Medicine in medieval England*, 1967.

WAGNER, R.: *Clemens von Pirquet; his life and work*, 1968.

ZIEGLER, P.: *The Black Death*, 1969.

EPILEPSY

A series of four articles with an introduction by J. W. Aldren Turner

This issue of the *Journal* will have achieved a useful purpose if it causes some students of medicine, undergraduate and postgraduate, to take more interest in epilepsy. The thoughtful reader will be at least as impressed by the ignorance as by the knowledge of the several authors and should find much to criticise in the personal and sometimes idiosyncratic views which have been expressed. This is as it should be for it merely reflects our lack of understanding about why fits occur at all and why they occur when they do.

It is none the less my personal opinion that the advances which have recently taken place in the sciences basic to medicine and in technology now provide us with an opportunity to invade the territories of our ignorance with some real chance of success. Doctors must be increasingly concerned with those disorders which are chronically disabling and socially incapacitating and our neglect of the study of epilepsy should be a source of shame to us. The epileptic patient has a raw deal not only from the community in general but from the medical profession in particular. Despite the manifest need for such studies, I can find little or no evidence of any research into epilepsy being carried out here at Bart's over the last decade.

I have talked to a number of medical students about epilepsy and found them surprisingly ill informed about the condition: and yet it has not been difficult to arouse their interest. Lennox[1] refers to Osler's affirmation that the physician who knew syphilis in all its manifestations knew all of medicine and continues:

"Today's scope for the person who would know all of epilepsy is even wider. In addition to the expanding preclinical sciences, including genetics, he would encounter problems that belong in the fields of paediatrics, endocrinology, neurosurgery, neurology, psychiatry, laboratory diagnosis, and preventive medicine. He would delve also into medical history, sociology, legal medicine, philosophy, religion, social work and medical economics."

Thus there are some grounds for supposing that the study of convulsions should embrace many different aspects of medicine and therefore have a particular appeal and indeed value for the medical student. This particular issue of the *Bart's Journal* has necessarily been restricted to a very small fraction of the disciplines related to epilepsy but the hope is that it may have provided a stimulus to wider reading.

If a clinic for those suffering from epilepsy can be established, not only will the patients themselves benefit but staff and students, medical and nursing, should gain more insight into the condition and become more proficient in its management. The Cohen Report[2], referred to in the Introduction to this issue, stated a pressing need for such clinics to be established as long ago as 1956 and it is a sad commentary on the Health Service that, in the main, this recommendation has not yet been implemented.

1. Lennox, W. G. (1960). *Epilepsy and Related Disorders.* Churchill: London.

2. *Report of the Sub-Committee on the Medical Care of Epileptics* (1956). Her Majesty's Stationery Office.

INTRODUCTION

by

Dr. J. W. Aldren Turner, D.M., F.R.C.P.

Consultant Neurologist

The subject of epilepsy has rightly been chosen for several articles in this issue of the *Journal* as the condition is common, the Cohen Committee of 1956 giving a rough estimate of four sufferers per 1,000 of population in the United Kingdom. The study of the disorder involves many branches of clinical medicine and careful study of the phenomena of epileptic fits has helped in the understanding of brain function. A definition of epilepsy is not easy, but Hughlings Jackson's definition of epileptic seizures as the expression of occasional, sudden, excessive, rapid, local discharges in the grey matter has not really been improved on, although it was formulated almost a century ago as a result of clinical observation. There has been some controversy as to whether one should speak of epilepsy or of "the epilepsies". When however, one realises that the clinical expression of the epileptic fit depends on the part of the brain in which it originates and that there are many causes, structural and metabolic, which may precipitate the occurrence of epilepsy, this controversy seems a sterile and unrewarding one.

The patient who presents complaining of blackouts, turns or attacks, often poses a difficult diagnostic problem. Apart from the various types of epileptic attack one has to consider syncope, and in particular cough syncope and micturition syncope, Stokes-Adams attacks associated with heart block, migraine, transient cerebral ischaemic attacks, metabolic disturbances especially hypoglycaemia, and psychogenic attacks. Although special investigations, in particular electro-encephalography may be of considerable value in this differential diagnosis, the most important element in diagnosis is a detailed history from the patient covering the circumstances in which the attack took place, the first symptoms, his recollection of the course of the attack and its sequelae. It is essential, if possible, to obtain an adequate witnessed account of the patient's attacks. When the diagnosis of epilepsy has been made, the second part of the diagnosis is to discover, if possible, the aetiology. Here again, adequate history taking, including the past and family history and physical examination are essential but it is at this stage that special investigations, in particular electro-encephalography and neuro-radiological procedures including isotope scanning of the brain may be necessary.

The care of patients suffering from epileptic attacks is a challenging problem, involving not only the adequate use of the various drugs available and a knowledge of their side effects, the occasional place of surgical treatment in post-traumatic epilepsy and in some cases of temporal lobe epilepsy, but possibly more important the adequate social care and psychological handling of the patient. There have been arguments for and against the value of special out-patient clinics for epileptic patients. The main point against such clinics has been the view that it is a mistake to segregate such patients and make them feel that they are different from other patients. This view is one which might be taken about any special clinic and if special facilities for the investigation and management of the patients can be provided there seem to be good reasons for the formation of such clinics and it is planned to start one at the hospital in the near future.

It is of interest that a mistaken view about epilepsy has led to an important advance in pyschiatric treatment. In 1935, Meduna introduced Cardiazol convulsion treatment for schizophrenia on the incorrect assumption that epilepsy and schizophrenia were biologically antagonistic. This fallacy led in due course to the introduction of electrically induced convulsions and to the refined methods of convulsion therapy in use today for the treatment of certain types of depressive illness.

EPILEPSY —

HISTORICAL PERSPECTIVE

by

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Is there any point in studying the history of medicine? I believe that there is. From such study we should learn that only to the extent that it is grounded in valid concepts, themselves usually derived from the basic sciences, can the practice of medicine be clearly beneficial to the patient. The medical profession rarely gives more than token assent to this viewpoint and more or less blindly follows the current dogma. In the past the great bulk of the dogma has proved unsound and, not uncommonly, dangerous. "Cured yesterday of my disease, I died last night of my physician."[1] It is at least arguable whether at this present time, when the physician's capacity to do harm has been greatly increased, iatrogenic disease is any less common than in former centuries.

The degree of stress laid upon the "art" of medicine—not defined but supported perhaps by reference to the first Hippocratic aphorism beginning "Life is short and the Art long"[2]—seems often to have been inversely related to the amount of useful knowledge available. Certainly the history of medicine shows that, at all times, those engaged in its practice have tended to assume special powers and indeed special moral qualities which make them, by inference, superior to other mortals. The supportive evidence is never remotely convincing and the most important lesson for any student may be that which demonstrates the fallibility of his teachers. Legitimate authority in some particular domain of knowledge may carry an unwarranted penumbra of wisdom over much wider territories. History demonstrates the dangers which may accrue from this halo effect, in medicine as in other spheres of activity. Thus, Thomas Willis, author of Cerebri Anatome—this book was illustrated by Sir Christopher Wren—while clearly a great and distinguished observer and thinker, none the less believed that epilepsy might be brought about by the Devil.[3] Doubtless he still has his supporters. The student must try to identify the strengths and weaknesses of his teachers in the sure knowledge that much of what he is taught will necessarily be false.

But why the history of epilepsy in particular? Firstly perhaps because, in marked contrast to many disease entities, as, for example, coronary thrombosis and schizophrenia, it has been recognised as such since time immemorial. Thus the code of Hammurabi (2080 B.C.), king of Babylon, includes legislation affecting the marriage of epileptic persons and the validity of their court testimony. Similarly, the "Sushruta Samhita" which according to Pirkner[4] is the "most representative work of the Hindu system of medicine" and "the most carefully written medical treatise of remote antiquity" (600 B.C.) lists causes, symptoms and treatments of "Apasmara" or epilepsy to which disease there are also references in the ancient Hebrew sanitary laws. It seems very probable that trephining or trepanning was carried out in prehistoric times in order to allow egress for the evil spirit with which the

victim was thought to be possessed during a fit and these neurosurgical procedures have also been pursued by more modern primitives as, for example, in the South Pacific Islands,[5] Caucasia and Algeria. History can be approached horizontally as well as vertically and there exist in various parts of Africa today mystical views about the aetiology and treatment of seizures which are not very different from those held in Western civilisation in antiquity.[6] The history of epilepsy presents a paradigm from which to gain insight into the history of medicine as a whole.

Temkin,[7] to whose classical and beautiful book on the Falling Sickness I have had frequent recourse in the preparation of this article, opens his first chapter as follows:

"Diseases can be considered as invasions by gods, demons, or evil spirits, and treated by the invocation of supposedly supernatural powers. Or they are considered the effects of natural causes, and are consequently treated by natural means. In the struggle between the magic and the scientific conception, the latter has gradually emerged victorious in the western world. But the fight has been long and eventful, and in it epilepsy held one of the key positions. Showing both physical and psychic symptoms, epilepsy more than any other disease was open to interpretation both as a physiological process and as the effect of spiritual influences. And whereas purely mental afflictions, such as neuroses and certain maniac and melancholic reactions, often were, and even are, not recognised as pathological, epilepsy, on the other hand, was always considered a disease. As soon, therefore, as a rational pathology was established claiming to explain all diseases as merely physical processes, its explanation of epilepsy became the test for the validity and persuasive power of the whole system. And it is thus that the history of epilepsy becomes, at the same time, an example of the history of magical beliefs and of their refutation by scientists and scientific physicians." It is a mistake to suppose that the irrational approach to medicine no longer persists and that modern dogmas may not be at least as menacing as their more primitive counterparts.

Not only are many of our concepts about epilepsy deep rooted but much of the current terminology was introduced many years ago and it helps us to a sensible appreciation of our limitations if we know something of the origins of the words we use. Esquirol, who carried out most of his work on epilepsy at the Bicêtre and Saltpêtrière hospitals, stated in 1815: "sometimes the attacks alternate in intensity: there are severe and slight attacks: this is what is called le grand and le petit mal in the hospitals."[8] Although these descriptions were understandably vague, it is sad to find most of the staff and students at Bart's still using the same terms with no more precision than Esquirol and this the more particularly when sloppiness in terminology can result in the misuse of anticonvulsants.

I shall now try to illustrate some of the foregoing ideas and, because a vast canvas is covered by the history of epilepsy, largely restrict myself to that part of it which is occupied by treatment.

In the prehumoral period, epilepsy was attributed to "possession", often as punishment for sin, so that its cure had to be supernatural. Even at the time of Hippocrates the testicles of the hippopotamus and the blood of the tortoise were in vogue, most probably because of their magical associations, and as late as 200 B.C., Serapion of Alexandria was prescribing the faeces of the land crocodile, the heart and genitals of the hare, and the blood of the sea tortoise, or, if these failed, the testicles of a boar, ram or poultry cock.[9]

The Hippocratic school is noteworthy for its attacks upon irrational explanations of disease. Hippocrates is usually considered to be the author of "The Sacred Disease", in which book he stated quite clearly that epilepsy was not caused supernaturally, that it reflected abnormal cerebal function and that he supposed it to be, like all other diseases, hereditary.[10] He was among the earliest humoral theoreticians and ascribed the cerebral abnormality to an excess of phlegm.

Two schools of medicine, the dogmatist and the empiricist, were emerging at about that time and Hippocrates should probably be assigned to the former. He was something of a therapeutic nihilist in his reaction to the more favoured specific treatments but advocated a régime for the epileptic patient involving a special diet, exercise, washing and cathartics. This has its counterpart in the régimes imposed upon institutionalised epileptic patients here in Britain within my own memory and similar attitudes can still colour out patient management by the well meaning but ill informed physician.

As the humoral theory became increasingly sophisticated so views on the aetiology of the falling sickness became more stylised and were expressed with growing confidence. Thus Temkin[11] quotes a late Aristotelian explanation of a seizure as follows: "For the thickness of the particles of the odours carried up through

the nose thickens and condenses the psychic pneuma, which is already thick and cold, and renders the psychic pneuma unfit for functions of the soul. Now the body, if not supported by the soul, is overcome by its own weight and falls down." The danger of the theoreticians who at that time classified epilepsy as a wet and cold disease is that, granted a false premise, they can lead us into all manner of error. Almost equally nonsensical and definitely more dangerous present day theories about epilepsy are not hard to find.

Galen, perhaps because he considered the soul to be resident in the brain, systematised Graeco-Roman medicine in such a way that epilepsy became fair game for mediaeval medicine. Equally, however, he was the first to draw a distinction between idiopathic and symptomatic or, as he would have it, sympathetic epilepsy.

A supposed link between epilepsy and lunacy was by then already well established, the goat which was the taboo animal of the moon goddess being especially prone to fits; more generally, most disorders characterised by a periodicity of symptomatology were related to the phases of the moon and brought within the province of the mind or soul doctor. Epilepsy has continued to be the concern of the psychiatrist and indeed it was in the wards of the mental hospitals that serious studies were first mounted; hospitals for the special needs of persons with epilepsy are a relatively modern innovation.

During the dark ages and for a long time thereafter, although the wet cold disease was still treated by hot drying substances, in general white and black magic prevailed often assisted by some such long standing specific as peony root or mistletoe. There are many examples of white magic in the Bible, for epilepsy was, and is, a very useful disorder for those who wish a dramatic portrayal of their short term therapeutic powers in that most people emerge unscathed from a major seizure. Thus the falling sickness became identified with the names of saints and sorcerers and was again regarded either as sacred or profane. Temkin refers to one such treatment. " Write these three names with blood taken from the auricular finger, Jasper[12]"—surely prophetic—"Melchior, Balthazaar, and put gold, frankincense and myrrh into a box. Let the patient say three Pater Nosters and three Ave Marias daily for the souls of the fathers and mothers of these three Kings for a month, and let the patient drink for a month of the juice of the peony, with beer or wine." Note the link with earlier treatments—the blood, the peony root.

I can only urge you to dip into Temkin's book in order to cover the long period between the Renaissance and the 19th century. It tells, among other things, of the attempts of the iatrophysicists and the iatrochemists—the latter included Sir Thomas Willis—to explain health and disease in rational terms during a century, the seventeenth, in which others still relied upon the Royal Touch for the cure of scrofula.

Our more modern myths are no less amazing than those of the centuries before Christ. There had always been a strong association between epilepsy and sexual behaviour and the phrase " Coitus brevis epilepsia est " was attributed to Democritus by Galen. It is interesting that a similar opinion has recently been expressed by Kinsey et al.[13] How far this represents the perpetuation of mysticism and how far it indicates an area for research remains unknown but there can be no doubt that during the 18th and 19th centuries masturbation was regarded by the bulk of the medical profession as the most common proximal cause of fits. Thus in a report of a B.M.A. meeting in 1880,[14] " Dr. Bacon had castrated two male epileptics, with the result, in one case, of great improvement. Dr. Hack Tuke asked under what conditions such an operation would be indicated, and Dr. Bacon replied: in cases of confirmed masturbation, in incurable cases of epileptic insanity." As late as 1907 we find Turner[15] writing: " The presence of a tight prepuce . . . is a well recognised cause of fits in boys. Its removal is frequently followed by great improvement, if not indeed by complete arrest." And again, " Masturbation is an ascribed cause of epilepsy, but it is doubtful whether it is ever the exciting cause of convulsions. As an associated symptom in epilepsy, as in all forms of degeneracy and mental defect, it is of common occurrence." If our distinguished immediate forebears held such views, you may be sure that we ourselves are guilty of errors in thinking of at least the same order.

These theoretical misconceptions none the less resulted in the first break-through in the chemotherapy of epilepsy. At a meeting of the Royal Medical and Chirurgical Society of London in 1857,[16] Sir Charles Locock—physician accoucheur to Queen Victoria and some time lecturer at St. Bartholomew's Hospital—stated that epilepsy might be caused by crowded teeth, that the practice of onanism might be responsible for a great rise in the incidence of epilepsy, and that he had read an account by a German physician to the effect that Potassium Bromide caused temporary

impotence in men. He had tried the drug firstly in cases of hysteria in women and then in cases of " hysterical epilepsy " occurring during the catamenial phase of the menstrual cycle.

Only very gradually did the importance of Bromide as an anticonvulsant agent become realised. Turner reported favourably upon it in 1907[17], though without mention of the side effects. I myself met a number of instances of bromism in a long stay in hospital for epileptics during the late 1950s. The fact remains that Potassium Bromide remains a useful standby for dealing with the acute phase of a recalcitrant case of major epilepsy.

The 19th century was also characterised by many vagaries of treatment, often very harmful, and usually based on the false premises of famous men. Thus Marshall Hall (1790-1857) whose important discoveries about the reflex activity of the spinal cord long remained unacknowledged in his own country, overelaborated them to the point where many types of seizures were considered to be produced by the spinal nervous system[18]. His concept of spinal reflex epilepsy led many, notably perhaps Brown-Séquard, to recommend local cauterisation of the limbs or skull[19] and even to undertake the amputation of extremities for what would now be regarded as focal cerebral epilepsies. Similarly, because closure of the larynx was thought to be responsible for cerebral congestion, Marshall Hall proposed tracheotomy as a possible cure for epilepsy[20]. Brown-Séquard believed that the same result might be achieved by cauterisation of the larynx with silver nitrate which happened also to be in more general use for the medical treatment of seizures. Indeed argyrism was so common among epileptic patients that Todd—of Todd's Paresis — complained about the indelible results of this ineffective therapy[21]. The therapeutic thread linking the pre-Christian and the more modern era is well indicated by the proposal of Reynolds in 1861 that a new trial be made of mistletoe[22].

There have been three major advances in the medical treatment of epilepsy over the last half century. Phenobarbitone was introduced by Hauptmann just prior to the First World War[23], the hydantoinates by Merritt and Putnam just before the Second[24] and tridione by Lennox in 1945[25]. Although it may still be too soon to evaluate the contribution of the succinimides which, like the others, are derived from the barbituric acid radical, most epileptologists—they do exist, though unhappily not in Britain—certainly opt for a succinimide as the drug of first choice in the treatment of clearly defined petit mal.

Many other anticonvulsants have been introduced since the last war and various claims made for their efficacy. In fact the large majority of so called drug trials do not stand up to impartial scrutiny and it would seem that, for the most part, neither manufacturers nor physicians understand how to set about testing anticonvulsants in humans. It therefore behoves those who would prescribe new preparations to proceed with particular caution and perhaps also to read the history of epilepsy.

In my view, iatrogenic fits have greatly increased in number over the last decade. A few of these are the residiuum from enthusiastic psychosurgery, itself often based on theoretical considerations of the most dubious validity—shades of prehistoric trephining. More commonly drugs are exhibited, particularly psychotropic drugs, which have convulsant properties and one is subsequently asked to explain a late onset epilepsy. Similarly, radical alterations are made in hormonal state and/or in fluid and electrolyte balance only for surprise to be expressed that fits have occurred when in fact such a possibility might well have been foreseen. In short one does well to look at the previous treatment in assessing the causes of fits in the 1960s.

It would be almost sacrilegious to comment, even briefly, on the history of epilepsy without reference to Hughlings Jackson. All students know of the unilateral motor seizures which bear his name. In fact he was by no means the first to describe such attacks and would probably have rejected the concept of cortical epilepsy now used to explain them. The essential point about Jackson's formulation is that he conceived an epilepsy as a recurrent, abnormal and localised discharge of cerebral neurones. In the same epoch Fritch and Hitzig (1870)[26] identified the motor cortex in dogs and evoked unilateral, focal motor seizures by electrical stimulation. Parallel work was being pursued by Ferrier in Britain[27] and Jackson's views were in substantial accord with the observations of these early experimental physiologists. At about the same time a number of eminent German neuropathologists were commenting upon the fact that they had identified a particular type of lesion, involving more especially the antero-infero-medial portion of the temporal lobe, in autopsy material from epileptic patients. Jackson seems to have paid little attention to this work but the way in which the several disciplines were merging towards a common notion that clinical seizure

patterns reflect a localised abnormality of cerebral function emphasises the need to maintain a continuing discourse between clinical and laboratory practice.

By the end of the 19th century Jackson himself described an association between gross lesions of the anteromedial portion of the temporal lobe and a special type of epilepsy of complex symptomatology, not uncommonly characterised by the so-called "dreamy-state" (Jackson and Beevor, 1889[28]; Jackson and Colman, 1898[29]; Jackson and Stewart, 1899[30]). This was the beginning of "Temporal Lobe Epilepsy". His diagnosis was based on careful, methodical, clinical observation which makes a mockery of the lamentably common current practice of referring patients for EEG investigations "T.L.E." by those who appear to have made little effort to take an adequate history, much less to understand the phrase.

Just to remind ourselves of the fallibility of heroes, it is worth pointing out that Jackson was opposed to epileptic patients eating meat on the somewhat tenuous grounds that the leopard often exhibits explosive muscular movements and the leopard is a meat eater!

The growth of electrophysiology has had an immense impact upon our knowledge of epilepsy. Only one aspect of this development can be dealt with here, namely that it led to the identification and location of paroxysmal electrical discharges in electroencephalographic tracings taken from the living subject. The detection of pathological function resulted, for example, in the concept of temporal lobe epilepsy being refined and made it possible for significant associations to be established between the clinical and EEG features. This in turn gave rise to a greater understanding of the functions of the temporal lobes and their associated structures and enabled predictions to be made and tested which in due time provided the basis for a more rational neurosurgical approach to therapy. Furthermore, the attention of the neuropathologist was redirected to the histological observations made many years previously and work was undertaken to validate clinical and EEG predictions either from biopsy specimens obtained at operation or from autopsy material.

Although many important matters have been missed, it is hoped that sufficient has been included to demonstrate that the history of epilepsy is not only interesting in its own right but also of more general value. It can certainly lead towards scepticism about current dogma and perhaps prevent the student from swallowing the views of his teachers without at least a substantial pinch of salt. If, for example, one has analysed the elaborate classification of epilepsy proposed by the humoral theoreticians, it materially helps the appraisal of similar modern attempts at classification which seem to fail as did the earlier efforts because they outstrip the known facts to the point of absurdity.

Above all history teaches us that one of the golden rules in medicine is to avoid doing harm.

"Nevertheless, I tell you, concerning epilepsy, that I have had in my treatment many people, young and old, rich and poor, men and women, suffering from almost every kind of epilepsy. . . . When patients come to you, do not dishonour yourself with empty and false promises in the treatment of epilepsy, since almost all epilepsy is eradicated with great difficulty if indeed it can be eradicated."[31] Bernard of Gordon, 1542.

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WHAT IS A FIT?

by

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Definitions

If epilepsy is a liability to recurrent seizures, then the nature of a fit is central to the definition of this disorder. So also, the diagnosis of epilepsy depends upon the recognition of seizures; although in many patients the fits may be only too obvious, in others it may be very difficult to establish with any certainty whether or not seizures occur.

An inability to define a term generally reflects a lack of comprehension in those seeking a definition, or suggests the concept itself to be of doubtful validity. Both may possibly be true of epilepsy; certainly I know of no definition of the epileptic fit, whether in medical textbook, dictionary or monograph, which does not beg more questions than it answers. The major seizure with convulsions and unconsciousness has been recognised since classical times but, as Kinnier Wilson wrote in 1935, "a faint, a cry, a laugh, can be a fit and no rigid semilogical framework can embrace all its phenomena." He would presumably not have accepted the definition, typical of its period, offered by Lennox and Cobb (1928): "We define it" (epilepsy) "as a syndrome characterised by the sudden appearance of paroxysms, of which convulsive movement or loss of consciousness, or both, are a principle element."

The date of this last quotation is significant, for the following year, Hans Berger (1929) reported his discovery of the human electroencephalogram, a development which rapidly transformed the concept of epilepsy. Lennox (1960), in his massive monograph on "Epilepsy and Related Disorders", rejects his earlier definition in favour of one based entirely on cerebral physiology: "Epilepsy is a disorder of the brain expressed as paroxysmal cerebral dysrhythmia. This dysrhythmia, *if symptomatic** is associated with seizures . . ." He then enumerates the typical clinical manifestations as:—

1. Loss or derangement of consciousness or remembrance.
2. Excess or loss of muscle tone or movement.
3. Alteration of sensation, including hallucinations of special senses.
4. Disturbance of the autonomic nervous system.
5. Other psychic manifestations, abnormal thought processes or moods.

Note that the central feature is now the dysrhythmia to which the clinical expression of the disorder is almost incidental.

*My italics.

The Neurophysiology of Epilepsy

But what is meant by a "cerebral dysrhythmia"? The normal EEG is characterised by various rhythmic phenomena; the most striking of these is the so-called alpha rhythm, an electrical oscillation at between 8 and 13 c/s which can be recorded from the back of the head in most normal adults resting quietly with closed eyes. When it is considered that the EEG is detected through scalp, skull, meninges and c.s.f., and that the cerebral neurones number some 10^{10} but the recording electrodes less than two dozen, there may seem to be little chance of obtaining useful information about cerebral physiology by electroencephalography. However, the very fact that brain-waves can be detected at all is instructive. If the vast population of nerve cells were firing independently and producing random action potentials and post synaptic potentials, no signals would be recordable from electrodes on the scalp.

The existence of an EEG indicates that large numbers of cells must undergo similar electrical changes at about the same time. Indeed the amount of activity recorded from the scalp is a crude measure of the non-random behaviour and degree of interaction of the neurones. This proposition will be readily understood by anyone who has listened, with disappointment, to an amateur tape recording of a particularly lively party. Twenty different conversations close to the microphone produce merely a confused murmur; only if the revellers can be persuaded to indulge in some group activity, such as the singing of rugby songs, does the recording become intelligible. The analogy can be pursued further, for just as community singing may be considered a lower level of social activity than private conversation, so also the non-random neuronal behaviour which generates the EEG is probably not concerned with the most obviously important cerebral functions, such as sensation, perception, thought, recall, and motor control. Thus the alpha rhythm is commonly reduced or abolished when the eyes are open, and during visual imagery or mental arithmetic. Conversely, the largest signals to be found in the normal EEG are recorded during deep sleep. It is a fair generalisation that higher cerebral function is associated with asynchronous neuronal activity, while hypersynchronous firing accompanies (either as cause or as effect) impaired brain function or reduced awareness.

In the 1930s, to record brain-waves at all was technologically difficult and only the crudest features of the EEG were demonstrable. None the less, it rapidly became apparent that during a seizure the EEG changed dramatically. The most characteristic electrical correlate of a fit was the occurrence of spikes or spikes intermixed with slow waves. It was conjectured that the abrupt change of electrical potential required to produce a spike must reflect an unusually high degree of neuronal synchronisation. More recently intracellular recordings in animals have shown this explanation to be essentially correct; spikes in the EEG coincide with depolarisation of underlying neurones (Creutzfeldt *et al.*, 1965). The mechanism of the electrical changes is poorly understood, some neurones remain hypopolarised throughout the discharge while others exhibit multiple excitatory post synaptic potentials (Sawa *et al.*, 1968).

A second discovery which rewarded the early electroencephalographers was that various clinically distinct types of seizure were accompanied by different patterns of electrical discharge. During the generalised convulsion of grand mal, spikes were recordable from all regions of the scalp. Generalised paroxysmal activity of more organised waveform appeared during various types of minor seizures. Runs of spike and slow wave complexes with a repetition rate of about three per second accompanied the brief absences of petit mal, while variants of this EEG pattern were claimed (on doubtful evidence) to characterise myoclonic epilepsy, akinetic attacks and other kinds of minor seizures. By contrast, localised EEG discharges were seen in those types of epilepsy in which the clinical manifestations suggested a localised disturbance of cerebral function. Thus the gradual progression of a Jacksonian seizure was accompanied by a spread of spike activity over the corresponding motor area.

Classification of the Epilepsies

The variety and complexity of clinical phenomena encountered in epilepsy render the taking of an accurate history both difficult and time consuming. An intelligent and articulate patient may be able to distinguish half a dozen distinct seizure patterns, each made up of several separate disorders of experience or of behaviour. Thus a classification of fits is essential both as an aid to communicating the clinical details of the individual and as a preliminary to documenting the results of research into epilepsy. Again the EEG findings prove to be of crucial importance, for the epilepsies fall into two natural groups, distinct with respect to clinical features and pathology, but most readily defined in terms of electrophysiology (Gastaut, 1954; Penfield and Jasper, 1954):—

Centrencephalic Seizures are characterised by a diffuse electrical disturbance of fairly symmetrical onset and distribution. It is believed that the diencephalon and upper brain stem are not only involved in the propagation of the discharge to all regions of the cerebrum but also represent its primary source. The clinical picture is either that of generalised convulsive seizures or of petit mal and its variants. Cerebral pathology is not ordinarily detectable and there is evidence of a genetic predisposition to the disorder. Although a family history of seizures is uncommon (3-6%), the patient's relatives often have EEG abnormalities; among the siblings of children with classical petit mal, the incidence of spike and wave activity in EEGs recorded between the ages of 8½ and 12½ years approaches 50% —implying dominant inheritance (Metrakos and Metrakos, 1961).

Focal Cerebral Seizures are accompanied by an electrical discharge of local onset which may remain discrete or may spread to involve other areas of the brain. The source of the disturbance appears to be cerebral grey matter, usually cortex, and the clinical pattern reflects dysfunction or hypofunction of the affected region. Thus involuntary jerking may spread through the limbs of one side of the body when the contralateral motor area is involved, while a temporal lobe disturbance can produce a variety of symptoms, as hallucinosis, disordered affect, dysphasia and automatism. In patients with focal seizures, cerebral pathology is commonly to be found in the region implicated by the localised EEG disturbance and by the fit pattern. Thus among long-stay hospitalised epileptics, an association between temporal spikes and temporal lobe pathology is found in about 80% of subjects (Margerison and Corsellis, 1966).

