In cases that take longer than 8 days for cure sulphonamide treatment is generally not needed, as the affection is no longer one of purulent ophthalmia but of a rather resistant conjunctivitis with some muco-pus. The proper treatment of such cases is still a matter of doubt; some, at any rate, are best left alone. In one case (non-gonorrhoeal) profuse purulent discharge continued for nearly a month in spite of intensive treatment with the various sulphonamides. In another, also non-gonococcal, a false membrane formed on the palpebral conjunctiva of the two lids of one eye, and is disappearing only now, three months later; all active treatment was suspended at the end of the first month, and at no time was there any apparent danger to the cornea.

Few relapses have been seen in the present series, and this is perhaps due to the greater initial dosage (0.25 g.) and maintenance dosages of 0.125 g. four-hourly, not only during the active phase of the affection but for three days after apparent clinical cure. Apart from minor toxic effects no untoward results were observed.

Causal Organisms

The 198 non-gonococcal cases in the present sulphonamide series included 50 in which pus cells only, but no organisms, were found, 52 with various bacilli (mostly diphtheroids), 88 with staphylococci, and 8 with other coccal organisms. Inclusion bodies were found 27 times; they occurred twice in association with organisms and 25 times in cases in which no organisms could be found. The actual distribution of the various organisms in this series, taken together with the 11 cases not treated by sulphonamides (but by newer methods of local therapy), is shown in Table III, which includes comparative figures from the previous series.

TABLE III.—*Differential Incidence of Organisms in 269 Consecutive Cases of Ophthalmia Neonatorum treated in the Period Jan. 1 1942, to Dec. 31, 1943, with Comparative Findings for 322 Cases seen during Sept., 1939, to Dec., 1941*

	Sept., '39, to Dec., '41	Jan., '42, to Dec., '43	Total
No organism found in smear or culture	53	51	104
Gonococcus	89	64	153
Staphylococcus:			
Staphylococci	55	48	103
" and diphtheroids	25	20	45
Staphylococcus albus	17	13	30
" aureus	13	11	24
Other coccal organisms:			
Gram-positive cocci	6	1	7
Meningococci	4	2	6
Pneumococci	3	2	5
Streptococcus haemolyticus	2	3	5
" viridans	1	—	1
Micrococcus catarrhalis	2	—	2
" tetragenus	2	1	3
Bacilli:			
Diphtheroids	20	31	51
Koch-Weeks	8	2	10
Coliform	6	2	8
Gram-positive	3	1	4
Gram-negative	3	10	13
Xerosis	3	—	3
Morax-Axenfeld	3	3	6
Hoffmann	2	—	2
Friedländer	1	4	5
Total	322	269*	591

* Inclusion bodies found 27 times: twice in association with organisms and 25 times without organisms.

Summary

In the earlier series of 273 cases of ophthalmia neonatorum treated by the oral administration of sulphapyridine, 61.9% showed clinical cure within 8 days.

In the present series, in which a standard dosage was used in 258 cases, 133 were treated with sulphapyridine, 43 with sulphathiazole, 28 with sulphamezathine, and 31 with sulphadiazine; two or more sulphonamides were used in the remaining 23 cases, which were either resistant or intolerant to the sulphonamide used initially. In these 258 cases clinical cure was obtained within 8 days in 85.7%.

A high proportion of cases (29.9%) showed clinical cure within 3 days; 55.8% required from 4 to 8 days for clinical cure; and 14.3% showed a protracted course.

There was no appreciable difference in the action of the four sulphonamides used. Clinical cures within 8 days were obtained in 88.7% of cases treated with sulphapyridine, 93.0% of those treated with sulphathiazole, 92.9% of sulphamezathine cases, and 83.9% of

sulphadiazine cases. Sulphapyridine, because of its greater toxicity, appears to be the least desirable of these sulphonamides.

Gonococcal cases responded more rapidly to sulphonamide therapy than did the non-gonococcal cases; 51.7% of the 60 gonococcal cases showed clinical cure within 3 days, against 23.2% of the non-gonococcal cases. By the end of 8 days 90.0% of all gonococcal cases and 84.3% of non-gonococcal cases were clear.

In the present series inclusion bodies were found in 27 instances: twice in association with organisms and 25 times without such association. These cases responded well to sulphonamide therapy.

Only exceptionally is a case completely resistant to sulphonamide therapy. It is more a matter of sluggish response than of total resistance.

The routine at White Oak Hospital now includes administration of the sulphonamides for 3 days after clinical cure; fewer relapses are now observed.

