Contemporary Themes

Congenital Malformations and the Problem of their Control*

C. R. LOWE

British Medical Journal, 1972, 3, 515-520

Summary

The possibility of preventing or reducing the prevalence of human congenital malformations is discussed in the light of current knowledge about their aetiology. The original data presented are derived from an epidemiological study of all the infants (92,982) born in the three years 1964-6 to women resident in a defined area of South Wales and all the congenital malformations (3,242) discovered in those infants by the second anniversary of their birth.

Introduction

There are four good reasons why I have chosen the topic of congenital malformations and the problem of their control as the subject for my contribution to this distinguished series of lectures.

The first is that in the developed countries as the tide of mortality from infectious disease has receded malformations have been left exposed as the leading and therefore most challenging cause of death and disability in infancy and childhood. For example, in England and Wales about one-quarter of all deaths between the 28th week of pregnancy and the end of the first year of postnatal life are now attributable to these conditions. Next, there is much evidence to suggest that they have a large environmental component in their aetiology, and this appears to offer the hope that they may be prevented or at least subjected to some measure of control, as the infectious diseases have been controlled. Thirdly, for some of the major malformations advances in surgical and other therapeutic techniques are proving of life-saving if not always of life-enhancing importance. Reliable information about the frequency with which they occur and the results of treatment is therefore of great importance.

The final reason is that I know a little about the subject—a necessary if not sufficient reason for a public lecture. When I moved to Cardiff in 1962 I was told that births in coal-mining valleys of South Wales were thought to carry an unusually high prevalence of malformations of the central nervous system.¹ So in 1964, with the financial support of the Association for the Aid of Crippled Children, I launched a three-year study of the prevalence of all congenital malformations in South Wales.

How the study was conducted has been described elsewhere.² All I need say here is that the original data I shall be using relate to all the infants (92,982) born in the three years 1964-6 to women resident in a defined area in South Wales and all the congenital malformations (3,242) discovered in those infants by the second anniversary of their birth.

To effect any measure of control some understanding of aetiology is necessary. For much of my presentation, therefore I shall be discussing what is known about this, with illustrations, where possible, from the South Wales survey. In this discussion I shall not be concerned with subcellular mechanisms or with the metaphysics of causality. Epidemiological knowledge has often provided a key to the control of a disease before, sometimes long before, the bacterial, biochemical, or other mechanisms involved in its aetiology were understood—for example, cholera and scurvy. Control becomes possible when enough is known about the chain of cause and effect for us to be able to break that chain.

Nature and Nurture

There has been much unproductive controversy about the relative importance of nature and nurture in the genesis of malformations. At the beginning of this century the balance of the argument tilted heavily to the genetic side. This tended to inhibit aetiological research, for at that time the belief that malformations were due to the drawing of an unlucky number in the genetic lottery that is set up when our father's spermatozoa meet our mother's ovum carried the implication that nothing could be done to prevent them. This, of course, is not true. We have not yet learnt how to manipulate the human genotype but we can sometimes change the phenotype. For example, until quite recently the phenotype of phenylketonuria was characterized by severe mental subnormality. Nowadays it is sometimes marked not by subnormality but by the need to take an expensive and unpalatable diet.

The controversy is best forgotten. Although some of the rarer malformations show straightforward Mendelian ratios in their familial aggregations most of the common malformations do not conform to the pattern of dominant, recessive, or sex-linked inheritance. To explain this geneticists have retreated through the hinterland of low penetrance into the shelter of polygenic inheritance with environmental triggers. Genetic models so flexible that they will fit almost any observed pattern of familial distribution are not very helpful. Most malformations have a multifactorial aetiology which includes both genetic and environmental components. It is the epidemiologist's concern to define the environmental components in the hope that some of them may be amenable to control.