Beyond the division of epilepsies into these two broad groups, attempts at rigorous categorisation commonly break down. Thus the recent proposed international classification (Gastaut *et al.*, 1964) becomes lost in a maze of semantic complexity, due partly to a confusion as to whether the terms "partial" and "generalised" relate to the EEG or to clinical phenomenology (obviously a localised temporal lobe disturbance can produce a generalised disorder of behaviour). It may, however, be useful to divide the two groups described above, after the manner of Esquirol, into major and minor, thus:—

	Major	*Minor*
Centrencephalic	Grand mal	Petit mal, Akinetic seizures, Myoclonic seizures, etc.
Focal Cerebral	Major seizures of focal onset	Jacksonian temporal lobe, other rarer focal seizures

There remain a number of disorders not readily classifiable which may belong among the epilepsies. They include migraine, trigeminal neuralgia, palatal myclonus and such rarities as cardiac arrhythmias associated with spikes in the EEG (Walsh *et al.*, 1968). It is to be hoped that further physiological studies will lead to a functional definition of epilepsy which will either embrace these conditions or give valid grounds for their exclusion.

"Subclinical Seizures"

Even within the range of phenomenology commonly accepted as epileptic, seizures differ not only in nature but also in severity. The quantitative variability of fits is particularly striking in the case of centrencephalic minor seizures such as petit mal. Careful observation of a patient with frequent discharges of spike and wave activity indicates that such clinical signs as "absence", staring, eye-lid flutter and the like appear only during bursts of paroxysmal activity more than two or three seconds in duration. The length of discharge necessary to produce a clinically recognisable change in the patient depends upon the skill of the observer, but the briefest spike and wave episodes are usually without apparent clinical concomittants. A number of workers have been interested, therefore, to study the effect of increasing the sensitivity of the methods used to detect altered behaviour. If, for example, the patient performs a continuous task intended to measure reaction time or vigilance, it is found that during bursts of spike and wave activity, errors increase and reaction time is lengthened, even in the absence of overt seizures (Tizard and Margerison, 1963; Hutt *et al.*, 1963). Thus psychological testing would appear more sensitive to impairment of cerebral function than is clinical observation, and the EEG may be more sensitive than either. However, anomalies often occur. In a series of 18 patients with minor seizures, Mirsky and

van Buren (1965) found that episodes of inattention occurred without impaired performance of psychological tests and, most interestingly, that the onset of inattention sometimes preceeded the bursts of paroxysmal activity by several seconds.

Whether or not the clinician elects to describe episodes of increased reaction time and EEG disturbance as fits, it is a matter of no small concern that such attacks can occur and escape detection by an experienced observer. One implication of this fact is that the clinical opinion that a patient's seizures are controlled offers no guarantee that he is not subject to episodes of impaired cerebral function which would be extremely dangerous if they occured, for example, while he was driving. Another aspect of the problem of "subclinical seizures" is represented by certain children referred from a variety of sources for EEG investigation of apparent subnormality of intelligence. The history is often of a few major seizures in infancy but otherwise normal development, followed by a rather sudden falling off in scholastic performance. The EEG reveals short bursts of centrencephalic paroxysmal activity, sometimes repeated many times in every minute and accompanied by an inability to continue simple tasks such as counting. It is hardly surprising that a child who is effectively unconscious for about 5% of the time has educational difficulties, but these are often dramatically ended by the use of anti-convulsants.

Lennox's (1960) definition of epilepsy as paroxysmal cerebral dysrhythmia, together with the implication that a seizure can be asymptomatic, is more likely to be acceptable to electroencephalographers than to neurologists. The validity of his concept cannot be assessed, nor can "cerebral dysrhythmia" itself be rigorously defined until much more is known concerning the pathophysiology of epilepsy and the relationship between paroxysmal activity and psychological variables. Meanwhile, for most practical purposes any disturbance of mental function or of motor or autonomic control consistently associated with paroxysmal discharges in the EEG may usefully be regarded for purposes of management as a fit.

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THE PATHOLOGY OF THE BRAIN IN EPILEPSY

by

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In England and Wales about five people in every thousand are epileptic; the incidence is much the same in other countries. Fits are particularly liable to occur in the early years of life and about 60% of epileptic patients develop their illness before the age of twenty (Alajouanine, Castaigne and Laflane, 1959).

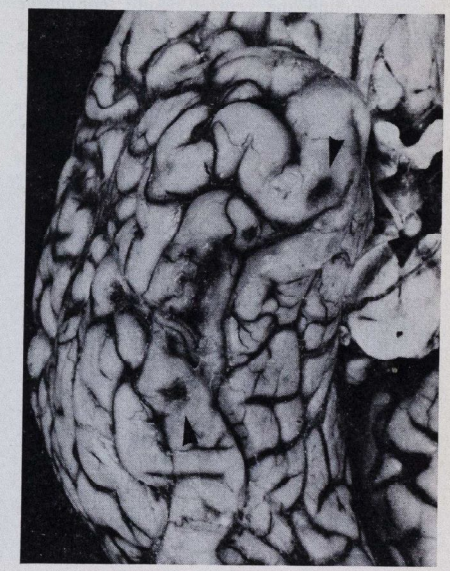
Although the length of life must sometimes depend on the cause of the fits, many epileptic people live a full life into old age. Death attributable to epilepsy is recorded more often than may be realised. In 1967 for example over six hundred were recorded, almost half of which were due to status epilepticus.

There are many different ways of classifying the common forms of epilepsy. Pathologists usually divide them into two: one division is the symptomatic (or secondary) type; the other used to be known as "idiopathic" but is now often referred to as "cryptogenic". This is a convenient split with which to start, although it is not so easy to sustain.

Symptomatic Epilepsy

Epileptic attacks may occur in association with many different illnesses that are centred outside the nervous system. Uraemia, and hypoxia, however they are caused, are examples. Most symptomatic or secondary fits however occur because of abnormalities within the brain. These may be either diffuse or focal

Fig. 1 Cortical scarring running along the inferior temporal gyrus of the right temporal lobe. The limits of the scarring lie between the two arrowheads.



but it is difficult to be precise about the incidence of the different kinds of abnormality, since the figures will vary according to the source, and to the age, of the patients being examined. For example, Bridge (1949) in his analysis of some seven hundred children, found that "birth injury" was the commonest cause since it accounted for nearly a quarter of the cases. Vascular accidents and head injury after the neonatal period accounted for a further 12% and 7% respectively, while infection (3%), malformation and tumour were all considerably rarer.

When fits develop in the adult, tumour and trauma become the more important of the identifiable factors. Although generalisations are of limited value, almost 50% of patients with *intracranial tumours* suffer from epileptic attacks at some time during the illness and not uncommonly an attack is the presenting symptom. As might be expected, tumours are more likely to give rise to an epileptic fit when they are situated in the cerebral hemispheres than when they lie below the tentorial opening. Within the hemispheres, the position of the tumour and also its nearness to the cortex are important. Lund (1952), for example, in his detailed study of "epilepsy in association with intracranial tumour" found that the most sensitive region was the frontal and parietal cortex around the central fissure, convulsive attacks having occurred in 75% of patients with a tumour in this area. The figure for the temporal lobes was 50%, and for other areas it was still lower. Lund also noted that fits tended to occur less often in glioblastoma than with meningioma and the more slowly growing gliomata.

Epilepsy following *head injury* is more likely to occur when the wound is open than when it is closed. Russell and Whitty (1952) found that attacks had developed in 40% of their patients during the five year period after a penetrating injury to the skull. Much as with tumours, the most sensitive area was the cerebral cortex adjacent to the central fissure. In closed head injury the incidence is much lower. Jennett (1965) gave the overall figure as less than 10%, but he emphasised that the risk varied greatly according to the severity of the injury and the nature of any complications. Thus the combination of prolonged post-traumatic amnesia with a depressed fracture of the skull carried a much greater risk than either on its own.

In later adult life and in old age epilepsy is also found in various degenerative conditions of the brain. Alzheimer's disease, cerebral infarction due to vascular disease, and sometimes senile dementia, are examples. As at any age, very occasionally elderly epileptic patients will be found with cerebral infection, such as an abscess or even an encephalitis.

At all ages, however, there are many epileptic patients in whom there is no underlying disturbance to be found either within the nervous system or outside it. The figures quoted earlier from Bridge's study illustrate this, for probable aetiological factors could only be identified in about half the children. In an interesting study of late onset epilepsy in fifty-one patients who were also psychiatrically ill, Serafetinides and Dominian (1963) found no cause for fits in as many as 74%. Such patients make up the cryptogenic group.

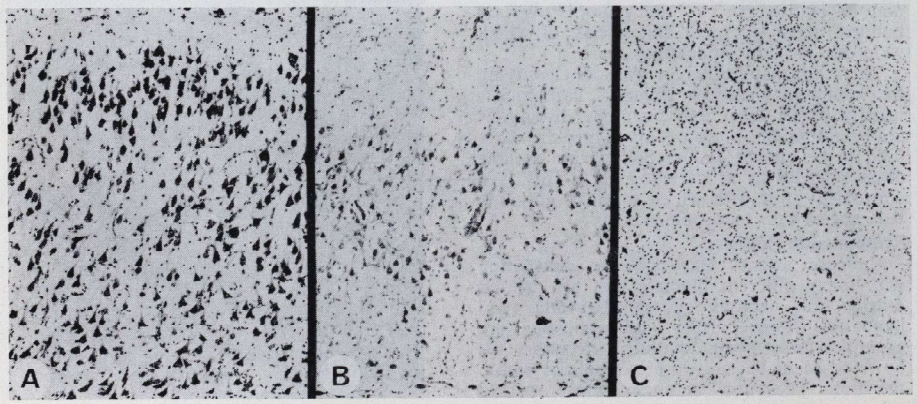


Fig. 2 **A** Neuronal population in a segment of a normal hippocampus.
B Acute necrotic lesion in the same area of an epileptic patient dying after severe epileptic attacks.
C The end result of **B** with almost complete loss of nerve cells and an increase in glia.
A, **B**, and **C**, x 70

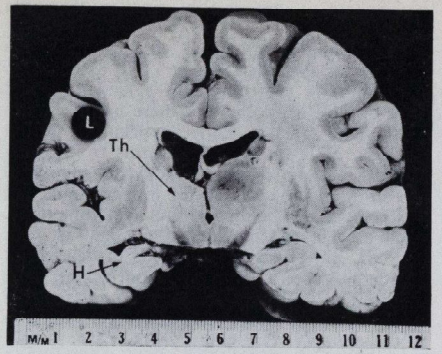


Fig. 3 Coronal cut through the brain of a patient with long-standing epilepsy. There is atrophy of the left hemisphere but the hippocampus (H) and the thalamus (Th) are particularly severely affected.

Cryptogenic Epilepsy

The title of this group implies that the cause of the epilepsy is unknown, or at least obscure. This is not, however, the same thing as saying that the brain appears normal, and in many cases of cryptogenic epilepsy it does not. The simple, if dogmatic, explanation of this paradox is that epilepsy can itself lead to cerebral damage.

This may happen in two ways. First, there is always the risk of patients falling and hurting themselves during a fit. In so doing they may damage their brains severely and appreciable bruising or cortical scarring can be found surprisingly often in patients with chronic epilepsy. The distribution and the appearances of the scar are typical of a closed head injury and are sometimes referred to as "contrecoup". The lateral convexity and the pole of the temporal lobe tend to bear the brunt of the damage along with the orbital frontal cortex. The convexity of the frontal and of the parietal lobe is much less often affected. Damage of this kind is usually bilateral but is often more severe on one side than on the other. If it is recent, haemorrhage and necrosis will be found; if it is old, as it usually is, the surface of the cerebral cortex appears eroded as if by an ulcer, with the floor formed by the underlying white matter (fig. 1).

It may of course be impossible in some cases to decide whether such damage is the result of epilepsy and not its cause, particularly as epilepsy following trauma is not uncommon. There is little doubt, however, that in most cases the epilepsy antedates the injury and occasionally this is well demonstrated by the presence in the same brain of an old cortical scar and a recent bruise. The possible influence of such scarring on the course of the epilepsy is discussed later.

The second type of damage is more common. It consists of patchy or more diffuse loss of nerve cells with the proliferation of glia.

Not all parts of the brain are equally vulnerable, but in about half the patients with cryptogenic epilepsy (and much less often in those with symptomatic epilepsy) the medial temporal grey matter is affected and "sclerosis of the Ammon's horn", or hippocampus, is the result. Fig. 2 shows this sequence of events. The first field (A) illustrates an area of normal hippocampal cortex; the adjacent field (B) is taken from the same area in the brain of a patient who died shortly after epileptic attacks. Most of the nerve cells in this view have been destroyed and the remainder are shrunken; the background consists of proliferating glia and a few prominent vessels. The third field (C) shows the scar that develops when the patient survives long enough. There is almost total loss of neurones, the field consisting of little more than the nuclei of fibrous astrocytes.

The human hippocampus is a sizeable

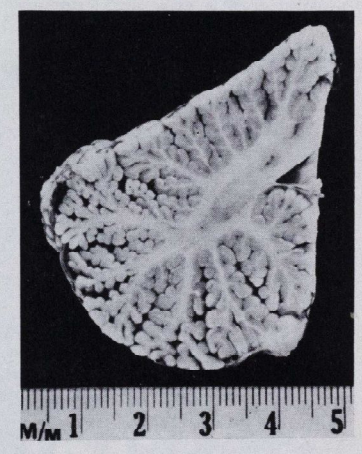


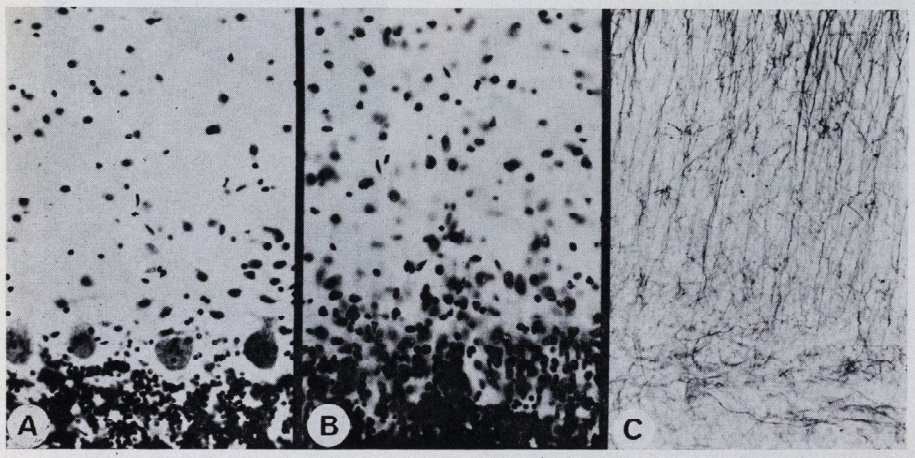
Fig. 4 Oblique cut through the cerebellum showing the normal appearance dorsally but marked atrophy of many folia on the under (convex) surface.

structure, measuring some 4.0 cms. in length and up to 1.5 cms. across. Its atrophy can therefore usually be seen naked-eye, particularly as in most patients only one side is affected. Fig. 3 shows the typical shrinkage and loss of grey matter in the left hippocampus (H) compared with the normal appearance on the right.

Cerebellar atrophy is also common. This too is often gross enough for the shrinkage of folia to be easily recognisable (fig. 4) but it may at times be detectable only under the microscope. The tissue reaction is similar in nature to that seen in the hippocampus. The most vulnerable cerebellar neurones are the pear-shaped Purkinje cells, and Fig. 5 illustrates how they are destroyed while the glia proliferate in the molecular layer and a dense fibrous gliosis is laid down by astrocytes. The neurones of the granular layer are also often destroyed but this is not illustrated.

Other areas in the brain that are not infrequently affected in the same way include the thalamus (Fig. 3) and various parts of the cerebral neo-cortex. Very occasionally one entire cerebral hemisphere is smaller than the other owing to diffuse atrophy. Such a hemiatrophy is seen in Fig. 3 in which, as well as the localised hippocampal and thalamic atrophy on the left side, the whole of the left hemisphere is appreciably smaller than the right.

Fig. 5 **A** Normal cerebellar cortex, with molecular layer, 4 Purkinje cells and a small part of the granular layer.
B Cerebellum in epilepsy. Loss of Purkinje cells and marked increase of glial nuclei in molecular layer.
C Fibrous gliosis in similar area of cerebellum.
A. B. and **C.** x 250



The origin of all these changes has been debated for the last 150 years. No explanation of them is as yet completely satisfactory, and least of all a brief one, but there is little doubt that the damage derives primarily from hypoxia, or in more general terms from a disturbance of the blood and oxygen supply to the brain, regions such as the hippocampus and the cerebellum being particularly vulnerable. Such a view is strengthened by the fact that hypoxia, however caused, results in damage of the same type with a predilection for the same areas. The difficulty is to account for the hypoxic episode and then to establish its relation to the epilepsy. For many years it was taught that this hypoxic scarring was no more than the effect of the physiological disturbances occurring during or after an epileptic attack and that it could therefore be discounted as a possible factor in the causation of fits. There would then be no contradiction in the fact that although brain damage was present the epilepsy was classified as cryptogenic.

This traditional view of the inertness of the scar, however, began to be questioned, particularly in so far as the hippocampal sclerosis was concerned, when psychomotor attacks, and the E.E.G. abnormalities associated with them, were found to occur most often when there were also structural abnormalities, including atrophy, of the medial temporal areas. It was then postulated by Penfield and his colleagues (1953) that the hypoxic insult was not

post-epileptic in origin but that the damage occurred at birth by deformation of the skull and herniation of the medial temporal gyri through the incisura of the tent. The distortion of the brain impedes the blood supply to these areas, in which the hippocampus is situated, the neurones die, and the resultant scar or sclerosis is liable to "ripen" in the course of time and to develop into an epileptogenic focus. If this view is correct, the epilepsy should be classified as "symptomatic". Gastaut (1959) supported Penfield's view that the brain damage antedated the epilepsy but he attributed it to the effect of infection or injury occurring during the first few years of life rather than injury at the time of birth.

Although both hypotheses may apply in some instances, neither is completely satisfactory, for there are too many adult epileptic patients with hippocampal sclerosis in whom no evidence can be found of such an eventful early life. The one fact that does seem to emerge from the study of such patients with hypoxic scarring, is the relative frequency with which severe fits, and often status epilepticus, had occurred for the first time during infancy or childhood (Falconer, Serafetinides and Corsellis, 1964). Moreover, if a young patient dies in status it is more than likely that acute hypoxic damage will be found in the hippocampus as well as in one or more of the other vulnerable parts of the brain (Norman, 1964). Since the cause of the status brain damage in such children often cannot be found, the neuronal degeneration and the gliosis are attributed to cerebral hypoxia occurring as a consequence of the severe and often prolonged convulsions. In this sequence of events, the damage must be a sequel to the epilepsy. Thus the apparent paradox is reached in which identical cerebral lesions may in one case originate prior to, and in another be the consequence of fits. The important thing is that the damage has occurred at all for there is now evidence that the hypoxic scar may act as an epileptogenic focus in much the same way as other kinds of brain damage. Like them moreover it may influence the pattern of attacks according to its localisation. For example, an epileptic patient, whether he has a small tumour or a hypoxic lesion in the medial temporal grey matter, is much more likely to suffer from attacks of a "temporal lobe" type than a patient without such demonstrable abnormalities (Margerison and Corsellis, 1966).

The implication of all this may now be summed up. Many epileptic patients, and particularly those living in hospital or in a sheltered community, suffer from major fits the causes of which are unknown. Occasionally when such patients die, an unsuspected malformation such as tuberous sclerosis, or a cerebral scar or even a tumour, is found, and the diagnosis of symptomatic epilepsy would have been more appropriate.

In most patients, however, the brain either appears normal or shows the hypoxic type of damage. It has been customary in the past to include both in the cryptogenic group since the hypoxic damage was considered to be of no consequence. This, however, can no longer be accepted for, when it occurs, it most often affects the medial temporal areas and there is then the strong possibility that "temporal lobe epilepsy" will sooner or later appear.

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THE MANAGEMENT OF EPILEPSY

by

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This is a broad subject which can be split into many sub-divisions, depending for instance on the age of the subject, his intelligence and emotional stability, the cause of the epilepsy, the frequency of attacks—and their nature, and the extent to which the attacks interfere with day-to-day life.

First, I am assuming that no surgically or medically treatable condition has been found to be causing the epilepsy—*explanation* is needed to the parents or the patient and/or spouse. Many patients—and the public are still unable to accept the expression "epilepsy", feeling that it implies mental retardation and that the epileptic attacks are something to be feared and treated with a mixture of disgust and intolerance. Relatives, friends and employers (I think in ascending order) still tend to have the feeling that attacks should be kept hidden and employers often feel unwilling to accept an employee to whom the label "epileptic" has been applied. They expect, I think, unpredictable and perhaps dangerous behaviour on the part of the sufferer. Parents and patients often think that the condition is hereditary and that marriage, or at least childbearing, are unthinkable for an epileptic subject. This is of course not true in the vast majority of cases.

Explanation must first consist of a statement that the attacks *are* epileptic, for this has to be faced first and then a few necessary adjustments made in the patients life. One must follow the statement immediately—as it is often a shock—by the further statement that *anyone* is, to some degree, subject to epilepsy and attacks can be induced in virtually anyone. This is a matter of the threshold at which the brain produces the electrical disturbances which result in what we call epilepsy. This threshold is a little lower in an epileptic subject than in the average population. One can cite the fact that many people feel odd when faced by flickering light—and say that this will in particularly susceptible people provoke an attack.

Next, having used the expression epilepsy deliberately, so that the patient can accept a few limitations on his life, one must explain what is doubtless already known—that the general population, despite our efforts at educating them better, still consider epilepsy in many ways unacceptable and that the patient is, therefore, better off to refer to his epilepsy as (e.g.) "attacks". He must be advised not to call them "Faints" or "Migraines" as is sometimes done, as this can be very misleading. The term "Blackouts" is also often used, and is

acceptable, but again sometimes misleading to the doctor. The patient should carry with him a card containing details of his condition, the tablets he takes for it, and preferably also what to do if an attack occurs. It is *not* usually necessary to call an ambulance, for instance, and a note of the hospital the patient attends and registration number of the patient at that hospital, and the names of the general practitioner and consultant he attends. This is an ideal, which is not unfortunately widely accepted as yet. School: Normal if possible with discussion with school staff. Condition not to be treated as anything unusual.

Limitations on Activity in the patient able to lead an otherwise normal life: (1) No driving within three years of an attack whether on or off medication. This, at present, applies legally to all subjects of epilepsy, but there may well be a change in the law in the future so that people who have epilepsy only when asleep may be allowed to drive, and people whose attacks are controlled by medication for three or more years may be allowed to drive. (Both these imply that there is no physical handicap to prevent driving).

(2) In children and younger adults—participation in "sports" in which minor head injury is likely is unwise as it can lead to an increase in attacks (e.g. playing rugby football, particularly when the head is not protected; swimming is dangerous as an attack in the water often goes unnoticed. Any epileptic swimmer *must* be accompanied by a friend who is constantly on the watch for an attack and who is capable of rescuing the subject if necessary. Diving is contra-indicated because of the minor blow to the head involved. Small boat sailing is unsafe even with a "safety" harness.

(Other sports which are contra-indicated for clear reasons are flying, gliding, climbing, mountain-walking, unless accompanied and within reach of adequate cover, and aqualung diving).

Cycling is inadvisable in a severely epileptic subject, but not in one with prolonged warning of attacks, nor in the nocturnal epileptic.

These limitations have to be repeated to the patient over and over again as he often finds them very difficult to accept.

Limitations at Work: Any situation involving danger to the patient or others—climbing ladders, scaffolding, working at heights, working with unprotected machinery or open fire (and usually working in a chemical laboratory—although I do not think that this should be an absolute rule). Employers often prefer that people do not have attacks in front of their clients and so are disinclined at present to allow patients to take on work which involves interviewing the public, but again this involves some prejudice and is by no means an absolute rule.

The Type of Attack

(a) Petit Mal—if these attacks are really brief they are often unnoticed except by parents and the experienced observer. They do not necessarily lessen as a child gets older and may indeed be accompanied or replaced by some major attacks—either of a temporal lobe variety or grand mal. Driving is contra-indicated, as is swimming, and the other sports already mentioned, but there is very little prejudice against this type of attack and work may, therefore, be more easily come by.

(b) Temporal Lobe Epilepsy—"uncinate" attacks may consist only of a brief feeling of nausea, perhaps fear, sweating and pallor and momentary difficulty in communication. Many people with attacks of this kind find that their attacks go unnoticed and they can lead virtually normal lives within the limits above. More severe uncinate attacks can go on to loss of consciousness and attacks arising elsewhere in the temporal lobe may cause automatic behaviour or go on to a major attack. The automatism can involve anti-social or belligerent activity and this may restrict life more than other types of attack. (As belligerence may occure with full awareness in some patients and may be caused to some degree by medication, it needs detailed assessment before one simply limits a patient's activity because of it.)

(c) Attacks of any kind involving incontinence—this is a serious social problem, particularly when it accompanies attacks which would be otherwise unnoticed. It is *sometimes* helpful, if the attacks cannot be entirely controlled, for the patient to *induce* an attack (a few patients can do this) while in safe private surroundings, as he often knows that he will then be free of attacks for a definite period afterwards, and can for instance work in public, or attend board meetings etc., without the likelihood of embarrassment.

(d) Frequent Minor Attacks: often appear as unusual behaviour on the part of the subject. They may occur despite heavy medication, and reduction and/or change of medication in hospital is usually best.

The E.E.G. may be the most reliable means of knowing that this type of status (minor or petit mal status) exists.

At this point I think it should have become increasingly clear from what has been said so far that the management of epilepsy and the recognition of changes in the patient's state, are best carried out by the same person or by a small group of people all of whom know the patient well.

Thus, anyone knowing a patient subject to minor attacks well would be aware of a change in behaviour and aware of the possibility of minor status. Someone seeing the patient for the first time might well be given the impression of anxiety or inattention and not realise the true state of affairs.

(e) Frequent Major Attacks: These are often not easily controlled by medication, although a change in drugs may help. Admission to hospital and change of drugs while under observation is often most satisfactory. If the attacks remain frequent and treatable pathology is excluded (and "epilepsy surgery" not indicated) then institutional care may be required (this should be aimed at producing reasonable control of the attacks, while rehabilitating the patient to enable him to work and to enjoy a social life—even if both these are somewhat limited).

(f) Groups of attacks of all kinds, may occur at very infrequent intervals. It is of help to know the circumstances surrounding the onset of a group of attacks—whether the patient is unusually fatigued, has not been eating normally, has had an intercurrent infection, has appeared depressed, has perhaps been forgetful and omitted to take regular medication, or has had some change in general health which may perhaps have altered the amount of his drugs which he has absorbed (conversely, the patient with drug intoxication from Phenytoin for instance, may while maintaining a normal dose of the drug absorb much more than usual of it, due for example to severe constipation). Insufficient is known about the causes of variability of frequency of attacks, and it is hoped that more will be learned by the trained observer who is responsible for the management of a patient's epilepsy, if he sees the patient himself at regular intervals. As groups of attacks or even single attacks can be very infrequent, if one suspects that they are occurring, one should maintain regular medication despite long periods of freedom from attacks.

(g) Focal or major status epilepticus must be treated as a medical emergency by admission to hospital and appropriate drug therapy with or without ventilation and curarisation. Relatives and friends must be warned of the dangers of repeated attacks and advised to seek medical advice *early*. They should also be warned that memory loss for a period of several months preceding the status may occur on recovery.

(h) "Epileptic psychosis". While this can almost certainly occur when attacks are frequent or after a long period of status, it is very probable that it is most often caused by medication. It may be necessary to substitute one anti-convulsant for another before gradual recovery occurs.

(i) In the brain-damaged patient who also has epilepsy, or in the few *hereditary conditions* (*e.g. epiloia*) accompanied by epilepsy, institutional care may be necessary, but often parents prefer to look after their children themselves not often realising the problems that can arise when the child becomes adult. The parents are no longer able to look after him and he *then* has to be transferred from his familiar surroundings to an unfamiliar hospital. It is perhaps wiser to foresee future difficulties and give advice to the parents along these lines—but *only* give advice, for the decision about institutional care will rest with the parents unless the patient's behaviour is such that the matter must be taken from the parent's hands.

(j) Heredity: Having mentioned above that a few hereditary conditions *are* associated with epilepsy, one must emphasize that they are rare, and that epilepsy alone is *not* hereditary—but is a sufficiently common condition to be found in more than one member of most families. Some children are brought up to believe that their epilepsy could be conveyed to any of their own children and they face a very difficult time when they wish to marry, come for advice, and have to try to accept the opposite of what their parents have always told them about the condition. One has to be very careful not to appear to put the parents in the wrong over this matter—it is best to explain that "our knowledge has increased and we know that your epilepsy is not hereditary".