We are indebted to Dr. E. N. Young of the Southern Group (L.C.C.) Laboratories for her painstaking bacteriological examinations. We are also obliged to Dr. G. Heisler, and to the Matron and Sister Condon of White Oak Hospital, for their collaboration.

REFERENCES

- Sorsby, A., Hoffa, E. L., and Smellie, E. W. (1942). *British Medical Journal*, 1, 323.
Wong, R. T. (1942). *Arch. Ophthalm.*, Chicago, 27, 670.

THE BLOOD PRESSURE IN MIDWIFERY

BY

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A study of the blood pressure during the later months of pregnancy is a recognized part of adequate ante-natal care, yet often it is neglected as soon as labour begins. This is all the more surprising since the value of blood-pressure records has been well established on the operation table and in the prevention and treatment of traumatic shock, and the same influences—drugs, haemorrhage, and trauma—are present to some extent in most labours.

The literature on this subject is very scanty; the most useful articles in the English language are those by Cook and Briggs (1903), Bailey (1911), Donaldson (1913), Schwarz (1923), Simons and Rasmussen (1925), and Matthews (1939). The subject is also practically ignored, apart from mentioning the blood pressure in eclampsia and toxæmia, in all the recent textbooks on midwifery that I have seen.

I have made records of the blood pressure, both systolic and diastolic, in all my midwifery for over two years, and I find them very instructive in assessing the effects of management and treatment, especially analgesia and anaesthesia. The technique is simple, and well within the scope of the general practitioner, even when working alone. The chief difficulty arises when the doctor is distracted from the mother to attend to urgent foetal asphyxia, for it is at this time during the third stage of the labour that the most important changes in blood pressure take place. I firmly believe that if more attention were paid to the blood pressure incipient shock would be recognized earlier while still amenable to simple treatment, and the lives of many of our mothers would be saved.

Types of Blood-pressure Curve

The first problem to be solved is that of the normal—that is to say, the blood-pressure curve in a straightforward delivery uninfluenced in any way by drugs or obstetric manipulations. It has been stated by Halls Dally (1936) that "during labour there is a progressive elevation of blood pressure, even to considerable heights as the pains become more intense, reaching a maximum as the head of the infant is expelled. After delivery the pressure rapidly falls." In my experience this description is very misleading, and there is far less disturbance of blood pressure than one would suppose.

Graph A is a typical blood-pressure curve, and probably approximates to the normal, even though nitrous oxide analgesia was given towards the end of the second stage. Exactly similar curves are recorded when no form of analgesia is given, and also when the agent used is weak chloroform or trichlorethylene. It will be seen that in labour there is a rise of about 20 mm. Hg, followed by a short-lived fall after delivery, partly due to reduced pulse pressure, but also shown in the diastolic pressure as well. In this particular case the blood pressure as the head was being born was not recorded, but I have rarely found it much higher. Soon

after delivery the blood pressure rises again, and this coincides with the return of uterine contractions. After the first day there is a gradual fall during the puerperium, but Donaldson (1913) has pointed out that this fall is no more than one would expect to find in any patient who was confined to bed.

I have recorded not only the systolic pressure but also the diastolic, and no account has been taken of the rise in pressure during pains, the recorded figures being taken only between each contraction. Although it may not be strictly accurate, I have assumed for all

delivery is also negligible, but soon afterwards there is a sharp increase in the pulse pressure. In my experience such an increased pulse pressure soon after delivery invariably coincides with a sharp bout of shivering. This is an observation which, so far as I am aware, has not previously been made, but I think it is of considerable importance in explaining the changes which take place in the third stage of labour.

A theory has been handed down from the time of William Smellie that the sudden emptying of the uterus at birth tends to cause

GRAPH A.—Primipara aged 23. Labour Feb. 26-27, 1942. R.O.P. rotating spontaneously to R.O.A. Dry labour. No drugs given other than N_2O + air. Full-term male child, 6½ lb.

GRAPH B.—Primipara aged 23. Labour Dec. 26-27, 1941. Persistent R.O.P. Full-term female child, 7 lb.

practical purposes that the diastolic pressure is a measure of the peripheral resistance to the circulation, and therefore an index of the vasomotor tone, and that the systolic pressure represents the sum of this peripheral resistance plus the pulse pressure, representing cardiac output. In studying these records I would stress the value of the diastolic pressure, and point out that the systolic pressure alone tells us only part of the story.