To conclude this section I must refer to the curious and puzzling lack of twin concordance for neural tube defects. Identical twins by definition are more identical than non-identical twins in respect of everything, including disease in idence. Curiously this is not true of malformations of the central nervous

* This paper is based on the Cutter Lecture read at the Harvard School of Public Health, Boston, in 1971.

system. Yen and MacMahon³ reported 108 instances of affected twins in eight consecutive series of cases of neural tube defect reported in the literature, and in none of the twin pairs, like-sexed or unlike-sexed, was the co-twin also affected. Furthermore the sex distribution in these sets of twins was not disturbed in the way that would be expected if the concordant like-sexed twin pairs had been aborted early in pregnancy.

In a small way our data from South Wales tell the same story. In our series we found 12 twin pairs of which one member had a neural tube defect. There was no concordance. It is difficult to reconcile lack of concordance in identical twin pairs with any genetic hypothesis. It is as difficult to reconcile it with any environmental hypothesis. This very curious incident of the dog that did not bark in the night still awaits its Sherlock Holmes for elucidation.

Laboratory Evidence of Environmental Influences

There is now a wealth of evidence from animal laboratories that given the appropriate animal, the appropriate dose, and the appropriate timing very many physical and chemical agents can be teratogenic. These include, to mention only a few, ionizing radiations, hypoxia, hypothermia, ultrasonic waves, folic acid antagonists, vitamin deficiencies, vitamin excesses, and a number of commonly used therapeutic substances such as salicylates, insulin, streptomycin, the hypoglycaemic sulphonamides, androgens and progestogens, and the corticosteroids. However, this is not the main contribution that experimental teratology has made to our understanding of human teratogenesis. More important is the way in which it has enlarged our understanding of how mammalian embryos react to teratogens. The following three general principles have particular relevance to the study of malformations in man.

Dose Response.—As the dose of a teratogen is increased there is first a no-effect range, then a teratogenic range, and finally embryolethality. This has two implications: because a valuable therapeutic substance has been shown to be teratogenic to laboratory animals at high doses, this does not necessarily mean that it will be teratogenic to the human embryo when the mother is on much lower therapeutic doses; and the fact that the teratogenic range is often very narrow and moves rapidly into embryolethality suggests that epidemiological studies of human malformations should pay much more attention to the incidence of early pregnancy loss than they have in the past.

Timing of Insult.—Different fetal organs are sensitive to teratogenic agents at different durations of gestation. One teratogen—for example, anoxia—can be made to produce widely differing malformations if it is exhibited at the appropriate points in pregnancy; one type of malformation—for example, of the neural tube—can be produced by widely different teratogens if they are exhibited at the appropriate point in pregnancy. We should be wise, therefore, not to think in terms of a search for “the” environmental cause of, say, neural tube defects in man, since almost certainly many different environmental factors are involved.

Maternal Tolerance.—The teratogenic and even the embryolethal range of most teratogens is usually well below the level of the threshold dose for maternal toxicity. This means that a fetus can be severely damaged, even killed, by substances well tolerated by its mother, a fact brought home with tragic force by thalidomide. Agents causing only marginal disturbances of the maternal metabolism, perhaps undetectable by ordinary clinical methods, may therefore be playing an important part in the aetiology of some human malformations.

Direct Evidence of Environmental Influences in Man

The list of specific agents known to affect the human fetus is still disappointingly short. This is all the more surprising when a belief that a mother's experiences in pregnancy can affect her

unborn child is part of every country's folk-lore. In the seventeenth century Sir Thomas Browne wrote in *Religio Medici* that “Every man is some months elder than he bethinks him; for we live, move, have a being, and are subject to the actions of the elements, and the malice of diseases, in that other World, the truest Microcosm, the Womb of our Mother.” Three-hundred years later we can be certain that the human embryo is indeed subject to the actions of the elements and the malice of disease, but that certainty is still for the most part based on indirect evidence. Syphilis and rubella mark the beginning of the disease story, and toxoplasmosis and cytomegalic inclusion virus pretty well end it. We can bring in ionizing radiations under the heading of actions of the elements. We can add a very short list of confirmed drug teratogens: thalidomide, aminopterin, the androgens, progestogens and oestrogens, cortisone, and iodides. We can compile a longer but still short list of reports of malformations suspected to have been associated with the taking of drugs early in pregnancy. And that, at present, is the end of the story. Taken together all the proved specific teratogens probably account for less than 5% of human developmental anomalies. For example, in non-epidemic years rubella accounts for not more than 2-4% of all cardiac malformations.