(k) Changing pattern of attacks: Here again the trained observer seeing his patient regularly will notice a change in the pattern or type of attacks. This may well be an indication for further investigation, in case a previously undiscovered neoplasm has started to grow and may require surgery, or an arteriovenous malformation—previously undiscovered—might have bled and require removal if this is feasible or evacuation of a haematoma if necessary.

(k) Social Management: Apart from acquiring a good relationship with the patient and his family and/or friends, one may need to give advice about work, and steer patients towards the most suitable work—or earlier give some indication of the most helpful post-school education. It is often *better* that the patient is *not* registered "disabled" (he is not at all disabled) but is encouraged to find work for himself without help from the Labour Exchange. It may be a positive disadvantage to be on the Disablement Register, partly because of the still present prejudice against epilepsy. The social workers are encouraged to join with the medical staff in giving advice to patients about this matter.

It is a help for the patient to visit the British Epilepsy Association.

MEDICAL TREATMENT: (some variation in thought on this subject).

(1) Petit Mal in children—treatment of choice now Ethosuximide, (dose varying up to 250 mg three or four times a day after meals) with or without Phenytoin or Phenobarbitone in small dose to prevent any major attacks.

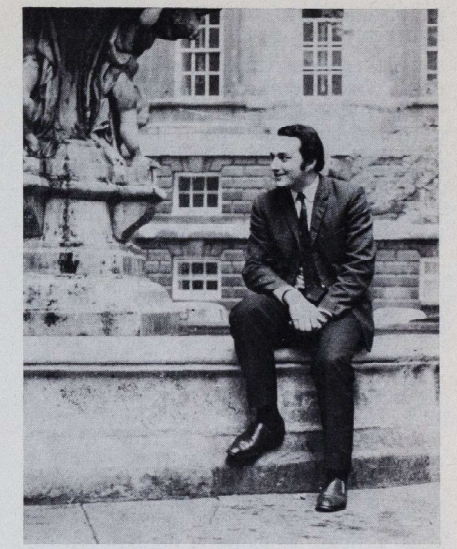
(2) Minor-Major and Major Epilepsy ("grand mal")—usually Phenytoin 100 mg three (or four) times a day and either Phenobarbitone 30 to 60 mg three times a day *or* Primidone (Mysoline) 250 mg three times a day or four times a day (either/or, as they resemble each other chemically closely—in theory not necessary to use them together, although in practice sometimes useful.

(3) "Temporal lobe" attacks: Phenytoin and Mysoline. Methsuximide (Celontin) has also proved practically very helpful though in theory it ought to help petit mal more. Many other anti-convulsants have been used. These are the most useful ones.

(4) Status Epilepticus: Initially drugs which do not cloud consciousness, i.e. Phenytoin 200 mg stat. i.m. and then 100 mg 6 hourly i.m. for 48 hours, reducing if possible to 8 hourly, plus (if necessary) Phenobarbitone 200 mg i.m. stat. followed by 100 mg i.m. 6 hourly for 24-48 hours, and then 60-100 mg i.m. 8 hourly (reducing to 30-60 mg *orally* when this route is feasible). If this does not help within half an hour add Diazepam 20 mg i-v slowly (for an adult) and warn the resuscitation unit that their help may be needed with ventilation. If no improvement, with anaesthetist, ventilation and endotracheal tube at hand, give Diazepam 40 mg i-v slowly and then if necessary in an i-v drip, 40 mg (or so) i-v every 4 to 6 hours, reducing the dose gradually until consciousness lightens and/or fits begin again—if control not gained quickly, tracheostomy helpful. Aim to *control* convulsions, which increase oxygen consumption of cerebral cells considerably and for this reason if necessary give *curare* to prevent movement while maintaining ventilation mechanically and giving smaller doses of anti-convulsants than above. On the whole Paradehyde is not now required—it can be dangerous even though an effective anti-convulsant and can cause abcesses if used repeatedly. Intravenous barbiturates are no more helpful than Diazepam and less safe. Intravenous diuretics such as Frusemide, Mannitol or Urea may help by reducing cerebral oedema. Give antibiotics to prevent chest infection or treat pre-existing infection and meanwhile make certain that nothing treatable has precipitated the status—such as a minor head injury and possible contusion or haematoma; maintain food and fluid intake. Serial E.E.G.'s may be very helpful in controlling medication in status in a patient who has been paralysed or is unconscious.

AN INTERVIEW WITH THE CHAIRMAN OF THE STUDENTS UNION

As Richard Page comes to the end of a year's experience as Chairman of the Union we put the following questions to him . . .



RICHARD PAGE

What are the functions of the Chairman of the Students Union?

There are two things, first of all he acts as a link between students and the Medical College staff and administrators, and secondly, he sees that the affairs of the Union run smoothly. The Union has been pretty well organised over the years in its actual functions. The main aims are stated in the Constitution, that is to say organising sporting activities and various sub-committees, such as the Teaching Committee and the Wine Committee, and to see that these carry out their normal functions.

How much time do you devote to the Union? Do you enjoy doing it?

On average about seven hours a week. Yes, I do enjoy it but I think that being the Chairman is not necessarily the best job. These are probably done in the various sub-committees where you are doing one particular job and you can devote all your time to this one thing.

What things did you particularly wish to see done during your term of office?

I wished to ensure that the Union continued to function effectively. I had some ideas about teaching and other ideas about representation of students on Medical College committees. I would like to see greater communication between staff and students, so that we could each air our problems. In this respect I have pressed for student representation on various committees, so that we may know what is being decided for us, and also have a chance to make constructive criticism before the decisions are irrevocably made.

How successful have you been?

So far I haven't been very successful but I think it's something that these ideas have been expressed. Whether they are acted on or not is something completely different. It's terribly difficult because whenever you put forward any suggestions on these lines people will meet and say " Well, of course, we can't do anything at the moment because . . .", and it's terribly difficult to get any action. I know I'm talking about my own ideas but I think they are shared by lots of other people. I'm not criticising

the Medical College in any way, but it's just that people don't seem to be prepared to act.

Would you say that the Medical College office is even receptive to your ideas?

Yes. I have spoken to the Dean and he is certainly sympathetic towards these ideas and is, in fact, in agreement with a lot of them. But, of course, he sees the problem of putting them into practice better than I do, and, therefore, has to move a bit slowly because he knows what he is up against.

As you may know we approached the Dean for an interview but we have had no reply to our letter. There is a rumour that the Dean has resigned. Is this true?

The best way in which I can answer this question is to refer you to the minutes of the S.U. Council meeting of Thursday, July 24, in which with the Dean's permission I made the following statement. "In order to allay rumours Mr. Page said that when talking to the Dean he had been informed that the question of the Dean's resignation had arisen because he, the Dean, did not feel that he could perform his duties in the Radiotherapy department and as Dean as fully as he would like under the present conditions. He would like to see his staff in the Radiotherapy department increased and was pressing for this."

Do you think that the time has come when students should take into their own hands more effective action to bring their grievances to the attention of the Medical College? Do you think that the time has come for a sit-in?

No I do not. I don't think that this is the right way to deal with this. We have not yet exhausted all the available channels of communication.

Do you think that the Journal could be more instrumental in putting forward the students' points of view?

I certainly do think so, yes. I think it's a great shame that the *Journal* doesn't take a more steadfast line. It doesn't really take a line at all. It puts forward people's views occasionally but it is never really radical in its approach. And it tends to have enthusiasm for these things one month, and the next month they're dropped. I know it's difficult for the *Journal* because it's difficult to get contributors and also it's difficult to get any correspondence going. I think this reflects on the general lack of interest there is about medical politics.

What can one do about this breakdown of communication?

It's very difficult to try and do anything if you haven't got people giving you ideas. It's terribly discouraging if you say you want to put forward a certain policy, and you haven't got the backing of anybody. Not because it's a bad policy necessarily, but just because people aren't interested in anything at all. And you cannot get people together to have a discussion in large numbers. You can get one or two people together usually but not the majority. It's impossible to get things moving along these lines if you don't turn round to the Dean and say "Well, look, I know for certain that there are at least fifty students who would like to see such and such is done . . .", when you know jolly well that fifty students at the moment don't really care.

It's a difficult situation, isn't it, when both students and teachers appear to have lost all interest.

Quite. But I am sure that students would be very willing to do more if they felt that they had some real responsibility. We've got the big incentive of examinations, but I would prefer to see the attitude that you have got to clerk this patient because if you don't nobody else will, and a good deal more of the patient's care is the responsibility of the student than it is at present.

In fact a system could be introduced whereby students who have completed their first medical and surgical firms could take on this increased responsibility.

Yes, it could be introduced. The only problem is that in order for this to work you've got to be there for twenty-four hours a day. Well, this is obviously quite impossible at Bart's because first there is nowhere for students to stay in the hospital. Also the patient : student ratio is not as high as it could be, but some advance has been made in making use of the peripheral hospitals, and I hope this will continue.

In his 1968 report Sir Michael Perrin expressed his confidence that Bart's would continue to play a leading role as an institute of research and as a teaching hospital providing a district responsibility and care. I was wondering if you thought that Barts could still maintain this position. Can we link this up with what you have said about not wanting the character of Bart's to change?

It is difficult to reply to someone else's quote. I think it can maintain its position. From the student's point of view there's no reason why all his time should be taken up with study, and I think it's a bad thing if this is so. I'm very keen, when I say that the character of Bart's shouldn't change that it shouldn't become a purely academic institution.

You were thinking then weren't you about the changes that are going to be made in the pre-clinical course, I mean the introduction of a three year B.Sc. course for everyone?

Yes. That's with reference to the introduction of the B.Sc. course. Basically what was on my mind was that in order to get students of a sufficiently adequate intelligence to manage their course one would have to cut out certain kinds of students that we have at present who are not purely academic but have other interests as well.

This new course may lead to the selection of a type of student which could alter the present character of Bart's.

Yes, yes, I agree with that. This was a possibility that one could see arising. I raised the point with the Dean and he was well aware of this and had obviously thought about it a long time before I had.

Can you tell us about the association of the Students' Union with the Junior Hospital Doctors Association since this is going to cost the Union money?

It is going to cost £30 p.a. en bloc. We wish to know what policies are being put forward by the Association as we would obviously be affected by them when we qualify. Also we have student representation on the Council of the J.H.D.A. and this provides an opportunity for students to put forward their views about the policies which should be adopted by the J.H.D.A. The J.H.D.A. is a pressure group and acts as it sees fit. It will first of all use the formal channels and if it is felt that this is not sufficient it will find some stronger way of putting its views forward, for instance in the Press.

Does this mean that the J.H.D.A. will be more concerned with our future than our present or are doctors and students going to act collectively?

Yes, it has been clear from the start that they would not concern themselves with student affairs except where student affairs affected them.

Can we ask about the finances of the Union? What is the total income, how is it allocated?

We haven't got the figures yet for this year but in 1968 the total income was £7,414 2s. 8d., of which subscriptions from students represented £6,602 3s. 1d., the rest coming from investments and so on.

The grants are allocated to the various clubs and societies according to the number of people in the club, the amount of interest taken, and expenses incurred in running the club.

You are the Chairman of the Students' Union, you are also Verger of the Vicarage Club, a club which appears, at least to some of us, to be a secret society. Are these two positions compatible?

I think the two are entirely compatible. They are so far apart. They do not interfere with each other at all.

What are the aims of the Vicarage Club?

There are approximately twenty-five student members of the club at any one time and once elected you are a life member. People are put up for election by members and are voted for by the students and only students can be elected. The students are responsible for running the club. There is one aim, which is to provide an opportunity for people who have left Bart's to come to social functions, say three or four times a year, and renew acquaintance with other members of the club.

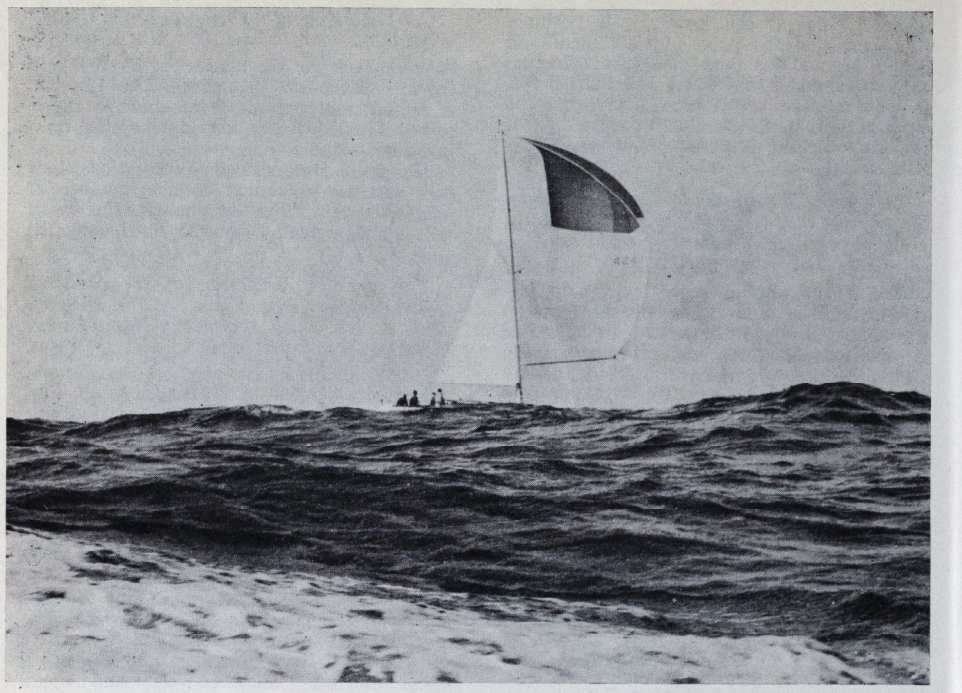
Does being a member confer a special advantage?

No. How could it? This was something that was never in the mind of the people who started the club. Really what you mean is does it confer some privilege, some kind of contact between the members and the staff. Yes, it does in that we now have two members who are consultants. Surely this is not a bad thing. It has never been in the mind of the Vicarage Club that it should provide a staircase by which you can rise in the medical profession.

But why is it so restricted?

Because the members like to feel exclusive.

INTERVIEWERS: J. BURMAN
J. HALL



Atlantic Swell

More and more people take up sailing every year and from among the student population at Bart's are to be found sailors of all degrees of ability and experience.

As the season draws to a close, here are three people's personal accounts of their experiences at sea this summer.

OCEAN RACING
THE FASTEST RACE

An ocean race is defined as more than 250 miles in the open sea, and ocean racing bears little resemblance to dinghy racing. Much the same physical stamina, concentration, and teamwork are required day and night for up to a week, and without boats in close contact to judge ones efforts at sail trimming by. On the other hand, although there is little close racing, there is the exhilaration of driving your boat to the limit of its ability in every kind of weather.

The navigation involved is more than knowing where you are, and the tactics of ocean racing are to be in the right place at the right time to take advantage of currents and tides. Hours may be gained or lost by gambling on a change of wind, by a combination of hunch and the BBC shipping forecasts (respectively!). Experience is vital in knowing which combinations of sails are optimum for the changing conditions of wind and sea.

The 605 mile Fastnet Race is acknowledged as the classic of the ocean racing season, and the most testing course for both boats and crew. With all the prestige attached to doing the Fastnet, most people secretly hope for one of the early August westerly gales that occur while the British traditionally take their holidays. This year no fewer than 189 boats from 20 countries set off from Cowes under a dark electric sky in pouring rain. Once through the Needles, the navigators start calculating the tides that will hold them up and sweep them past Portland Bill, Start Point, the Lizard, and Lands End. One attempts to arrange ones tacks so as to be close inshore while the tide is favourable, and well out to sea when the tide changes. The wind is never as predictable as the tides though! Then comes the real essence of the Fastnet—the 160-mile passage to the Fastnet Rock off the south-west coast of Ireland, and usually dead up-wind. Once round the rock, the fleet heads for home with every imprudent scrap of canvas set. The crews can dry out and relax a little, and with the easier motion, some will be glad of the chance to keep a meal down. From the Scillies to Plymouth, the Scandinavians and Americans are again bewildered and infuriated by our tidal phenomena, and even at this stage the race is frequently won and lost.

Compared to straight courses such as the Bermuda Race or the Sydney/Hobart, it is easy to see why the Fastnet Race is the greatest race of all, and why crews come from all over the world to compete in it.

A very complicated handicapping system embraces everything from the smallest boats, about 25 foot and with a crew of four, up to several boats about 70 foot long and with crews of around twenty. With tactics, weather, and luck all playing their part, every boat is in with a chance,—this year the winner got home on corrected time by a margin of only 68 seconds, after five days at sea. I was on "Zulu" which is fast becoming outdated compared with the latest water-tank designed boats, but we still race against our own regular rivals.

There is phenomenal prestige at stake for the designer of the winning boat, and eternal fame for every member of the crew,—every yachtsman remembers who won the Fastnet.

PADDY SMYTH

A CRUISING AND COASTAL NAVIGATION COURSE

Why a course at all? We had both done quite a lot of dinghy sailing and one of us at least had read extensively on coastal navigation. Our original idea had been to charter a yacht of about twenty-six feet. However, after wading through a pile of literature about chartering in England, Scotland and Ireland, we realised that unless we could put some supervised navigational experience on paper, the cost would be prohibitive. The cheapest charter companies obviously have the most stringent insurance requirements.

Several more piles of literature later, we chose Plymouth sailing school, on purely economical grounds. The course lasted six days from Sunday morning until the following Friday night, at a cost of £30 each, including full board. It consisted of day cruises, starting at about 9.45 a.m. and finishing about 5.00 p.m. with a picnic lunch at midday. We slept in a small hotel not far from the school.

The boat was a four ton Kestrel which is twenty-two feet long. Our instructor was a very experienced sailor who had originally started the school. In fifteen years he had never been late back from a cruise and that had involved sailing through some very heavy weather. There were two other people on the course besides ourselves, who were fortunately more interested in the ride than in the course so that we had the instructor more or less to ourselves.

As it was the end of the season the boat looked even older and more battered than it really was. The instructor was also rather tired but with some vigorous prodding he taught us a lot. Each day we plotted the course we planned to take, made allowances for tides and drift, and repeatedly checked our position with compass and sextant. After the first day this became harder rather than easier because the spinner of the patent log disappeared overboard. The line had finally worn through and it was not replaced during the week. This meant that we had no means of knowing our speed but we managed to persuade ourselves that this was better than having all the navigational instruments to hand.

The cabin was excessively cramped and plotting our position in a rough sea required a strong stomach and a high level of gymnastic ability besides a knowledge of navigation. Although neither of us had been sea-sick before,

ten minutes below deck was more than enough. In fact some very blue cheese for lunch was too much for one stomach and the sight of that person collapsed over the lee rail did not help the rest of the crew. The boat itself was badly balanced with far too much weather helm which means that in a strong wind the helmsman needs to be built like a second row forward. It is bad enough trying to steer a compass course for the first time in any sort of boat, but in her, especially running, it was well nigh impossible. However, we felt that once we had mastered her, everything else would be sheer heaven! Another thing we shall expect in heaven is a guard-rail or at least a life-line. On the Kestrel we had neither. It was pretty hair raising reefing down the main or changing the jib as the wind got up. As it was, only one person went overboard and that was as he was trying to cast off the mooring while standing on the fore-deck. As we moved away, he was left dangling from the bollard. Our only other objection to the boat was the lack of a loo. After a day surrounded by all that water, the boat was tied up very rapidly in the evening.

On reflection we might have gained more experience if we had spent another £10 each and gone on a five day cruise on a bigger boat. This would have let us put our navigation to the ultimate test of being out of sight of land for a day or more. As it was, we were never more than twelve miles off-shore and only lost sight of land for a few minutes in a mist. We would also have been able to do some night sailing. Sailing into a strange harbour in daylight can be quite difficult as we found out trying to get into Newton Ferres in a force 6. At night it can be almost impossible with the lights of the buoys lost in the lights on the shore.

However, I doubt whether a five day cruise would have been such an enjoyable holiday. When you cannot choose your companions aboard a small boat there is a lot to be said for returning to dry land every evening. The weather was beautiful and the occasional shower makes no impression on a good set of oilskins.

The coast of Devon and Cornwall is a wonderful sailing ground and we probably learnt more from the boat's disadvantages than if it had been a more efficiently run course. In fact, it was a very successful week which combined a good holiday with a lot of useful instruction.

Gill and John Durham

SPORTS NEWS

*

receiving a bumper which hit him on his head. The Bart's batting was careless and consequently suffered under the locals' guile: the total score was 74 (Shepherd 24).

Due to some solid drinking and dubious singing resulting in much anger from the landlord round the corner, the fixture has been kept for next year.

CRICKET CLUB REPORT

The annual tour to Sussex took place during the first week of August. Based in Brighton, four matches were played against the surrounding villages. Our stay this year was somewhat shortened in duration due to various opponents changing their cricket weeks, and although the results were not those expected, a very enjoyable time was had by the whole party.

The opening match was against Ferring on Sunday, August 3, 2 p.m. at Ferring. Result: Lost by 62 runs.

Unfortunately, various people arrived at Ferring rather early, even though they had enjoyed various ports of call and so found their way to the local before the game. Hence the bowling and fielding was not by any means faultless, indeed it must have been very amusing to watch, Ferring were dismissed for 136 at tea.

Purcell and Shepherd opened for Bart's and soon the former had to leave the field, after

Mr. L. J. Chalstrey, M.D., F.R.C.S., has been appointed consultant surgeon to Hackney group of hospitals and senior lecturer in surgery, the Medical College of St. Bartholomew's Hospital.

On Monday, we played St. Andrew's at Burgess Hill. Batting first on a hard wicket, Bart's soon lost Leach, but then Purcell and Furness put on 112 for the next wicket. Purcell, having weathered two very shaky first overs then settled down to score his third century of the season. Furness scored 42 before being caught at long-on. At tea, we declared at 216 for 7 (Purcell 122).

St. Andrew's replied with 160 all out, so giving Bart's a 56-run victory. Rhys-Evans produced some incredible bowling, of very varied pitch, height and direction, even back-spin to take three wickets for 12 runs.

Tuesday found us in Rottingdean on the familiar "horse-shoe" pitch but this time sporting a new club house. Our hosts elected to bat first, and lost their opening batsman to Edmondson's first ball. The home team found it difficult to dig themselves in, and suffered under Berstock's "late-swingers". They struggled to 70 all out, Berstock 6 for 14.

Bart's soon found that perhaps the pitch was not going to favour them either; Purcell being bowled first ball also. H. Newton (ex-Sussex) was responsible for this, and his pace had the whole team in trouble. We were dismissed for 59 (Newton 6 for 17), and so lost by 11 runs.

In the following beer match, however, Bart's proved their worth. Festivities continued in the club house, and then the annual trip to the "Plough" resulted in the usual manner. Once more, we must never go there again, especially the captain.

The final match was played against Sir Geoffrey Todd's XI, at the King Edward VII Hospital, Midhurst. The home team batted first, and scored a very slow 127. Armstrong convinced us that he can turn the ball, and took three very good wickets.

After suitable pauses for nourishment and swims, Bart's set out to score the runs. The chase lasted a very exciting 105 minutes, and failed by 1 run. We were dismissed for 126, Sloan scoring 37. This is always a very enjoyable match, and Sir Geoffrey certainly is to be thanked for his generous hospitality.

And so the tour ended. For the first year, a fines system was instituted, and some members paid dearly for their dropped catches: perhaps this should be used more often to encourage players to keep awake!

Tour Party: D. Berstock, J. Shepherd, D. Edmondson, P. Furness, G. Purcell, P. Rhys-Evans, E. Rowland, D. Sloan, R. Page, B. Armstrong, S. Leach, K. Jones.

Sunday, August 10th vs. Harpenden, away. Lost by 61 runs.

A much weakened team visited Harpenden and the home side batted first, scoring 185 for 4 declared. Barts replied with 124, Purcell 65. Essex Tour, Sunday, August 17 vs. Arkesden won by 3 wickets.

Unfortunately Clavering cancelled the Saturday match, due to the harvest. On Sunday, August 17, we played Arkesden. The home side batted first, and scored 150 runs, D. Husband taking 5 wickets for 41 runs. Bart's reached the required total for 7 wickets, Purcell scoring 58, and Berstock 39.

Sunday, August 23 vs. Hill End Hospital. Away. Lost by 13 runs.

In the final match of the season, the home side scored 155 for 8 declared. Armstrong bowled his off-spinners well, taking 5 wickets for 58 runs. Bart's replied with 142; Burdett, in a guest appearance, scoring 57.

So the season came to a close. The final matches were not outstanding successes, but as a whole, the season was good. Winning the Junior Cup, and being the beaten finalists in the Senior Cup, are no small achievements. Although varying in strength sides were fielded, a good performance was usually given.

The final results and averages were:

Played: 34. Won: 16. Lost: 14. Tie: 1.

The batting averages were headed by G. Purcell who scored 1,078 runs.

D. Berstock produced the most consistent bowling, taking 75 wickets.

Batting (10 inns. completed)

	Innings	out	Not Highest score	Runs	Ave.
G. Purcell	30	1	122	1,078	37.2
P. Furness	25	2	76	714	31.0
R. Firmin	14	1	52	330	25.4
C. Reid	14	3	43	251	22.8
D. Lindsell	14	0	77	289	20.6
D. Sloan	14	1	59	240	18.0
D. Berstock	28	4	44*	420	17.5
D. Husband	17	3	57	242	17.3
E. Rowland	15	4	47*	171	15.5
J. Shepherd	13	3	24	130	13.0
S. Leach	17	4	27	107	8.0
R. Page	11	1	30	78	7.8

Also batted: E. Lloyd, K. Jones, B. Armstrong, I. Hann, P. H. Rhys-Evans, N. Offen, S. Kelly, L. Reddington, K. Burdett, C. Varton, A. Burke.

Bowling (10 wkts.)

	Overs	Maidens	Runs	Wkts.	Ave.
C. Varton	96	31	283	23	12.3
D. Berstock	376	100	985	75	12.9
D. Husband	136	30	405	30	13.5
E. Lloyd	114	28	286	16	17.9
E. Rowland	143	42	387	21	18.4
D. Edmondson	243	64	675	34	19.9
K. Jones	68	11	269	12	22.4

Also bowled: P. H. Rhys-Evans, L. Reddington.

John Shepherd.

RUGBY CLUB REPORT

BART'S v WOODFORD
23rd September, k.o. at 6.00 p.m.

Due to the late k.o. only 35 mins. each way were played. This was probably just as well for the first game of the season. An open game was played from the start by both sides. A very fast first half ended with the score 0-0. Bart's were tired at this stage.

Woodford scored first, a try far out on the right as they finally beat the cover. Mason taking the first overlap. The Bart's pack went back continuing to take the lineout ball at 3 and 5. The front row were working well and the loose ball was being gained. It was from a succession of loose balls that Bart's scored. Poustie, playing a good game at fly-half, came back inside and linked up with Boatman who sent Carroll over in the corner. Rhys-Evans was unlucky, with the kick as with an earlier kick from 50 yards. Lambert's leadership and change of direction was impressive as was Smith's speed off the mark. Packer, playing well at full-back, fielded the ball well and opened the game up on several occasions.

In all it was a fair result and both teams were well satisfied with the result 3-3.

Leach apparently enjoyed watching the game from wing-forward, where he played with great endeavour.

Team: N. Packer, P. Rhys-Evans, S. Smith, R. Lambert (capt), P. Mundy, J. Poustie, J. Wellingham, A. Boatman, N. Fairhurst, P. Furness, W. Grant, J. Carroll, S. Leach, A. Mason, C. Higgins.

BART'S v. TROJANS
September 27, 1969

This was an away game and Bart's warmed up very slowly. Peter Griffiths was injured after twenty minutes and had to leave the field. A very disorganised scrum failed to gain much ball in the tight and very little in the line-out. A. Mason tried hard and covered a great deal of ground. The deprived three-quarters were given very little chance to shine, which on occasions they showed they could. Cassidy failed with a couple of penalties.

In fact, a combination of injuries—R. Hill had to leave the field in the second half—and bad play, together with an unfit scrum led to a sad first Saturday of the season. Packer at full-back saved a possibly larger score against us. The result was 23-Nil to Trojans.

Team: N. Packer, S. Smith, D. Jefferson, R. Lambert (Captain), P. Griffiths, B. Cassidy, R. Hill, M. Skidmore, N. Fairhurst, P. Furness, A. Boatman, J. Carroll, C. Higgins, A. Rowsell, A. Mason.

AXV v TROJANS
Away – September 27, 1969

A scratch "A" side put up a very good performance in the first half against Trojans and, in fact, were in the lead until shortly before half-time, when they went 8-6 down. The second half did not, however, see play in the same vein and Trojans eventually finished 24-6 up.

SUNDAY, September 28, 1969
OLD ASKEANS
SEVEN-A-SIDE TOURNAMENT

The team consisted of:—

A. Mason, S. Leech, S. Fairbairn-Newton, C. Grafton, R. Lambert (Captain), S. Smith, N. Packer.

The First Round was against Old Dunstonians 1st VII. Bart's gained plenty of possession and this enabled Smith to score an individual try, which was not converted by Grafton. A further half-break by Lambert was backed up by Mason, who kicked ahead, whereupon Lambert dribbled on for Fairbairn-Newton to touch down. This time Grafton hit a post. The half-time score was 6-Nil to Bart's.

In the second half Dunstonians scored a goal but afterwards Smith again broke clear for Leech to convert.

Bart's won 11-5.