Graph B is a second type of blood-pressure curve, and is nearly as common as the first example. In this type there is very little or no increase in the blood pressure during labour, and the fall after

collapse. His pupil, John Harvie (1762), described it as "too great quantity of blood rushing into ye relaxed vessels of ye abdominal contents, whereby ye head is robbed of its due proportion, and dangerous faintings ensue." This phenomenon was thought comparable with the collapse which sometimes follows paracentesis abdominis. To it has been attributed not only obstetric shock but also the fall in systolic pressure (Cook and Briggs, 1903) and the shivering bout (Phillips, 1931). It is interesting to note that the splanchnic pool theory of shock has only recently been discarded. Bailey (1911) was the first to doubt the theory, and suggested that

the fall in systolic pressure seen after delivery might be due rather to an abrupt cessation of pain, anxiety, and muscular activity. The fact, unknown to him, that the drop is largely in the pulse pressure and that it also partially recovers when contractions begin again, supports his view. Soon afterwards Donaldson (1913) firmly rejected the whole idea on the good grounds that the splanchnic vessels have muscular fibres in their walls, and do not rely on the pressure of the abdominal viscera for their support. In other words, he recognized the presence of vasomotor tone. Clinical experience rejects also the idea of dangerous faintings, which we know are rare even when the patients have the most uncontrollable attacks of shivering. I suggest that the shivering bout is one symptom of a vasomotor reflex in which the skin vessels play a prominent part. Wiggers and Werle (1942) have pointed out that in such a skin reflex the salivary glands are included, and we know that our patients often at this time ask for a sip of water. Nevertheless there is an element of truth in the old theory, for in Graph A the fall in blood pressure definitely includes the diastolic pressure, and I would suggest that, while most of the drop in systolic pressure is due to the cessation of the pains, there is a temporary relaxation of the uterine sinuses in which a proportion of the maternal blood may be pooled until such time as the contractions of the third stage begin. This is a point of some importance in cases of atonic post-partum

The most dramatic changes in blood pressure are seen in complications of the third stage of labour. For example, Graph D is from a case of post-partum haemorrhage, and shows a rapid fall in the systolic pressure from over 130 mm. Hg to 88 mm., with a corresponding rise in the pulse rate. Yet in this case the diastolic pressure dropped only a few millimetres, showing that there was no vasomotor failure. As soon as the usual measures for controlling haemorrhage had been taken, recovery was steady and assured. Thus the effect of the haemorrhage as well as the effectiveness of the treatment can be seen in the blood-pressure curve.

Graph E affords a marked contrast, for although there was a small post-partum haemorrhage, the picture was completely overshadowed by shock, shown by faintness, giddiness, and a cold perspiration, and a very severe drop in both the systolic and the diastolic pressure to 80/60 mm. Hg, which is within the limits described by Hewer (1943) as critical hypotension. Fortunately energetic treatment brought about a prompt recovery. A point of practical importance arises in such cases; it will be seen that no attempt to repair the perineum was made until the blood pressure had been restored to normal limits, and then a local anaesthetic was used in preference to a general one rather than run any risk of upsetting the vasomotor balance again. It has been pointed out by Miles Phillips (1931) that even the small amount of trauma caused

GRAPH C.—Primipara aged 36. Labour May 3, 1942. L.O.A. Forceps used. Ether anaesthesia after N_2O -air analgesia. Full-term female child, 8½ lb. Exceedingly fat patient: abdominal examination impossible.

haemorrhage. It should be remembered that the uterine blood vessels during pregnancy lose their muscular coats and come to rely for their support at term on the muscle fibres of the uterus (DeLee, 1938).

A valuable clinical lesson which may be learnt from these records is the depressant effect of drugs used for analgesia and anaesthesia. The barbiturates are the worst offenders, and a drop of 20 mm. Hg, both systolic and diastolic, is commonly recorded after the normal doses. Graph C is an example of such a fall after the use of nembutal, and I have seen just the same after seconal. This graph, in addition, shows well the effect of prolonging ether anaesthesia, given in the first place for low forceps, through the third stage of labour to cover a small perineal repair operation. While it is well known that chloroform anaesthesia produces such hypotension, it is not always realized that ether does so too once the initial stimulant effect has passed off. The importance of hypotension under anaesthesia as a precursor of shock has recently been stressed in an article by Dodd and Prescott (1943). If such hypotension can be demonstrated in the mother, what must be the effect on the foetal circulation? Among the causes of foetal asphyxia anaesthetics and analgesic drugs administered to the mother play a very considerable part (Shaw, 1943). One is justified, I think, in assuming that any depression of the maternal circulation must be reflected in that of the foetus, at least up to the moment when independent respiration begins. Further research along these lines might lead to a reduction in the stillbirth rate.

by the operation of perineal repair may be sufficient to take away the patient's chance of recovery.