Indirect Evidence of Environmental Influences

Almost all the evidence relates to non-specific and uncertain markers of as yet unidentified specific environmental triggers. In this section I shall illustrate the nature of that evidence, mostly from the South Wales survey.

GEOGRAPHICAL DIFFERENCES

There are quite remarkable differences between countries in the reported incidence of various malformations. But the interpretation of international differences in disease incidence, always hazardous, is nowhere more beset with difficulties than in the field of congenital defects. However, if we limit ourselves to neural tube defects, which we would expect to be reasonably well ascertained, we find that there are indeed remarkable differences between different countries (Table I). Neural tube defects are eight times more common in South Wales than Japan. It is difficult to believe that all or even most of this difference is genetic in origin.

We are on much firmer ground when we point to geographical differences within a country as indirect evidence of environmental influence. Mortality from neural tube defects between the 28th week of pregnancy and the end of the first year of life (stillbirths plus infant deaths) is much higher in the north and the west of England and Wales than it is in the south and the east.⁸ In South Wales the incidence is much higher in the coal-mining valleys than it is in the coastal plain (Fig. 1). These differences are almost certainly environmentally induced.

TABLE I—Incidence of Neural Tube Malformations in South Wales compared with Other Selected Locations

Location	Incidence per 1,000 Total Births		
	Anencephalus	Spina Bifida	Anencephalus and/or Spina Bifida
South Wales (1964-6) ^a	3.09	3.92	7.01
England (Birmingham 1950-4) ^a	2.01	2.18	4.19
Hungary (Budapest 1963-7) ^b	1.10	1.63	2.73
Israel (1959-60) ^c	0.61	0.74	1.35
Japan (1948-54) ^d	0.63	0.26	0.89

TEMPORAL DIFFERENCES

A long-term epidemic of neural tube defects in the north east of the United States has recently been uncovered.⁹ The incidence

FIG. 1—Incidence per 1,000 births of malformations of the central nervous system in South Wales 1964-6.

of neural tube defects began to rise shortly after the first world war, reached a peak three times that of the original level around 1930, and then fell back to the earlier level in the 1950s. There have also been long-term swings in the incidence of anencephalus in Dublin, Scotland, and Birmingham.¹⁰ In these three localities the stillbirth rates from anencephalus declined during the second world war, reached their lowest levels a few years after the war, rose steadily to reach peaks round about 1958-60, and are now declining again. In Dublin the peak rate in 1960-1 was more than three times the trough rate in 1944-5. The greater part of the large differences in these long, slow epidemics can be due only to environmental changes.

Twenty years ago McKeown and Record¹¹ drew attention to a seasonal variation in the prevalence at birth of anencephalus (but not of spina bifida) for Scotland and for Birmingham. The rates were highest in December and lowest in May. In Scotland there is still some suggestion of seasonal differences in anencephalus stillbirth rates, but this is becoming much less marked than it used to be (Fig. 2). Curiously, in our own data we have found little evidence of cyclic variation for neural tube defects

FIG. 2—Anencephalus stillbirth rate by month of birth in Scotland 1939-58 and 1963-8.

SOCIAL CLASS

One of the most convincing of the indirect indicators of environmental influence is the association of malformations of the central nervous system with socioeconomic circumstances. In

FIG. 3—Anencephalus stillbirth rates by social class in Scotland 1963-8.

FIG. 4—Incidence of anencephalus and/or spina bifida by social class in South Wales 1964-6.

but strong evidence of a cyclic pattern for hypospadias, with November as the month of conception with the highest incidence.¹² Once again these temporal differences provide clear evidence of environmental influences at work.

Scotland for the period 1963-8 the anencephalic stillbirth rate increased regularly from 1.1 per 1,000 births in social class I (professional classes) to 3.8 in social class V (unskilled workers), a threefold difference (Fig. 3). Some difference is also in evidence

in South Wales. However, although the incidence is low in the professional classes there is no regular trend through the other social classes, and the differences appear to be mainly between manual and non-manual occupations (Fig. 4). In our study, apart from cleft lip, which was also commoner among the children of manual workers than non-manual workers, no other malformation showed any clear social class pattern.