In the Second Round lack of possession led to Bart's losing against Old Colefians, who scored straight from the kick-off and were eventually held to an 8-nil win.

An enjoyable day was had by all.

Book Reviews

Principles of Human Physiology (Starling and Lovatt Evans); 14th edition, edited by Hugh Davson, D.Sc. and M. Grace Eggleton, D.Sc., M.R.C.S. J. and A. Churchill Ltd., London, 1968, p. 1627, fig. 914. Price 120/-.

"Principles of Human Physiology", soon to be more familiarly known as "Starling", first appeared in 1912 and quickly established itself among the leading textbooks of physiology in the English language. It remained the single-handed work of Starling himself until his death in 1927. The task of preparing subsequent editions then fell to Lovatt Evans who at about this time left the Chair of Physiology at Bart's to become Jodrell Professor of Physiology at University College. With the advent of the thirteenth edition in 1962, "Starling" became the product of a team of contributors, the ever-increasing rate of growth of knowledge making the burden excessive for any single author. However, a link with Bart's still persists, since the sections on circulation and respiration are now written by Professor Daly.

In this latest, fourteenth, edition Principles of Human Physiology maintains its established position as the most comprehensive yet well-balanced textbook of physiology published in this country. That it succeeds in combining these qualities reflects both on the skill of its editors and the fact that the contributors are not only leading figures in research in their own fields but in the majority of instances have widespread experience of teaching, especially at the undergraduate level. Though the book is not aimed primarily at medical students, being fuller than the requirements of the average 2nd M.B. course demand, the emphasis throughout is on human physiology, and a more thoughtful appraisal reveals how much of the more detailed physiology constitutes the necessary basis for many of the recent advances in medical and surgical techniques. This increased application of physiological knowledge to clinical situations is exemplified by new or largely revised sections on acid-base balance, the ECG and its abnormalities in disease, changes in the work of the heart in myocardial ischaemia and infarction, the distribution of pulmonary blood flow to different regions of the lung, lung surfactant and the respiratory distress syndrome in newborn infants, hyperbaric oxygen therapy, and cardiac arrest and resuscitation by closed chest cardiac massage and electrical defibrillation. Equally apparent is the growing involvement of physiologists in the problems raised by competitive sport and by man's attempts to exploit those regions of the earth rendered inhospitable by their climate or altitude. Thus this edition includes new material on cardiac responses and the application of Starling's Law to the heart in exercise, on circulatory adjustments in hypoxia and diving, on acclimatisation to altitude, on accidental hypothermia in walkers and climbers and on the use of controlled hyperthermia to produce acclimatisation prior to exposure to heat stress.

Though of less immediate application, but almost certainly of immense potential significance, is the wealth of data emerging from the use of microelectrodes in studies of the central nervous system described in this latest edition. These have greatly aided in the interpretation of records of the electrical activity of the cerebal cortex particularly in regard to the nature of inhibition within the cortex and the role of interneurones. As a result of the use of this technique, principally by Eccles, great strides have also been made in our understanding of the mechanism of cerebellar action. Similar microelectrode studies of single units by Hubel & Wiesel have demonstrated the manner in which the retinal message is integrated in the lateral geniculate body and the striate cortex. Advances are also described in the identification of possible transmitters at synapses within the central nervous system, among which acetyleholine and monoamines are prominent. These findings are of significance in the pharmacological treatment of diseases such as Parkinsonism and psychological disorders.

The introduction of so much new material has inevitably necessitated an increase in size of this latest "Starling" over that of its predecessor. The number of pages has grown by 86 to 1627, though to some extent this is due to an increase in the number of figures from 851 to 914. However, this modest increase in the length of the text has only been achieved at the expense of some pruning. The loss of the section on statistics will not be regretted. In the previous edition this was too brief to be of much use and so condensed as to be unintelligible to the uninitiated. Opinion may be more divided on the fact that the basic biochemistry of proteins, lipids and carbohydrates and their metabolism are no longer included, and more particularly on the omission of the greater part of the description of the vitamins. While biochemistry has for many years now had the status of a subject in its own right, neglect of its origins in and close relationship to physiology is to the detriment of teacher and student in both disciplines. The omission of these topics, while doubtless justifiable on grounds of space and economy, inevitably emphasises this tendency and will therefore be viewed with mixed feelings.

One feature of this new "Starling" which remains unchanged is its format. However, with so many pages the book has become difficult to hold in the hand. More importantly, the binding now seems unequal to its task and several pages of this reviewer's copy fell out after only brief use. Perhaps the time has come to consider the use of larger pages and double columns in order to reduce the book's thickness. Such minor criticisms do not however detract from the prediction that this new edition of "Starling" will be held in the same high regard as its predecessors, and the present team of contributors are to be congratulated on so ably continuing the high standards set by Starling himself.

N. Joels.

●

"Experimental Physiology" 8th Edition. Edited by B. L. Andrew, D.S.c. Published by E. and S. Livingstone Ltd. Price: 42s.

Dr. Andrew has successfully filled a glaring gap in the literature available to both teacher and student at the 2nd M.B. level. The book is basically a laboratory manual describing a wide range of procedures which are suitable material for experiments or demonstrations and it introduces many little known variations of the more popular experiments.

A modern approach has been systematically adopted throughout, for example, the account of the storage oscilloscope will interest those who wish to perfect the demonstration of electrophysiological phenomena but it should have been pointed out that the use of local circuit television is essential if the face of a 10cm. cathode ray tube is to be adequately seen by a large audience.

The student will find that the understanding and use of electronic techniques has been ably simplified, even so, it is gratifying to note that the descriptions of methods involving the use of traditional apparatus show a practical awareness of the pitfalls that the student meets with.

The section on plethysmography is particularly stimulating, laying emphasis, as does the book whenever possible, on human experimentation so helping to bridge the gap between pure and clinical physiology which should aid the student to regard the pre-clinical course as a venture that is something more than an academic exercise.

The chapter on muscle and nerve is commendably arranged but students will enjoy a higher degree of success with the frog if they use the alternative "short" sciatic-gastrocnemius preparation which requires a dissection of the sciatic nerve from the thigh muscles only and leaves the skin over the gastrocnemius muscle intact.

The chapter on respiration is particularly good, combining as it does a relevant sample of experiments with a succinct appraisal of the Gas Laws. It is questionable, however, whether the experiment on progressive hypoxia has any place in a practical course because irregularities in the e.c.g. may occur before cyanosis is observed. This finding is related to the fact that the oxygen content of a spirometer may be reduced to 4% during rebreathing thus rendering the experiment as potentially dangerous. The description of the paramagnetic and infra-red analysers which are used for the rapid and accurate analysis of O_2 and CO_2 gas samples respectively is noteworthy inasmuch as these instruments eliminate the drawbacks of the laborious Haldane procedure; but such apparatus is best kept out of the hands of the average 2nd M.B. student; like a lot of the sophisticated apparatus described by Dr. Andrews these machines give satisfactory results only when they are in the hands of an experienced operator.

It is suggested that the chapter on the cardiovascular system would have benefitted from the substitution of the tortoise heart in the descriptions of the Llangendorf-type preparation and the experiment on Starlings Law. The tortoise heart is robust, easily dissected and its slow rhythm, unlike that of the rabbit, enables the

student to more easily appreciate the mechanical events of the cardiac cycle. The anaesthetised mammal can be used to demonstrate a much wider range of responses than those described, carotid clipping for instance is a simple procedure from which the student may learn a lot about the control of the cardiovascular system.

Although, in a book of such scope there are bound to be omissions, for example, the use of a nasal spray containing A.D.H. to demonstrate the hormonal regulation of urine output, Dr. Andrew has produced a book whose content and format will appeal to the student (the use of blank pages for notes, however, is not to be recommended). It is a book that the teacher should consult and it is a welcome improvement on any work to be found on the bookshelf.

R. W. Willmott.

●

Clinical Pharmacology (Dilling) 22nd Edition. Ed. by S. Alstead, J. G. Macarthur, T. J. Thomson. Balliere, Tindall and Cassell; pp. 760; price 50s.

The treatment and prophylaxis of disease are advancing so rapidly that it is difficult for a textbook on clinical pharmacology to be up-to-date by its time of publication. This volume is an exception and one of its strongest points of recommendation is that it is as up-to-date as any textbook at present available. For some years, I have recommended it as the textbook of choice in this subject because if a student reads it from cover to cover he will not only have learnt his clinical pharmacology but a great deal of general medicine besides.

One criticism must be that it is heavy reading, requiring concentrated effort. More figures and diagrams would, I think, be of value and help to retain the interest. It is difficult to see how structure-activity relationships of drugs can really be discussed without giving some formulae as examples. Another minor criticism is the strange location of reserpine for discussion under drugs acting on the heart, when other antihypertensive drugs are discussed under the autonomic nervous system. In general there is not enough discussion on basic mechanisms of action of drugs but when one considers the size of this volume and its reasonable price it would be difficult to decide where to draw the line with further discussion. Once again, I think that this is the textbook of choice for general reference in clinical pharmacology for the medical student.

Paul Turner.

●

"Modern Trends in Obstetrics" Edited by Professor Kellar. Published by Butterworth and Co. Ltd., 1969. Price £3 4s.

This book is the fourth in a series of deservedly popular works on modern trends in Obstetrics. Professor Kellar has carefully edited, as usual, authoritative articles by multiple authors on subjects which illustrate the growing edge of scientific knowledge in relation to human reproduction. No one planning to take a higher degree in this subject can ignore the content of this book with impunity. Even established gynaecologists will find to their surprise that although the orientation of the articles is scientific, the subjects discussed are intensely practical and information forthcoming is clinically useful. Such a man will find that his "hunch" in relation to the cause of spontaneous abortion is confirmed. The "blighted ovum" of his earlier days is now found to be due to a malshuffle of chromosomes. The losses of human pregnancies from this cause alone are reckoned to be as high as the total perinatal loss.

The importance of the interdependence of the foetus and its own placenta is emphasised and studies of one without the other meaningless. From a philosophical point of view it is interesting to note that, although the mother provides environment for development, the foetus in utero depends for its ultimate salvation on its own placenta which belongs to it and not to the mother. Left wing socialists please note.

Anyone who has any knowledge or the slightest sympathy for the professional activities of our colleagues who work in relatively underprivileged countries, will be fascinated by the chapter on obstetrics in India by Dr. Krishna Menon. He writes from a professional lifetime of experience and considering the problems that he has had to face in obstetrics, consultants in our National Health Service with all its deficiencies may regard themselves as fortunate.

Donald Fraser.

ROAD TEST

by RALPH SMALLHORN

1. Rolls-Royce Silver Shadow and Bentley T-Series.

It is proposed in the coming months to feature road tests of cars of interest to our readers. To start the series we decided on a test of the car which most regard as the pinnacle of the British Motor Industry—the Rolls-Royce Silver Shadow—and Rolls-Royce were kind enough to allow an afternoon with one of their cars.

The Silver Shadow and its Bentley cousin are powered by a V-8 cylinder 6230cc engine made almost entirely of aluminium. Transmission is now fully automatic and the specification includes power steering and independent suspension all round with a triplicated power assisted disc brake system.

The first impression is that one is about to step into and drive off in a car costing some £8,148 19s. 2d. and that even one corner might cost a year's grant to repair.

Our Shadow was beautifully finished in dark maroon, suiting the new styling which can best be described as modern Traditional. The doors open smoothly and close with that satisfying coach built "clunk".

Once installed one is immediately confronted by the long wide bonnet crowned by the flying lady, symbol for so long of high quality motoring. The steering wheel is large by modern standards and the latest fascia panel incorporates crash padding, fewer dials, more warning lights and a large legible speedometer. The interior can best be described as sumptuous. The seats are leather, extremely comfortable, and the front ones are adjustable in all directions including up and down. In the back, leg room is considerable and individual reading lights (to choice), ashtrays and cigarette lighters are supplied. Curiously placed

mirrors are on the rear quarters above ones shoulders . . . possibly for greying temples. It is with much regret that I announce the passing of the picnic trays. These were in the rear of the front seats and are also victims of the great American safety crusade. Although of doubtful value they were supreme examples of the craftsman's art. They are replaced by large pockets for Vogue and Hansard (or Lancets?) The test car had a refridgerated air conditioning plant (£189 6s. 1d.) which undoubtedly renders the electric windows valueless.

The engine is barely discernible on tickover and in fact, only when accelerating hard, is anything heard at all. Suffice it to say performance is more than adequate with 115 m.p.h. top speed and 0-60 m.p.h. in some 11 secs. To cope with this a braking system which crash stopped from 70 m.p.h. produced no drama except the speedometer needle dropping like a well-shot pheasant.

The ride is soft at the expense of some roll when cornering enthusiastically and there was some tyre roar on abrasive surfaces. The power assisted steering seemed rather vague in the straight ahead position. However this is largely a matter of what one is used to. Directional stability was of the first order.

The gear box on the test car has now been superceded by a General Motors gearbox, which if it is better than the Rolls Royce one, as we were told it is, must be unbelievably good. All gear changes except under full acceleration were imperceptible; one grumble was a rather slow acting kickdown, possibly our reservations stopped us pushing hard enough!

Prospective owners apparently ask the m.p.g. figures which are 14 m.p.g. on a 24 gallon tank (with low level warning light), giving a range of 360 miles. In conclusion, for those with gold bath taps and heated loo seats, this is an essential, for the rest, a dream.

The sort of criticisms one has are petty in the extreme, but should a car costing as much as a house or ten Minis have any faults? The standard for future tests has now been set, albeit a very high standard. By the way, the clock doesn't even tick,—it's electric.

Saint Bartholomew's Hospital

JOURNAL

contents

Vol. LXXIII No. 12 1st December, 1969

journal staff

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EDITORIAL

The number of scientific papers published each year increases phenomenally. Librarians must already be aware of the problems so created, whether of space or finance. There are journals whose rate of growth, if continued at the present tempo, will, in a few years time, cause them to be accumulating at the speed of light along the bookshelves! Luckily the problem of paying for all this print arises earlier, before such a catastrophe could occur. It would seem, however, that the writers are less aware of the chaos they are creating than the poor librarians. Despite the appearance of a new profession and new publications which endeavour to sort out, digest and reissue the facts in a more critical and balanced form, it is becoming increasingly difficult to have heard about all the *important* experiments and papers in any particular field. In the search for useful information one is too often thwarted by the publicity hounds who smother the journals with articles often more or less reprints from several other journals and containing perhaps one new observation buried somewhere in the central pages. The dangers and stupidity of any system of reward and promotion based on quantity of matter published are obvious. We must therefore search for better methods of controlling the number of specialist articles going to press and of keeping everyone well informed of advances in their field and of all important general developments.

The latest attempt by the B.M.S.A. to keep the country's medical student body informed appeared this month (October). In newspaper form, with vivid red motif carrying name *Scope*, it successfully covers a range of subjects from medical politics to aids to buying a second-hand car.

In the *Bart's Journal's* attempt at communication, though we do expect it to be less widely read than *Scope* and though we shall generally leave politics to the B.M.S.A., we try to reflect everything in work or leisure which goes on in and around the Hospital and Medical College.

One important local item to "reflect" immediately is the appointment of the first woman editor of this *Journal*. The *Journal* also took note of the recent government announcements on equal pay and is happy to be able to offer both editors (honorary) equal remuneration! We wonder when other honorary bodies like the Vicarage and Cambridge Bart's clubs will take a similar step and allow the gentle sex into *their* privileged communities.

ANNOUNCEMENTS

Engagements

DEAN-LANKESTER—The engagement is announced between Dr. Richard Simon Dean and Miss Judith Lankester.

HOPKIN-HOWLETT—The engagement is announced between Dr. Graham Owen Hopkin and Miss Rita Janice Howlett.

Births

GREEN—On August 27, to Sheila (née Minns) and John Green, a second daughter (Hannah Mary).

ILES—On October 6, in Sidney, to Gillian (née Walker) and Dr. David Iles, a son.

WOOLRYCH—On September 23, to Ann (née Taylor-Marsh) and Dr. Michael Woolrych, a daughter, sister for Thomas, Jonathan and Caroline.

Deaths

DANIEL—On September 16, Dr. William Raymond Daniel, M.R.C.S., L.R.C.P., D.A., aged 47. Qualified 1947.

GILBERTSON—On September 28, Herbert Marshall Gilbertson, M.R.C.S., L.R.C.P., aged 82. Qualified 1912.

LAWN—On August 4, Dr. John Arthur Ernest Lawn, M.R.C.S., L.R.C.P., aged 61. Qualified 1933.

PUSEY—On October 22, Dr. John Harrington Pusey, M.B., B.S., M.R.C.S., L.R.C.P. Qualified 1963.

YOUNG—On September 22, Dr. Frederick Hugh Young, O.B.E., M.D., F.R.C.P., aged 77. Qualified 1915.

Appointment

Drug Control Committee

Prof. E. F. Scowen is to become chairman of the Committee on Safety of Drugs, to succeed Sir Derrick Dunlop.

Mr. L. J. Chalstrey, M.D., F.R.C.S., has been appointed consultant surgeon to Hackney group of hospitals and senior lecturer in surgery, the Medical College of St. Bartholomew's Hospital.

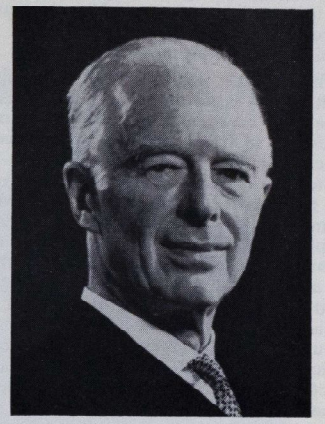
Change of Address

Dr. and Mrs. J. M. Robinson have moved to 154 Knightscroft, New Ash Green, Dartford, Kent.

OBITUARY

DR. F. H. YOUNG, O.B.E., M.D., F.R.C.P.

Freddie Young died in Bart's after a short illness on September 22, 1969, and a whole host of friends and patients will be sad that he is no longer with us.



Frederick Hugh Young was born in 1892. He was educated at Shrewsbury School and Trinity College, Cambridge, obtaining his B.A. in 1913. He then came to Bart's, qualified by L.S.A. and promptly entered the R.A.M.C. Serving in Salonika, he attained the rank of Captain, was mentioned in despatches three times and was appointed O.B.E. On his return to London at the end of the war, he obtained his M.B., B.Ch. in 1920, his M.R.C.P. and D.P.H. in 1922 and his M.D. in 1924. He soon became interested in diseases of the chest and in 1922 was elected tuberculosis officer in charge of the Corporation of the City of London tuberculosis clinic at Bart's. This consisted of two out-patient clinics a week, which he personally conducted for the next forty years. He was extremely well informed upon the medical and social aspects of all his regular patients; he came to regard many of them as friends and the relationship was reciprocated. He kept his notes meticulously, in a rather illegible handwriting. He was delighted to have been a member of the Medical Council during his last few years at Bart's.

In 1925 he was appointed assistant physician to the Charing Cross Hospital and two years later became dean of the Medical School. He rapidly established himself as one of the leaders in chest medicine, but unfortunately in 1929 he became ill with pulmonary tuberculosis. He

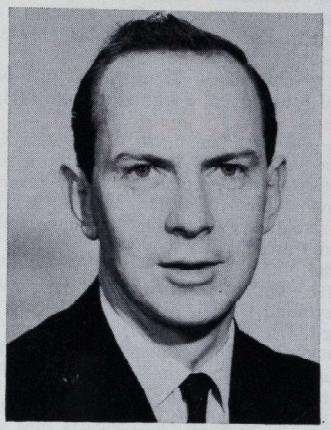
made a good recovery and resumed his increasing commitments, but a relapse in 1934 forced him to curtail his activities. He reluctantly resigned from Charing Cross Hospital, being appointed consulting physician.

Whilst Bart's was in many ways his first loyalty, there is no doubt that his professional interest was based upon the Brompton Hospital. Starting as a house physician at the country branch, Frimley, he eventually became senior physician, a member of the Board of Governors and, for several years, chairman of the Medical Committee. In this last capacity, he had the complete confidence of his colleagues and, by means of painstaking thought and consultation, was able to maintain a friendly efficiency combined with progress during his term of office. In his years at Brompton he saw the treatment of pulmonary tuberculosis pass from bed rest, artificial pneumothorax and thoracoplasty to resection and chemotherapy. During the stage of medical and surgical collapse therapy he was probably the best tuberculosis physician in the country. His skill was based upon clinical observation and an ability to balance the socio-economic aspects with the medical needs rather than upon scientific acumen. His large private practice, much of which was taken up with pneumothorax refills, bears testimony to the excellence of the results he was able to achieve.

Freddie was a member of the Association of Physicians of Great Britain and Ireland and was awarded the Parkes Weber prize in 1963 for his contributions to the investigation and treatment of pulmonary tuberculosis. He was president of the Thoracic Society, perhaps the highest honour that his specialist colleagues could confer upon him. Indeed, quite apart from his professional attainments, his bearing and appearance added distinction to any gathering. Some of his patients may have thought him to be austere, but they rarely disobeyed his instructions, as many of them may have been tempted to do in the days when tuberculosis was treated by prolonged bedrest and graduated exercise. His house physicians could hardly fail to gain from the training he gave them in method and judgement, although some of them might have preferred a little less insistence upon orderly routine and attention to detail, and a little more opportunity for critical discussion.

Freddie was a cultured and somewhat reserved man, and a keen golfer of moderate ability. He was blessed with an extremely happy family life, and our thoughts at this time must be with his charming wife, son and daughter.

JOHN HARRINGTON PUSEY, M.B., F.R.C.S.Eng., F.R.C.S.Edin., D.Obst.R.C.O.G.
Born: January 19, 1940.
Died: October 22, 1969.



John Pusey graduated from Bart's in 1963. He will be remembered as a quiet and much liked student who worked hard but left ample time for a number of other interests amongst which were rowing and the managing of this *Journal*.

In addition to his post in the Obstetrics and Gynaecology Department at Bart's he has held, since qualification, a succession of appointments within the Wessex Region becoming a Fellow of the Royal College of Surgeons earlier this year. It is particularly sad that his death should come at a time just before his return to his old hospital as Registrar within the Orthopaedic Department.

Anyone who knew John both before and after graduating was impressed by his kindness and thoughtfulness over small matters that might otherwise have passed unnoticed but meant so much to those concerned. He died as a result of a shooting accident while relaxing in the country of which he was so fond.

John had a bright future ahead of him and Orthopaedics, in which he had a particular interest, has lost an able surgeon. His friends will miss him but it is to his family and fiancée that our sympathy is mainly extended.

CONSULTANT OPINION

What they think of us...

Three of the consultants who teach first year clinical students here present their views on us, the students.

What a tempting ball to hit! "Please write between 300 and 600 words on what you think of the students." I can call them all the names they deserve. They are in no position to make reprisals, and surely would not dare to have a return match. Lazy, spoon-fed, illiterate, unenterprising, unpunctual, moaners without critical faculty or constructive ability, insolent, arrogant, disloyal. All these things they are. Try lecturing to them: prepare it afresh, get out the appropriate slides, make a few new ones, look up some references, ring up a Stockbroker to get the latest funny story, and then when you arrive find that only a handful of students are there, and that another handful clatters in late without thought of apology, only to settle down noisily to their usual noughts and crosses.

They are so lazy that they are prepared to go all the way to Whipps Cross to see just one patient; so keen on being spoon-fed that they commonly ask quite impossible questions; so illiterate that the Pot-Pourri and the annual Smokers are amongst the best entertainments of the year; so unenterprising that they travel to America, to Yugoslavia, to the Arctic, to Rhodesia in order to meet more stimulating teachers; so unpunctual that every now and then they get to Medical Out-Patients before I do; so without critical or constructive thought that some of the recommendations of their Teaching Committee are going to be acted upon; so insolent that my wife declares she has never yet met a Bart's man she dislikes (interesting, those Firm parties: in my day we would never have thought of spending money on our Chiefs); so arrogant that a Senior Administrative Officer at Whipps Cross declares he prefers them to the products of any other School; so disloyal that special security precautions have to be taken during and after Cup matches.

They vary greatly, of course. And they infect each other, whether with enthusiasm or with boredom, whether with surliness or with good humour, so that some of the Firms we have are thoroughly tedious and hard work and one does not feel like bothering with them at all, and yet other Firms can be so bright and stimulating that they are a delight to teach and one could happily take them on every day of the week (assuming one knew that many words).

I choose to believe (having no proof, and desiring none) that our Medical Students are in general more intelligent, more mature, more sensible, and, despite the obviously etiolating effects of their grants, just as interested in their work as in my day. Very few indeed are, or ever have been, of top flight intelligence—double-firsts and the like—for medicine does not attract such people. (Fortunately it seems to get by pretty well without them, and it is dubious if it would know what to do with them if it met them). On the other hand it mysteriously attracts those people who at the age of 12 know that they want to do it, and at the age of 65 are intensely reluctant to give it up.

Two sixteenth century proverbs: "Young men think old men fools and old men know young men to be so." But: "Young men may die, old men must."

And the reason for all this? It can only lie in the inexpertness of student selection during the years 1960 to 1963 when I was Sub-Dean.

H. Wykeham Balme

My views of Bart's students are based on my contact with them during the past 18 months. It is difficult enough to generalize on this kind of topic, but my very limited experience makes my task impossible.

The students with whom I have had to deal have been charming, polite and have contributed very much to the work of the wards and also to the teaching. They may not always know whether Howell-Jolly bodies are present in Bagdad Fever, but they are kind and considerate to the patient and are interested, not only in their immediate operative treatment but also the management of the patient as a whole, the social problems and how their relatives are dealt with. They seem to me much more aware and concerned about many ethical and social factors than possibly earlier generations of students were.

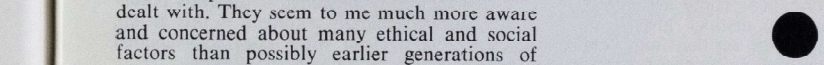
The attitude to lectures is probably right, and those who arrange the lecture courses have in fact listened sympathetically to their views and altered the curricula accordingly. Lectures should deal with some of the controversial subjects or areas where there have been recent advances and where the overall picture is somewhat confused. On the whole they feel that lectures should provide something that text books do not. They favour symposia and a tutorial system. On the surgical unit the tutorial system seems popular, not only among the students, but among those who conduct them. This Medical School is part of a University and it is important to encourage them to think and solve the problems rather than to have information pumped into them. Education implies a leading out rather than a stuffing in.

Bart's students have been accused of lacking a certain amount of enthusiasm. This has not been my experience. We have had no trouble at all in getting students to come at 7.30 or 8 a.m. in the morning for tutorials regularly every week. Where there appears to be a lack of enthusiasm all that is required is a bit of spark to light some latent fire.

There is one field where I find it difficult to understand their point of view. It is difficult to reconcile their wish to be given more responsibility with their reluctance to take blood from patients and make sure that the blood samples arrive at the correct destination. A student who wants the experience of putting up an intravenuous infusion but resents taking the blood over for cross matching to the laboratory seems to me to have a strange sense of values. The acceptance of responsibility inevitably includes the acceptance of some of the more mundane and menial tasks.

Bart's students are as friendly and charming and co-operative as any I have met. They do not lack enthusiasm and the ability to work hard. Their performance would probably be improved with a tutorial system and progressive assessment throughout their clinical years. At the end of every appointment the registrar and both consultants should independently assess each student.

I. McColl



A reply to a request for one's views on today's student and how he compares with those of previous generations is bound to be coloured by one's own age and the memory of one's own attitude as a student. A very fundamental difference between the students of my generation and those of today is that all the students today start on their University career having been indoctrinated with the belief that they are entitled to free University education as long as they wish; it is therefore everyone else's responsibility to make this time an enjoyable one with as much profit as possible. Most of the students of my generation were dependent on their parents whom they knew to be making sacrifices in order to provide their offspring with a good career.

I think on the whole that a student's attitude is fairly consistent throughout the whole of his career. When I was a demonstrator of anatomy immediately before the 1939 war I felt that one's contact with the students was very close. Though there was a constant dread on their part of failing their vivas there was nevertheless a reciprocal respect between students and teachers. There are several members of the senior staff today who were victims of my teaching at that time whose qualities as students were quite outstanding—they could have been picked for success at that stage. After an absence of six years on active service I returned to Bart's in 1946 and my impression was that the students on the whole had changed very little. However, during the following decade there was a remarkable change in attitude of students generally with the advent of the fully

developed welfare state, the universal application of the grant system to students and the increase of student intake. When I became Dean in 1957 I had the opportunity of seeing all the students at the beginning of their career and I think if one knows students over a long period of years one forms a very different impression of the individuals. It must be said that the majority of students are intelligent, enthusiastic, loyal, honest and reasonably consistent. Perhaps I might be allowed to classify the students. One might describe the good qualities of the students as being congenital or acquired. Some are clearly outstanding from the start in every way, others are very weak at the start but finish with an outstanding performance. There are nevertheless in my view four distinct groups of students. First, there are the brilliant men and women with a high intellectual capacity and a good upbringing who know how to use their opportunities to the full and never cause any trouble. They are easy to converse with on any subject.

Then there is the less brilliant group who are conscientious and hard working, but who need help and encouragement at times though rarely requiring rebuke or discipline—the average student.