On Graph C I have noted the rise of systolic pressure produced by a first-stage uterine contraction. I have frequently noted down the blood-pressure changes produced by the pains, and generally have found an increase of from 18 to 25 mm. systolic and 8 to 18 mm. diastolic. I have never seen anything like the 50-mm. rise in the systolic pressure mentioned by Donaldson (1913), and think this must be very exceptional. It is not difficult to explain the systolic increase as the result of muscular exertion and physical pain (Cannon, 1929), but this will not account for the diastolic rise, which is not affected by them (McDowall, 1941). As I have already mentioned, the uterine sinuses are supported by the muscular fibres of the uterus and therefore the uterine circulation must be compressed with each contraction. It has been shown by Chassar Moir (1933-4) that this pressure is of the order of 50 mm. Hg during the first stage of labour, rising to about 85 mm. during the second stage. Since the normal diastolic pressure is between 70 and 80 mm. it follows that during each pain the uterine pressure would close these sinuses and stop the circulation if there were not an increased diastolic pressure to raise the mean blood pressure over this normal figure. It is possible that the increased diastolic pressure is brought about by an increased tone in the peripheral circulation in order to maintain a sufficient supply of blood to the contracted uterus.

Apart from example C, in which a mild degree of toxæmia before labour is shown to have responded rapidly to treatment, I have

regarded as a danger sign, and the blood pressure should be restored before anything else is done, the only exception to this rule being the minimum steps necessary to stop haemorrhage. It is not always appreciated that in cases of traumatic shock the precipitating injury precedes the secondary factors, such as pain, exhaustion, drug depression, and injudicious rough handling, which join in to complete the clinical picture; whereas in labour most of these factors will have been present for some hours before the final trauma which determines and initiates shock. The ultimate state of shock is the same, but the sequence is different. When, therefore, a long and difficult labour is anticipated we have a particularly favourable opportunity of controlling these accessory factors, especially if we study the blood pressure.

I would therefore urge the general practitioner to use his sphygmomanometer at the bedside in his midwifery, and cannot do better than quote in support of my plea the words of Prof. Miles Phillips (1936), who made it his practice to do so, although he has not published his figures. Speaking before an American Association of Surgeons, he said: "Neither she nor her husband was concerned with the maternal mortality rate—only four, five, or six in a thousand—but they did expect you to take every precaution."

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REFERENCES

- Adair, F. L., Hunt, A. B., and Arnell, R. E. (1936). *J. Amer. med. Ass.*, **107**, 1036.
 Bailey, H. C. (1911). *Amer. J. Obstet.*, **64**, 260.
 — and Driscoll, W. P. (1926). *Amer. J. Obstet. Gynec.*, **11**, 287.
 Cannon, W. B. (1929). *Bodily Changes in Pain, Fear, Hunger and Rage*, New York.
 Cook, H. W., and Briggs, J. B. (1903). *Johns Hopk. Hosp. Reps.*, **11**, 451.
 Dally, J. F. Halls (1936). *Brit. Ency. med. Pract.*, **2**, 505, London.
 DeLee, J. B. (1938). *Principles and Practice of Obstetrics*, Philadelphia.
 Dodd, H., and Prescott, F. (1943). *British Medical Journal*, **1**, 345.
 Donaldson, M. (1913). *J. Obstet. Gynaec. Brit. Emp.*, **24**, 133.
 Harvie, J. (1962). Manuscript Lectures at the Liverpool Med. Inst. Library.
 Hewer, C. L. (1943). *Recent Advances in Anaesthesia*, p. 215, London.
 Hirst, J. C. (1910). *N.Y. St. J. Med.*, **91**, 1204.
 McDowall, R. J. S. (1941). *British Medical Journal*, **2**, 41.
 Matthews, H. B. (1939). *J. Amer. med. Ass.*, **113**, 1183.
 Moir, Chassar (1933-4). *Trans. Edinb. obstet. Soc.*, **54**, 93.
 Phillips, Miles (1931). *British Medical Journal*, **1**, 833.
 — (1936). *Trans. Amer. Ass. Obstet. Gynec. abd. Surg.*, p. 14.
 Schwarz, O. H. (1923). *Amer. J. Obstet. Gynec.*, **6**, 155.
 Shaw, Wilfred (1943). *Textbook of Midwifery*, p. 493, London.
 Simons, E. J., and Rasmussen, C. C. (1925). *Minnesota med. J.*, **8**, 303.
 Wiggers, C. J., and Werle, J. M. (1942). *Amer. J. Physiol.*, **136**, 421.