FIG. 5—Incidence of Down's syndrome by maternal age in South Wales 1964-6.

MATERNAL AGE AND PARITY

With one striking and well-known exception maternal age does not appear to be of great consequence either in our own survey or in the conflicting reports in the literature. The exception is, of course, Down's syndrome (Fig. 5).

Again with one notable exception the evidence in the literature of a parity effect is also conflicting and inconclusive. The exception is neural tube defects, for which all agree that there is a striking first-birth effect, a much lower incidence in second and third births, and a raised incidence at higher parities (Fig. 6).

FIG. 6—Incidence of anencephalus and/or spina bifida by maternal age and parity in South Wales 1964-6.

To complete the record I will mention that in our series in addition to anencephalus and spina bifida several other malformations showed significant but less striking relationships with parity. The incidence of malformations of the heart and great vessels increased slightly with parity, talipes decreased with parity, and urogenital malformations (other than hypospadias) showed both a primiparity and a high-parity effect.

OTHER FACTORS

I must now draw attention to the loose way in which I have used the term "incidence." What in fact I have been describing is the prevalence of malformations at the time of birth. We must remember that in the animal laboratory most influences harmful to the developing fetus have only a narrow teratogenic range, and above that range the fetus dies and is aborted or absorbed. A low prevalence at birth may therefore indicate either a low incidence at conception or a greater loss than usual of malformed embryos or fetuses early in pregnancy. A high prevalence at birth may mark a high incidence at conception or a uterine environment favourable for the survival through to the 28th week of pregnancy and beyond of a higher proportion than usual of malformed fetuses. We are in the surrealist world of Groucho Marx's remark that "the contrary is also true."

For this reason a history of previous abortions in relation to the prevalence at birth of malformations is of great interest. A few investigations have, in fact, disclosed a history of repeated abortion in mothers giving birth to certain malformations, notably neural tube defects and defects of the heart. Inevitably all such inquiries if they are to include the crucial first trimester are retrospective and therefore subject to well recognized biases (women who have been delivered of a malformed infant are more likely to remember and report previous abortions than women who have had normal infants). For what it is worth we have found in South Wales that malformation rates were significantly higher among women reporting one or more previous abortions than among those not reporting abortions for spina bifida, anencephaly, cardiac defects, alimentary atresias (but not cleft lip), urogenital defects other than hypospadias, dislocation of the hip, and polydactyly. In our series there were 102 women with their first registered birth—that is, of 28 weeks' gestation—who reported that they had previously had three or more miscarriages. Of the 102 infants born to them 12 had congenital defects—four with neural tube, four with cardiac, and four with other defects—(Table II).

TABLE II—Incidence of Malformations per 100 Births related to History of Previous Miscarriage

No. of Previous Miscarriages	Maternal Parity		
	0	1	2 and Over
0	3.9 (29,774)	3.1 (23,685)	3.2 (24,714)
1	4.8 (1,879)	4.0 (2,605)	4.5 (4,778)
2	5.4 (295)	4.9 (553)	4.3 (1,076)
3	11.8 (102)	4.5 (202)	3.7 (539)

Numbers of related births are given in parentheses.

Direct evidence of the loss of high proportions of malformed fetus by spontaneous abortion has now been provided by Nishimura¹³ in his unique survey of the incidence of gross malformations in a very large population of early fetuses obtained from women whose pregnancies had been artificially terminated (mostly for socioeconomic reasons and therefore of rather higher age and parity than all deliveries). In his series the prevalence of gross malformations in the fetuses of 8-18 weeks' gestational duration was about 5% (the prevalence at birth of correspondingly gross malformations is about 1%) and the prevalence of neural tube malformations was 13 per 1,000 (the prevalence of birth of these malformations is, as we have seen, remarkably low in Japan—a little over 1 per 1,000). This suggests that at the most only about one out of five of the gross malformations laid down in early pregnancy survive to be recognized at birth. What comes down at birth is no guide to what went up at conception.