Third, there is the dull student who has been exhausted by the pre-clinical course and discouraged by his relative inability to cope with the torrents of scientific jargon with which he has been presented. This is the student who might have been helped by a more realistic tutorial system in the pre-clinical years, or by the curriculum being pruned of irrelevant material. Such students were helped in former days in that the Anatomy Department demanded a full and detailed dissection of the human corpse. This provided long periods of relaxation and social intercourse around the coal fires of the old dissecting rooms and provided an opportunity for contact with junior teaching staff who were actively engaged in hospital practice. I am sure that the almost complete separation of pre-clinical from clinical staffs today is a very retrograde development which is to be made worse if the recommendations of the Todd Commission for London University are carried into effect.

Fourth, there is the clever student who is lazy, irresponsible, not correctly motivated, defiant of authority with little sensitivity to the needs of other people-in fact immature. I believe that this group of students is increasing numerically because of the lack of discipline and the confused curriculum which is broken up into many periods of short duration. This type of student is able to get away with a very minimal attendance, neglects his routine responsibility in clinical work and expects his deficiencies to be covered by his fellow students. The distorting factor in such a student's life may be sport, love, debt or a combination of all three. When he does turn up he is pleasant, convincing, full of excuses but clearly not the sort of person one would want as a family doctor. He may already have taken shelter in the idea of a future in one of the Armed Forces. Perhaps it would be kinder if the College took action and got rid of these students who will never be any credit to the profession. It is of course very easy for seniors to misjudge a student's potentiality and occasionally a student in this group, when put in a clean white coat and labelled House Officer, turns out to be superb.

When one comes to consider the main bulk of the students in the first three groups there is no doubt that they are much better educated generally with wider background information than were the students of my own generation. They are more adventurous, better travelled, perhaps more ambitious. These qualities have largely developed as the result of progress in communication, thorough mixing of communities and races, and a general levelling of society. These students are obviously less respectful of seniority or age. Conversely senior staff I think find it easier to talk with students on an informal level, than did our predecessors. Consultants are still far from infallible but now this fact is recognised—even by the consultants!

I am asked to say whether I think the student of today is generally idle and apathetic. The answer clearly is that today's student is neither idle nor apathetic if he falls into one of my three first groups, but that there is an idle group cannot possibly be denied. I think it is up to the fellow students of these academic loafers to stop doing their work so that at least the authorities may find out who they are before they reach their final examinations. It would be wrong to say that today's students have any faults apart from those of previous generations. There was a period, post-war and in the early fifties, when students' dress was careless, usually untidy and often dirty; whether this is no longer so, or we have got used to seeing a different type of dress it is difficult to be certain, but on the whole I would say without reservation that Bart's students are as well turned out as any in London. Much of the apparent sloth, apathy and inattention of students is I am convinced induced by the educational

system in which they find themselves, partly because there is no discipline or compulsion, and partly because the curriculum is now so confused it is not possible for anyone to check on students' movements. As a classicist I found my introduction to science in the pre-clinical years exciting. Now students start on their science curriculum at 10 or 11 and there is very little intellectual adventure in the crowded "A" level and pre-clinical course. I believe I am not alone in saying that I very much regret that I do not see more of the students. The recent change in the curriculum giving up the early full 3-month periods of attachments to specific firms made it extremely difficult to get to know any group of students properly. Until the recent introduction of an examination at the end of the first clinical year there has been no standard or milestone against which a student can assess his progress. War has to be waged from the 2nd M.B. to the final examination without any knowledge as to how the battles are going. This state of affairs may well be corrected by the proposed system of progressive assessment.

In conclusion, whatever may be said about the student or the educational system, there is not the slightest doubt that after qualification the first few months may be a bit sticky, but at the end of the pre-registration year the vast majority of our graduates are far better informed than we were at the corresponding time. There is of course much more to learn, and the knowledge of any student is spread over a wider field. One cannot therefore expect the depth of knowledge in any particular department which some of my generation may have had at a corresponding period of their career. Unfortunately today's students had their picture of medicine distorted by 20 years of grumbling on the part of many members of the profession but it is significant that a very large number of students come from medical homes where clearly the picture of the profession has not been painted too drab. In spite of years of broken political promises, committees, working parties and commissions and much frustration, there is something enduring, stable and pretty constant—the quality of product of our medical school. I had the privilege of regulating the entry of students for five years and had little to regret!

D. F. Ellison Nash

STUDENTS UNION

ANNUAL GENERAL MEETING

The Annual General Meeting of the Students' Union was held in the Clinical Lecture Theatre on October 30.

Elections were held and the following appointments were made:

President:
Mr. J. O. Robinson, M.D., M.Chir., F.R.C.S. (*no election necessary—two-year term of office*)
Treasurers:
Dr. A. G. Spencer, G.M., M.D., F.R.C.P.;
Professor O. J. Lewis, M.D.;
Mr. I. P. Todd, M.D., M.S., F.R.C.S.
Chairman: Mr. M. G. Britton.
Hon. Secretary: Mr. P. J. Simpson.
Financial Sec.: Mr. P. M. R. Millard.
Asst. Hon. Sec.: Mr. T. Fenton.
Vice-Pres. Clubs Union:
Mr. R. G. Lambert.
B.M.S.A. Vice-Pres.: Mr. D. Stringer.

Year Representatives:
1st Year Preclinical—
no nomination.
2nd Year Preclinical—
Messrs. Dickenson-Hennessy and Henderson.
3rd Year Preclinical—
Messrs. Wall and Dehn.
Women Preclinical—
Miss W. Mansi
B.SC.—none (less than 25 students)
Dental—no nomination.
1st Time Clerks—Mr. M. Knowland.
1st Time Dressers—Mr. B. Cassidy.
Kids and Psychiatry—
Mr. B. Grimaldi.
Mid. and Gynae.—Mr. N. Packer.
Finalists—Messrs. Rymer and Hunt.
Women's Clinical Rep.—
Miss J. Hall.

Wine Committee: Messrs. M. Navin, R. Lambert, S. Leach, C. Yates, M. Knowland, D. Wilkinson, J. Carroll, D. Jefferson, M. Skidmore.

Teaching Committee: *A vote was taken but after lengthy discussion it was decided that voting procedure should first be improved and the election of the committee was therefore postponed for a few weeks.*

At the meeting, the following reports were amongst those given:

CHAIRMAN'S REPORT 1968/69

This has been a busy year in the Union and I would like to outline the main events as briefly as possible.

1. **Constitutional Amendments:**
Various amendments were proposed at the A.G.M. last year and several of these were adopted after careful consideration. The main changes were:—
(a) A B.Sc. Representative if there were over 25 B.Sc. students;
(b) Alteration in the election of the Wine Committee to ensure continuity of members;
(c) Canvassing by candidates for Union Council and Executive.
2. **Refectory:**
After much discussion, the student canteen in the Hospital will be closed in the near future and, as an alternative, the Lay Staff Canteen will be made available for our use.
3. **Journal:**
Owing to continued financial difficulties, the grant by the Students' Union to the *Journal* was increased from 1/- to 1/6 per student. It seems that as a result of this the *Journal* has been able to break even over this year.
4. **J.H.D.A.:**
Following moves by certain London teaching hospitals, we are now affiliated to this organisation: the object of this is to ensure continuity between students and junior doctors by giving us some representation on the J.H.D.A. Executive.
5. **Damage in College Hall:**
Owing to the alarming rise in damage ocurring in College Hall early in the year, various measures were introduced in an attempt to reduce this. So far these measures appear to have been reasonably successful and it is hoped that they continue to be so.
6. **Nuffield Foundation Bursaries:**
Following a generous grant from the Nuffield Foundation, it is possible for 14 students in one year to be given £25 each for the purpose of visiting other medical schools within the United Kingdom, with the object of increasing the contacts between students.
7. **Diving Club:**
This Club was formed in July and a substantial sum of money was given by the Union to the Club for the purchase of equipment to be used in a Nitrogen Narcosis Experiment in Scotland, and subsequently for training purposes in London. The experiment appears to have been successful and a report is due to be submitted.
8. **Teaching Committee:**
The Teaching Committee this year has met regularly and has discussed many topics. It has been instrumental in putting forward students' views to teachers, both Preclinical and Clinical. The Teaching Committee provides an excellent opportunity for staff and students to meet to discuss differences and it is hoped in the future to invite members of the staff to a larger proportion of meetings.
9. **Resignations:**
We are very sorry to hear of the resignation of Dr. Arthur Jones after only one year as Dean. During this time he has shown a

great deal of interest in student affairs and we are very grateful to him for the effort that he has put in on our behalf. We wish his successor, Dr. Malpas, every success. Also this year Laurie and Mrs. White have retired from Chislehurst. A party was given in their honour, at which they were presented with a substantial cheque, made up of donations from past and present students. They have been succeeded by Mike Carruthers and his wife and we wish them every success and happiness.

Finally I must thank Mr. Robinson and the Treasurers for their support throughout the year and the interest they have shown in the affairs of the Union. I would also like to thank Mr. Morris for once again keeping a watchful eye on our accounts and for his help and advice in many matters. My thanks are also due to Mark Britton who has worked hard and efficiently as Secretary, Peter Simpson as Assistant Secretary and Chris Hunt as Financial Secretary, and all others who have been involved in Union matters. It just remains for me to wish the Union every success in the coming year.

Richard Page,
Retiring Chairman

REPORT FROM THE TEACHING SUB-COMMITTEE FOR THE YEAR 1968-1969 TO THE ANNUAL GENERAL MEETING OCTOBER 30, 1969

At the last A.G.M. in 1968 Tony Newman-Taylor (Chairman), John Burman (Secretary), George Lodge, Roger Rolls and Oliver Smales were elected to the Teaching Committee.

On the committee this year we have had the help of several co-opted members who deserve our thanks. They are: Elisabeth Macdonald, Jonathan Rendall, Paul Dieppe, and with pre-clinical affairs Paul Millard, Primrose Watkins and several others. But our thanks must go particularly to Dick Page who has shown a great interest and has been very active in representing our views to the Dean.

Last November the Teaching Committee produced a report on the Royal Commission on Medical Education (Todd report) and its relevance to teaching at Bart's. In this report the Teaching Committee made a number of recommendations including the recommendation that house appointments should be for six months instead of a year.

The report was well received and the Committee discussed it with a number of members of the staff of the Medical College and the Hospital. During the year the Committee held meetings with: Mr. Bourne, Prof. Quilliam, Dr. Franklin, Dr. Balme, Dr. Malpas, Prof. Taylor, Dr. Spencer, and Mr. McColl, and on a number of occasions, Dr. Jones and Mr. Hill. We received the impression that the Teaching Committee was being listened to and that some of its ideas met with agreement.

If there has been any progress it is that the Teaching Committee now gets a better hearing and that many of the more recently appointed consultant staff are beginning to express more clearly their interest.

The pre-clinical committee has been active. Paul Millard and Primrose Watkins conducted a survey of teaching in Charterhouse and published their findings in the *Journal*. The pre clinical committee has had discussions with the heads of the pre-clinical departments. It is thought that the pre-clinical committee should continue to work as a separate committee.

Over the year there has been close co-operation with the *Journal* and it has published a number of articles by members of the Committee. The present editorial staff tells me that it hopes to continue this co-operation.

The B.M.S.A. organised with the Teaching Committee a symposium on teaching at Bart's and people from Bart's attended a national conference on medical education organised by the B.M.S.A.

We have the feed-back from members of the staff but reaction from students has been discouraging. Until students as a whole show interest and involve themselves in discussion with their teachers and their representatives on the teaching committee there will be little progress. In future it is hoped that the meetings of the Teaching Committee will be more widely publicised and that everyone, both staff and students, will feel free to attend and take part in the discussion.

J. F. Burman,
Secretary
of the Teaching Committee

INTERVIEW : THE DEAN

DR. J. S. MALPAS D. Phil., B.Sc., M.B., B.S., M.R.C.P. WAS APPOINTED DEAN OF THE MEDICAL COLLEGE on 1st. November 1969

Dr. Malpas is Senior Lecturer in Medicine at Bart's and Consultant Physician to St. Leonard's Hospital. From 1964-1965 he was Deputy Director of Clinical Studies at the University of Oxford.

What do you consider to be the most important aspects of being Dean?

I think the most important aspect of the Dean's work is obviously to help to produce good doctors. This is undoubtedly the prime duty of the Medical College. It is very difficult to particularise how this should be done. How one can enable this to be done is the whole process of being Dean.

Do you think it is a question of selection? What sort of students do you want to admit and how do you decide if they are going to be the right people?

I was going to get round this very difficult question by telling the story of the Head of an Oxford college who was asked how many students were in his college. He thought for some time and then said about 10 per cent! I thought that what I would like to do was to admit 100 per cent. Presumably by the second part of this question you mean whether they are going to become good doctors. I don't think anybody has found the answer to this. Various methods have been tried but I don't think anybody really knows who's going to make a good doctor.

You say it's impossible to say who is going to make a good doctor. Do you have any criteria of your own which you are going to apply?

I can only point out some of the ways in which other schools have been going. For instance, the Edinburgh Medical School has not interviewed candidates for the last five years. There appears to be no deterioration in the doctors being produced by the Edinburgh Medical School.

Have they gone purely on academic achievements?

I think selection has to go almost entirely on academic achievement and in the majority of cases the "A" level results or equivalent are really as sound a test of academic ability as can be achieved in an imperfect world.

What about the Head's report? Is this important?

This may help, particularly I think, if you know the Headmaster concerned.

Will you, then, admit students primarily on academic achievement?

I think one always does if one is honest. This must in the long term be the major reason for admitting a person.

Although in the past at Bart's one has got the impression that this may not have been exactly true.

Yes, I think it might have been just the impression. You tend to remember the particular instance. I don't know about this. I haven't been involved with the admission of students at this college until recently.

Are you happy that enough people are applying?

Here again I can't tell you the figures though, of course, many more people apply than we have places for.

The numbers of students applying from Oxford and Cambridge to come into the clinical part of the course have gone down, especially in the last year. This year I think there were only four, which does seem rather a shame.

I'm not sure of the exact figures but I quite agree with you it is a pity that there are not more.

I would like very much to encourage, as far as is possible, the return to a reasonable entry from both the older universities. This is going to be more difficult in the future as one of these has got a very flourishing medical school, the other is proposing to undertake clinical teaching so you may see even fewer people applying to London anyhow.

Could we ask you a bit more about being Dean? You have mentioned admitting students as part of this job of producing good doctors but you have to deal with all sorts of other authorities as well, the University, the University Grants Committee for example. Do you have any idea relatively what parts of your job will be taken up with this kind of work?

Of course, this is a very essential part of a Dean's work but I haven't any idea yet as to how much time will be spent on one or the other.

The other question was whether you consider the Dean is more of a servant or a leader. Are you going to be able to apply your own ideas?

I don't see the Dean's job as fitting into either of these categories. I think such a complex task as providing a very stimulating course depends on co-operation. It is possible to give a lead on the introduction of new teaching techniques perhaps, or helping to make alterations in the curriculum but these really would have to be introduced in a spirit of co-operation. I think that the job of the Dean is that of helping and co-ordinating the various departments that are going to provide teaching.

How much co-operation with the University does this involve?

Well, I think it is going to involve more and more. I say this because the implementation of the Todd report recommendations will inevitably mean a great deal more discussion and planning both with the University, with Queen Mary College and with the London Hospital.

We have heard that it has been proposed that there should be a three-year pre-clinical course at Bart's in the future. Is this going to be introduced?

I think this question of a three-year pre-clinical course is one of the things that has interested a lot of people. You know the Todd report recommended that there should be a three-year pre-clinical course leading to a first degree, whether it is called B.Med.Sc. or B.Sc. Amongst other reasons this was thought necessary because a number of people who wished to take a variety of biological subjects to degree standard didn't necessarily want to go on to do clinical medicine. The Commission now suggested that a wide spectrum of course modules be offered for a first degree.

The next part of the course is, if you like, a clinical general education as distinct from a vocational training. It was one of the main ideas of the Todd Commission that this should be distinct from a vocational training and so it is possible if you cut out the idea of trying to produce an all round doctor at the end of three years to cut down the clinical course to two years. One still envisages a five-year course.

Are we going to have such a course at Bart's?

I think it is very likely that it will come through eventually. But I don't think that we shall see this developing until such time as we can implement the whole of the Todd recommendations or something similar to this.

Would it be followed by one year's pre-registration as it is now or would that be expanded to two years to complement the cut down in the clinical course?

I think the vocational training programme is still under discussion and of course depends on a number of variables which at the present time it is very difficult to determine. I don't think that there is a move to extend the so-called pre-registration time although I'm sure that the total time spent in the initial training up to and before the entry to the specialists register will be somewhat longer.

If the Todd recommendations are going to be implemented what will be the first step? Will it mean an amalgamation of the medical schools?

I know people are very worried about this term amalgamation and the possibility that we shall lose autonomy. I think if you look at the paragraph in the Commission's report, paragraph 435, you will see it doesn't amount to loss of autonomy. A dreadful term "twinning" has been used, and in this "twinning" they envisage the provision of new departments, and the provision of facilities which one medical school or one hospital by itself could not afford to provide. I think it may also help us with some problems that already exist in established departments. It would, for example, enable the facilities for paediatric teaching to be improved. I think that twinning is inevitable and I personally would welcome this. I look forward very much to co-operating with colleagues at the London Hospital and I think that we could in this way help considerably to improve the course. Whether you have to have this twinning available immediately before you start the new Todd course is very much open to discussion. I think it would probably be best worked out on an *ad hoc* basis first.

Can you say how far things have gone with discussions about twinning?

They have started.

Has anyone thought about a date?

I don't think anybody has given a date. I think it would depend very much on what is offered by the University Grants Committee in the next quinquennium, beginning in 1972.

Many people feel the need for a system of tutors meaning a body of people prepared to look after students throughout their course and to whom the student can turn for advice on academic and personal problems. The need is pressing because students feel anonymous when they first arrive in Charterhouse, they often have doubts as to the relevance of certain parts of the course, there is a high failure rate in the second M.B. B.S. examination and towards the end of their course students are in need of guidance concerning exams and careers. Would such a system help and would you like to see it introduced at Bart's?

I am very well aware of this feeling about the tutorial system. To have such a system would be an advantage. I am very sympathetic to the idea. I think I would like to look upon it as an adjunct to teaching rather than in the nature of a so-called "moral" tutor. I really can't go into this further but I will certainly give it very careful consideration.

Are you in general happy with the teaching methods that are employed and will you be able to direct people in what they teach and how they teach?

I am sure I can't do that!

If you look at the appendix in the Todd report on a survey carried out into teaching methods it certainly appears that from a very wide survey, bedside teaching was the most popular. The difficulty is, of course, the increasing numbers, and one has got to make sure that the amount that a student can get from bedside teaching is not hampered by firms becoming over large.

Do you think the responsibility of the medical college ends when the student qualifies? Despite the change to six months house jobs here, it is getting harder to find jobs because of the greater numbers of students qualifying.

Yes, I understand this. I personally don't think that responsibility ends there. This is another feature of the Royal Commission on Medical Education that with the implementation of this report there will be a duty on the part of the Medical College towards vocational training and this will be not only just a question of seeing that suitable posts are available but also to some extent advising people who are qualified on where they can best go with regard to say some specialist training programme that they will eventually want to enter into.

Can you tell us how housemen are chosen at Bart's?

I have never actually had to sit on a committee choosing housemen so really I can escape that one.

How much importance do you attach to the views of students about their course? Would you be prepared to see students represented on College committees at Bart's?

I am certainly prepared to listen to what students have to say about the course. I don't think there's any doubt about that. With regard to representation on committees I think there has been considerable discussion in various universities recently on how this can best be done. In summary it seems that there are two ways: either you can have a very small number who sit on the various committees that have existed previously or you can have a separate properly elected student committee or possibly a joint consultative committee of students and teachers to whom the relevant questions are referred and on the whole I think the decision at most universities has been that the latter is the best solution and the vast majority of students have, in fact, found this acceptable.

I should say clearly that I don't think that it is the function of the student body to deal with the final planning of the curricula or indeed with the management of examinations and I think that academic standards must really be the prerogative of the relevant departments conducting the teaching.

You have already mentioned the danger that teaching may become less effective because of increasing numbers of students, yet Bart's must be alone among the London teaching hospitals in having no designated hospitals attached to it at which it can appoint medical staff and nurses and where it can organise

undergraduate teaching. In view of the increasing demands on teaching is this a dangerous situation?

I don't think this question is altogether correct. As you know there is undergraduate teaching going on at two general hospitals: St. Leonard's and the Hackney Hospital, although I will agree with you that these hospitals are not designated as the definition goes. Nevertheless students are being taught there and they are being staffed in medicine and surgery at all levels. There are certainly plans in operation for further development of teaching in these hospitals and in fact if you look at the Wade report you will see that we have a number of hospitals that are very closely associated with us with regard to the provision of pre-registration jobs. We don't lag behind other hospitals in this respect and as far as undergraduate teaching is concerned we have well over a thousand extra beds available for undergraduate teaching between the two hospitals concerned.

Do you think these other hospitals are being utilised to the full?

Not yet. But they will be and will need to be.

Will a more formal arrangement be made to extend the influence of the teaching hospital over these hospitals or is this a gradual matter of co-operation?

It is impossible to say. What we want is undergraduate teaching and this is what we are getting.

From the point of view of the people running the units there are all sorts of disadvantages in not having a formal attachment, the training of nurses and so on.

Yes, but this doesn't really impinge on student training all that much. This is more a problem for the provision of services.

Are you going to be able to keep up with your other interests now that you are Dean or did you have to decide that you would have to, say, do less research?

I think it can be answered simply, I don't know yet. I will certainly try and keep all my activities going because I think that only if you are involved in teaching yourself and the other problems of clinical medicine can you really be of help to your colleagues.

Do you do most of your teaching at St. Leonard's?

Yes. Most of my teaching is done there.

Could you just tell us how much teaching you do at Bart's?

This really amounts to teaching informally in out-patients and also some more informal postgraduate teaching, tutorials and so on, but I don't actually have a teaching round at Bart's. But then I do have at least two full mornings teaching at St. Leonards.

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"A Factual Study of The Clinical Facilities Available for Medical Education in London. Couitenay C. Wade 1967

INTERVIEWERS: J. BURMAN
J. HALL

MACCABAEAN PRIZE AND MEDAL

Entries for the 1970 Maccabaean Prize of 25 guineas and a bronze medal are now invited for an essay of 5,000 to 7,000 words on some aspect of the history of medicine or pharmacy. Intending candidates, who must be under thirty years of age on March 15, 1970, may apply for further particulars to the Honorary Secretary of the Faculty of the History of Medicine, Dr. F. N. L. Poynter, The Wellcome Institute of the History of Medicine, 183 Euston Road, London, N.W.1.

A CHORAL CONCERT

given by

ST. BARTHOLOMEW'S HOSPITAL CHOIR

and

ST. PETER'S CANTATA PLAYERS

will take place in

SOUTHWARK CATHEDRAL

on December 10th at 7.30 p.m.

Mozart Programme:

"Davidde Penitente" K.V. 469
"Song and Vespers" K.V. 339
Ave Verum: "Jesu, Word of God Incarnate"

Tickets: 7/6 (students and nurses 5/-), from members of the choir or at the door.

The Interaction of Automation and Chemical Pathology

by

P. G. Sanders M.A.,M.C.B.,A.R.I.C.

Department of Chemical Pathology

University Departments of Chemical Pathology have a number of functions. These include the teaching of undergraduates, the post-graduate training of doctors and biochemists wishing to specialise in the subject and the participation in the training of technicians. Research, both fundamental and methodological, is undertaken as well as the provision of a routine and specialised analytical service. This last function is by no means the least, since the aim is to help the clinician and thus the patient. Few patients would agree with this at the time a blood sample is being drawn!

The workload in Departments of Chemical Pathology all over the world increases by an average of 15% per year. As this is an exponential growth curve the number of analyses doubles every five years. At Bart's, 23,000 tests were made in 1954. The number doubled by 1959, and rose eightfold in the 14 year period to 1968 when 180,000 were performed. In 1969, despite severe limitations in space during the reconstruction of the Pathology laboratories, a 30% increase is expected, so that the quarter of a million mark is likely to be reached. Perhaps this figure is more readily appreciated when translated to 1,000 investigations per working day. The rise comes partly from an increase in the frequency of requests from clinicians and partly from the proliferation of multiple simultaneous requests. It must be remembered that the introduction of new tests often leads to an increasing complexity in the work, as well as an increase in numbers. Without economies, this increase would have involved a quite prohibitive expenditure. At present-day rates, salaries for the Chemical Pathology Department would have totalled £16,000 for the year 1958, when 37,000 tests were completed. By extrapolation, the 250,000 tests in 1969 would require a total of some £110,000. In fact approximately one quarter of this sum is spent.

Prior to the late 1950's, an increase in staff was the only method of providing an expanding service, but when the Technicon Company marketed the automated analytical system developed by Skeggs, it was possible to improve the "productivity" of laboratories. Most routine analyses consist of an initial pipetting of the sample, which is then diluted to give a volume easy to handle. Protein is removed by the addition of a suitable precipitating agent and an aliquot of the clear supernatant obtained after centrifuging is treated with suitable reagents, the reaction conditions being chosen to yield a colour whose intensity is proportional to the concentrations of substance. The concentration is measured in a photoelectric colorimeter by comparing the amount of light absorbed by the sample in comparison with that of a standard solution of known concentration.

The heart of the Technicon "Autoanalyzer" system is the proportioning pump. Plastic tubes of specified internal diameter can be stretched across a flat bed and compressed by a series of moving rollers, giving a pumping movement similar to intestinal peristalsis. This can be used to replace manual pipetting of reagents. With suitable glass connections, dilutions and addition of reagent can occur. In this analytical system, the sample is sucked into a plastic tube, and remains in the plumbing of the system until finally discarded. Protein cannot be removed by precipitation, so that dialysis is used to separate large from small molecules. Heating is achieved by passing the liquid stream into a long coil of glass immersed in an oil bath maintained at the requisite temperature. The colorimeters are fitted with flow-through cells into which the reagent flows continuously, so that a continuous record of the light transmission can be read off from a potentiometric strip-chart recorder. This continuous flow system of analysis has been introduced extensively

in Britain. Although Bart's entered the field of automation some time ago, it is only recently that it has been possible to increase the apparatus available so that 95% of the analyses are performed automatically.

Alternative analytical systems have been developed, usually of the discrete type in which reactions occur in individual tubes. With this approach protein removal is somewhat difficult, unless centrifugation is employed. Complete systems of discrete analysis are now on the market but for many years individual steps of routine procedures have been made easier. This has been termed 'work simplification'. Included in this category are automatic pipettes and dilutors, both manually operated and motor driven, colorimeters sufficiently stable to be fitted with flow-through cells and even colorimeters fitted with digitalised output so that a print out of results is possible.

The immediate problem of the early 1960's was the vast increase in analytical work being demanded by the changes in clinical practice. Increased productivity in the form of work simplification and more complete automation permitted laboratories to cope with this workload without a parallel increase in staffing and space requirements. The ease with which extra analyses could be made permitted close investigation of the accuracy and reproducibility of results. In clinical biochemistry the term precision is used to indicate the reproducibility of results, and is not synomymous with accuracy. Thus a technique which invariably produced a result of 33 mg/100 ml. for a blood urea of real value 30mg/100 ml. would be a method of high precision but poor accuracy. To a large extent, the precision obtainable with a particular method is determined by the operator and the quality of the apparatus used. With automatic apparatus, the calibre of the operator has a much smaller effect on the result, so that good design of the machinery leads to improved precision.

Analytical errors can be classified into random and systematic components. Random errors are difficult to recognise, but are fortunately not frequent. Most analytical errors are systematic, and therefore show up when sufficient numbers of results are examined critically. There has been a growing appreciation and use of statistical methods in laboratory work. Good laboratories should be able to quote the precision for each of their analyses, and in certain instances, knowledge of this figure is vital for interpretation of the result. For example, creatinine clearance may be used as one parameter in the assessment of progressive renal damage. Providing that urine collections are made properly, the result is largely influenced by the figure obtained for serum creatinine. If the precision for serum creatinine estimation is ± 0.1 mg/100 ml. then a true figure of 1.0 mg/100 ml. may be reported as 0.9, 1.0 or 1.1 mg/100 ml. With constant urine output, these results may give creatinine clearances of say 110, 100 and 90 ml/min. respectively. Using these results alone, it would be impossible to decide whether deterioration had occurred, as all the results fall within the limits of experimental accuracy. However, at higher abnormal levels of serum creatinine, the percentage error is correspondingly lower, provided that a precision of ± 0.1mg/100 ml. is maintained. The improved precision obtainable when suitable work simplification procedures and automation are used have been demonstrated recently. Surveys of the quality of results in clinical laboratories have been carried out in various countries over the past twenty years. The 1964 survey of about 200 laboratories in Britain made by the Association of Clinical Biochemists gave results that were better than any produced previously, but were still poor. The Scandinavian clinical chemists have been participating in an interlaboratory quality control scheme for some years with constantly improving precision. A similar scheme is now operating in Britain, and as in Scandinavia, the results are displayed prominently in the laboratory for all to see. This scheme enables a laboratory to check both its precision and its accuracy, since the mean result for about 185 laboratories in Britain, together with the standard deviation from the mean, is quoted. The ability to work to much improved standards of accuracy and precision has led to a world-wide investigation of the range of results found for people without obvious illness. This work, still in progress at various centres, is giving a better understanding of the 'normal range' so enabling a more exact interpretation of minor changes of results. This is a vital step in the introduction of biochemical screening for latent disease. Automated analysis has reached the point where a new problem arises. This is the handling of the data necessary to initiate and report the test. In some laboratories, the proportion of time spent by the technical staff on clerical operations has reached 75%, so that methods of reducing this must be introduced. It is often simple to group tests together by combining apparatus to provide simultaneous results. When this is done, it is usually more convenient for the laboratory to measure all the constituents, than to take the clerical steps necessary to omit the un-requested analyses. At Bart's, this technique has been applied to the electrolytes—sodium, potassium, chloride and bicarbonate being measured simultaneously on the same specimen. Calcium and phosphorus form a pair, total protein and albumin another. In the near future, the process may be carried further when total protein, albumin, bilirubin and alkaline phosphatase are measured simultaneously. Indeed, there is now machinery available to analyse some twelve or more components simultaneously at rates of up to 300 samples of blood per hour. These techniques often lead to a reduction in the sample volume required, but may well lead to a problem outside the laboratory. This is the digestion of the vast amount of numerical data received by the clinician. It may be possible in certain instances, to reduce this problem by reporting only those requests falling outside the acceptable normal limits.