NERVOUS BREAKDOWN IN THE NAVY DOMESTIC DIFFICULTIES AS A CAUSAL FACTOR

BY

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While it is generally agreed that prolonged psychotherapy has no place in the treatment of psychiatric casualties in the Services in wartime, this need not mean that the psychiatrist should concern himself only with diagnosis and institutional efforts at rehabilitation. In this paper the part played by the domestic affairs of the sailor in determining the onset and gravity of nervous breakdown is considered and suggestions are made for investigating and treating these difficulties more effectively. Psychological motivation is said (Kretschmer, 1934), to be due to combinations of causes, of which the least socially and morally acceptable possesses the greatest driving force and is furthest removed from consciousness, whereas that which the patient considers to be the most easily acceptable is in the forefront of consciousness and is the manifest cause of any reaction. Wartime propaganda is directed towards assigning to patriotism and devotion to duty the highest ethical values and stressing the subordination of the individual and the family to the State. In this present struggle, waged by conscript armies and navies, such a fundamental change of outlook might well give rise to conflicts, of which the most potent cause need not be the most obvious.

Aetiological Classification of Naval Psychiatric Material

In classifying the causal factors in mental illness it is usual to make a broad distinction between those that are determined mainly by the personality of the patient and those that are chiefly environmental. Though it is seldom possible to make a

rigid distinction between endogenous and exogenous, yet for purposes of treatment and prognosis it is almost essential to divide the facts of the history in this way. Exogenous factors can be considered as forming a graded series, the members of which will vary in their closeness to the centre of the patient's personality. On theoretical grounds it would be reasonable to assume that those factors which most intimately affect this hypothetical nodal point of the personality would have the greatest strength in causing a nervous breakdown: the effects of domestic trouble are explicable on this basis.

Wartime psychiatry is greatly simplified because the individual environmental stresses in war are much more constant than they are likely to be in peacetime; for example, the living conditions of a sailor in his ship, which may be his home for years at a stretch, are so invariable as to be comparable to those artificially produced in a psychological laboratory. The effects of stress are evenly distributed throughout the ship's company, and, although there are differences in responsibility among the various ranks and ratings, so far as exposure to what are usually termed "the hazards of life afloat" are concerned all can truly be said to be in the same boat. The realization of this has led to the conception that the breakdown of a small percentage of a ship's company following an action is an indication of the poor morale of those who succumbed, and, because the majority stood up to the test, cannot be attributed to the conditions of the action. In some cases this may be true, but it is probable that this assumption accounts for only a small number of the men who break down under the stress of action.

On the basis of the observation of fairly large numbers of these cases, it appears more likely that unfavourable reactions to stress are due, in order of frequency, to the following causes: (1) constitutional weakness of the personality; (2) combinations of environmental factors; (3) simple bad morale, in the sense of "yellowness" or lowered social responsibility. It is with the second of these groups that this paper is chiefly concerned.

Effects of Stress

By the time a man becomes a frank psychiatric casualty, unless he belongs to the low-morale group, he has usually had to readjust or repress his former scale of ethical values, and the ease with which he does this depends upon his personality. Both in the last war and in this it has been noted (Beaton, in *The Naval Medical History of the War*; Curran and Mallinson, 1941) that the better types suffer affective changes in the direction of either depression or elation, usually combined with anxiety, whereas their less meritorious messmates react with hysterical symptoms. It has been observed, moreover, that affective disturbances in good personalities, when they are due to the effects of war service uncomplicated by other factors, respond surprisingly well to ordinary hospital conditions. It is those cases in which the causal factors are multiple that sometimes present unusual clinical states and, on the whole, do badly. In the Services, the return of a man to duty is a fair measure of the success of treatment; invaliding usually implies failure, and the invaliding rate may be regarded as an indicator of the virulence of a condition.

In the Navy there appear to be two groups of exogenous factors, which are not directly connected with the hazards of war: (a) the conditions and type of service, including reactions to authority, disappointments over promotion or the lack of official recognition of services, and other specifically Naval determinants; (b) domestic difficulties. Of these two groups the former is the commoner; but the latter, as will be shown, can give rise to the most virulent and intractable reactions, leading to invaliding in a high proportion of cases.

The home life of the sailor has been the subject of ribaldry and a cause of unhappiness for as far back as the lives of sea-going men are recorded. Among sea-faring peoples the subject has become part of the national mythology; it is not recorded that Jason or Ulysses suffered from anxiety states, but their behaviour on landing after long voyages showed considerable apprehension as to what might have happened at home in their absence, and the Norse sagas abound with references to these additional hazards of sea life. The Admiralty has evidently given much thought to this matter, and in peacetime every effort is made to enable the sailor to lead a double life with the minimum of discomfort and disruption of his material home.