Descriptive epidemiology is fruitful when it leads to the formulation of hypotheses which can be put to the analytical or experimental test. What hypotheses does our meagre harvest of epidemiological facts suggest? If we limit ourselves to neural

tube malformations, the relation to social class and high parity and the cyclic and secular changes have suggested that infection or nutrition may be involved. The search for infective agents has been thorough and almost completely unrewarding, and there is no evidence that overt maternal malnutrition plays any part in the story. If nutrition has a role it is in respect of deficiencies that are of no importance to the mother, such as, perhaps, relatively low serum folic acid levels, or of food contaminants or additives, such as the preservatives, detergents, weed-killers, antibiotics, and colouring materials so many of our foods now contain. But this is mere speculation and the search is still on.

There is one hopeful lead from another direction altogether. Last year Stocks⁸ pointed out that in the 15 hospital regions of England and Wales mortality from malformations of the neural tube had a high positive correlation with mortality from cardiovascular disease in women aged 25-54 whereas other malformations had insignificant negative correlations. He suggested that because mortality from cardiovascular disease is known to be associated with the softness of the local water supplies there may also be a soft-water factor in the aetiology of neural tube defects. We have been able to test this hypothesis in South Wales by relating the incidence of spina bifida and anencephalus in 48 local authority areas to not very reliable estimates of the mean total hardness of the related water supplies. There was a significant negative correlation.¹⁴ We are not yet convinced, however, that this is a direct relationship, and further studies are in progress.

Possibilities of Control

The purpose of this exploration of the epidemiology of congenital malformations has been to see what hope there is of our being able to control this now major cause or group of causes of uterine and postnatal death and disability. It would be wrong to say of our journey, as Gertrude Stein said of Oakland, California, "when you get there, there is no there there." But I have to admit that the landscape is rather bleak.

Firstly, of course, universal vaccination of girls against rubella would effect a small reduction in the prevalence of congenital heart disease and cataract. But so far that and the early diagnosis of syphilis are the only preventive measures of the traditional type that are available to us.

Secondly, there are the possibilities opened up by the clinical discipline of genetic counselling. But nearly all the developmental defects known to be due to chromosomal abnormalities and mutant genes of large effect are extremely rare, and if genetic advice were freely available to all married couples known or suspected to be at risk (and the advice taken) the effect this would have on the general prevalence of congenital defects would be very small indeed, while its effect on the pool of harmful genes in the population would be negligible. Rather more hopeful is the growing possibility of identifying a small proportion of the seriously malformed fetuses early in pregnancy by means of amniocentesis, ultrasonics, refinements of radiography, and the sampling of fetal blood and amniotic fluid. There are difficult ethical problems involved here, and not only those concerned with the taking of life by abortion. There is also the danger of applying research techniques to the general population before their costs measured in terms of morbidity and mortality to mother and fetus have been reliably assessed.

Then there is the need to control the hazard of pharmacologically-induced malformations. The risk is small and uncertain, but the cohort of thalidomide children moving through the schools in England and Wales and in Germany bears witness to its reality. In Britain at the present time the birth rate for all women (married and unmarried) aged 20-29 years is 160 per 1,000; this means that about 1 in 20 young women at the time that they are consulting their doctors or buying drugs from a pharmacy are in the vulnerable first trimester of pregnancy. When this is related to the injections, tablets, and pills which women are already taking and to the rate at which more and

more active pharmaceutical preparations are being elaborated—tranquillizers and stimulants, steroids, and antibiotics—there is clear cause for concern. The obvious preventive measure is to advise all women of childbearing age that when they are at risk of pregnancy (and certainly when they suspect that they may be pregnant) they should avoid all drugs, including those bought at the pharmacy without prescription (even salicylates have come under suspicion¹⁵) unless they are necessary for the saving of life or the prevention of serious disability. Unfortunately good advice is easier to give than to take. The next line of defence is the screening of all new drugs for potential teratogenicity before they are unleashed on the public. But for well known reasons laboratory screening cannot provide complete protection. As a safeguard we must also have an effective early-warning system. This presents difficult problems for which even with ready access to computers there are no ready answers. Another thalidomide-type disaster can and probably will occur. Our hope is to identify and control it much more quickly.