The vast increase in data handling, together with the mathematical calculations necessary for quality control systems have led to the introduction of computers to clinical laboratories. This is the most expensive field of development, and is not so far advanced in Britain as it is in Scandinavia. To some extent, this has been brought about by the easier labour market in Sweden, where automation is not as widely applied as in Britain, and the data handling problem has been given priority. Finance appears to be a much greater problem in Britain than it is in Scandinavia. The whole hospital syystem of data-handling is feasible, but involves enormous sums of money, and covers so many aspects of patient records that the problems are legion. The delay in the introduction of such a comprehensive system may be greater than can be tolerated by pathology laboratories, so that individual systems must be developed preferably suitable for later inclusion in the complete system. Most systems in use in Britain have developed "off-line" i.e. with no direct connection between the analytical apparatus and the computer. 'On-line' facilities are now available at a few centres so that colorimeters, fluorimeters and flame photometers can be used to give a digital output suitable for computer handling. The transport of reports to wards, or even to distant hospitals, can be speeded using tele-type machines remote from the laboratory, suitably sited to serve one or more wards.

Integrated with this changing pattern of working, is a perceptable change in staffing. The greater productivity of automation can be applied to estimations made in fairly large numbers, but it is not economic to deal with the smaller batches of analyses or with single estimations. In certain cases it is realistic to send analyses to a regional centre. Protein bound iodine determinations are most successfully automated using the Technicon "Autoanalyser". The apparatus required costs approximately £4,000, and in ideal circumstances, can complete 100 analyses in a normal working day. Most Regional Hospital Boards in England and Wales have set up central units for P.B.I. determinations. There is a growing tendency to centralise laboratory facilities so that one unit serves a total of 2,000 beds or more for a group of hospitals. Automation, together with the introduction of disposable apparatus has removed much of the drudgery from laboratory work, since there is now little washing-up. The staff liberated from work on the bulk analyses are employed on the 10% of manual estimations. It must be realised that this 10% of the work may occupy more than 50% of technician time. In general terms fewer staff are employed, but they need to be of a higher calibre. Technicians of the requisite academic standard are difficult to recruit. Males, in particular, usually obtain grants for University training. At the same time the increasing complexity of the chemistry, together with the growing ability of clinicians to interpret results has led to the partial replacement of chemical pathologists by the paradoxically named 'non-medical' biochemist. (In an official terminology a 'non-medical biochemist' is a graduate scientist who has specialised in the application of chemistry to medicine). Perhaps clinical biochemist is a more reasonable title.

Mechanical aptitude is a great asset for the modern laboratory worker. The screwdriver has replaced the spatula as the badge of office of the biochemist. When will the boiler suit replace the laboratory coat? Some of the apparatus now under development is reminiscent of model railways. Shall we work or play in the laboratory of the future? The massive Swedish "Autochemist" now being installed at the B.U.P.A. laboratory in London weighs 4 tons, and the moving machinery will surely need the "engine driver" with his oil can. This latter machine costs approximately £200,000, a far cry from twenty or so years ago when £100 was regarded as an enormous expenditure on any one item of apparatus.

It was stated earlier that an estimated

£85,000 per year in salaries was being saved by automation. In Copenhagen, where labour problems are different from those in Britain, Professor Paul Astrup is in charge of the clinical chemistry laboratory at the University Hospital. Little automation has been introduced, work simplification having been used instead. His new laboratory in a 1,600 bed hospital will occupy an area of 3,000 square metres—approximately the size of one half of a football pitch. 125 staff are employed—some of them part-time. Approximately 800,000 analyses are completed per year. At Bart's 250,000 tests are made by a staff of 16. Of course, here there has been extensive expenditure on apparatus—about £100,000 on capital items. It is reasonable to expect a life of 7 or 8 years for these items, so that an annual outlay of some £15,000 to £20,000 is required, for maintenance and replacement. As the total hospital budget for this type of expenditure is less than £40,000 per annum, it will be difficult to provide adequate finance without restricting other departments. The solution will be to amalgamate laboratories from several hospitals, allowing greater economy of labour and greater efficiency in the use of apparatus.

Thus, mechanisation of the chemical pathology laboratory has solved some problems, but in doing so has raised others. Greater precision of results has been obtained, but it must be remembered that erroneous results can be produced quite unblushingly by machines. There is an even greater need for both laboratory workers and clinicians to be aware that mistakes do occur. The Chemical Pathology at Bart's, reporting 1,000 results a day, would send out 5 faulty figures if the success rate were as high as 99.5%, and this figure is quite difficult to attain even though perfection is the aim. It is almost impossible to inspect thoroughly all these reports before issuing them to the wards. A 'feedback' from the clinicians is a vital portion of the quality control systems employed. The laboratories need to know when clinicians are worried about the veracity of results, especially when specific examples can be quoted.

The developments in chemical pathology have been very rapid over the past decade. Extension into the field of data handling is the next step. This, together with the advances of quality control, automation, and a better understanding of normal ranges may permit screening of the whole population, leading to a true National Health Service, rather than the present National Disease Service.

ON LIVING WITH A LIVER

Confessions of a chronic tonic drinker

by

N.H.S. 3177857, Patient

Paterson, David C.

"Your liver," said the white-jacketed medicine men clustered around Bed 11, Rahere Ward, "is all swole up and got hobnails on."

I lay there, a tiny tongue surrounded by enormous teeth.

"That a degree of hepatomegalia exists," I replied coldly, "and a coarsely nodular palpation, are phenomena of which I am apprised. And of course the percutaneous needle biopsy one of your fellows with ginger fur on its knuckles brought to a successful conclusion after Sister explained that no, no, it's the *heart* on the left-hand side, was to demonstrate histo-chemically . . ."

What I said out loud was: "I suppose . . . this means . . . cirrhosis?"

"Chronic parenchymal disease we prefer to say reely," said left canine. Reely.

"Or fibrosis or siderosis as the case may be," —this from right incisor—"and there's one thing sure, if there's an 'osis' on the end it must be somefink bad, very bad."

I struggled desperately to find some exception to this monstrously arbitrary generalization. "What about meiosis then?"

"Progressive degeneration of the ego, very narsty indeed," cried right incisor quick as a flash.

There was a general movement towards the intrinsically more interesting case of shingles in Bed 12. I grasped left molar before he could join his cronies and asked him that question most frequently posed to the late Mr. Robb Wilton by his lady wife. Left molar was intrigued. Nobody had ever asked him that question before. He fingered his pink-striped tie.

"*Do* about it," he said, "well I dunno as there's much as we can *do* about it, Mind you, a kidney, summat simple like that, we'd have it out of you quick as kiss-my-arse. Now your liver, on the other hand, your actual liver, well that's a highly inscrootable organ and what we tend to do is, we leave well enough, or what is in your case as you might say *ill* enough, sort of well alone."

His brow wrinkled in concentration. "There's salt," he added learnedly, "that's sodium thingummyide. Sometimes they keep them low on salt, that's what they call a low-salt diet, see? But you get too little salt, not enough salt see? that can well muck up what they call your electric light balance. Same as meat, a valuable source of protein, eat too little and you're back here in a coma, eat too much and likewise. You got to eat exactly the right amount of meat, and nobody knows what the right amount is, not exactly."

He leaned nearer, exhaling fumes of last night's whisky. "And for Christ's sake, mate, lay off the booze. You'll f*****g *kill* yourself!"

Forty-eight hours after this objurgation was delivered, I met my wife by arrangement in a City pub. I ordered two gins and two tonics. My wife drank two gins and one tonic. I drank one tonic. Neat, without ice or lemon. The round came to 8s. 4d. My career as a teetotal, band-of-hope, water-wagon, pussy-foot, judge-sober abstainer and pariah had begun.

I've never been what you might call a heavy drinker, or rather what *I* might call a heavy drinker. Let's say I held an honourable ranking among the cruiser-weights. The sun rarely went down on my drouth. But it was equally rarely that I got drunk enough to do something really foolish, like buying a round out of turn.

However, I freely admit to expert status in the study of alcohol absorption. My credentials might, it is true, be more readily accepted by *Brewers' Guardian* than by *Brit. J. of Prev. & Soc. Med.* Yet three months' sober experience in the field has garnered enough observed data to support the tentative formulation of several analcoholic postulates. The first is the saddest, but they are all sad.

1. **You can't be a semi-abstainer.** It's useless to argue that alcohol in homoeopathic doses —all you get in half a pint of English bitter— can't possibly do any harm. This is figuratively true, but irrelevant. The point is that if you have half a pint with a friend or friends, you will inevitably have several half pints. And if you're on your own, why go into a pub at all?

This proscription is reinforced in my case by a course of unselective reading in the literature of hepatic disorder, which has proved more efficacious than any aversion therapy. I come out in a cold sweat if I touch the *outside* of a stoppered and sealed whisky bottle. A man with whom I was trying to curry favour forced a glass of sherry on me before lunch the other day. For hours I could feel my liver bobbing up and down, a hob-nailed, inscrootable yo-yo.

2. **There isn't a soft drink you can drink.** Not without feeling sick. Ever stood at a bar and swallowed *six* tonics, *three* tomato juices, *four* bitter lemons? Remember the Spanish Inquisition's leather funnel?

3. **There isn't a soft drink you can afford.** Your licensed victualler has it in for the soft-drink brigade. I suppose if you went into an opium den and asked for chewing gum they'd charge you the earth. So you put a splash of beer in tuppence worth of lemonade to produce a glass of shandy, that'll be 1s. 6d. thank you Sir. I've paid prices for tiny bottles of apple juice that would have made the down payment on a fair-sized orchard in Kent.

The agony is compounded when you find

it's your turn to buy a fiver-busting round that nets you 100 millilitres of nauseous, carbonated, sodium-cyclamated swill.

So why go into a pub at all? Alas . . .

4. **More than 94 per cent of all London business is transacted in London pubs.** I'm a journalist by trade. I used to think that we did all our serious work in pubs because we liked drinking a lot. But I confused cause and effect. Lord Malvern (at that time Prime Minister of the Federation of Rhodesia and Nyasaland, but better known as the rowdiest houseman in St. Thomas's, 1906-1909) once told me that there was no difference between two men that couldn't be settled in two leather armchairs over a bottle of Scotch. It remains to be said that quite a number of differences yield less readily to a formica-topped table and two cups of Joe Lyon's tea. This leads us to . . .

5. **Nobody likes a man who doesn't drink.** You can get away with it once or twice by pleading a hangover; even here, the odd proponent of hair-of-the-dog therapy is liable to give you a hard time. What's invariably fatal is the admission that you're off the hard stuff *on medical grounds*. This makes you as welcome as Poe's eponymous Red Death at the masque. You have stirred hidden insecurities that many of your audience are drinking to forget.

This last sentence, with its clinical and contemptuous analysis of the harmless toper, is itself a product of the next postulate.

6. **Nobody who doesn't drink likes a man who does.** When the shambling wreck that two hours before was a witty and cultured citizen throws his arm around your neck and spends twenty long minutes telling you, very slowly and badly, a joke *you told him in the first place;* as you stand there with a frozen smile on your face and a fruit juice in your hand and murder in your heart you will readily understand why every voluntary abstainer in history has been such an obnoxious prig. Spiritual pride is the abstainer's neurosis.

So much then for Paterson's Six Analcoholic Postulates on the social effects of abstinence. To round off my research notes, here are two observations on adverse physical effects.

The average human organism reaches its efficiency peak between 8 and 9 p.m. (This is the converse of the low ebb it reaches in the small hours.) I often take advantage of this to work in the evening. It had previously been my practice to administer a mild alcoholic depressant before getting down to it. In the absence of this medicament, my memory and concentration are showing recurrent lapses.

Can I be the only man in Britain to suffer the manifestations of Korsakov's syndrome *in reverse*?

The other point is that getting drunk at hygenic intervals seems to have a markedly therapeutic effect. The point was made, I think in Huxley's *Doors of Perception*, that things were bloody and getting bloodier. We all need an escape from reality now and then. Huxley advocated the so-called psychedelic drugs, but they have their drawbacks: on the most practical level, you need a laboratory in which to foment them.

There's always pot, of course, which has the added attraction of being illegal. It's also, *ipso facto*, hard to come by and pretty expensive. My G.P. reacts quite violently to the suggestion that he might prescribe it for me on the National Health.

So here I am, firmly shackled to the wheel of *karma*. And here I'll stay until someone proves that cannibis gives you cancer; which will remove the only logical objection to legalising its use.

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The editor would like to say, I think, that those white-jacketed medicine men we met in the first few paragraphs are merely grotesques imported for dramatic purposes. Neither they, nor the third-year medical students on whom they are based, bear any resemblance to any human being, living or dead.

ART EXHIBITION

The Bart's Art Society held an exhibition of paintings in the Great Hall from 7th to 9th October. About 150 *works were shown.*

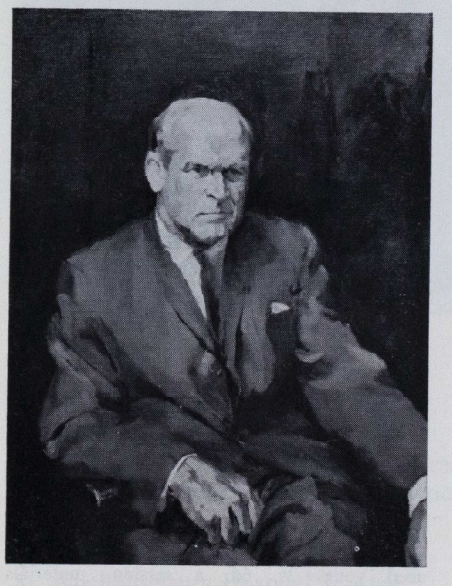
I enjoy going to art galleries, yet I never seem to get to any of the exhibitions in London. Luckily Bart's Art Society recently arranged one on the spot and I just managed to reach the Great Hall and see the exhibition before it closed.

Putting several excellent paintings by Simon Brett at the start of the exhibition set a high standard for the other paintings. Not surprisingly this standard of technical achievement was rarely attained but, though technique may have been lacking, originality, sparkle and the enjoyment of painting were evident.

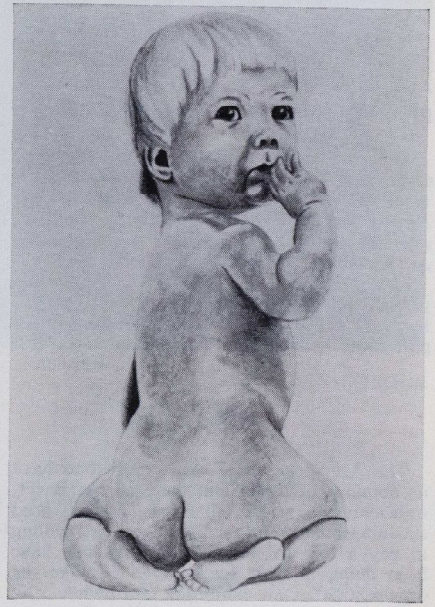
As I wandered round several pictures especially appealed to me. First the lively little kid in two *Childhood Studies* by J. Scorer—how *do* you catch that fleeting, cheeky expression on a child's face? A. B.'s *Durham Cathedral Doornocker* was the first of his exhibits to catch my eye and the heron in foliage near it aroused my envy of those who can portray life boldly and quickly. In contrast the sketch of irises by Y. P. Banks was admirable for the way it captured the essence of these flowers. I noted particularly the dry, wrinkled fold at the base of the flower.

I liked *Early Morning on the Thames* by P. Voak. Reflection on water is so difficult to paint; I thought it had been done successfully in this picture which stirred pleasing memories of boats and rivers.

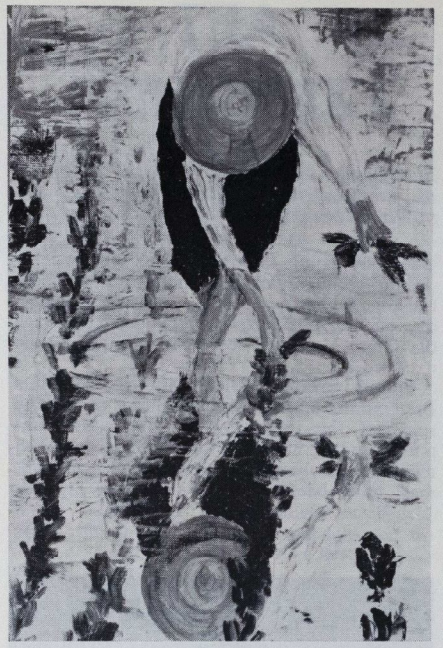
Vietnam by Vivienne Durkee—a drawing of the wizened old face of a war-worn woman—and several other works in the same part of the room showed well the less idyllic side of life.



"The Steward"—Simon Brett



"Childhood Study"—J. Scorer



Above: Irises-Y. P. Banks
Right: The Rice Picker-Alison Corcoran

Others that caught my eye were *The Gamecock* by Ann Gaythorne Jackson, *Still Life in the Studio* by Virginia Phillips, *The Rice Picker* by Alison Corcoran as well as splendid brass rubbings, several fine photographs and coiled copper jewellery.

Congratulations to the Art Society on this year's exhibition; I hope I get to next year's—I should do, so long as I do not walk blindly past the artistic posters advertising it!

C. Reisner.

●

The Art Society was very gratified to have its opinion confirmed that there are, at Bart's, sufficient vain artists (all artists, in fact, need encouragement) and sufficient critics, willing to pay a bob to see what their friends have been doing in their spare time, to make the organisation of an exhibition worthwhile.

We have, however, one suggestion. Everyone would like to see the standard at the Bart's exhibitions maintain its upward trend. We therefore ask the person who knows what he likes and wishes that he could paint too, to consider what he could perhaps do if someone showed him how. The critic we hear cry, "I could do that much better if only I were not leading such a busy, dedicated life and just had the time." We ask him the loaded question, "Is it dedicated to the really important things in life, such as painting?" And we hear the struggling amateur mutter, "Well, I'm not really happy with what I've painted, but my masterpiece is being formulated." We suggest that the masterpiece might actually be painted if he were put back on the tracks and given a smart push in the right direction. One neither realises one's potential nor how little one knows until someone points it out.

The point is this: Art classes are held every Wednesday evening between 5 p.m. and 7 p.m. at the top of Gloucester House: the professional artist appears to live on the bread line and keeps demanding money; and we need yours—2s. 6d. a session, materials supplied!

Graham Kidd, Secretary of the Art Society.

JOURNAL CHRISTMAS CARDS — 1969

"Pool of Bethseda" – Hogarth. **IN COLOUR**

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BLOOD, SALT AND WATER WITHOUT TEARS

Current thought on fluid replacement

by Tom B. Boulton

Consultant Anaesthetist to St. Bartholomew's Hospital

and at the present time to

The Children's Medical Relief International, Saigon

Readers of this *Journal* may well have seen the two excellent cartoons drawn by Mr. Peter Cull, the Medical Artist to the Hospital, which appeared in the May and June issues [1], [2]. There were entitled the "Great Fluid Controversy" and represented the two extremes of currently held theories on fluid replacement for the surgical patient.

The author has a personal interest in this subject for some 30 years ago at the age of fifteen, he suffered, and has never subsequently forgotten the desperate agony of the symptoms of fluid deprivation after having had his appendix out in a cottage hospital. The regime used, which it is hoped would now be considered old fashioned, included dehydration by massive enema, and complete deprivation of fluid from 6 p.m. on the night before operation for over 48 hours after its conclusion. Even today patients are made to suffer a degree of dehydration during the surgical period which not only causes unnecessary suffering but also delays their recovery to a greater or lesser extent.

On the positive side, adequate fluid replacement, besides improving the comfort of the patient, results in improved perfusion, and thus improved function of, the vital tissues and organs. Adequate replacement of fluid loss means vasodilation and adequate perfusion of the microcirculation with consequent avoidance of tissue anoxia and metabolic acidosis, adequate coronary and renal perfusion and function, and the elimination of hypovolaemia, which is the commonest cause of oliguria in the post-operative patient. As a further bonus, satisfactory hydration means that the sputum in the respiratory tree is prevented from becoming viscous and bronchial obstruction and infection are thereby avoided.

PHYSIOLOGY

The body fluid is the means of maintaining homeostasis in the tissues and of transporting metabolites and waste products to and from the cells. The body water forms about 70% of body weight in the lean adult male, a rather greater proportion in children and rather less in women and relatively more obese men.

Figure 1 demonstrates that the functional fluid can be divided into two main compartments, the intracellular and extracellular. Water equivalent to about 50% body weight is contained within the cells and an amount equal to 20% body weight outside the cells in the extracellular fluid. The extracellular fluid can be divided into the intravascular compartment amounting to about 5% of body weight and an extravascular compartment, consisting of water between the cells, which is equivalent to approximately 15% body weight.

The cells expend energy to maintain active transport through the cell membranes, which, although permeable to salts and water, nonetheless retains potassium ions within the confines of the cell while "pumping" sodium ions out into the extracellular compartment. By contrast, transport through the capillary wall between the intravascular and extra-

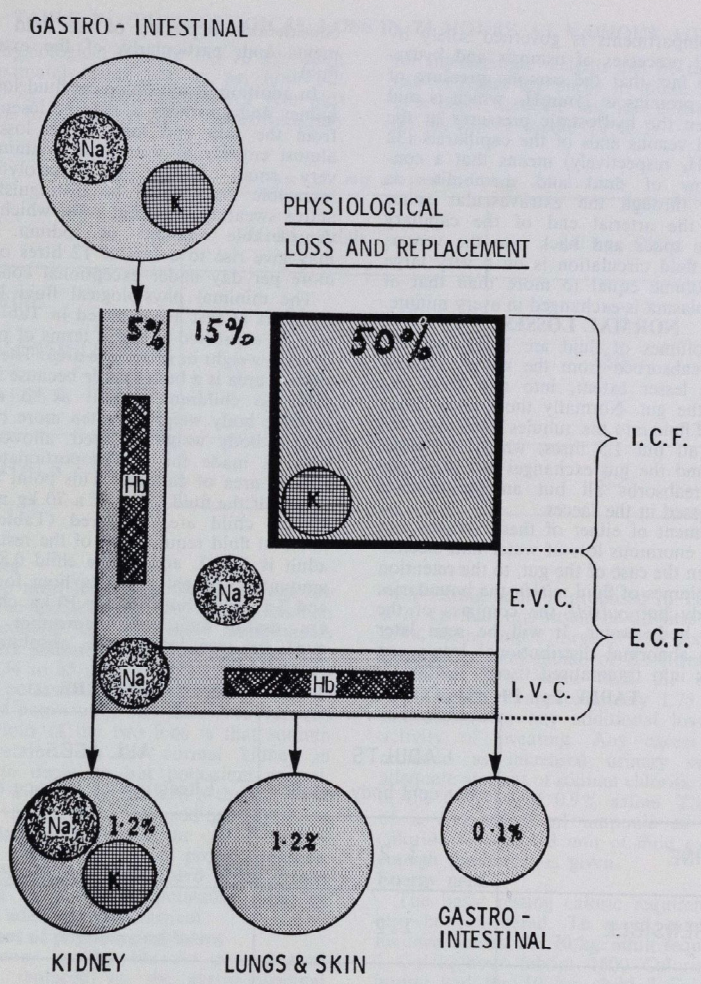


Figure 1. The body water forms about 70% of body-weight (B.W.). The intracellular fluid (I.C.F.) accounts for 50% B.W. and the extracellular fluid (E.C.F.) about 20%. The I.C.F. and E.C.F. are separated by an active membrane (thick black line) which 'pumps' sodium out of the cell and retains potassium within it. The E.C.F. is further subdivided into the intravascular compartment (I.V.C.)—about 5% B.W.—and the extravascular compartment (E.V.C.)—about 15% B.W. These are divided by the capillary epithelium, filtration through which depends on physical principles. Normal losses occur via the kidney, lungs and skin (insensible loss) and the gastro-intestinal tract. Average net adult losses are shown as percentages of body weight. In normal physiological circumstances, replacement occurs into the I.V.C. via the gastro-intestinal tract (top left).

vascular compartments is governed solely by the physical processes of osmosis and hydrostatics. The fact that the osmotic pressure of the plasma proteins is 25mmHg, which is mid way between the hydrostatic pressures at the arterial and venous ends of the capillaries (32 and 12mmHg respectively) means that a continuous flow of fluid and metabolites is maintained through the extravascular space (out from the arterial end of the capillary through the space and back into the venous end). This fluid circulation is on a very large scale—a volume equal to more than that of the whole plasma is exchanged in every minute.

NORMAL LOSSES

Large volumes of fluid are being secreted into and reabsorbed from the kidney tubules and, to a lesser extent, into and from the lumen of the gut. Normally the kidney filters 170 litres of fluid into the tubules each day but reabsorbs all but 1.5 litres, which is passed as urine, and the gut exchanges 8-9 litres per day but reabsorbs all but an infinitesimal amount passed in the faeces.

Derangement of either of these systems can lead to an enormous loss of water and electrolytes and, in the case of the gut, to the retention of large volumes of fluid *within* the boundaries of the body but *outside* the compass of the functional body water. It will be seen later that large abnormal distributional losses can also occur into traumatised tissues leading to considerable depletion of the fluid compartments and, particularly, of the extracellular fluid.

In addition to physiological fluid losses from kidney and gut there is also the insensible loss from the skin and lungs. This loss consists almost entirely of water and contains only a very small amount of electrolytes. This insensible loss should be distinguished from active sweating, the fluid from which contains considerable amounts of sodium. Sweating may give rise to a loss of 12 litres of fluid or more per day under exceptional conditions.

The minimal physiological fluid losses per 24 hours at rest are depicted in Table 1. They may be expressed either in terms of percentage of body weight or of surface area. Theoretically, surface area is a better guide because it is applicable to children as well as to adults. In practice body weight is often more convenient but, if body weight is used, allowance must then be made for the proportionately larger surface area of children. This point is demonstrated if the fluid losses of a 70 kg. man and a 10 kg. child are compared (Table 2). The minimal fluid requirement of the resting 70 kg. adult is 1.75 L and of the child 0.85 L. This amounts to roughly 1 ml/kg/hour for the adult and 3.5 ml/kg/hour for the 10 kg. child. These are useful figures to remember but they must be regarded as the absolute minimal requirement.

TABLE 1: PHYSIOLOGICAL FLUID LOSS IN 24 HOURS

	ADULTS Per cent body weight	ALL AGES Litres per m^2 surface area
URINE	1.2	0.5
INSENSIBLE	1.2	0.5
GASTRO INTESTINAL	0.1	0.05
TOTALS	2.5	1.05

Per cent. body weight is an acceptable means of assessing fluid losses for the adult of over 50 kg. Litres per square meter surface area is essential for children unless due allowance is made for the proportionately large surface area in the smaller patient (see Table 2).

TABLE 2: PHYSIOLOGICAL LOSS IN 24 HOURS AT VARIOUS AGES

The physiological loss in the 70 kg. adult is approximately 1 ml per kg. per hour but in the 10 kg. child (approximately 1 year old) it amounts to 3.5 kg. per hour. Approximations for intermediate weights can be made if these two extremes are borne in mind. Surface area is a more accurate but less convenient guide for all ages (compare Table 1).

	Loss in mg per kg. per 24 hours	
	70 kg. Adult	10 kg. Child
URINE	12	50
INSENSIBLE	12	30
FAECES	1	5
TOTALS	25	85

Electrolytes

The two most important inorganic ions are sodium (the extracellular ion) and potassium (the intracellular ion). The daily requirements of these are sodium (as sodium chloride) 2 to 5 grams (34 to 85 mEq of sodium) and potassium (as potassium chloride) about 4 grams (52mEq of potassium). The difference between the behaviour of the two ions is that sodium can be retained by the normal kidney in response to depletion but potassium cannot. Potassium will only be retained if there is renal failure. It is fortunate that sodium can, to a point, replace potassium in the cell if there is potassium depletion but, in proper practice, this fact should not be regarded as an excuse for failing to anticipate potassium loss by providing adequate replacement.

Replacement of physiological losses

In ordinary healthy life the physiological losses are replaced via the gastro-intestinal tract in food and drink. Normally an excess of fluid is ingested with a compensatory excretion of additional urine. In the adult a minimum volume of urine of 600 ml is required to get rid of waste nitrogenous products. If normal feeding processes are denied to the patient, the fluid requirement must be supplied intravenously or by some other route. A suitable intravenous regime based on body weight for the maintenance of physiological fluid balance in a resting adult is proposed in Table 3. For a 70 kg. man this would be 0.5 litre of isotonic 0.9% saline and 2 litres of 5% dextrose. This provides a total intake of 2.5 litres of water, which is 30% more than the basic fluid requirement of approximately 1.75 litres, but this allows for any additional losses due to activity of sweating. Any excess water is removed as increased urinary output. An adequate amount of sodium chloride is supplied in the 0.5 litre of 0.9% saline. The addition of a 1 gram 5 ml ampoule of potassium chloride to each 0.5 unit of fluid ensures that enough potassium is given.

Energy needs

The basic resting caloric requirement must also be considered. To avoid catabolism of his own tissues, the 70 kg. adult requires about 1 Cal/kg/hour (about 1600 Calories per 24 hours) and the 10 kg. child 2 Cals/kg/hour (500 Calories). To avoid ketosis an intake of carbohydrate supplying about a quarter of these amounts is required (about 400 Calories for the 70 kg. adult and about 125 Calories for the 10 kg. child). This must be regarded as the absolute minimal requirement.

The energy derived from the regime described above is thus enough to prevent ketosis but *not* enough to prevent tissue catabolism. Intravenous feeding with carbohydrate and

TABLE 3: INTRAVENOUS REPLACEMENT OF PHYSIOLOGICAL, PRE-EXISTING AND OBSERVED FLUID LOSSES IN ADULTS OF OVER 50 KG BODY WEIGHT (B.W.)

SOURCE OF LOSS			REQUIREMENT PER 24 HOURS			
			Blood or Plasma Expander	0.9% Saline	5% Dextrose	Total
Physiological	(at rest)		0	1%BW	2%BW	3%BW
Pre-existing	Dehydration (salt + water)	Clinical	0	3%BW	3%BW	6%BW
		Severe	0	4%BW	3%BW	7%BW
	Blood		measured loss + 10%	2xmeasured loss	0	3xmeasured loss
Observed	Body fluids		0	80% loss	20% loss	100%loss
	Blood		measured loss + 10%		0	110% loss

The regime recommended for "physiological" replacement provides 30% more fluid than the absolute basic requirement (see Tables 1 and 2) to allow for additional insensible loss. It contains enough sodium chloride (4.5 grams) but potassium chloride, at least 2 grams per litre, must be added to provide the requisite potassium.

amino acid solutions and fat emulsions must be considered if a patient remains on such a regime for more than 2 or 3 days.

PATHOLOGICAL ALTERATIONS IN FLUID AND ELECTROLYTE BALANCE

It is fortunate that the body possesses considerable powers to compensate automatically for all but gross fluid and electrolyte derangements. Probably fully 80% of surgical patients will make a rapid and satisfactory recovery with little attention to fluid replacement. However, in a small number of cases, meticulous attention to detail is necessary if mortality is to be avoided. In a considerably larger number morbidity can be reduced and the patient's comfort improved by intelligent attention to fluid replacement.

Disturbances in body fluid can be divided into those of loss of volume and those of change (usually deficiency) in composition.

The possible sites of pathological extracorporeal loss have already been considered including abnormal loss from the kidney and from the gut, as vomit, diarrhoea or loss of other intestinal fluids from fistulae or drainage sites and surface loss due to sweating.

Distributional loss must also be considered. The term "distributional loss" implies losses due to "translocation" of a volume of fluid within the confines of the body which is thus denied to the effective circulating blood volume, but still forms part of the body weight. Translocations of this nature will be considered later in the paper but it is pertinent to mention at this stage that it is in this area that important research by Shires and other workers has led to major changes in our attitude to fluid replacement in patients who suffer accidental or surgical trauma. 3-7

Losses of electrolytes are the most important potential changes in composition and sodium and potassium by far the most important ions concerned. It is true that changes in pH can have disastrous consequences, particularly on cardiac action, but automatic compensation of the acid/base state usually follows adequate replacement of volume and electrolytes.

The importance of the supply of adequate calories in long term replacement has also been briefly considered earlier. Other substances which might be depleted include the haemoglobin and the plasma proteins. Surprising though it may seem, these have relatively minor relevance in the present context unless the patient was *initially* anaemic or suffering from hypoproteinaemia. The following factors should be considered in this context. Neither haemoglobin nor plasma proteins (colloids) are lost by any of the routes we have discussed up to this point. Given a normal red cell mass, an individual can lose more than half his haemoglobin without suffering anoxia. Except in specific conditions such as proteinuria or malnutrition, plasma proteins are not easily depleted. Even in haemorrhage this constituent of whole blood appears to be rapidly replaced probably from the lymphatic system. Burns have long been considered to cause a major loss of colloid but recent experience suggests that the deficiency in these cases is largely made up of electrolytes and water alone.[8]

Supportive Terapy

Replacement of fluid either in volume or composition must not be considered in isolation. Support may be required for the cardiopulmonary circulation.

Such therapy may well include additional oxygen and mechanical ventilation in cases of severe haemorrhage, vasoconstriction or acidosis, the use of inotropic drugs such as digitalis or isoprenaline to increase cardiac output and permit rapid replacement when cardiac failures threaten, and the use of diuretics.

CLINICAL ASSESSMENT AND REPLACEMENT OF LOSSES

Before, during, or after surgery we must assess the fluid requirement of the patient at any given time in terms of (a) the pre-existing deficit which requires replacement; (b) the continuing physiological loss; and (c) continuing current abnormal losses. The relative need for the three categories of replacement (water, electrolytes, and blood) must also be considered.

Since sodium has the primary function of controlling the distribution of water in the body, sodium and water deficits and excesses are inextricably linked. Ever since Marriott's classic articles [9, 10, 11] it has, however, become customary to consider water and sodium deficits separately in order to simplify the understanding of the phenomenon of "clinical dehydration".

Pre-existing water deficiency

Almost pure water deficiency (Figure 2) occurs classically in diabetes insipidus, sometimes when patients cannot take water by mouth due to coma or an oesophageal or pharyngeal cancer and, occasionally, when a high protein diet is given; in such a case a large quantity of water may be required to excrete the extra urea.

Water deficiency is an *intracellular* dehydration; water passes from the intracellular space to maintain the volume of the extracellular space in general and the intravascular space in particular. The initial symptoms are thirst and dryness of the mouth. The urinary output will be reduced and the specific gravity of the urine may be as high as 1035. The intravascular space must, ultimately, become depleted and circulatory failure will follow, sodium is then retained and coma ensues at serum levels of 170 mEq or more.

Pre-existing sodium depletion

Almost "pure" salt depletion may occur if a mixed salt and water depletion, usually from the gastro-intestinal tract losses, is replaced by water alone either by mouth or by 5% glucose parenterally, (Figure 3.)

This is an *extracellular* dehydration. Water passes out of the extravascular compartment to maintain the osmotic pressure by concentrating the available sodium. The red cells swell and, initially, the volume of the intravascular compartment will be maintained. The urine will be normal both in volume and in specific gravity.

The somatic cells, including the nerve cells, are overhydrated and the patient will suffer from apathy, loss of energy, weakness and

cramps. The skin will feel inelastic due to depletion of the extra-vascular space. Ultimately circulatory failure and coma will ensue.

Pre-existing mixed water and sodium depletion

The surgical emergency suffering from clinical "dehydration" due to major fluid loss as vomit or diarrhoea, or into obstructed or atonic bowel or from a fistula has a "mixed" salt and water deficiency and an appropriately "mixed" symptomatology consisting of thirst, apathy, inelastic tissues, and scanty urine.

A patient with overt clinical dehydration short of circulatory failure has probably lost up to 6% body weight. If the inevitable vasodilation of an anaesthetic is imposed upon him he will suffer cardiovascular collapse. In practice

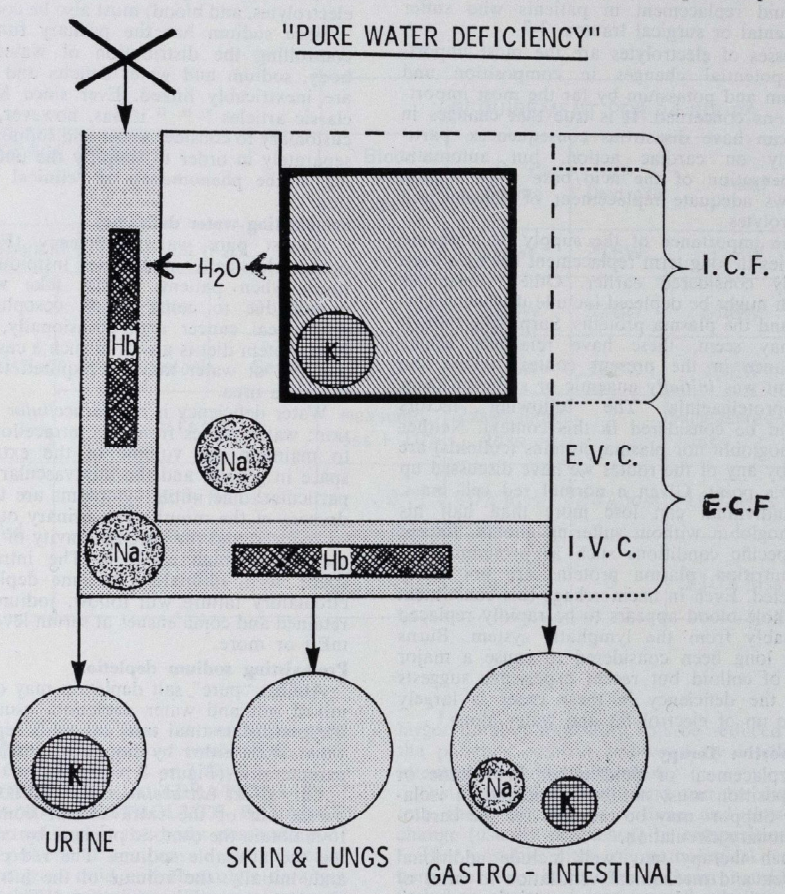
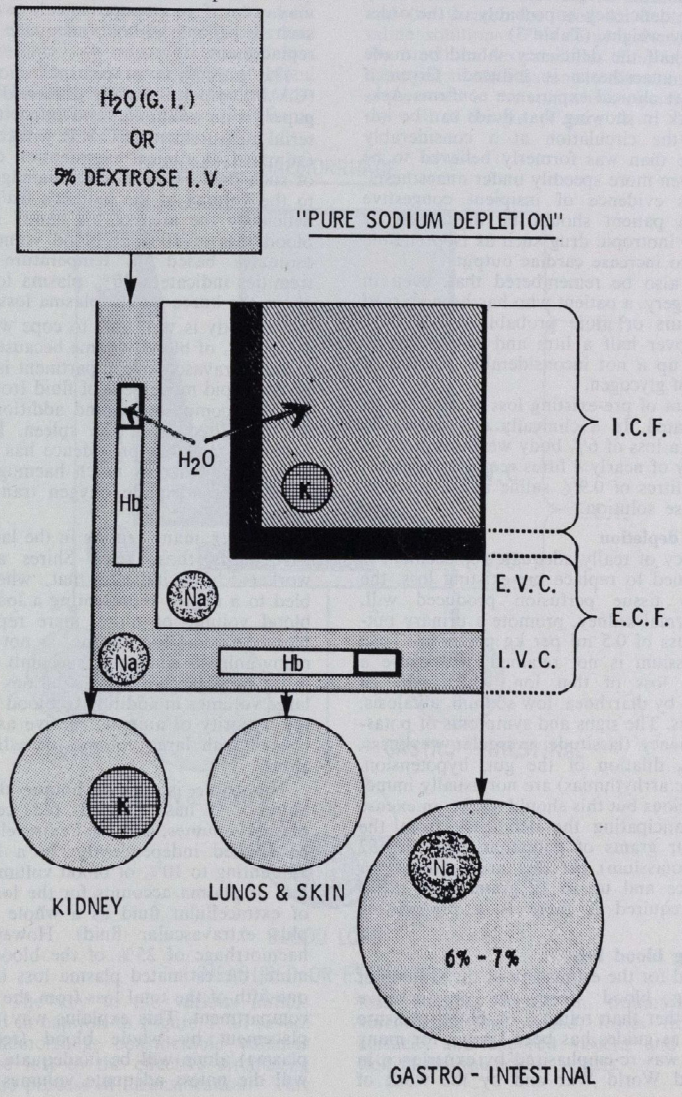


Figure 2. The key to the various initials will be found in the caption to Figure 1. " Pure " water deficiency occurs when there is abnormal predominant water loss without equivalent replacement either orally or parenterally (X). Water deficiency results in an intracellular dehydration. Water passes from the cell to compensate for the water deficit in the E.C.F. and, in particular, to maintain the volume of the I.V.C. for as long as is possible.

Figure 3. The key to the various initials will be found in the caption to figure 1. " Pure " salt deficiency occurs when a loss of salt and water is replaced by water alone. An extracellular dehydration ensues; water passes from the E.V.C. into the red blood cells thus initially compensating for lowered plasma volume and into other cells resulting in the symptoms and signs of cellular overhydration.



the best preliminary replacement procedure is that recommended by Darrow.[12] This is to make up the deficit with alternate bottles of 0.9% saline and 5% dextrose. If dehydration has gone further and circulatory failure is already present, the deficiency is probably of the order of 7% body weight. (Table 3).

At least half the deficiency should be made up before anaesthesia is induced. Given a normal heart clinical experience confirms Askrogg's[13] work in showing that fluids can be imposed on the circulation at a considerably greater rate than was formerly believed to be safe and even more speedily under anaesthesia. If there is evidence of insipient congestive failure, the patient should be digitalised or some other inotropic drug such as isoprenaline employed to increase cardiac output.

It must also be remembered that, even in elective surgery, a patient who has been starved for six hours or more probably has a fluid deficit of over half a litre and will probably have used up a not inconsiderable amount of his stores of glycogen.

The extent of pre-existing loss should not be underestimated. In a clinically dehydrated 70 kg. patient a loss of 6% body weight represents a deficiency of nearly 4 litres requiring replacement of 2 litres of 0.9% saline and 2 litres of 5% dextrose solution.

Potassium depletion

If a policy of really adequate replacement is being pursued to replace pre-existing loss, the satisfactory tissue perfusion produced will, given a normal kidney, promote a urinary output in excess of 0.5 ml per kg per hour. This, since potassium is not retained, will cause a continuous loss of that ion, which will be aggravated by diarrhoea, low sodium, alkalosis, and diuresis. The signs and symptoms of potassium deficiency (lassitude, muscular weakness, drowsiness, dilation of the gut, hypotension, and cardiac arrhythmias) are not usually immediately obvious but this should not be an excuse for not anticipating the deficiency from the start.[14] Four grams of potassium chloride (52 mEq of potassium) per day are required for maintenance and up to 12 grams (156 mEq) may be required if there is a pre-existing deficiency.

Pre-existing blood loss

The need for the estimation of the volume of pre-existing blood losses in pre-operative patients rather than reliance on blood pressure and pulse as guides has been known for many years and was re-emphasised by experience in the Second World War and by the work of Grant and Reeve and many other workers.[15, 16]

A fit young man can lose 30% or more of his blood volume without a significant fall in systolic blood pressure but all anaesthetists are aware of the catastrophic hypotension which may occur if an attempt is made to anaesthetise such a patient without adequate preliminary replacement.

The estimation of central venous pressure (C.V.P.), which is briefly discussed later in the paper, is an additional and important aid. The serial measurement of C.V.P. will confirm losses estimated at clinical examination on the basis of such data as "soft tissue damage equivalent to the volume of the patient's fist" or "a skin deficiency the area of his hand" represents a blood loss of 10% of blood volume or other estimates based on temperature—"cold extremities indicate a 30% plasma loss, coldness above the knees a 40% plasma loss."[16, 17]

The body is well able to cope with losses of 10 to 12% of blood volume because the volume of the intravascular compartment is maintained by the rapid movement of fluid from the extravascular compartment and additional red cells are mobilised from the spleen. It is also a fortunate fact that providence has endowed us with about twice as much haemoglobin as we require for adequate oxygen transport to the tissues.

However, many reports in the last few years, particularly those from Shires and his coworkers.[3, 4, 5, 18] indicate that, when dogs are bled to a degree representing a loss of 25% of blood volume or more, mere replacement of blood "volume for volume" is not enough and many animals so treated succumb. If, however, saline or balanced saline solutions are given in large volumes in addition to blood replacement, the majority of animals survive as do animals treated with large volumes of saline solutions alone.

The triple isotope technique developed by Shires,[3, 4, 18] has enabled red cell volumes, plasma volumes, and total extracellular fluid to be studied independently. In a haemorrhage amounting to 10% of blood volume, the actual loss of plasma accounts for the loss of volume of extracellular fluid as a whole (i.e., plasma plus extravascular fluid). However, after a haemorrhage of 25% of the blood volume or more, the estimated plasma loss is only about one-fifth of the total loss from the extracellular compartment. This explains why it is that replacement by whole blood (red cells and plasma) alone will be inadequate. The animal will die unless adequate volumes of balanced saline solution amounting to two or three times the measured blood loss are administered.

Shires,[3, 4, 18, 19] extended his study to 18 humans using volumes measured by the isotope technique one week after admission and treatment for severe haemorrhage as control values. He showed that where losses of red cells and plasma represented an apparent external loss of just over 1 litre (just over 20% of blood volume) of whole blood there was a hidden loss of extracellular fluid over and above the plasma loss amounting to some 4.5 litres.

In severe haemorrhage (20% of blood volume or over) there is a loss of a large volume of extracellular fluid due to translocation. The more the trauma accompanying the haemorrhage, the more the loss of extracellular fluid and the greater the need for replacement by saline solutions. This large volume is lost to the functional extracellular fluid both into the damaged tissue adjacent to the wound and also sequestered in the splanchnic circulation and in the wall of the gut, (Figure 4).

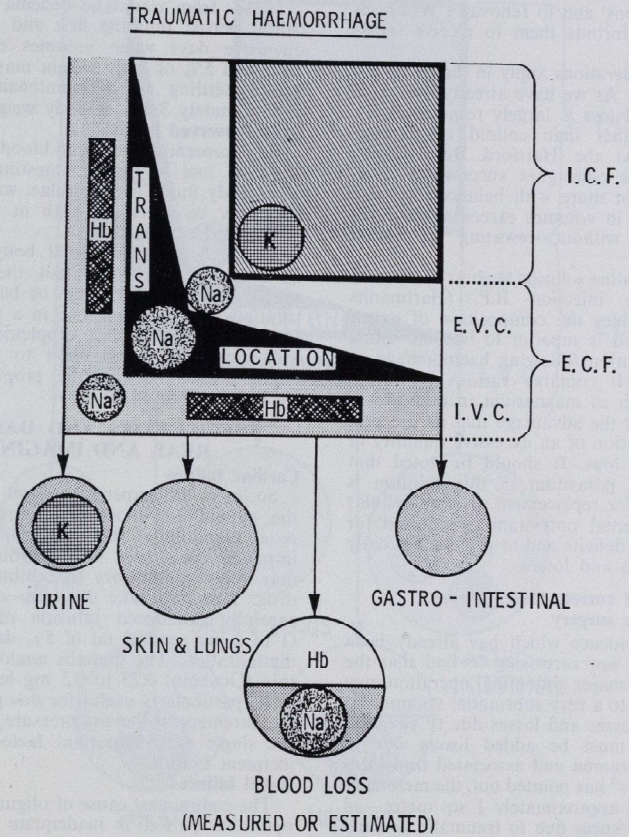


Figure 4. The key to the various initials will be found in caption to Figure 1. In severe traumatic haemorrhage considerable volumes of E.C.F. are lost to the effective circulating volume by the process of "translocation." This loss includes losses in traumatic oedema surrounding the wound, losses from wound surfaces and exposed viscera and sequestration in the splanchnic vessels.

It is probable that adequate therapy for severe traumatic loss only be achieved by the replacement of the measured loss of whole blood plus at least twice, and possibly three times, that volume of isotonic saline solution. If no blood is available, the blood volume lost should also be replaced by twice the amount of isotonic saline solution (i.e., a total of four times the measured loss of whole blood). Within limits the haemoglobin dilution is much less important than the need for adequate fluid replacement with saline solution—a fact important in disaster conditions[7] and to Jehovah's Witnesses whose religion forbids them to receive transfused blood.[20]

Similar considerations apply in the treatment of severe burns. As we have already observed, electrolytic fluid loss is largely responsible for burn shock rather than colloid or plasma-protein loss. At the Hartford Burn Clinic, Moyer[8] and his colleagues successfully treat burns of 40% or more with balanced isotonic saline solutions in volumes exceeding 20 litres in 30 hours without resorting to blood transfusion.

A balanced saline solution such as compound sodium lactate injection B.P. (Hartmanns solution) resembles the composition of extracellular fluid and is superior to isotonic saline as a repair solution following haemorrhage or burn loss.[5-8, 20] It contains various important electrolytes such as magnesium in addition to sodium and has the advantage that its use precludes the injection of an excessive quantity of acidic chloride ions. It should be noted that the amount of potassium in this solution is sufficient only for replacement of extra-cellular fluid. Supplemental potassium is required for replacement of deficits and to provide for daily metabolic needs and losses.

Replacement of current losses during and after major surgery

From the evidence which has already been presented, it is not surprising to find that the losses during a major abdominal operation may in fact amount to a very substantial volume. To physiological losses and losses due to sweating under drapes must be added losses due to haemorrhage, trauma and associated translocation. As Jenkins[18] has pointed out, the mesentery has an area of approximately 1 sq. metre—an increase in thickness due to traumatic oedema of only 1 mm represents a loss to functional fluid volume in translocation of 1 litre. Losses in thoracic operations are rather less than in abdominal surgery, except in such operations as pulmonary decortication but they are, nonetheless, substantial as are the fluid deprivations in orthopaedic surgery especially in hip operations and when tourniquets are applied, (Figure 5).

It is small wonder that many clinicians are finding that in major surgery given normal cardiovascular and renal function, replacements of 5 to 15 ml/kg/hour of balanced saline preparations such as Hartmanns solution are the order of the day and greatly improve the patient's condition both during the operation and in the post-operative period.[3-7, 18, 19, 21]

Losses from traumatic oedema and translocation persist into the first and second post-operative days when volumes equivalent to 10% and 5% of body weight may be required before settling to a maintenance level of approximately 3.5% of body weight.

Other observed losses

Replacement of measured blood loss and any observed loss of gastric, intestinal, biliary or other body fluids from fistulae, wounds drains, etc., may be necessary both in the pre- and post-operative period.

Although gastro-intestinal body fluids vary quite widely in ionic content, they can be replaced with isotonic saline or balanced saline solutions and 5% dextrose in a proportion of 4 to 1 plus potassium supplementation. The kidney can be relied upon to sort out any detailed differences in the proportion of the electrolytes infused.

DIFFICULTIES AND DANGERS: REAL AND IMAGINED

Cardiac failure

So far in this paper it has been assumed that the patient has a normal heart and normal renal function. If the myocardial function is impaired (i.e., there is a "cardiac deficit") it may become necessary to exhibit an inotropic drug. This may take the form of a slow and carefully monitored infusion of isoprenaline (1 to 2 mg per 500 ml of 5% dextrose), or of digitalisation. The digitalis analogue strophanthin (Ouabain) 0.25 to 0.5 mg has been found to be particularly useful for this purpose.[22] The measurement of venous pressure (see below) is the single most important factor in assessing incipient failure.

Renal failure

The commonest cause of oliguria in the post-operative period is inadequate fluid replacement but, in the case of true renal failure, fluids must be restricted to 400 to 500 ml (0.6% BW) in the adult plus the replacement of urinary output and other observed losses. The osmotic diuretic 20% mannitol (100 to 200 ml)

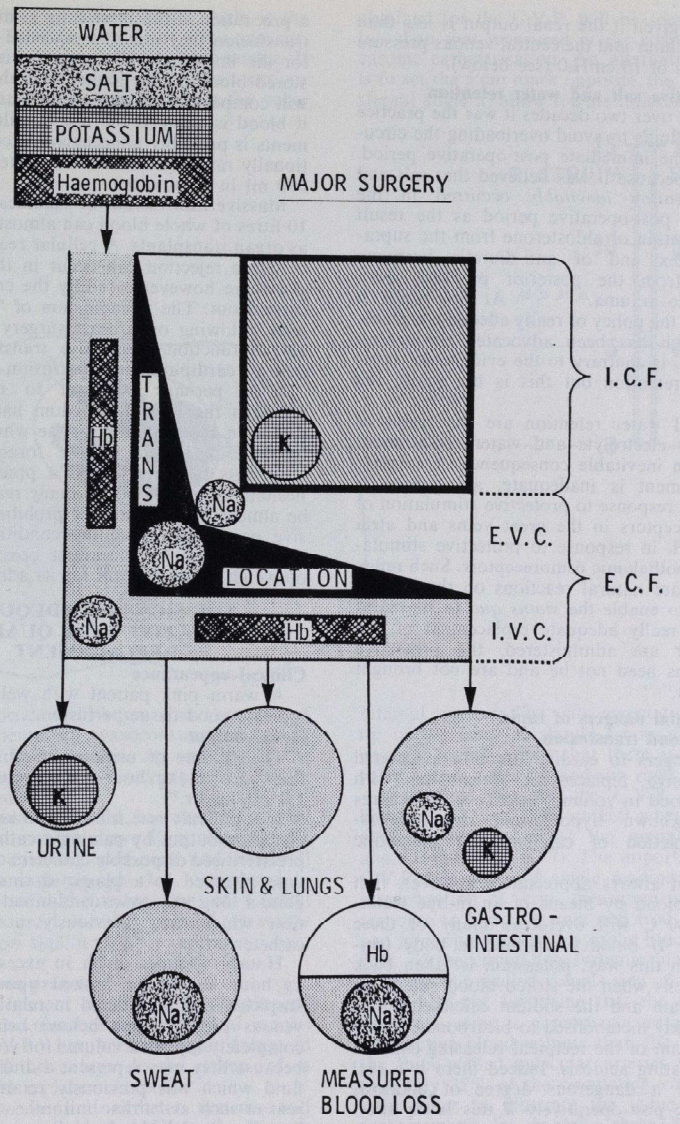


Figure 5. The key to the various initials will be found in the caption to Figure 1. In major abdominal surgery losses occur into and from the exposed gut, as sweat, as measured blood loss and by translocation and sequestration. All such losses must be replaced intravenously.

should be given if the renal output is less than 0.5 ml/kg/hour and the central venous pressure is in excess of 10 cmH_2O (see below).

Post-operative salt and water retention

For well over two decades it was the practice to restrict fluids to avoid overloading the circulation in the immediate post-operative period. This was because it was believed that salt and water retention *inevitably* occurred in the immediate post-operative period as the result of the secretion of aldosterone from the suprarenal cortex and of anti-diuretic hormone (A.D.H.) from the posterior pituitary as a response to trauma.[11, 18, 23, 24] At first sight it seems that the policy of really adequate replacement, which has been advocated throughout this article, is contrary to the evidence of post-operative retention but this is not really the case.

Salt and water retention are the result of *inadequate* electrolyte and water replacement and *not* an inevitable consequence of surgery. If replacement is inadequate, aldosterone is secreted in response to protective stimulation of volume receptors in the great veins and atria and A.D.H. in response to protective stimulation of hypothalamic osmoreceptors. Such reflex responses are natural reactions on the part of the body to enable the *status quo* to be maintained. If really adequate replacement of salt and water are administered, the protective mechanisms need not be and are not brought into action.[18, 24]

The potential dangers of rapid massive blood transfusion

The dangers to cardiac function associated with massive replacement transfusion with citrated blood in volumes in excess of 10 litres are well known—hypothermia, excess potassium, depletion of calcium and metabolic acidosis.[25]

It is not always appreciated, however, that warming blood by means of an in-line water-bath at 40°C will overcome many of these difficulties. If blood is warmed to body temperature in this way, potassium is taken back into the cells when the stored blood enters the blood stream and the sodium calcio-citrate is quite quickly metabolised to bicarbonate in the blood stream of the recipient releasing calcium and combating acidosis. Indeed there is a real danger of a dangerous degree of alkalosis developing post-operatively if this latter effect is not anticipated and full correction of metabolic acidosis deliberately avoided. The work of Howland[26, 27] has demonstrated that if calcium chloride and bicarbonate are given as a precaution at the same time as massive blood transfusion they must be regarded as treatment for the immediate effects of the impact of the stored blood. In the longer term the body itself will compensate for these effects and, therefore, if blood is warmed the use of calcium supplements is probably unnecessary except in exceptionally rapid transfusion at a rate in excess of 500 ml in 5 minutes.

Massive transfusions of volumes in excess of 10 litres of whole blood can almost be regarded as organ transplants. A cellular reaction similar to tissue rejection can occur in the lungs and elsewhere however carefully the cross-match is carried out. The phenomenon of "pump lung" seen following open-heart surgery is undoubtedly a function of massive transfusion rather than of cardipulmonary perfusion as such.