What of the at-one-time promising possibility of uncovering controllable environmental factors in the aetiology of malformations? As we have seen there is good evidence that environmental factors are important in the aetiology of the central nervous system malformations. For example, in South Wales young women of low socioeconomic class living in the mining valleys who are having their first child are at four times the risk of that child being born with a neural tube defect than women of high socioeconomic class living on the coastal plain who are having their second or third child. Some part of the poverty complex is clearly involved, but the contribution of that factor or factors appears to be becoming less—an unplanned and certainly an unexpected bonus from our rising standard of living. The quite large geographical differences may possibly be due to a water factor, certainly in England and Wales they are related to water supplies, but we are a long way from confirming this. The truth of the matter is that everything we so far know about the environmental influences suggests that we are unlikely ever to have much control over them. The lack of concordance in twins, the primogeniture effect, and the early pregnancy losses all indicate that the environmental factors are quite unlike those with which we are familiar in the world outside the womb—city smog, the traffic in the streets, overcrowding, bad working conditions, and polluted water supplies. In that fine and private place, the womb, the environmental influences at work are likely to remain obscure and unpredictable accidents occurring at the time of fertilization or implantation or during early fetal development due in part, perhaps, to minor disturbances of delicately balanced maternal metabolic factors beyond our control.

There remains the hope of more effective treatment. Over the past two decades we have witnessed remarkable advances in the treatment of many congenital malformations, advances made possible by the enterprise of surgeons moving forward under the cover of new antibiotics and improved anaesthetic techniques. And this is only the beginning. Advances in bioengineering, transplants and prostheses will provide increasing opportunity for further surgical enterprise. Some of the major malformations—e.g., cleft lip and congenital dislocation of the hip—have always been compatible with a reasonable life span, and advances in plastic and orthopaedic surgery have now greatly improved the quality of that span. In the case of a few malformations, such as some of the abnormalities of the heart and great vessels and some of the alimentary atresias, new methods of treatment offer the hope of a reasonable span of normal and active life where previously early death was the rule. There are other abnormalities for which medical advances now provide extra years for what in earlier days would have been a very short life. But the years of life given are not in any sense normal, because they are burdened with gross residual handicaps. In South Wales, where the incidence is high, spina bifida raises this dilemma in an acute form. Until quite recently about 85% of liveborn children with myelomeningocele died in infancy or early childhood. With advances in surgery about 60% are now

surviving into school life.¹⁶ Half of these survivors are grossly disabled, incontinent of urine, and walking with great difficulty or not able to walk at all. Medicine in making some sort of life possible for these unfortunate children has presented their families and the society in which they live with the sometimes intractable problem of making that life tolerable.

References

- ¹ Laurence, K. M., Carter, C. O., and David, P. A., *British Journal of Preventive and Social Medicine*, 1967, 21, 146.
- ² Richards, I. D. G., and Lowe, C. R., *British Journal of Preventive and Social Medicine*, 1971, 25, 59.
- ³ Yen, S., and MacMahon, B., *Lancet*, 1968, 2, 623.
- ⁴ Leck, I., McKeown, T., and Edward, J. H., *Teratology*, 1968, 1, 263.
- ⁵ Czeizel, A., and Revesz, C., *British Journal of Preventive and Social Medicine*, 1970, 24, 205.
- ⁶ Halevi, H. S., *British Journal of Preventive and Social Medicine*, 1967, 21, 66.
- ⁷ Neel, J. V., *American Journal of Human Genetics*, 1968, 10, 398.
- ⁸ Stocks, P., *British Journal of Preventive and Social Medicine*, 1970, 24, 67.
- ⁹ MacMahon, B., and Yen, S., *Lancet*, 1971, 1, 31.
- ¹⁰ Leck, I., and Rogers, S. C., *British Journal of Preventive and Social Medicine*, 1967, 21, 177.
- ¹¹ McKeown, T., and Record, R. G., *Lancet*, 1951, 1, 192.
- ¹² Roberts, C. J., Lloyd, S., and Lowe, C. R., *British Journal of Preventive and Social Medicine*. In press.
- ¹³ Nishimura, H., in *Proceedings of Third International Conference on Congenital Malformations, The Hague 1969*, ed. F. C. Fraser and V. A. McKusick, p. 275. Amsterdam, Excerpta Medica, 1970.
- ¹⁴ Lowe, C. R., Roberts, C. J., and Lloyd, S., *British Medical Journal*, 1971, 2, 357.
- ¹⁵ Richards, I. D. G., *British Journal of Preventive and Social Medicine*, 1969, 23, 218.
- ¹⁶ Cook, C. M., *British Medical Journal*, 1971, 2, 796.