It is perhaps pertinent to remark that, although the United Kingdom has the advantage of a blood donor service which is second to none, it should not be forgotten that in other less favoured places a practitioner may hesitate to give blood for many reasons; it may be almost unobtainable or prohibitively expensive or, in the prevailing conditions the risk of infection or an improper cross-match may outweigh the indication for its administration.[25]

THE SIGNS OF ADEQUATE QUANTITATIVE AND QUALITATIVE REPLACEMENT

Clinical appearance

A warm pink patient with well filled veins indicate good tissue perfusion.[7]

Renal output

The volume of urine secreted should be at least 0.5 ml/kg/hour and preferably nearer 1.0 ml/kg/hr.[29]

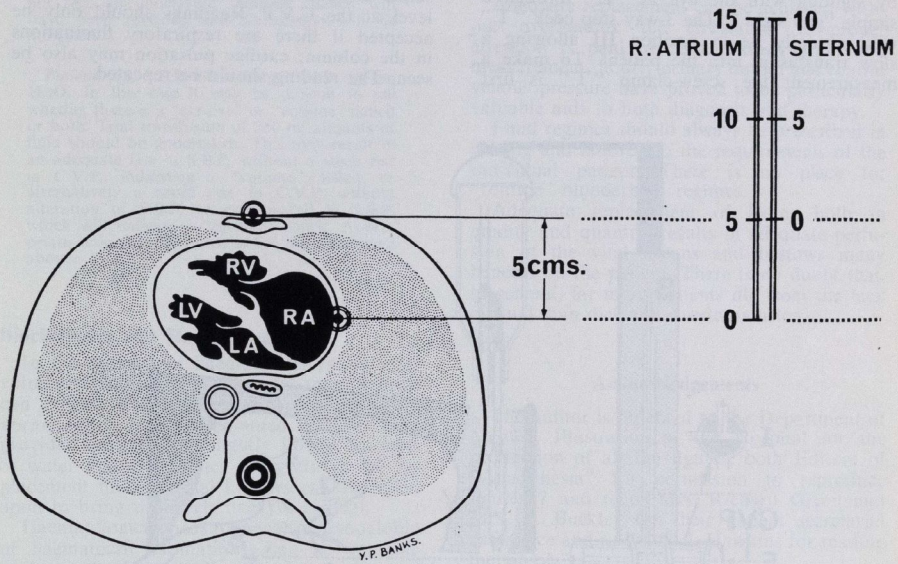
In a difficult case it is wise to keep an hourly check on output by passing a catheter. Modern pre-sterilised disposable catheters of the Gibbon type attached to a plastic drainage bag have gone a long way towards eliminating the infection which was previously a deterrent to catheterisation.

Hourly volumes much in excess of 1.0 ml/kg/hour should be looked upon with some suspicion and considered in relation to central venous pressure (see below) before they are completely replaced volume for volume. This is because they may represent a diuresis of excess fluid which was previously retained for some reason such as cardiac failure.

Systolic arterial blood pressure

Systolic arterial blood pressure on its own is a poor guide to adequate volume replacement. Anyone who has dealt with severe trauma

Figure 6. The manubrio-sternal angle is the most convenient reference point when measuring C.V.P. with a simple saline manometer. If the scale zero is opposite this surface marking it must be remembered that the value obtained for the C.V.P. will be some 5cmH_2O less than that measured at the right atrium in cardiac catheterisation. An alternative method is to set the 5 cm mark opposite the manubrio-sternal angle to allow for the difference.



in war or peace will know that, because of active compensatory vasoconstriction, a fit young individual may lose up to 30% of the blood volume, with little accompanying fall in systolic pressure.[30]

Systolic pressure should, however, be usually maintained at a value above 80 mmHg in order to maintain coronary perfusion and we shall see in the next paragraph that, considered in conjunction with the estimation of Central venous pressure, systolic arterial pressure assumes a new significance.[31]

Central venous pressure

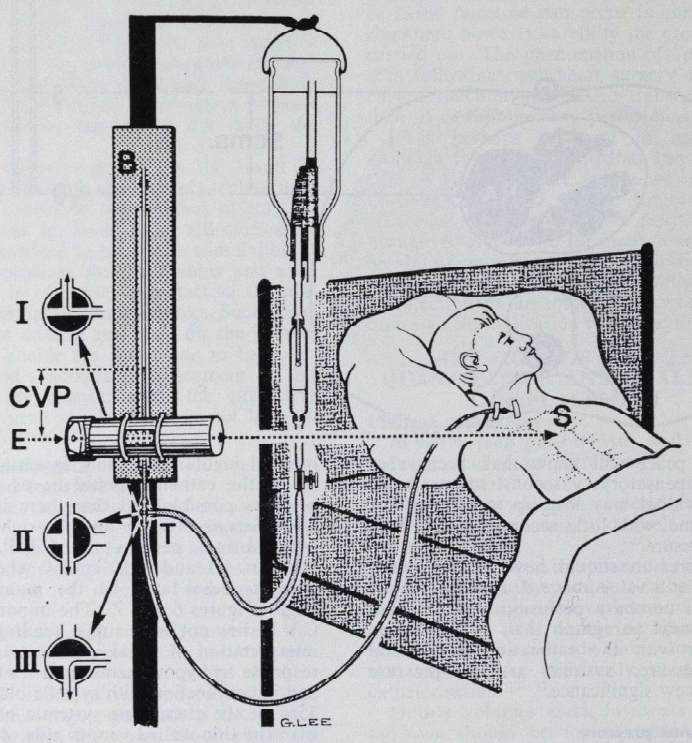
The development of relatively simple apparatus for the measurement of central venous pressure (C.V.P.) has been a major factor in enabling full advantage to be taken of a vigorous fluid replacement policy.[31]

Various needle and catheter systems and techniques are available to facilitate the passage of a catheter into the great veins in proximity to the heart either from one of the antecubital or basilic veins or through the external or internal jugular. The only essential is that the tip of the catheter should lie as close to the heart as possible and that there is no venous valve between the tip and the right atrium.[32]

In normal persons the C.V.P. may vary between −1 and +10 cmH_2O when measured with its zero taken at the manubrio-sternal angle (Figures 6 and 7). The importance of the C.V.P. lies not in a single reading but in the interpretation of serial measurements taken in response to hypovolaemia and trial transfusion and in conjunction with systolic blood pressure. 75% of the circulating systemic blood volume is in the thin-walled venous side of the circulation; it is not surprising that the pressure in the great veins is a more accurate indication of effective blood volume than the pressure in the thick-walled muscular arteries which contain only 25% of the circulating volume.

In order to illustrate the value of C.V.P. measurement, let us take three hypothetical patients each of which has been shown to have a systolic arterial blood pressure (S.B.P.) of 60 mmHg.

Figure 7. The reference point, in this case the manubrio-sternal angle "S," is first checked against the scale zero by moving the base board "B" up and down on the drip pole either by eye by using a rod, by rod and spirit level, or by sighting with the eye at "E" through a simple "gun sight". The 3-way stop cock "T" will normally be in position III allowing a slow transfusion into the patient. To make a measurement the screw-clamp "T" is first opened to make sure that the cannula is not obstructed. The stop-cock is now turned to position I and the open manometer filled to well above the expected C.V.P. The cock "T" is then turned to position II and the saline allowed to run in until it establishes a level at the C.V.P. Readings should only be accepted if there are respiratory fluctuations in the column; cardiac pulsation may also be seen. The reading should be repeated.



Patient (*A*). S.B.P. 60 mmHg, C.V.P. initially —5 mmHg. An obvious case of hypovolaemia or "volume deficit." The patient is probably pale, vasoconstricted and sweating and showing all the signs of peripheral circulatory failure which is somewhat nebulously referred to as "shock," a term which, in the author's opinion, is best avoided.

Transfusion should result in a gradual rise in C.V.P. to a level not greater than 10 cmH_2O and a rise in S.B.P. to an acceptable level (say 100 mmHg ∓10 mmHg). Transfusion should be continued until the point where S.B.P. no longer rises but the C.V.P. continues to do so.

If the C.V.P. exceeds 12 cmH_2O and the S.B.P. is still below 80 mmHg. it is probable that there is a "cardiac" deficit as well as a "volume" deficit: the use of inotropic drugs such as isoprenaline or one of the digitalis group should then be considered.

Patient (*B*). 60 mmHg, C.V.P. 15 cmH_2O. Obviously a "cardiac deficit." The neck veins will be congested and there may be signs of ankle, sacral or pulmonary oedema.

The administration of an inotropic drug should result in a fall of C.V.P. and a rise in S.B.P. If the C.V.P. falls but the S.B.P. does not rise, an underlying "volume" deficit will have been revealed and transfusion will be indicated.

Patient (*C*). S.B.P. 60 mmHg. C.V.P. 10 cm H_2O. In this case it may be difficult to tell whether there is a "cardiac" or "volume" deficit or both. Trial transfusion of 250 ml aliquots of fluid should be undertaken. This may result in an adequate rise in S.B.P. without a steep rise in C.V.P. indicating a "volume" deficit, or alternatively a rapid rise in C.V.P. without alteration of S.B.P. or even a fall in S.B.P. which will indicate a "cardiac" deficit. Appropriate action can be taken as has been discussed above.

Biochemistry and Haematology

In the absence of haemoconcentration after volume replacement biochemical measurement can indicate whether the electrolytes are within normal limits. It should be noted, however, that, provided adequate, or slightly liberal amounts of water and electrolytes are given in the replacement fluid, normal kidneys can be relied upon to bring the levels back to normal.

Haematological measurements—haemoglobin or haematocrit estimations—can be valuable guides to the degree of haemoconcentration and the need for blood transfusion; a packed cell volume of less than 35% would certainly be considered an indication for blood in the United Kingdom.

Acid-base balance

If adequate amounts of water and electrolytes are administered, the natural blood buffers and renal function will normally adjust the pH and bicarbonate levels unless there has been severe tissue anoxia due to hypotension or vasoconstriction.

If metabolic acidosis persists, an assessment of the base deficit must be made with an appropriate apparatus such as the Micro-Astrup.[9] The deficit can then be corrected according to the formula:

$$\text{Sodium bicarbonate (mEq)} = \frac{\text{Body weight (kg)}}{3} \times \text{base deficit (mEq)}$$

SUMMARY AND CONCLUSION

There is much evidence to show that the fluid and electrolyte requirement of the surgical patient and seriously ill medical patient is much greater than had previously been supposed.

Adequate replacement can be achieved without overloading the circulation if proper attention is paid to clinical observation and measurement; hourly urinary output and central venous pressure have proved to be particularly valuable aids to both diagnosis and therapy.

Fluid regimes should always be prescribed in writing and tailored to the requirements of the individual patient. There is no place for "routine" blunderbuss regimes.

Adequate replacement of losses both in quality and quantity results in adequate perfusion of the vital organs and bestows many benefits on the patient. There is no doubt that, in general, far more patients die from the lack of fluid than die from overtransfusion.

Acknowledgements

The author is indebted to the Department of Medical Illustration at the Hospital for the production of all the figures, both Editors of "Anaesthesia" for permission to reproduce figures 7 and 6, to Mrs. Richard Green and Mrs. L. Buckley for their patient secretarial assistance and to Dr. T. J. Hawkins for reading the proofs.

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Book Reviews

Antibiotics in Clinical Practice by Hillas Smith. Published by Pitman Medical Publishing Co. Ltd. Price 30s.

In March, 1886, Pasteur received a letter from a person living in Orleans who greatly admired his work on infections and particularly his treatment of rabies. The letter was simply addressed to "The one who does Miracles, Rue d'Ulm, Paris". It would be interesting to see that person's reaction to the account in this book of the vast range of antibiotics, antiviral and antifungal agents that have now been developed and brought into clinical use. It is only 28 years ago that a small quantity of penicillin was used in an attempt to save the life of an Oxford policeman stricken with staphylococcal septicaemia, and for those who have watched the introduction of each new antibiotic since then, the task of keeping abreast has been difficult. To the student just starting medicine, the array of antibiotics is overwhelming and it is for him that this book will prove of greatest value.

The text has been divided into two parts. The first section deals with each antibiotic or group of antibiotics in considerable detail. A very useful table of the properties, routes, dose and precautions for the use of each agent is included at the end of each account together with a very full list of references; rather more references, one would think, than are really necessary for a book like this. The second part of the book discusses the main types of infection and gives a guide to diagnosis, investigation and management of bacterial endocarditis, bacterial meningitis, and tuberculosis, for example. The regimes advocated follow the best accepted practice. In dealing with drug combinations in meningitis one of the early chapters may be misleading. It is suggested that the combination of penicillin and chloramphenicol can be given in purulent meningitis. This particular combination was an early example of the inadvisability of giving a bacteriocidal with a bacteriostatic drug. To be fair, in a later chapter on meningitis the advice is modified to the recommendation that penicillin and chloramphenicol could be used as initial treatment in a child where the purulent meningitis is of unknown aetiology. A better drug would be ampicillin which has a wide spectrum and crosses the blood brain barrier.

The paperback presentation is to be commended, but the size of the type used for legends on the figures requires first class eyesight to read it. The habit of frequent underlining detracts from the appearance of the page and does nothing to emphasise a point, and while possibly helpful in lecture notes is not necessary in a book. Occasional incorrect spelling could well be corrected in the next edition.

J. S. Malpas.

The Chemotherapy of Chronic Bronchitis and Allied Disorders. By J. Robert May, London. English Universities Press, 1968; pp. 115; 35s.

The bacteriology of chronic bronchitis is very much Professor Robert May's own subject and this review and comprehensive account of his own work and views is particularly welcome. He deals successively with the bacteria responsible for chest infections, with the relevant antibiotics, and with the principles and details of treatment. There is a section on allied disorders including asthma, bronchiectasis and cystic fibrosis and in an appendix he gives some technical details. The arrangement produces a certain amount of repetition which is no doubt necessary if the chapters are to be consulted separately but makes continuous reading somewhat laboured. Students of this common and important disease will find a great deal to interest and enlighten them here, and even those who are little concerned with the details of management will learn much about the general approach to the antibacterial therapy of respiratory infection from reading the earlier sections.

Francis O'Grady.

"Anorexia Nervosa" by Peter Dally M.B., F.R.C.P., D.P.M., Physician in Psychological Medicine, Westminster Hospital, London. William Heinemann Medical Books Ltd., London, 1969, 137 pages, Price: 30s.

This monograph is based on a study of 140 women diagnosed as suffering from Anorexia Nervosa. To one who has handled only about a dozen such patients, this fact alone seems commendable. Anorexia Nervosa—like states, including six men, are also discussed.

It is more important, that Gull's and Lasègue's original classic descriptions have been resurrected and put on a more satisfactory basis by giving clear cut definitions of the diagnostic features and by attempting an acceptable working classification into three groups.

The controversial problems of the treatment are made easier to sort out by a chapter on "Criteria for assessing improvement" and by two on the "Possible factors affecting outcome".

To sum up, Anorexia Nervosa is firmly established as a psychological disorder of women (by making amenorrhoea one of the diagnostic criteria), although the Klein-Levin syndrome is accepted as the equivalent in men. Using the above mentioned method, the last chapter can proceed to a logical prognosis of the disorder. Finally, there is a very satisfactory section of 232 references.

V. C. Medvei.

Hewer's Textbook of Histology for Medical Students, 9th edition. Revised by S. Bradbury, M.A., D.Phil. Published by Heinemann. Price 70s.

This book is very well known to many generations of students. In the past its virtues of reasonable size and modest cost have ensured its popularity. However in its earlier editions it could be stated that the text was barely adequate for the requirements of the second and third year medical student, while the illustrations were, in many cases, poor. The chapter on the central nervous system was very well written and adequately illustrated.

Dr. Bradbury has effected a complete change, and is to be congratulated on the fruits of his efforts. The book has been rewritten, and the text is now very suitable for the second M.B. course of London University. The illustrations have been greatly improved, while a considerable number of electron micrographs have been included.

Naturally these improvements have not been achieved without paying a price. In fact the cash price is now 70s. instead of 42s. and the old but still excellent chapter on the central nervous system has vanished. However, both these features are more than offset by the overall improvement in quality. Finally it can be argued that as several schools no longer insist upon the use of staining techniques by their students, it might have been better to have retained the central nervous system, and reduced the bulk by omitting Appendix 2.

These criticisms are on the whole minor, and it can now be stated that Hewer has become a. if not the. "best buy" in Histology textbooks.

F.J.A.

"Fundamentals of Obstetrics and Gynaecology. Vol. I. Obstretrics" by Derek Llewellyn-Jones. Faber and Faber. Price £4. First Edition. 1969.

This is a brand new obstetrics textbook, and covers the subject in the widest possible sense. Its real value lies in the up-to-date coverage of the physiology of pregnancy and labour, obstetric operations, the epidemiology of maternal and perinatal mortality, and a particularly well written section on the problems of the newborn infant. Normal and abnormal pregnancy and labour make up the rest of the book.

There is constant debate among students about the wisdom of settling down to read the larger textbooks. A small book is cheaper and can be carried in the pocket, but excessive condensation of the subject matter often makes them difficult to read and they invariably suffer from a paucity of illustrations. The tomes are often left on the shelf as reference books for the more obscure points. How many of us, for example, have a dusty and unread Gray's Anatomy tucked away somewhere.

However, this book is no tome. It is a first edition published this year. There are over 300 illustrations. The line drawings and graphs are clear and simple and the photographs carefully selected. The text is precise, informative and very readable, and printing errors almost non-existent.

Professor Llewellyn-Jones is from Sydney University and has also had much obstetric experience in Malaya. His comparison of statistics from the UK, USA, Malaya and Australia make interesting reading, as does his method of not only describing what happens in obstetrics and what is done about it, but also, wherever possible, explaining why it happens.

At £4 this book may seem expensive, but for the clarity with which the subject is expounded and the wealth of illustrations, it is good value indeed.

A second volume by the same author on Gynaecology is expected in late 1970.

GEOFF HYDE

SPORTS NEWS

RUGBY CLUB REPORT
Wednesday, 1st Ocober, 1969
Bart's v. Met. Police 'D' Division

A strong Bart's Wednesday XV started off well against the Police. A brilliant interception by Rhys-Evans started the scoring with a try in the right-hand corner. Then Wellingham scored a good blindside try from a scrum on the 25-yard line. This was followed up by an excellent cross-kick from Rhys-Evans, picked up by Laidlaw, who went over on the left wing for another try. Good movement in the three-quarters set Smith off to score between the posts for May to convert. Half-time came with a penalty by May, making the score 17-Nil.

Bart's had slacked off somewhat in the second quarter of the game and this continued into the third quarter, the Police playing a tighter game. Bart's held on well but failed to dominate the game during this time.

The beginning of the fourth-quarter saw Bart's scoring again; May took a pass from Jefferson after a brilliant cross-kick by Lambert: This was converted by May. An exciting piece of handling by the outsides led to Rhys-Evans flinging a brilliantly timed inside pass for Carroll to score between the posts, May converting. The performance as far as the result goes was very creditable. D. Rowlands hooked very well giving us much possession.

Final score 27-Nil.

Team: Sowden
Rhys-Evans
Smith
Jefferson
Laidlaw
Lambert (Captain)
Wellingham
Fairhurst
Rowlands
Furness
Carroll
Skidmore
Barrett
May
Davies

Saturday, 4th October
Barts. First XV v. Southend

Bart's tried exceptionally hard in the first half, the line-out going well and Court hooking a couple against the head. The loose was however very infrequent and consequently Bart's failed to cross the Southend line, Lambert and Davies both going close. The score at half-time was 3 all by virtue of a penalty from Cassidy and a drop goal from Southend.

The second half saw increased vigour from Southend, although Bart's held together well, substantially aided by the efforts of Mark Britton, the Captain. The change in the game came about after an individual foray by Lambert, who kicked ahead, charged down the return kick, picked up and ran to the line for Cassidy to convert. Southend struck back well with a goal after a defensive mistake by Bart's. Bart's were soon ahead again after an individual burst by Laidlaw led to a try by him on the left wing. Southend levelled with a drop goal. This was followed by a brilliantly taken penalty from Cassidy, which bounced on the cross-bar, to put us three points up. A further drop goal by Southend again levelled things. It was left to May to run clear and score on the right wing after excellent handling by the outsides.

The final score—Bart's 17: Southend 14.

Team: Packer
May
Jefferson
Lambert
Laidlaw
Cassidy
Hill
Fairhurst
Court
Cotterell
Britton

Carroll
Fenton
Davies
Barrett

Results of games played on 4th October

1st XV W. 17-14 against Southend
A.XV L. 26-9 against Southend
Preclinical 3rd XV L. 33-8 against Southend
Brigands XV L. 19-14 against Southend
Wanderers XV W. 14-Nil against Alleynians
Veterans XV W. 46-Nil against Standard Cosmos.

Saturday, 18th October, 1969

Bart's 1st. XV v Sandhurst
Won 33-14
Bart's 2nd. XV v Sandhurst
Lost
Bart's Preclinical "A" v Blackheath
Lost
Bart's Brigands v John Fisher Old Boys
Lost
Bart's Clinical Wanderers x London Transport
Won
Bart's Veterans v Surrey University
Won

Report on 1st XV Game:

After a short space Bart's became fully alive and some excellent rucking by the forwards and the subsequent feed of the loose ball, followed by some more second phase possession, resulted in a try by Mason backing up his three-quarters on the inside. This was converted by May. There followed some more hard drives by the forwards and some intelligent running by the three-quarters. In the remainder of the first half, Hill and May added tries, one of which was converted by May, who also notched a penalty goal. A short drop goal by Cassidy made the half-time score 19-11 to Bart's. There had, however, been some moments of loose tackling by Bart's, both in the forwards and in the outsides. On the whole, however, things went well even with the absence of Cotterell for a considerable period.

In the second half an opportunist try by Fenton, which was converted by May, started the scoring. May continued in excellent form by kicking three second half penalties. The final score was 33-14 to Bart's. The return of Mason at No. 8 made a great deal of difference to the rucking and Davies at wing forward had an excellent first half.

Rugby Results: 25th October, 1969

Bart's 1st XV v Sidcup
Lost 9-18
Bart's A. XV v Sidcup
Lost 11-22
Bart's 3rd. XV v Sidcup
Lost Nil-55
Clinical Wanders v Sidcup
Won 6-3
Brigands v Sidcup
Lost 34-Nil
Veterans v Hampstead
Lost 6-3

Report on 1st XV game against Sidcup:

In the first half, due to accurate kicking by Cassidy, Bart's were in the lead by 6-3 and turned round at half time to play down the slight slope with the advantage of having played very well in the last quarter. Things in the second half were not so good; a distinct lack of possession hindered Bart's attack. The three-quarters looked especially dangerous with what loose ball they did get, though passing out to the wings was virtually non-existent. As regards the scrum, it is unfair to comment too much, but the heavy going for the first time in the season might have contributed towards lack of cohesion.

In the A.15 Game v Sidcup, under Gilmore's captaincy, a very efficient back row played with distinction, especially James.

We are pleased to note the continued success of the Clinical Wanderers.

From the *Evening Standard*, Thursday, September 18, 1969

MIDDLESEX PUT MCINTYRE IN COMMAND

Trinidad born Keith McIntyre, who led St. Bartholomew's to their first Hospitals' Cup win for 38 years last season, takes over the captaincy of Middlesex next week.

They were so impressed with flanker McIntyre's leadership of the hospital side, and his general play in the County Championship, that he seemed the ideal choice to spearhead the county's challenge in the South-East group.

HOCKEY CLUB REPORT

The trials at the beginning of the season showed that we have a large number of talented "freshers", this not only means that competition for the 1st. XI is even greater than normal but also that we must stand a good chance of repeating last season's success in the 1st. and 2nd. XI Cup matches. A 3rd. XI and a mixed XI are also being run this season.

The first match was lost to Beckenham (fitness!). We then beat St. Mary's Hospital using the 4-2-4 system to very good effect.

The Cambridge tour was enjoyed by all despite the departure (due to qualification) of some of our more extrovert members! The first match against Fitzwilliam was won 4-3 (Robinson scoring two goals), the second was lost to Jesus 2-0 due to a very dispirited effort —possibly the bad effort at "clap-clap" the night before had permeated onto the hockey field. However we then recovered to win the Selwyn match 1-0 and to draw a particularly good game with Pembroke. We hope to return to Cambridge next year should the water (bomb) level have subsided.

On the Sunday following the tour we competed in the U.L.U. Sixes Tournament and proceeded to come top of our division, only to lose by a goal scored from a short-corner to St. Thomas', the eventual winners.

ST. BARTHOLOMEW'S HOSPITAL GOLFING SOCIETY

The 34th Autumn Meeting was held at Tandridge Golf Club on Thursday, October 2nd. Twenty-four Members attended and enjoyed this lovely course.

The Milson-Rees Cup (handicap) was won by Dr. C. M. B. Pare (37 points).

The Graham Trophy (scratch) was won by Dr. H. Bevan Jones (33 points).

The Robinson Cup (handicap 18 or more) was won by Mr. R. McNab Jones (36 points).

Anyone wishing to play in future meetings should contact one of the Secretaries.

The next meeting is at Porters Park on Wednesday, June 24th, 1970.

I. Kelsey Fry)
H. Harvey Ross) Hon. Secretaries

SWIMMING CLUB REPORT

The annual general meeting of the swimming club was held on the 4th of July, after an excellent dinner at the Royal Automobile Club. Mr. George Ellis was in the chair.

The following officers were elected:

President: Mr. George Ellis.
Vice-president: Mr. Julian Britton.
Captain: Paddy Weir.
Secretary: Charles van Heyningen.
Charterhouse Rep.: Chris Fenn.

Attention was drawn to the club's fine performance in the U.H. and U.L.U. leagues; we were promoted to the premier division in both leagues. Doug Shearer and Chris Fenn were commended for their regular appearances for the university swimming team, and Chris Fenn was also congratulated on his appointment as secretary of U.H. water polo team.

A colours committee was formed, and it was decided to award colours to Pete Durcy and Chris Fenn.

Finally a vote of thanks to Mr. George Ellis was proposed, for making the hallowed premises of the R.A.C. club available to us for our dinner.

We have water-polo matches arranged for October against St. Mary's, Guy's, and St. George's hospitals. On the 22nd of October we are holding a swimming gala, with the Nurses team, to which several other hospital teams have been invited.

Paddy Weir

NURSES SWIMMING CLUB REPORT

During this year we have certainly had a very eventful and successful time. We have participated in numerous invitation races including a Grand Social Gala at the Westminster followed by a discotheque and free drinks. The highlights of the season were no doubt at the annual Summer and Autumn Galas of the London Hospitals' Swimming Association.

At the Summer Gala, held at the London Hospital, the team consisted of Daphne Polston, Jan MacArthur, Gill Jones, Pauline Marschall, Jane Timmis and Rosalind Ingram. Here we won the Freestyle Championship, the Diving, the Freestyle Relay and the Medley Relay.

At the Autumn Gala Bart's really shone, not only the team but also the supporters who were all in very good form. In the team were Daphne Polston, Jan MacArthur, Gill Jones, Rosalind Ingram and Jane Timmis and again we brought back with us six of the nine cups presented. I also had great pleasure in receiving the Nursing Mirror Aggregate Shield from Miss Hadman, Matron of the Royal Free (Bart's trained), for the hospital which obtained the most points in the year. We ended the year by holding an inter-hospital gala of our own, against Westminster and King's College. Bart's won the concluding race of the evening—the leap frog race—thus winning the gala. We also had a social gala from which team members were exempt to give a chance to the other swimmers in the Hospital. This proved to be quite a successful evening and most people enjoyed themselves.

I would like to conclude this short report of the year's swimming by thanking all the people who have supported the team. Hoping that next year in the swimming world will be even more successful and enjoyable.

ROSALIND INGRAM

CROSS-COUNTRY CLUB

At the beginning of the season we welcomed three new members to the club, B. Campbell and R. Miller, both freshers, and D. Pinkard, a post-graduate member from I.C. These three, together with J. Brooks, P. Taylor and R. Moody have formed the basis of the team so far this season, and when H. Glennie returns from Redhill the club should easily be able to maintain its traditionally high standard of middle-distance running. John Brooks has been elected captain for this season and he is also the secretary of United Hospitals Hare and Hounds.

Match results so far this season:

U.C. Relay 6 x 1.8 miles at Hampstead, October 11th.

D. Pinkard ran the first leg for Barts and performed very creditably to bring us home in 14th position. However we slipped back somewhat over the succeeding stages and eventually finished 25th out of 45 teams. Winners: Birmingham University 47m. 17 secs.

Barts results: D. Pinkard, 8m. 34s.; B. Campbell, 9m. 55s.; P. Taylor, 9m. 57s.; R. Moody, 9m. 11s.; J. Brooks, 8m. 30s.; W. Garson, 10m. 11s.

R. Miller recorded 9m. 34s. for I.C. 2nd team.

2nd U.L.U. Trial plus Leagues 1 and 2, 21st October.

Parliament Hill Fields. 5½ miles.

Barts ran a weakened team in the opening match of the league due to the absence of P. Taylor (injured) and H. Glennie, and we therefore finished 10th out of the 14 teams in Div. 1. However Brooks, Pinkard, and Moody all ran well and were selected to represent U.L. against Cambridge University later in the month. Mention too must be made of Miller and Campbell both of whom performed well in their first major race for Barts.

Result: 20. J. Brooks, 33:08. 42.—D. Pinkard, 34:35. 50.—R. Moody, 35:05. 107.—R. Miller, 38:44. 136.—B. Campbell, 40:29. 180 ran.

●

SPORTS DIARY

Dec.
1st Boat club dinner.
3rd 1st. XV v. Westminster Bank, away.
4th Rugby Club Ball.
6th 1st. XV v. Old Alleynians, home.
13th 1st. XV v. Old Askeans, away.
27th 1st. XV v. Metropolitan Police, away.

RECENT PAPERS BY BART'S MEN

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