Emotional Problems of Childhood and Adolescence

How to Use Child Psychiatry*

J. D. HALDANE

British Medical Journal, 1972, 3, 520-522

To guide and advise the family doctor in his use of child psychiatry services, of community resources for children and young people, or in his decision whether to refer a child or adolescent for inpatient psychiatric treatment, is a task hampered by the lack of universally agreed criteria. This lack of agreement reflects several factors which affect the organization, policy, and practice of present psychiatric services for children and adolescents. In Scotland most consultants in this specialty work on a full-time basis in the National Health Service; to a greater or lesser degree provide the psychiatric services to this age group in a defined area; and have no formal appointments to local education authority child guidance clinics. In England and Wales, on the other hand, many psychiatrists work in child guidance clinics and many have sessional appointments in two or more agencies. Some child psychiatrists are based on paediatric, others on psychiatric, hospitals, and appointments to mental subnormality hospitals are being developed. Some child psychiatry services accept only those patients referred by medical colleagues, others also from non-medical agencies.

Services are uneven in distribution throughout the country and vary in staffing resources, facilities available, policy about the type of children and adolescents accepted for assessment and treatment, and in the range of treatments offered. It is rare for a department of child psychiatry to be responsible for the psychiatric services for the age group up to the age of 18 years or for any one department to have inpatient facilities for this whole age group. There is also a shortage of places in children's homes, residential schools for the handicapped and mal-

adjusted, hostels, approved schools, and borstals. In all of these are found young people of the type regularly referred to psychiatric services for children and adolescents and many psychiatrists have commitments to such institutions.

The child psychiatrist sees his patients only at the request of another professional colleague, his authority for assessing or treating a child being given him by this request and, he hopes, by child and parents. Clearly, just as referral is often still only a "last resort" so the necessary authority is often given reluctantly, without expectation of "success" even by the referring family doctor. While the psychiatrist must define for himself the nature and limits of his professional responsibility, he has an accountability to patients and their parents and also to colleagues. Some family doctors expect the child psychiatry service to undertake the whole responsibility of seeing child and family through a process of assessment and treatment, even if this involves non-medical agencies and do not expect to be regularly informed of progress or lack of it. Others expect to be kept informed of progress, though not all consider that they should keep the psychiatrist informed. Some family doctors expect advice on what they might do in exercising their responsibility for primary care; others resent such advice. These attitudes, and frequent comments from psychiatrists and family doctors about lack of adequate communication, express different and not easily reconcilable views of both child psychiatry and general practice. It would be helpful to both if the professionals concerned were to consider this a serious issue worth discussing, in order to reach some measure of agreement in each local situation.

Non-psychiatric Services

Child psychiatry services represent only one of the many services, agencies, and organizations responsible for providing help to children, adolescents, and parents in need and see only a minority of the young people who might be referred to them. The family doctor has a choice: to refer a child to a non-psychiatric service, as a decision of preference, or as a step towards referral to a psychiatric service. He has both a responsibility to know something of the non-psychiatric services in his

*Based on a paper read to a Symposium on "The Disturbed Adolescent," 16 April 1972, to the Dunbartonshire Division of the B.M.A.